Essentials of FACIAL GROWTH

Second Edition

DONALD H. ENLOW, M.S., Ph.D
Thomas Hill Distinguished Professor Emeritus
Department of Orthodontics
Case Western Reserve University
Cleveland, Ohio

MARK G. HANS, D.D.S., M.S.D.
Professor and Chairman
Department of Orthodontics
Director, Bolton-Brush Growth Study Center
Case Western Reserve University
Cleveland, Ohio

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CONTRIBUTORS

ROLF BEHRENTS, D.D.S., M.S., Ph.D.
Professor and Chairman
Department of Orthodontics
Saint Louis University, St. Louis, Missouri
Adult Craniofacial Growth

ALAN BOYDE, Ph.D.
Hard Tissue Research Unit, Dental Biophysics
Centre for Oral Growth and Development, Institute of Dentistry
Barts and The London School of Medicine and Dentistry
Queen Mary University of London, New Road., London, England
Bone Growth Remodeling of the Early Human Face

B. HOLLY BROADBENT, D.D.S.
Director Emeritus, Bolton-Brush Growth Study Center
Case Western Reserve University, Cleveland, Ohio
Craniofacial Imaging

TIMOTHY BROMAGE, Ph.D.
Hard Tissue Research Unit, Departments of Biomaterials & Basic Sciences
New York University College of Dentistry, New York, New York
Bone Growth Remodeling of the Early Human Face

DAVID S. CARLSON, Ph.D.
Vice President for Research and Graduate Studies
Texas A&M Health Science Center, Dallas, Texas
Maturation of the Orofacial Neuromusculature

EEMAN DAJANI, B.D.S., M.S.
Private Practice, Tipp City, Ohio
Intrinsic Biological Basis for Accurate Prediction of Facial Growth

JOS DIBBETS, D.D.S., Ph.D.
Professor and Chair, Department of Orthodontics
Philipps University, Marburg, Germany
Introduction to the Temporomandibular Joint

DANA DUREN, Ph.D.
Lifespan Health Research Center, Boonshoft School of Medicine,
Wright State University, Kettering, Ohio
Genetics of the Craniofacial Complex

ROBERT E. MOYERS, DDS., Ph.D.*
Professor Emeritus, School of Dentistry and Director Emeritus
Center for Human Growth and Development
University of Michigan, Ann Arbor, Michigan
Maturation of the Orofacial Neuromusculature

J. MARTIN PALOMO, D.D.S., M.S.D.
Associate Professor, Department of Orthodontics, and Director of the Craniofacial Imaging
Center, School of Dental Medicine
Case Western Reserve University, Cleveland, Ohio
Craniofacial Imaging

RICHARD SHERWOOD, Ph.D.
Lifespan Health Research Center, Boonshoft School of Medicine
Wright State University, Kettering, Ohio
Genetics of the Craniofacial Complex

MEROPI N. SPYROPOULOS, D.D.S., M.S., Ph.D.
Professor Emeritus, School of Dentistry
University of Athens, Athens, Greece
Mechanobiological Perspectives of Orthodontic Tooth Movement Related to Bone Physiology

APOSTOLOS I. TSOLAKIS, D.D.S., M.S.D., Ph.D.
Research Associate, School of Dentistry
University of Athens, Athens, Greece
Mechanobiological Perspectives of Orthodontic Tooth Movement Related to Bone Physiology

To Martha (DHE)
and
Susan (MGH)

Preface to the Second Edition

It has been more than 10 years since the first publication of *Essentials of Facial Growth*. During the first half of this decade changes in clinical orthodontics were largely driven by improvements in bracket design, attachment mechanisms and arch wire materials. Unfortunately, this focus on the mechanical aspects of treatment led to decreased clinical interest in the underlying biology of facial growth and, for a time, craniofacial practitioners often ignored the *bio* in biomechanics. There was even more disappointment after the completion of the Human Genome Project. Although this project promised exciting possibilities for biologically based manipulation of the growing face, the resulting data was interesting but in most cases not clinically useful. In the second half of this decade, two advances in clinical orthodontics fueled increased interest in craniofacial biology and facial growth. The first was the introduction of low cost, high resolution, computed tomographic imaging. This technology provides researchers and clinicians with the tools needed to study three dimensional changes in craniofacial anatomy associated with the growth process and clinical care. In the 20th century, radiographic cephalometry was a pioneering advance that led to many fundamental insights into the behavior of the face and neurocranium during growth. CBCT imaging promises similar advances in the 21st century. However, to take advantage of this opportunity, clinicians will need to interpret 3D anatomic changes in the context of the underlying growth process. Another clinical advance that has stimulated interest in craniofacial biology and facial growth is the use of temporary anchorage devices (TADs). TADS give the clinician the ability to move teeth in any direction. With greater control comes increased responsibility. Clinicians using TADs need to know how teeth drift naturally within the face and how TADs influence this drift. To achieve optimal results the modern clinician is compelled to study craniofacial biology in general and facial growth in particular.

This Second Edition of *Essentials of Facial Growth* is designed to meet the needs of the craniofacial clinician as well as the student of craniofacial biology. The book has been totally updated and important chapters have been added on timely topics in craniofacial biology. All of the chapters that were included in the First Edition have been extensively updated and are included in the second edition. The Second Edition is divided into two parts. Part One-Enlow's Essentials-focuses on the life work of Don Enlow and his classic and timeless characterization of growth fields of the face. In addition to Enlow's histologic and anatomic descriptions we have attempted to frame the concepts in a clinical context. Thus, the beginning clinician can use Part One as a comprehensive course in the biology of facial growth and as an introduction to the clinical manipulation of the human face. Part Two-More Essentials-includes chapters on the TMJ and Adult Facial Growth that were first published as part of Enlow's *Facial Growth 3rd Edition*. These chapters were not included in the first edition due to publishing constraints but are back by popular demand in the Second Edition. This material has been updated and represents the current state of the art. In addition, the chapter on imaging has been totally rewritten to reflect the impact

of 3D technology on the field. Other new chapters focus on early hominid growth, tooth movement, genetics, muscle maturation and growth prediction. The authors in Part Two are all experts in their field and represent a modern Who's Who in the area of growth and development. We are proud to have collaborated with this fine group of contributors to create what we feel is the most complete reference text on human facial growth available.

The preparation of this updated and expanded edition has been strengthened by the talents and gracious tolerance of our secretary, Mrs. LaVerne Vogel. We would also like to acknowledge the technical assistance and cover design talents of Dr. J. Martin Palomo. We all value and appreciate the professional expertise and helpful collaboration provided by Susan Leonard of Typesetting and Book Design, Dr. Bruce Tracy of Indexing Services and the good folks at Needham Press.

THE PATH

This is the story, in a few short paragraphs, of studies that proceeded along a twisting and turning research path. They were about how the remodeling process in growth works, and they took more than fifty years. We will look at the bone tissues of fossils who were alive and growing many millions of years ago. We will go through ancient geologic history on to modern vertebrates, and finally to the human face itself. The findings showed that bone structure and the mode of growth of whole bones vary widely. How the growth process does this is unknown. If the manner of the developmental operation can be understood, the growth of bones can be used as indicators to explain how complex morphological assemblies, such as the face, can be better understood. Bright students will properly ask: How in the world can looking at old fossil bones possibly lead to understanding how the human face grows? Here is the story of the unique research path that pulled this off. For me personally, it has been a most interesting and exciting fifty year adventure. I hope you will share that interest and excitement with me.

One particular working biological process was found on this Path almost immediately. From the start it was always in play. That process is "remodeling". It is so important and so heavily involved that we now recognize it as a major tool in how "facial growth" operates. It's manner of function is far more elaborate and extensive that any of its old and simplistic perceptions. Awareness of the significance of the remodeling process in growth, for me, first began to emerge from an article I wrote long ago for our Natural History Museum titled "Written in the Rocks: An Autobiography of Texas". The message was that the earth's rocky crust undergoes constant rebuilding into ever-changing patterns of stratified layers through an ongoing process of cyclic erosion and sedimentary deposition. This geology can be "read" from the nature of the changing stratification patterns and analyses of the circumstance-specific content of each layer. This yields a successful *history* and precise *understanding* of the earth's "growth" through time. The analogy with bone growth is remarkable in many ways. As in inorganic rock, rock-like bone undergoes a constant, quite extensive rebuilding process necessary for its enlargement during growth. This rebuilding process, termed "remodeling" by the biologist, produces changing, stratified layers composed of specific varieties of different basic kinds of bone tissue. In bone as in geology, the nature of the sequencing and the details of patterns of the changing layering tells the story of what happened. Also in bone as in geology, each variety of content in each layer provides biological insight for the multitude of circumstances existing at the time of formation. All of his now presents an effective means to "read" the growth history of each bone, and in composite study of all the bones together, a precise way to accurately reconstruct the development of the entire craniofacial complex. Beautiful. This had never been done before.

To begin this story of what happened and what the key events were on the Path, it started soon after World War II ended, I left the Service and started all over again in college changing to a pre-med major. I took Biology as required, and that particular

subject hit me like a freight train. It opened my eyes to a fantastic, whole new world. I found that everything in Biology is purely fascinating, exciting, and absolutely compelling. Changing my major to pure Biology, I took every course in the catalog. Soon I found one particular professor who was, in every way, the dynamic kind of scholarly professor I wished to be. I signed up for all the courses he presented… Comparative Anatomy, Embryology, Histology, Vertebrate Paleontology… Oh brother, was I ever hooked. He was a paleontologist and had done his Harvard doctorate under Al Romer, the Modern Father of Paleontology.

After receiving my Master's degree I began teaching in the Department, and with "My Professor", did extensive fossil field prospecting all over the widespread Permian and Triassic geologic outcroppings of West Texas. These Permocarboniferous Red Beds go back 225 million years ago. I had become a paleontologist. I had also done a minor at the University in geologic sedimentary petrology, which proved to be a key factor on my quest and was about to pay off. During one particular fossil expedition, I was looking over some bone fragments I just found, and a small piece of a neural spine of Edaphosaurus (note to typist; please underline or italicize), an early mammal-like reptile, really caught my interest. I thought I could make out some **structure** in its broken end. I asked My Professor if ground sections of fossil bone have ever been made. "I don't think so" was the response with a negative head shake, and "why don't you try it?"

Well, back at the lab I did just that. And what I saw just absolutely FLOORED me. I tell you I was just ASTOUNDED. I remember that my hands were shaking as I stared at that first section for long minutes, almost disbelieving. Most of the key histology was there. I was transfixed in silence, pondering the compact and cancellous areas, the lacunae, the lamellae and the vascular canals. Great Heavens, can you believe, there were even elaborate Canaliculi flowering out from each lacuna, perfectly and beautifully intact. I could not help but think that what I was seeing was just impossible. After all, I was looking at bone tissue over 200 million years old, TWO HUNDRED MILLION YEARS OLD. Yet I had to believe my eyes. I was seeing something that no one had ever seen. Yes, profoundly exciting. I think it must have been something like an explorer's feeling when discovering something like a new continent. Big!

It hit me hard that there were no Haversian Systems. Like every other histologist in the world, I had been duped into believing that Secondary Osteons were the fabled "units" of bone. But importantly, even more than that, the organization of everything I saw in that first section was quite different from anything in the textbooks. At this point I was beginning to see that there are important things about bone we surely don't understand. I scrounged around the Department and found some bones from several different vertebrate species and made ground sections. Then, indeed, I realized I was truly on some kind of Path, but I had no ideas about this path and what was beginning to happen along it. What I found were beautiful histologic patterns. Again, all of them were quite different from all of the others. I made more sections from more bones and more species. Yes, everything was all different. I just had one thought, "what is happening?" I intended to find out. But before I could really start serious work there was a survival need I must attend to. Now it was clear that an Academic Career was my goal. To hope to get a permanent academic position anywhere, I must first succeed in entering an appropriate university and complete the Doctorate degree. I did this, and the time invested and adventure of the experience were truly exciting, This doctorate-level experience did much to shape me into an "academic",

and providing for me so much of the academic and scholarly attitudes and skills needed in this business.

I had begun a particular career direction, but until beginning the Doctorate and my realization that I had to get down to serious business, I had just been on a marvelous lark having a young man's great time looking for dinosaurs. I did not realize that I had entered, unexpectedly, a long research road which I did not realize could end up with a working understanding of how the vertebrate face, especially the complex human craniofacial assembly, grows and develops. What would transpire over full five decades would be a continuing series of random career events that happened to keep nudging me along this relentless research course. But without these particular, unrelated "happenings", the whole thing would come apart. If that happened, then our BIOLOGICAL understanding of how the human face and neurocranium grow and develop would very likely have remained back at the old 1946 level.

Let me briefly interrupt this story of The Path and the quest along it. I wish to point out a serious problem existing in orthodontics and why it is important. The next two paragraphs outline this disturbing situation, and why The Path, going somewhere, is truly significant. The idea is so you know I did not have any idea at all what was going to happen, or where, if anywhere, the Path would go. After this, so you have a handle on a big problem, then we will return to the Path.

What virtually everybody has thought about Facial Growth and what largely continues today in Modern Orthodontics is essentially a non-biologic package First, it was emphasized that the face and all of its component parts grow "Forward and Downward". Hard to believe, but that timeless cliche is STILL taught, without apology, in almost all universities, day after day. Now, look at the problems that have resulted. It is still insisted that the mystical mandibular condyles especially control virtually everything about mandibular growth. This has been a rock-solid presumption, yet everybody also agrees that the condyles grow "Upward and Backward". This is another basic conflict that has caused endless confusion temper eruptions. Further, sutures were solidly presumed to be important, proactive "growth regulating centers", just as the condyles were also without question presumed to be. But if the sutures are surgically removed in an experiment, everything nonetheless develops naturally as though nothing happened. What? Note the conflict. Here is another big, and quite major, problem. All bones are presumed to grow by periosteal deposition and endosteal resorption. This is always regarded as the rule, and its simplistic nature allows everyone to "understand" how bones of the face grow without understanding how "remodeling" works to size and shape each bone. Yet, about half of all the periosteal surfaces in the face are actually resorptive, not depository. It is an enigma and seemingly impossible that a bone can enlarge even though extensive outer surfaces are resorptive. Yet, as seen throughout this book, that is exactly how remodeling works. Another conflict. There is another major, very significant biologic fact that almost never has been taken into account. It concerns an old presumption long regarded as the fundamental-most tenent in bone biology and deals with growth effects of muscle traction on bone. The idea holds that muscles pulling on bone induces bone deposition, and this dangerously incomplete idea has always been the major thrust in explaining how "growth" works. But so many key muscles in the face attach onto **resorptive** surfaces that this simplistic notion falls quite short. Yet serious study to resolve it and then proceed in a corrected direction, is truly major need but falls on deaf ears among the biomechanical

researchers. Muscles can routinely insert on either a resorptive or depository surface and in the same bone, some muscles attach to both at the same time. The nature of the complex insertion of the Temporalis muscle is virtually never considered, yet part of its insertion on the coronoid process is on a resorptive surface, and another part is on a depository surface. The conflict is apparent. Another clinical shortfall is that Cephalometrics has been a useful and tremendously used tool in attempts to understand how the face grows. Considering the extreme complexity of craniofacial anatomy, it is indeed has been a daunting and absolutely essential tool in the absence of understanding how the biological process of growth operates. However, Sella-Nasion is a contrived (not natural) radiographic plane without any significant anatomical meaning relating to facial development, yet it was adopted long ago as the vital centerpiece for analyzing almost everything, including how the face grows. It cannot represent with anatomic reality either the "cranial base" or the "upper face", yet it has been presumed almost daily to do both. Further, it is mid-line, yet the condyles, maxillary tuberosities, major basicranial and cerebral relationships, etc. are all bilateral in placement. Attempting to understand facial growth, this line is still in everyday use. More than just ineffective, it is a genuine block that precludes real biologic understanding.

So, very little true and real biological understanding about how the face grows was known, continuing decade after decade. The non-biologic perceptions held worldwide were all that existed to try to develop meaningful cephalometric procedures and to design treatment procedures and materials. The old, extraordinarily simplistic teaching of Facial Growth, much of it incorrect or misleading, virtually cemented those ideas into standard orthodontic principles. Unfortunately, the many purely non-biologic facial growth perceptions today are the basis for nearly all of orthodontics as presumed to be built on "growth". A central point is this; Virtually everything once presumed to be reasons why clinicians must "Work With Growth" (an old orthodontic cliche) were obsolete perceptions that existed BEFORE there was a working understanding of craniofacial development. Thus, orthodontics as a clinical discipline is markedly out of date. I do not see a groundswell of interest throughout orthodontics that we should do what badly needs to be done. This greatly saddens me, and I do not understand it.

My Doctoral Dissertation was a comprehensive, comparative study of the earliest bone tissues in fossils and all major vertebrate groups, fossil and modern This went all the way back to the early Cambrian period, over 500 millions of years ago, and the primitive jawless fishes that existed then. And then back up through the Geologic Periods, with each period including major fish groups, amphibians, reptiles, birds, and all mammalian classes. Everything was described and evaluated. A major finding was that, again, every histologic section was basically different from every other section. I was at the University of Michigan School of Medicine then, teaching histology and embryology year after year after year. This really served to establish in my thinking, the basic "ground rules" for the REASONS for the distribution of tissues throughout the body. This was yet another factor in pushing me along on "The Path". In dental classes I found myself teaching how "Mesial Drift" of the developing dentition works. That subject requires that one be able to recognize "depository" and "resorptive" bone surfaces, and this led to understanding the mode of formation of all the various "periosteal and "endosteal" types of bone tissue. Like everything else on this Path, this was an essential step and had to occur. My geologic training in Sedimentary Petrology demonstrated how to interpret multiple erosion/

deposition patterns seen in sedimentary cycles to the variety of complex stratification patterns in bone. And also, importantly, how to read and interpret underlying meanings of the histologic composition and the histogenic sequencing of the layers in bone. All of these factors were combining to begin explaining WHY bone growth behaves as it does during the formations of the seemingly endless histogenic variations among all of the bones in all of the different vertebrate groups, and finally, in the human face and neurocranium. THERE MUST BE SOME KIND OF "RULES BOOK" that underlies the reasons determining the widely varied histologic patterns of bone everywhere. This cannot simply be random differences that have no organizing causes. So, my goal was to find out what the basic Rules are, and why.

But the "Rules" determining bone tissue microstructure, for so many years the baffling puzzle I just described, have now at last been worked out and I think are fairly well understood. They closely parallel geologic systems of sedimentation, and to biologists are known by our old, long used term of "Remodeling", but greatly expanded beyond the old, simplistic understanding. The developmental principle involved is quite simple. When this principle hit me I can't recall. It was not like a clap of thunder, but whenever and however, the title I've given to it is simple and descriptive… just as simple as how the principle itself is so straightforward. This key developmental process is "Area Relocation", and how it works is described next. It is the fundamental reason all bones must Remodel as they grow. It fully explains the seemingly endless diversity seen in the microscopic structure of bone.

Bones generally grow by adding new bone at their proximal and distal ends together with remodeling everywhere in between. Let's say that the proximal end of a bone is P1 and the distal end is D1. When the whole bone lengthens, P2 is added onto P1, and D2 is added at D1. Now, the old ends P1 and D1 became "Relocated" from their former end positions to New positions. Neither actually "moves"; this is a RELATIVE movement. But the CONFIGURATION and the SIZE of both P1 and D1 become changed, and both are Remodeled to accommodate their relative movements and changed conditions. Now, repeat this exchange system in all parts of a growing bone. That is, add P3 and D3, then P4 and D4, and so on. This continuous growth process is the KEY to understanding histological pattern variations of bone structure. It is termed AREA RELOCATION. The growth ENGINE that carries it out is the resorptive and depository process of REMODELING. The relative movements of Relocation are carried out by Remodeling sequentially along the entire length of every bone as they grow.

To complete the body's growth system for (1) each individual bone (mandible, maxilla, etc.), and (2) all of them growing together in concert, another essential, fundamental process produces their MOVEMENTS and composite PLACEMENTS into functional, perfect fitting positions. This is the important DISPLACEMENT process. It positions all the separate bones into functional skeletal composites (e.g., the nasomaxillary complex) as they simultaneously undergo Remodeling. Now add the widespread SOFT TISSUE growth systems controlling skeletal development. Clinicians enter into the body's intrinsic growth control systems in order to introduce clinical signals that override the body's own systems in order to produce desired results by manipulating the Remodeling and Displacement processes. Remodeling and Displacement are the two key, interrelated developmental processes that carry out Growth and Development. They are the fundamental ENGINES that drive the Growth Process.

At this point all of the principal "tools" of the growth process as seen and interpreted in bone were functionally in place. The development of the face and neurocranium could now be accurately traced through detailed analyses of the "geologic formations" recorded in the substance of the bones. The body records its own growth in the substance of its bones, and by understanding the "language" used, the manner of the craniofacial growth process itself can be determined. We were then in a position to study the sedimentary cycles of the bone tissues throughout the growing human face, and by determining through analyses of RELOCATIONS of all parts of all the individual bones of the whole head, we could accurately determine how each separate bone develops through childhood to adult. Then by analyzing how each bone becomes precisely DISPLACED into all of the groupings of all the separate bones, the overall development of the entire facial and neurocranial composite could be fully worked out. This we did, and the results of this 50 year research study are presented in the chapters of this new book edition.

Don Enlow
June 1, 2007

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Part 2: More Essentials of Facial Growth

PROLOGUE

Historical Perspective

The morphogenic *interrelationships* among the diverse families of soft tissues and the growth and development of the craniofacial skeleton are highlights of the present monograph. However, the early historical story of "bone" is especially interesting because of bone's unusual "oxymoron" character. That is, how in the world can this unique rock-like substance actually grow and develop into constantly changing shapes and sizes *perfectly* matching the developing soft tissues it serves. This question festered in early scholars' minds and has been a particular wonderment since Biblical times. In the book of Ecclesiastes (11:5), for example, it was said that "As thou knowest not the way of the spirit, nor how the bones do grow in the womb of her that is with child, even so thou knowest not the works of God who maketh all." Many Old and New Testament passages make frequent reference to bone in health and disease. That bone is actually a living substance is certainly not a modern notion at all. Greek philosophers and physicians, including Hippocrates (*De carnibus*), Aristotle (*De generatione animalium*), Galen (*Opera omnia*), and Plato (*Timaeus*), all recorded allegories on bone formation, describing how the earthy, less fluid, and thicker seminal parts become solidified by internal body heat, comparable to the manner in which moist clay (i.e., cartilage) is kiln-fired (endochondral ossification) into earthenware. A reasonable analogy at the time, considering that microscopes, histology textbooks, and the Cell Doctrine were in the distant future. Arnobius accounted for the control of childhood bone formation by a goddess "who hardens and solidifies the bones in infants." Then, century by century, many of the great names in the anatomy and medicine Hall of Fame assembled our foundations of bone knowledge in a long series of plateaus, each following some technological advance or conceptual breakthrough. These familiar names include Albinus, Vesalius, Bartholin, Harvey, Sue, Havers, Nesbitt, Monro, Leewenhoek (who observed canals in bone years before Havers, but that doesn't detract from the latter's classic monograph "*Osteologia Nova*"), Todd, Bowman, Tomes, Demorgan, Von Ebner, Gagliardi, Malpighi, Bell, Howship, Belchier, Hales, Hunter, Volkmann, Wolff, Hassall, Meckel, Virchow, Purkinje, Sharpey, and Schwann. All, and many more, were directly involved in the quest. (See Enlow, 1963, for a more extensive historical review, including the specific landmark contributions of these early scholars.)

Part 1

Enlow's Essentials of Facial Growth

1

Overview of Craniofacial Growth and Development

"Growth" is a general term implying simply that something changes in magnitude. It does not, however, presume to account for **how** it happens. For the professional clinician, such a loose meaning is often used quite properly. However, to try to understand "how" it works, and what actually happens, the more descriptive and explanatory term "development" is added. This connotes a maturational process involving progressive differentiation at the cellular and tissue levels, thereby focusing on the actual biologic mechanism that accounts for growth.

"Growth and development" is an essential topic in many clinical disciplines and specialities, and the reason is important. Morphogenesis is a biologic process having an underlying **control** system at the cellular and tissue levels. The clinician intervenes in the course of this control process at some appropriate stage and substitutes (augments, overpowers, or replaces) some activities of the control mechanism with calculated clinical regulation. It is important to understand that the actual biologic process of development itself is the same. That is, the histogenic functioning of the cells and tissues still carry out their individual roles, but the **control signals** that selectively activate the composite of them are now clinically manipulated. It is the rate, timing, direction, and magnitude of cellular divisions and tissue differentiation that become altered when the clinician's signals modify or complement the body's own intrinsic growth signals. The subsequent course of development thus proceeds according to a programmed treatment plan by "working with growth" (an old clinical tenet). Of course, if one does not understand the workings of the underlying biology, any real grasp of the actual basis for treatment design and results, and why, is an illusion. Importantly, craniofacial biology is independent of treatment intervention strategy. Therefore, although some clinicians may argue about the relative merits of different intervention strategies (e.g., extraction versus arch expansion). The biologic rules of the game are the same.

Morphogenesis works constantly toward a state of composite, architectonic balance among all of the separate growing parts. This means that the various parts developmentally merge into a functional whole, with each part complementing the others as they all grow and function together.

During development, balance is continuously transient and can never actually be achieved because growth itself constantly creates ongoing, normal regional imbalances. This requires other parts to constantly adapt (develop) as

they all work toward composite equilibrium. It is such an imbalance itself that fires the signals which activate the interplay of histogenic responses. Balance, when achieved for a time, turns off the signals, and regional growth activity ceases. The process recycles throughout childhood, into and through adulthood (with changing magnitude), and finally on to old age sustaining a changing morphologic equilibrium in response to ever-changing intrinsic and external conditions.

For example, as a muscle continues to develop in mass and function, it would outpace the bone to which it inserts, both in size and in mechanical capacity. However, this imbalance signals the osteogenic, chondrogenic, neurogenic, and fibrogenic tissues to immediately respond, and the whole bone with its connective tissues, vascular supply, and innervation develops (undergoes remodeling) to work continuously toward homeostasis.

By an understanding of how this process of progressive morphogenic and histogenic differentiation operates, the clinical specialist thus selectively augments the body's own intrinsic activating signals using controlled procedures to jump-start the remodeling process in a way that achieves an intended treatment result. For example, in patient's with maxillary transverse deficiency rapid palatal expansion can be used to separate the right and left halves of the maxilla (displacement. This in turn initiates a period of increased remodeling activity in the midpalatal suture and dentoalveolus.

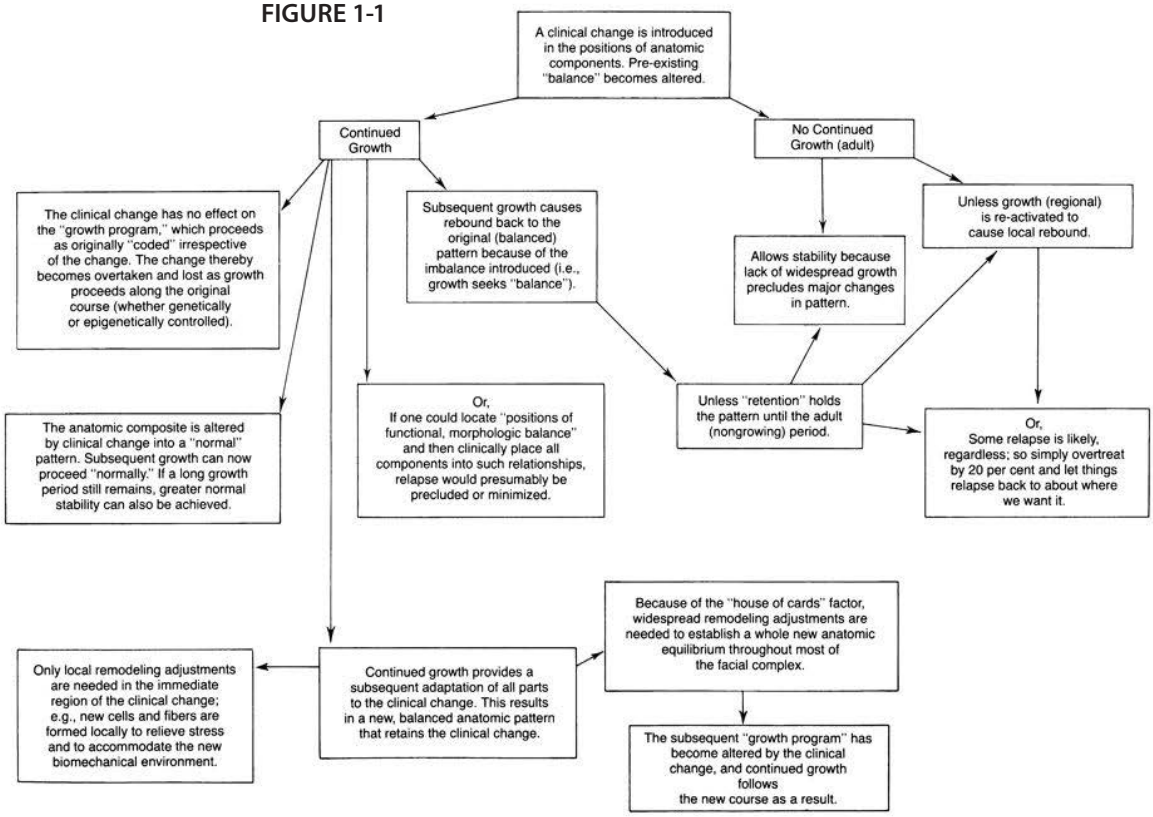
The genetic and functional determinants of a bone's development (i.e., the origin of the growth-regulating signals) reside in the composite of soft tissues that turn on or turn off, or speed up or slow down, the histogenic actions of the osteogenic connective tissues (periosteum, endosteum, sutures, periodontal membrane (aka periodontal ligament)). Growth is not "programmed" within the bone itself or its enclosing membranes. The "blueprint" for the design, construction, and growth of a bone thus lies in the muscles, tongue, lips, cheeks, integument, mucosae, connective tissues, nerves, blood vessels, airway, pharynx, the brain as an organ mass, tonsils, adenoids, and so forth, all of which provide information signals that pace the histogenic tissues producing a bone's development.

A major problem with therapeutic modification of the growing face can be relapse (rebound subsequent to treatment). The potential for relapse exists when the functional, developmental, or biomechanical aspects of growth among key parts are clinically altered to a physiologically imbalanced state. The possibility of instability exists because clinicians strive to bring about a state of aesthetic balance that at times produces physiologic imbalance. Rebound is especially strong when the underlying conditions in the genic tissues that led to the pretreatment dysplasia still exist and thus trigger the growth process to rebound in response to the clinically induced changes in morphology. The "genic" tissues (see below) are attempting to restore physiologic balance,* thereby returning in a developmental direction toward the pretreatment state or some combination between. Physiologic compensation is, in effect, a built-in protective mechanism that allows the final occlusion of the teeth to vary only a mere 6mm despite enormous variation in the human face (See Fig. 1-1).

* A malocclusion or other dysplasia (including congenital malformations), although clinically abnormal, is nonetheless in a "balanced" state.

The evolutionary design of the human head is such that certain regional clinical situations naturally exist. For example, variations in headform design establish natural tendencies toward different kinds of malocclusions. The growth process, in response, develops some regional imbalances, the aggregate of which serves to make corrective adjustments. A Class I molar relationship with an aesthetically pleasing face is the common result in which the underlying factors that would otherwise have led to a more severe Class II or III malocclusion still exist but have been "compensated for" by the growth process itself. The net effect is an overall, composite balance.

FIGURE 1-1



As pointed out above, clinical treatment can disturb a state of structural and functional equilibrium, and a natural rebound can follow. For example, a premature fusion of some cranial sutures can result in growth-retarded development of the nasomaxillary complex because the anterior endocranial fossae (a template for midfacial development) are foreshortened, as in the Crouzon or Apert syndromes. The altered nasomaxillary complex itself nonetheless has grown in a balanced state proportionate to its basicranial template, even though abnormal in comparison with a population norm for esthetics and function. Craniofacial surgery has disturbed the former balance, and some degree of natural rebound can be expected. The growth process attempts to restore the original state of equilibrium, since some extent of the original underlying conditions (e.g., the

basicranium) can still exist that were not, or could not be, altered clinically. These are examples in which the biology of the growth process is essentially normal, either with treatment or without, but is producing abnormal results because of altered input control signals.

The Big Picture

The following paragraph outlines a growth concept basic to the overall developmental process. It deals with the separate but **interrelated** and **interdependent** nature of the assembly of all the regional parts comprising the neurocranium (for the brain and associated sensory organs) and viscerocranium (face). It underscores the variety of developmental conditions in any given local region, but at the same time points to the necessary morphogenic and functional interplay among them.

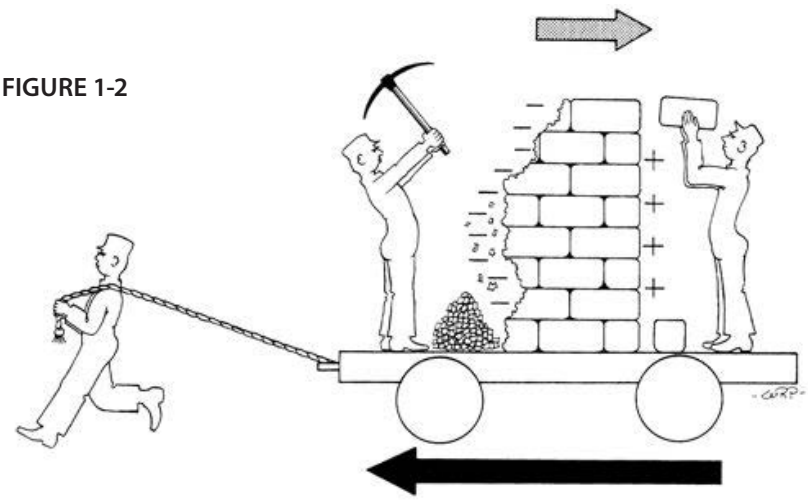
No craniofacial component is developmentally self-contained and self-regulated. Growth of a component is not an isolated event unrelated to other parts. Growth is the composite change of all components. While this seems self-evident, it might be perceived, for example, that the developing palate is essentially responsible for its own intrinsic growth and anatomic positioning, and that an infant's palate is the same palate in the adult simply grown larger. The palate in later childhood, however, is not composed of the same tissue (but with more simply added), and it does not occupy the same actual position. Many factors influence (impact) that growing palate from without, such as developmental rotations, displacements in conjunction with growth at sutures far removed, and multiple remodeling movements that relocate it to progressively new positions and adjust its size, shape, and alignment continuously throughout the growth period. Similarly, for the mandible, the multiple factors of middle cranial fossa expansion; anterior cranial fossa rotations; tooth eruption; pharyngeal growth; bilateral asymmetries; enlarging tongue, lips, and cheeks; changing muscle actions; headform variations; an enlarging nasal airway; changing infant and childhood swallowing patterns; adenoids; head position associated with sleeping habits; body stance; and an infinite spread of morphologic and functional variations, all have input in creating constantly changing states of structural balance. As emphasized above, **development** is an architectonic process leading to an aggregate state of structural and functional equilibrium, with or without an imposed malocclusion or other morphologic dysplasia. Very little, if anything, can be exempted from the "big picture" of factors affecting the operation of the growth control process, and no region can be isolated. Meaningful insight into all of this underlies the basis for clinical diagnosis and treatment planning. Ideally, the target for clinical intervention should be the control process regulating the growth and development of the component out of balance. However, gaps in our understanding of these processes limit the clinician's ability to treat malocclusions in this manner. Since cause is unknown, clinician's target the effect of the imbalance. Therefore, a thorough understanding of the process and pattern of facial growth serves as the foundation for craniofacial therapies.

A Cornerstone of the Growth Process

A grasp of how facial growth operates begins with distinction between the two basic kinds of **growth movement**. These are (1) remodeling and (2) displacement (Fig. 1-2). Each category of movement involves virtually all developing hard and soft tissues.

For the bony craniofacial complex, the process of growth **remodeling** is paced by the composite of soft tissues relating to each of the bones. The functions of remodeling are to (1) progressively create the changing **size** of each whole bone; (2) sequentially **relocate** each of the component regions of the whole bone to allow for overall enlargement; (3) progressively **shape** the bone to accommodate its various functions; (4) provide progressive fine-tune **fitting** of all the separate bones to each other and to their contiguous, growing, functioning soft tissues; and (5) carry out continuous structural adjustments to **adapt** to the intrinsic and extrinsic changes in conditions. Although these remodeling functions relate to childhood growth, most also continue on into adulthood and old age in reduced degree to provide the same ongoing functions. This is what in freshman histology is meant when it is stated that bones "remodel throughout life," but without an explanation of the reasons. Added to this, now, is that all soft tissues *also* undergo equivalent changes and for all of the same reasons.

FIGURE 1-2



In Figures 1-3 and 1-4, note that many external (periosteal) surfaces are actually resorptive. Opposite surfaces are depository. This is required in order to sculpt the complex morphology of the facial bones.

As a bone enlarges, it is simultaneously carried away from other bones in direct articulation with it. This creates the "space" between bones and allows bony enlargement to take place. The process is termed **displacement** (also called "translation"). It is a physical movement of a whole bone and occurs while the bone simultaneously models by resorption and deposition. As the bone enlarges in a given direction within a bony interface, it is simultaneously displaced in the **opposite** direction (Fig. 1-5). The relationships underscore why facial articulations (sutures and condyles) are important factors; they are often direct clinical targets.

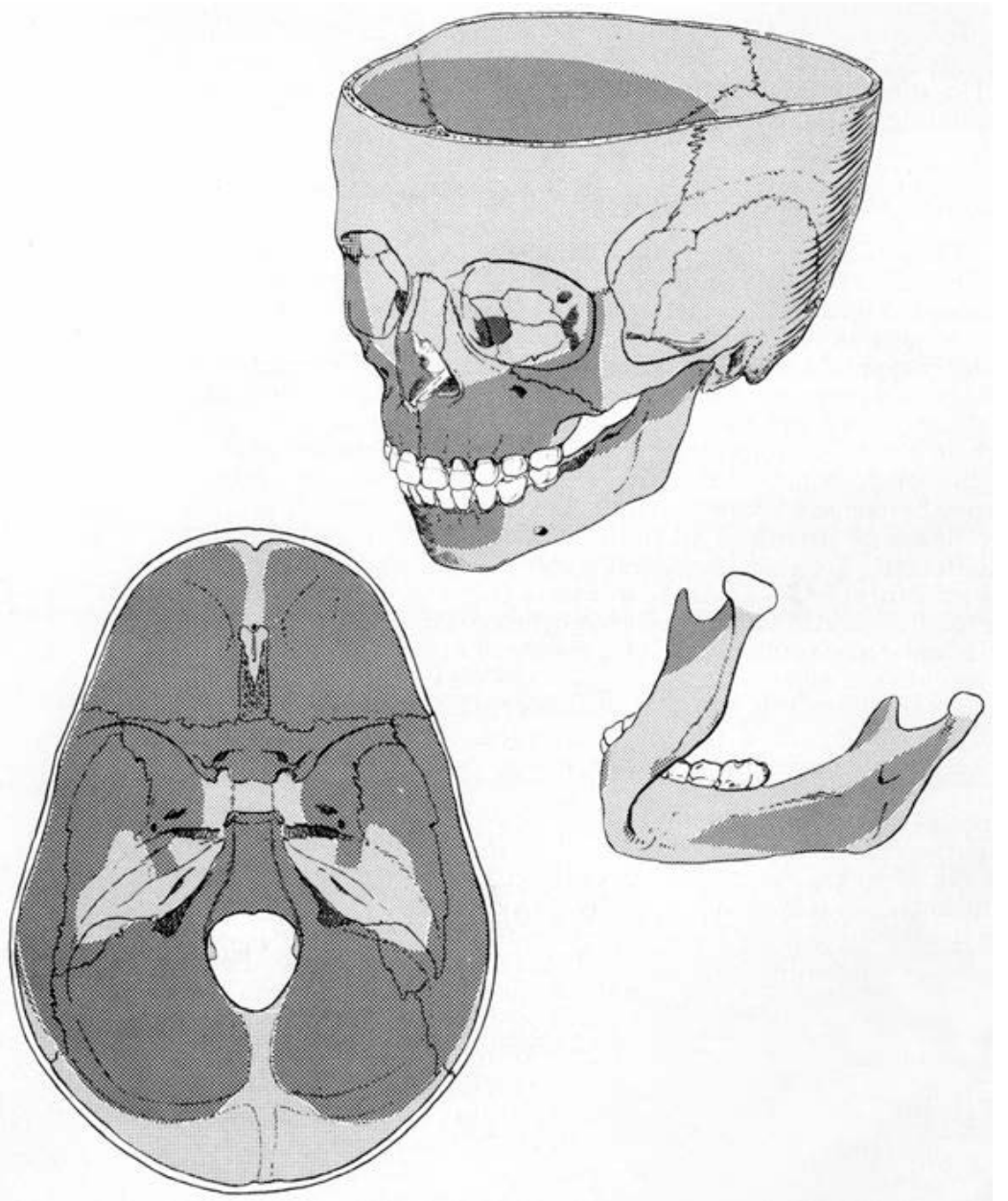


FIGURE 1-3.
Summary diagram of the resorptive (darkly stippled) and depository (lightly stippled) fields of remodeling (From Enlow. D. H. T. Kuroda, and A. B. Lewis: The morphological and morphogenetic basis for craniofacial form and pattern. Angle Orthod., 41:161, 1971, with permission.)

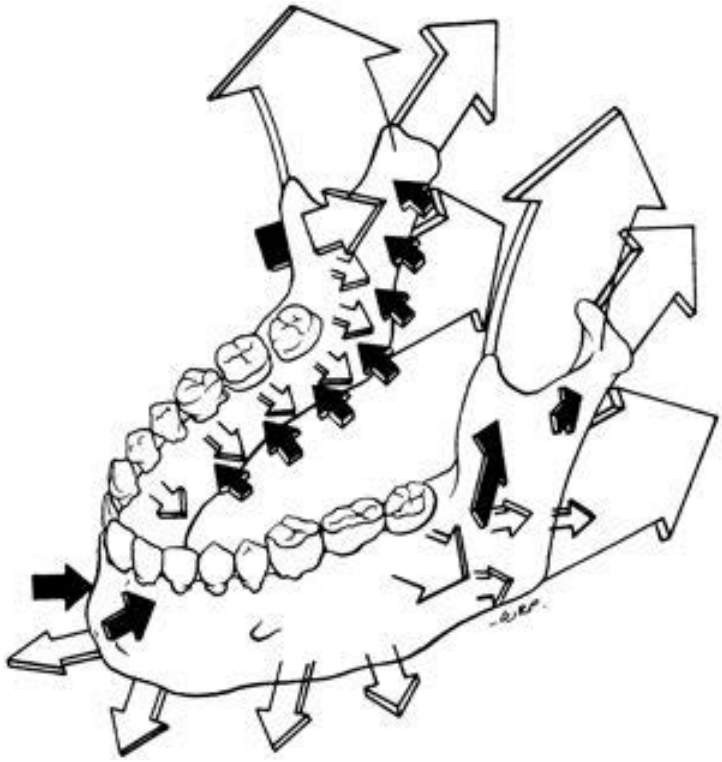


FIGURE 1-4.
Black arrows are surface resorptive, and white arrows are depository.

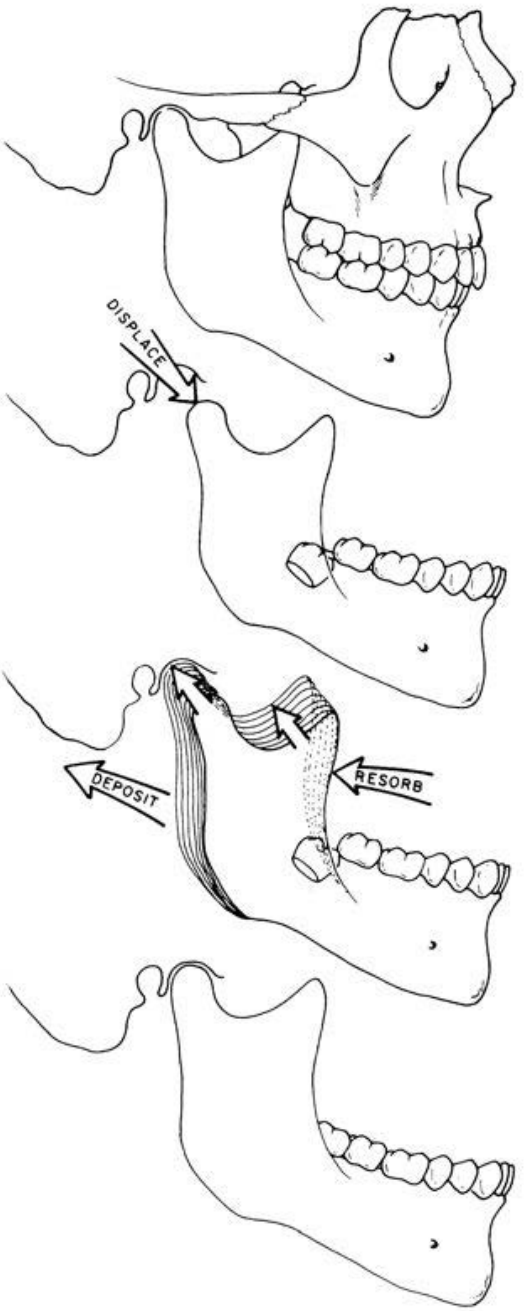
The process of new bone deposition does not cause displacement by **pushing** against the articular contact surface of another bone. Rather, the bone is **carried** away by the expansive force of all the growing soft tissues surrounding and attached to it by anchoring fibers. As this takes place, new bone is added immediately (remodeling), the whole bone enlarges, and the two separate bones thereby remain in constant articular junction. The nasomaxillary complex, for example, is in sutural contact with the floor of the cranium. The whole maxillary region, **in toto**, is **displaced** downward and forward away from the cranium by the expansive growth of the soft tissues in the midfacial region (Fig. 1-6*A*). This then triggers new bone growth at the various sutural contact surfaces between the nasomaxillary composite and the cranial floor (Fig. 1-6*B*). Displacement thus proceeds downward and forward an equivalent amount as maxillary remodeling simultaneously takes place in an opposite upward and backward direction (i.e., **toward** its contact with the cranial floor).

Similarly, the whole mandible (Fig. 1-5) is **displaced** "away" from its articulation in each glenoid fossa by the growth enlargement of the composite of soft tissues in the developing face. As this occurs, the condyle and ramus grow upward and backward (relocate) into the "space" created by the displacement process. Note that the ramus also changes in both shape and size due to the remodeling process as it relocates posterosuperiorly. It becomes longer and wider

to accommodate (1) the increasing mass of masticatory muscles inserted onto it, (2) the enlarged breadth of the pharyngeal space, and (3) the vertical lengthening of the nasomaxillary part of the growing face.

A beginning student is always confused because it is repeatedly heard and read that the face "grows forward and downward." It would seem reasonable, then, that the growth activity of the mandible or the maxilla would be in their anterior, forward-facing parts. However, it is mostly the displacement movement that is forward and downward, thereby complementing the predominantly

FIGURE 1-5



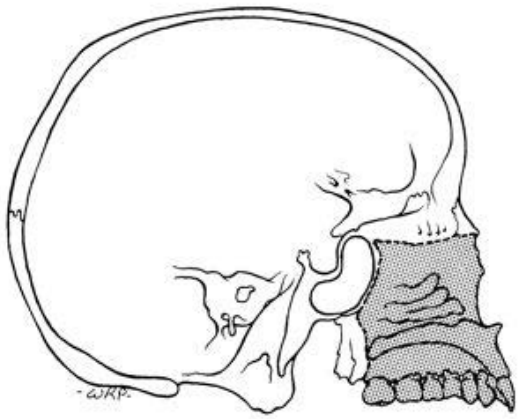
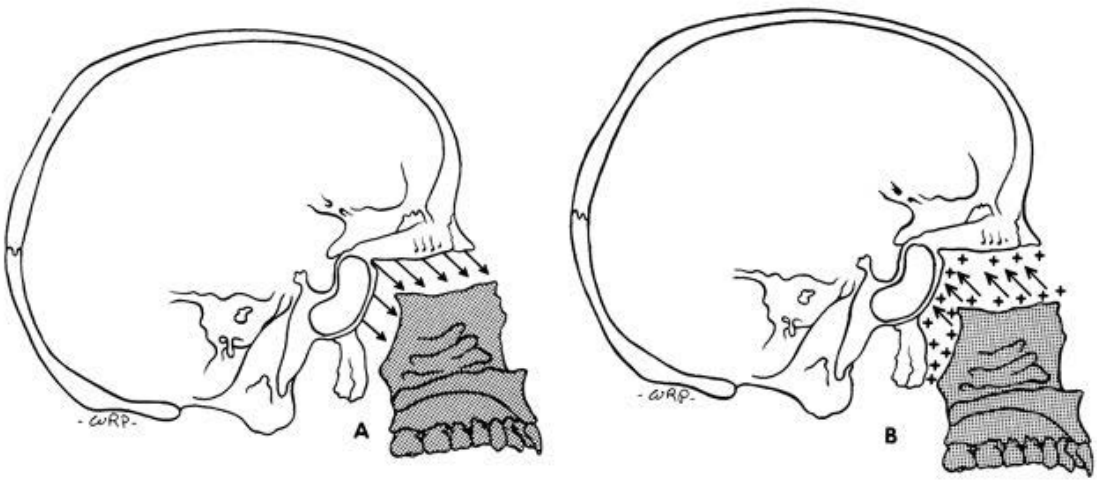


FIGURE 1-6



posterosuperior vectors of remodeling. This is one fundamental reason, as mentioned above, that all joint contacts and bone ends are of basic significance in the growth picture. They are the points away from which displacement proceeds and, at the same time, the sites where remodeling lengthens a given bone. Thus, they are key locations where clinical procedures can alter the growth process.

Note this significant point. If a non-biologic material, such as a metal or plastic plate or other prosthetic appliance, is implanted within the developing craniofacial complex, it lacks both of these two systems of growth movement! It cannot (1) move by patterns of REMODELING since resorptive and depository fields do not exist. It (2) cannot become moved by DISPLACEMENT through traction growth forces because the enlarging soft tissues are not anchored into its substance by Sharpey's fibers. The growing bone contiguous with any non-biologic material, thus, simply grows away from it as they become progressively disjoined. The skeletal as well as soft tissue parts previously around it when originally implanted thereby continue to (1) model and (2) displace, while the non-biologic material itself remains behind without dual biologic growth movement capacity. It now becomes a developmental block against any advancing tissues behind.

The "Genic" Tissues

The histogenic "blast" cells and tissues are activated by a shower of intercellular signals targeted toward the signal-sensitive cell membrane receptors of each cell type, including chondroblasts, osteoblasts, myoblasts, fibroblasts, neuroblastic (satellite) cells, and any other progenitor or undifferentiated cellular types. Signals include mechanical forces, bioelectric potentials, hormones, enzymes, oxygen tension, and other similar agents. Within each cell, a chain of reactions then passes through the cytoplasm to and from the nucleus, endoplasmic reticulum, or organelles such as lysosomes and mitochondria, ending either in (1) output of secretions such as alkaline and acid phosphatase, ground substance (protein mucopolysaccharides), and collagen; or (2) differentiation by cell divisions and maturation into specific tissue types comprising cartilage, bone, periosteum, muscle, epithelia, blood vessels, and lengthening nerves. Ongoing activating signals are "intrinsic" during development, but, as emphasized herein, are subject to clinical modification that then alters regulation of the same underlying biology. This affects the **timing** and **duration** of the cellular activity, and the growth **vectors** (magnitude and direction). It is the selective nature of the signals that governs the pattern of developmental activity that leads to variations in morphology, not any real change in the growth biology itself.

Regional Control of Development

Replacing the archaic notion of "master growth control centers" of yesteryear, is the understanding that tissues within each **local** area contain an array of cell types carrying out the specific developmental requirements of that area. Sensitive to the play of "primary messengers" (activating signals) relating to particular localized **functions** and structural relationships, each and every location has a developing size and shape that is custom-made by its own "genic" cells receiving the local information that determines it. Because the local signals continuously change, regional size and shape correspondingly and progressively adapt. Complex architectonic combinations of regional parts, such as those comprising the mandible and maxilla as a whole and all of the soft tissues associated with them, achieve their differentiating morphology by continuous adjustments among the developing local parts (condyles, coronoid processes, tuberosities, alveolar sockets, tubercles, etc.). This provides a precise and ongoing "fit" among all of them. Everything continues to function all the while.

With regard to the "goodness of fit" of separate bones, muscles, teeth, blood vessels, and all other such anatomic parts to each other, consider several examples illustrating the remarkable developmental interplay characterizing "growth." This interplay is a key factor that makes the whole thing work. When dealing with the growth process, we sometimes forget to appreciate this, or, actually, don't even think of it at all.

For example, a tooth **precisely** matches its alveolar socket in shape, size, and the timing of developmental changes and movements during growth. The osteogenic and fibrogenic periodontal connective tissue (1) shapes and progressively

reshapes the bony socket, (2) allows movement of teeth independent of other surface remodeling or displacement of bone (drift and eruption), (3) moves the socket by remodeling, and (4) remodels its own periodontal connective tissue (fibrogenic) to sustain continuous attachment and to move itself in precise lock step with the moving tooth and bone.†

Another example of "goodness of fit" is a cranial nerve with its sheath of vascular connective tissue passing through a basicranial foramen. The configurational and dimensional fit and the positioning of the foramen must be absolutely perfect. As the nerve constantly moves with the growing brain, the remodeling of the bony passage precisely conforms. If such were not the case, development itself would reach a dead end. (See page 119 Chapter 6 for further phylogenic insight.) Another example is the precise match of a bony tuberosity to which a muscle inserts. There can be no misfit whatever between the two. The match is perfect because of their constant histogenic interplay. Also, any given bone fits precisely within its articular joint. Actually, tissues everywhere throughout the whole body involve virtually limitless adaptive interactions as a part of the growth process, and function continues all the while it happens.

Figure 1-7 schematizes this process. Although the growth activities involved are separated into little boxes, in real life such isolation of relationships, of course, is not possible because of the interdependence among them. This is one basic reason why so many laboratory experiments addressing the "determinants of growth" have historically yielded equivocal results: either (1) **all** of the categories were not

† It is this same histogenic ("growth") process that is utilized in orthodontic tooth movement. Only the signals are changed in order to alter the directions and amounts of tooth movement.

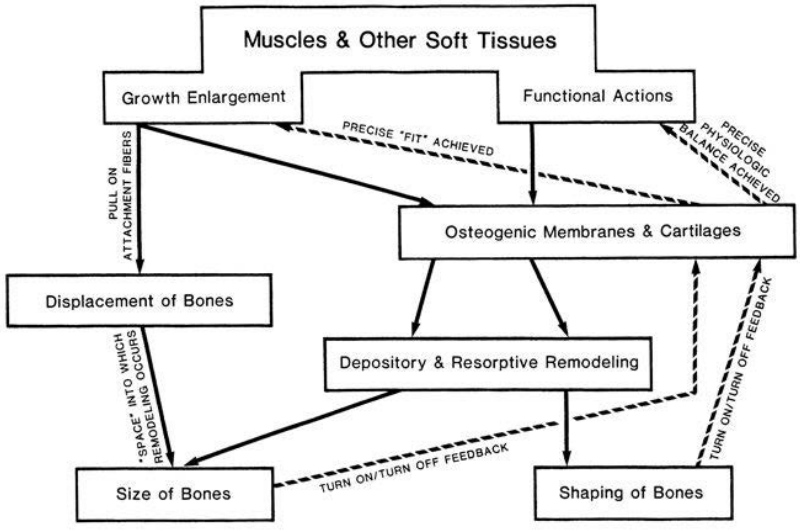


FIGURE 1-7.
See text for discussion. (From Enlow, D. H.: Structural and functional "balance" during craniofacial growth. In: *Orthodontics: State of the Art, Essence of the Science.* Ed. by L. W. Graber, St. Louis, C. V. Mosby, 1986, with permission.)

taken into account (almost always), or (2) the experimental design often calls for separation of the categories in order to attempt to control variables, but which simply cannot be done. (See also later chapters.)

THE THREE PRINCIPAL REGIONS OF FACIAL AND NEUROCRANIAL DEVELOPMENT

The major but mutually interrelated form/function components involved in development are the brain with its associated sensory organs and basicranium, the facial and pharyngeal airway, and the oral complex. Although discussed below separately, they are, of course, developmentally inseparable. The fact that all three are interrelated becomes important when applying growth concepts to clinical situations since the developmental factors underlying most craniofacial dysplasias involve all three. And very few clinical procedures address malocclusions at the level of the cranial base.

The Brain and Basicranium

The configuration of the neurocranium (and brain) determines a person's headform type which, in turn, sets up many of the proportionate and topographic features characterizing facial type. A long and narrow basicranium (dolichocephalic) with its more elongate and open-angle configuration, for example, programs the developmental process so that it characteristically leads to an anteroposteriorly and vertically elongate facial pattern and a more frequent built-in tendency for mandibular retrusion (Fig. 1-8, top).

A rounder basicranium (brachycephalic) is characterized by a proportionately wider but anteroposteriorly shorter configuration, a more closed basicranial flexure, and a vertically and protrusively shorter but wider midface (nasomaxillary complex). These features generally underlie a more orthognathic (or less retrognathic) profile or, in the extreme, a tendency for mandibular protrusion (Fig. 1-8, bottom).

These characteristic features exist because the basicranium is the template that establishes the shape and perimeter of the facial growth fields. The mandible articulates by its condyles onto the ectocranial side of the middle endocranial fossae, and the bicondylar dimension is thus determined by this part of the cranial floor. The nasomaxillary complex is suspended from the anterior endocranial fossae, and the width of the facial airway, the configuration of the palate and maxillary arch, and the placement of all these parts are thus established by it.

The Airway

The facial and pharyngeal airway is a space determined by the multitude of separate parts comprising its enclosing walls. The configuration and dimensions of the airway are thus a product of the composite growth and development of many hard and soft tissues along its pathway from nares to glottis.

Although determined by surrounding parts, those parts in turn are dependent upon the airway for maintenance of their own functional and anatomic

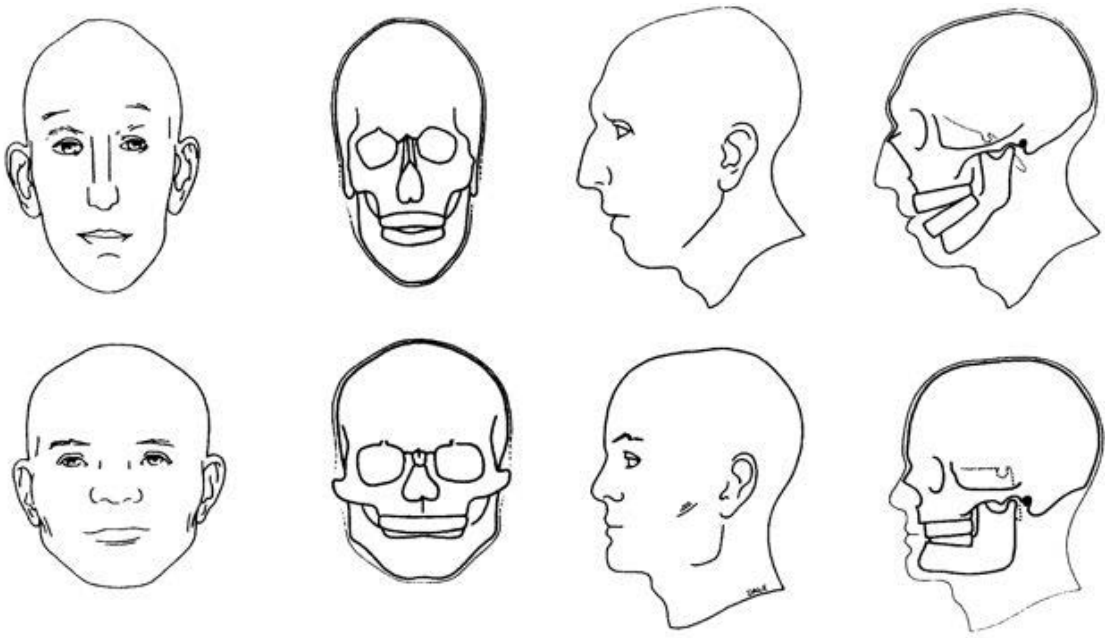


FIGURE 1-8.
(From Enlow, D. H., and J. Dale: In: *Oral Histology*, 4th Ed. Ed. by R. Ten Cate, St. Louis, C. V. Mosby, 1994, with permission.)

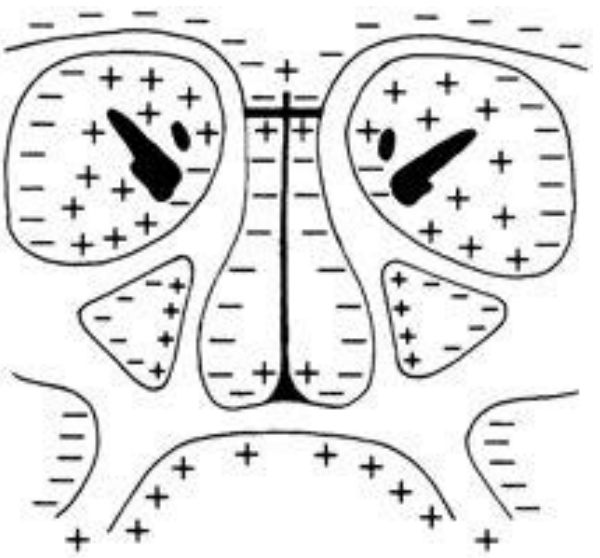
positions. If there develops any regional childhood variation along the course of the airway that significantly alters its configuration or size, growth then proceeds along a different course, leading to a variation in overall facial assembly that may exceed the bounds of normal pattern. The airway functions, in a real sense, as a keystone for the face. A keystone, as you know, is that part of an arch which, if of proper shape and size, stabilizes the positions of the remaining parts of the arch. In Figure 1-9 a few of the many "arches" in a face can be recognized, and the bony remodeling (+ and -) producing them. Horizontally and vertically, the archform of the orbits, the nasal and oral sides of the palate, the maxillary arch, the sinuses, the zygomatic arches, and so forth are all subject to airway configuration, size, and integrity. Note that the airway is strategically pivotal to all of them.

Two easy personal tests can be performed illustrating the airway as a significant factor in programming the developmental course of the facial "genic" tissues. This is useful in explanations of malocclusion etiology for patients or their parents.

First, starting with an open mouth, close the lips and jaws, noticing that you likely raise the tongue against the palate and, momentarily, swallow. This evacuates the oral air into the pharynx, creating an oral vacuum. The effect is to stabilize the mandible and hold it in a closed position with minimal muscle effort. Now, open the jaws and lips, feeling a rush of air into the mouth. To hold the lower jaw in this "mouth breathing" posture requires a different pattern of muscle activity, and the osteogenic, chondrogenic, periodontal, fibrogenic, and other histogenic tissues thereby receive a correspondingly different pattern of signals. This causes

different developmental responses to a different functional morphology adapted to the conditions. As emphasized before, the operation of the growth process itself functions normally. It is the nature of the **activating signals** that produces emerging deviations in the course of development that results in any morphologic variation and perhaps malocclusion.

FIGURE 1-9



Another test is similar. With closed jaws and lips compared to open, try swallowing. Open-jawed swallows are possible, but can be difficult when one is accustomed to a closed mouth. Note the very different pattern of masticatory and hyoid muscle actions required. As with the mouth-breathing test outlined above, altered signals are generated, and the genic tissues work toward a different balance combination, producing a variation in facial morphology. A factor often overlooked by clinicians is that these altered signals may result in different treatment responses to the same intervention. For example, a patient's response to a removable orthopedic appliance such as a bionator or twin block may vary dramatically based upon the patient's mode of breathing.

The Oral Region

In addition to the basicranial and airway factors described above affecting mandibular and maxillary shape, size, and positioning, other basic considerations are involved. If a brain and basicranial asymmetry exists, this condition can either be (1) passed on to cause a corresponding facial asymmetry, or (2) compensated by the facial developmental process to either offset or reduce its magnitude. For the latter, remodeling adjustments produce an actual opposite asymmetry in the nasomaxillary complex and/or mandible that counteracts the basicranial condition. Advances in craniofacial imaging such as cone beam computed tomography have made it easier for clinicians to identify the site of facial asymmetry and plan treatment accordingly.

For the maxilla, if not developmentally compensated or only partially so, the maxillary arch can become deviated laterally, matching the lateral asymmetry of the anterior endocranial fossae. (See Thimaporn et al., 1990.) Or, vertically, one side can become lowered or elevated relative to the other, including the orbits, palate, and maxillary arch. For the mandible, the middle endocranial fossae determine the placement of the temporomandibular joints and, if asymmetric, one or the other will be lower or higher, forward or back. Whole-mandible alignment necessarily follows if not fully or partially adjusted by remodeling during development.

Many other such compensatory adjustments by the remodeling process occur throughout growth and development in many ways, as discussed in subsequent chapters. It involves the development of certain regional imbalances to offset others, resulting in a composite overall structural and functional equilibrium.

Craniofacial Levels

When the face is in balance, there exists a descending, cause-and-effect stratographic arrangement of structural **levels** in the design of the face. Beginning with the frontal lobes of the cerebrum, the floor of the anterior endocranial fossae become adapted in size and shape during their interrelated development. The ectocranial side of this floor is the roof of the nasal chambers, thus programming the perimeter of that key facial part of the airway. This configuration, in turn, is projected inferiorly to the next level, establishing the proportions and configuration of the nasal side of the palate. Then, the perimeter of the apical base of the maxillary dental arch is set by the oral side of the hard palate, all representing configurational projections from the anterior endocranial fossae. The next level following is maxillary cuspid width, and then mandibular cuspid width, all preprogrammed in configuration and in proportion to the basicranium.

The mandible has a component not represented in the maxilla, and that is its **ramus**. The anteroposterior size of the ramus develops by an amount approximating the horizontal span of the pharynx, which has a programmed anteroposterior dimension established by **its** ceiling, which is the ectocranial side of the middle endocranial fossae underlying the temporal lobes of the cerebrum. The ramus, thus, places the mandibular arch in occlusion with the maxillary arch following a pattern set up by the basicranium. Vertically, the developing ramus lowers the corpus by progressive amounts, adapting to the vertical growth of the middle cranial fossae (clivus) as well as the vertical expansion of the nasal airway and developing dentition.

The face, thus, is a stratified series of vertical levels all sharing a common developmental template. This makes possible a workable morphogenic system having a structural design allowing large numbers of separate parts to develop together in harmony and to carry out respective functions while it happens.

The Two Basic Clinical Targets

There is one developmental concept that needs to be addressed with particular emphasis because of its great significance to the old clinical axiom

"working with growth." While a factor such as the basicranium can prescribe and determine a "growth field" in the contiguous facial complex, as described above, it is within the boundaries of that field that **remodeling** then engineers the **shape and size and functional fit** of all parts and develops them through time. However, it can be misunderstood if one presumes that all "local growth" is regulated solely by a single local, intrinsic growth system. Remember, there are **two** kinds of growth activity: (1) localized, regional **remodeling** ("genic" tissues), and (2) the **displacement** movements of all the separate parts as they model. Thus, there are two corresponding histogenic recipients of clinical intervention.

To illustrate this fundamental concept, the incisor and premaxillary alveolar region of the maxilla develops into its adult shape and dimensions by the local remodeling process. But the principal source of the considerable extent of its downward-and-forward growth movement is by displacement, and **that** comes from biomechanical forces of growth enlargement occurring **outside** the premaxillary region itself. Thus, most of the growth movements responsible for the anatomic **placement** of this region, along with, passively, its teeth, are not controlled within its own tissues or any genetic blueprint therein, even though this might be a natural presumption. **Two** clinical targets thereby exist for orthodontists: local remodeling and, separately, the displacement of some whole part produced by the sum of developmental expansions occurring everywhere. There are certain clinical procedures that relate specifically to one or the other target, and some that involve both. For example, rapid palatal expansion mimics displacement; incisor retraction primarily involves remodeling of the anterior portion of the alveolar arch, and bionator treatment involves both remodeling of the alveolar process and displacement of the mandible triggering changes in the remodeling of the ramus.

These two basic growth movements are difficult to separate in clinical interventions since the majority of therapeutic procedures require the teeth to be used to deliver biomechanical forces to the surrounding tissues. This limits the clinician's ability to separate displacement from remodeling using traditional cephalometric techniques. It is likely that the new three dimensional imaging modalities currently available will help with this problem.

Child-to-Adult Changing Proportions

The three principal craniofacial growing parts (brain and basicranium, airway, oral region) each has its own separate timetable of development even though all are inseparately bound as an interrelated whole. Some body systems, such as the nervous and cardiovascular systems, develop earlier and faster compared to others, including the airway and oral regions. The reason is that airway growth is proportionate to growing body and lung size, and the oral region is linked to developmental stages involving the fifth and seventh cranial nerves and associated musculature, the suckling process, dental eruption stages, and masticatory development.

The infant and young child are characterized by a wide-appearing face because of the precociously broad basicranial template, but the face otherwise is vertically short (Fig. 1-10). This is because the nasal and oral regions are yet diminutive, matching the smallish body and pulmonary parts and with

masticatory development in a transitory state. The mandibular ramus is vertically yet short because it is linked in developmental feedback with the shorter, later-maturing nasal and dental regions. Masticatory musculature is proportionately sized and shaped to progressively match increasing function and to interplay developmentally with the ramus.

During later childhood and into adolescence, vertical nasal enlargement keeps pace with growing body and lung size, and dental and other oral components have approached adult sizes and configuration. The mandibular arch is lowered by increasing vertical ramus length. Overall, the early wide face has become altered in proportion by the later vertical changes. The end effect is particularly marked in the dolichocephalic long-headed and long-face pattern, and less so in the brachycephalic headform type.

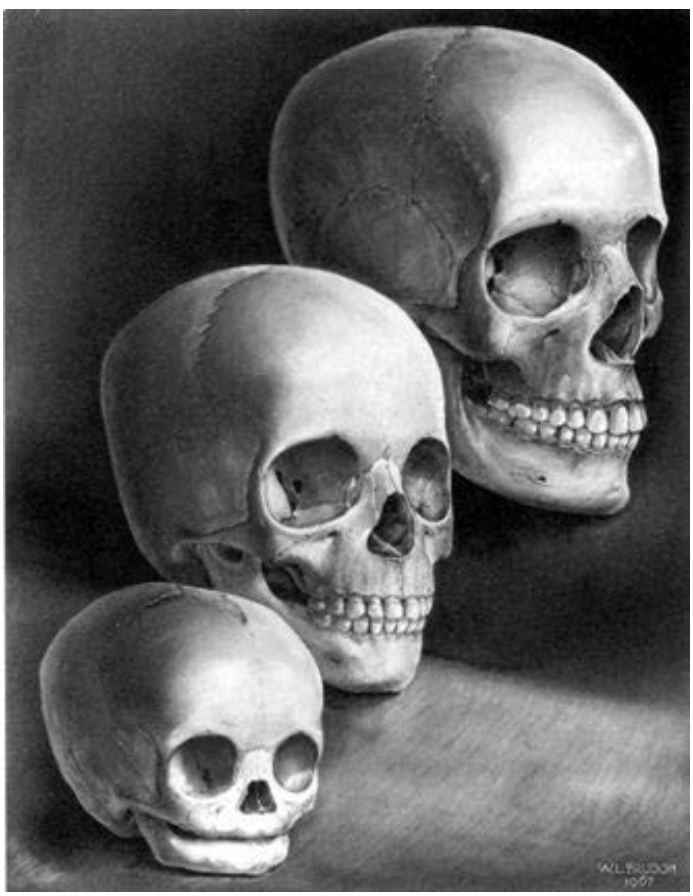


FIGURE 1-10.
(Courtesy of William L. Brudon. From Enlow, D. H.: *The Human Face*. New York, Harper & Row, 1968, with permission.)

Tooth Movement

As with all other sections of this introductory chapter, the subject of how a tooth undergoes intrinsic growth or clinical movements is elaborated in subsequent pages.

To begin, a tooth is moved by either or both of two developmental means: (1) by becoming actively moved in combination with its own remodeling periodontal connective tissue and alveolar socket; and (2) by being carried along passively as the entire maxilla or mandible is displaced anteroinferiorly during facial morphogenesis. A second basic and clinically significant concept is that **bone and connective tissues** (such as the periodontal connective tissue, periosteum, endosteum, and submucosa, all of which participate directly and actively in a tooth's movement) have an intrinsic remodeling process that, when activated, move themselves as a growth function. When a tooth is moved, these other contiguous parts move with it by their own "genic" remodeling process to sustain relationships. A tooth, however, **cannot move itself** in a comparable manner by its own remodeling. Teeth erupt "fully grown" and are mobile, but not motile. A tooth is **moved** by biomechanical forces external to the tooth itself, and there is an elaborate "biology" in the composite process that produces a tooth's growth movements. A tooth must move (drift, erupt, etc.) during maxillary and mandibular growth in order to become properly placed in progressively changing anatomic positions (see Chapters 3, 4, and 5). Whether the force producing the tooth's change in position is intrinsic *or clinically induced*, the actual biology is the same. As mentioned again because the point is important, it is the nature of the activating **signals** that is different, and this causes (1) the multiple array of genic tissues to alter the course of remodeling or (2) the displacement process of a whole bone to become altered in direction or magnitude.

Drift

A worthy advance was made when it was realized that teeth undergo a process of ***drift***. For many years this fundamental concept was limited to horizontal (mesial and distal) movements, and the essential function was held to be a stabilization of the dental palisade to compensate for interproximal attrition. Added to this, now, is that drift has a basic **growth** function. It serves to anatomically place the teeth as the maxilla and mandible enlarge. Such movements are significant considering that a jawbone lengthens considerably from prenatal to adult sizes. Also, the original drift concept was for horizontal movement. Important to the clinician, now, is awareness that teeth, especially maxillary, have a marked **vertical** extent of drift. This is in addition to "eruption" and should not be so termed. **Vertical drift** is a basic growth movement the clinician "works with" because it can be modified by clinical intervention (i.e., orthodontic treatment).

Just as teeth undergo a drifting movement, the bone housing them also moves. Unlike a tooth, however, bone moves by the remodeling action of its enclosing osteogenic membranes, and this is also a direct target for clinical intervention. The intrinsic coordination of these bone-tooth movements is remarkable.

A Fundamental Principle of Growth

It has been emphasized in the preceding pages that facial growth is a process requiring intimate morphogenic **interrelationships** among all of its component growing, changing, and functioning soft and hard tissue parts. No part is developmentally independent and self-contained. This is a fundamental and very important principle of growth. As underscored earlier, the growth process works toward an ongoing state of composite functional and structural equilibrium. In clinical treatment, no key anatomic part can be fully segregated and altered without affecting "balance" with other parts and their state of physiologic equilibrium as well.

In essence, orthodontic treatment seeks to maximize the effectiveness of anatomic compensations to achieve an aesthetically harmonious masticatory system.

2

Basic Growth Concepts

An in-depth understanding of facial morphogenesis is essential so that the clinician as well as the research biologist can properly grasp (1) differences between "normal" and ranges of abnormal; (2) the underlying biologic process that accounts for these differences and the virtually limitless variations involved; (3) reasons for rationales utilized in diagnosis and treatment planning as well as selection of appropriate clinical procedures; and (4) the biologic factors underlying the important clinical problems of retention, rebound, and relapse after treatment.*

It was emphasized in the previous chapter that one of the most important elements of the facial growth process is that two separate but closely interrelated systems of **movement** exist—remodeling and displacement. For remodeling, a bone (or any other kind of organ or tissue composite) is not simply "modeled" when it first appears prenatally. It cannot simply grow by new additions keeping the same form. That is not possible given the architectonically complex designs involved. Because some areas of any given part grow faster or to a greater extent than others, *r*emodeling is a necessary growth function. Then, when bones and all other kinds of organs enlarge, they must necessarily all move away from each other to allow for the enlargement. Because modern orthodontic treatment is a series of decisions on which compensations to remove and which to augment or keep, understanding **how** compensations work is a fundamental consideration. Historically, there have been spirited arguments over the underlying theory (Functional Matrix modulation versus orthopedic effects resulting from sutural modification). Surprisingly, however, even to this day, the rationale of many clinical procedures (e.g., Functional appliances, light forces with self ligating brackets to "grow bone"), are seldom discussed in terms of their effects on remodeling and displacement. This is one of the basic reasons why the actual biological basis of these treatments is often overlooked leaving the clinician in danger of designing treatments that break the biologic rules and thus a doomed to failure. The following pages will address the remodeling process first. The craniofacial skeleton initially is emphasized because the bones represent the head as a whole as seen in radiographs. This displacement process is then described, and a developmental merger of the two is presented in the following chapter.

* The rapid acceptance of "lifetime retention" by some clinicians underscores the need for understanding the biology of post treatment changes. Without biology, orthodontists are left with poorly designed retrospective clinical studies on relapse and retention that suggest nothing is stable. These studies conducted in the late 1980s were a major setback for modern orthodontic thought and helped fuel a return to Angle's faulty arch expansion philosophy.

REMODELING

A lay person's natural perception of "growth" is often quite incorrect. A bone such as the mandible **does not** grow simply by generalized, uniform deposition of new bone (+) on all outside surfaces (Fig. 2-1), with corresponding resorption (-) from all inside surfaces, as one might erroneously presume (and as has often been incorrectly taught). It is not possible for bones having the complex morphology of, for example, the mandible or the maxilla to increase in size by such a growth process. Because of the topographically complex nature of each bone's shape, the bone must have a **differential** mode of enlargement, in which some of its parts and areas grow much faster and to a much greater extent than others. Many of the **external** surfaces of most bones are actually **resorptive** in nature. In Figure 2-2, **fields** of surface resorption (darkly shaded) and deposition (lightly shaded) blanket the whole bone. How can a bone increase in size, even though many outside (periosteal) surfaces undergo resorptive removal as the bone grows? Keep this question in mind as the processes of facial growth are explained in the pages that follow.

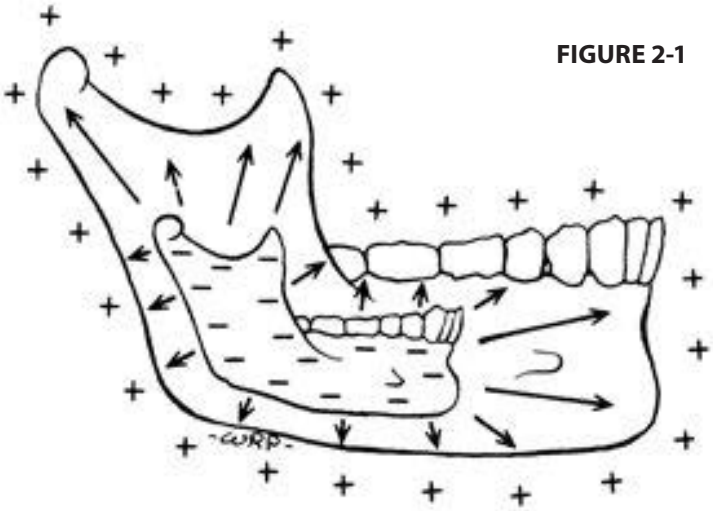


FIGURE 2-1

The reason a bone must remodel† during growth is because its regional parts must become **moved** (Fig. 2-3). This calls for sequential remodeling changes in the shape and size of each region. The mandibular ramus, for example, moves progressively posteriorly by a combination of deposition and resorption. As it does so, the anterior part of the ramus becomes **remodeled** into a new addition for the mandibular corpus. This produces a growth elongation of the corpus. This

† Four different kinds of remodeling occur in bone tissues. One is biochemical remodeling, taking place at the molecular level. This involves the constant deposition and removal of ions to maintain blood calcium levels and carry out other mineral homeostasis functions: Another type of remodeling involves the secondary reconstruction of bone by haversian systems and also the rebuilding of cancellous trabeculae. A third kind of remodeling relates to the regeneration and reconstruction of bone during or following disease and trauma. The remodeling process that we are dealing with in facial morphogenesis, however, is **growth remodeling**. In order for a bone to grow and enlarge, it must also undergo a simultaneous process of remodeling.

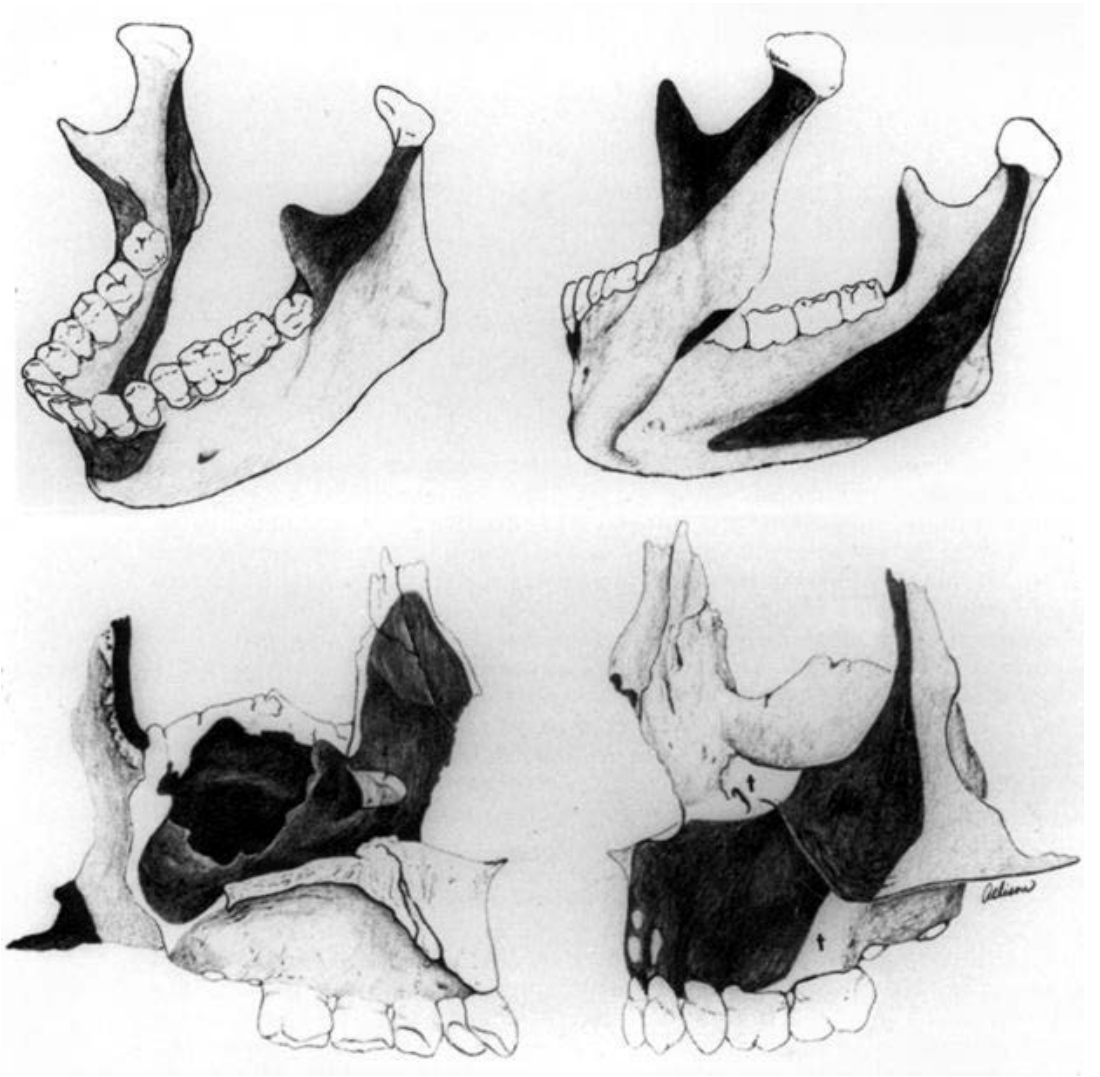
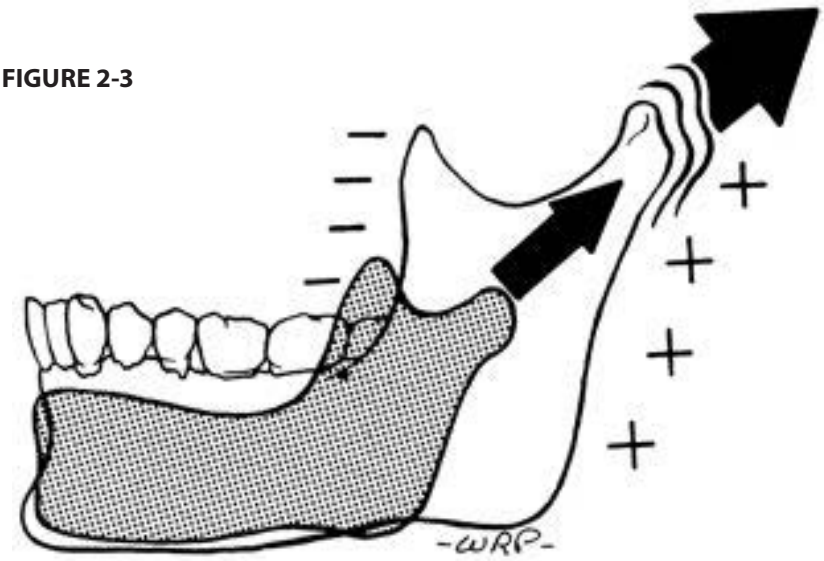


FIGURE 2-2.
Top, Mandibular remodeling. Resorptive surfaces are dark shaded, and depository surfaces are unshaded. Bottom, Maxillary remodelling. (From Moyers, R., and D. Enlow. *Growth of the Craniofacial Skeleton*. In: *Handbook of Orthodontics,* 4th Ed. Chicago, Mosby-Year Book, Inc. 1988, with permission.)

progressive, sequential movement of component parts as a bone enlarges is termed **area relocation**. The whole ramus is thus relocated posteriorly, and the posterior part of the lengthening corpus becomes relocated into the area previously occupied by the ramus. Structural remodeling from what **used to be** part of the ramus into what then **becomes** a new part of the corpus takes place. The corpus grows longer as a result.

The mandible remodels differentially in directions that are predominantly posterior and superior. Even though successive remodeling of one part into another

FIGURE 2-3



constantly takes place as the whole bone enlarges, the form of the bone as a whole is sustained (with some characteristic age changes in shape). It is remarkable that the external morphologic characteristics of any given bone are relatively constant, even though its substance undergoes massive internal changes and all its parts experience widespread alterations in regional shape and size as they are relocated. This is the special function of growth remodeling; it **maintains** the form of a whole bone while providing for its enlargement at the same time. Thus, remodeling is not a process that functions essentially to alter overall shape, although some degree of this is also involved. Although the term "remodeling" implies such change, the actual changes produced by growth remodeling are mostly those that deal with the sequential **relocation** of the bone's component parts.

Bone produced by the covering membrane ("periosteal bone") constitutes about half of all the cortical bone tissue present; bone laid down by the lining membrane ("endosteal bone") makes up the other half (Fig. 2-4). In this diagram, note how the cortex on the right was formed by the periosteum and the cortex on the left by the endosteum as both sides shifted (drifted) in unison to the right.

The surface that faces **toward** the direction of movement is depository (+). The opposite surface, facing away from the growth direction, is resorptive (-). If the rates of deposition and resorption are equal, the thickness of the cortex remains constant. If deposition exceeds resorption, overall size and cortical thickness gradually increase. In Figure 2-5, the pattern of growth fields results in a **rotation** of the skeletal part shown. Such rotations are a significant part of the developmental process of the face and cranium, as will be seen later. See also page 38.

The operation of the remodeling fields covering and lining the surfaces of a bone is actually carried out by the osteogenic **membranes** and other surrounding tissues, rather than by the hard part of the bone. The bone does not "grow itself"; growth is produced by the **soft tissue matrix** that encloses each whole bone. The genetic and functional determinants of bone growth reside in the composite of soft

tissues that turn on and turn off, or speed up and slow down, the histogenic actions of the osteogenic connective tissues (periosteum, endosteum, sutures, periodontal membrane, etc.). Growth is not "programmed" within the calcified part of the bone itself. The "blueprint" for the design, construction, and growth of a bone thus lies in the muscles, tongue, lips, cheeks, integument, mucosae, connective tissues, nerves, blood vessels, airway, pharynx, the brain as an organ mass, tonsils, adenoids, and so forth, all of which provide information signals that pace a bone's development by its osteogenic tissues.‡

FIGURE 2-4

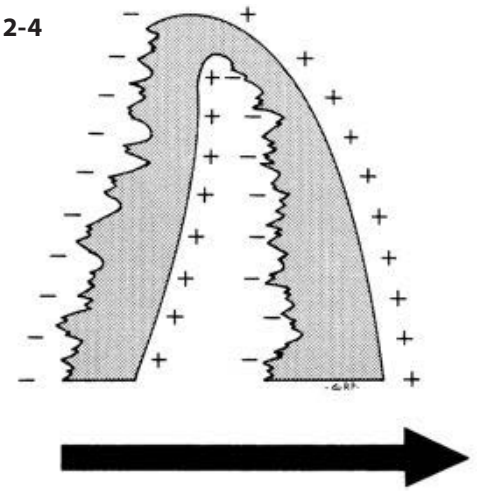
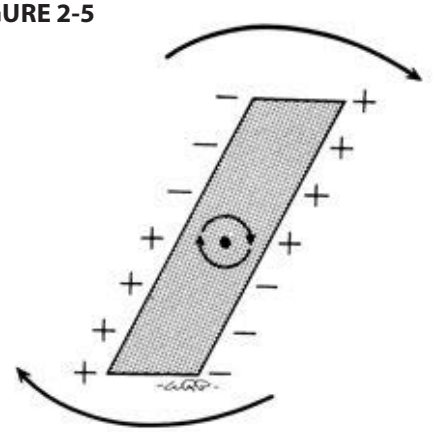


FIGURE 2-5



Fields that have some special significance or noteworthy role in the growth process are often called growth **sites**. The mandibular condyle, for example, is such a growth site (Fig. 2-6). Remember, however, that growth does not occur just at such special growth sites, as is sometimes presumed. The entire bone participates. **All** surfaces are, in fact, sites of growth, whether specially designated or not. The old term "condylar growth" is still often used. It is misleading, however, because it mistakenly implies that the condyle is **the** growth center largely responsible for overall mandibular growth and development. If only condylar growth were operative, the condyle would sit on an elongated neck as a giraffe's head perches high on its neck. The **entire** ramus, together with its condyle, participates actively and directly. Clinical interventions designed to manipulate mandibular growth must necessarily achieve therapeutic effects by altering the entire ramus and not merely by affecting "condylar growth."

In Figure 2-7, the osteogenic connective tissue overlying field *a* moves to a^1. The underlying area of bone beneath it is remodeled and moves under its control. This remodeling field is thus relocated to occupy the region located just posteriorly. A **reversal line** (*x*) separates field *a* from the area of the ramus behind it, and moves to x^1. The resorptive field in the larger mandible (a^1) occupies the

‡ By osteogenic tissues we mean the biologically active cells of the periosteum and endosteum including osteoblasts and their precursors, neurons, capillaries and fibroblasts.

same **relative** position as when it was smaller during the former growth stage (*a*). The overlying osteogenic field, however, is now larger and has moved to a new location, relocating its underlying bone with it by continuous bone deposition and resorption (i.e., remodeling growth). Remember, the osteogenic connective tissue of the field moves by its own remodeling; the bone deep to it moves by remodeling (deposition and resorption) produced by this connective tissue. The actual bone tissue present in the ramus of the smaller stage has been replaced by a whole new generation of bone in the location occupied by the ramus of the larger stage following relocation. The **patterns of distribution** of all the various resorptive and depository fields, however, have not changed; the fields have only moved from one position to another as the whole bone enlarged. This requires sequential remodeling as any one field expands into areas previously occupied by other fields which, in turn, have moved on to hold successively new locations. The same developmental process continues, over and over again.

Although these growth movements are carried out by the osteogenic membranes and cartilages, the bone itself contributes feedback information to them so that as the size, shape, and biomechanical properties of the bone come into equilibrium with functional requirements, the histogenetic activity then becomes adjusted. Removable orthopedic devices such as the bionator seek to modify this biomechanical equilibrium to achieve a specified orthodontic treatment goal.

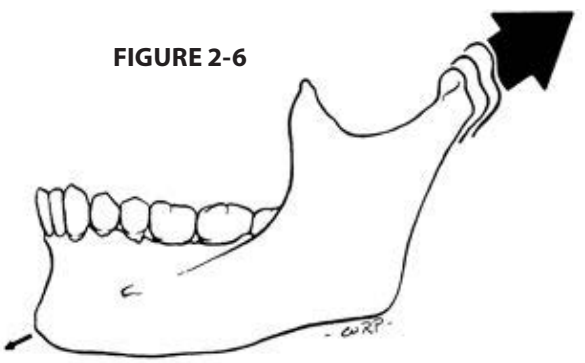


FIGURE 2-6

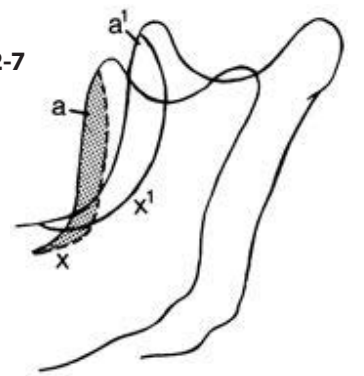


FIGURE 2-7

Fields of Remodeling

As already seen, resorptive and depository **fields** of growth blanket all of the outside and inside surfaces of a bone (Fig. 2-2). This mosaic pattern is more or less constant for each bone throughout the growth period, unless a major change in the shape of a region becomes involved. As the perimeter of each of these growth fields enlarges, the parts of the bone associated with them correspondingly increase in size. As emphasized earlier, of course, the actual operation of these fields of growth is performed by the enclosing osteogenic connective tissues. The bone itself is the product of this field activity. Thus, during the operation of the relocation process, it is the growth fields formed by the "genic" connective tissues that first move and control the relocation movements of the underlying bony parts associated with each

field. The growth movement of the bone **follows** the pace-setting movement of the overlying growth field. There is virtually no lag time, however, between the two.

Variations in facial configuration are always the rule. No two faces are quite alike. Morphologic variations, normal and abnormal, are produced by corresponding developmental variations that take place during the growth process. Some can be genetically established by characteristic soft tissue relationships that are hereditary determinants of bone growth. Other variations are largely determined by functional changes in soft tissue relationships during an individual's own development. The results, however, are all based on the following factors that establish the nature of anatomic variations in an individual person:

1. Fundamental differences in the **pattern** of the fields of resorption and deposition, that is, the distribution of the growth fields in an individual person.

2. The specific placement of the **boundaries** between growth fields; that is, the size and shape of any given growth field.

3. The differential **rates** and **amounts** of deposition and resorption throughout each field.

4. The **timing** of the growth activities among the different fields.

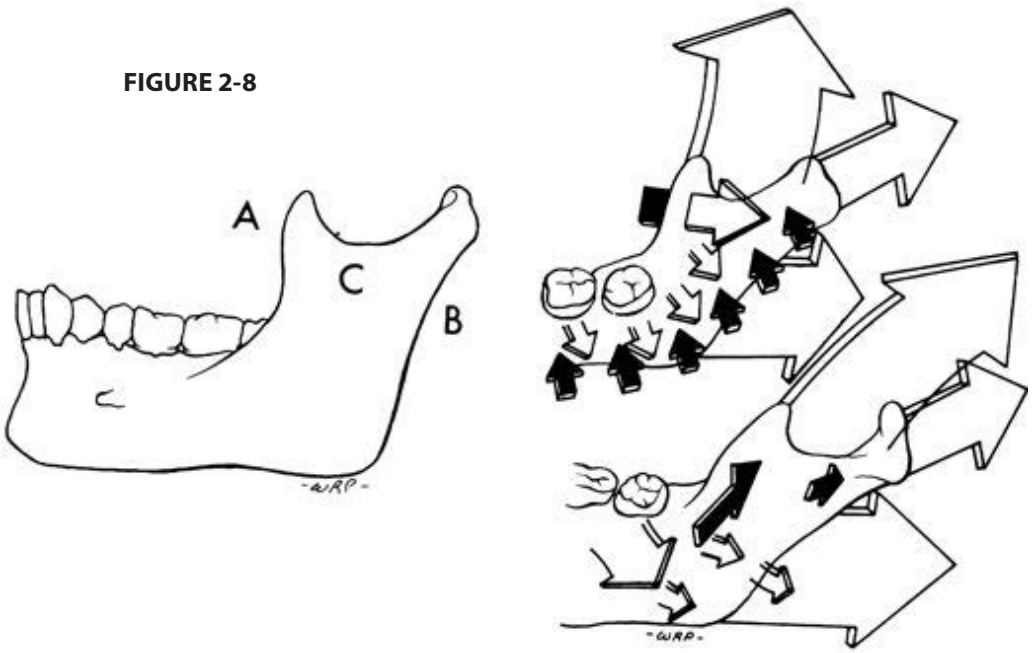
Understanding the regional growth field concept and the operation of the remodeling process is basic for the clinician. Clinicians must also understand how the complementary process of displacement operates (described later), and how an individual's growth variations can occur. The question can be one of working "with" or "against" the intrinsic morphogenic processes in the use of different orthodontic procedures. Does a given procedure, for example, harness the same intrinsic regional remodeling or displacement direction, but with alteration of magnitude? For example, the use of extra oral forces to distalize the maxillary molars moves these teeth into an area of depository growth activity. Such tooth movement may favorably augment the remodeling pattern in the tuberosity area, resulting in a more stable orthodontic correction. Or, is a direction actually changed, perhaps with severe violations of remodeling field boundaries and balance, leading to rebound? For example, labial movement of lower incisors to reduce crowding places those teeth in an area of bone normally undergoing resorption. This creates the potential for biologic failure because normally new bone is not forming in this area.

As mentioned, the mandibular condyle is a specially recognized growth **site**. The condyle and some other special sites have sometimes been termed growth **centers**. This label has come into disfavor, however, because it is now understood that such a site does not actually control the growth processes of the bone as a whole. They are not "master centers" that directly regulate the overall morphogenic process of the entire bone and all its regional parts. Although developmentally unique (see Chapter 4), they represent only regional fields of growth adapted to the localized morphogenic circumstances in their own particular areas, just as

all other regional developmental sites are locally adapted. Growth "centers" are a conceptual anachronism.

Routine headfilms, of course, are **two-dimensional,** and this is a limitation that presents many troublesome problems. Only the anterior and posterior **edges** of the ramus, for example, can be visualized (as at *A* and *B*) in lateral cephalograms (Fig. 2-8). Important changes on surfaces in the span **between** these edges (*C*) cannot be visualized. This is all the more reason for the clinician and researcher to thoroughly understand what happens when such areas grow in a **three-dimensional** manner not representable by the headfilm itself.

FIGURE 2-8



Implant Markers

Metallic implants (tiny pieces of tantalum or some other appropriate metal) are often used as radiographic markers in clinical and experimental work to study bone remodeling and displacement in headfilms. Using the markers as registration points when superimposing serial headfilm tracings, one can readily determine the amount and direction of remodeling as well as displacement movements.

If a metallic marker is implanted on the depository side of a cortex, it becomes progressively more deeply embedded in the cortex as new bone continues to form on the surface and as resorption takes place from the other side. Eventually, the marker would become translocated from one side of the cortex to the other, not because of its own movement (the marker itself is immobile), but because of the "flow" of the drifting bone around it.

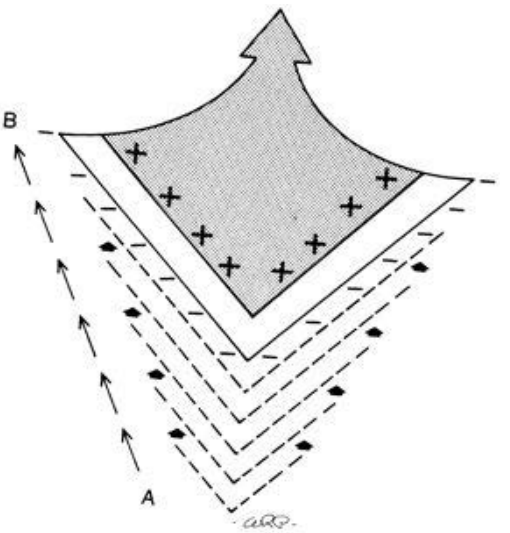
If two implants are placed across from each other on the two sides of a joint (suture, synchondrosis, temporomandibular joint [TMJ]), the distance of their separation, subsequent to a period of growth, indicates the direction and

amount of displacement as well as the total extent of bone deposition on these two joint surfaces. Vital dyes (alizarin, procion, tetracycline, etc.) can also be used to determine the sequence and amount of new bone formation as well as specific locations utilizing histologic sections.

The "V" Principle

A most useful and basic concept in facial growth is the V principle (Fig. 2-9). Many facial and cranial bones, or parts of bones, have a V-shaped configuration (or a funnel-shape in three dimensions). Note that bone deposition occurs on the **inner** side of the V; resorption takes place on the outside surface. The V thereby **moves** from position *A* to *B* and, at the same time, **increases** in overall dimensions. The direction of movement is toward the wide end of the V. Thus, a simultaneous growth movement and enlargement proceeds by additions of bone on the inside with removal from the outside. The V principle will be referred to many times in later explanations of the facial growth process.

FIGURE 2-9

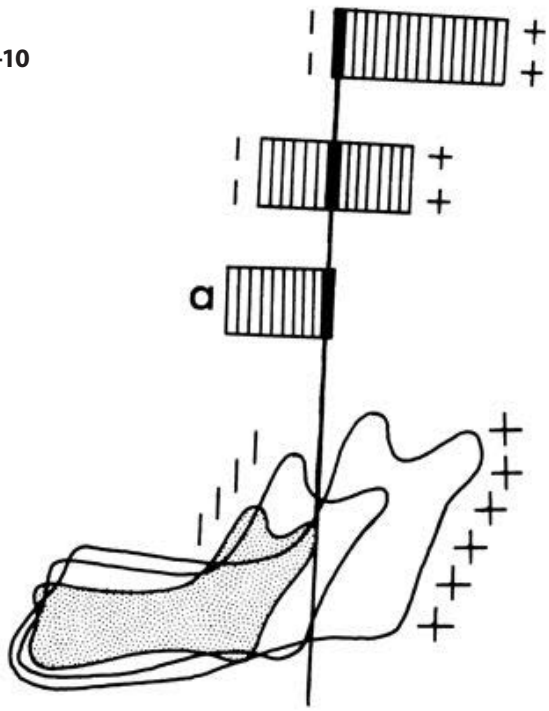


The Relocation Function of Remodeling

Why do bones remodel as they grow? The key factor is the process of **relocation**. To illustrate, in the stack of chips in Figure 2-10, the black chip is at the right end in *a* at the level of the condyle in the smallest mandibular stage. This location then becomes translocated "across" the ramus to lie at the level of the anterior margin in the third stage. As "growth" has continued to take place, the black chip became progressively "relocated"—not by its own movement, but because new chips have been added on one side and removed from the other. This changes the **relative position** of the black chip within the stack, even though this chip itself does not move. Let the stack of chips represent a whole growing area having complex topographic shape, such as the ramus, rather than a perfectly cylindrical form. It is apparent that the changing relative positions of the black

chip would require continuous **remodeling** of the shape and sectional dimensions to conform with each successive position the chip comes to occupy. A **sequence** of continuous remodeling changes is required level by level. Remodeling is a process of **reshaping** and **resizing** each level (chip) within a growing bone as it is relocated sequentially into a succession of new levels. This is because additions and/or resorption in the various **other** parts cause changes in the relative positions of all levels. Note that the **position** of the condyle in the smallest mandibular stage becomes relocated into the middle of the ramus and then onto the anterior border of the ramus. Continuous remodeling is thus involved as this and all other areas change in relative position.

FIGURE 2-10



In the face of a young child, the levels of the maxillary arch and nasal floor lie very close to the inferior orbital rim. The maxillary arch and palate, however, **move** downward. This process involves (in part) an inferior direction of **remodeling** by the hard palate and the bony maxillary arch (Fig. 2-11). Bone deposition occurs on the downward-facing oral surface, together with resorption from the superior-facing nasal surface of the palate. The combination results in a downward **relocation** of the whole palate and maxillary arch composite into the progressively lower levels, so that the arch finally comes to lie considerably below the inferior orbital rim. The vertical dimension of the nasal chamber is greatly increased as a result.

About half of the external surfaces involved in these growth and remodeling examples are resorptive and half depository. About half of the bone tissue of the palate is thus endosteal and half periosteal. (The cortex on the nasal side of the palate is produced by the endosteum of the medullary cavity.)

Because of the **relocation** process, the inferior nasal region of the adult occupies an area where the bony maxillary arch **used to be** located during earlier childhood (Fig. 2-12). What was once the bony maxillary arch and palatal region has been converted into the expanded nasal region. This is "growth remodeling"; the basis for it is **relocation**.

FIGURE 2-11

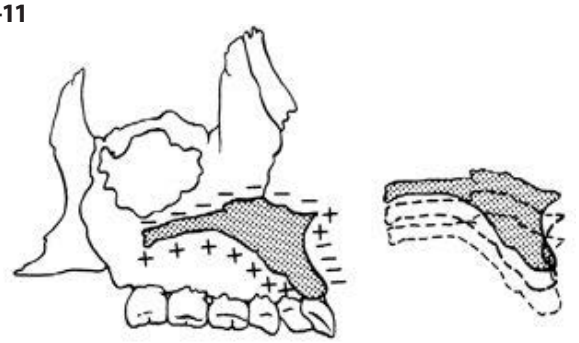
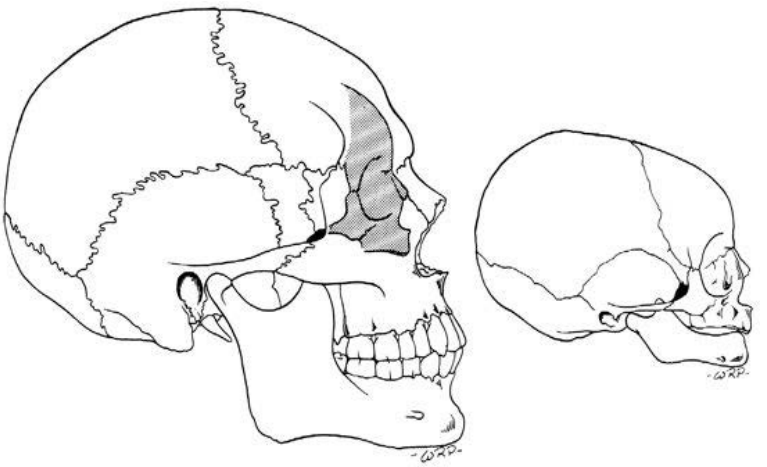


FIGURE 2-12



As the palate and arch grow downward by constant deposition of new bone on one side and resorption of previously formed bone from the other, the bone tissue that comes to house the teeth at older age periods is not the same actual bone enclosing them during the succession of former growth levels. This is significant because the growth movement and the exchanges of bone involved are **used** by the orthodontist to "work with growth." (See "vertical drift" Chapter 3 and 5.)

It was shown above that as the mandible grows, the ramus moves in a backward direction by appropriate combinations of resorption and deposition. As the ramus is **relocated** posteriorly, the corpus becomes **lengthened by a remodeling conversion** from what was at one time the ramus during a former growth period (Fig. 2-3). During growth from the fetus to the adult, the "molar"

region in the younger mandible, for example, undergoes relocation to occupy the "premolar" region of the larger, older mandible. It is apparent that remodeling is a process of relocation and that the same **deposition and resorption producing growth enlargement is the same that also carries out the growth remodeling process.** See page 5 for the multiple functions of remodeling.

A transverse section through the zygomatic arch in Figure 2-13 demonstrates how a bone relocates laterally as the whole bone simultaneously grows in length. The zygomatic arch is moving and enlarging laterally and also inferiorly as the entire face, brain, and cranium widen and expand into space formerly occupied by the zygomatic arch. It does this by progressive deposition on the lateral-facing and downward-facing periosteal and endosteal surfaces, with resorption from the opposite cortical sides. The remnants of the old cortical contours can be recognized in microscopic sections. The right and left zygomatic arches thus grow out and away as the rest of the head enlarges between. The arches also increase in size to accommodate the growing muscles attached to them. (Note: Half of the bone tissue is endosteal in origin, and half is periosteal. Half of the inner and outer surfaces are resorptive, and half are depository.)

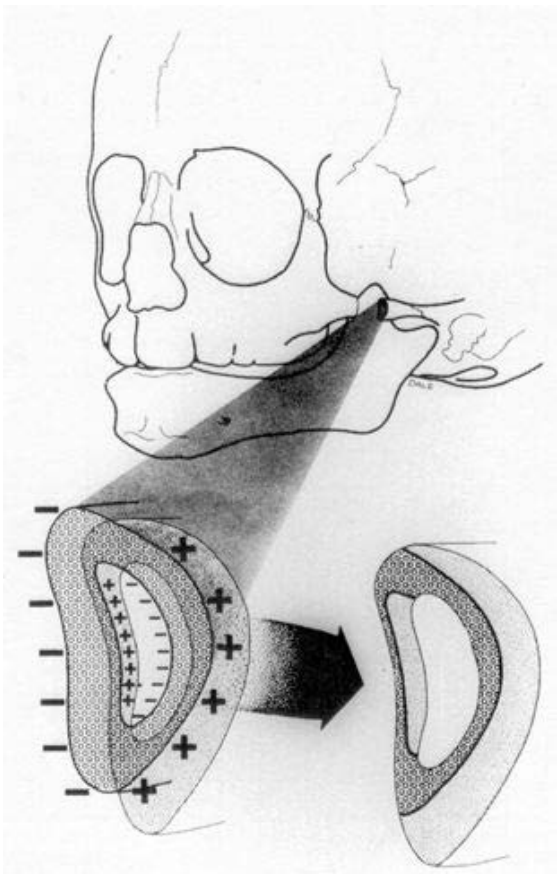


FIGURE 2-13.
(From Enlow D., and J. Dale. *Childhood Facial Growth and Development*. In: *Oral Histology,* 4th Ed. Ed. by R. Ten Cate. St. Louis, C. V. Mosby, 1990, with permission.)

THE DISPLACEMENT PROCESS

In **remodeling**, the bone surface moves (relocates) by deposition on the side facing the direction of growth movements, as seen in Figure 2-13. In the process of **displacement**, however, the **whole** bone is **carried** by mechanical force as it simultaneously enlarges. Remodeling and displacement are separate processes, but always where **joints** (sutures, TMJ, synchondroses) are involved, they necessarily occur in conjunction with one another, since either one without the other is not possible (See Fig. 1-5).

The growth expansion of single bone is a process by which its size, shape, and fitting develop in response to the composite of all the functional soft tissue relationships associated with that individual bone. The bone does not grow and enlarge in an isolated way, however. Its increases in size involve one or more articular contacts with **other** bones that are also enlarging at the same time. For this reason, as emphasized above, all articular contacts are important because they are the sites where displacement is involved. Articulations are the interface surfaces "away" from which the displacement movements proceed as each whole bones enlarges. The amount of enlargement equals the extent of displacement. That is, a bone grows into the space being created as the whole bone is displaced by amounts determined by the extent of surrounding soft tissue enlargement. The enlargement of each bone thereby keeps pace with that of the soft tissues it serves in a mutually interrelated and controlled manner.

In the analogy shown in Figure 2-14, the expansion of a single balloon does not "compete" for space. However, if **two** enlarging balloons are in contact with each other, a displacing **movement** takes place until their positions become adjusted as either one or both expand. This movement proceeds away from the interface between the two balloons. What happens when the mandible, for example, grows in a direction **toward** its articular contact with the cranium? A "displacement" takes place in which the whole mandible moves **away** as it enlarges by an equal amount toward the temporal bone (Fig. 2-15).

Do the balloons **shove** each other apart **because** of the pushing force produced by the expansion? Or, are the balloons **carried** apart by other (outside) mechanical forces, with growth expansion **responding** by an equal amount to the separation, thereby maintaining the precise contact between them (Fig. 2-16)? In the first possibility, the extent of push (displacement) equals, but **follows**, the combined amount of expansion. In the second possibility, the extent of combined enlargement equals, but follows (virtually simultaneously), the amount of separation (displacement), with the balloons "growing" into the potential space being created. In other words, which is the primary (pacemaker) movement, displacement or remodeling enlargement? The question is more than academic; clinical treatment procedures utilize one or the other or both kinds of growth movements in response to clinical signals activating the appropriate "biology."

The above question has been, and still is, one of the great historical controversies in craniofacial biology. The mandible does not enlarge by simple, symmetrical expansion, as shown in Figure 2-17(*A*). Rather, it remodels by deposition and resorption in the manner shown in Figure 2-17(*B*). The predominant

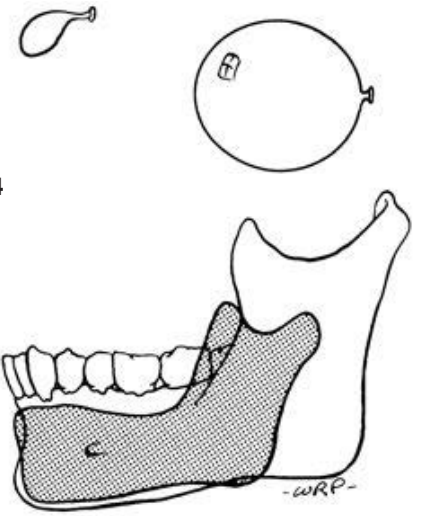


FIGURE 2-14

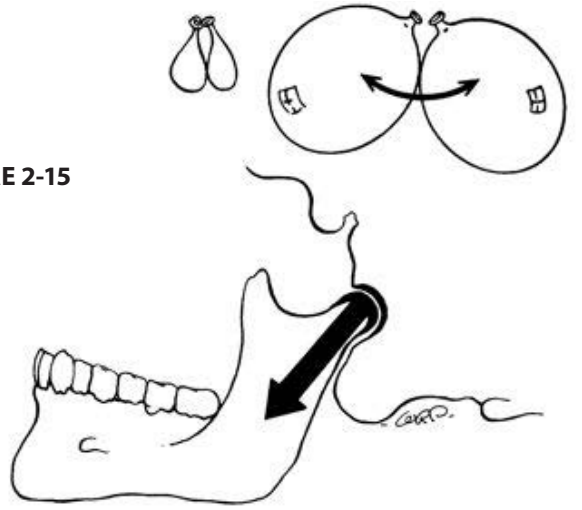
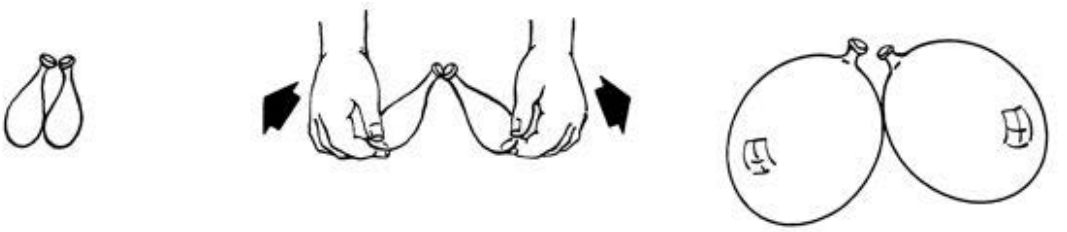


FIGURE 2-15

FIGURE 2-16



vectors (direction and magnitude) are posterior and superior. Thus, the condyle enlarges directly **toward** its articular contact in the glenoid fossa of the cranial floor.

As this takes place, the whole mandible is moved forward and downward by the same amount that it remodels upward and backward (Fig. 2-17, lower right). The direction of remodeling by new bone additions on the ramus and at the condyle and, separately, the direction of displacement are opposite to each other.

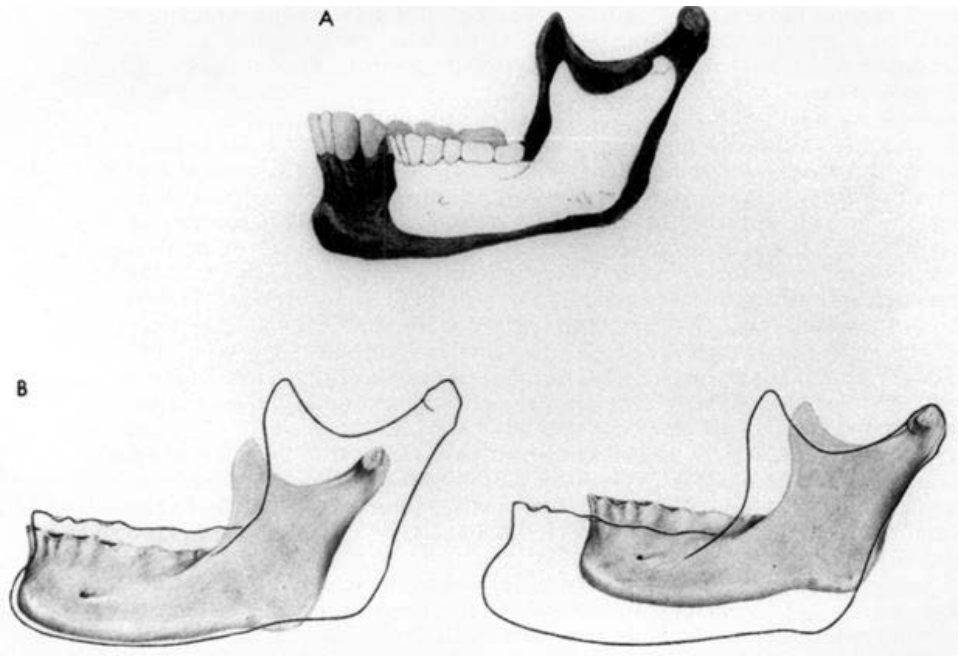


FIGURE 2-17.
(From Moyers, R. E., and D. Enlow. *Growth of the Craniofacial Skeleton*. In: *Handbook of Orthodontics,* 4th Ed. Chicago, Mosby-Year Book, Inc., 1988, with permission.)

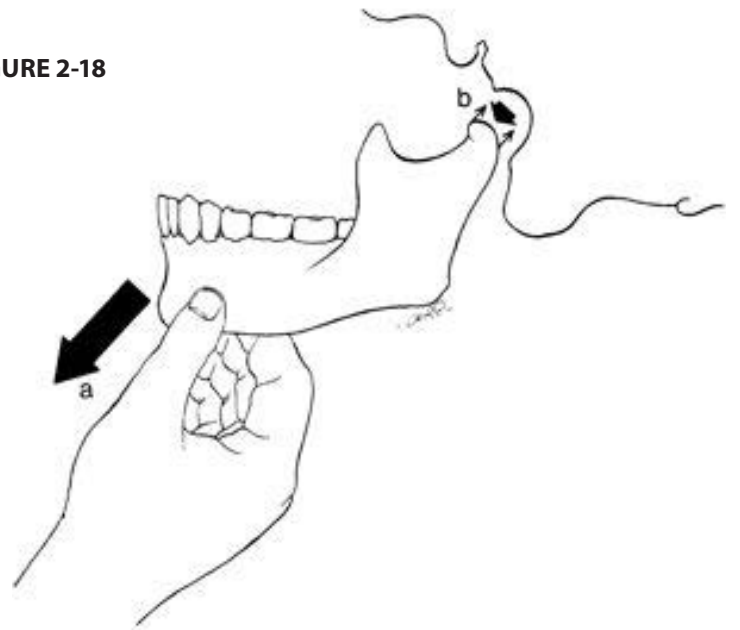
Is the forward and downward displacement movement of the mandible accomplished by a **shove** against the articular surface caused by the growth of the condyle, or conversely, by a **carry** of the entire mandible away from the basicranium[§] by mechanical forces (described in following chapters) extrinsic to the mandible itself (Fig. 2-18)? If the latter is true, bone remodeling follows secondarily (but virtually simultaneously) at the condyle and entire ramus to maintain constant contact with the temporal bone. As "force *a*" carries the mandible anteriorly and inferiorly, the condyle is **triggered to respond** in response to mutual developmental and functional signals by an equal amount of growth at *b*.

§ The proper anatomic term in the context used here is **basicranium**, not "cranial base." The latter, more properly, is a radiographic term used in cephalometry and has a two-dimensional meaning with "collapse" to the midline. **Basicranium**, however, connotes the entire cranial floor, including the lateral parts where the condyles articulate, which is important to the present discussion.

Is condylar growth thus the active cause of displacement (a "push" or "thrust") or the passive response to it? This was a heated controversy for many years. Current theory is outlined next.

In summary, two basic modes of skeletal movement take part in the growth of the face and neurocranium. **Remodeling** involves deposition of bone on any surface pointed toward the direction of enlargement of a given area; resorption usually occurs on the opposite side of that particular bony cortex (or cancellous trabecula). **Displacement** is a separate movement of the **whole bone** by some physical force that carries it, in toto, away from its contacts with other bones, which are also growing and increasing in overall size at the same time. This two-phase remodeling-displacement process takes place virtually simultaneously. The displacement movement is presently believed by many researchers to be the pacemaking (primary) change, with the rate and direction of bone growth representing a transformative (secondary) response. It can be argued, however, that the terms primary and secondary are biologically inappropriate for these two processes. Rather, they are each respondents to common signals that separately but simultaneously activate both to operate in unison. A widespread symphony of such interdependent movements proceeds throughout the craniofacial complex throughout growth.

FIGURE 2-18



There are, however, two different kinds of displacement that utilize these same two commonly used terms—primary and secondary. The word application here seems appropriate, since there is a basic difference in the source of movement. In **primary displacement**, the process of physical carry takes place in conjunction with a bone's **own** enlargement (Fig. 2-19). Two principal remodeling vectors in the maxilla, for example, are posterior and superior. As this occurs, the whole bone is displaced in opposite anterior and inferior directions. Primary displacement

produces the "space" within which the bone continues to enlarge. The amount of this primary displacement exactly equals the amount of new bone deposition that takes place within articular contacts. The respective directions are always opposite in the primary type of displacement. Because primary displacement takes place at an interface with other, contiguous skeletal elements, **joint contacts** are thus important growth sites involved in this kind of remodeling change.

In **secondary displacement**, the movement of a bone and its soft tissues is not directly related to its own enlargement. For example, the anterior direction of growth by the middle cranial fossae and the temporal lobes of the cerebrum **secondarily** displaces the entire nasomaxillary complex anteriorly and inferiorly (Fig. 2-20). Midfacial growth and enlargement itself, however, is not "primarily" involved in this particular kind of displacement movement. Thus, as any bone develops, remodels, and becomes displaced in conjunction with its own growth process, it is also secondarily displaced, in addition, resulting from the growth of **other** bones and their soft tissues. This can have a "domino effect." That is, growth changes can be passed on from region to region to produce a secondary (spinoff) effect in areas quite distant. Such effects are cumulative.

Note that much of the anterior part of the midfacial region is **resorptive** in nature (see Fig. 1-3). Yet the face grows **forward**. How can this be? The face does not simply "grow" directly anteriorly. The forward movement is a **composite** result of growth changes (1) by resorption and deposition that cause the maxilla to **enlarge backward** and (2) by primary and secondary displacement movements that cause it to be **carried forward**. The resorptive nature of the anteriorly facing surface of the premaxilla is concerned primarily with its downward, not forward, remodeling, as explained in Chapter 5.

To illustrate the composite nature of these different growth processes, the growth of the arm is used as an analogy (Fig. 2-21). The tip of the finger moves away from the shoulder as the whole arms increases in length. Most of this growth movement of the finger, of course, is not a consequence of growth at the fingertip itself. The aggregate summation of linear growth increments by all the separate bones in the arm at each particular interface between the phalanges, carpals, metacarpals, radius, ulna, humerus, and scapula is involved. The contribution by the tip of the terminal phalanx is only a relatively small part of the total. It is the secondary displacement effect produced by all the **other** bones in the arm that causes most of the growth movement of the fingertip, not its own remodeling and primary displacement.

Similarly, the greater part of the growth movement of the tip of the premaxilla is produced by the growth expansion of all the bones behind and above it and by growth in other parts of the maxilla. The premaxillary tip itself contributes only a very small part of its own forward growth movement. The enlargement of the maxilla proper and the frontal, ethmoid, occipital, sphenoid, lacrimal, vomer, and temporal bones and all of their soft tissues provides an aggregate expansion, the sum of which is the basis for most of the total forward movement of the premaxilla and its teeth. (It contributes somewhat more to its own downward movement, however, as illustrated in Chapter 5.) Keep in mind, also, that the biomechanical basis for these primary and secondary displacement growth movements is actually

FIGURE 2-19

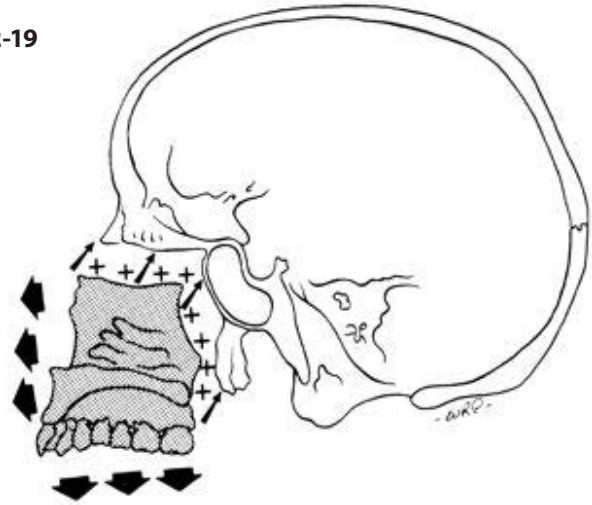
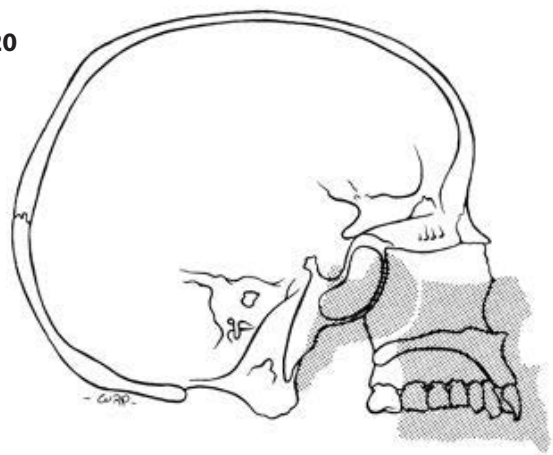


FIGURE 2-20



the "carry effect" produced by the expansion of the soft tissues associated with the bones, not a "pushing effect" of bones against bones.

Understanding of these concepts can be difficult for a beginning student. This is because it is natural to presume that (1) any given growing region is mostly responsible within itself for its own growth, and (2) since the maxilla "grows forward and downward," the forward/downward pointing area is where it should seem logically to "grow."

The points made in the previous paragraphs are basic and should be understood from day one in any postdoctoral specialty training program requiring an understanding of facial development. If one is to "work with growth" and has presumed that "growth" is exclusively within any given region itself rather than substantially elsewhere, a false start has been made that will seriously handicap the professional.

The factor of secondary displacement, as just seen, is a fundamental part of the overall process of craniofacial enlargement. Growth effects of skeletal parts far removed are passed on, bone by bone, to become expressed on the resultant topography of the face. Cranial floor-facial growth imbalances contribute materially to variable alignments and multitudes of positionings of the different facial bones. Secondary displacement is one of several basic factors involved in the developmental basis for malocclusions and most types of facial dysplasias. In Figure 2-22, note, for example, how a remodeling rotational alignment of the middle cranial fossae, in the manner shown, has the secondary displacement effect of maxillary retrusion and mandibular protrusion.

Growth Rotations

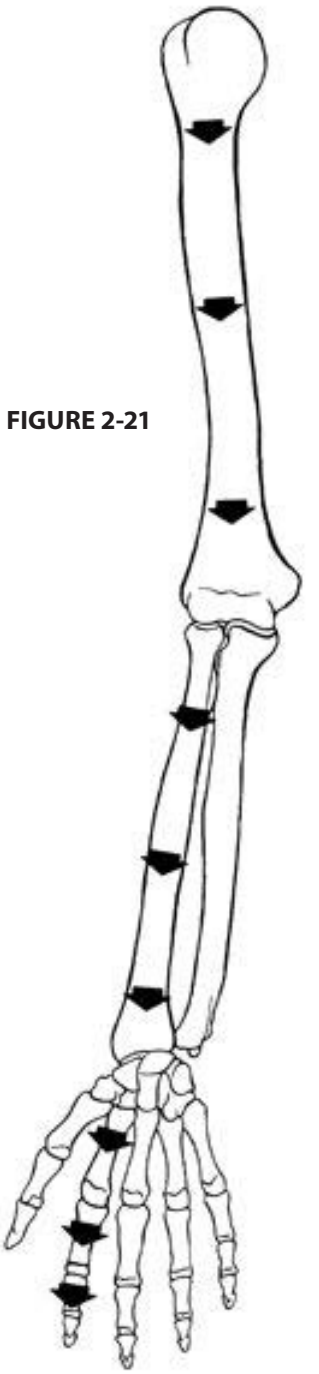


FIGURE 2-21

The subject of developmental **rotations** is a major consideration. Although confused by a jumble of terminologies in the literature, there are, simply, two basic categories of rotations: (1) remodeling rotations and (2) displacement rotations. Thus, rotational movements conform, understandably, to the same two categories of growth movements as described above.

While there are countless examples of **remodeling rotations** throughout the craniofacial complex, several in particular have considerable clinical significance. Refer to Figure 2-5.

A principal anatomic function of the mandibular ramus, in addition to providing insertion for masticatory muscles, is to properly position the lower dental arch in occlusion with the upper. To do this, it usually becomes more upright in alignment as development proceeds, closing the ramus-to-corpus ("gonial") angle. (See Chapter 4 for more details.) It is primarily remodeling of the ramus, not the corpus, that is responsible, and it is a combination of remodeling fields that carries out the **remodeling rotation** of the ramus, as illustrated in Figure 4-13. As this growth change proceeds, the entire mandible can also become rotated more downward and backward or upward and forward (as determined by the vertical height of the developing nasomaxillary complex.) See Fig. 10-14. This is a **displacement rotation** of the mandible as a whole as its ramus simultaneously rotates to a (usually) more closed position by an adjustive **remodeling** rotation.

A second example is the maxilla and its palate. The whole nasomaxillary complex is rotated by **displacement** in either a clockwise or counterclockwise direction, depending on growth activities of the overlying basicranium and also the extent of growth by the sutural system attaching the midface to the cranial floor. This would result in a canting and misfit of the palate and

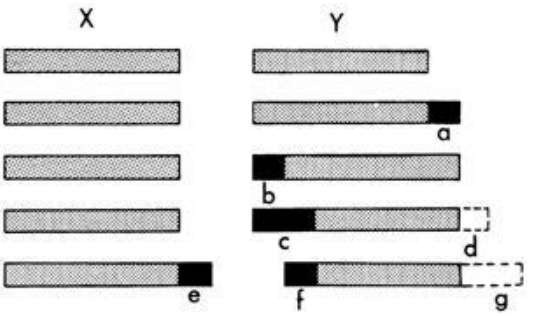
maxillary arch into either open or deep bite positions. However, the remodeling fields pictured in Figure 2-11 can become modified to provide adjustment by producing a counter-direction palatal **remodeling** rotation. This involves either gradients of depository (+) and resorptive (-) activity or actual reversals of remodeling fields along the nasal and oral sides of the palate to offset and compensate for the direction and magnitude of the whole-maxilla displacement rotation. (Note: dental and alveolar adjustments are also involved, as explained in Chapter 10.)

FIGURE 2-22

REMODELING AND DISPLACEMENT COMBINATIONS

Both primary and secondary displacements as well as remodeling are involved in the multiple-direction growth movements of all bones. A great many different combinations of all three processes are found throughout the craniofacial complex. As schematized in Figure 2-23, it is seen that essentially comparable results can be produced by quite different developmental combinations. It is the task of diagnosis and treatment planning to determine just which combination is at issue in any given real-life situation. Bones *X* and *Y* are in articular contact (as by a suture, condyle, or synchondrosis). A growth increment by bone deposition *a* produces a similar end-effect as deposition at *b*, with accompanying primary displacement of the whole bone to the right. Or increment *c* is added at the contact interface, with accompanying primary displacement of the whole bone to the right. Resorption at *d* occurs, however, producing an end-result equivalent to the two examples above. Or, secondary displacement of segment *Y* is caused by separate segment *X*, owing to growth addition *e*. Primary displacement accompanies growth at *f*. With resorption at *g*, it is thus seen that this combination also produces end-results similar to all the examples above.

FIGURE 2-23



The analysis of composite growth changes is always difficult in headfilm evaluation because, as just seen, the **same** growth results can be theoretically attained by many different combinations of remodeling and displacement. The purpose of Chapter 3 is to analyze just which of the many hypothetical combinations actually take place in each of the many regions of the face and cranium.

Note this important point. The word "growth" is a loose term that we all use, quite properly, when a more descriptive meaning is not needed.

However, in many instances, a specific and precise meaning should indeed be used. For example, it is frequently heard that some given clinical procedure "stimulates growth." One should always attempt to specify, whenever appropriate, just what kind of "growth" is indeed involved. Remodeling? Primary or secondary displacement? A particular combination? The biologic reason is apparent. If one is to control the growth process, just what is to be controlled must be understood, and the specific local targets involved must be identified. Trying to understand, biologically, how a functional regulator or an activator actually works, explanations that take into account these different movement types, and the specific biologic targets of each, are seldom encountered in the literature.

Superimposing Headfilm Tracings

The conventional method used to show facial "growth" is to superimpose serial headfilm tracings on the cranial base, as shown in Figure 2-24. Sella and a plane from sella to nasion are usually used for registration of the superimposition. Tracings are ordinarily employed instead of the headfilms themselves because superimposed x-ray films pass insufficient light.

Superimposing on the midline cranial base demonstrates the "downward and forward" (one of the most common clichés in facial biology) expansion of the whole face relative to the cranial base. Great caution must be exercised, however; one must understand possible misrepresentations of just what this really shows, because multiple, complex combinations of regional remodeling and primary and secondary displacement are all involved. This is the subject of Chapter 3.

First this method of superimposition is appropriate and valid because we all naturally tend to visualize facial enlargement in **relation** to the neurocranium (and brain) behind and above it. That is, the characteristically small face and larger brain of early childhood **change** progressively in respective proportions.

Then, as the facial airway and oral region progressively enlarge, the face grows and develops rapidly throughout the childhood period. The size and shape of the brain and the cranial vault also continue to develop, but at a much lesser pace and much less noticeably. The parent **sees** the structural transformations of the child's face, month by month, as it "catches up" with the earlier maturing calvaria and forehead (Fig. 2-25). Superimposing on the cranial base (sella, etc.) thereby represents what we all actually visualize by direct observation as the face progressively enlarges.

Superimposing headfilm tracings "on the cranial base" is not valid, however, if the following **incorrect** assumptions are made:

1. The incorrect assumption that the cranial base is truly stable and unchanging. It is not. This notion has often mistakenly been made. The floor of the cranium continues to grow and undergo remodeling changes throughout the childhood period (although this is much more marked in some regions than others at different age levels). Properly taken into account, however, this is not necessarily a factor, since the purpose, really, is only to show facial growth changes **relative** to the cranial base, whether or not it is actually stable.

2. The incorrect assumption that "fixed points" actually exist (i.e., anatomic landmarks that do not move or remodel). All surfaces, inside and out, undergo continued, sequential displacement movements and remodeling changes during morphogenesis (with the exception of no size changes by the ear ossicles once formed; see previous editions of the present book). Although the **relative** position of some landmarks can remain constant, the structures themselves actually experience significant growth movements and remodeling changes along with everything else. Sella has often been presumed to be a true "fixed" point or one that represents the "zero growth point" in the head. Of course, it is not. Sella changes during continued growth. This, however, does not invalidate the use of sella to represent a registration point on the cranial base **if** these various considerations are properly taken into account. *Nasion* is another such landmark. So many marked growth and remodeling variations are associated with this point relating to age, sex, headform types, and ethnic and individual differences, however, that the use of nasion as a cephalometric landmark requires great caution. (Note: There are other basic reasons why points such as nasion and sella are misleading if improperly used, as explained in Chapter 5.)

3. The incorrect assumption that the traditional "forward and downward" picture of facial enlargement, seen when serial tracings are superimposed on the cranial base, represents the **actual mode of facial growth**. Many workers have believed, quite incorrectly, that this is how the face really grows, that is, the facial profile of the younger stage expands straight to the profile of the older stage by direct "growth" and expansion from one to the other. This has been one of our most common misconceptions and one of the most difficult to overcome. The face is a multifactorial, cumulative composite of diverse, multi-directional changes throughout the head, the summation of which produces the "forward and downward" expansion seen in the overlay.

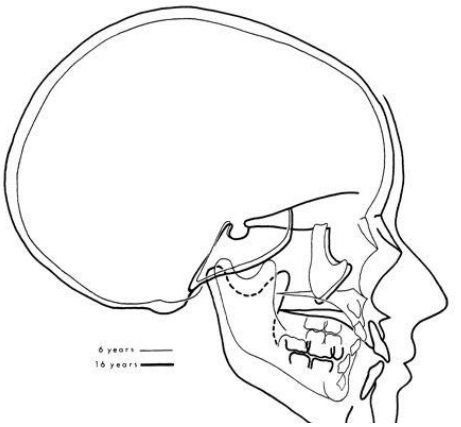
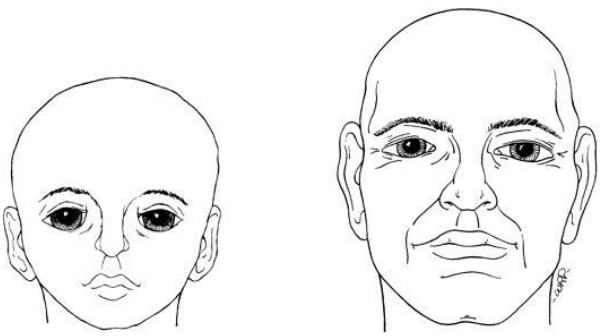


FIGURE 2-24.
(From Enlow, D. H.: *The Human Face*. New York, Harper & Row, 1968, with permission.)

FIGURE 2-25



As mentioned above, superimposing headfilm tracings on the cranial base shows the **combined** results of (*a*) deposition and resorption (remodeling) and (*b*) primary and secondary displacement **relative** to a common reference plane (such as sella-nasion). The superimposing procedure, however, does not provide an accurate representation for either remodeling or displacement in most facial regions. Note that the two placements of the mandible in the preceding Figure 2-24, for example, do not properly represent either its growth by deposition and resorption (*B*, left) or its primary displacement (right), as shown in Figure 2-17. The overlay positions for the mandible in Figure 2-24 (and all other facial bones as well) simply indicate their successive **locations** at the two age levels represented relative to the cranial base, not their actual modes of development. To presume the latter is to completely misunderstand how any treatment procedure actually works, and to miss as well the rationale for a true morphogenic diagnosis and treatment plan.

One basic problem always encountered with routine methods of superimposing headfilm tracings on the cranial base is that the **separate** effects of growth by disposition and resorption and by displacement are **not distinguishable**. This is an important consideration. The purpose of the following chapters is to demonstrate these separate effects and to explain how the process of craniofacial development is really carried out.

3

The Developmental Sequence

This short chapter summarizes the overall pattern of combined remodeling and displacement movements representing the essence of the "big picture." Keep in mind that, although the basic growth changes are illustrated using two dimensional line drawings of the bones their actions are representative of all the other growing parts and could be extended to three dimensions. Reducing the data to two dimension drawings derived from cephalometric radiographs, follows the traditional approach used in orthodontics diagnosis and treatment planning. The following chapters elaborate on the underlying biology associated with the morphologic changes described in this chapter.

The multiple growth processes in all the various parts of the face and cranium are described separately as individual "regions" or "stages." The sequence begins arbitrarily with the maxillary arch. Changes are then shown for the mandible, followed by growth changes in parts of the cranium and then those of the other regions, one by one. Keep in mind that even though these regional growth processes are presented here as a sequence of separate stages, in our patients they all take place simultaneously.

Growth increases are shown in such a way that the same craniofacial form and pattern are maintained throughout; that is, the proportions, shape, relative sizes, and angles are, purposely, essentially unaltered to the extent possible as each separate region enlarges. Thus, the geometric form of the whole face for the first and last stages is the same; only the overall size has been changed. Each sequential region incorporates all the changes that precede it. The final stage is a cumulative composite.

Facial and cranial enlargement, in which form and proportions remain constant, constitutes "balanced" growth. However, a perfectly balanced mode of growth in all the parts of the face and cranium never occurs in real life. Because regional imbalances always occur during the actual developmental processes, changes in facial shape and form always take place as the face grows into adulthood. That is, imbalances in the developmental process lead to corresponding imbalances in structure. Most of these "imbalances" are perfectly normal and are a regular part of the developmental and maturation process. They are unavoidable because of the complex design of the craniofacial composite in which many different parts develop at different times, in different directions at different velocities and perform many diverse functions. Making everything actually fit requires certain normal "imbalances." These factors are why the face of a child undergoes sequential

alterations in profile and in facial proportions as developmental differentiation progresses.

The reason the facial growth descriptions that follow are presented first as a "balanced" series is twofold. First, they will show just what constitutes the concept of "growth balance" itself and how to understand what this actually means. Second, in order to be able to recognize and explain facial imbalances, normal and abnormal, one must know what constitutes deviations from the balanced mode of development, that is, exactly where disproportions develop to cause a given facial pattern, and how much is involved in terms of dimensional and angular departures from balanced growth. Only by understanding the balanced process can one accurately identify, measure and, importantly, account for the imbalanced changes. The face of each of us is the aggregate sum of all the many balanced and imbalanced craniofacial parts combined into a composite whole. Regional imbalances often tend to compensate for one another to provide functional equilibrium (growth works toward composite balance: Chapter 1). The process of compensation is a feature of the developmental process; it provides for a certain latitude of imbalance in some areas in order to offset the effects of disproportions in other regions. It is this natural tendency toward balance that results in the precise fit (within 6mm from Class II to Class III) of the first molar teeth despite large variations in craniofacial form and facial appearance.

The regional descriptions of the growth process outlined below are not randomly presented. Rather, a system is used that, in fact, is the same developmental plan utilized in the growth process itself. This is the counterpart principle of craniofacial growth. It states, simply, that the growth of any given facial or cranial part relates specifically to other structural and geometric "counterparts" in the face and cranium. For example, the maxillary arch is a counterpart of the mandibular arch. The anterior cranial fossae and the palate are counterparts, as are the palate and maxillary apical base. The middle endocranial fossa, mandibular ramus (it bridges the pharyngeal space established by the middle cranial fossa), and zygomatic arch (which bridges both the cranial fossa and the ramus) are all respective counterparts. These are regional relationships throughout the whole face and cranium. If each regional part and its particular counterpart enlarge to the same extent, balanced growth between them is the result. This is the key to what determines the presence or lack of balance in any region. Imbalances are produced by differences in respective amounts or directions of growth between parts and counterparts. Many part-counterpart combinations exist throughout the skull, and these provide a meaningful and effective way to evaluate the growth of the face and the morphologic relationships among all its structural components.

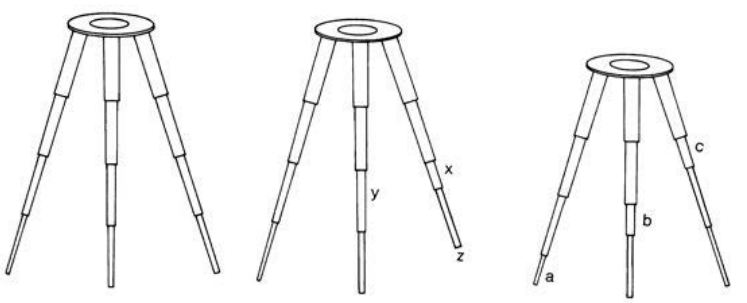
The "test" for a part-counterpart relationship in the face and cranium is not difficult. The question is simply asked: "If a given increment is added to a specific bone, or soft tissue part, where must an equivalent increment to be added to other bones or parts if the same form and balance are to be retained?" The answer to this question then identifies which other specific bones or parts of bones or soft tissue parts are involved as counterparts. This counterpart concept will be used repeatedly in this chapter as well as in following chapters dealing with facial variations and abnormalities. (See also page 159.)

Each regional growth change is presented as two separate processes. First, the changes produced by deposition and resorption (remodeling) are described and are shown by fine arrows in the illustrations. Second, the changes produced by displacement are described and are represented by heavy arrows. These two processes, it is understood, take place at the same time, but they must be described separately because their effects are quite different. Then the question asked is, "Where do counterpart changes also occur if the same pattern is to be maintained?" This identifies the next anatomic region, which is described in turn.

To illustrate the counterpart principle, an expandable photographic tripod is used here as an analogy (Fig. 3-1). The tripod has a series of telescoping segments in each leg; the length of each segment matches the length of its "counterpart" segments in the other two legs. If all the segments are extended to exactly the same length, the tripod retains geometric balance and overall symmetry. If, however, any one segment is not extended equal to the others, the leg as a whole is either shorter or longer, although the remainder of all the segments in that leg match their respective counterparts. One can thus identify which particular segment is different and determine the extent of imbalance. Segment x, for example, is short relative to y, thus causing a retrusion of z. In addition, the relative (not actual) length of a whole leg can also be altered by changing its alignment. A leg "rotated" into more vertical alignment, for example, increases the expression of that dimension without actually lengthening its real size.

Many other hypothetical combinations exist. For example, segments a, b, and c in Figure 3-1 are short with respect to their segment counterparts in the other legs. Overall symmetry is balanced, nonetheless, because of all these regional imbalances offset one another, and the total length of each leg is, therefore, the same.

FIGURE 3-1



Regional Change (Stage) 1

Note that two reference lines are used, a horizontal and a vertical,* so that directions and amounts of growth changes can be visualized (Fig. 3-2). The bony

* This vertical line is not arbitrary; it is the PM boundary, which is one of the most basic and important natural anatomic planes in the head (see Chapter 9). The horizontal line is the functional occlusal plane.

maxillary arch lengthens horizontally in a posterior direction (the fact that new bone is added in the posterior of the arch always comes as a surprise to those new in the business). This is schematized by showing a backward movement of the posterior border of the maxilla Note its new location behind the vertical reference line (Fig. 3-3).

The Pterygomaxillary fissure (PTM) is a historic cephalometric landmark used to identify the maxillary tuberosity, and it appears on cephalometric radiographs as an "inverted teardrop" produced by the gap between the pterygoid plates of the sphenoid bone and the posterior border of the maxilla.

The overall length of the maxillary arch has increased by the same amount that PTM moves posteriorly. Bone has been deposited on the posterior-facing cortical surface of the maxillary tuberosity. Resorption occurs on the opposite side of the same cortical plate, which is the inside surface of the maxilla within the maxillary sinus.

FIGURE 3-2

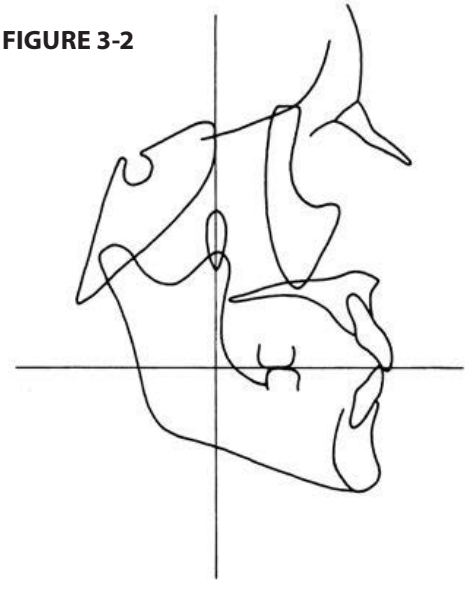
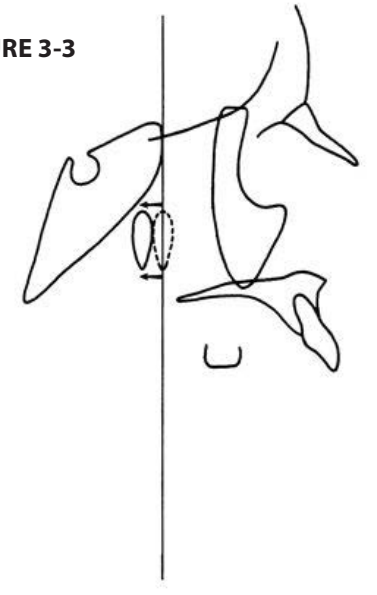


FIGURE 3-3

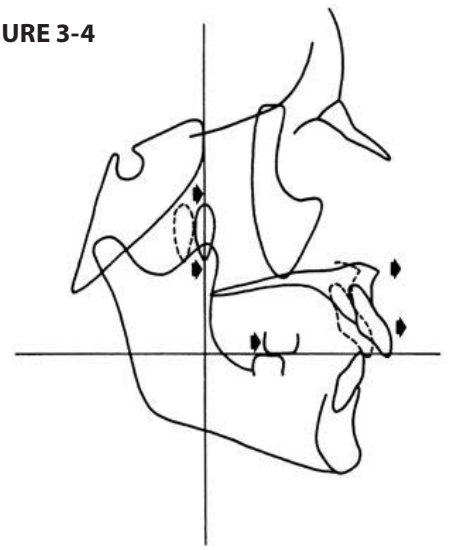


Regional Change (Stage) 2 –Maxillary Displacement

The preceding stage is the first of the two-part growth process described for each region, that is, remodeling by deposition and resorption. The second part involves displacement, described in the present stage (Fig. 3-4). As the maxillary tuberosity grows and lengthens posteriorly, the whole maxilla is simultaneously carried anteriorly. The amount of this forward displacement exactly equals the amount of posterior lengthening. Note that PTM is "returned" to the vertical reference line. Of course, it never actually departed from this line because backward growth (Stage 1) and forward displacement (Stage 2) occur at the same time. This is a primary type of displacement because it occurs in conjunction with the bone's own enlargement; that is, as the bone is displaced, it undergoes remodeling growth

that keeps pace with the amount of displacement. A protrusion of the forward part of the arch now occurs, not because of direct growth in the forward part itself, but rather because of growth in the posterior region of the maxilla as the whole bone is simultaneously displaced anteriorly. Displacement is a necessary action because the process of bony deposition CANNOT push the maxillary complex forward. It is biologically impossible for bones to push on other bones to cause displacement. So the question naturally arises, what is the source of the biomechanical force producing this maxillary displacement? The answer, in brief, involves the developmental expansion of all the enclosing soft tissues which, attached to the maxilla by Sharpey's fibers, as well as growth of the nasal septal cartilage attached to the premaxilla via the septopremaxillary ligament, carry the maxillary complex anteriorly. (See also Figs. 1-6, 5-1, and 5-2.)

FIGURE 3-4



Regional Change (Stage) 3 –Mandibular Corpus Lengthening

The question is now asked: "When the elongation of the maxilla in Stage 1 is made, where must equivalent changes also be made if structural balance is maintained?" In other words, what are the counterparts to the bony maxillary arch? Several are involved, including the upper part of the nasomaxillary complex, the anterior cranial fossa, the palate, and the corpus of the mandible. The mandible is described in this stage. The mandible is not to be regarded as a single functional element; it has two major parts, the corpus (body) and the ramus. These two parts must be considered separately because each has its own separate counterpart relationships with other, different regions in the craniofacial complex.

The bony mandibular arch relates specifically to the bony maxillary arch; that is, the body of the mandible is the structural counterpart to the body of the

maxilla. The mandibular corpus now lengthens to match the elongation of the maxilla, and it does this by a remodeling conversion from the ramus (Fig. 3-5). The anterior part of the ramus remodels posteriorly, a relocation process that produces a corresponding elongation of the corpus. What was ramus has now been remodeled into a new addition for the corpus. The mandibular arch lengthens by an amount that equals the remodeling of the maxillary arch (Stage 1), and both elongate in a posterior direction. However, note that the two arches are still offset; the maxilla is in a protrusive position even though upper and lower arch lengths are the same, as seen in Figure 3-6. A Class II type of relationship still exists between the maxillary and mandibular molars. The proper Class I position is seen in Stage 1; the mandibular posterior tooth shown in the diagram should normally be about one-half cusp ahead of its maxillary antagonist, as seen in Figure 3-2.

Regional Change (Stage) 4 –Mandibular Ramus Remodeling

The second of the two growth processes (i.e., first, growth by deposition and resorption, and second, displacement) will now be described. Remember that these two changes actually occur at the same time. The whole mandible is displaced anteriorly, just as the maxilla also becomes carried anteriorly while it simultaneously grows posteriorly. To do this, the condyle and the posterior part of the ramus remodel posteriorly (Fig. 3-6). This returns the horizontal dimension of the ramus to the same breadth present in Stages 1 and 2 above; to keep the ramus the same width, the amount of anterior ramus resorption is equaled by the amount of posterior ramus addition. This purpose is not to increase the width of the ramus itself, but to relocate it posteriorly resulting in lengthening of the corpus.

FIGURE 3-5

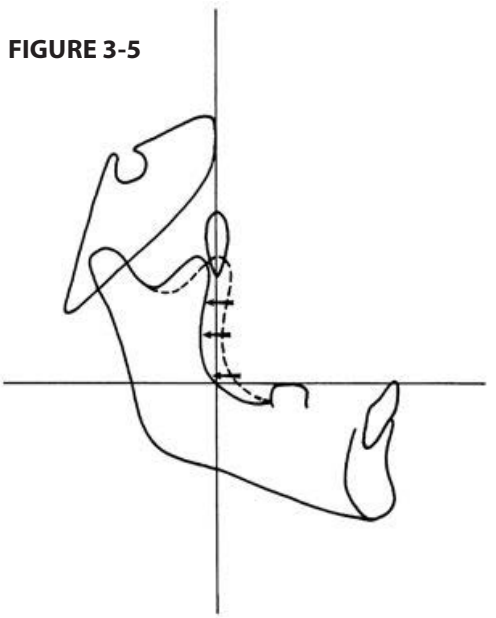
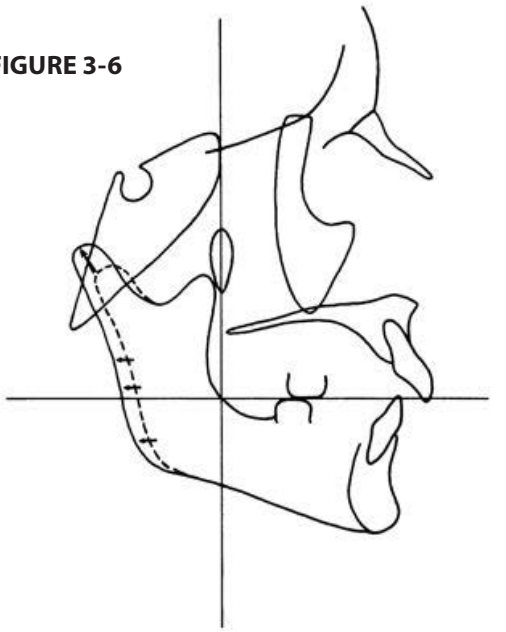


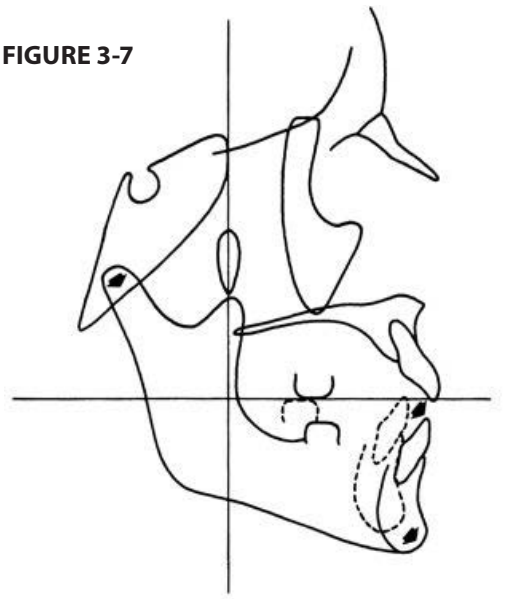
FIGURE 3-6



Regional Change (Stage) 5 –Mandibular Displacement

The whole mandible, now, is displaced anteriorly by the same amount that the ramus has relocated posteriorly (Fig. 3-7). This is the primary type of displacement because it occurs in conjunction with the bone's own enlargement. As the bone becomes displaced, it simultaneously remodels (the stage just described) to keep pace with the amount of displacement.

FIGURE 3-7



Note the following:

1. The corpus of the mandible elongates primarily in a posterior direction, just as the maxilla also lengthens posteriorly (Stage 1). It does this by remodeling from what was ramus into what then becomes a posterior addition to the mandibular arch. In this respect, mandibular arch elongation differs from maxillary arch elongation because the maxillary tuberosity is a free surface, unlike the posterior end of the mandibular corpus.

2. The whole ramus has moved posteriorly. However, the only actual change in horizontal dimension involves the mandibular corpus, which becomes longer. The horizontal dimension of the ramus remains constant during this particular remodeling stage (the widening of the ramus itself is part of another stage).

3. The anterior displacement of the whole mandible equals the amount of anterior maxillary displacement assuming everything is perfectly balanced (which is unlikely; see Chapter 10.) This places the mandibular arch in proper position relative to the maxillary arch just above it. The arch lengths and the positions of the maxilla and mandible are now in balance, and a Class I position of the teeth has been "returned."

4. Note, however, that the obliquely upward and backward direction of ramus remodeling must also lengthen its vertical dimension in order to provide for horizontal enlargement. This separates the occlusion (contacts between the upper and lower teeth) because the mandibular arch is displaced inferiorly as well as anteriorly.

5. Like the displacement of the maxilla, this type of mandibular displacement is primary because it takes place in conjunction with the bone's own enlargement. The biomechanical force causing this displacement movement involves the developmental expansion of the soft tissues attached to the mandible by Sharpey's fibers which carry the mandible forward with concomitant endochondral bone formation in the area of the mandibular condyle to help maintain the mandible in the displaced position. Thus the displacement of the mandible occurs in two closely choreographed steps. The expansion of the associated soft tissues provides the tissue separating force necessary to displace the mandible and condylar growth locks the change in place.

6. In summary thus far, the increment of backward growth at the maxillary tuberosity (Stage 1), the amount of forward displacement by the whole maxilla (Stage 2), the extent of remodeling on the anterior part of the ramus and the amount of corpus lengthening (Stage 3), the increment of backward growth by the posterior part of the ramus (Stage 4), and the amount of forward displacement of the whole mandible (Stage 5) are all precisely equal in this "balanced" sequence of growth. What happens when they are not all exactly equal (as usually happens), or when differentials in timing occur, or if developmental "rotations" occur to cause variations in alignment (which change the expression of actual dimensions) is described later.

Regional Change (Stage) 6 –Middle Cranial Fossa Growth

While all of the growth and remodeling changes described in the preceding stages have been taking place, the dimensions of the temporal lobes of the cerebrum and the middle cranial fossae have also been increasing at the same time (Fig. 3-8). This is done by resorption on the endocranial side and deposition of bone on the ectocranial side of the cranial floor. The spheno-occipital synchondrosis (a major cartilaginous growth site in the cranium up to age 14) provides endochondral bone growth in the midline part of the cranial floor.† The total growth expansion of the middle fossa would now project it anteriorly beyond the vertical reference line, except that this line itself is moved in the next stage.

† Note the change in the position of the sella turcica. This is a highly variable structure, however, and other patterns of remodeling movements are also common. See Chapter 5.

Regional Change (Stage) 7 –Secondary Displacement Nasomaxillary Complex

All cranial and facial parts lying anterior to the middle cranial fossa (in front of the vertical reference line) become displaced in a forward direction as a result (Fig. 3-9). The whole vertical reference line moves anteriorly to the same extent that the middle cranial fossa expands in a forward direction. This is because the line represents the anterior boundary between the enlarging middle cranial fossa and all of the cranial and facial parts in front of it. The maxillary tuberosity remains in a constant position on the vertical reference line as this interface line moves forward. The forehead, anterior cranial fossa, cheekbone, palate, and maxillary arch all undergo protrusive displacement in an anterior direction. This is a secondary type of displacement because the actual enlargement of these various parts is not directly involved. They are simply moved anteriorly because the middle cranial fossa behind them expands in this direction. The floor of the fossa, however, does not push the anterior cranial fossa and the nasomaxillary complex forward. Rather, they are carried forward as the frontal and temporal lobes of the cerebrum enlarge by respective growth increases. That is, the tissue separating biomechanical force is generated by the growth of the brain. The nasomaxillary complex, suspended by sutures from the anterior cranial fossae and frontal lobes, is thus carried anteriorly as the combined frontal and temporal lobes progressively expand.

FIGURE 3-8

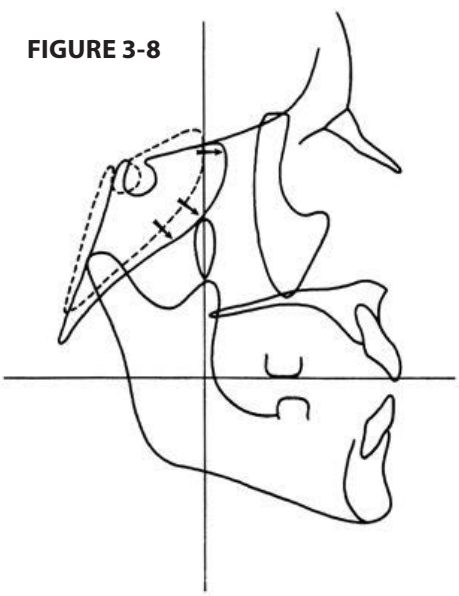
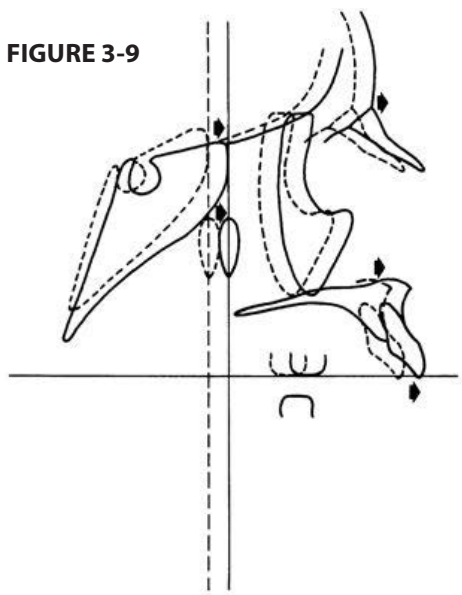


FIGURE 3-9



Regional Change (Stage) 8 –Secondary Displacement Mandible

The expansion of the middle cranial fossa, just described, also has a displacement effect on the mandible (Fig. 3-10). This too is a secondary type of displacement. The extent of the displacement effect, however, is much less than that for the maxilla. This, importantly, is because the greater part of middle cranial fossa growth occurs in front of the condyle and between the condyle and the maxillary tuberosity. The spheno-occipital synchondrosis also lies between the condyle and the anterior boundary of the middle cranial fossa. Thus, the extent of maxillary protrusive displacement far exceeds the amount of mandible protrusive displacement caused by middle fossa enlargement. The result is an offset horizontal placement between the upper and lower arches. The upper incisors show an "overjet," and the molars are in a Class II position, even though the mandibular and maxillary arch lengths themselves are matched in respective dimensions. Sella-nasion (again, a historic cephalometric plane) should not be used to represent the "upper face" or "anterior cranial base" in comparisons with the entire mandibular dimension, ramus and corpus, as is often done. The comparison is invalid because dissimilar effective spans (counterparts) are being compared and because sella-nasion itself does not represent any anatomically meaningful dimension, either for the cranial base or for the upper face.

FIGURE 3-10

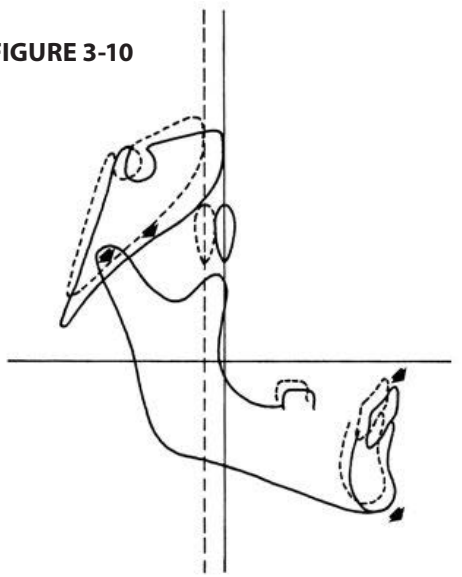
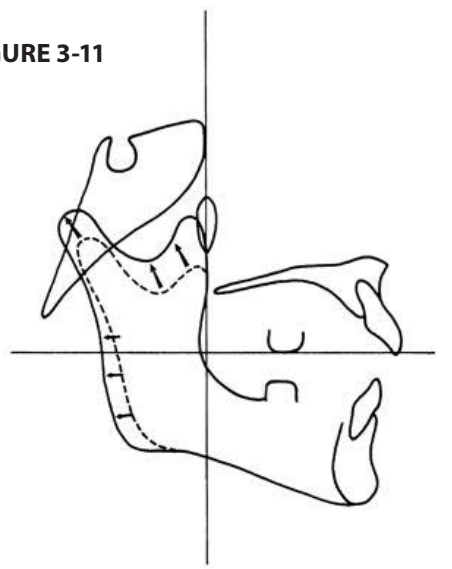


FIGURE 3-11



Regional Change (Stage) 9 –Counterparts: MCF-Ramus

The question is now asked: "When this change in the middle cranial fossa takes place, where must an equivalent change also occur if balance is to be sustained?" This identifies the "counterpart" of the middle fossa and shows where facial growth must take place to match it.

Just as the lengthening of the middle cranial fossa places the maxillary arch in a progressively more anterior position, the horizontal growth of the ramus places the mandibular arch in a like position. What the middle cranial fossa does for the maxillary body, in effect, the ramus does for the mandibular body. The ramus is the specific structural counterpart of the middle cranial fossa. Both are also counterparts of the pharyngeal space. The skeletal function of the ramus is to bridge the pharyngeal space and the span of the middle cranial fossa in order to place the mandibular arch in proper anatomic position with the maxilla. The anteroposterior breadth of the ramus is critical. If it is too narrow or too wide, the ramus places the lower arch too retrusively or too protrusively, respectively. This dimension and also the alignment must be just right. As will be described later, the horizontal dimension of the ramus can become altered during growth to provide intrinsic adjustments and compensations for morphogenic imbalances that may occur elsewhere in the craniofacial complex.

The horizontal extent of middle cranial fossa elongation is matched by the corresponding extent of horizontal increase by the ramus (Fig. 3-11). The horizontal (not oblique) dimension of the ramus now equals the horizontal (not oblique) dimension of the middle cranial fossa. The effective span of the latter, as it relates to the ramus, is the straight line distance from the cranial floor-condyle articulation to the vertical reference line. Recall that the ramus was previously involved in remodeling changes associated with corpus elongation (Stage 4), but the actual breadth of the ramus was not increased during that particular stage. The present stage represents that increase and is considered separately here. Both stages proceed simultaneously.

Regional Change (Stage) 10 –Maintaining Vertical Balance

The entire mandible is displaced anteriorly at the same time that it remodels posteriorly (Fig. 3-12). The amount of the anterior displacement equals (1) the extent of posterior ramus and condylar growth (Stage 9); (2) the amount of middle cranial fossa enlargement anterior to the mandibular condyle (Stage 6); (3) the extent of anterior movement of the vertical reference line; and (4) the extent of resultant anterior maxillary displacement (Stage 7).

The oblique manner of condylar growth necessarily produces an upward and backward projection of the condyle with a corresponding downward as well as forward direction of mandibular displacement. The ramus thus becomes vertically as well as horizontally enlarged. This results in a further descent of the mandibular arch and separation of the occlusion (it was also previously lowered during Stages 5 and 8). The total extent of this vertical growth (Fig. 3-12) must match the total vertical lengthening of the nasomaxillary complex (Fig. 3-15) and the upward eruption and drift of the mandibular dentoalveolar arch (Fig. 3-18) if the same facial balance is to be achieved.

Note that the protrusion of the maxilla during Stage 7 has now been matched by an equivalent amount of mandibular protrusion. The molars have once again been "returned" to Class I positions, and the upper incisor has no overjet. Note also that the anterior border of the ramus lies ahead of the vertical reference line. The

"real" junction between the ramus and corpus, however, is the lingual tuberosity housing the last molar, not the "anterior border." The lingual tuberosity lies on the vertical reference line behind the anterior border, which overlaps this tuberosity (not shown in the figure because it cannot be seen in a lateral cephalometric radiograph; observe on a dry mandible or a three dimensional computed tomograph). This protruding overlap is an evolutionary result of the distinctive upright remodeling rotation of the ramus among higher primates relating to the more vertically elongate rotation of the midface (see Chapter 9) and the extension of a flange of the anterior border to accommodate the temporalis muscle.

Regional Change (Stage) 11

The floor of the anterior cranial fossa and the forehead grow by deposition on the ectocranial side with resorption from the endocranial side (Fig. 3-13). The nasal bones are displaced anteriorly. The posterior-anterior length of the anterior cranial fossa is now in balance with the extent of horizontal lengthening by its structural counterpart, the maxillary arch (Stage 1). Because these two regions have undergone equivalent growth increments, the profile retains its originally balanced form. (Actually, age differentials, both in time and amount together with male/female and headform differences, always occur, but our present purpose is to describe perfectly "balanced" growth.) The enlarging brain displaces the bones of the calvaria (domed skull roof) outward. Each bone enlarges by sutural growth. As the brain expands, the sutures respond by depositing new bone at the contact edges of the frontal, parietal, occipital, and temporal. This expands the perimeter of each. At the same time, bone is laid down on both the ectocranial and endocranial sides to increase the thickness.

The upper part of the face, which is the ethmomaxillary (nasal) region, also undergoes equivalent growth increments. This facial area increases horizontally to an extent that matches (if the same balance is retained) the expansion of the anterior cranial fossa above and the maxillary arch and palate below it. These areas are all counterparts to one another. The growth process involves direct bone deposition on the forward-facing cortical surfaces of the ethmoid, the frontal process of the maxillary, and the nasal bones. Most of the internal bony surfaces of the nasal chambers are resorptive. In addition, anterior displacement takes place in conjunction with growth at the various maxillary and ethmoidal sutures. The composite of these changes produces an enlargement of the nasal chambers in an anterior (and also lateral) direction.

Regional Change (Stage) 12

The vertical lengthening of the nasomaxillary complex, as with its horizontal elongation, is brought about by a composite of (1) growth by deposition and resorption, and (2) a primary displacement movement associated directly with its own enlargement. The latter is considered in a later stage. The combination of resorption on the superior (nasal) side of the palate and deposition on the inferior (oral) side produces a downward remodeling movement of the whole palate from

1 to 2 in Figure 3-14. This relocates it inferiorly, a process that provides for the vertical enlargement of the overlying nasal region. The extent of nasal expansion is considerable during the childhood period to keep pace with the enlargement of the whole body and lungs. (Note: The pattern and extent of downward palatal and maxillary arch remodeling often varies between the mesial and distal parts of the area, as described on page 39 in Chapter 2. This provides for a range of positional adjustments of the arch to compensate for developmental variations and displacement rotations.)

FIGURE 3-12

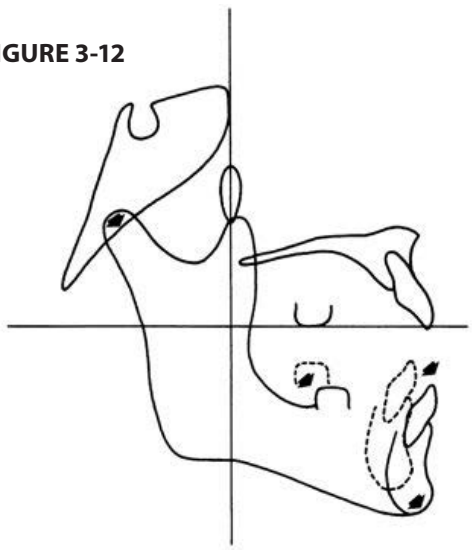


FIGURE 3-13

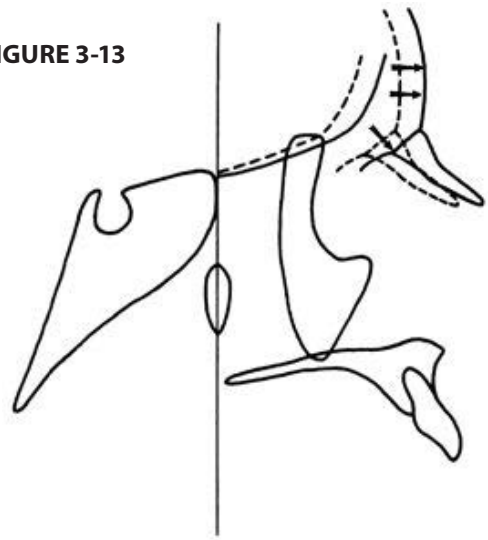
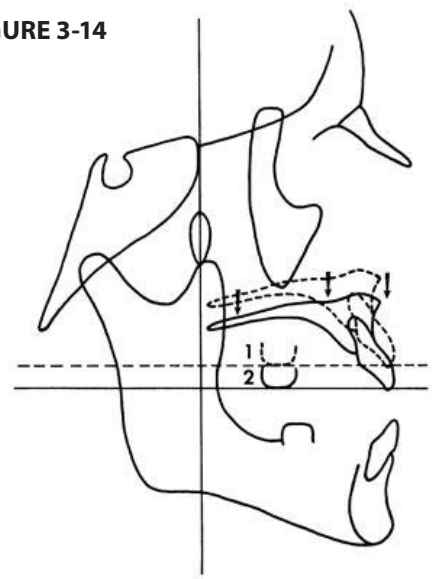


FIGURE 3-14



The anterior part of the bony maxillary arch has a periosteal surface that is resorptive (the face of the human, with reduced jaws, is the only species that has this). The reason is because this area grows straight downward, as schematized in Figure 2-11. In other species (including primates), the premaxillary region remodels forward as well as downward to produce a much more elongate, protrusive muzzle.

As seen in Figure 2-11, the labial (external) side of the premaxillary region faces mostly upward and away from the downward direction of growth, and it is thus largely resorptive. The lingual side faces toward the downward growth directions and is depository. The growth pattern also provides for the remodeling of the alveolar bone as it adapts to the variable positions of the incisors (Fig. 3-15).

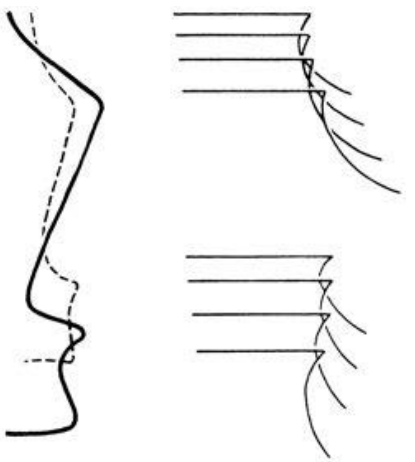


FIGURE 3-15.
(From Enlow, D. H.: *The Human Face*. New York. Harper & Row, 1968, p. 244, with permission.)

Regional Change (Stage) 13 –Vertical Drift

Vertical growth by displacement is associated with bone deposition at the many and various sutures of the maxilla where it contacts the multiple, separate bones above and behind it. Bone is added at these sutures by amounts equalling whole maxillary displacement inferiorly (Fig. 3-16). The addition of new sutural bone does not "push" the maxilla downward, as presumed in years past. Rather, the maxilla is carried inferiorly by the physical growth forces of enclosing soft tissues (See Fig. 1-7). This is accompanied by bone deposition in the sutures responding to mutual growth signals relating to both displacement and remodeling. New bone is thus simultaneously laid down on the sutural edges keeping the bone-to-bone junction intact. The increment of bone growth in the suture exactly equals the amount of inferior displacement of the whole maxilla. This is primary displacement because it takes place in conjunction with the bone's own enlargement.

Of the total extent of downward movement by the palate and maxillary arch, that part from 2 to 3 is produced in association with sutural growth and primary displacement (Fig. 3-17). The part form 1 to 2, which can be about half of the total (depending on palatal remodeling rotations), is direct relocation by resorptive and depository remodeling. Similarly, the movement of the teeth from 2 to 3 is by the downward displacement of the whole maxilla, carrying the entire dentition passively with it. The movement from 1 to 2 is produced by each tooth's own movement as bone is added and resorbed on appropriate lining surfaces in each socket. This is the vertical drift of the tooth, a process that is accompanied by the same deposition and resorption of alveolar bone that works with the familiar "mesial drift" of the dentition (see Chapter 5). Vertical drift takes place in addition to eruption, which is a separate growth movement. The vertical drift process is important to the clinician because it provides a great deal of growth movement to "work with" during treatment.

FIGURE 3-16

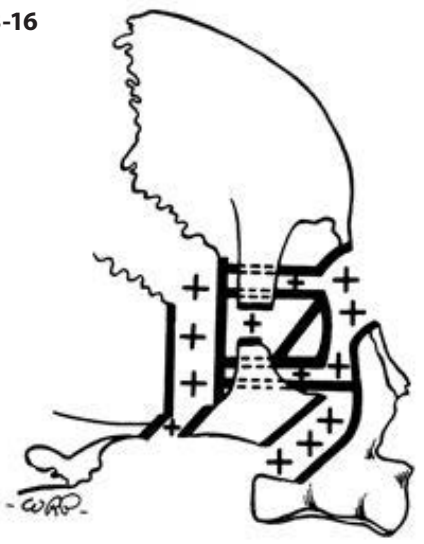
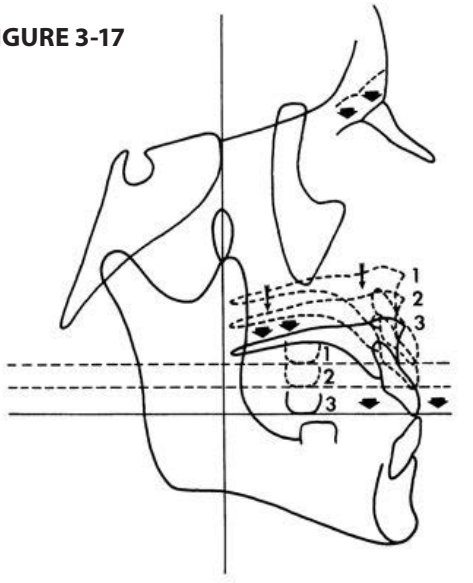


FIGURE 3-17



"Vertical drift" is a most significant concept that somehow has been bypassed in the dental curriculum and literature. For dentists, orthodontists, and other craniofacial practioners, this concept deserves day-one attention because of its clinical significance. Historically, vertical tooth movement has been called simply "eruption," which it is not and which misses the point entirely.

Tooth movement from 2 to 3 can also be clinically influenced. This involves the use of special appliances intended to either augment or retard the displacement movement of the entire nasomaxillary complex or to alter their directions. This in turn causes remodeling changes in the size or shape of the whole maxilla or of other separate bones (in contrast to remodeling of the alveolar bone supporting the teeth). Refer to the "Two Basic Clinical Targets" section in Chapter 1. In the nasomaxillary complex orthopedic forces can influence both displacement and vertical drift. This is an important concept to understand because although craniofacial imbalances usually involve a variety of facial parts and counterparts clinical treatment usually

targets the imbalance at the level of the maxilla and mandible. For example, most class II malocclusions have components of mandibular retrognathia and maxillary prognathia. Since the maxilla is more reposive to intervention it is important in class II's to eliminate any portion of the imbalance that is related to maxillary protrusion. Likewise, in class III patients the clinician should attempt to eliminate any contributions to the malocclusion related to maxillary retrognathia. Indeed, a good understanding of this basic tenet is critical to successful treatment.

Another significant concept relates to the reason why the palate and maxillary arch are subject to both remodeling and displacement (1 to 2 and 2 to 3). Figures 2-11 and 3-14 illustrate the palate and maxillary arch moving inferiorly in an idealized manner, with the anterior and posterior regions remodeling downward to the same extent. Mesiodistal variations, however, are common. In displacement movements, a clockwise or counterclockwise rotation of the whole palate and arch often occurs. In conjunction with this, the remodeling movement (1 to 2) can compensate by producing an opposite direction of rotation, thereby leveling and fine-tuning the palate into definitive adult position. This, indeed, represents a primary function of the remodeling phase of the composite downward growth process. That is, selective remodeling in the anterior versus posterior parts can serve to adjust and counteract rotations produced during primary nasomaxillary displacement as well as secondary displacement rotations caused by growth of the middle and anterior cranial fossae.

Awareness of the distinction between the two categories of movements 1 to 2 and 2 to 3 should also be highlighted in any orthodontic or surgical training program. The clinician addresses one or the other in every patient, or some combination of both. Either the magnitude or the direction can be influenced by substituting "clinical control" for nature's own intrinsic control. The underlying biologic process actually producing the movements is the same, however, whether intrinsic or clinical. If the potential for growth movement does not already exist, as in an adult, this movement must be clinically induced in addition to providing direction. Also, a different "stability" situation then exists, since no subsequent childhood facial growth is involved that can either lead to a new, composite equilibrium or, conversely, that could disrupt it (relapse). (See also Figure 1-1.)

Keep in mind, also, this key point: the teeth themselves have very little capacity for remodeling. They can, essentially, only be moved by the displacement process, either in conjunction with remodeling of an individual alveolar socket or displacement of the entire arch as a unit. It is the bone that must undergo any remodeling required. (See "Anterior Crowding" in Chapter 10.)

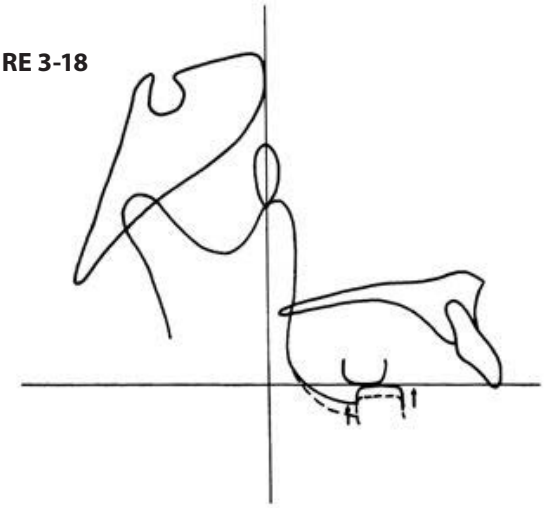
Regional Change (Stage) 14 –Mandibular Alveolar Drift

In three previous stages (5, 8, and 10), it was seen that the mandibular corpus becomes lowered by the vertical enlargement of both the ramus and the middle cranial fossa. Their combined vertical dimensions represent the growth counterpart of the vertical dimensions of the nasomaxillary complex and the dentition. In other words, the amount of vertical separation between the upper and lower arches caused by the vertical growth of both the middle cranial fossa and the

ramus must balance an equivalent amount of lengthening in the nasomaxillary complex and the dentoalveolar region of the mandible.

The maxillary arch has grown downward to Level 3 in Stage 13. Now, the mandibular teeth and alveolar bone drift upward to attain full occlusion (Fig. 3-18). This is produced by a superior drift of each mandibular tooth, together with a corresponding remodeling increase in the height of the alveolar bone. The extent of this upward growth movement plus that of the downward growth movement by the maxillary arch equals the combined extent of vertical remodeling by the ramus and middle cranial fossa if the pattern of the face is not changed. Note this factor: The extent of downward drift of the maxillary teeth can exceed the extent of upward drift by the mandibular teeth. Much less growth is thus available to "work with" in major orthodontic movements of the mandibular, as compared with the maxillary, teeth. However, there is a significant extent of vertical drift by the mandibular anterior teeth if a curve of Spee exists (See page 216). It is in fact within this vertical drift of the lower teeth and alveolus that favorable compensations can be introduced during clinical care. Because most orthodontic force systems are extrusive most compensations introduced therapeutically augment vertical. The use of temporary anchorage devices (microscrews) to reduce vertical drift should double the clinicians ability to therapeutically modify the alveolus during the vertical drift process.

FIGURE 3-18



Working with Growth and the Extraction Nonextraction Decision

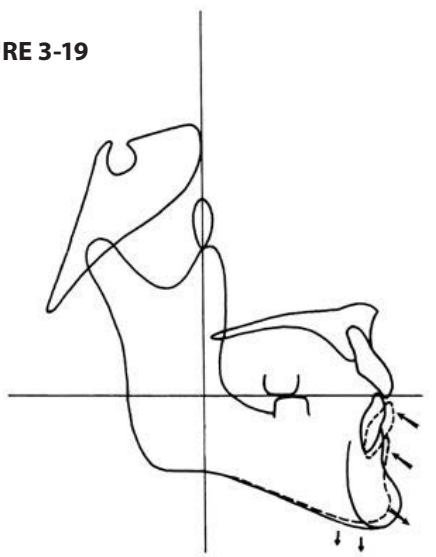
Much has been written about the clinician's ability to treat malocclusions without the removal of permanent teeth. A major factor that has been largely overlooked in this debate is the potential for therapeutic modification of the vertical remodeling process. As stated, vertical remodeling occurs in response to and coincident with vertical displacement of the mandibular corpus and nasomaxillary complex. The remodeling that occur around the individual sockets of maxillary and mandibular teeth can be modified by orthodontic forces. Indeed, it is this

modification that allows the clinician to develop arch length by redirecting the alveolar remodeling process toward areas of natural deposition. In the maxilla the area of the tuberosity and in the mandible the posterior portion of the corpus are areas that can be used to biologically augment arch length. Interestingly, it is in the case of deep bite malocclusion that differential vertical alveolar remodeling can most easily be harnessed by the clinician. The reason deep bite malocclusions are more easily treated in this manner is that most orthodontic treatment mechanics tend to "extrude" (vertical drift) posterior teeth. As posterior teeth are moved, vertical overbite is reduced. This treatment response is desirable in the deep bite case, but would be disastrous in the open bite patient. The relationship between vertical remodeling and deep bite may be one reason for the observed clinical relationship between deep bite and nonextraction treatment. Variable extents of vertical drift of the mandibular teeth, from distal to mesial, occur during normal facial development, just as for the maxillary teeth (described earlier). This functions to adjust the occlusal plane to compensate for various displacement rotations. When the orthodontist "extrudes" a tooth, it is the equivalent of the vertical drifting movement of the tooth together with remodeling of its companion alveolar socket, and the essential purpose is the same.

Regional Change (Stage) 15 –Anterior Dental Changes

While the upward movements of the mandibular teeth and remodeling of the alveolar sockets are taking place, remodeling changes also occur in the incisor alveolar region, the chin, and the corpus of the mandible (Fig. 3-19). The lower incisors undergo a lingual tipping (a "retroclination"), so that the uppers overlap the lowers for proper overbite. This involves a posterior rotational movement of the mandibular incisors as they simultaneously drift superiorly. The movement of the teeth is accompanied by resorption on the outside (labial) surface of the alveolar region just above the chin, and deposition on the lingual side. The alveolar bone

FIGURE 3-19



thus moves backward as the incisors undergo lingual drift. This does not occur to the same extent in individuals having an "end-to-end" incisor relationship or an anterior crossbite.

Bone is progressively added to the external surface of the chin itself, as well as along the underside and other external surfaces of the corpus. This is slow accretion that proceeds gradually throughout childhood. At birth, the mental protuberance is small and inconspicuous. Many anxious parents naturally worry about the chinless appearance of their little child. However, the whole mandible usually tends to lag in differential growth timing and will later catch up to the maxilla in the normal face. This is because of the difference in the timing of growth expansion between the frontal cerebral lobes and anterior cranial fossae versus that of the temporal lobes and middle cranial fossae (Stages 10 and 11). The chin takes on more noticeable form year by year. The combination of new bone growth on the chin itself and the posterior direction of bone remodeling in the alveolar region just above it gradually causes the chin to become more prominent. The whole mandible, meanwhile, is also becoming displaced anteriorly in conjunction with continued remodeling additions at the condyle and ramus producing overall mandibular lengthening.

Regional Change (Stage) 16 –Zygoma

The forward part of the zygoma and the malar region of the maxilla remodel in conjunction with the contiguous maxillary complex, and their respective modes of growth are similar. Just as the maxilla lengthens horizontally by posterior remodeling growth, the malar area also remodels posteriorly by continued deposition of new bone on its posterior side and resorption from its anterior side (Fig. 3-20). The front surface of the whole cheekbone area is thus actually resorptive. This remodeling process keeps this area's position in proper relationship to the lengthening maxillary arch as a whole. They both relocate backward, thereby maintaining the proper anatomic positions between them. The amount of deposition on the posterior side, however, exceeds resorption on the anterior surface, so that the whole malar protuberance becomes larger. Another way of understanding the rationale for the growth of the zygomatic process of the maxilla is to compare it with the coronoid process of the mandible. Just as the coronoid process relocates backward by anterior resorption and posterior deposition to keep pace with the overall posterior elongation of the whole bone, the zygomatic process similarly remodels posteriorly by anterior resorption and posterior deposition. (Refer to page 109.)

Note that the vertical length of the lateral orbital rim increases by sutural deposits at the frontozygomatic suture. The zygomatic arch also enlarges considerably by bone deposition along its inferior edge. The arch remodels laterally (not seen, of course, in lateral headfilms) by bone deposition on the lateral surface, together with resorption from the medial side within the temporal fossa.

Regional Change (Stage) 17

Just as the whole maxillary complex is displaced anteriorly and inferiorly as it simultaneously enlarges in overall size, the malar area is moved anteriorly and inferiorly by primary displacement as it enlarges (Fig. 3-21). The cheekbone thereby proportionately matches the maxilla in (1) the directions and amount of horizontal and vertical remodeling relocation and (2) the directions and amount of primary displacement.

This completes the introductory survey of the regional growth changes taking place in the basicranium and face. The final result is a craniofacial composite that has essentially the same form and pattern present when the first stage was begun. Only the overall size has been altered. All the growth changes among the specific parts and counterparts have been purposefully balanced to give an understanding of the meaning of "balanced growth" and to provide a basis for analyzing imbalanced growth changes in a later chapter.

FIGURE 3-20

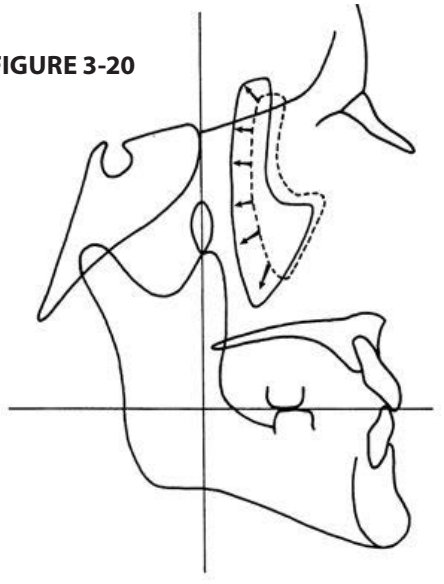
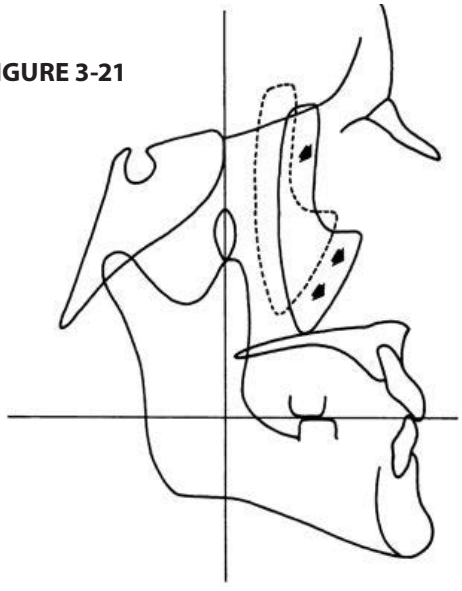


FIGURE 3-21



In Figure 2-24, the first and last stages are superimposed with sella as a registration point. When the sequence of changes described in Stages 1 through 17 are considered, it is apparent that the face does not simply grow directly from one profile to the other. Rather, all the regional changes just outlined are involved, including the complex array of many additional details as explained in following chapters. The overlay seen here is the traditional way of representing the results of the overall process of facial enlargement. This overlay does not, however, represent the actual growth processes themselves—that is, the changes produced (1) by remodeling resorption and deposition; (2) relocation; (3) by primary and (4) secondary displacement. Historically, this basic and important fact was not generally appreciated. The overlay shows the cumulative summation of all four processes and demonstrates the locations of all the regional parts, before and after, when registered on a plane such as sella-nasion.

4

Growth of the Mandible

Because the maxillary complex can respond to treatment procedures in ways that are similar to the mandible, but also in ways that are different, an evaluation of the **differences and the similarities** of the morphology, functions, and development of the mandible **compared** to the nasomaxillary complex is important. A listing is presented below. This is not merely academic, since virtually every factor herein is directly pertinent to basic treatment rationale. (Note, in seminar meetings with postdoctoral residents, a goodly number of significant additions were made to this preliminary list. Try your hand.)

1. The mandible has a ramus jutting from the distal end of its arch (the lingual tuberosity), whereas the maxillary tuberosity has a free posterior surface (in childhood) with the pterygoid plates directly, but separately, behind.

2. The mandible is a separate bone with a movable articulation with the basicranium; the maxilla has fixed sutures with the cranial floor and also among its own multiple, separate bony elements.

3. The temporomandibular joint is lined with cartilage, a pressure-tolerant articular tissue. The maxillary sutures are composed of collagenous connective tissue, which is tension adapted, but pressure sensitive at low force thresholds.

4. The mandible has a condyle capable of forming endochondral bone. The maxilla is made up of entirely intramembranous bone.

5. The mandible has masticatory (and other) muscles attached. The maxilla is not functionally mobile, and the maxilla itself is a paired bone i.e. left and right maxillae, with a midline suture.

6. The mandible is a single bone (in primates). The nasomaxillary complex is an elaborate grouping of many separate bones bounded by the circum maxillary and circumfacial collageneous suture systems. Most areas are without direct muscle attachments.

7. The human mandible has a chin; the maxilla has a nasal spine attached to

a septal cartilage with a septopremaxillary ligament. The mandible lacks a midline, vertical cartilage attached to the basicranium.

8. Both are of "first pharyngeal arch" embryonic origin, and therefore both are innervated by the fifth cranial nerve, but by different divisions.

9. The maxilla incorporates orbital and nasal components not directly represented in the mandible, with diverse functions, structure, and development all involved. This is a major factor relating to clinical considerations. The vertical span of the mandibular ramus, however, is the vertical architectonic "counterpart" of these developing maxillary components.

10. The mandible has a coronoid process; the maxilla has a zygomatic process.

11. The maxilla has a maxillary tuberosity, the mandible has a lingual tuberosity, each a counterpart to the other.

12. The maxillary teeth drift inferiorly; the mandibular dentition drifts superiorly.

13. Both the maxilla and mandible remodel in a predominately posterior manner, and both become similarly displaced in an anteroinferior direction.

14. The maxillary dental apical base is linked directly to the perimeter of the hard palate. The mandible lacks a palate-equivalent altogether.

15. The vertical drifting alveolar processes housing the teeth in both the maxilla and mandible have considerable adjustive capacity and potential for compensations relating to morphogenic variations elsewhere in the craniofacial composite.

16. The positioning of the mandibular corpus and dental arch is a function of remodeling adjustments in alignment, vertical height, and anteroposterior breadth of the ramus. The placement of the maxilla is primarily by the basicranium, but adjustive capacity occurs in sutural growth potential, both intrinsically and clinically.

17. Because the temporomandibular joint is located toward the rear of the middle cranial fossa, endochondral bone formation at the sphenoccipital synchondrosis affects the final position of the mandible in relation to the maxilla. Also, the secondary displacement effect caused by temporal lobe and middle cranial fossa expansion and endochondral bone formation at the sphenoethmoidal synchondrosis has a much lesser effect on the mandible than the maxilla (see page 52).

MANDIBULAR REMODELING

As introduced in Chapter 1, the mandible does **not** simply "grow" as pictured in Figure 2-17*A*. It "remodels" (*B*) and is simultaneously "displaced" as "forward and downward" movement proceeds from the temporomandibular interface (lower right).

The Ramus

In terms of gross anatomy, the significance of the **ramus** of the mandible is mostly that it provides an attachment base for masticatory muscles, which, of course, is a basic function. What usually isn't mentioned, however, is the key role of the ramus in placing the corpus and dental arch into ever-changing fit with the growing maxilla and the face's limitless structural variations. This is provided by critical remodeling and adjustments in ramus alignment, vertical length, and anteroposterior breadth. A best fit with the maxillary arch and middle cranial fossa is thereby provided. Indeed, the **special developmental significance of the ramus is a highlight of craniofacial growth**. Of course, it is not the bony ramus itself that does the job, but rather its osteogenic, chondrogenic, and fibrogenic connective tissues receiving local input control signals that produce the adjustive shape and size of the ramus through time.

Contrary to some older theories of mandibular growth, the mandible is not a product of, or singularly controlled by, a "master center." **Every** area and surface throughout the entire mandible participates directly in its remodeling process. Some parts, of course, represent more active growth sites than others; it would not be possible for a bone having such a complex architectonic configuration to be otherwise. Keep in mind, as emphasized in Chapter 1, that each local area has regionally local conditions, functions, and relationships. The growth signals generated locally are largely responsible for progressive maturation of each local region in concert with corresponding, but different, growth activities in all the other regions.

The mandibular remodeling description begins below with one of the most important structural parts, the ramus. It is important because (1) it **positions** the lower arch in occlusion with the upper, and (2) it is continuously **adaptive** to the multitude of changing craniofacial conditions.

As briefly described in Chapter 3, the principal vectors of mandibular "growth" are posterior and superior. The ramus is thereby **remodeled** in a generally posterosuperior manner while the mandible as a whole becomes **displaced** anteriorly and inferiorly, as schematized in two dimensions in Figures 2-17*B*; 2-17, *right*; 4-1; and 1-5. This allows posterior lengthening of the corpus and dental arch.

The posterior development of the mandibular bony arch simultaneously proceeds into the region that was previously occupied by the ramus. This requires a **remodeling conversion** from what used to be ramus into what then becomes mandibular corpus. That is, the whole ramus becomes relocated posteriorly by resorptive and depository remodeling, and the former anterior part of the ramus is structurally altered into an addition to the corpus, which thereby becomes lengthened by this remodeling process.

The remodeling movement of the ramus in a backward direction has usually been pictured as essentially a two-dimensional process (Fig. 2-3). This is not merely an incomplete explanation; it is inaccurate as well. The problem is that some of the key anatomic parts that participate in the relocation and remodeling process of the ramus and corpus cannot be seen or represented in conventional two-dimensional headfilms and tracings. Among these is the **lingual tuberosity.**

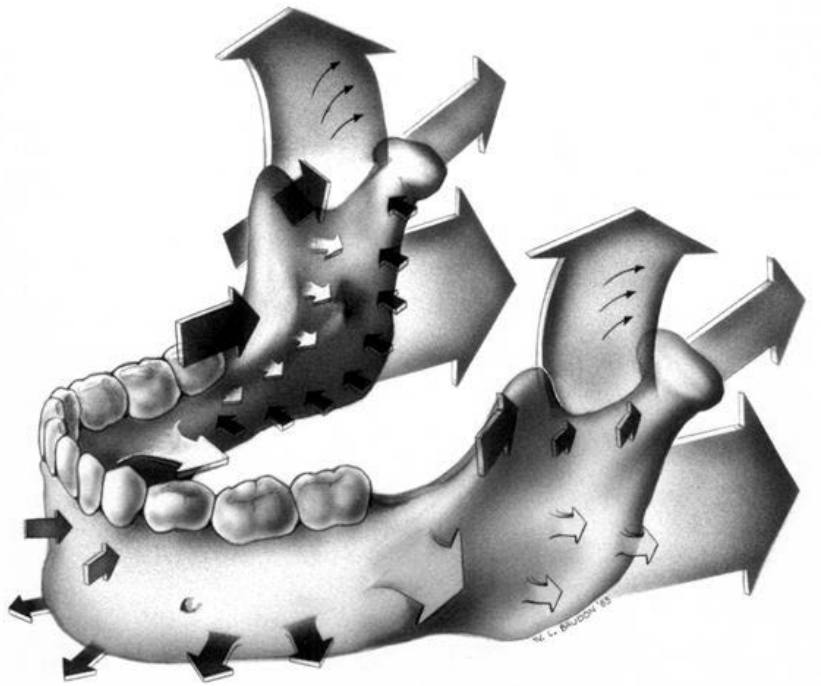


FIGURE 4-1.
Summary diagram of the growth of the mandible. Growth directions involving periosteal resorption are indicated by arrows pointing into the bone surface, and growth directions involving periosteal deposition are represented by arrows pointing out of the bone surface. (From Enlow, D. H. and D. B. Harris: A study of the postnatal growth of the human mandible. Am. J. Orthod., 50:25, 1964, with permission.)

The Lingual Tuberosity

This is an important structure because it is the direct anatomic equivalent of the maxillary tuberosity (Fig. 4-2). Just as the maxillary tuberosity is a major site of growth for the upper bony arch, so is the lingual tuberosity a major site of growth for the mandible. Yet, this structure is not even included in the basic vocabulary of cephalometrics. The reason, simply, is that it is not recognizable in the lateral or frontal headfilm. This is one limitation of cephalometry that was overcome with the introduction of three dimensional cone beam computed tomography in craniofacial diagnosis and treatment planning. The lingual tuberosity is not only a major growth and remodeling site but it also the effective boundary between the

two basic parts of the mandible: the ramus and the corpus. The changes that this important structure undergoes during growth **must** be understood by modern craniofacial practitioners.

The lingual tuberosity grows posteriorly by deposits on its posterior-facing surface, just as the maxillary tuberosity undergoes comparable growth additions. Ideally, the maxillary tuberosity closely overlies the lingual tuberosity (i.e., both are aligned on **PM**, a vertical reference line). Moreover, the lingual and maxillary tuberosities ideally have proportionate rates and amounts of respective remodeling. Variations are explained in Chapter 10.

Note that the lingual tuberosity protrudes noticeably in a lingual (medial) direction, and that it lies well toward the midline from the ramus. The prominence of the tuberosity is augmented by the presence of a large resorptive field just below it. This resorptive field produces a sizable depression called the **lingual fossa**. The combination of periosteal resorption in the fossa and deposition on the medial-facing surface of the tuberosity itself greatly accentuates the contours of both regions (Figs. 4-3 and 4-4*A*).

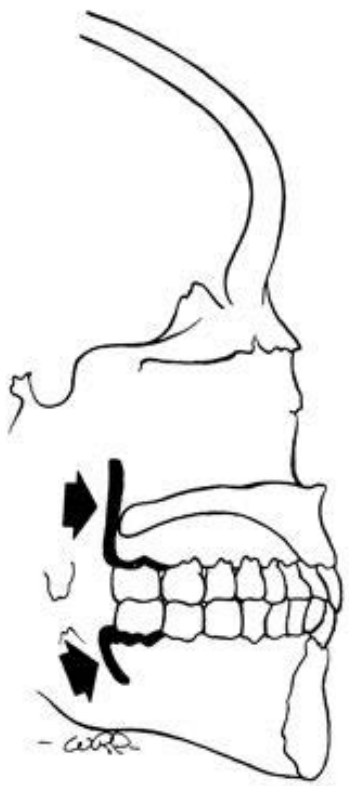


FIGURE 4-2.
The tuberosity remodels (relocates) in an almost directly posterior direction, with only a relatively slight lateral shift. The latter is because bicondylar width does not increase nearly as much as mandibular length beyond the early childhood period, since most of the bilateral growth of the basicranium has occurred by about the second and third years. Even so, the human basicranium is notably wide (and thus also the bicondylar dimension), and this calls for a key remodeling movement to accommodate the more narrow arch (described next).

FIGURE 4-3

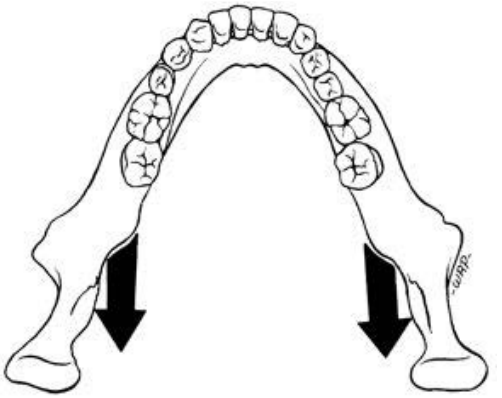
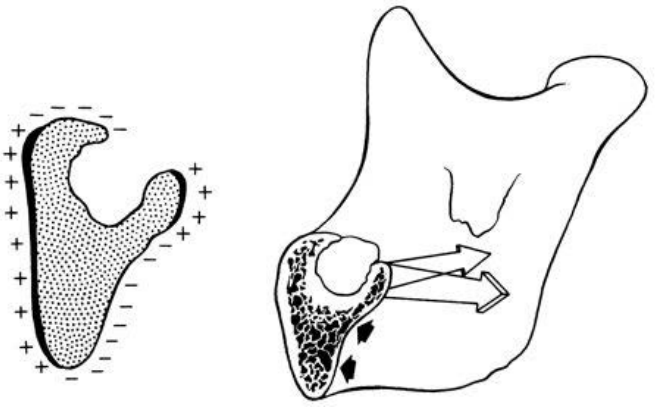


FIGURE 4-4*A*



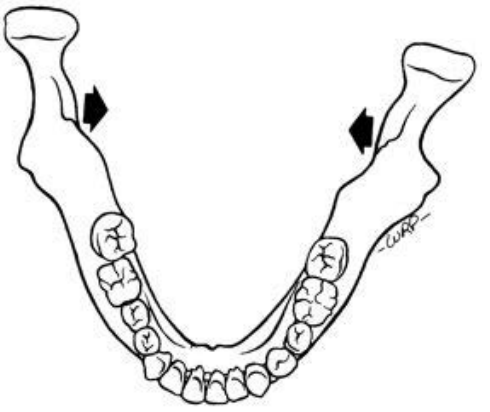


FIGURE 4-4*B*.
The posterior growth of the tuberosity is accomplished by continued new deposits of bone on its posterior-facing exposure. As this takes place, that part of the ramus just behind the tuberosity remodels medially (Fig. 4-4B). This area of the ramus is coming into line with the axis of the arch in order to join it and thus become a part of the corpus, thereby lengthening it. As pointed out above, the whole ramus lies well lateral to the dental arch.

The Ramus-to-Corpus Remodeling Conversion

Keep in mind that the whole ramus is also becoming relocated in a posterior direction at the same time. What has happened, in summary, is that bony **arch length** has been increased and the corpus has been lengthened by (1) deposits on the posterior surface of the lingual tuberosity and the contiguous lingual side of the ramus and (2) a resultant lingual shift of the anterior part of the ramus to become added to the corpus.

The presence of resorption on the anterior border of the ramus is often described as "making room for the last molar." It is doing much more than this! The resorptive nature of this region is directly involved in the whole process of progressive relocation of the entire ramus in a posterior direction; this movement continues from the tiny mandible of the fetus to the attainment of full adult mandibular size. The overall extent of ramus movement amounts to several centimeters, not merely the width of the molar.

Another key point is that the traditional description of posterior ramus movement implies a **straight line** backward growth process in a two-dimensional plane, as represented by *a* and *b* in Figure 4-5. This is not the case at all. Such a picture of ramus growth shows, simply, resorption on the anterior edge and deposition on the posterior edge. Development actually takes place as indicated by *c*. (Refer to the "V" principle.) In *d*, the growth direction thus follows the *x* arrows, rather than the straightline axis shown by the *y* arrows. As pointed out above, because the bicondylar dimension is established much earlier in childhood, bilateral growth separation between the right and left condyles is minimal beyond the early childhood years.

Remodeling activity does not occur **only** on the anterior and posterior margins of the ramus. The various parts of the ramus are oriented so that the span **between** also necessarily comes into play. The **coronoid process** has a propeller-like twist, so that its lingual side faces three general directions all at once: posteriorly, superiorly, and medially. When bone is added onto the lingual side of the coronoid process, its growth thereby proceeds **superiorly**, and this part of the ramus thereby becomes increased in vertical dimension (Fig. 4-6). Notice that each coronoid process lengthens vertically, even though additions are made on the medial (lingual) surfaces of the right and left coronoid processes. This is an example of the enlarging V principle, with the V oriented vertically.

These **same** deposits of bone on the lingual side also bring about a **posterior** direction of growth movement, because this surface also faces posteriorly. A backward movement of the two coronoid processes is the result, even though deposits are added on the inside (lingual) surface. This is also an example of the expanding V principle, with the V oriented horizontally. Notice further that this enables the whole posterior part of the mandible to **widen** (although not very much except during the period of fetal and early childhood basicranial growth in width), even though deposition occurs on the inside of the V.

In all the above relationships, the **buccal** side of the coronoid process has a **resorptive** type of periosteal surface. This surface faces away from the combined superior, posterior, and medial directions of growth. The remainder of most of the

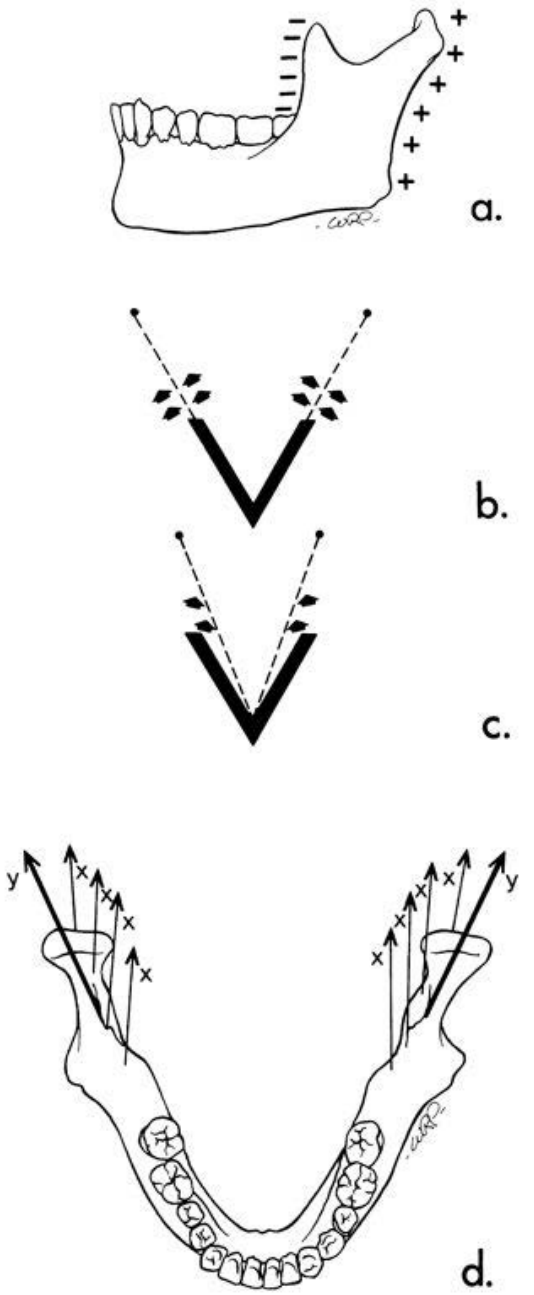


FIGURE 4-5.
These same deposits of bone on the lingual side also function to carry the base of the coronoid process and the anterior part of the ramus in a medial direction in order to add this part to the lengthening corpus, which lies well medial to the coronoid process. This was underscored above, and, again, is an example of the V principle because a wider part undergoes relocation into a more narrow part as the whole V moves toward its wide end. Thus, the area occupied by the anterior part of the early childhood ramus (Fig. 4-7) in 1 is relocated and its former location becomes remodeled into the posterior part of the corpus in 2.

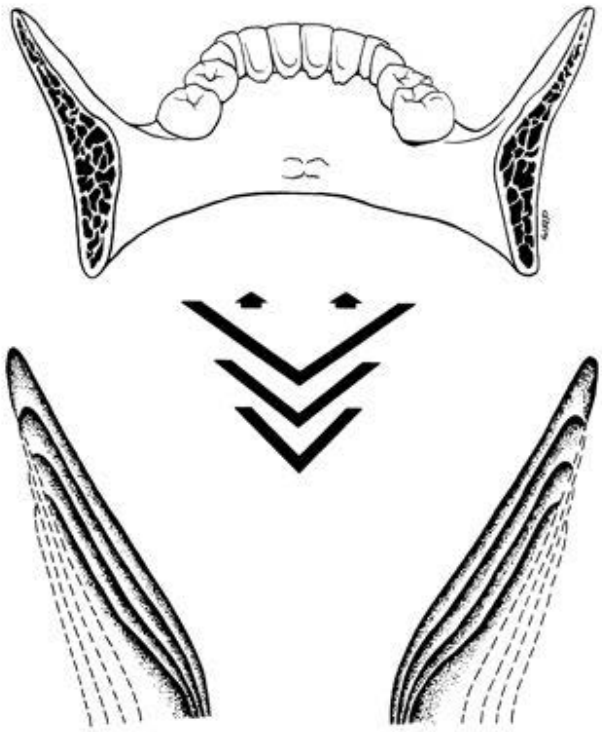


FIGURE 4-6

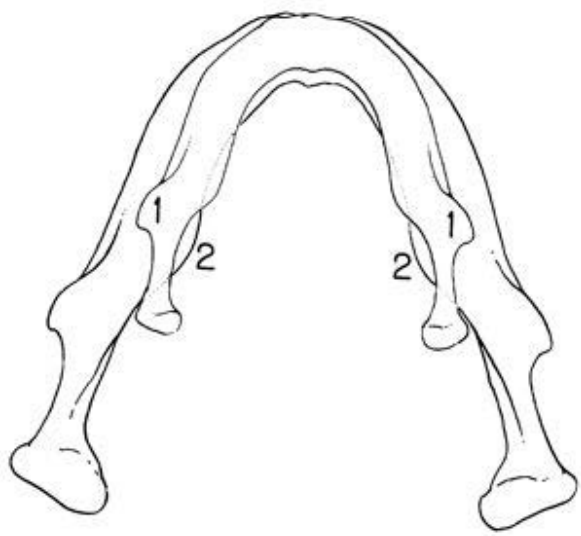
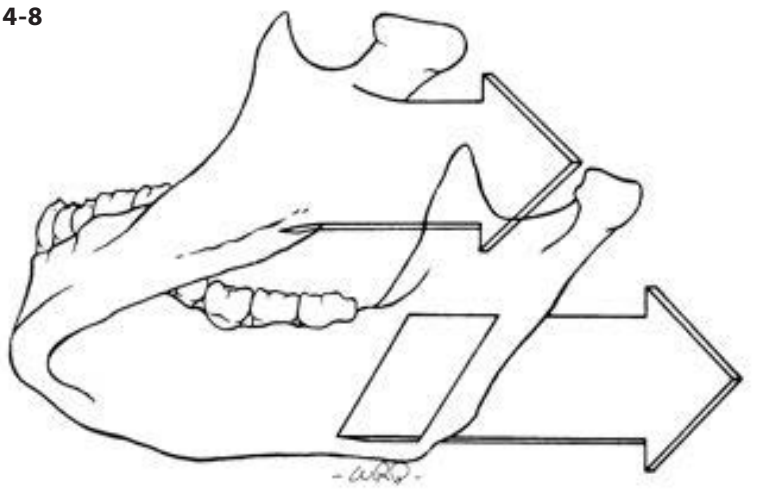


FIGURE 4-7.
(Adapted from Enlow. D. H., and D. B. Harris: A study of the postnatal growth of the human mandible. Am. J. Orthod., 50:25, 1964, with permission.)

superior part of the ramus, including the whole area just below the mandibular (sigmoid) notch and the **superior** (not lateral or medial) portion of the condylar neck, grows superiorly by deposition on the lingual side and resorption from the buccal side. The lower part of the ramus below the coronoid process also has a twisted contour. Its buccal side faces posteriorly toward the direction of backward growth and thus, has a depository type of surface (Fig. 4-8). The opposite lingual side, facing away from the direction of growth, is resorptive.

Keep in mind, in all such growth activities, that it is the enclosing distribution of osteogenic, fibrogenic, and chondrogenic connective tissues in respective local growth fields that actually conduct these remodeling activities. And, it is the signals from the composite of all related soft tissue parts and their growth and functioning that orchestrate the local remodeling patterns. (See Fig. 1-7.) The result is the complex configuration of the mandible that then carries out *its* diverse, regional functions, and grows and develops as it does so.

FIGURE 4-8



Only one field of surface resorption is present on the inferior edge of the mandible. It is located at the ramus-corpus junction and forms the **antegonial notch. The antegonial notch is the result of** remodeling of the ramus just behind it as the ramus relocates posteriorly (Fig. 4-9). Clinically important mandibular growth rotations also involve a sizable resorptive field on the ventral edge of the ramus, as illustrated by Figure 4-15.

The posterior margin of the ramus is a major remodeling site. The condyle generally has an obliquely upward and backward growth direction; the trajectory of growth involved (i.e., how much upward and how much backward) is variable. Such variations have led to the common terms "horizontal or vertical grower" with respect to the mandible. One problem with this concept is that it implies that the condyle is leading such rotational changes when in fact it is more likely that the condylar head is responding to rotational force vectors acting on the ramus. **To maintain an effective articulation the condyle responds with selective cell divisions** in those parts around the periphery of the condyle pointing toward the

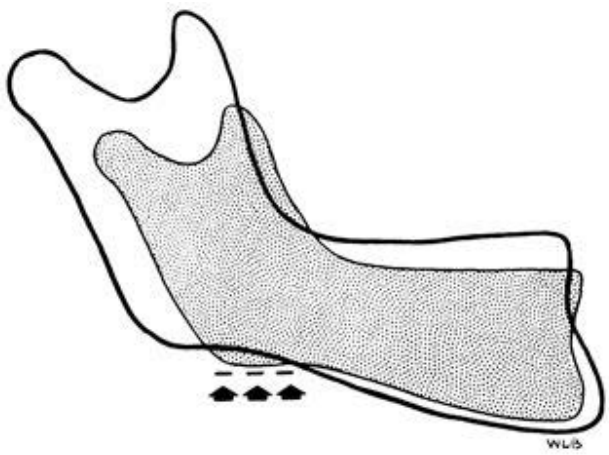


FIGURE 4-9.
(Modified from Enlow, D. H.: *The Human Face*. New York, Harper & Row, 1968, p. 232, with permission.)

growth direction, with retardation in other parts of the condyle. However, the growth of the rest of the ramus necessarily keeps pace (or actually determines) the amount of condylar proliferation (see Fig. 4-19). Although correlated, these two regional growth sites (posterosuperior part of ramus and condyle) are essentially separate and develop under different regional conditions, but interrelate in response to the common activating signals that both share. Together they represent the most active areas during mandibular growth in distance moved and amount of histogenic activity. Because of the relatively rapid rate of ramus growth, the bone tissue in the posterior part of the ramus is characteristically one of the "fast-growing" types (see Chapter 14). Ramus development often involves a remodeling rotation of the whole ramus, and a resorptive field then occurs on the posterior margin below the condyle, as illustrated by Figures 4-13 and 4-15.

The gonial region is anatomically variable and, therefore, much variation is involved in its pattern of growth. Depending on the presence of inwardly or outwardly directed gonial flares, the buccal side can be either depository or resorptive, with the lingual side having the converse type of growth. However, many different histogenetic combinations can be encountered because of the variability of this region.

While the whole ramus grows posteriorly and superiorly, the mandibular foramen likewise relocates backward and upward by deposition on the anterior and resorption from the posterior part of its rim. The foramen, from childhood through old age, maintains a constant position about midway between the anterior and posterior borders of the ramus. Even when the ramus undergoes marked alterations associated with edentulism (during which it may become quite narrow), this foramen usually sustains a midway location. This is anatomically important because the mandibular branch of the trigeminal nerve exits the foremen ovale of the skull and enters the corpus of the mandible at this point. To minimize

movement of the nerve during mandibular function the foramen is located at the center of rotation of the mandible during masticatory movement. This makes the foramen a stable point for superimposition of serial images of the mandible to determine both normal growth changes and treatment effects.

The Mandibular Condyle

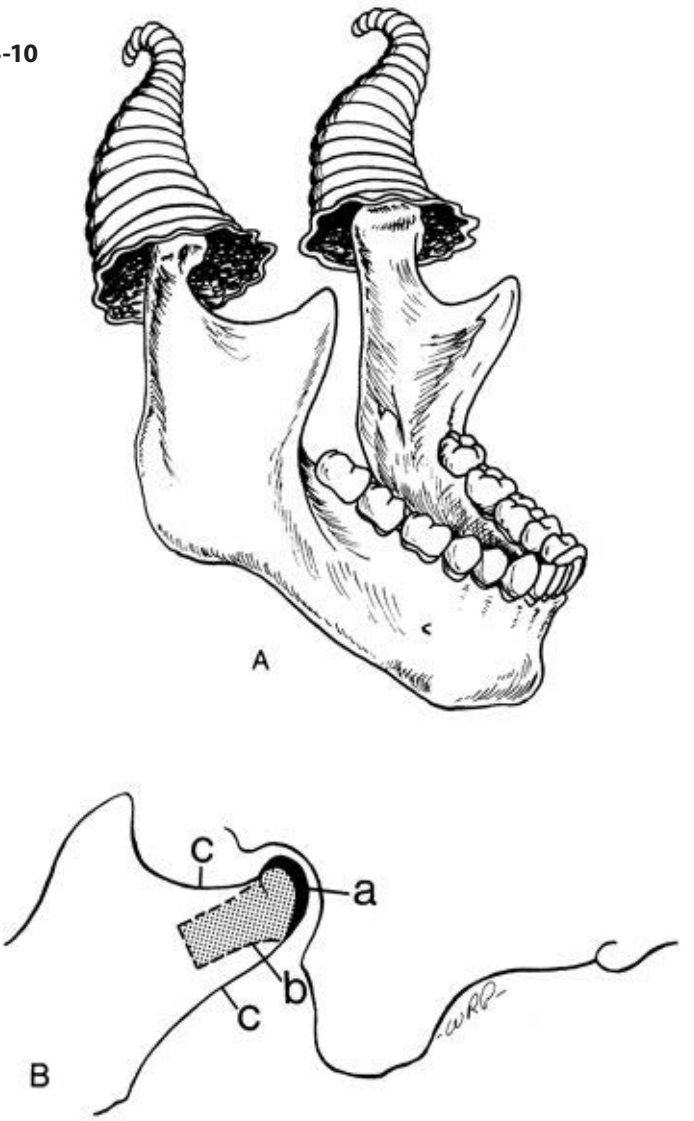
This is an anatomic part of special interest because it forms the articulation for the mandible and as such determines at least in part the relationship of the upper and lower teeth. In addition, it is a major site of growth, having considerable clinical significance. Historically, the condyle was incorrectly regarded as a kind of cornucopia from which the whole mandible itself pours forth (Fig. 4-10*A*). The condyle was believed (and unfortunately is still incorrectly regarded by some) to be the ultimate determinant of the mandibular rate of growth, the amount and direction of growth and thus the determining factor in overall mandibular size, and shape. Present-day biologic theoreticians regard the condyle as a unique structure but it is no longer believed to represent a pacesetting "master center" with all other regional growth fields subordinate to and dependent on it for direct control. Yet, the condyle is a major field of growth, nonetheless, and it is an important one. What, then, it must be asked, could be even more important than serving as a master center? That question is answered in the pages that follow. Indeed, there is a key function that **does**, in fact, transcend a mythical "master center" notion.

During mandibular development, the condyle functions as a **regional** field of growth that provides an adaptation for its own localized growth circumstances, just as all the other regional fields accommodate their own particular (but different) localized growth conditions. The growth of the mandible as a whole is the product of all the different **regional** forces and regional functional agents of growth control acting on it to produce the topographically complex shape of the mandible as a whole. Growth is the aggregate expression of the composite of all these localized factors. Every local growth site is independently self-contained, although all are bound as an interrelated mosaic proceeding as a "symphony of developmental movements."

The condylar growth mechanism itself is a clear-cut process. **Cartilage** is a special **non-vascular** tissue and is involved because variable levels of **compression** occur at its articular contact with the temporal bone of the basicranium. There are no capillaries in cartilage that can be collapsed by a compressive surface force. In addition, importantly, the intercellular matrix of cartilage is markedly **hydrophilic** and, therefore, is turgid and unyielding to surface pressure. An **endochondral** growth mechanism is required for this part of the mandible because the condyle grows upward and backward towards its articulation. Although there is some debate as to the degree of compressive loading of the condyle there is no doubt that condylar growth takes place in an area where direct pressure is at least intermitantly applied. An intramembranous type of growth could not operate in this location because the periosteal mode of osteogenesis is not pressure adapted and has a low threshold for compressive forces. Endochondral growth occurs only at the articular contact part of the condyle, because this is where pressure

exists at levels that would be beyond the tolerance of the bone's vascular soft tissue membrane (ie the periosteum). As seen in Figure 4-10*B*, the endochondral bone tissue (*b*) formed in association with the condylar cartilage (*a*) is laid down only in the medullary portion of the condyle. The enclosing bony cortices (*c*) are produced by periosteal-endosteal osteogenic activity; these vascular membranes are not subject to the compressive forces of articulation, but, rather, are essentially tension related because of muscle and connective tissue attachments. The real functional significance of the condylar cartilage thus involves an avascular and matrix-firm adaptation for regional pressure and movable articulation. This regional, **endochondral** bone-forming mechanism develops as a specific response to this particular **local** circumstance. The cartilage itself is **not** genetically programmed

FIGURE 4-10



to grow and certainly does not govern the course of growth in other areas of the mandible. The pressure-tolerant condylar cartilage, however, provides another basic and significant growth function that will be described later.

The condylar cartilage is a **secondary** type of cartilage, which means that it does not develop by differentiation from the established **primary** cartilages of the fetal skull (i.e., the cartilages of the pharyngeal arches, such as Meckel's cartilage, and the definitive cartilages of the basicranium). Phylogenetically, the original ancestral reptilian endochondral bone (the *anticular*) that provided for mandibular articulation became converted to an ear ossicle (the *malleus*) in mammals. Then, in mammalian evolution, a "secondary" cartilage was subsequently developed on the ancestral mandible (the intramembranous *dentary bone*) to provide for lower jaw articulation with the basicranium. It is believed that the unique connective tissue covering (capsule) of the "new mammalian" condylar cartilage was actually an original periosteum. Its undifferentiated connective tissue stem cells, however, metaplastically developed into chondroblasts, rather than osteoblasts, because of the compressive forces acting on this membrane. An adventitious type of "secondary" cartilage thereby developed, rather than bone, because of the changed functional and developmental conditions imposed upon this part of the mandible. It is thus not an "endochondral" bone in the sense that phylogenetically, the bones of the basicranium are endochondral in type. The mammalian mandible is essentially a membrane bone in which one part (i.e., what has become the new condyle) has developed in response to a phylogenetically altered developmental situation. This involved the ectopic presence of pressure that, in turn, caused localized ischemia and anoxia, factors known to induce chondrogenesis from the pool of undifferentiated connective tissue cells, rather than osteogenesis.

The condylar cartilage is thus phylogenetically and ontogenetically unique and differs in histologic organization from most other growth cartilages involved in endochondral bone formation. It is NOT structurally comparable to a long bone's cartilaginous epiphyseal plate. Further, it is now generally recognized that the secondary cartilage of the condyle is NOT the genetic pacemaker for the growth of the mandible. Its real contribution is to provide **regional adaptive growth** (i.e., growth that is secondarily responsive to a variety of circumstantial conditions) thus giving yet another meaning for the term "secondary." This maintains the condylar region in proper anatomic relationship with the temporal bone as the whole mandible is simultaneously being carried downward and forward. Thus, the condyle is not a "primary" center of growth, but rather (1) secondary in evolution; (2) secondary in embryonic origin; and (3) secondary in adaptive responses to changing developmental conditions.* The condyle has a special multidirectional capacity for growth and remodeling in selective response to varied mandibular displacement movements and **rotations** (described below). The special structure of the condyle provides for this, unlike the committed unidirectional linear growth

* Another common definition is that secondary growth cartilages are those, simply, that have a type of structure that puts them in a separate category from the typical epiphyseal growth plates of long bones. Articular cartilages are another example of the secondary type. As shown by Moss, the condylar cartilage is comparable, both in structure and growth behavior, to an articular, rather than an epiphyseal plate, cartilage. It is not directly "articular," however, because of its special fibrous covering.

of long bone epiphyseal plates produced by the characteristic linearly oriented direction of chondroblast proliferation.

In summary, the primary role of the condyle is to maintain the articulation of the mandible with the neurocranium through the glenoid fossa. The condyle is also capable of adaptive and responsive growth and under normal circumstances can withstand the compressive forces of mandibular function. Thus the condyle (1) provides a pressure-tolerant articular contact, and (2) makes possible a multidimensional growth capacity in response to ever-changing, developmental conditions.

A unique **capsular layer** of poorly vascularized connective tissue covers the articular surface of the condyle (*a* in Fig. 4-11). This membrane is highly cellular early in development, but becomes densely fibrous with age and function. Just deep to it is a special layer of prechondroblast cells (*b*). This is the predominant site for cellular proliferation, and it is responsible for the tissue-feeding process providing an ongoing flow of new developing cartilage (layer *c*) for endochondral replacement by bone as the deeper layers advance.

The proliferative process produces the "upward and backward" growth movement of the condyle (Fig. 4-11). The condylar cartilage **moves** by prechondroblast cell divisions with an equal amount of cartilage removal at the cartilage-bone interface. The removal phase involves replacement with endochondral bone. A trail of continually forming endochondral bone thus follows the moving cartilage, as schematized by layer *d*.

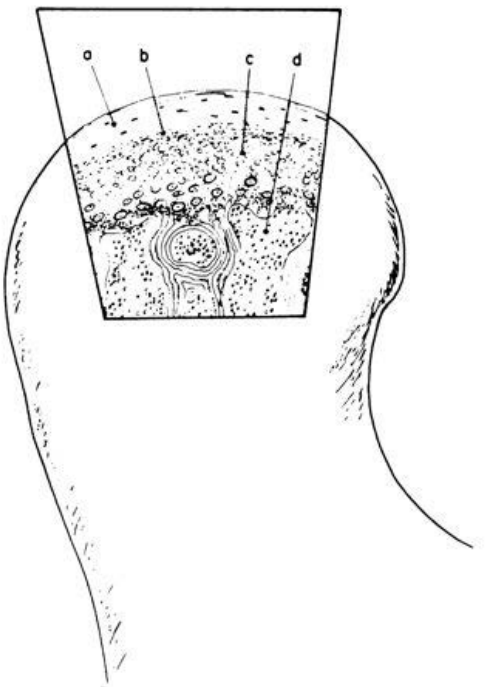
The prechondroblast cells are closely packed, and very little intercellular matrix is present. This is due to their rapid division. A relatively thin transitional zone or immature zone then occurs deep to the proliferative layer as new cells feed into it, with a somewhat increased amount of matrix. This layer does not appear to contribute materially to the cell division process. The deeper cells then become transformed into the next layer as it "moves up" behind the moving layers ahead of it and is composed of densely packed chondroblasts that are undergoing hypertrophy (*c*). The matrix is also noticeably scant.

The small amount of matrix in the deepest part of the hypertrophied zone becomes calcified, and a zone of resorption and bone deposition follows (*d*). Unlike the arrangement in typical *primary* growth cartilage (i.e., epiphyseal plates of long bones and synchondroses), these various zones **do not have linear columns of daughter cells. This is a notable histologic difference between primary and secondary types of growth cartilage**. The nonlinear arrangement of the daughter cells in the condylar cartilage is consistent with the condylar cartilage's **multidirectional proliferative capacity**. This is one of the most significant developmental features of the condyle. Depending on *where* in the condylar cartilage that mitotic divisions occur, **that** part of the condyle (and ramus) thereby proliferates more vertically or more posteriorly, or virtually any point between, as determined by input signals. These input signals are related to both the demands of dynamic and static articulation of the teeth as well as the architectonic pattern of "fitting" among the multitude of craniofacial parts.

While all this is going on, the periosteum and endosteum are active in producing the **cortical** bone that encloses the **medullary core** of endochondral

bone tissue. This arrangement is like a cork in a bottle with the cork representing the condylar cartilage with its associated endochondral bone and the glass surrounding the cork representing the cortical ring of intramembranous bone. The overlying cap of pressure-tolerant cartilage has taken the brunt of the compressive forces acting on the condyle. The cortical ring of intramembranous bone continues down onto the condylar neck.

FIGURE 4-11



The lingual and buccal sides of the neck characteristically have **resorptive** surfaces (Fig. 2-2). This is because the condyle is quite broad and the neck is narrow. The neck is progressively relocated into areas previously held by the much wider condyle, and it is sequentially derived from the condyle as the condyle **moves** in a superoposterior course. What used to be condyle in turn becomes the neck as one is remodeled from the other (Fig. 4-12). This is done by periosteal resorption combined with endosteal deposition. Explained another way, the **endosteal** surface of the neck actually faces the growth direction; the periosteal side points away from the course of growth. This is another example of the V principle, with the V-shaped cone of the condylar neck growing toward its wide end. (See Figs. 2-2 and 2-9.)

All the while, as condylar and ramus development proceeds, the mandible, as a whole, is becoming displaced anteroinferiorly (Fig. 2-17). What is the physical force that produces the forward and downward primary **displacement** of the mandible? For many years it was presumed that growth of the **condylar cartilage**, because it is known that cartilage is a special pressure-adapted type of tissue, creates a "thrust" of the mandible against its articular-bearing surface in the glenoid fossa. The proliferation of the cartilage **toward** its contact thereby was presumed to **push** the whole mandible away from it.

Some students of facial biology still accept this explanation. However, most contemporary investigators and biologic researchers have concluded that this is either an incomplete or an off-target answer. The biologic reasons follow.

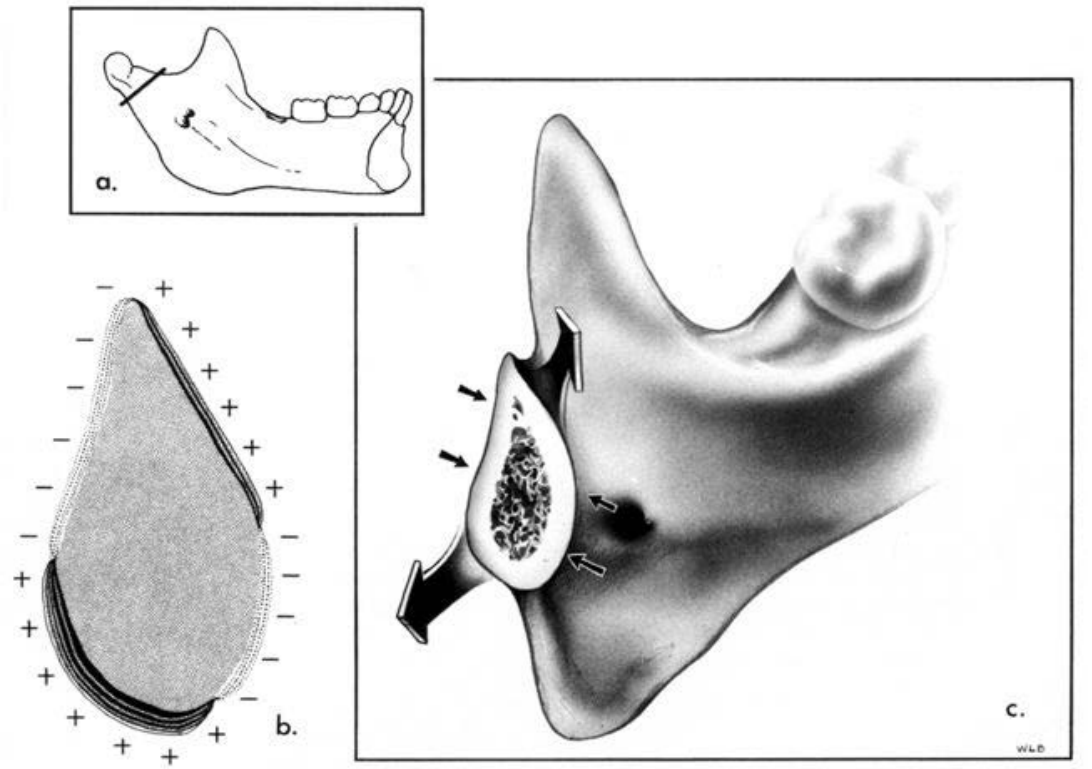


FIGURE 4-12.
(Adapted from Enlow, D. H., and D. B. Harris: A study of the postnatal growth of the human mandible. Am. J. Orthod., 50:25, 1974, with permission.)

The Condylar Question

A great puzzle was created when it was pointed out that functional mandibles totally lacking condyles exist in nature. And surprisingly, the morphology of these mandibles was more or less normal in all other respects; only the condyle and part of the condylar neck were congenitally missing. Moreover, these **bilaterally** condyle-lacking mandibles occupied an essentially normal anatomic **position**; the bony arch was properly placed for occlusion, and the mandible functioned (albeit with distress) in masticatory movements even though it lacked an articulation. These revealing observations suggested two conclusions. First, the condyles may not play the kingpin role of a "master center" pace-setting the growth processes in the other parts of the mandible. Second, the whole mandible can become **displaced** anteriorly and inferiorly into its functional position without a "push" against the basicranium. Many experimental studies have subsequently been carried out with

similar results, although investigators are still arguing about the proper way to interpret their meaning.

These observations led to a consideration of the fabled "functional matrix" by students of facial biology. The idea is essentially that the mandible is **carried** forward and downward, just as the maxilla is presumably carried anteroinferiorly in conjunction with the growth expansion of the soft tissue matrix associated with it. It is a passive type of carrying in which condylar remodeling acts with displacement as co-participants but not as the driving force. They proceed together in mutual response to common activating signals. Thus, as the mandible is displaced away from its basicranial articular contact, the condyle and whole ramus **secondarily** (but virtually simultaneously) remodels toward it (see Fig. 1-5), thereby closing the potential space without an actual gap being created (unless the condyle does not develop at all, as mentioned above). There are still, however, actual but variable levels of pressure being exerted on the articular surface of the condyle because it is a movable joint; it is presumably a **relief** of the **amount** of pressure that relates to condylar growth. The enlarging soft tissue mass draws the mandible protrusively to cause this.

The clinical implications are apparent. Just how involved is the condyle as an underlying and causative factor in facial abnormalities? What happens to the mandible if the condyle is injured during the childhood period? To the orthodontist, a key question is whether the condyle itself is the **direct** and **primary** target of any given clinical procedure, or whether it **follows** in response to clinical signals acting on soft tissues (e.g., the masticatory musculature), which in turn activate the composite of osteogenic and chondrogenic connective tissues of the ramus and its condyle as a **whole**. How can overall mandibular length be clinically increased or decreased for Class II and III individuals by physiologic or mechanical intervention in this composite growth mechanism?

The current thinking is that the condylar cartilage **does** have some limited measure of intrinsic, genetic programming. Of major importance is the condyle's capacity for continued cellular proliferation. That is, the cartilage cells are coded and geared to divide and continue to divide, but extracondylar factors are needed to sustain this activity. The **rate** and **directions** of condylar growth are presumably subject to the influence of extracondylar agents, including intrinsic and extrinsic biomechanical forces and physiologic inductors. Perhaps the single most important factor that stimulates growth of the condylar cartilage is functional movement of the mandible. (See Moussa et al 1992, Duterloo et al 1971, Tsolakis et al 1997.) One hypothesis is that increased amounts of pressure on the cartilage serve to inhibit the rate of cell division and proliferation and decreased amounts of pressure appear to stimulate and accelerate growth. Presumably, forces applied to the mandible in such a way that they increase the level of pressure on the condyle would result in a shorter mandible **if** this were done during the period of active condylar growth. This is the biologic basis for the use of chin-cup therapy to reduce mandibular growth. Similarly, a **release** of some of the compressive force, using a bionator or similar device to protrude the mandible, would produce a larger mandible if done during the active growth period. Animal experiments have yielded inconsistent results with regard to experimental increases in mandibular length.

And in humans, three randomized clinical trials showed that mandibular growth could be stimulated to occur earlier but the total amount of growth was similar between treatment and controls. Current thinking is that mandibular growth can be restricted if compressive force is applied to the condyle and the mandible for the entire growth period (until 18 in women and 25 in men!). However, this approach has not been universally adapted because of the high degree of patient compliance needed to achieve meaningful long term results (see Sugawara J et. al 1990). Moreover, recent research studies show that the nature of the condylar stimulus is more complex than simple forces acting directly on the condyle; rather, nerve-muscle-connective tissue pathways are involved, and changes utilize a **composite** of such tissue responses and chain feedbacks with the condyle as well as the other parts of the mandible that also participate. Sensory nerve input from the periodontal membranes and from the soft tissue matrix throughout the face pick up stimuli that are passed on via motor nerves to muscles that, in turn, alter the displacement and the positioning of the mandible, which then affects the course of growth and remodeling by the condyle and all other areas of the growing mandible. These signals must ultimately affect the **amount** of ramus growth in different **directions**, thereby continually adjusting both the alignment and the shape of the ramus to accommodate its multiple and changing anatomic and functional relationships. Clinically, the impact of most "functional appliances" designed to increase mandibular growth cause changes in the remodeling process of the alveolar bone and the relative position of the alveolus with respect to the underlying corpus. These effects on the dentoalveolus combined with alterations in ramus growth likely result in the observed clinical effects.

This whole discussion of the condylar role, however, bypasses a most fundamental consideration. Is it the condyle itself or, more basically, the **whole ramus** with its condyle that is the real issue. This is evaluated later.

The Adaptive Role of the Condyle

The random arrangement of the condylar prechondroblasts, described earlier, is in contrast to the linear columns of daughter cells associated with the essentially unidirectional growth of long bones. This is a histogenetic adaptation of the condylar cartilage that provides opportunity for selected, multidirectional growth potential. Consider the virtually limitless range of anatomic variations that occur in the structural patterns of the nasomaxillary complex and basicranium. There are dolichocephalic and brachycephalic types of headforms, vertically long and short nasomaxillary regions, wide and narrow palates and upper arches, widely separated versus closely placed glenoid fossae, steep versus shallow cranial floor flexures, broad versus narrow pharyngeal regions, large versus small tooth sizes, male versus female patterns, and so on. If the growth, shape, and dimensions of the mandible were actually "preprogrammed" within the genes of condylar chondroblasts (according to old theory), and if the condyle were indeed to function as an ultimate "growth control center," without taking into account structural and developmental vagaries in the rest of the craniofacial complex, there is **no way** that a fitting of the mandible to the basicranium on one end and to the maxilla on the

other could be achieved. If the condyle functioned as a self-contained, independent structure with its growth coded in an isolated cartilage unresponsive to variations and continual changes in the growth and morphology of contiguous regions, these many developmental and functional relationships simply could not be workable. However, it is the **adaptive responsive** nature of the condylar growth process that allows for a latitude of morphologic and morphogenic adjustments and a working (if not perfect) functional relationship among all of them. What could be more exalted in status than serving as a "master center of growth"? **The answer is the condyle's adaptive capacity.** A remarkable histogenic function.

The New Image of the Condyle and "Condylar Growth"

One specific point, however, needs to be clearly understood. Historically, it has been the **condyle** that has been given all the glory, whether as the primary **determinant** of mandibular growth or, as we now see it, as the **respondent** structure that makes possible adaptive, truly interrelated growth. The problem, however, is that we still use and endure the anachronistic, held-over term "condylar growth." This old term, unfortunately, implies an incomplete and inaccurate understanding of the whole picture. The condyle, of course, plays a role. It is directly involved as a unique, regional growth site; it provides an indispensable latitude for adaptive growth; it provides movable articulation; it is pressure tolerant and provides a means for bone growth (endochondral) in a situation in which ordinary periosteal (intramembranous) growth would not be possible; and it can also, all too frequently, become involved in TMJ (temporomandibular joint) pathology and distress. With regard to the growth and adaptive requirements for the mandible, **it is not just the condyle**, however, that participates as the key component. The **whole ramus** is directly involved. This is an all important point. The ramus bridges the pharyngeal compartment and places the mandibular arch in occlusal position with the maxillary arch. The horizontal breadth of the ramus determines the anteroposterior position of the lower arch, and the height of the ramus accommodates the vertical dimension and growth of both the nasal and masticatory components of the midface. The dimensions and morphology of the ramus are directly involved in the attachments of the masticatory muscles, and the ramus must accommodate their growth and size. It is the growth and development of the **whole** ramus, not merely the condyle, that accomplishes these multiple and basic ends. As already seen, the growth and remodeling of the ramus are complex maneuvers and involve many regional growth sites, only one of which is the condyle. The term "condylar growth" is misleading and conveys a biologic misconception. More properly, the terms needs to be "**ramus** and condylar growth." In a real sense, the condyle **follows** the growth of the whole ramus and does not lead it. This is important, because studies have shown, and continue to show, that the entire ramus and the muscles attached to it, not just the condyle, are a principal clinical target for many orthodontic procedures. Compare Figure 2-6 (an incomplete picture conveyed by the old term "condylar growth") with Figure 4-1 (whole ramus growth). Significantly, the "adaptive capacity" of the condyle discussed in

the preceding paragraph **also** involves the entire ramus. The ramus, importantly, is also an important anatomic part directly involved in growth **compensations**, as described later.

Clinicians have traditionally regarded the mandible as less predictable in its response to orthopedic forces than the maxilla. One possible biologic explanation of this clinical finding is that the whole ramus is directly involved and not just the condyle. Clinical forces must overwhelm not "the condyle" but the massive masticatory musculature, a significant restraining factor. Furthermore, clinically induced changes in condylar cartilage growth may be negated by equal and opposite growth changes in the ramus. It follows then that mandibular orthopedics must modify growth signals targeted at both the ramus, the condyle and the dentoalveolus to be maximally effective.

The Ramus and Middle Cranial Fossa Relationship

This is a kinship that, at first thought, could seem unlikely considering their distant functions. However, the relationship becomes real during development and is indeed a significant consideration.

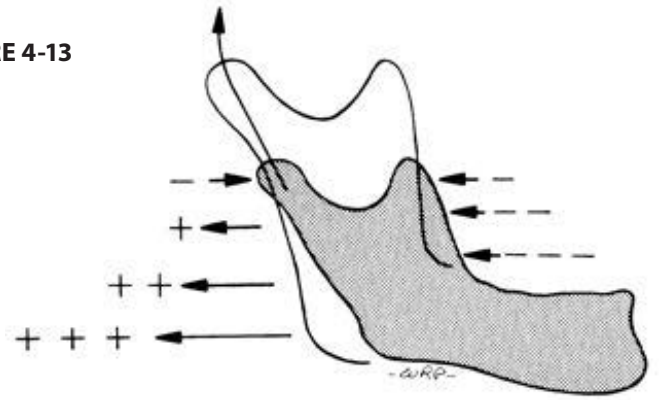
As the horizontal enlargement of the middle cranial fossa and brain growth advance the nasomaxillary complex by forward displacement, the horizontal span of the pharynx correspondingly increases (Fig. 3-9). The skeletal dimension of the pharynx is established by the size of the middle cranial fossa because the floor of this basicranial fossa is the roof of the pharyngeal compartment. The ramus must necessarily increase to an equivalent extent. The effective anteroposteral dimensions of the ramus and middle cranial fossa (not their respective oblique dimensions) are direct counterparts to each other. One structural function of the ramus, in spanning the pharynx, is to provide developmental potential for adaptations required to place the corpus in a continuously functional position because of variations elsewhere in the face and neurocranium. If such adjustments are fully or even only partially successful, a better occlusal fit is achieved. This is done by the same remodeling process that simultaneously relocates the ramus posteriorly as it becomes displaced anteroinferiorly.

Ramus Uprighting

The ramus normally becomes more vertically aligned during its development. As long as the ramus is actively growing in a posterior direction, this is accomplished by greater amounts of bone additions on the inferior part of the posterior border than on the superior part (Fig. 4-13). A correspondingly greater amount of matching resorption on the anterior border takes place inferiorly than superiorly. A "remodeling" rotation of ramus alignment thus occurs. Condylar growth becomes directed in a more vertical course along with the rest of the ramus. See page 34.

The reason the ramus becomes progressively more upright as childhood development proceeds is that it must lengthen vertically to a much greater extent than it broadens horizontally, and this creates a developmental problem for the "genic" tissues involved (Fig. 4-14). In this schematic diagram, the pharynx (and

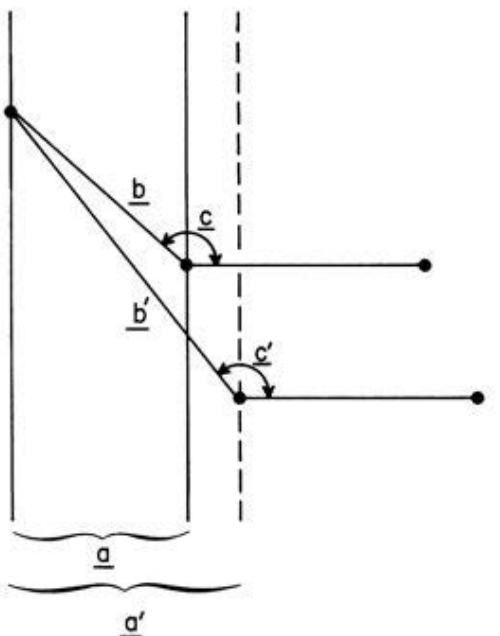
FIGURE 4-13



middle cranial fossa) enlarges horizontally from *a* to *a′*. The ramus enlarges, correspondingly, from *b* to *b′* to match it. It also **lengthens vertically,** however. Angle *c* is thereby reduced to *c′* in order to accommodate the vertical **nasomaxillary growth** also taking place at the same time. The "gonial angle" thus must undergo change (close) in order to **prevent** change in the occlusal relationship between the maxillary and mandibular arches.

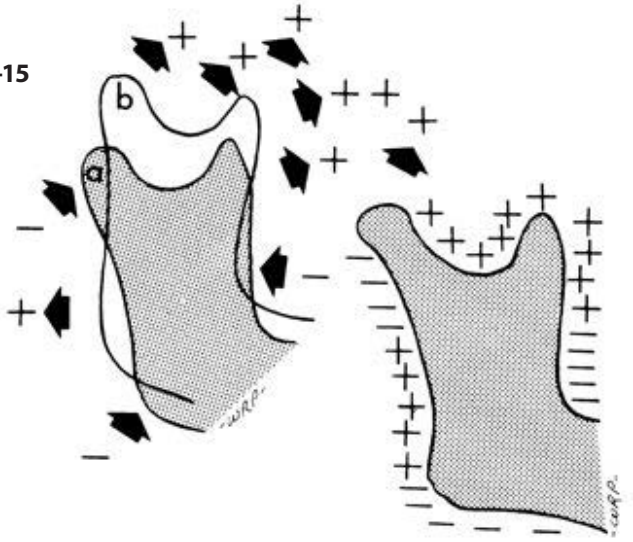
However, vertical lengthening of the ramus continues to take place **after** horizontal ramus growth slows or ceases (when the horizontal growth of the middle cranial fossa begins to slow and cease). This is to match the continued vertical growth of the midface. To achieve this, condylar growth may become more vertically directed, and a different pattern of ramus remodeling can also become operative (Fig. 4-15). The direction of deposition and resorption reverses. A **forward** growth direction can then occur in some individuals on the **anterior**

FIGURE 4-14



border in the upper part of the coronoid process. Resorption takes place on the upper part of the posterior border. A posterior direction of remodeling takes place in the lower part of the posterior border. The result is a more upright alignment and a longer vertical dimension of the ramus without a material increase in breadth. This remodeling change, when it occurs, appears to be more marked when the backward relocation of the ramus, to provide for corpus lengthening, has decreased. There are probably other relationships involved as well, including different facial and headform types, although the biologic basis is presently not fully understood. (See Hans, et al., 1995.)

FIGURE 4-15

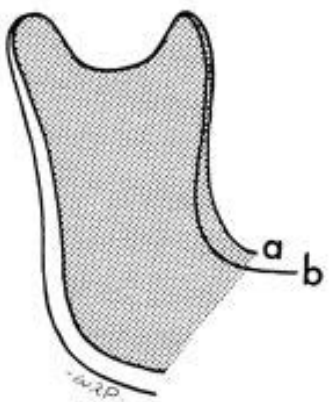


An important growth track jump of the growing ramus. This critical remodeling change is diagrammed in Figures 4-13 to 4-16 (and intermediate combinations). They show the adaptive remodeling capacity of the **whole** ramus for providing fitting of the mandibular corpus and dental arch to BOTH the temporal bone and to the growing, changing nasomaxillary complex. The WHOLE ramus itself, and the intrinsic signals controlling its growth, are the key agents that achieve this. These compensatory remodeling changes occur during the long childhood span as (1) the **vertical** enlargement of the nasal region and the maxillary arch significantly outpace (2) the **anterior** (protrusive) expansion of the temporal lobe, the Middle Cranial Fossae, and the underlying pharynx crossed by the ramus. Then, later is childhood, (1, above) continues by a significant extent while (2, above) slows and ceases. Ramus growth, thus, becomes confronted with a major problem. **Its protrusive development is required to significantly change and jump from one growth track to a different growth track** to accommodate this major timing transition. Progressive changes in the gonial angle are a consequence. Any developmental disturbance that prevents changing between these ramus tracks results in an undesirable variation in growth direction and possible malocclusion. THIS IS A ROUTINE RAMUS REMODELING ADAPTATION AND PLAYS A SIGNIFICANT ROLE IN THE EVERY DAY FACIAL GROWTH PROCESS.

The ramus thus undergoes a remodeling alteration in which its angle becomes changed in order to **retain** constant positional relationships between the upper and lower arches. Otherwise, development among all the diverse parts involved at different times, by different amounts, and in different directions would result in a marked misfit between the upper and lower jaws. This is another example of a developmental "compensation" (intrinsic adjustment) at work.

If mandible *a* in Figure 4-16 is superimposed over *b* in the anatomically functional position, it can be seen that all the complex remodeling changes outlined above serve simply to alter the ramus angle without increasing its breadth. This also accommodates the growing muscle sling and muscular adaptations associated with mandibular rotations. In addition, increased space for third molar eruption is provided.

FIGURE 4-16



The composite of vertical growth changes of the mandibular dentoalveolar arch, the ramus, and the middle cranial fossae must **match** the composite of vertical nasomaxillary growth changes to achieve continuing facial balance. Any differential will lead to a displacement type of mandibular rotation, either downward and backward or forward and upward. Normal variations of facial type and headform pattern are a common basis for such mandibular rotations.

The remodeling sequence shown in Figures 4-13 through 4-16, thus, are representative of countless intermediate variations taking place during development of the ramus. As documented by Cevidanes, et, al. (2003; 2005; 2005). The significant remodeling capacity of the ramus is clearly a key factor operating throughout the complex growth of childhood face.

During the descent of the maxillary arch and the vertical drift of the mandibular teeth, the anterior mandibular teeth simultaneously drift **lingually** and superiorly (Fig. 4-17). This produces a greater or lesser amount of anterior **overjet** and **overbite**. The remodeling process that brings this about (Fig. 4-18) involves periosteal resorption on the labial bony cortex (*a*), deposition on the alveolar surface of the labial cortex (*b*), resorption on the alveolar surface of the lingual cortex (*c*), and deposition on the lingual side of the lingual cortex (*d*).

At the same time, bone is progressively added onto the external surface of the mandibular basal bone area, including the mental protuberance (chin). The reversal between these two growth fields usually occurs at the point where the

concave surface contour becomes convex. The result of this two-way growth process is a progressively enlarging mental protuberance. Man is one of only two species having a **"chin"** (the elephant is the other, although the analogy is loose). Whatever its mechanical adaptations, the human chin is a phylogenetic result of downward-backward whole-face rotation into a vertical position, decreased prognathism (as described in Chapter 9), the marked extent of vertical facial growth, and the development of an overbite (in comparison to an end-to-end type of occlusion).

FIGURE 4-17

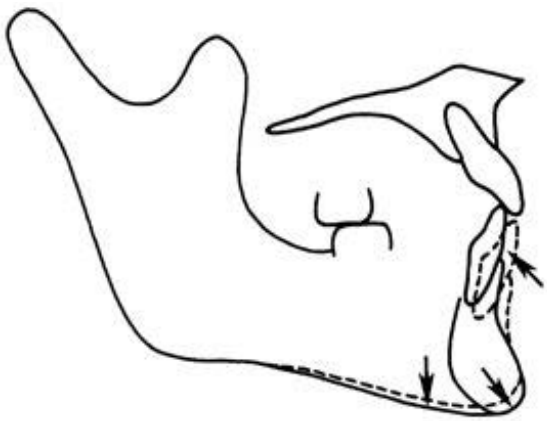
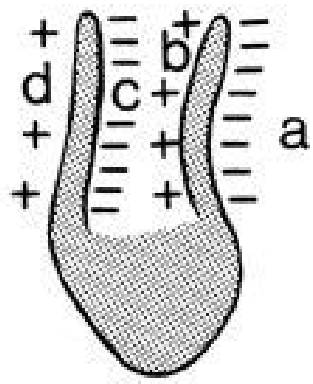


FIGURE 4-18



The Human Chin

In Chapter 9, the evolutionary factors of upright (bipedal) body posture and the greatly enlarged human brain, as they relate to a marked downward and backward rotational placement of the nasomaxillary complex, are described. For the mandible and its development, that situation has led to a serious evolutionary problem. This midfacial rotation has caught the human mandible in a closing vice, with the maxilla on the one side and the airway and other cervical and pharyngeal parts on the other. The result has placed these vital parts in real jeopardy. However, there have been three evolutionary adjustments, each based on contrasting headform and facial types. First, in many long-midface individuals, the "fitting" of the lower jaw has led to maxillary dental protrusion, deep overbite, and mandibular dental protrusion, thus relieving the closing vise. Second, in many short-face types, a tendency toward an anterior cross bite or bimaxillary protrusion has achieved an alternate adaptation to the problem. A third way is by anterior crowding, which shortens the mandible. All three adaptations result in a "malocclusion" in the clinical context, but nonetheless have served as phylogenetic answers (biologic compensations) to this evolutionary problem.

There is considerable variation in the placement of the reversal line between the resorptive alveolar and the depository parts of the chin; it may be fairly high or low. Variations also occur in the relative amounts of resorption and deposition. There are, correspondingly, marked variations in the shape and the size of the chin among different individuals. It is one of the most variable (but slow-growing)

areas in the entire mandible as seen among the different basic facial types and patterns. Interestingly, no orthopedic strategies target the bony chin. Increases in chin prominence occur via one or more of the following three pathways. First, natural bony deposition associated with the onset of puberty. Second, relative chin prominence related to uprighting of the lower incisors either as the result of orthodontic treatment or natural drift of the lower anterior teeth. And third, by surgically altering the bony chin with a genioplasty.

The Ramus-Corpus Combine

Most of the outer surfaces of the mandibular corpus receive progressive deposits of bone on both its buccal and lingual sides, with resorption occurring from the endosteal surfaces. (Resorptive periosteal areas occur, however, on the labial side of the incisor region and below the lingual tuberosity, as previously described.) The above changes enlarge the breadth of the corpus; the buccal side remodels, to a slightly greater extent than the lingual side because bony arch width increases slightly during postnatal mandibular development, but not as much as the bony maxillary arch increases in width. The ventral border of the corpus is also depository; this is a prolonged growth process, however, and progresses in concert with long-term masticatory development and dental arch maturation.

The amount of upward alveolar growth greatly exceeds the extent of downward enlargement by the "basal bone." (Note: Basal bone is a term sometimes used to denote that part of the corpus not involved in "alveolar" movements of the teeth. This area has a higher threshold of resistance to extrinsic forces than alveolar bone, which is extremely labile. There is no distinct structural line, however, separating basal from alveolar bone tissue. This is more of a physiologic than an anatomic difference.)

Whenever a change in the angle between the ramus and corpus develops, multiple sites of remodeling are involved. The adaptive trajectory of condylar growth is usually a factor (Fig. 4-19), as shown by *a, b*, and *c*. **Variable growth directions are produced by selective proliferation of prechondroblasts in some parts around the periphery of the condyle, with retardation of cell divisions in other parts.** Thus, "condylar growth" is an active **respondent** in developmental function that can adapt to the widely variable conditions imposed on it.

If **backward** (but not yet upward) condylar *and* ramus growth has slowed or largely ceased (Fig. 4-13), remodeling can produce angular changes of the ramus relative to the corpus by direct remodeling. Such remodeling processes can either close or open the "gonial angle." In fact, some clinical intervention strategies (most notably the vertical chin cup) attempt to alter gonial angle by **ramus** and dentoalveolar (not just "condylar") remodeling to achieve the desired clinical result.

Note these two fundamental points: It is the **entire ramus** that is involved, not just "condylar growth." Also, any change in the ramus-corpus ("gonial") angle is largely produced by **ramus** remodeling, not the corpus, and **is determined by the remodeling direction of the ramus with its condyle** (Fig. 4-20). This is a most important point because the **whole** ramus (not just the condyle) is a primary clinical target. It is remodeling combinations such as those shown in Figure 4-13

that are primarily responsible for ramus and corpus alignment positions relative to each other. Direct upward remodeling of the corpus, involving resorption on its inferior surface, does not ordinarily occur. A marked superior extent of alveolar bone growth and the drifting of anterior mandibular teeth, however, is common (see curve of Spee). The size of the antegonial notch is determined largely by the nature of the ramus-corpus angle and also by the extent of bone deposition on the underside (inferior margin) just posterior or anterior to the notch. The notch itself is also increased in size owning to its resorptive periosteal surface. A mandible characteristically has a less prominent antegonial notch (Fig. 4-20*b*) if the angle between the ramus and corpus becomes closed, and a much more prominent antegonial notch (a) if it becomes opened. The antegonial notch itself is surface resorptive because it is relocated posteriorly, as the corpus lengthens, into the former gonial region of the ramus (Fig. 4-9).

FIGURE 4-19

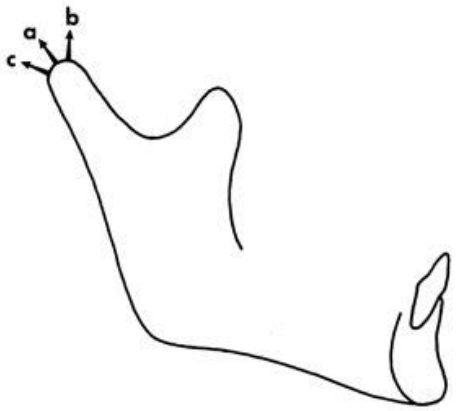
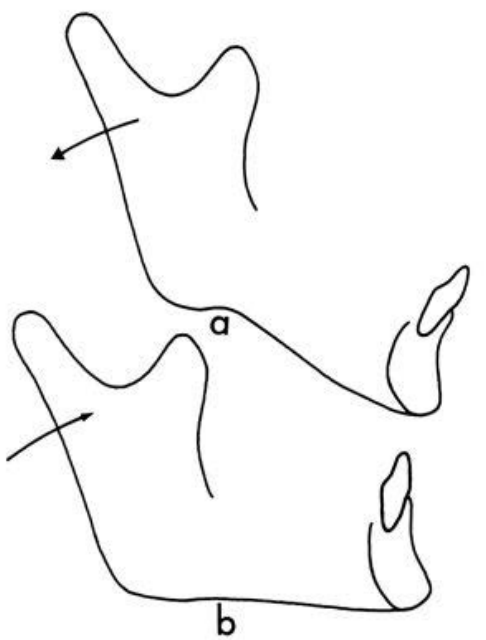


FIGURE 4-20



A significant point is that clinical manipulation of the ramus is effective only so long as it is actively engaged in mandibular growth. Thereafter, how to unlock its responsive remodeling capacity is poorly understood, since a developmental "balance" (Chapter 1) has long since been achieved with the vertical, anteroposterior and bilateral relationships with the basicranium, airway, nasomaxillary complex, dentition, tongue, and the masticatory and hyoid musculature.

5

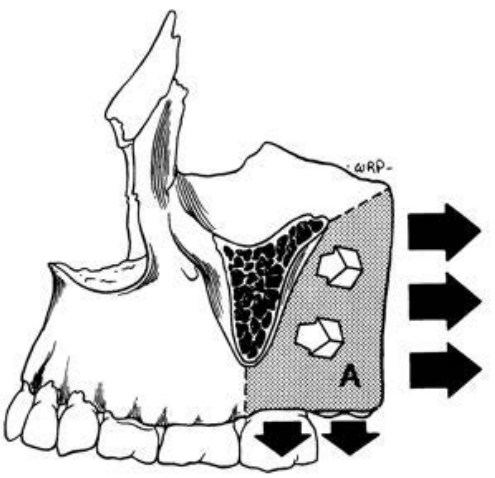
The Nasomaxillary Complex

Just as the mandible **remodels** in a predominately posterosuperior manner as it simultaneously becomes **displaced** in an opposite anteroinferior direction, the nasomaxillary composite also "grows" in a generally comparable way. Because of the notable differences between mandibular and nasomaxillary circumstances, however, the midface necessarily involves additional and significant developmental operations.

The Maxillary Tuberosity and Arch Lengthening

The horizontal lengthening of the bony maxillary arch is produced by remodeling at the maxillary tuberosity. The area shown in Figure 5-1*A* is the specific growth field that carries this out. It is a **depository** field in which the backward-facing periosteal surface of the tuberosity receives continued deposits of new bone as long as growth in this part of the face continues. The arch also widens, and the lateral surface is, similarly, depository. The endosteal side of the cortex within the interior of the tuberosity (the maxillary sinus) is resorptive. The cortex thus moves (relocates) progressively posteriorly and also, to a lesser extent, in a lateral direction. The maxillary sinus increases in size as a result. In the newborn, this sinus is quite small but becomes greatly expanded as growth continues and eventually occupies the greater part of the large suborbital compartment. (See page 179 for the interesting evolutionary significance of this region.)

FIGURE 5-1



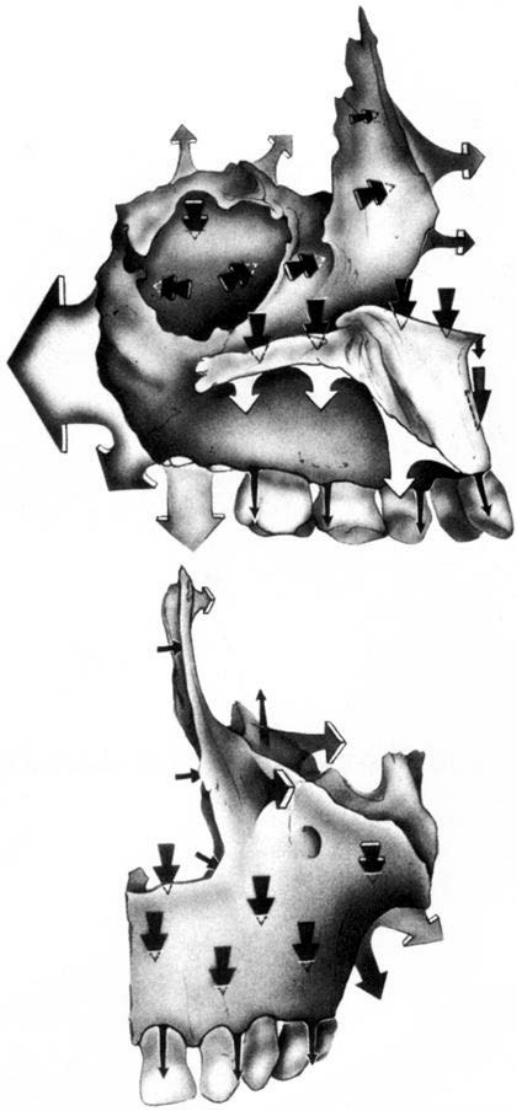
Because distal movement of the maxillary first molar is often part of an orthodontic treatment plan, the maxillary tuberosity is important in clinical orthodontics. Every mechanical option designed to move the maxillary first molar distally exploits the growth potential of the tuberosity. It is **this** depository field that allows the clinician to "increase arch length " by moving teeth into an area of bone deposition. When orthodontists decide to extract teeth to adjust arch length, this decision is based primarily on the **lower** arch discrepancy because of the potential to expand the upper arch both laterally and distally. A second reason is that, in a Class II molar relationship, such distal molar movement aids the clinician in achieving the treatment goal of a Class I molar relationship.

The maxillary tuberosity is a major "site" of maxillary growth. It does not, however, provide for the growth of the whole maxilla, but relates only to that area associated with the posterior part of the lengthening arch. Many other basic and important sites of growth also exist throughout the various parts of this architecturally and functionally complex bone (Figs. 5-2 and 5-3). Remember, also, that the position of the maxillary tuberosity is actually established by the posterior boundary of the anterior cranial fossa, and any clinically induced deviation could result in a developmental rebound. (See the **PM Plane** in other chapters.)

The whole maxilla undergoes a simultaneous process of **primary displacement** in an anterior and inferior direction as it grows and lengthens posteriorly (Fig. 5-4). Protraction face mask therapy to correct maxillary retrognathia finds its biologic basis for action in this displacement process. In Figure 5-5, extensive remodeling occurs throughout the nasomaxillary complex (*B* and *C*) as the entire region undergoes inferior (and anterior) displacement (*D*). The nature of the force that produces this anterior movement has, historically, been a subject of great controversy. One early theory (long since abandoned) suggested that additions of new bone on the posterior surface of the elongating maxillary tuberosity "push" the maxilla against the adjacent muscle-supported pterygoid plates. This presumably would cause a resultant shove of the entire maxilla anteriorly because of its own posterior bone growth activity. The idea was aborted, however, when it was realized that a bone's osteogenic membrane is pressure sensitive, and that the bone growth process does not have the physiologic capacity to actually push the whole bone away from the other bones by itself. The reason, simply, is that a surface force exerting pressure causes compression that would press closed the sensitive capillary plexus within the vascular osteogenic connective tissue. This renders it inoperable and leads to necrosis.

Another theory held that bone growth within the various maxillary *sutures* pushed apart the bones, with a resultant thrust of the whole maxilla anteriorly (and inferiorly as well). Although this old explanation is still sometimes heard, it has been soundly rejected for the reason just mentioned: bone tissue is not capable of growth in a field that requires the levels of compression needed to produce a "pushing" type of displacement. Bones cannot push on other bones to create the tissue separating force necessary for displacement. The sutural connective tissue is not adapted to a pressure-related growth process (in contrast to cartilaginous mediated bone-to-bone articular contacts, which are much more compression tolerant). The suture is essentially a **tension-adapted** tissue. This is a basic

FIGURE 5-2



difference. Its collagenous fiber construction is a functional design for traction accommodation across the connective tissue bridge between separate bones. The presence of any unusual pressure on a suture triggers bone resorption, not deposition, to relieve the pressure. This decreases the pressure by removing some of the bone.

It is believed that the stimulus for sutural bone growth (remodeling) relates to the tension produced by the **displacement** of that bone. The deposition of the new bone occurs in tandem with displacement, rather than the force that causes it. (See later in this chapter and also Chapter 14 for further discussion of the sutural growth process.) Thus, as the entire maxilla is **carried** forward and downward by displacement, the osteogenic sutural membranes form new bone tissue that enlarges the overall size of the whole bone and sustains constant bone-to-bone contact via the sutures.

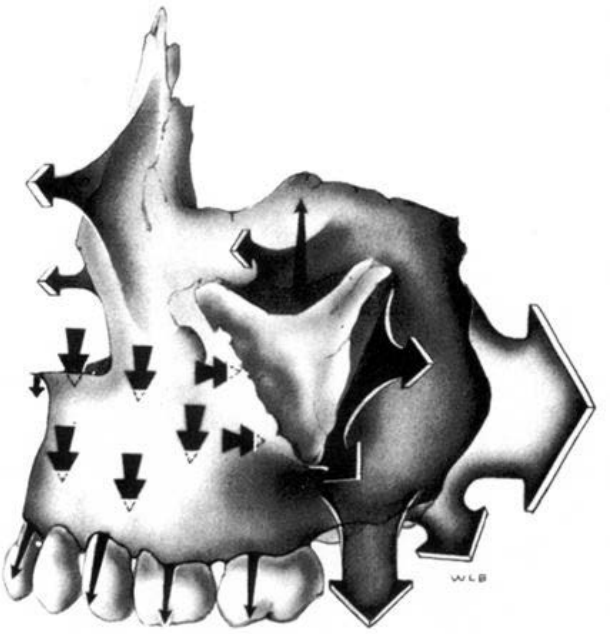


FIGURE 5-3.
(From Enlow, D. H.: *The Human Face*. New York, Harper & Row, 1968, p. 164, with permission.)

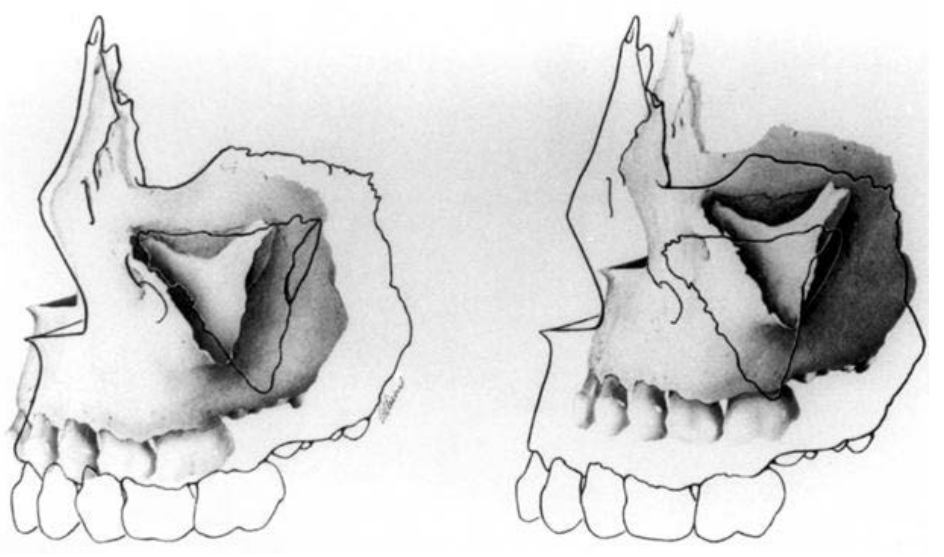


FIGURE 5-4.
(From Moyers. R. E., and D. Enlow: In: *Handbook of Orthodontics*. 4th Ed. Chicago, Mosby-Yearbook. Inc., 1988, with permission.)

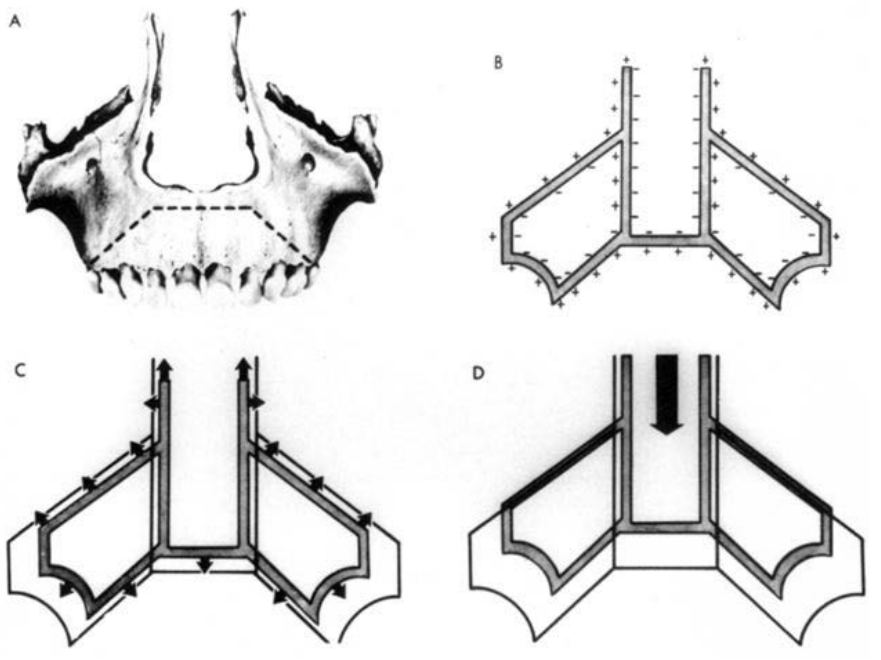
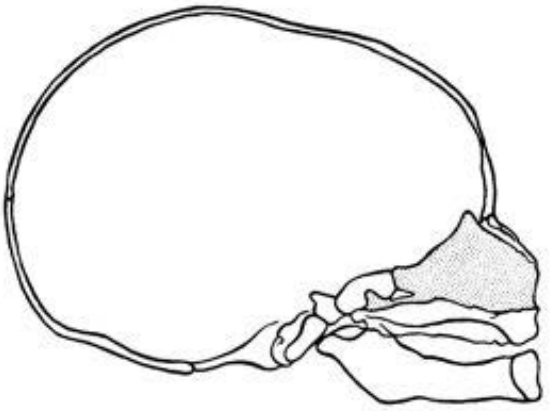


FIGURE 5-5.
(From Moyers, R. E., and D. Enlow: In.: *Handbook of Orthodontics*, 4th Ed. Chicago, Mosby-Yearbook, Inc., 1988, with permission.)

The Biomechanical Force Underlying Maxillary Displacement

An early explanation for maxillary displacement is the now famous "*nasal septum*" theory (Fig. 5-6). This was developed largely by Scott, and the premise for the idea was quite reasonable. It developed from the criticisms of the "sutural theory" described above. The hypothesis was soon adopted by many investigators around the world and became more or less the standard explanation for a number of years, replacing the sutural theory. **Cartilage** is specifically adapted to certain pressure-related growth sites, as mentioned before, because it is a special tissue uniquely structured to provide the capacity for growth in a field of compression. (See Chapter 14.) Cartilage is present in the epiphyseal plates of long bones, in the synchondroses of the cranial base, and in the mandibular condyle, where it relates in each case to linear growth by endochondral proliferation. Whereas the cartilaginous nasal septum itself contributes only a small amount of actual endochondral growth, the basis for the "septal" theory is that the pressure-accommodating **expansion** of the cartilage in the nasal septum provides a source for the physical force that displaces (pushes) the whole maxilla anteriorly and inferiorly. This sets up fields of tension in all the maxillary sutures. The bones then secondarily, but virtually simultaneously, enlarge at their sutures in response to the tension created by the displacement process. (See also page 236.)

FIGURE 5-6



As with any important explanatory theory, the nasal septum concept subsequently received a great deal of laboratory study to test validity. Ingenious experiments led to inconclusive results subject to multiple and uncertain interpretations However, the consensus today is that growth of the septum is one of a number of factors that actively displace the maxilla.

The reasons why the source of the forces causing displacement of the maxilla have remained unresolved are that the source of maxillary displacement is multifactorial in nature. Although the nasal septum itself is indeed involved, many other factors contribute as well, and it is very difficult to separate respective effects even in controlled laboratory experiments. For example, when experimental studies involve surgical removal of parts (such as the septum) to presumably test the nature of their functional roles in growth, the studies simply cannot account for the multiple variables introduced by the experimental procedure itself. These variables include the destruction of tissues, blood vessels, and nerves playing a role in the growth process. Critics of these studies point out that the experimental removal of a given part does not necessarily demonstrate what the role of that part actually is when present *in situ*. It merely shows how the growth process functions in the absence of that part, rather than in its presence. A basic point is usually not acknowledged: if one experimentally changes some structure, as by surgical deletion, and this, in turn, affects the growth process, one simply cannot thereby conclude that this structure, thus, "controls the growth process."

Another important biologic consideration is the concept of "*multiple assurance*" (Latham and Scott, 1970). The processes and mechanisms that function to carry out growth are virtually always multifactorial. Should any one determinant of the growth process become inoperative (as by pathology or by experimental deletion of an anatomic part), other morphologic components in some instances have the capacity to "compensate." That is, they provide an alternative means to achieve more or less the same developmental and functional end result, although perhaps with some degree of anatomic distortion. This concept has far-reaching implications, necessitating caution in the interpretation and evaluation of facial growth experiments utilizing laboratory animals.

As with the cartilaginous mandibular condyle, there can be no actual genetic determinates within the septal cartilage itself (a blueprint for the maxilla).

Another theory that attempts to account for the biologic force leading to displacement is the **functional matrix** concept. Melvin Moss was the primary architect and proponent of this theory. The functional matrix concept states, in brief, that any given bone grows in response to functional relationships established by the sum of all the soft tissues operating in association with that bone. This means that the bone itself and its osteogenic membranes do not genetically regulate the rate and directions of their own growth; the functional soft tissue matrix, rather, is the "epigenic" governing determinant of the skeletal growth process. The course and extent of bone growth are secondarily dependent upon the growth and the functioning of pacemaking soft tissues. Of course, the bone and any cartilage present are also involved in the operation of the functional matrix, because they participate in giving essential feedback information to the governing soft tissues (muscles, etc., see Fig. 1-7). This causes the osteogenic and chondrogenic tissues to inhibit or accelerate the rate and amount of subsequent bone remodeling activity, depending on the status of functional and mechanical equilibrium between the bone and its soft tissue matrix. A basic concept is that genetic as well as functional determinants of the growth process reside wholly in the related soft tissues, giving control signals to the "genic" tissues, and not in the hard part of the bone itself.

The functional matrix concept is basic to an understanding of the fundamental nature of a bone's role in the overall process of growth control. This concept, historically, has had great impact in the field of facial biology.

The functional matrix concept also comes into play as a source for the mechanical force that carries out the process of displacement. According to this now popular explanation, the facial bones grow in a subordinate growth control relationship with all the surrounding, pace-making soft tissues. As those tissues continue to grow, the bones become passively (i.e., not of their own doing) **carried** along (displaced) with the soft tissues attached to the bones by the Sharpey fibers. Thus, for the nasomaxillary complex, the growth expansion of the facial muscles, the subcutaneous and submucosal connective tissues, the oral and nasal epithelia lining the spaces, the vessels and nerves, and so on, all combine to move the facial bones passively along with them as they grow. This continuously places each bone and all of its parts in correct anatomic positions to carry out its functions, **because the functional factors are the very agents that cause the bone to develop into its definitive shape and size and to occupy the location it does.**

The functional matrix concept is a useful paradigm and provides an intellectual framework to discuss the complex interrelationships that operate during facial growth. It is to be realized, however, that this principle is not intended to explain **how** the growth control mechanism actually functions. This concept describes essentially **what** happens during growth; it does not presume to account for the regulatory processes at the cellular and molecular levels that carry it out. This is a basic point.

The term "functional" matrix, however, can be misleading in the sense that it unintentionally implies that function, as in the forces of muscle contractile function, is the only or primary determinant. Just as basic to the big picture of "growth" is the expansive biomechanical **force of growth itself**. For example, increases in body size require a larger airway, and the expansive growth of the

nasal airway influences midfacial growth. A significant force is exerted by the process of growth expansion that has a major traction effect on attachment fibers to bone, thereby moving the bones in displacement. (See Fig. 1-7). The forces of (1) function and (2) growth itself are equally basic, and **both** must be taken fully into account; either without the other is incomplete.

NASOMAXILLARY REMODELING

An important concept, clinically as well as biologically, is that *all* inside and outside parts, regions, and surfaces participate directly in growth (Figs. 5-2 and 5-3). The old idea of centralized and self-contained "growth centers" (such as presumed growth-controlling sutures) is contrary to the actual biology involved and prevents any realistic grasp of the developmental interplay that occurs. There are, of course, differentials in the timing and magnitude among all the localized regions, but they all nonetheless take active part in response to the activating signals that trigger their local "genic" tissues. Furthermore, because of the developmental and functional interrelations among them, what occurs in any one region is not developmentally isolated from the others. This has profound clinical implications in terms of responses to treatment procedures presumably targeting on some particular area. See also page 103.

The Lacrimal Suture: A Key Growth Mediator

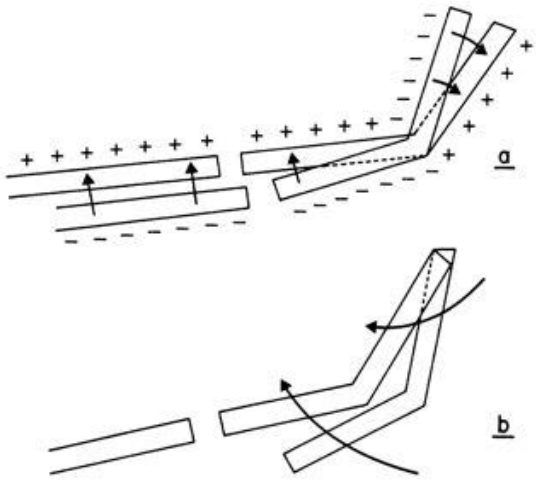
This is a significant but unsung growth site that provides a developmental function so important and so basic and so interesting that it merits special consideration. What it does as a growth function has gone unrecognized, yet its unique role in facial development has made possible a headform design that **works**. Without it, human (and mammalian) craniofacial development could not have evolved and could not have resulted in a functional assembly of parts. It is simply that important and fundamental.

The **lacrimal** bone is a diminutive flake of a bony island with its entire perimeter bounded by sutural connective tissue contacts separating it from the many other surrounding bones. As all these other separate bones enlarge or become displaced in many directions and at different rates and different times, the **sutural system** of the lacrimal bone provides for the "slippage" of the multiple bones along sutural interfaces with the pivotal lacrimal as they all enlarge differentially. This is made possible by collagenous linkage adjustments within the sutural connective tissue (see page 282). The lacrimal **sutures** make it possible, for example, for the maxilla to "slide" downward along its orbital contacts. This allows the whole maxilla to become displaced inferiorly, a key midfacial growth event, even though all the **other** bones of the orbit and nasal region develop quite differently and at different times, amounts, and directions. Without this adjustive developmental "perilacrimal sutural system," a developmental **gridlock** would occur among the multiple developing parts. The lacrimal bone and its suture is a developmental hub providing key traffic controls.

The lacrimal bone itself undergoes a remodeling rotation (Fig. 5-7), because

the more medial **superior** part remains with the lesser-expanding nasal bridge, while the more lateral **inferior** part moves markedly outward to keep pace with the great expansion of the ethmoidal sinuses. This remodeling change is illustrated by *a*; the primary rotational displacement that accompanies it is shown by *b*. (See page 249 for additional information on lacrimal and orbital development and the biologic rationale involved.) Refer also to page 98.

FIGURE 5-7



The Maxillary Tuberosity and the Key Ridge

In the growth of the bony maxillary arch, area *A* in Figure 5-1 is moving in three directions by bone deposition on the external surface: it lengthens **posteriorly** by deposition on the posterior-facing maxillary tuberosity; it grows **laterally** by deposits on the buccal surface (this widens the posterior part of the arch); and it grows **downward** by deposition of bone along the alveolar ridges and also on the lateral side, because this outer surface slopes (in the child) so that it faces slightly downward. The endosteal surface is resorptive, and this contributes to maxillary sinus enlargement.

In Figure 5-8, note that a major change in surface contour occurs along the vertical crest just below the malar protuberance (small arrow). This crest is called the "key ridge." A **reversal** occurs here. Although a range of variation occurs in the exact placement of the reversal line, anterior to it most of the external surface of the maxillary arch (the protruding "muzzle" in front of the cheekbone) is **resorptive**. This is because that part of the bony arch in area *b* is **concave**, and the labial (outside) surface faces upward, rather than downward. The resorptive nature of this surface provides an inferior direction of arch remodeling in conjunction with the downward growth of the palate. This is in contrast to area *a*, which grows downward by periosteal **disposition**. See page 91 for descriptions of this region as it participates in the posterior (distal) remodeling of the tuberosity and malar region.

In Figure 5-9, surface *a* is resorptive; *b* is depository. A reversal occurs at "*A* point" (indicated by arrow, a much-used cephalometric landmark). Periosteal surface *c* is resorptive, *d* is depository, *e* is resorptive, and *f* is depository.

FIGURE 5-8

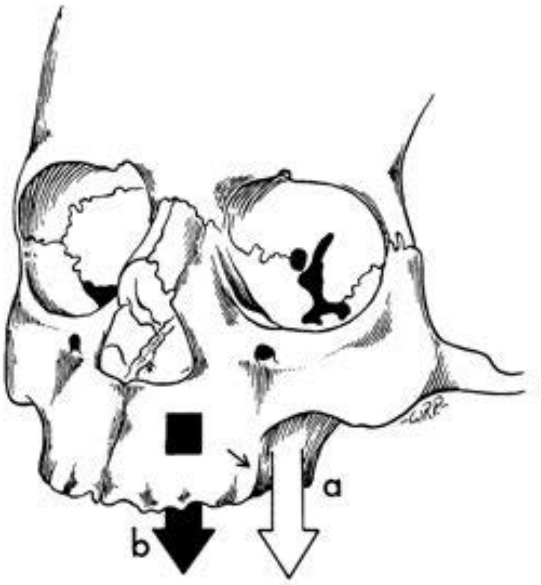
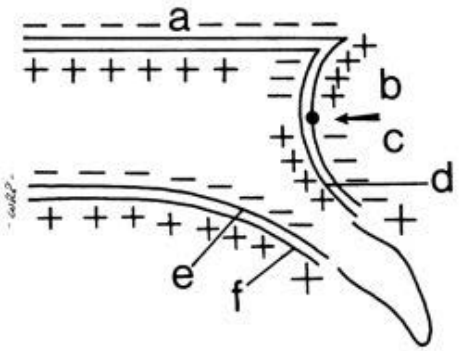


FIGURE 5-9



The Vertical and Mesial Drift of Teeth: Important Clinical Concepts

Because they are supported by a periodontal membrane, in the absence of pathology, teeth never become directly attached to the alveolar bone. After teeth erupt into occlusion there is continued movement of the teeth within the alveolar process. The naturally occurring movements are in the mesial direction in the anteroposterior dimension, towards the occlusal plane in the vertical dimension, and buccally in the transverse dimension. These movements are absolutely essential for normal development of the maxilla and mandible and allow teeth to maintain contact during active growth. Since these movements occur in the absence of extrinsic tooth moving pressure and are not part of the eruption process, we refer them as "**Mesial, Lateral and Vertical Drift**". Although some texts refer to all

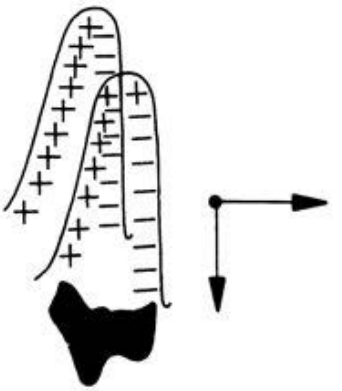
vertical movements of teeth as simply "eruption" or "extrusion/intrusion" when applied clinically, this terminology is misleading. The developmental **vertical, mesial and lateral** movements of teeth that occur after teeth are in occlusion are marked in extent and play a key role in maxillary and mandibular morphogenesis. Many orthodontic clinicians emphasize that "working with growth" is a primary objective in treatment and one of the main reasons that orthodontists can treat a wider range of malocclusions in the rapidly growing patient. Therefore it is critical for the clinician to differentiate tooth eruption from tooth drift. The only instances where clinicians target the process of dental eruption itself are when impacted teeth are surgically exposed and mechanically erupted into the mouth. ALL other instances of tooth movement are intimately related to the process of drift.

An important clinical point is that orthodontic tooth movements in the direction of natural drift occur more rapidly attempting to move teeth in opposite directions. Put more simply, in the growing patient it is easier to move teeth toward the occlusal plane in the vertical dimension, mesially in the anteroposterior direction and laterally in the transverse dimension.

As a tooth drifts mesially (or distally, depending on species and which tooth), note that the same process of alveolar remodeling (resorption and deposition) relates to a vertical movement of the tooth as well. Any tipping, rotations, or buccolingual tooth movements are also simultaneously carried out by the same versatile remodeling process. As a tooth bud develops and its root elongates, the growing tooth undergoes **eruption**, bringing its crown into definitive occlusal position above the bone and gingiva. Now, the vertical drift of a tooth **thereafter** (which is considerable) is **in addition to** eruption, and use of the term eruption for this vertical drifting is inappropriate. As the maxilla and mandible enlarge and develop, the dentition drifts both vertically and horizontally to keep pace in respective anatomic positions. The process of drift moves the whole tooth **and** its socket; that is, the tooth does not drift vertically out of its alveolar housing as it does in eruption (or as implicit in the term "extrusion"). Rather, in vertical drift, the socket and its resident tooth drift **together** as a unit (Fig. 5-10). Actually, unerupted tooth buds also undergo drifting in order to sustain their anatomic positions. The periodontal connective tissue also moves together with drifting teeth, but it does not merely "shift" along with its tooth. Rather, it undergoes extensive **remodeling** within itself to relocate (see page 131). It is this important periodontal connective tissue membrane that (1) provides the intramembranous (hence periodontal "membrane" rather than "ligament") bone remodeling that changes the location of the alveolar socket and (2) moves the tooth itself. The horizontal, lateral and, especially, the vertical distances moved by the socket, its tooth, and the periodontal membrane can be substantial. By harnessing dental drift movements, the orthodontist can more readily guide teeth into calculated positions, thereby taking advantage of the growth process ("working with growth"). In cases where only the upper and lower anterior teeth are bonded along with the first molars, the clinician will use the vertical drift of the teeth without braces as a guide. In these partially banded cases, the clinician is attempting to selectively modify vertical drift in one area and allowing normal drift to occur in others. This concept may be useful to understand treatment response in partially banded cases. The effect of

drift can be seen in almost every patient where the second molars are not bonded at the same time as the rest of the permanent teeth. In fact, unbonded second molars give the clinician a intraoral guide to the magnitude and direction of the normal drift of the dentition.

FIGURE 5-10



When all of the teeth are bonded, drift of the entire dental arch is influenced by the orthodontist. Although the braces are attached to the teeth the biologic goal is bring about the remodeling ("relocation") of each individual alveolar socket as the connective tissue simultaneously moves the tooth as directed by the clinically controlled "signals." The mediator of these therapeutic signals and the biologic target is the periodontal membrane. If all teeth are included in the orthodontic appliance, the clinician does not have any vertical drift reference to help gauge treatment response. This can be problematic and may require progress imaging to determine treatment response. Since it is possible for clinicians to induce "signals" that move teeth into biologically unstable positions, care must be taken when all biologic markers of drift (i.e. the teeth) are incorporated into the orthodontic appliance.

With the advent of temporary anchorage devices (TADs) the ability of the clinician to harness the effects of dental drift for therapeutic benefit is increased. Clinicians in the Pre-TADs era were able to substantially augment drift in the naturally occurring directions (lateral, mesial, toward the occlusal plane) but were limited in their ability to influence drift in other directions (Medial, Distal, away from the occlusal plane). Clinicians in the Post-TAD will be able to control drift in all three dimensions and in all directions. With increased therapeutic power comes increased biologic responsibility. Clinicians of the future must fully understand the impact of drift on treatment results or risk serious complications for their patients.

The Nasal Airway

The lining surfaces of the bony walls and floor of the nasal chambers are predominantly resorptive except for the nasal side of the olfactory fossae (See Fig. 1-9.) This produces a lateral and anterior expansion of the nasal chambers and a downward relocation of the palate; the oral side of the bony palate is depository. The small, paired olfactory fossae have a resorptive endocranial surface that lowers

them in conjunction with the downward cortical remodeling of the entire anterior cranial floor.

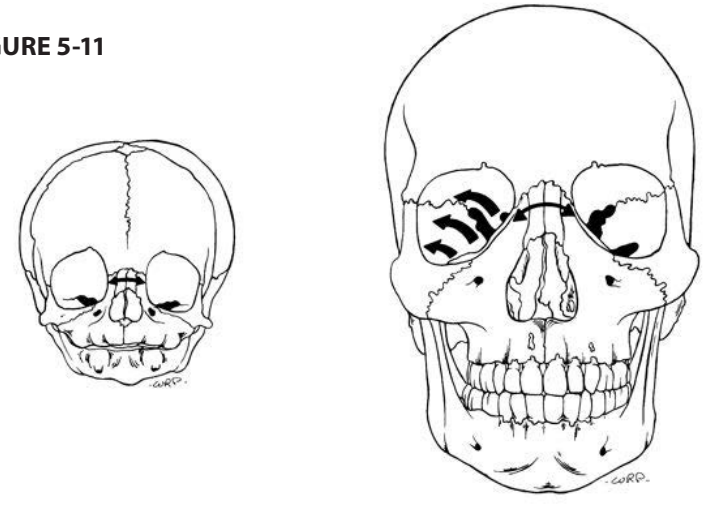
The ethmoidal conchae generally have depository surfaces on their lateral and inferior sides and resorptive surfaces on the superior and medial-facing sides of their thin bony plates. This moves them downward and laterally as the whole nasal region expands in like directions. (The developmentally separate inferior concha, however, can show remodeling variations because it is carried inferiorly, to a greater extent than the others, by maxillary displacement.) The lining cortical surfaces of the maxillary sinuses are all resorptive, except the medial nasal wall, which is depository because it remodels laterally to accommodate nasal expansion (Fig. 5-5).

A basic and important concept of the facial growth process is underscored in Figure 1-9: it is the **entire** facial complex that participates in the growth process. **All** parts and bony surfaces are directly involved, not merely certain special sites and "centers." All are necessarily interrelated, and the developmental positioning, shaping, and sizing of any one part affects all the others. (See the "keystone" analogy, page 13.)

The bony portion of the internasal septum (the vomer and the perpendicular plate of the ethmoid) lengthens vertically at its various sutural junctions (and to a much lesser extent by endochondral growth where the cartilaginous part contacts the perpendicular plate of the ethmoid). The bony septum also warps in relation to variable amounts and directions of **septal deviation**. The remodeling patterns involved are individually variable, and the thin plate of bone typically shows alternate fields of deposition and resorption on the right and left sides, producing a buckling to one side or the other.

Note that the breadth of the nasal bridge in the region just below the frontonasal sutures does not markedly increase from early childhood to adulthood (Fig. 5-11). More inferiorly in the interorbital area, however, the medial wall of each orbit (lateral walls of the nasal chambers between the orbits) expands and balloons out considerably in a lateral direction in conjunction with the considerable extent of lateral enlargement of the nasal chambers. The ethmoidal sinuses are thereby enlarged greatly.

FIGURE 5-11



Palatal Remodeling

Even though the external (labial) side of the whole anterior part of the maxillary arch (the protruding "muzzle") is resorptive, with bone being added onto the **inside** of the arch, the arch nonetheless increases in width, and the palate becomes wider (Fig. 5-12). This is another example of the V principle. In addition, growth along the midpalatal suture is known to participate to a greater or lesser extent in the progressive widening of the palate and alveolar arch (not shown in this schematic diagram). The extent can vary between the anterior and posterior regions. (See also pages 19, 57, and 214 for other palatal growth adjustments.)

As the palate grows inferiorly by the remodeling process, a nearly complete exchange of old for new hard and soft tissue occurs. At each succeeding descending level, the palate becomes, literally, a different palate. It occupies a different position and is composed of different bone, connective tissue, epithelia, blood vessels, nerve extensions, and so on. When one visualizes the palate of a newborn and a young child, it should be realized that the palate in that same person at an older age is not the same palate at all

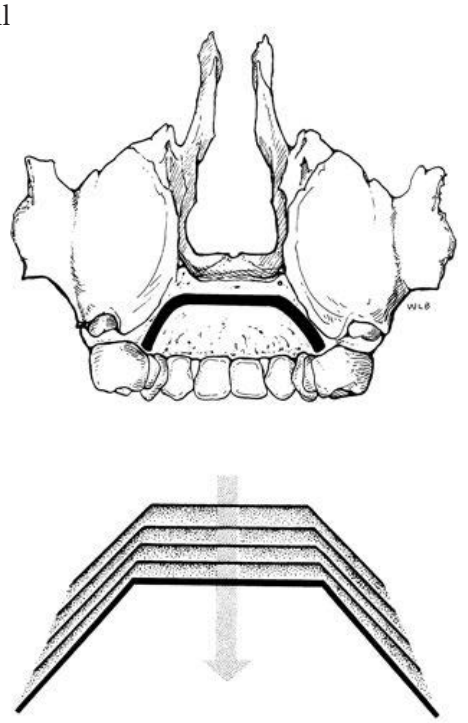


FIGURE 5-12.
(From Enlow, D. H., and S. Bang: Growth and remodeling of the human maxilla. Am. J. Orthod., 51:446, 1965, with permission.)

The rotations, tipping, and inferior drift of the individual maxillary teeth, in combination with the characteristic external bony resorptive surface of the whole forward part of the maxilla, sometimes result in a localized rupture and protrusion of a tooth root tip through the bony cortex. Such penetration results in a normal defect (i.e., a tiny surface hole in the bone) called a fenestra.

Rapid or slow palatal expansion has become a very common clinical technique. Historically, the process of expanding the maxilla by "splitting" the midpalatal suture has been thought of as "biologic" procedure. Such is not the case. Natural increases in palatal width are the result of vertical drift of the posterior teeth with expansion laterally occurring according to the V principle of growth. Therapeutically induced expansion of the midpalatal sutures is an entirely different process. In rapid palatal expansion the maxillae are first displaced laterally. Remodeling of the displaced maxillae follows the clinically induced displacement. Understanding that this process is **not** the same as the natural biology of increasing maxillary arch width has two important clinical consequences. First, it **is** possible to expand the maxilla into an unstable (imbalanced) position and, if the clinician retains the maxillary shelves with a tooth-born device, fenestration of the molar and bicuspid roots is an almost certain consequence. (See Herberger, Thomas 1990) Remember that the lateral aspect of much of the maxilla is resorptive, **not** depository. Moving teeth into areas of natural resorption is problematic at best and disastrous at worst. The second important clinical point is that, because the midpalatal suture probably plays only a small role (if any) in the displacement of the maxillary shelf laterally, it should be clinically possible to increase maxillary arch width even after fusion of the midpalatal suture. Such increases in arch width would necessarily result from remodeling of the alveolar process laterally and inferiorly. Stability of such nonsutural expansion should be subject to the same rules of biologic balance that apply to expansion achieved by the separation of the suture. The underlying biology of expansion underscores the importance of allowing for a period of physiologic rebound after rapid or slow palatal expansion.

How palatal remodeling participates in adjustments for maxillary rotations was explained in Chapters 2 and 3.

Downward Maxillary Displacement

The **primary displacement** of the whole ethmomaxillary complex in an inferior direction (Fig. 5-5) is accompanied by simultaneous remodeling (resorption and deposition) in all areas, inside and out, throughout the entire nasomaxillary region.

New bone is added at the frontomaxillary, zygotemporal, zygosphenoidal, zygomaxillary, ethmomaxillary, ethmofrontal, nasomaxillary, nasofrontal, frontolacrimal, palatine, and vomerine sutures. These multiple sutural deposits accompany displacement and are not the pacemaker for it. The process of displacement produces the "space" within which remodeling enlargement occurs. Sutural bone growth does not **push** the nasomaxillary complex down and away from the cranial floor. The displacement of the bones is produced by the expanding soft tissues (Fig. 1-7). As the bones of the ethmomaxillary region (Fig. 1-6) are displaced downward (*a*), sutural bone growth (*b*) takes place at the same time in response to it, thus enlarging the bones as the soft tissues continue to develop. This places all the bones in new positions in conjunction with the generalized expansion of the soft tissue matrix and maintains continuous sutural contact as the bones become "separated." See also page 57.

The balance between the greater or lesser amounts of displacement and remodeling growth in the posterior and anterior parts of the maxilla is a response to the clockwise or counterclockwise rotatory displacements caused by the downward and forward growth of the middle cranial fossa. The nasomaxillary complex must correspondingly undergo a compensatory ***remodeling rotation*** in order to sustain its proper position relative to the vertical reference (PM) line and to the neutral orbital axis (see also descriptions for Figs. 2-11 and 3-14).

Maxillary Sutures

Most sutures in the facial complex do not simply grow in directions perpendicular to the plane of the suture itself. This was pointed out in a previous stage with respect to the lacrimal sutures. Because of the multidirectional mode of primary displacement and the differential extents of growth among the various bones, a slide or slippage of bones **along** the plane of the interface can be involved. As the whole maxillary complex is displaced downward and forward, or as it remodels by deposition and resorption, it undergoes a frontal **slide** at sutural junctions with the lacrimal, zygomatic, nasal, and ethmoidal bones. This is schematized by a slip of *b* over the sutural front of *a* as shown in Figure 5-13. The process requires adjustment remodeling and relinkages of the collagenous fiber connections within the sutural connective tissue across the suture (see Chapter 14).

It is apparent that the downward **and** forward directions of movement occur at the same time, and that they are produced by the same actual displacement process. A suture is another regional site of growth adapted to its own localized, specialized circumstances, just as all the other parts of the bone have their own regional growth processes. It is not possible for a bone to grow **just** at its sutures, as was sometimes implied in years past. Nor is it possible for a bone to have "generalized surface growth" without sutural involvement (in areas where sutures are present, of course; nonsuture regions may enlarge by direct remodeling). Although sutures become partially fused as skeletal growth slows, bone continues to enlarge in the sutural areas as depicted in Figure 5-14 where remodelling on surface *x* enlarges the surface area of the bone, but additions must **also** be made by deposits at sutural surface *y* in order to maintain morphologic form. It is apparent that it would not be possible for the bone to enlarge in surface area without corresponding additions at the sutural contacts.

The downward movement of the teeth from *1* to *2* in Figure 3-17 is accomplished by a **vertical drift** of each tooth in its own alveolar socket as the socket itself **also** drifts (remodels) inferiorly with it in lock-step by deposition and resorption. The movement of the dentition from *2* to *3*, however, is a passive **carrying** of the maxillary dental arch as a whole, the palate and bony arch, all associated soft tissues, and all of the alveolar sockets as the **entire** maxilla is **displaced** downward as a unit. The *1* to *2* and *2* to *3* movements are shown separately, but, of course, actually proceed simultaneously. Recognition and understanding of the biologic difference between them are of basic importance because each represents a separate biologic target for different clinical procedures.

Some orthodontic procedures are designed to alter the vector (magnitude and

FIGURE 5-13

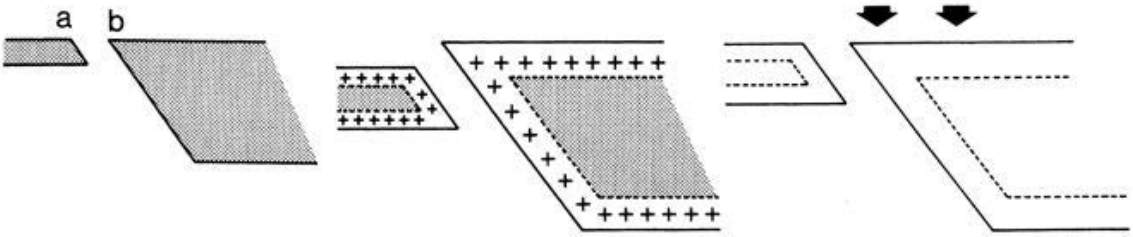
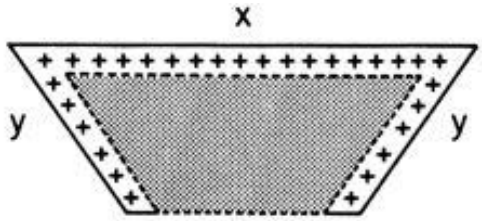


FIGURE 5-14



course) of the displacement movement (e.g., to accelerate or restrain it or to change direction). The specific target is thus the growth activity of the various maxillary sutures and other regional growth sites associated with the displacement process. A good example of an orthopedic force system designed to modify displacement at the sutures is the use of maxillary orthopedic traction, using a face mask attached to an intraoral device that is attached to the maxillary dentition and in contact with the palatal tissue (a bonded rapid palatal expander is often the method of choice). If an expansion screw is incorporated into the attachment device it can be used to separate the maxillary sutures by lateral movement of the maxillae. Subsequent to this lateral movement, anterior traction is applied to literally pull the maxilla forward and transiently disarticulate the bone at its sutural margins. To the extent that the bonded device contacts the palatal tissue and thus prevents tooth movement, this orthopedic approach is in contrast to the use of full bonded orthodontic appliances in which the periodontal connective tissue and drift movements of individual teeth (*1* to *2*) are the direct clinical target. In the mandible, similarly, the displacement movement is one target for treatment (as by a restraining chin cup), and horizontal and vertical drift movements of teeth, separately, are another. The former utilizes (it is hoped) regulation of ramus (and with it, condylar) growth, and the latter involves control of growth movements related to the periodontal "genic" tissues. In both the maxilla and the mandible, **both** types of movements occur most actively during childhood growth, of course. Utilization of such techniques in adult patients is not supported by the underlying biology of facial growth. Of some clinical importance is the fact that since drift and displacement are significantly reduced in adults, the outcome of surgically induced skeletal changes are more stable in skeletally mature adults (over age 25). The biologic rationale is that because the ability of the body to physiologically rebound is limited post treatment change will also be limited. Clinical studies

support this rationale for anteroposterior and vertical movements but not for transverse changes.

The remodeling and displacement changes of both the ramus and the middle cranial fossa produce a lowering of the mandibular arch. This accommodates the vertical expansion of the nasomaxillary complex. To bring the upper and lower teeth into full occlusion, the mandibular teeth must drift (not simply erupt) vertically (Fig. 3-18). The extent can vary considerably among different individuals having different facial types, and it can also vary markedly between the anterior and posterior parts of the arch. The latter is involved in occlusal plane rotations (see Figure 10-31). Significantly, the amount of upward mandibular tooth drift can be much less than the downward drift and displacement of the maxillary teeth, depending on headform type and location within the arch. This is one of several reasons that orthodontic procedures in the past often attacked the maxillary dentition, even though a given malocclusion was based on positioning of the mandible. This produced an "imbalance" in the maxilla to offset the effect of an existing skeletal situation in the mandible (or basicranium). Although this approach established a class I occlusion the resulting facial appearance was often less than optimal. Because of this, more modern approaches to orthodontic diagnosis and treatment planning attempt to position the upper incisor in an esthetically pleasing position within the face rather than adapting upper incisor position to an underlying skeletal imbalance.

The Cheekbone and Zygomatic Arch

The growth changes of the malar complex are similar to those of the maxilla itself. This is true for the remodeling process as well as the displacement process (Figs. 5-15 to 5-17).

The posterior side of the malar protuberance within the temporal fossa is depository. Together with a resorptive anterior surface, the cheekbone relocates **posteriorly** as it enlarges. It would seem untenable that the whole front surface of the cheek area can actually be **resorptive**, considering that the face "grows forward and downward." However, as the maxillary arch remodels posteriorly, the malar region must also move backward at the same time to keep a constant relationship with it. The extent of malar relocation is somewhat less in order to maintain **relative** position along the increasing length of the maxillary arch. The zygomatic process of the maxilla thus behaves in a manner similar to that of the coronoid process of the ramus. Both move posteriorly as the maxillary and mandibular arches develop posteriorly to complement each other.

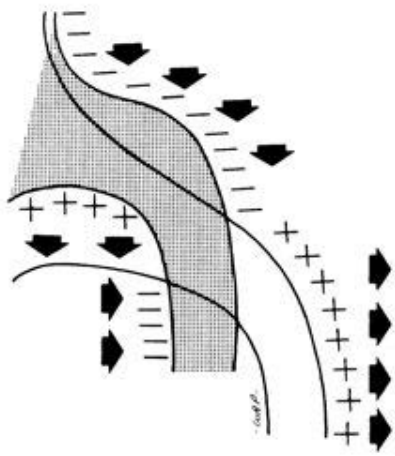
Some published implant-growth studies have not detected this posterior remodeling (relocation) movement of the malar region and anterior part of the zygoma. The reasons are twofold. First, implant insertion can be too close to the reversal lines between resorptive-to-depository remodeling fields (see Fig. 1-3), or too medial, and would thus not show the relocation movement because the remodeling extents here are not great enough to detect in serial headfilms. **Second, importantly, posterior relocation of the malar area slows and ceases after dental arch length is achieved during childhood development, and implant studies**

subsequent to this will not demonstrate the prior active posterior relocation of the malar protuberance, but which is no longer active. Histologic sections, however, clearly demonstrate the active resorptive nature previously present. This factor was not taken into account in previous implant studies. (See also Kurihara and Enlow, 1980a, in which resorptive surfaces are documented in histologic sections and the timetable involved.)

The inferior edge of the zygoma is heavily depository. The anterior part of the zygomatic arch and malar region thereby become greatly enlarged vertically as the face develops in depth.

The zygomatic arch moves **laterally** by resorption on the medial side within the temporal fossa and by deposition on the lateral side (Fig. 5-15). This enlarges the temporal fossa and keeps the cheekbone proportionately broad in relation to face and jaw size and the masticatory musculature. It also moves the arches bilaterally, thus increasing the space between for overall head and brain enlargement. The anterior rim of the temporal fossa moves posteriorly by the V principle.

FIGURE 5-15



As the **malar** region grows and becomes relocated posteriorly, the contiguous **nasal** region is enlarging in an opposite, anterior direction (Fig. 5-16). This draws out and greatly expands the contour between them, resulting in a progressively more protrusive-appearing nose and an anteroposteriorly much deeper face (see Fig. 5-17). This is a major topographic maturational change in the childhood-to-adult face. Note how the facial contours become opened, the protrusions more prominent, and the depths all increased.

The zygoma and cheekbone complex becomes **displaced** anteriorly and inferiorly in the same directions and amount as the primary displacement of the maxilla. The malar protuberance is a part of the maxillary bone and is carried with it. The separate zygomatic bone is displaced inferiorly in association with bone growth at the frontozygomatic suture and anteriorly in relation to growth at the zygotemporal suture. The growth changes of the malar process are similar to those of the mandibular coronoid process, its counterpart. Both remodel backward, along with the backward elongation of each whole bone, by anterior resorption and posterior deposition (Fig. 5-18). Both become displaced anteriorly and inferiorly along with each whole bone.

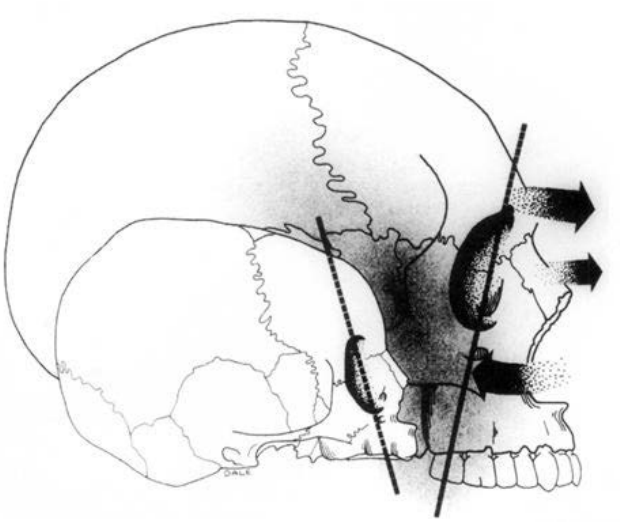


FIGURE 5-16.
(From Enlow. D. H., and S. Bang: Growth and remodeling of the human maxilla. Am. J. Orthod. 51:446. 1965, with permission.)

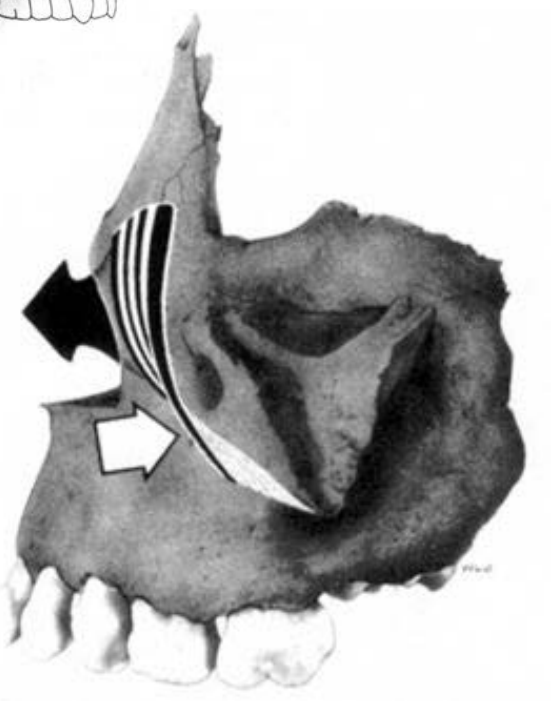
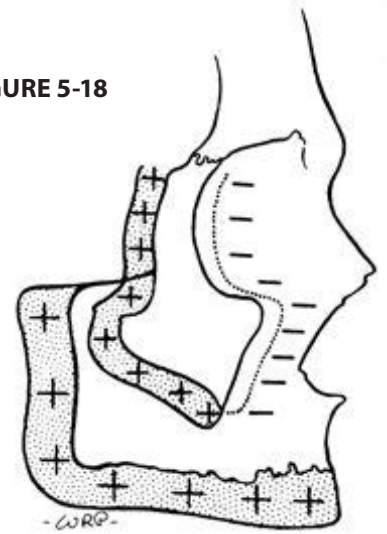


FIGURE 5-17.
(From Enlow, D. H., and J. Dale: *Oral Histology*, 4th Ed. by R. Ten Cate, St. Louis, C. V. Mosby. 1994, with permission.)

FIGURE 5-18



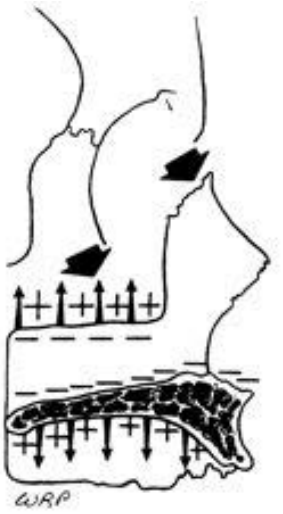
Orbital Growth

The remodeling changes of the **orbit** are complex. This is because many separate bones comprise its enclosing walls, including the maxilla, ethmoid, lacrimal, frontal, zygomatic, and the greater and lesser wings of the sphenoid. Many different rates, timing, directions, and amounts of (1) remodeling growth and (2) displacement occur among these multiple bony elements and their parts, thereby adding substantially to the developmental complexities.

The elaborate remodeling activities in the medial wall of the orbit, including the lacrimal and ethmoid bones, were mentioned above. In other parts of the orbit, most of the lining roof and the floor are depository. The orbital roof is also the ectocranial side of the floor of the anterior cranial fossa. As the frontal lobe of the cerebrum expands forward and downward (until about 5 to 7 years of age), the orbital roof remodels anteriorly and inferiorly by resorption on the endocranial side and deposition on the orbital side. It might seem that a depository type of orbital roof and orbital floor would decrease the size of the cavity. However, two changes come into play that actually increase it, although the amount is relatively small in the older child. First, the orbit grows by the V principle (Fig. 6-13). The cone-shaped orbital cavity **moves** (relocation by remodeling) in a direction toward its wide opening; deposits on the inside thus enlarge, rather than reduce, the volume. Second, the factor of enlarging **displacement** is directly involved. In association with sutural bone growth at the many sutures within and outside the orbit, the orbital floor is displaced and enlarges in a progressive downward and forward direction along with the rest of the nasomaxillary complex.

An interesting developmental situation is seen involving the nasal and orbital parts of the **same** bone (the maxilla). The floor of the nasal cavity in the adult is positioned **much lower** than the floor of the orbital cavity (Fig. 2-12). Compare this with the situation in the child, in which they are at about the same level. As described earlier, about half the process of palatal descent is produced by downward **displacement** of the whole maxilla accompanied by maxillary sutural growth. The greater part of the orbital floor is a component of the maxillary bone. Because both the orbital and nasal floors are regional portions of the **same** bone, the same displacement process that carries the palate downward **also** carries the floor of the orbit inferiorly at the same time. The extent of this downward palatal and nasal/oral displacement, however, greatly exceeds the much smaller amount required for orbital enlargement; that is, a lesser increase is needed for the much earlier growing eyeball and other orbital soft tissues than for the marked expansion carried out by the longer-growing nasal chambers. The floor of the orbit offsets this by **remodeling upward** as the whole maxilla **displaces inferiorly**. Deposition takes place on the intraorbital (superior) side of the orbital floor and resorption on the maxillary (inferior) sinus side (Fig. 5-19). This sustains the orbital floor in proper position with respect to the eyeball above it. The nasal floor, in contrast, approximately doubles the amount of displacement movement by **additional** downward cortical remodeling. Thus, the orbital and nasal floors are necessarily displaced in the same direction because they are parts of the same bone, but they undergo remodeling relocation movements in opposing directions.

FIGURE 5-19



The floor of the orbit also remodels laterally. It slopes in a lateral manner, and deposits on the surface of the floor thus relocate it in this same direction (as shown in Fig. 5-11). The lateral wall of the orbital **rim** remodels by resorption on the medial side and by deposition on the lateral side. This intraorbital field of resorption continues directly onto the anterolateral surface of the orbital roof beneath the overhanging supraorbital ridge. This is the only part of the orbital roof and lateral wall that is resorptive, and it provides for the lateral expansion of the domed roof. The cutaneous side of the supraorbital ridge is depository, and this combination causes the superior orbital rim to become protrusive. An upper orbital rim that extends forward beyond the lower rim is a characteristic of the adult face, particularly in the male because of the larger nose associated with larger lungs. (See Chapter 6 and refer to Figure 6-12 for an account of forehead and frontal sinus development in relation to nasal protrusive growth.)

The growing child's facial topographic profile undergoes a characteristic clockwise rotation (facing right). Several developmental relationships underline this maturational change. **The two-way combination of (1) forward remodeling of the nasal region and superior orbital rim together with (2) backward remodeling growth of the inferior orbital rim and the malar area, and (3) the essentially straight downward remodeling of the premaxillary region, all combine to produce a developmental rotation in the alignment of the whole of these middle and upper facial regions** (Fig. 5-17; see Figs. 5-16 and 5-18). Keep in mind that all parts, regardless of regional remodeling directions, become **displaced** in an anteroinferior direction (Fig. 3-16).

The lateral orbital rim undergoes remodeling growth in an obliquely posterior and a lateral direction at the same time. The lateral growth change increases the side-to-side dimension of each orbit and also contributes to the lateral movement of the whole orbit added to the small increase in the interorbital (nasal) dimension. The backward growth change of the lateral orbital rim keeps it in proper location with respect to the posterior direction of remodeling by the zygoma. **The forward remodeling of the superior orbital ridge and the whole**

anterior part of the nasal region, combined with the backward remodeling of the lateral rim and cheekbone, cause the orbital rim in the adult human face to slant obliquely forward, in contrast to all other mammalian faces. This reflects the forward remodeling rotation of the entire upper part of the human face and the backward rotation of the lower part. Additional discussion is provided in Chapter 9.

The resorptive nature of the cheekbone surface area, combined with the depository nature of the whole external nasal region of the maxilla, **greatly expands the surface contour between them and markedly deepens the topography of the face**. This changes the relatively flat early childhood face into the much bolder adult topography. The medial rim of the orbit is only slightly in front of the lateral rim in the young child. In the adult, the medial rim has grown forward with the anterior growing nasal wall, and the lateral rim has remodeled backward with the cheekbone. The medial and lateral rims are thus drawn apart in divergent posterior-anterior directions as the face deepens. Note the greatly increased depth of the contour of the lateral orbital rim and the midface as a whole resulting from these topographic changes.

Note this Feature of Facial Growth

In many of the growth and remodeling processes described throughout this chapter, one major difference exists between the female and the male. In the female, skeletal changes in the developing face slow markedly shortly after puberty. In the male, however, topographic and dimensional changes continue through the late adolescent period. The distinct facial similarities that exist between the sexes during earlier childhood, therefore, become substantially altered and divergent in the teenage years. This includes the preteenage composite of a more upright and bulbous forehead with lesser eyebrow ridges, the smaller and less protrusive nose, a lower nasal bridge, a more rounded nasal tip, flatter face, a wider appearing face with more prominent-appearing cheekbones, and a vertically shorter midface, all features of the prepubertal facial complex characterizing both sexes.

6

The Neurocranium

The housing for the brain impacts directly on many aspects of the developing facial complex (the latter known also as the viscerocranium or the splanchnocranium). The basicranium is involved in this fundamental and important relationship because the ectocranial side of the cranial floor is the interface with the face suspended beneath it. The perimeter, alignment, and configuration of the basicranium prescribes a "template" that establishes the growth fields within which both the mandible and nasomaxillary complex develop. In very simple terms, "the face is built on the base of the brain". However, the calvaria is largely removed from direct growth effects on the face.

The skull roof is described first, the basicranium second. There are basic growth differences between them and the developmental conditions relating to each.

The Calvaria

First, the proper singular spelling is calvar*ia*, not calvar*ium*, even though the latter seems to make sense given the related term "cranium," which is correct. The proper plurals are calvariae and crania, respectively. This common error is so pervasive, even by some anatomists, that one medical dictionary now even includes the incorrect form as a second spelling. Proper use, however, is a badge of scholarship.

The lining bony surface of the whole cranial **floor** is predominantly resorptive (darkly shaded, Fig. 6-1). This is in contrast to the endocranial surface of the **calvaria**, which is predominantly depository (lightly shaded; note the **circumcranial reversal line** indicated by the arrow). The reason for this major difference is that the inside (meningeal surface) of the skull roof is not compartmentalized into a series of confined pockets. The cranial floor, in contrast, has the **endocranial fossae** and other depressions, such as the sella turcica and the olfactory fossae. Why this calls for a difference in the mode of growth is explained below.

As the brain expands (*a* in Fig. 6-2), the separate bones of the calvaria are correspondingly displaced in outward directions (*b*). This is a passive movement on the part of the bones themselves in conjunction with the brain's expansion. Brain enlargement does not directly "push" the bones outward; rather, each separate bone is enmeshed within a connective tissue stroma attached to it. This stroma, in turn, is continuous with the meninges endocranially and the integument outside.

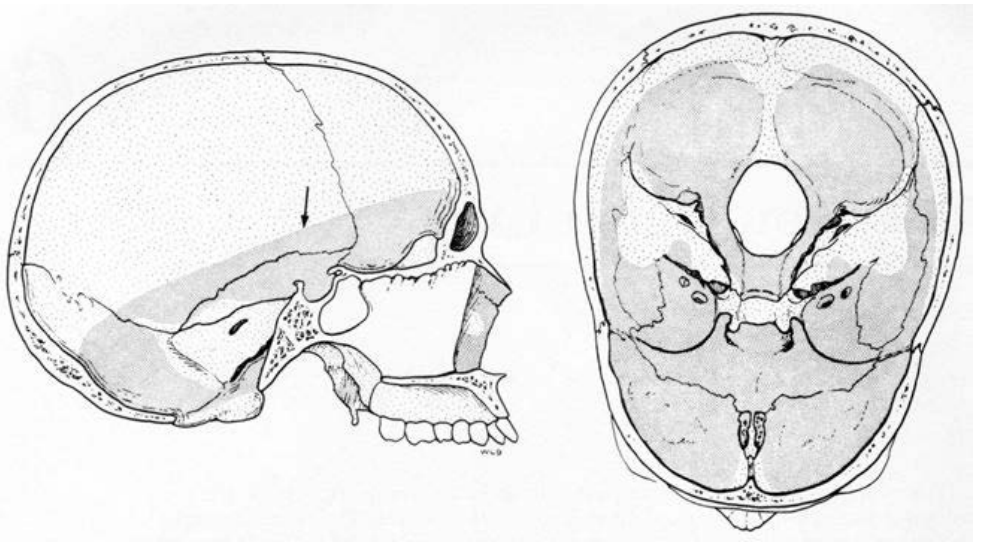


FIGURE 6-1.
(From Enlow, D. H.: *The Human Face*. New York, Harper & Row, 1968, p. 197, with permission.)

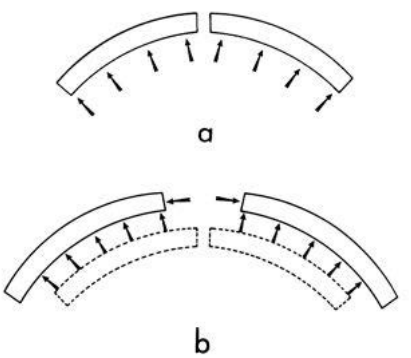


FIGURE 6-2

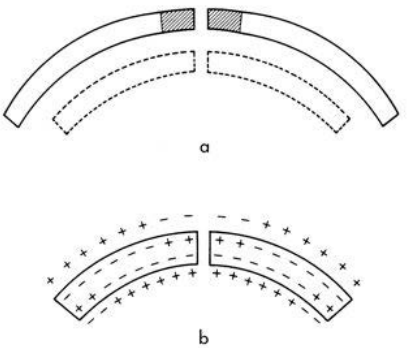


FIGURE 6-3

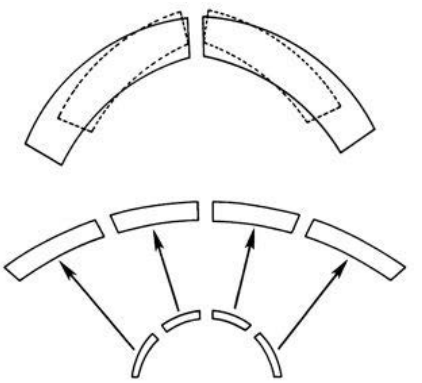


FIGURE 6-4

As these enclosing connective tissue membranes anchored to the bones enlarge with the growing brain, the bones are carried outward (displaced) by them, thereby "separating" all of the bones at their sutural articulations. In Figure 6-3, the primary displacement causes tension in the sutural membranes, which, according to present theory, respond immediately by depositing new bone on the sutural edges (*a*). Each separate bone (the frontal, parietal, and so forth) thereby enlarges in circumference. At the same time, the whole bone receives a small amount of new deposition on the flat surfaces of **both** the ectocranial and endocranial sides (*b*). The endosteal surfaces lining the inner and outer cortical tables are resorptive. This increases overall thickness and expands the medullary space between the inner and outer tables. The deposition of bone on the ectocranial surface, however, is **not** the growth change that causes the entire bone to move (displace) outward. Note that the endocranial surface, which is in contact with the dura that functions as a periosteum, is **not** a resorptive surface. This is an error in the older literature, still sometimes encountered.

The arc of curvature of the whole bone decreases, and the bone becomes flatter (Fig. 6-4). Although **remodeling** is not extensive in any of these "flat" bones because of their relatively simple contours, reversals can occur in areas mostly adjacent to the sutures. Here, either outside or inside surface resorption can take place (Fig. 6-3*b*), depending on the local nature of the changing contour.

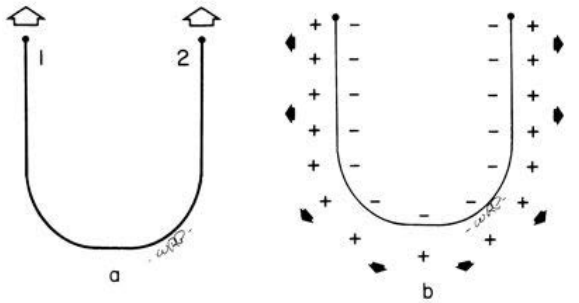
The Basicranium –The Foundation for the Face

It has often been presumed that the face is more or less independent of the basicranium, and that facial growth processes and the topographic features of the face are unrelated to the size, shape, and growth of the floor of the cranium. This is not the case at all. What happens in the floor of the cranium very much affects the structure, dimensions, angles, and placement of the various facial parts. The reason is that the cranial floor is the **template** from which the face develops. How differences in the architecture of the basicranium as a whole affect facial pattern is explained in other chapters.

The neural side of the cranial floor requires an entirely different mode of development compared to the calvaria because of its topographic complexity and the tight curvatures of its fossae. The endocranial surface of the basicranium, in contrast to the roof, is characteristically **resorptive** in most areas (see Fig. 6-1). The reason for this is that the alignments of the sutures do not have the capacity to provide for the multiple directions of enlargement and the complex magnitude of remodeling required. The relatively simple system of sutures inherited from our mammalian ancestors cannot fully accommodate the markedly deepened endocranial fossae of the massively enlarged human brain and basicranium. Additional, widespread remodeling of the cranial floor is necessarily involved. For example, Figure 6-5*a* schematically represents an enlarged human basicranial fossa with sutures located at *1* and *2*. These produce unidirectional sutural growth as indicated by the arrows. However, the two sutures present cannot produce the growth for the **other** directions also needed to accommodate brain expansion, as shown in Figure 6-5*b*. Fossa enlargement is accomplished by direct remodeling,

involving deposition on the outside with resorption from the inside. This is the key remodeling process that provides for the direct expansion of the various endocranial fossae in **conjunction** with sutural (and also synchondrosis) growth. Thus, the pattern of sutures present (as inherited from the flat basicranium of our early ancestors) cannot provide for this more elaborate remodeling design.

FIGURE 6-5



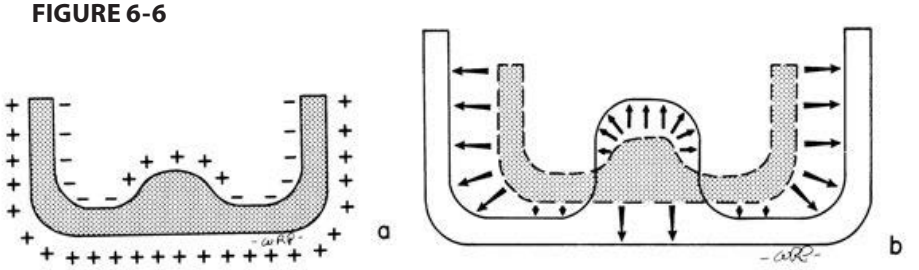
The various endocranial compartments are separated from one another by elevated bony partitions. The middle and posterior fossae are divided by the petrous elevation; the olfactory fossae are separated by the crista galli; the right and left middle cranial fossae are separated by the longitudinal midline sphenoidal elevation just below the sella turcica; and the right and left anterior and posterior cranial fossae are divided by a longitudinal midline bony ridge. All these elevated partitions, unlike most of the remainder of the cranial floor, are **depository** in nature (Figs. 6-1 and 6-6*a*). The developmental basis for the depository nature of these partitions is schematized in Figure 6-6*b*. The reason, simply, is that, as the fossae expand outward by resorption, the partitions between them must enlarge inward, in proportion, by deposition.*

The **midventral segment** of the cranial floor grows much more slowly than the floor of the laterally located fossae. This accommodates the slower development of the medulla, pons, hypothalamus, optic chiasma, and so forth, in contrast to the massive, rapid expansion of the hemispheres. Because the floor of the neurocranium enlarges by remodeling in addition to sutural and synchondrosis growth, these

* The activity of the bone lining the sella turcica, however, is quite variable and can be either depository or resorptive in different areas. Several reasons contribute to this, including the varying degrees of cranial base flexure and the variable amounts of downward and forward displacement of the midventral segments of the whole basicranium by the different shapes and proportionate sizes of the cerebral lobes. The sella turcica, however, must remain in contact with the hypophysis and also adjust to the variable size of the growing gland itself. If the pituitary fossa is carried downward by whole basicranial displacement disproportionately to the hypophysis itself, the floor of the sella will correspondingly rise by surface deposition to maintain contact with the pituitary, or the floor may be partly or entirely resorptive in other individuals to adjust to the balance between cranial base displacement and hypophyseal contact. A common combination is a resorptive posterior lining wall of the hypophyseal fossa and a depository surface on the sphenoidal part of the clivus. This causes a backward flare of the dorsum sellae to accommodate a pituitary gland that is being displaced to a lesser extent than the sphenoidal body below it. The jugum sphenoidal, like the floor of the sella turcica, shows variations for the same reasons. Its dorsal surface may be resorptive in some individuals, but depository in others.

differential extents and rates of expansion can be carried out. A markedly decreasing and tapering gradient of sutural growth occurs as the ventral midline is approached, but direct remodeling also occurs to provide for the varying extents of expansion required among the different midline parts themselves and between the midline parts and the much faster growing lateral regions (see Fig. 6-10*b*).

FIGURE 6-6



The following point is developmentally significant. Unlike the skull roof, the floor of the neurocranium provides for the passage of cranial nerves and the major cerebral blood vessels. Because the expansion of the hemispheres would cause marked displacement movements of the bones in the cranial floor if only sutural growth mechanism were operative (as in the skull roof), the process of **remodeling** growth in the basicranium provides for the changing stability of these nerve and vascular passageways. That is, they do not become disproportionately separated because of the massive expansion of the hemispheres of the brain, as would happen if the basicranium enlarged primarily at the sutures. The foramen enclosing each cranial nerve and major blood vessel also undergoes its own drift process (+ and -) to constantly maintain proper position. The foramen moves by deposition and resorption, keeping pace with the corresponding movement of the nerve or vessel it houses as the brain expands carrying the nerves with it. This relocation movement is differential in magnitude and direction related to the remodeling movements of the lateral walls of the fossa, thus requiring sensitive differences in respective regional remodeling.

The differential remodeling process maintains the proportionate placement of the spinal cord, even though the floor of the posterior cranial fossa, which rims the cord, expands to a considerably greater extent than the circumference of the foramen magnum (Fig. 6-7). Note the much larger growth increments of the hemispheres and the squama of the occipital bone, in contrast to the much smaller growth increments of the spinal cord and foramen magnum. Differential remodeling, not merely sutural growth, again provides for this. Recall, as emphasized in Chapter 1, that bone and soft tissue remodeling proceeds in whatever mode is required by the **regional** conditions that produce **local** developmental control signals in response to those architectonic circumstances.

The midline part of the basicranium is characterized by the presence of **synchondroses**. They are a retention left from the primary cartilages of the chondrocranium after the endochondral ossification centers appear during fetal development. A number of synchondroses are operative during the fetal and early postnatal periods. During the childhood period of development, however,

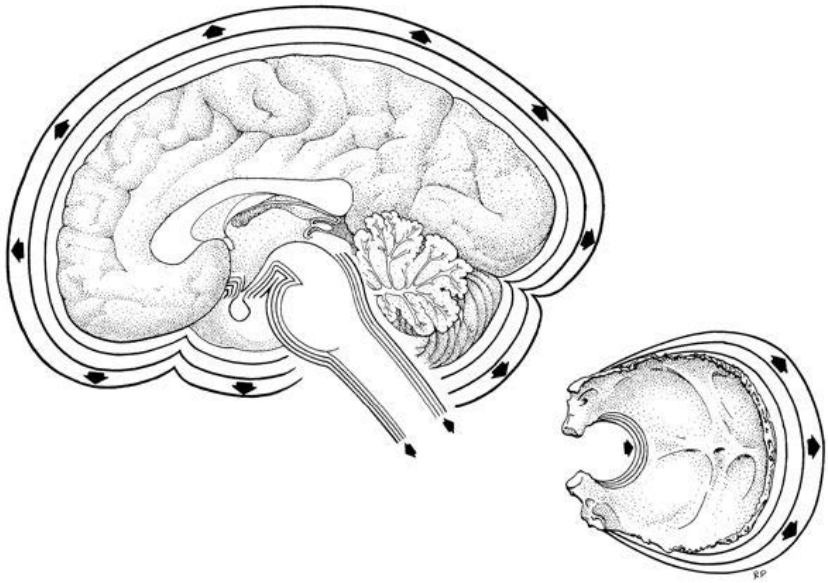


FIGURE 6-7.
(Modified from Enlow, D. H.: *The Human Face*. New York, Harper & Row, 1968, p. 202, with permission.)

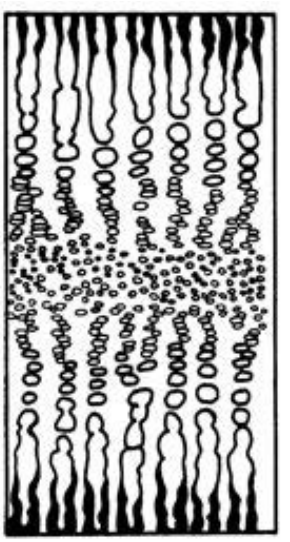
it is the spheno-occipital synchondrosis that is the principal "growth cartilage" of the basicranium. As with all growth cartilages associated directly with bone development, the spheno-occipital synchondrosis provides a pressure-adapted bone growth mechanism. This is in contrast to the tension-adapted sutural growth process of the calvaria, lateral neurocranial walls, and the endocranial fossae. Compression is involved in the cranial floor, unlike the calvaria, presumably because it supports the mass of the brain and the face, which bear on the fulcrum-like synchondrosis in the midline part of the cranial floor, and also presumably because it is more subject to cervical and masticatory muscle forces. Beyond these presumed pressure-adapted conditions, what other possible factors may be involved are not presently understood. The spheno-occipital synchondrosis is retained throughout the childhood growth period as long as the brain and basicranium continue to develop and expand. It ceases growth activity at about 12 to 15 years of age, and the sphenoid and occipital segments then begin to become fused in this midline area through about 20 years of age.

The presence of the spheno-occipital synchondrosis provides for the elongation of the **midline** portion of the cranial floor by its pressure-adapted mechanism of endochondral ossification. The floor of the cranium also has sutures in the lateral areas, but (1) the force of the compression produced by the growing neural mass is accommodated by the synchondrosis, not the sutures, and (2) the expansion of the laterally located **hemispheres** produces tension in these lateral sutural areas, unlike the more slowly growing midline part of the brain and basicranium not related directly to the hemispheres. Sutures are connective tissue membranes that provide tension-adapted sites of intramembranous bone growth, as described in Chapter 14.

Historically, the spheno-occipital synchondrosis has been regarded as **the** growth "center" and pacemaker that programs the development of the basicranium. This overly simplistic notion, however, as with the mandibular condyle, is a conceptual anachronism. The development of the basicranium is **quite** multifactorial and not merely the product of localized, midline cartilages that do not relate to the many regional growth circumstances throughout all parts of the basicranium as a whole. Only a very small percentage of the actual bone of the cranial floor is formed endochondrally in conjunction with the synchondroses, a parallel truism previously noted for the mandibular condyle.

The structure of the synchondrosis is similar to the basic plan for all "primary" types of growth cartilages, in contrast to the secondary variety of cartilage, which is basically different (see Chapter 14). As in the epiphyseal cartilage plate of long bones, the synchondrosis has a series of "zones," including the familiar reserve, cell division, hypertrophic, and calcified zones (Fig. 6-8). Similar to an epiphyseal plate, but unlike the condylar cartilage, the chondroblasts in the cell division zone are aligned in distinctive columns that point along the line of growth. Unlike the epiphyseal plate, the synchondrosis has **two** major (bipolar) directions of linear growth. Structurally, the synchondrosis is essentially two epiphyseal plates positioned back-to-back and separated by a common zone of reserve cartilage.

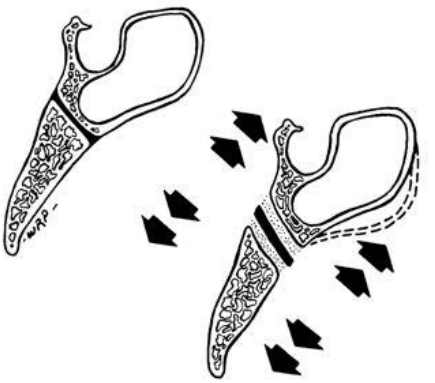
FIGURE 6-8



Endochondral bone growth by the spheno-occipital synchondrosis relates to primary **displacement** of the bones involved. The sphenoid and the occipital bones **are actively separated** by the primary displacement process (Fig. 6-9), and at the same time, new endochondral (medullary fine-cancellous) bone is laid down by the endosteum within each bone. Compact cortical (intramembranous) bone is formed around this core of endochondral bone tissue. Each whole bone (the sphenoid and the occipital) thereby becomes lengthened. Both bones also increase in girth by periosteal and endosteal remodeling. The interior of the sphenoid bone eventually becomes hollowed to form the sizable **sphenoidal sinus**. This sinus is just behind and in direct line with the bony nasal septum of the nasomaxillary

complex. As the midface becomes progressively displaced forward and downward, the sphenoidal body must remodel to retain contact with it. The sphenoidal sinus is thereby formed and progressively enlarges. Sphenoidal sinus expansion does not "push" the maxilla, however. This sinus secondarily "grows" as the body of the sphenoid bone expands around it to maintain contact with the moving nasomaxillary complex.

FIGURE 6-9



Two key questions exist with regard to the lengthening of the basicranium at a synchondrosis and the process of displacement that accompanies the elongation of each whole bone. First, do the synchondroses **cause** displacement by the process of growth expansion, or is their endochondral growth a **response** to displacement caused by other forces (such as brain expansion)? Second, does the cartilage have an intrinsic genetic program that actually regulates the rate, amount, and direction of growth by the cranial base? Or, is the cartilage dependent on some **other** pacemaking factors for growth control and secondarily responsive to them?

Traditionally, the cranial cartilages (and the whole basicranium in general) have been regarded as essentially autonomous growth units that develop in conjunction with the brain, but somehow independent of it. This explanation is incomplete. We know that disturbances in synchondrosal growth, such as those seen in achondroplasia, result in significant shortening of the cranial base while growth of the calvaria and mandible is largely unaffected. This observation strongly suggests that normal basicranial growth depends on genetically coded biologic processes occurring within the cartilage cells of the synchondrosis. In addition, experimental studies show that the synchondrosis has some independent proliferative capacity and as a growth cartilage is capable of generating tissue separating force via interstitial growth. However, the force levels generated in the synchodrosis are an order of magnitude smaller than those generated by the epiphyseal plates in long bones. Similarly, in cases of agenesis of the brain, growth of the basicranium is also affected. Therefore, it seems likely that the shape, size, and characteristics of the cranial floor have evolved in direct phylogenetic association with the brain it supports i.e., a "phylogenetic type" of functional matrix. This suggests that although there is some intrinsic growth capacity in the basicranium (not calvaria), extrinsic control factors are also required. In contrast, the calvaria is largely dependent on its surrounding endocranial and ectocranial matrix for growth control.

As previously pointed out, the contribution of the synchondrosis relates to the midventral axis of the cranium and not the **entire** cranial floor. Note that the overall enlargement of the midline part of the basicranium is much less than the marked expansion of the more laterally located middle (and posterior) cranial fossae. This is because the lateral fossae house the various lobes of the huge hemispheres, which enlarge considerably more than the closer-to-midline medulla, pituitary gland, diencephalon, hypothalamus, optic chiasma, and so forth. Endocranial resorption occurs on the endocranial surface of the clivus and, laterally, the sizeable floor of the middle cranial fossa. For the clivus, this produces an oblique anteroinferior remodeling movement **in addition to** the linear growth by the synchondrosis.† For the middle and also posterior cranial fossae, it produces massive expansion in conjunction with the sutural growth also taking place. The clivus also lengthens by bone deposition on the ectocranial side of the occipital bone at the lip of the foramen magnum.

The expansion of the middle cranial fossa and its neural contents have a major **secondary** displacement effect on the anterior cranial floor, the underlying nasomaxillary complex suspended from the latter, and the mandible. Because the posterior boundary of the maxillary complex is developmentally positioned to exactly coincide with the boundary between the anterior and middle cranial fossae, forward displacement of both the anterior cranial fossa and the nasomaxillary complex suspended beneath it occurs harmoniously. The amount of horizontal displacement for the mandible, however, is much less because most of the enlargement of the middle cranial fossa takes place **anterior** to the mandibular condyles. This basic developmental fact has important clinical significance. The nasomaxillary complex is naturally in balance with the displacement of the anterior cranial base, the mandible does not share this inherent developmental advantage. Being more independent in development, means that the potential for mandibular imbalance during growth (e.g mandibular micrognathia or prognathia) is much greater.

The enlarging middle cranial fossa does not in itself **push** the mandible, anterior cranial fossa, and maxillary complex forward. Visualize the enlarging temporal and frontal lobes of the cerebrum as two expanding rubber balloons in contact. They are each displaced **away** from the other, although the net effect is a forward direction of movement from the foramen magnum. The temporal and frontal "balloons" have fibrous attachments to the middle and anterior cranial fossae, respectively. As **both** balloons expand, these two fossae are thus **pulled away** from each other, but both also being moved together in a protrusive direction. This sets up tension fields in the various frontal, temporal, sphenoidal, and ethmoidal sutures, and this presumably triggers sutural bone responses (in addition to direct basicranial remodeling expansion by resorption and deposition all over all other inside and outside surfaces). Both fossae are thus enlarged, and the nasomaxillary complex is carried along anteriorly with the floor of the anterior cranial fossa from which it is suspended. At about 5 or 6 years of age, frontal lobe growth and anterior

† The dorsum sellae, however, shows much variation in shape and size. In some individuals it flares markedly in an upward and backward direction, and the sphenoidal part of the clivus may be correspondingly depository, rather than resorptive.

cranial fossa expansion are largely complete. Thus, any further developmental protrusion of the forehead is a result of thickening of the frontal bone and enlargement of the frontal sinus within it (Fig. 6-12). The temporal lobe and middle fossa, however, continue to enlarge for several more years, and ongoing expansion of each temporal lobe continues to displace the frontal lobe forward, and this, in turn, causes tension in the osteogenic suture systems between these two areas. The anterior fossae and the maxillary complex are carried anteriorly by the frontal lobes, which is moved forward because of temporal lobe enlargement behind it. This "tension" trigger in response to **brain** and other soft tissue enlargements is a theoretical explanation but is consistent with the underlying biology of soft and hard tissue growth.

As schematized in Figure 6-10*a*, the composite picture shows that resorption occurs from the lining side of the forward wall of the middle cranial fossa (*1*), deposition on the orbital face of the sphenoid and in the sphenofrontal suture (*2*), and forward displacement of the anterior cranial fossae as the frontal lobes are displaced anteriorly (*3*). The petrous elevation (*4*) increases by deposition on the endocranial surface, and lengthening of the clivus occurs by growth at the spheno-occipital synchondrosis (*5*). The foramen magnum is progressively lowered by resorption on the endocranial surface and deposition on the ectocranial side. This also contributes to the lengthening of the clivus (*6*), and the perimeter of the foramen enlarges to match myelination and further enlargement of the spinal cord. Inferior to the circumcranial reversal line (see Fig. 6-1), the endocranial fossae enlarge by a combination of endocranial resorption and ectocranial deposition (*7*) that occurs in addition to growth at the basicranial sutures.

In Figure 6-10*b*, a decreasing gradient of sutural growth occurs approaching the midventral part of the basicranium is schematized (lightly shaded areas, *1*). The endocranial fossae enlarge by a corresponding gradient of direct cortical remodeling, as shown by the darkly shaded areas (*2*). The clivus lengthens by endochondral bone growth at the spheno-occipital synchondrosis (*3*) and also by direct downward remodeling of the basicranial floor around the rim of the foramen magnum. The sphenoid and occipital complex remodels and rotates anteriorly and inferiorly by endocranial resorption (*0*) and ectocranial deposition.

The vertical enlargement of the middle cranial fossae has a major effect on the respective vertical placements of both the mandibular and maxillary arches. The effect is a progressive separation of the arches.

Each anterior cranial fossa enlarges in conjunction with the expansion of the frontal lobes. Wherever sutures are present, they contribute to the increases in the circumference of the bones involved. Thus, the sphenofrontal, frontotemporal, sphenoethmoidal, frontoethmoidal, and frontozygomatic sutures all participate in a closely coordinated, traction-adapted bone growth response to brain and other soft tissue enlargements. The bones all become **displaced** "away" from each other as a consequence. This is a primary type of displacement, because the enlargement of each bone is involved. Together with this, the bones also enlarge outward by ectocranial deposition and endocranial resorption, as described below. The aggregate of all these processes produces the composite growth changes seen in Figure 6-10*b*.

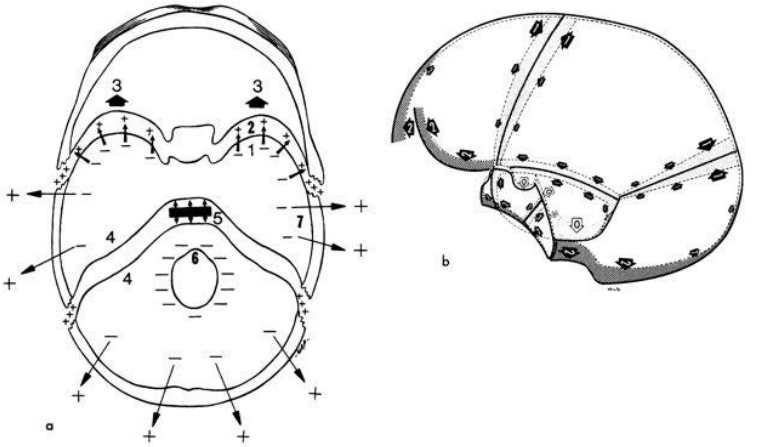
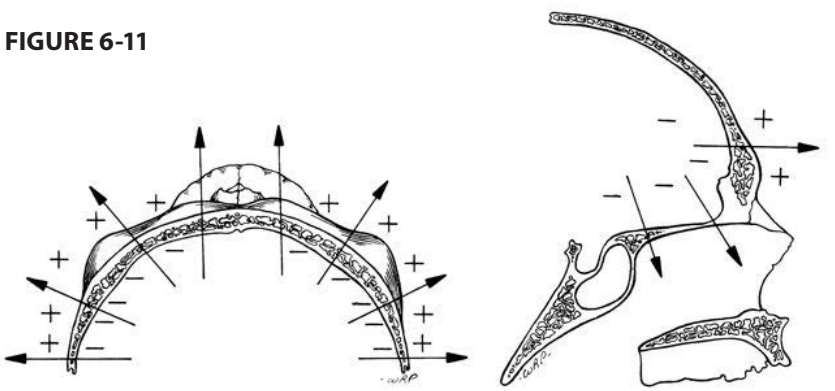


FIGURE 6-10.
(b) From Enlow, D. H.: *The Human Face*. New York, Harper & Row, 1968, with permission.

As previously pointed out, sutural growth alone cannot accomplish the extent of cranial fossa expansion required. In addition to bone additions at the various sutures, direct cortical remodeling also takes place extensively (Fig. 6-11). About midway up the forehead a reversal line encircles the inner side of the skull and separates the resorptive endocranial remodeling fields of the basicranium from the separate depository field of the roof (see arrow in Fig. 6-1).

As long as the frontal lobes of the cerebrum enlarge, the **inner** table of the forehead correspondingly remodels anteriorly (Fig. 6-11). When frontal lobe enlargement slows and largely ceases sometime before about the sixth year, the growth of the inner table stops with it. The outer table, however, continues to remodel anteriorly (Fig. 6-12). This progressively separates the two tables, and an enlarging frontal sinus develops by resorptive replacement of the cancellous medullary bone (diploë). The size of the sinus, however, and the amount of forehead slope vary considerably according to age, sex, and headform characteristics (see Chapter 8). The reason the frontal sinus develops is that the upper part of the nasomaxillary complex continues to remodel protrusively, and the outer table of the contiguous forehead necessarily must remodel with it.

FIGURE 6-11



Note that the floor of the anterior cranial fossa is also the roof of the underlying orbital cavity (Fig. 6-13). The endocranial side is resorptive, and the orbital side of this very thin bony plate is depository; it relocates by remodeling progressively downward and outward. While this serves to enlarge the bottom part of the cranial fossa, does it also then reduce the size of the orbital cavity? The answer is no, for two reasons. First, the orbits relocate anteriorly by the V principle, which itself serves to enlarge, not reduce, orbital size (Fig. 6-13). Second, the multiple parts of the whole orbit are also becoming **displaced** out and away from each other at the same time in association with bone deposition at the various orbital sutures, as described in the chapter dealing with the maxilla.

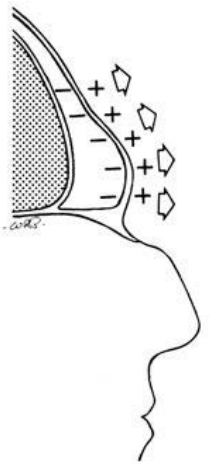


FIGURE 6-12

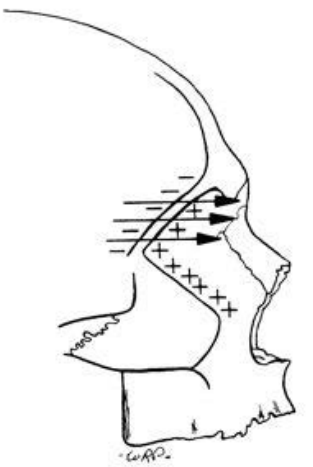


FIGURE 6-13

7

The Role of the Dentition in Facial Growth

As previously highlighted in Chapter 1, "tooth movement" has functions beyond just placing the dentition into occlusion. It is a key part of facial growth and is a major biologic process that can be therapeutically modified. Tooth movement (1) positions a tooth into changing functional locations, and (2) sustains progressively changing anatomic relationships as the entire craniofacial assembly around it continues to undergo massive development. Additionally, (3) the periodontal membrane (PDM) serves as a pressure-to-traction converting buffer to masticatory forces. These basic growth functions require an elaborate and intricate biologic system of closely orchestrated histologic actions involving multiple "genic" tissues. An intrinsic control process selectively activates and coordinates the complex histogenic interplay.

Therepeutic interventions designed to "work with growth" introduce extrinsic control signals to augment, modify, or replace the intrinsic, regionally distributed intercellular messengers already ongoing. One objective of craniofacial clinicians is to manipulate (1) the directions, and (2) the magnitude of the underlying biologic actions sensitive to these signals. Importantly, the biologic processes being modified are the same as those occurring during the normal growth process; it is the signal input to the genic tissues that is altered by clinical procedures. The result is to selectively accelerate or inhibit **regional** cellular responses, and to alter directions of movement. The clinician, thus, is engaged in biologic engineering and must design input signals (e.g. class II elastics, headgear, temporary anchorage devices) to elicit the desired biologic outcomes. Indeed, the manipulation of the growing face requires constant monitoring of signal inputs and biologic responses to insure optimum results. It is imperative that clinicians engaged in this activity have an in depth understanding of the underlying biology involved. Since tooth movements (eruption and drift) account for about one third of post natal facial growth, this chapter is directly applicable to clinical care.

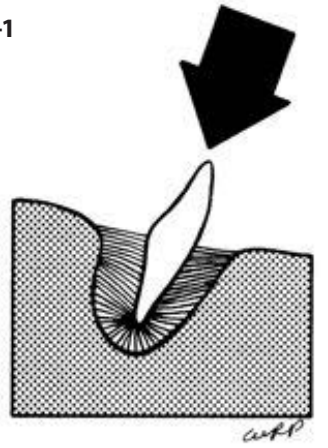
The **periodontal membrane*** is an osteogenic connective tissue comparable to the periosteal membrane and, because it is a "back-to-back," double-sided

* Also commonly called the periodontal ligament. It is indeed a mature ligament in terms of its histologic structure in the more stable, adult form. However, the term "membrane" is much more appropriate for the childhood growth period. The periodontium has a connective tissue membrane that is quite active and dynamic, not one that merely physically supports a tooth (i.e., a ligament). It

histogenic membrane, it is also comparable to a sutural histogenic membrane. Phylogenetically, it is the adaptive answer to a basic functional problem. A vascular membrane, such as the periosteum, is known to be quite pressure sensitive, and resorptive necrosis results when a surface compressive force acts to close off its vascular supply (see page 274). Pressures produced by chewing teeth would seem to cause such destructive compression on a jaw bone's osteogenic membranes, and a neutralizing factor is thereby needed. Would cartilage, a tissue specially resistant to pressure, function satisfactorily as a neutralizing buffer between the tooth root and the bony alveolar surface? No, because cartilage is severely limited in its capacity for remodeling and could not accommodate the dynamic changes required for tooth development, eruption, and the drift needed as an integral part of the facial growth process.

The phylogenetic problem of pressure on the bone surface beneath a tooth has been overcome in a simple but effective way (Fig. 7-1). Pressure is converted directly into **tension** (to which fibrous membranes are adapted and can handle) by the suspension of each tooth in a connective tissue **sling** of fibers within a socket.† By this means, the compressive force by a tooth being pushed into its socket is translated, not as pressure, but as direct tension on the alveolar bone. Thus, the sensitive, vascular periodontal membrane is not exposed to the killing effects of compression as the tooth is depressed into the socket or as it is tipped or rotated in

FIGURE 7-1



(1) contributes to the growth and development of the tooth; (2) is involved directly in the eruption of the tooth; (3) is involved directly in the drifting, tipping, and rotation movement of the tooth; (4) provides for the formation of the bone tissue lining the alveolar socket; (5) is an active and essential sensory receptor and vascular pathway; and (6) is involved directly in the extensive remodeling of the bone associated with the movements of the teeth. For these reasons, the term periodontal "membrane" is more closely associated with the truly dynamic functions of this connective tissue layer. "Ligament," on the other hand, connotes a more stable, inactive, nonchanging type of tissue that has a single function—fibrous attachment. Alveolar bone, of course, is of intra-membranous origin, being produced by the periodontal membrane.

† A violation of this biologic relationship is the basis for many of the problems encountered by the prosthodontist. Tissue supported dentures are pressure-causing appliances fitted onto bone without a tension-converting sling of periodontal fibers. Uncontrolled resorption can be a consequence. Implant supported dentures eliminate compressive forces on the alveolus.

one direction or another by masticatory forces. This relatively simple plan accomplishes several needed functions. It provides effective mechanical support for the tooth, gives resilient yet nonbrittle stability, provides a biologic system (connective tissue remodeling) for eruption, enables each individual tooth to acquire a functional occlusal position, provides for the growth and remodeling maintenance of the alveolar bone, provides a vascular and nerve supply as well as a pool of undifferentiated cells that are needed for continued development, and provides for the vertical and horizontal drifting of the tooth and the accompanying remodeling movements of the alveolar bone. These are all major requirements essential to the biology of tooth movement. All of these functions performed by the PDM are lacking in the rigid osseo-integrated implants being used to replace teeth prosthetically and from the interface between bone and temporary anchorage devices (TADs). The impact of this important difference between implants/TADs and natural tooth-membrane physiology is sometimes not fully appreciated by the dental profession.

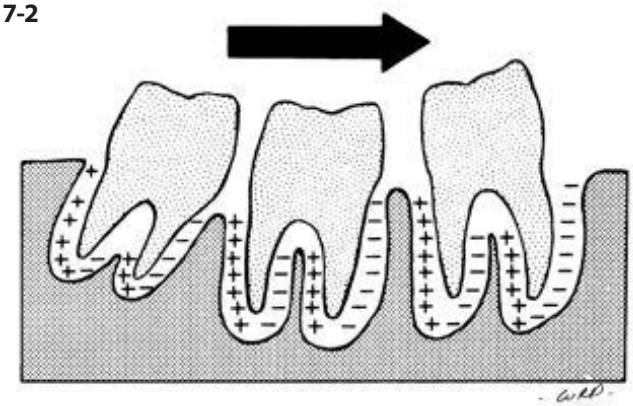
Teeth are unique structures in the human body. For example, teeth are the only structures that attain their adult size at formation (i.e. the crowns of the teeth erupt fully formed). And although there is some ionic exchange with saliva, the uptake of fluoride is one example, the part of the tooth that is in the oral cavity is remarkably stable and inert. Dental enamel is static when compared to all other tissues in the body. The static nature of teeth often misleads dentists into thinking that other components of the human body are similar. The fact of the matter is that teeth are different. Another unique feature of the dentition is that teeth exist in both a septic and aseptic environment. The interface between these environments consists of a complex histologic structure that both resists bacterial invasion yet allows physiologic movement of individual dental units within bone. An intact attachment apparatus is necessary for normal biologic tooth movements to occur and allows tooth movement to be an osteogenic process. Breakdown of the attachment apparatus results in a septic environment in which tooth movement is no longer osteogenic but is now osteolytic. A complete description of the this complex structure is not relevant to this book, however, all processes described herein assume an intact periodontal attachment apparatus with a clear demarcation of the septic and aseptic environments.

Teeth drift for two basic, functional reasons. One, as described in all basic oral histology texts, is to close-up the dental arch during growth and keep it closed as the contact edges along interproximal contacts of the teeth progressively wear. Tight contact points allow the dental arch to better withstand masticatory forces. **The second reason, much less known but of great importance, is to anatomically place and progressively relocate the teeth as the whole mandible and maxilla grow and remodel.** Each tooth (and the unerupted tooth buds as well) must drift vertically, laterally, and either mesially or distally in order to sustain proper but changing anatomic position. The "molar" region at an early age level, for example, becomes the "premolar" region of the jaw at a later age as the corpus lengthens posteriorly. The maxillary teeth, another example, must drift (not merely "erupt") inferiorly for a considerable distance as the whole bony maxillary arch relocates downward to provide (1) enlargement of the overlying nasal chambers, and (2)

adjustments for palatal displacement rotations to "level" the occlusal plane. Anterior versus posterior differences in the extents of this vertical dental drift, in conjunction with palatal remodeling, function to achieve constant and ongoing proper palate and dental arch alignment (see pages 213 and 214). Thus, the function of horizontal **and** vertical **drift** is far more significant than merely "closing up" the dentition. It is one of the basic developmental factors involved in facial growth. As pointed out in Chapter 1, importantly, it is the "growth" a clinician "works with" in tooth movements.

The customary diagram used to illustrate "mesial drift" shows deposition and resorption on the "tension" (depository) and "pressure" (resorptive) sides of the alveolar socket, respectively (Fig. 7-2). A posteroanterior section through the jaw showing several tooth roots gives the familiar histologic picture schematized here. However, only the **mesial** direction of drift movement is pointed out in most standard textbooks; the important **vertical** drift movements that **also** take place are never explained. This other movement is carried out by the **same** alveolar bone deposition and resorption usually associated only with mesial drift. Drift is a vertical as well as horizontal growth process, and the diagrammatic picture illustrated here depicts only mesial drift (see page 101). With the advent of temporary anchorage devices (TADs) an understanding of the vertical component of drift becomes exceedingly important. Before TADs, clinician's were able to modify vertical drift toward the occlusal plane and increasing drift toward the occlusal plane was a significant factor in the correction of deep bite malocclusion (See Hans et. al 2007). However, an alveolar socket can move by remodeling in virtually **any** direction, when accompanying its resident tooth's movement, by appropriate patterns of its periodontal remodeling fields. TADs allow clinicians to modify vertical drift in all directions providing a powerful, biologically based, clinical tool.

FIGURE 7-2



The classic "pressure and tension" model for mesial drift depicts resorption on the mesial and deposition on the distal side of the alveolar socket. This model is an oversimplification, and masks the actual biology involved. The collagenous fibers of the periodontium on the pressure side are actually under tension during normal physiologic tooth movements such as drift (see Fig. 7-8). On the "tension" side,

furthermore, the cells of the periodontal membrane are actually under compression between the taut collagenous fibers. In contrast to ordinary physiologic (growth) conditions, tooth-to-periodontal membrane-to-alveolar bone compression can be involved in some **clinical orthodontic** tooth movements.

It has been a major point of controversy for many years whether the pressure presumed to trigger alveolar bone resorption acts first on the connective tissue membrane or directly on the bone, which, in turn, causes the membrane to respond (see below). One concept is that very minute **distortions** of alveolar bone caused by shifting of the tooth's root, or through other forces created by growth, serve to trigger alveolar remodeling. The **piezo effect** is held by many investigators to be the response to this stress trigger, and it is widely believed that this bioelectric stimulus serves as a "first messenger" that fires receptor sites on osteoblastic and osteoclastic cell surfaces within the periodontium. Some investigators have suggested that the viscous intercellular fluid matrix functions as the biomechanical intermediary. It presumably acts as a "hydraulic system" that can transmit variable amounts of pressure to the alveolar bone surface by vessel or matrix distention or compression.

If the extent of pressure exerted by a tooth on the periodontal membrane produced, for instance by heavy orthodontic forces, results in a severe compression of the membrane, a closing-off of the blood vessels and cellular necrosis follows. The growth capacity of the membrane is destroyed, and remodeling changes on the alveolar bone surface are precluded. This is presumed to trigger **undermining resorption.** In this process, the resorptive changes then proceed from the endosteal cancellous spaces **deep** to the alveolar bone surface, since the hyalinized connective tissue on the alveolar surface itself is histogenically inert owing to vascular occlusion.

As pointed out above, the periodontal membrane is an equivalent of both the periosteum and sutures. Its general structure is similar, and its own internal mode of growth is comparable. The notable difference, of course, is that one side attaches to a tooth, rather than to a muscle or another bone. The periodontal membrane is a reflection of the overlying periosteum into the alveolar socket, and these two histogenic, vascular membranes are directly continuous.

In its "stable," nonremodeling and histogenically inactive form, the periodontal membrane is then essentially a mature ligament composed of dense bundles of thick collagenous fibers with correspondingly few fibro-blasts and little ground substance. During the active period of facial growth, dental development, and the establishment of occlusion, however, this membrane has a much more dynamic function, and its histologic structure is adapted to the complex developmental role in facial growth that it plays. During the growth period, the periodontal membrane is much more highly cellular, and much more than just ligament attachment fibers and a scattering of pyknotic fibroblasts are present. As comprising the histogenically active periosteal and sutural connective tissues, the periodontal membrane has three basic layers. The middle layer, called the "intermediate plexus," is composed of the same slender, precollagenous **linkage fibrils** that are present in the intermediate layers of the osteogenic periosteum and sutures. Linkage fibrils (layer *B* in Fig. 7-3) provide connections and sequential

reconnections between the innermost and outermost dense, coarse fibrous layers (layers *A* and *C*). Their key function is for **adjustments** involved in tooth drift, eruption, rotations, and alveolar bone movements produced by remodeling. This layer may be poorly differentiated or absent in nonremodeling periods and in regional locations (and species) where tooth movements are relatively slow. Or, the distribution of the linkage fibrils may be more diffuse rather than forming a separate recognizable zone. During active tooth movements, nonetheless, they are necessarily **always** present, as histochemical tests clearly show, whether or not a separate, discrete zone is apparent (Kraw and Enlow, 1967). During tooth movement and companion alveolar remodeling relocations, the PDM is not simply "moved," *in toto*, to progressively new positions. Rather, it undergoes **its own remodeling**, just as the bone does, to provide the movement, and this requires considerable and on-going re-linkages of the connecting fibers.

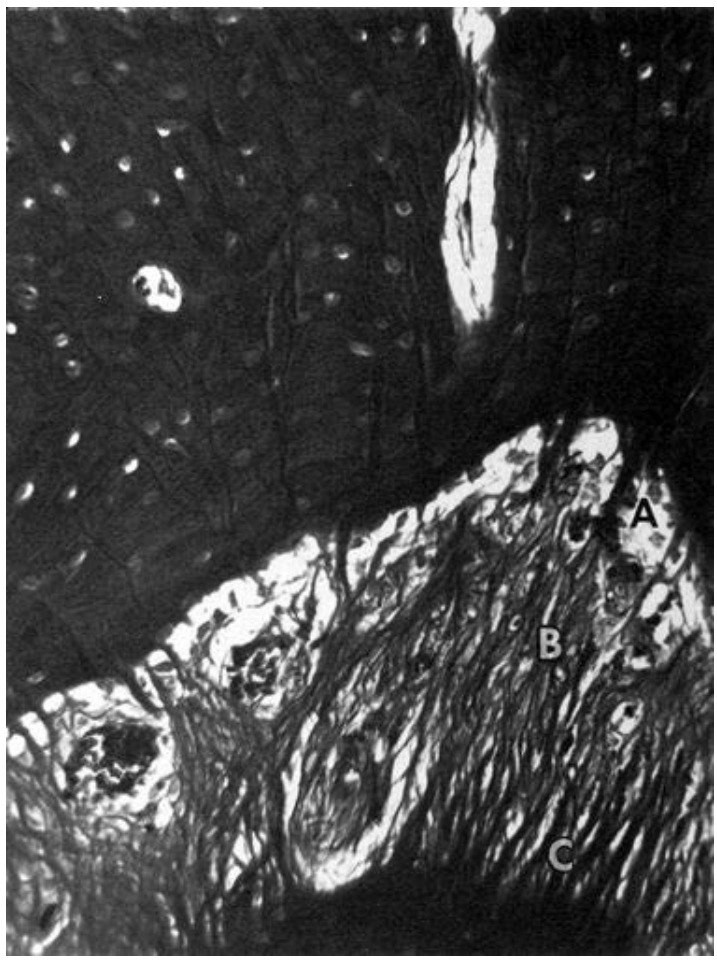
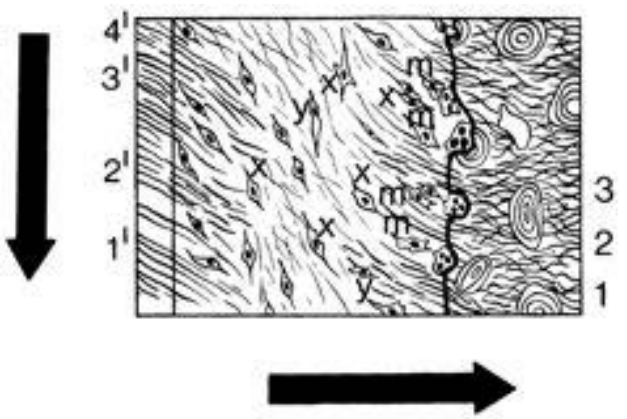


FIGURE 7-3.
(From Kraw, A. G., and D. H. Enlow: Continuous attachment of the periodontal membrane. Am. J. Anat., 120:133, 1967, with permission.)

It has been proposed that the actual source of the propulsive mechanical force that brings about eruption, vertical and horizontal drift, and other tooth movements is provided specifically by an abundant population of actively contractile fibroblasts ("myofibroblasts") on the **resorptive** sides of the socket (Azuma et al., 1975). The contraction of these special cells (*m* in Fig. 7-4) is believed to **pull** the collagenous framework within the periodontal membrane, and thereby the tooth, in the direction of the resorptive bone front. The contractile cells also presumably transport fibers into new linkage positions. These contractile fibroblasts are all interattached by desmosomes, and interconnected physiologically by a nexus between cells. The cells are anchored to fibers by hemidesmosomes. Simultaneously, special collagen-degrading and collagen-producing cells (*x* and *y* in Fig. 7-4) within the linkage zone provide the fiber remodeling and relinkages described below. This occurs in conjunction with ground substance degradation and synthesis, and the tooth is thus propelled in horizontal and vertical drift movements (arrows). Multinuclear osteoclasts resorb the bone in advance of tooth's movement. The same process also appears to provide for eruption and rotatory movements. The fibers at level *1*, formerly linked with 1^1, thus become relinked with fiber level 2^1, and so on, in the inferior direction of maxillary dental eruption. It is suggested, importantly, that these various cells are the specific targets of the clinical forces utilized by the orthodontist to move teeth. Some histogenic process such as this, or similar variation to it, must necessarily be operative.

FIGURE 7-4



As seen in Figures 7-5 to 7-7, on one side of the zone of linkage fibrils (*b*) is a layer of coarse collagenous fibers that attach to the alveolar bone (*a*), and on the other side a layer of coarse fibers attaching to the cementum of the tooth (*c*). The activity on the "tension side" is schematized in Figure 7-6. This old term is used because the pull of the tooth to the right presumably sets up tension on the bone surface by the periodontal fibers, and tension was presumed to be osteoblastic activating (see below). A new layer of bone is deposited on the alveolar surface. This embeds the periodontal fibers of layer *a* (Fig. 7-6). Note that the attachment fibers are not driven into the bone as with a nail; they become progressively enclosed and

buried as new bone deposits form around them. It is apparent that the fibers of zone *a* would soon be used up and become completely enclosed. However, the **linkage** fibrils of the intermediate zone *b* (or its histologic equivalent) become remodeled into *a*, thereby lengthening *a* in advance of the drifting alveolar wall. The fibers of layer *a* are thus enclosed by new bone on one side while being lengthened by an equal amount on the other. The conversion from *b* to *a* is accomplished by a bundling together of the thin, precollagenous linkage fibrils into the thick, "mature" fibers of layer *a*. Ground substance is believed to be the binding agent, and the process is carried out by the abundant resident population of periodontal fibroblasts. Layer *b* retains its breadth by elongation of the precollagenous linkage fibrils. It is not presently known whether this lengthening process occurs within zone *b* or at the interface between *b* and *c*. New unit fibrils are also constantly added as the tooth grows and as the membrane drifts in conjunction with tooth drift. The fibers of layer *c* are carried in the direction of the tooth's movement. Throughout this membrane remodeling process, continuous attachment between tooth and alveolar bone is thereby maintained. Note that the periodontal membrane as a whole is not simply pushed or pulled along as the tooth moves. It **grows** from one location to the next.

FIGURE 7-5

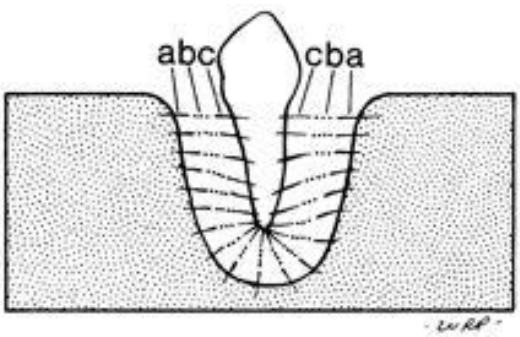


FIGURE 7-6

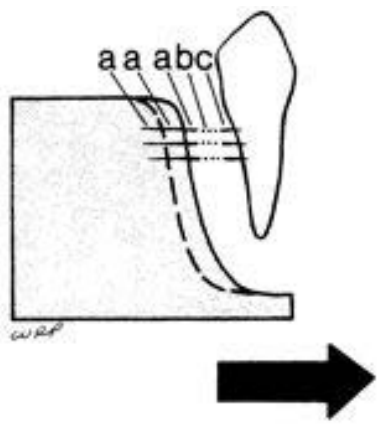


FIGURE 7-7

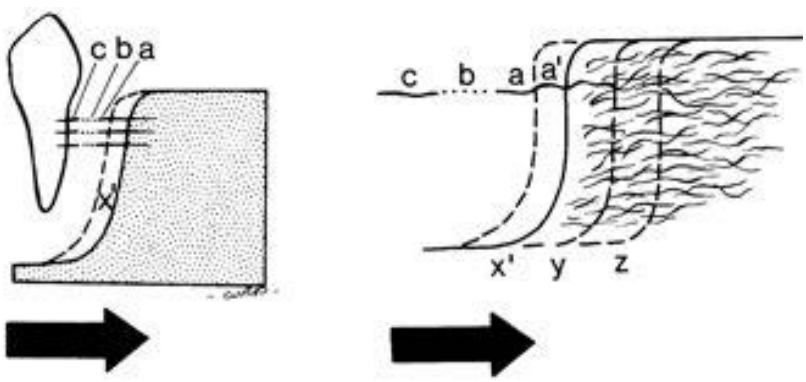


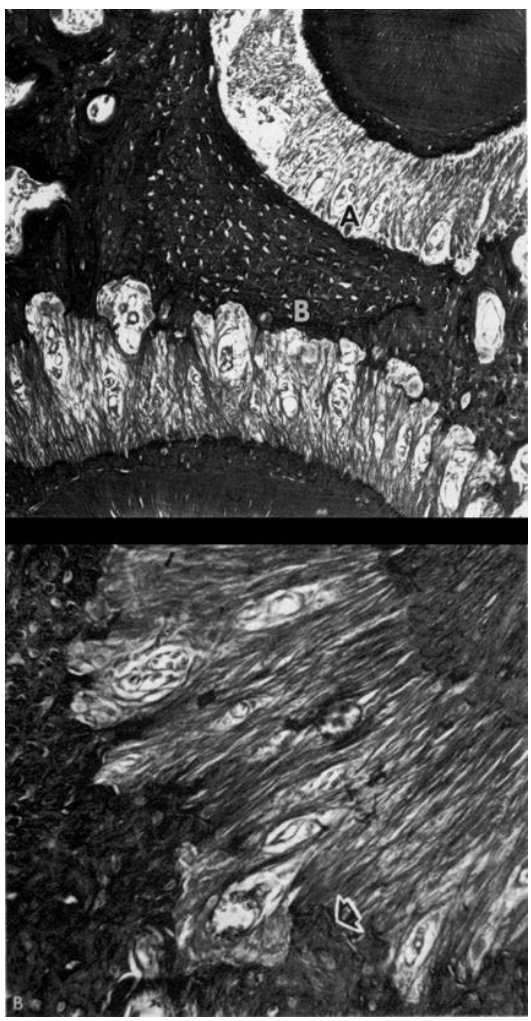
Figure 7-7 shows the activity on the "pressure side" of the tooth root ("pressure," as mentioned above, because the tooth root, according to longstanding but inadequate theory, has been presumed to exert direct compression on the periodontal membrane and bony alveolar wall). It is the reverse of the remodeling sequence on the opposite ("tension") side. A layer of bone is **resorbed** (x^1) from the alveolar surface by an abundant sheet of osteoclasts. The resorptive side of the alveolar socket can easily be distinguished microscopically from the depository side by the characteristic chiseled, pitted, eroded appearance of the bone's surface. If the resorptive process was active at the time, numerous osteoclasts are seen in the erosion pits (Howship lacunae).

An effective means for periodontal attachment onto resorptive alveolar bone surfaces is operative and involves an adhesive mode of new fiber attachment onto the resorptive bone surface. (Kurihara and Enlow, 1980b and 1980c). As the alveolar resorptive front (Fig. 7-7) proceeds from x^1 to y and on to z, the linkage fibrils (*b*), the attachment fibers on the bone side (*a* and a^1), and those on the tooth side (*c*) **remodel** to sustain their proportionate lengths, with new fibers and relinkages sustaining continuous attachments as the tooth moves. The whole membrane thus grows in a direction toward the bone surface and away from the tooth as the tooth simultaneously moves in a like direction. The process is continuously repetitive. Even though the bone on the resorptive side of the alveolar socket undergoes progressive removal, periodontal connection between bone and tooth nonetheless is still sustained. Periodontal membrane reattachment is rapidly achieved by deposition of a layer of adhesive ground substance (a component of proteoglycans) on the resorbed bone surface, followed by the formation of new precollagenous fibrils. This can be done almost immediately after the resorptive action of the osteoclasts. Indeed, the fibroblast-like cells that do this trail just behind the ameboid-moving osteoclasts and reestablish attachment as the osteoclast moves from its Howship lacuna. The new fibrils, embedded in the adhesive proteoglycans secretion on the bone surface, become "stuck" onto the bone. They link with older collagenous fibers deeper within the periodontal membrane, and transitory attachment between bone and membrane is thereby produced. As the resorptive front continues, such adhesive attachments undergo removal in turn, to be replaced by new ones. Should a reversal occur in which the bone surface becomes depository, rather than resorptive, calcification of the interface proteoglycans layer becomes a "reversal line." Such reversals may be more or less permanent, with substantial new deposits formed. Or they may occur, as frequently seen, as temporary, thin scales of bone ("spot" deposits) that serve to reinforce transient attachments (Fig. 7-8). In either case, the reversal shows clearly as a refractile line at the interface.

Wherever connective fiber-to-bone anchorage attachments are made on resorptive alveolar bone surfaces, the fibers of the periodontal membrane are **taut between bone and tooth**. Thus, even though the "resorptive" side of the socket is often referred to as the "pressure side," the fibers are actually under tension, as seen in Figure 7-8, top, side *B*. The periosteal and periodontal vascular, connective tissue membranes are constructed to function in a field of traction (as by the pull of a muscle or biting force on a tooth), not marked surface pressure. Covering

membranes are quite sensitive to direct compression because any undue amount causes vascular interference and impedes osteoblastic formation of new bone. Osteoclasts can function to "relieve" the degree of pressure by removing bone. A commonly heard cliché is that "bone" is "pressure sensitive," and that high-level pressure induces resorption. Actually, it is the covering membrane and not the hard part of the bone itself that responds in such a manner. However, there are two general targets for biomechanical forces acting on bone: (1) the bone's membrane and, (2) the bone's calcified matrix. The nature of response is different for each. If surface pressure is exerted on the membrane, the resultant compressive effect is to restrict the vascular bed with an osteoclastic result (if the compression is not so great as to cause complete necroses and a close-out of function), and the tissue response is resorption in the specific, localized area so involved. **Tension** acting on the membrane, in contrast, is generally osteoblastic, and the response is new bone deposition. These responsive actions presumably continue until physiologic and biomechanical equilibrium is attained, whereupon the blastic and clastic activities are turned off.

FIGURE 7-8



The above biomechanical relationships deal with growth actions on a bone's **vascular membranes**. An additional **bone matrix** factor exists. Stresses on bone's intercellular matrix have been shown to have a different but also important mode of remodeling action, as shown schematically in Figure 7-9. The **piezo** (bioelectric) response to a physical force results in a histogenic bone response accompanying a bone's displacement. The action of a muscle or tooth, the bearing of weight, and the forces of growth itself cause minute distortions within a bone at the ultrastructural level (arrows). This leads to regional changes in configuration involving localized surface convexities and concavities. A concavity results in matrix compression and a negative surface charge (*B*), and a convexity causes tension in the bone matrix and a resultant positive surface charge. This triggers bone deposition and resorption (*C*), respectively, by the piezo effect (page 240) acting on surface cell receptors of osteoblasts and osteoclasts. The bone thereby remodels until biomechanical and bioelectric neutrality (remodeling equilibrium) is attained (*D*), and the signals activating the whole process are turned off.

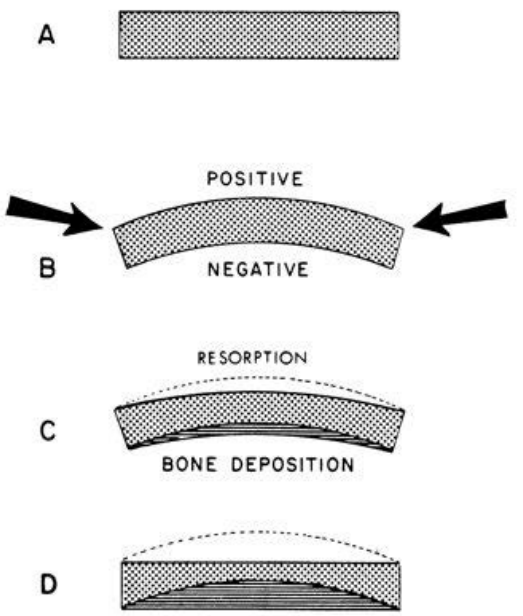


FIGURE 7-9.
Piezo response to forces acting on the bone matrix. See text for description.

Note that the nature of this bioelectric response is **opposite** to that seen for forces acting on a bone's covering soft tissue. This is significant. Pressure on the (1) periosteum or periodontal membrane leads to resorption, and tension can trigger deposition. Pressure in the bone's matrix (2), conversely, leads to deposition with tension relating to resorption. The nature of the operations and balance between these seemingly opposite remodeling effects is interesting and suggests a biologic interplay that is very significant.

As an exercise, see if an advanced student can account for orthodontic tooth movements on the basis of the membrane/bone matrix information provided in the preceding paragraph. A seeming obstacle, however, is that **all** of the alveolar surfaces are concave. One theoretical idea will be helpful in addressing that problem. If an existing concave surface becomes **more concave**, the effect is active compression and the action-response thereby depository. If an existing concave surface becomes **less** concave, however, the action results in less compression and in a "direction" toward tension; the response is thereby resorption. If a convex surface becomes either more or less convex, similarly, the results are believed to be resorption and deposition, respectively.

A reflective thought with regard to the biology of tooth movement: consider the **remarkable degree of precision** operative among the separate movements of a tooth, its alveolar bone, the periodontal connective tissue, and the remodeling of all the other surrounding hard and soft tissues affected by these movements. It is, in effect, a "symphony" of movements having **zero** latitude of misplay among them. Otherwise, everything would soon become developmentally mismatched, and the whole process would end quickly in gridlock. This wondrous growth system is a **precisely** coordinated, interactive composite of movements.

A tooth cannot move itself; it must be physically moved by the soft tissue surrounding its root. The directions, the amounts, and the timing of the tooth's movement must be **precisely** matched with no variance **at all** by the connective tissue remodeling of the periodontal membrane and alveolar bone remodeling movements. Attachments must be sustained all the while. If the tooth moves farther than, faster than, or at a time different from that of the resorptive bone side, for example, the periodontal space would become lost, and tooth-to-bone ankylosis would result. Similarly, the same matching growth actions on the depository alveolar bone surface must proceed in precise coordination between both and tooth for the same reason. Too much or too little, in any varying directions, one way or the other, or with off-timing, the periodontal space would either be lost or enlarged beyond functional tolerance. The PDM must remodel itself, sustain its breadth, provide relinkages, maintain attachments, and continue to relocate with the bone/tooth all the while. The whole process works because of the exceedingly finely tuned, exact operation of the signals activating the closely interrelated developmental responses involving all of these separate parts. This underscores concordance within a communal organization in which everything happens with mutual and reciprocal precision and harmony. Function continues uninterrupted throughout.

This remarkable developmental system operates under an intrinsic system of control and implementation responsive to functional conditions and complex developmental circumstances that spread throughout the craniofacial assembly during ordinary development. **Orthodontic** tooth movement harnesses and manipulates this control system through clinically induced signals that override and replace or modify the intrinsic signals. These signals can be produced by fixed and/or removable orthodontic appliances. The genic tissues respond to the signals without regard to their source. Therefore, more attention should be focused on identifying the type of biologic signals generated by various orthodontic devices and

bracket designs, rather than simply the way an archwire is attached or whether the appliance is fixed or removable. The common misconception that fixed appliances move teeth and removable appliances adjust bone is both inaccurate and incorrect. (See Hans et. al. 2007) Importantly, the system of operation itself, however, is biologically the same and utilizes the same intrinsic histogenetic mechanisms. That is, in conclusion, the essential point. Orthodontists should think in terms of "controlling biologic input" and regulating histogenic biologic systems rather than taking a purely mechanical approach to patient care.

8

Facial Form and Pattern

If a child's facial form and pattern are essentially balanced, "balanced growth" will then sustain it. Imbalanced growth, however, will alter the pattern into an imbalanced state. If a child's face is imbalanced, "balanced growth" will sustain the imbalance. An imbalanced child's face requires, in effect, imbalanced growth in order to achieve a structural craniofacial balance. Whichever, the infinite balance/imbalance mix and headform variations, population differences, and sex dimorphic variations result in a bewildering spectrum of facial "types." This chapter addresses basic developmental reasons. Other chapters evaluate anatomic patterns underlying categories of variations.

In your lifetime, you have seen the faces of thousands of people, and each face is recognizable to you as distinctively individual. No two are quite alike, even those of identical twins. Every person's face is a custom-made original; there has never been another face exactly the same before, and there never will be again. Yet consider how relatively few parts comprise a face: a lower jaw and chin, cheekbones, a mouth and upper jaw, a nose, and two orbits. Add a forehead and supraorbital ridges for the neurocranial parts relating to the face. How is it possible that so few components can underlie such great variation in facial form?

The answer is that we have the ability to perceive exceedingly subtle differences in the relative shape, spread, and proportions of both hard and soft tissue parts and minute variations in the topographic contours among all of them. Very slight alterations in the configuration of the nose, for example, make a substantial difference in the appearance and the character of one's face as a whole. (Fig. 8-1, shows a sketch from photographs of the same person before and after rhinoplasty; they look like two quite different individuals, although only a minor nasal contour has been altered.) Furthermore, there is the particular "set" to a person's mouth, the personal sparkle in the eyes, and the tone in the muscles of facial expression that are quite individualized. Often we ask, "Who does that person remind you of?" because there is some unique combination of nasal contour, lip configuration, jaw shape, and so on, that resembles some other face known to us.

Anthropologists can "reconstruct" the face from a dry skull by use of normative population data that provide integument thicknesses in the different areas of the face. However, the results can provide only a general approximation, because population "averages" can never match the delicate topographic features of a given individual in all, or even most, regards. Everybody is familiar with the method by which a police department artist attempts to draw a suspected felon's face from the recollections of eyewitnesses. Sometimes the artist's "composite"

picture can be close enough to give a more or less recognizable likeness, but often it is vague at best. It depends on how thoroughly a witness can recall and visualize key facial features. Also, the effectiveness of the artist's rendering depends on how accurately the witness can select the proper features from the police department's "catalogue," picturing different noses, cheekbones, hairlines, eyebrows, chins, and so on. As pointed out before, relatively subtle differences in a given feature can produce a quite noticeably different overall facial "character."

FIGURE 8-1

In the next few pages, the biologic rationale underlying common variations in facial features is described. Three general considerations are taken into account: (1) different facial types as they relate to variations in the development of overall form and shape of the whole head, (2) male and female developmental facial differences, and (3) child and adult facial differences. As you study these variations, you will begin to realize that most of the same characteristics relate to all three categories, essentially for similar physiologic, developmental, and morphologic reasons.

HEADFORM

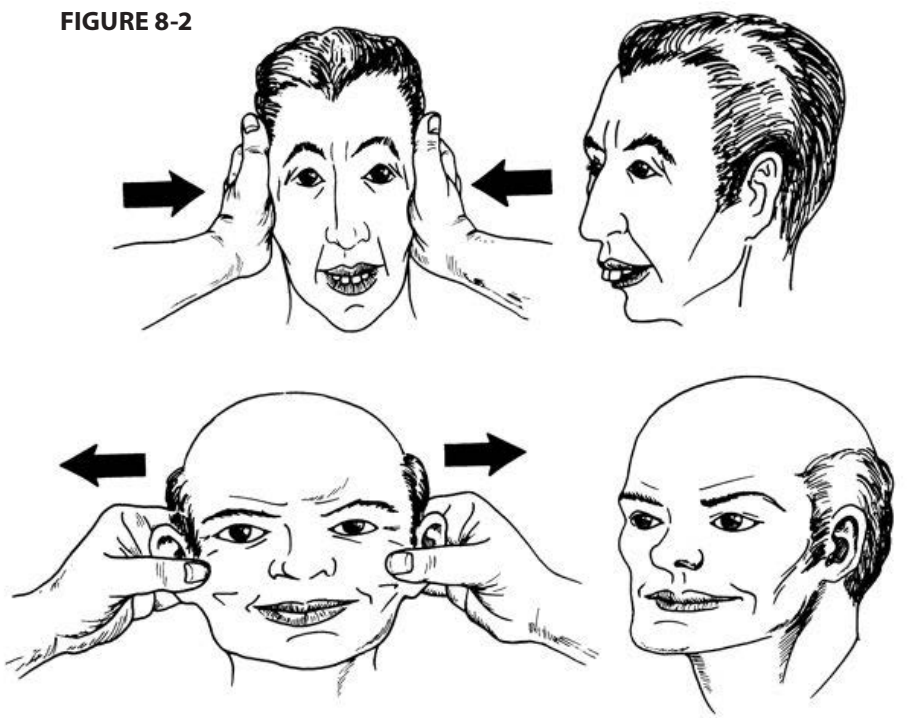
Two general extremes exist for the shape of the head: the long, narrow (dolichocephalic) headform and the wide, short, globular (brachycephalic) headform. The facial complex attaches to the basicranium, and the early growing cranial floor is the template that establishes many of the dimensional, angular, and topographic characteristics of the face. The dolichocephalic headform, therefore, sets up a developing face that becomes correspondingly narrow, long, and protrusive. This facial type is termed **leptoprosopic**. Conversely, the brachycephalic headform establishes a face that is more broad, but somewhat less protrusive, and this is called the **euryprosopic** facial type.

In Figure 8-2, observe what happens when a skull is dolicho- or brachycephalized. If faces are cast onto rubber balloons and the balloons then either squeezed or stretched, as shown, distinctively divergent facial patterns

occur with regard to the forehead, the shape of the nose, the set of the eyes, the prominence of the cheekbones, the contour of the facial profile, the degree of flatness (depth) of the face, and the position of the mandible. Note that the dolichocephalic nose is vertically longer and much more protrusive (Fig. 8-3). The pug-like brachycephalic nose is vertically and protrusively shorter, and it has a more rounded tip. Even though quite different in configuration, this latter nasal design is such that approximately equivalent airway capacity exists nonetheless, because it is proportionately wider. The vertically shorter midfacial feature of the wideface type (europrosopic), in turn, establishes a number of other facial features distinguishing it from the longer and more narrow midface of the leptoprosopic type (including differing basic malocclusion tendencies, as described in other chapters). Because the proboscis in the long and narrow facial form is also much more protrusive, the bridge and root of the nose tend to be much higher. In the dolichocephalic, also, the slope of the nasal profile tends to follow the same slope of the forehead, in contrast to the brachycephalic nose as it breaks from a more bulbous and upright forehead. Because the **upper** part of the dolichocephalic nose is also quite protrusive, the nose sometimes "bends" to produce an aquiline* ("Roman or Dick Tracy") type of convex nasal contour; and the end of the more pointed nose frequently tips down (the effect increases as age advances). The degree of the bending and downturning also increases with increasing height of the nose. Thus,

* Aquila is the generic name for the eagle, with its characteristic beak.

FIGURE 8-2



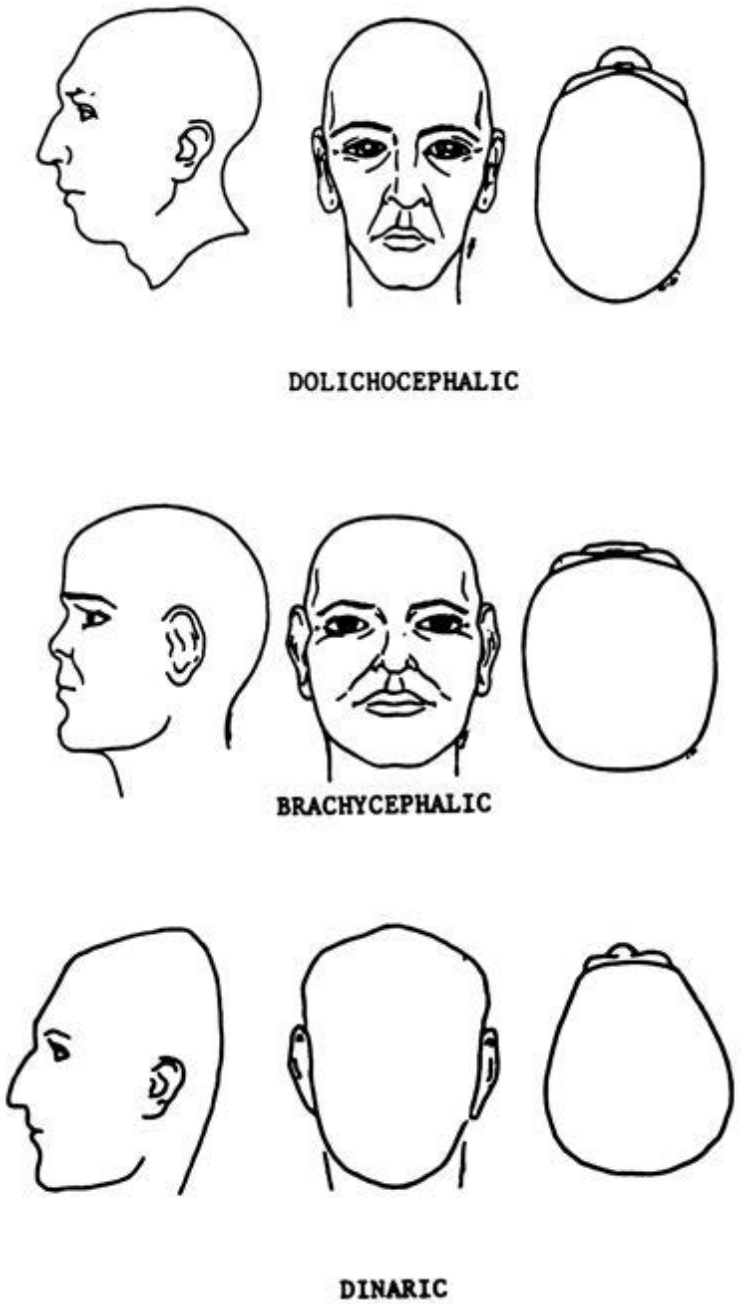


FIGURE 8-3.
The dolichocephalic headform has a cranial index of. 75 or less. The brachycephalic cranial index is .80 or greater. Between lies the mesocephalic (not shown). The dinaric cranial index is often hyperbrachycephalic (e.g., about .90 or greater). However, intermediate dinaric configurations with other headform types exist. In the lateral dinaric view, note the posterosuperior bossing. A variation is biparietal bossing (right). Note: The "cranial" index refers to a dry skull. The "cephalic" index includes covering soft tissue and has slightly different ratio values.

aquiline convexity becomes much more marked in persons having vertically longer noses. In contrast, the more stubby brachycephalic nose tends to be straighter or often concave, and it frequently tips up, with the external nares usually showing in a face-on view. (Note: A "third" nasal configuration also exists among some long-nosed dolichocephalics and dinarics in which the **middle** part of the external nose is protrusive relative to an upper part that is much less so. In this type, the nose displays a graceful, recurved, S-shaped configuration.)

Because the nasal part of the narrow (leptoprosopic) type of face is more protrusive, the external bony table of the contiguous forehead is correspondingly more sloping, and the glabella and upper orbital rims tend to be much more prominent. The forehead of the wide (euryprosopic) facial type is more bulbous and upright, and the frontal sinus tends to be thinner because of the lesser degree of separation between the inner and outer tables of the forehead. The more protrusive nature of the nasal region and the supraorbital ridges in the dolichocephalic type of headform gives the cheekbones a much less prominent appearance, and the eyes appear more deep-set for the same reason. As seen from above (Fig. 8-3) as well as laterally, the dolichocephalic face (Fig. 8-6, left) is more angular and less flat. In the brachycephalic headform (right), the wider, flatter and less protrusive face gives the cheekbones a noticeably squared configuration and a more prominent-looking character. The brachycephalic eyeballs are characteristically more exophthalmic (proptotic) because of the shorter anterior cranial fossa (the floor of which serves as the roof for each orbit). The orbital cavities are thus more shallow causing the eyeballs to bulge in appearance. The broad brachycephalic face also appears quite shallow in comparison with the deeper and topographically more bold contours of the dolichocephalic face.

The vertically long nature of the dolichocephalic midface and the "open" (obtuse) form of its basicranial flexure (see Chapter 10) relate to a downward-backward rotational alignment of the mandible. This results in a tendency for a retrusively placed mandible and retrusive lower lip with a retrognathic (convex) facial profile (Fig. 8-4). The brachycephalic face, conversely, relates to a more "closed" basicranial flexure. As a result, the lower jaw tends to be variably more protrusive, with a greater tendency for a straighter or even concave facial profile and a more prominent-appearing chin (Fig. 8-5) The vertically shorter midface in this facial type tends to highlight a more prominent appearance of the mandible. The more upright (closed) nature of the brachycephalic basicranium produces a tendency for more erect head posture, in contrast to a tendency for a more slumped stance and head posture in many individuals with a dolichocephalic headform. The narrow but longer anterior cranial fossa in the dolichocephalic headform (Fig. 8-6) results in a correspondingly longer but narrower and deeper (high vaulted) maxillary arch and palate. The broad but anteroposteriorly shorter brachycephalic type of anterior cranial fossa sets up a wider but shorter and more shallow palate and maxillary arch. **The palate is a configurational projection of the anterior cranial fossa. The configuration of the apical base of the maxillary dental arch, in turn, is established by the perimeter of the palate.** These are basic developmental and anatomic relationships. As emphasized in earlier chapters, the development of any given region is **not** wholly "preprogrammed" within itself. Rather, factors external

to the region can largely determine size and shape. A link thereby exists between brain and basicranium down to actual palatal and dental arch configuration. These same long-narrow and short-wide cranial and facial relationships are also routinely seen in other mammalian species (e.g., the Doberman pinscher or collie versus the bulldog or boxer).

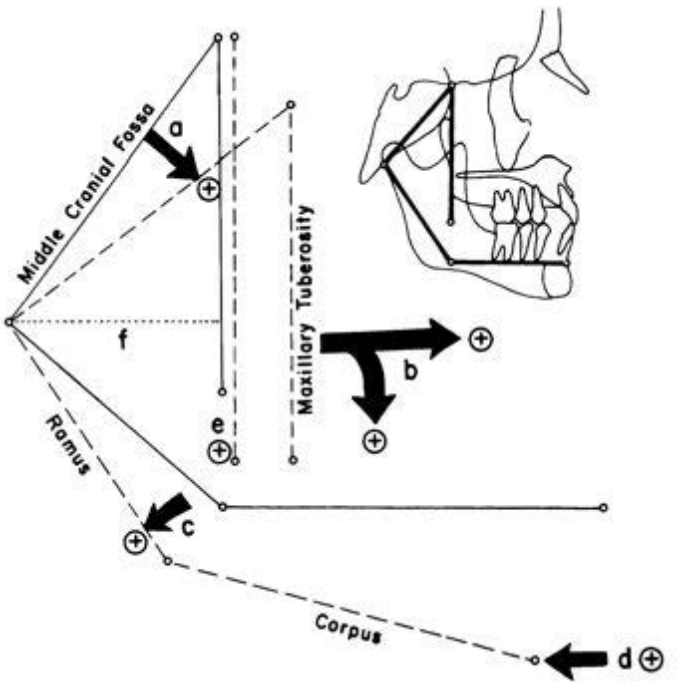


FIGURE 8-4.
Mandibular retrusive/maxillary protrusive effects (+) are seen when there is (a) an anterior inclination of the middle cranial fossa; (b) an anteriorly and inferiorly positioned maxillary complex due to the anterior inclination of the middle cranial fossa; (c) a downward and backward alignment of the ramus; (d) a posterior and inferior positioning of B point due to a backward rotation of the ramus; (e) a long nasomaxillary complex; (f) an increased span of the middle cranial fossa (MCF) due to an anterior inclination of the MCF. A closing of the gonial angle at c would add to the mandibular retrusive effect. (From Bhat, M., and D. Enlow. Facial variations related to headform type. Angle Orthod., 55:269, 1985, with permission.)

Among most of the world's different human population groups, either the brachycephalic or the dolichocephalic type of headform tends to predominate. Keep in mind that very few population groups are truly genetically homogeneous, even though "assumed" otherwise. Genetic admixtures and diverse population blends are nearly always operative, whether European, Asiatic, New World, or anywhere. A distribution **range** from one extreme headform or facial type to the other thereby usually exists within a given population, even though one or the other particular side of the range is the more common. An intermediate headform type (mesocephalic) can occur, and the facial features tend to be correspondingly

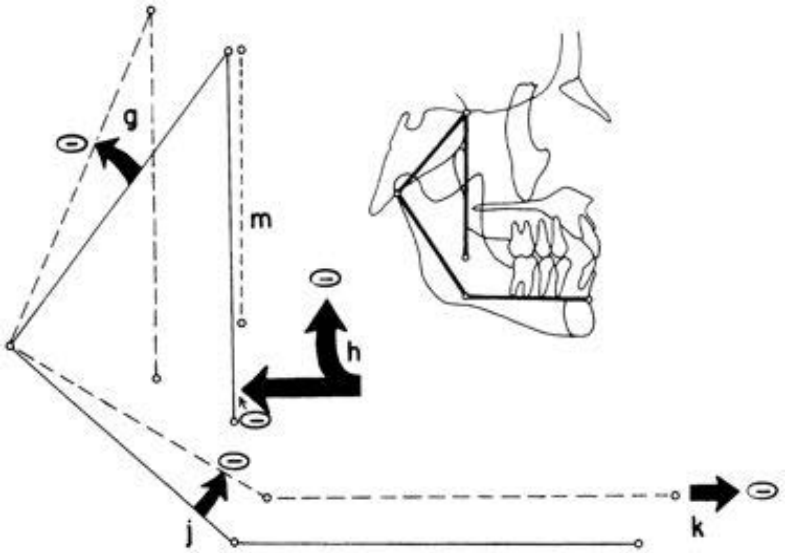
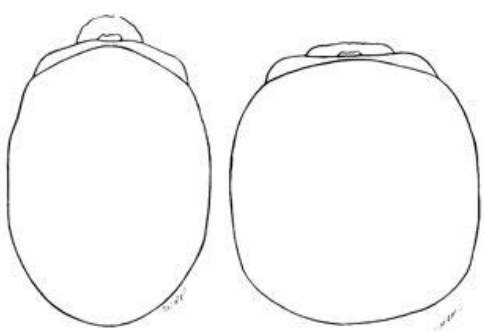


FIGURE 8-5.
Mandibular protrusive/maxillary retrusive effects (-) are seen when there is (g) a posteriorly inclined middle cranial fossa; (h) a posteriorly and superiorly positioned nasomaxillary complex due to a posterior inclination of the middle cranial fossa; (j) a forward and upward alignment of the ramus; (k) an anteriorly and superiorly positioned B point due to the forward alignment of the ramus; (m) a short nasomaxillary complex. Opening of the gonial angle at j increases the mandibular protrusive effect. (From Bhat, M., and D. Enlow. Facial variations related to headform type. Angle Orthod. 55:269, 1985, with permission.)

intermediate. In the northern and southern edges of continental Europe, as well as in most of England, Scotland, Scandinavia, northern Africa, and some Near and Middle Eastern countries (e.g., Iran, Afghanistan, India, Iraq, and Arabia), the dolichocephalic headform tends to predominate. In central Europe (the Alpine headform) and the Far East (Oriental), brachycephaly is predominant. In many worldwide areas, however, massive population migrations, wars, and the ease of travel interchanges have led to a much muddled distribution of the historical map of headform types just outlined. In each headform category, a **range** typically exists, from less to more, for the expression of features. See Chapter 10 for a morphologic evaluation of the overlaps and mixes of features.

FIGURE 8-6



Special Note. Figures 8-4 and 8-5 are specially highlighted and are referred to as the "Rosetta Stones" of Facial Growth. They summarize and demonstrate the principal structural features in a growing craniofacial assembly that can combine to form a mandibular retrusive face (Figure 8-4) and a mandibular protrusive face (Figure 8-5). It is suggested that framed copies be hung on your office wall where they can be very useful and referred to as needed for spotlighting key relationships underlying normal as well as abnormal faces described throughout this book. They are very effective for explaining during a teaching seminar or lecture the basic anatomic and developmental relationships predisposing malocclusions. The principal developmental variables include (1) the lead rotational positions of the Middle Cranial Fossae; (2) the resultant range of displacement positions of the Anterior Cranial Fossae and underlying Nasomaxillary Complex; (3) the resultant range of displacement positions of the whole Mandible by both the Middle Cranial Fossae and Nasomaxillary Complex; (4) the resultant opening and closing of the gonial angle; and (5) the displacement effects on the Mandible of a vertically long versus short Nasomaxillary Complex; Note how the sequence begins with the Middle Endocranial Fossae, then responding in turn by the Anterior Cranial Fossae, and then by the Nasomaxillary Complex, and finally by the Mandible. In each Figure, all of the retrusive and protrusive features may not, and often do not, necessarily occur in any given individual face. Many mixed combinations, both in numbers of features and in their magnitudes, occur with frequency, particularly in composite populations.

The Dinaric Headform

Interestingly, at certain geographic interfaces between the dolicho- and brachycephalic regions of the world, a "third" and quite distinctive type of headform commonly occurs, the **dinaric** (after the Dinaric Alps, in former Yugoslavia). The resultant admixture has, according to one theory, resulted in a "brachycephalized dolichocephalic" having a long face and large nose but with a brachycephalic cranial index. Such interface areas include regions located between middle and northern Europe, between central and southern Europe, and between Europe and the Near East. Thus, several geographically separate evolutionary lines of this headform have independently appeared and have become fairly common, although separate lines all share a number of similar craniofacial features. Admixtures of different headform types do not, thus, necessarily produce a consistent "mesocephalic" result, although this as well as pure "dolicho/brachy" offspring (mendelian-type) ratios can occur in an individual family. Rather, quite a different anatomic "blend" occurs in the dinaric. Although technically brachycephalic because it is anteroposteriorly short, it is primarily the **posterior** part of the dinaric head that has been brachycephalized (Fig. 8-3). Two basic variations exist, both of which involve **bossing** of the skull roof. First, the occipital or lambdoidal regions become widened and markedly **flattened**, with bilateral peaking (bossing) of the parietal region. The skull often has a distinctively triangular configuration when viewed from the top. Second, another common variation occurs in which the bossing is directed more upward than bilaterally, thereby forming an elevated hump or peak in

the posterosuperior part of the skull dome. Cranial configuration is less triangular when viewed from the top. Whatever the manner of bossing, it accommodates the volumetric mass of a brain that has undergone alteration in overall shape. The cranial index is often hyperbrachycephalic (i.e., 90 or greater).

The old practice of "cradling"† causes or at least intensifies occipital flattening by the external force acting on the skull. Indeed, the sleeping posture of an infant, whether or not cradled as such, is likely a factor in "dinarizing" a growing head to some greater or lesser extent. It has been argued that such mechanical influences during early infancy represent the dominant reason for causing the dinaric form if the sleeping position is predominately on the back and sustained through childhood. Interestingly, it has been shown that American descendants of Old World dinaric grandparents can lose the dinaric features because cradling is not practiced. It is also argued, however, that a "genetic" dinaric form also exists because dinaric individuals certainly exist who have not been cradled as infants and who have mixed sleeping positions. Observations suggest that the "biparietal bossing" type is a product of cradling or, at least, early backposition sleeping habits. The "peaked dome" type, in contrast, may be a more "genetic" type, although formal studies are now needed to test these hypotheses. The matter is significant because the dinaric headform, with its variations and degrees of magnitude (see below) shows different malocclusion tendencies and responses to different treatment procedures.

The ears of the dinaric characteristically appear much closer to the back of the head because of the occipital flattening, as noted in Figure 8-3. Of course, it is not the ears themselves that are back, but rather the posterior flattening making them appear so. The anterior part of the skull retains the relative narrowness that characterizes the dolichocephalic pattern. The narrow face from the dolichocephalic side of the ancestral heritage has perhaps "constrained" the anterior cranial fossa, or perhaps the converse, thereby sustaining a narrow dimension in this part of the basicranium. Even though the headform is technically brachycephalic, the form of the face itself is distinctively leptoprosopic, quite unlike the typical eurosopic brachycephalic pattern. The posterior facial parts (such as the mandibular ramus and temporomandibular joint [TMJ] region) tend to flare laterally in the "biparietal" (triangular) headform type of dinaric because they grade farther back on the widening cranial triangle. The forehead is often quite sloping in many individuals of all dinaric types, the supraorbital ridges are prominent, and the face is long and topographically protrusive. The nose tends to be very large and often aquiline (this occurs in many females as well as males), and the nasal bridge is high. The mandible tends to be less retrusive, the face less retrognathic, and the profile tending more toward orthognathic. This is because the basicranial flexure is compressed and more closed (see page 145). The midface, however, tends to be vertically long in proportion, even more so than among dolichocephalic leptoprosopics. These various leptoprosopic features often appear exaggerated in character in the dinaric headform, almost as though the brachycephalized, flattened posterior part of the cranium "pushed" the face even more protrusively than in a routine dolichocephalic headform. Any malocclusion in a dinaric will

† Immobilizing an infant on its back with a wrap of swaddling clothes on a hard board.

have a combination of structural features different from that in a dolichocephalic. Both, in turn, are different in malocclusion anatomy from a brachycephalic, and treatment responses and rebound tendencies will also be different (See Bhat and Enlow, 1985; Martone et al., 1992.)

While the dinaric headform has been historically perceived as a "brachycephalized dolichocephalic," it is probable that **any** headform type, including the brachycephalic and mesocephalic, is susceptible to this modification, whether by sleeping habits or through hereditary mix. Intermediate variations, further, certainly exist, but have yet to be studied and catalogued. "Partial dinarization" is commonly observed in individuals having, for example, a mesocephalic index.

Headform and Orthodontic Treatment

Because the "face is built on the brain", the three dimensional morphology of the cranial base influences the size, shape and position of the maxilla and mandible. Since the cranial base establishes the template for facial structures it must dictate the anatomic limits for those structures. For example, the maxillae of brachycephalalic individuals can be wider then maxillae of dolichocephalic headforms because the cranial base template is wider in brachycephalic individuals. The logical extension of this clinical thinking is that palatal expansion may be indicated more often in brachycephalic individuals since the lateral anatomic limits for maxillary width are greater in this group. Previous studies have shown that extraoral orthopedic traction and removable dental appliances have divergent responses in the different headform groups and subgroups (Enlow et al., 1988; DiPalma, 1983; Martone et al., 1992). Because clinical procedures harness "growth" by activating regional modifications in the directions and amounts of histogenesis that lead to the different headform types, awareness of headform variations and facial differences is a fundamentally important consideration for orthodontic treatment planning. With the increased use of low dose cone beam computed tomographic (CBCT) scans in clinical orthodontics greater interest in the 3D morphology of the cranial base and anatomic limits of orthodontic treatments is likely. Exploring the anatomic limits of treatment in three dimensions will be an important area for research in the future.

Male Versus Female Facial Features

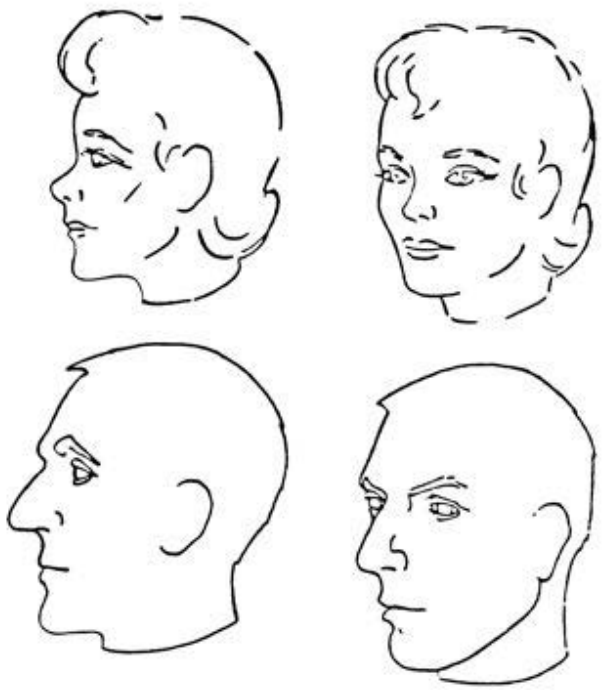
A talented artist can effectively render male versus female faces, and the viewer has no problem recognizing either gender from sketches or portraits of adults. (Children are another matter, as will be seen). However, many artists, as well as the average citizen, are not really conscious of the actual, specific anatomic differences involved. They just "know." In our mind's eye, we have all subconsciously associated, over the years, the topographic characteristics that relate to facial dimorphism.

The overall body size of the male tends to be larger than that of the female, and the male lungs are correspondingly more sizable to provide for the relatively more massive muscles and body organs. This calls for a larger airway, beginning with the

nose and nasopharynx. A principal sexual dimorphic difference, therefore, is the size and configuration of the nose, and this, in turn, leads to collateral differences in other topographic structures of the face, since the airway is a developmental keystone (see Chapter 1).

The male nose is proportionately larger than the female nose (Fig. 8-7). This is a "population" feature based on general comparisons among large numbers of people; any given individual female or male, of course, can display a smaller or larger nose. The male nose, in general, tends to be more protrusive, longer, wider, more fleshy, and tends to have larger and more flaring nostrils. The interorbital part of the nasal bridge in the male tends to be much higher. All of this is in contrast to a relatively thin and less protrusive female nose. The male nose usually ranges from a straight to a convex (aquiline) profile, whereas the female nose tends to range from a straight to a somewhat concave profile. The tip of the male nose is often more pointed and has a greater tendency to turn downward, and the somewhat more rounded female nose often tips upward. The external nares in the female are more often visible in a face-on view for this reason. A variation of the aquiline (Roman) type of nose, which is also much more prevalent among males than females, is the classic "Greek" nose, in which the nasal profile drops almost straight downward from a protruding forehead (Fig. 8-8). The reason for the multiple male variations in nasal configuration lies in the more protuberant nature of the whole nasal region. Both the upper and lower parts of the whole external nose are protrusive, but the lower part can be constrained to a degree by the septopremaxillary ligament, palate, and maxillary arch. The contour of the nose thus rotates"; it either "bends" to give an aquiline configuration or rotates into a straight, but more vertical, alignment.

FIGURE 8-7

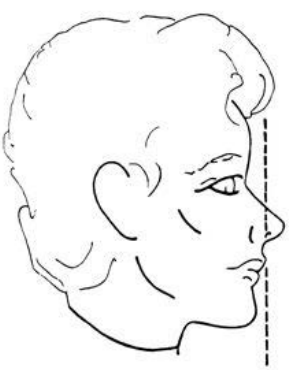


Because of the larger, more protuberant character of the male nose, the part of the forehead contiguous with it also necessarily remodels into a more protrusive position. Therefore, the male forehead tends to be more sloping, in contrast to a more bulbous, upright female forehead. The supraorbital and glabellar parts of the male forehead tend to be **quite** protrusive, as compared with the much **less** Neanderthal-like character of the female forehead. This, together with the differences in the relative size and vertical alignment of the nose, provide the features most readily recognizable in our subconscious perceptions of male and female faces.

In Figure 8-9, note the dashed line passing vertically along the surface of the upper lip perpendicular to the neutral orbital axis. In the female, this line usually crosses about midway along the upper nasal slope, and the forehead generally lies behind but seldom at the line. Conversely, in the male, the nose and forehead are often so protrusive that the forehead bulges out as far as this line or sometimes even beyond it, and the greater part of the nose often lies ahead of it.

FIGURE 8-8

FIGURE 8-9



Because of the greater extent of protrusiveness of the male forehead and nose, the eyes appear more deep-set. In the female the eyes appear more proptotic and "closer to the front" of the face. Female cheekbones also "look" much more prominent for the same reason; that is, the malar protuberances seem more apparent because the nose and forehead are less prominent. Indeed, "high cheekbones" are a classic feature of femininity, much emphasized by beauty analysts. Of course, the

malar protuberances are not actually "higher;" they are just more conspicuous. This topographic feature of the female face is better seen in a 45-degree view of the face (see Fig. 8-7). In addition, the temporal region along the side of the forehead tends to be more sloping and less bulgy in the female.

The composite of all these regional, topographic distinctions render the female face flatter, proportionately (not actually) wider in appearance, and more delicate in general character. The male face, in contrast, appears deeper, more irregular and knobby, and more coarse. The astute clinician will keep these general tendencies in mind when planning treatment to achieve aesthetic goals. For example, the male patient with a prominent chin may be more likely to accept an orthodontic compromise of his prognathism than a female with the same degree of mandibular prognathia.

The protuberant supraorbital part of the more sloping male forehead (because of the larger nose) is produced by a greater extent of remodeling separation of the outer table of the frontal bone beyond the earlier-stabilized inner table. In both sexes, the growth of the inner table stops when the enlargement of the frontal lobes of the cerebrum ceases around 5 to 6 years of age. The outer table, however, continues to remodel forward until contiguous nasal growth ceases, which is some years later. The inner and outer tables thereby diverge, and the cancellous bone between them is hollowed into the frontal sinus. Because the nasal part of the male face continues to grow for several years beyond that of the female, the frontal sinus is, therefore, much larger in the male face than in the more juvenile-like female face. Also, because of the smaller frontal sinuses in a female, the temporal regions of the lateral forehead often appear less full and thus more sloping. Because the forehead and nose are less protrusive in the female face, the upper jaw tends to "look" more prominent and muzzle-like. For this reason, the astute craniofacial clinician will identify the ideal position of the upper incisors in females as slightly more forward than the ideal position for their male patients. This creates a degree of relative mandibular retrognathia that is often quite acceptable and even desirable for female patients. "One size fits all" treatment planning negates the inherent anatomic differences between men and women and clinicians who ignore this fact do so at their own peril.

Now relate these male and female features to the facial features previously described that distinguish the dolichocephalic from the brachycephalic headform. This includes the degree of supraorbital protrusion, forehead slope, cheekbone prominence, nasal configuration, orbital depth, and depth or flatness of the whole face. These headform characteristics are **the same** as those that distinguish the male from the female face. The long, narrow **dolichocephalic** basicranium and facial airway lead to facial features that essentially parallel those of the **male** face. The short, wide **brachycephalic** basicranium and facial airway set up facial features that parallel those of the **female** face. The nasal part of the face underlies much of the overall character of a person's facial form. In the "brachy/dolicho" situation, it is the width and length of the basicranium that (1) establish the basis for nasal form and size and (2) the positions of all the various facial parts and their relative vertical, sagittal, and bilateral proportions. In the male/female comparison, it is **relative whole body and lung size** that leads to corresponding nasal characteristics,

which, in turn, establish the other facial features that are analogous to those also associated with headform type. Please do not misunderstand this "headform and male/female" convergence. We are not saying that males are dolichos, and that females are brachys. Headform and gender are independent variables, and all possible combinations are possible.

Then what about a female dolichocephalic? Or a male brachycephalic? Or a female dinaric? Most people naturally feel a comforting confidence that they can usually distinguish gender by a person's face. However, recognition tests have shown that it is actually not all that easy. If ancillary clues (e.g., hairstyle, cosmetics) are removed by masking from facial photographs and other kinds of clues are not available (e.g., voice, clothing, gait, presence or absence of a protruding larynx, neck circumference, breadth of shoulders), recognition tests in which facial photographs are presented can lead to dismay and despair. The chances of a correct identification for many individual faces or parts of faces are often not much better than a coin flip.

In a female **brachycephalic**, the headform characteristics of a wider and flatter face, smaller nose, squared cheekbones, and an upright forehead tend to augment and emphasize the **same** dimorphic features that also relate to gender. Conversely, in the female **dolichocephalic**, the larger-nosed, more angular face, and generally more protrusive facial characteristics associated with this headform type tend to give a more "male-like" cast to the face. Of course, these features are **not** really masculine at all, but, rather, headform-linked. Thus, a leptoprosopic female is characterized by a more sloping forehead, greater supraorbital protrusion, a higher nasal bridge, a longer nose, an aquiline or more vertically aligned nasal contour, a downturned and more pointed nasal tip, and, often, a more retrognathic mandible. The average citizen has subconsciously learned the difference between brachycephalic and dolichocephalic females, even though few have any idea what a brachycephalic or a dolichocephalic is. In a male brachycephalic, the reverse situation exists. The "female-like" characteristics of the brachycephalic headform present a flatter, wider face with more prominent cheekbones; a more bulbous forehead; a smaller, less protrusive nose with a lower nasal bridge, less deep-set eyes, and a tendency for a straight-to-concave nasal profile with a more rounded and upturned tip. One frequently sees female impersonators on TV (e.g., a detective disguised as a lady of the street); the realism of the disguise is favored if the male is a wide-faced, small-nosed brachycephalic. A male dolichocephalic, as in a female long-head, has the headform features augmenting the gender characteristics, including the nasal, forehead, cheekbone, deeper set eyeball, retrusive mandibular tendency, and so on. Female "impersonation" would be more difficult and less believable. These characteristic features do not compromise the masculine character of the face; they simply represent another facial variation that we all have learned to recognize.

Finally, a common misconception is pointed out. It is frequently heard that the female mandible, more often than in the male, has a typically retrusive set (e.g., a more "submissive" position). True anatomic retrognathia (as opposed to relative retrognathia described previously) is a headform feature, not a sexual dimorphic one.

Child Versus Adult Facial Features

The faces of prepubertal boys and girls are essentially comparable. How many times have you been embarrassed by calling a little boy a girl? In the female, facial development begins to slow markedly after about 13 or so years of age. At about the time of puberty for the male, however, the sex-related dimorphic facial features just described begin to be fully manifested, and this maturation process of the facial superstructures continues actively throughout the adolescent period and into early adulthood. This is a factor to be taken into account by all craniofacial practitioners in treatment planning for girls and boys because it often has a greater aesthetic impact on the adult facial profile than some treatment interventions. For example, the effect of post pubertal nasal and chin growth has a much greater impact on the facial profile than the removal of four bicuspid teeth.

Whether a young child's headform is dolichocephalic or brachycephalic, the youthful face itself appears more brachycephalic-like because it is still relatively wide and vertically short. It is wide because the brain, and therefore the basicranium, is precocious relative to facial development. The neurocranium grows earlier, faster, and to a much greater extent that the contiguous facial complex. The wider basicranium, because it establishes TMJ positions for the mandible and the facial-to-cranial sutures for the nasomaxillary complex, is thereby a template that also paces the early **width** of the growing face. The face is **vertically** short because (1) the nasal part of the face is still diminutive (overall body and lung size still correspondingly small); (2) the primary and secondary dentition has not yet become fully established; and (3) the jaw bones have not yet grown to the vertical extent that will later support the full dentition, the enlarging masticatory muscles, and the airway.

Note (Figs. 8-10 and 8-11) the features of the child's face compared to the adult, regardless of sex or headform type: the nose is short, rounded, and pug-like; the nasal bridge is low; the nasal profile is concave; the nares can be seen in a face-on view; the forehead is bulbous and upright; the cheekbones are prominent; the face is flat; and the eyes seem wide-set and bulging.

The same situation, noted twice before, thus exists with regard to facial pattern: most of the **same** facial features that characterize **both** headform and sexual dimorphism **also** relate to the differences between the facial features of the child and the adult. In all three categories, the character of the nasal part of the face is a key factor that relates directly to the other facial features (forehead slope, nasal configuration, height of nasal bridge, cheekbone prominence, flatness of the face, and the general extent of facial protrusiveness). Because the facial and pharyngeal airway is yet small because of diminutive body and lung size, it thus sets up the early developmental conditions paralleling a topographic similarity with the brachycephalic headform (i.e., the basicranial template) **and** the male/female difference (i.e., the airway). The childhood situation, however, is developmentally independent of both the headform and sex dimorphic factors.

How does one recognize advancing age in an **older adult** by external facial appearance? There was a time when edentulism caused major changes in facial structure and topography. In many parts of the world, modern dentistry

FIGURE 8-10

FIGURE 8-11

has effectively precluded much of this. Other facial changes, however, still occur. The velvety, soft, pink, tightly bound, resilient, and firm skin of the child becomes replaced over the years by the more leathery, crinkled, open-pored, limp, blemished skin that progressively characterizes more aged persons. As one advances through middle age, the integument begins to droop and sag noticeably. Several physical and biochemical changes are occurring in the connective tissue of the dermis and hypodermis that cause the skin to become less firmly anchored to underlying bone or facial muscles. First, if general loss of body weight occurs for whatever reason as one ages, resorption of subcutaneous adipose results in a "surplus" of skin which, with gravity, leads to sagging, wrinkling, and creasing. Loss of adipose thus exaggerates age appearance. After dieting, for example, a face often looks older. The effect can occur in children as well; the sight of a severely malnourished child with a wrinkled, lined, hollow face is not easily forgotten. Second, the distribution and character of the collagenous matrix change with advancing age. Fibers increase in massiveness, and the whole skin decreases in resilience. Third, fibroblasts decline in number as well as cellular activities. The latter includes a marked decrease in the secretion and overall amount of hydrophilic (water-bound) protein mucopolysaccharides (proteoglycans). Because of this, a widespread subcutaneous dehydration occurs that contributes significantly to shrunken facial volume and skin surplus, with consequent skin wrinkling. In advanced old age, a person's face can become an expansive carpet of noble ripples and lines. Resorption of adipose in the orbit leads to a sunken appearance of the eyes, and the more visible venous plexus in the thinned suborbital hypodermis produces a darkening of the skin below the eyes. The suborbital integument can also begin to sag perceptibly to form "bags." Something also happens to the youthful "sparkle" in many persons' eyes as they age. By waxing artificial wrinkles onto the face and blue-tinting the suborbital region, a good Hollywood make-up technician can "age" a face in minutes. Observe closely, however, and you will note that, unlike real skin, the artificial furrows are not as motile during attempts at expressive facial movements.

Facial lines and wrinkles develop in specific and characteristic locations, particularly during the middle-age period. One of the initial lines to appear is the prominent **nasolabial furrow**. This "smile line" is seen at any age when one grins, but it becomes a fixed feature of the face sometime during the late 30s or 40s in many people and continues to deepen and become more marked. It extends from the lateral side of each nasal down to the corners of the mouth. This is a particular facial feature that we have subconsciously learned to associate with the onset of middle age. Stopping smiling does not seem to help.

Other wrinkles and creases begin to develop as crow's feet at the lateral corners of the eyes, horizontal lines on the forehead, vertical corrugations overlying the glabella, vertical furrows along the upper lip, lines extending down from the corners of the mouth lateral to the chin, a horizontal crease just above the chin, suborbital lines, drooping jowls over the sides of the mandible, and a "turkey gobbler" bag of skin sagging down over the neck below the chin. The placement, alignment, prominence, and number of such lines and creases, as well as many other topographic facial features, are utilized as presumptive clues by the physiognomist (a practitioner of the ancient Chinese art of face reading) to judge

a person's character, temperament, and ultimate fate. However, no real functional, preprogrammed, cause-and-effect relationships are likely to exist in most such correlations; there are just too many physiologic, anatomic, environmental, social, developmental, and ethnic variables involved in the biology of the human face.

How about the person who "looks younger than his or her years"? Or older? For reasons only partially understood, the onset of the smile line and some other facial wrinkles is delayed, or at least such lines look less marked, in youthful-appearing individuals. Conversely, in others, lines can appear more harsh and begin to develop at an earlier age. Intrinsic physiologic as well as environmental factors can contribute to this. For example, sun and UV damage to the facial integument, particularly in lighter-complexioned individuals, is known to accelerate the aging process of skin. Furthermore, chronic alcoholism causes the muscles of facial expression within the skin to sag because of the long-term anesthetized-like and limp state of their tone. Alcohol, further, is a dehydrator. Smoking tends to intensify wrinkling because tobacco is a peripheral vasoconstrictor. Major loss of adipose can also accelerate the onset of facial wrinkles, as previously explained. In addition, a euryprosopic (brachycephalic) type of face appears more juvenile-like because it resembles the more wide and short configuration characterizing the face of a child. A dolichocephalic adult face "looks" more mature because the nasal region is vertically longer and the face less wide in proportion. A fat face looks younger (1) because the subcutaneous adipose tends to smooth out wrinkles and (2) because it resembles the labial and buccal fat-padded face of a child. Thus, a wide-faced, more chunky, sober, nonsmoking, darker-skinned individual, particularly one protected from undue sun exposure, tends to retain a more youthful appearance somewhat longer.

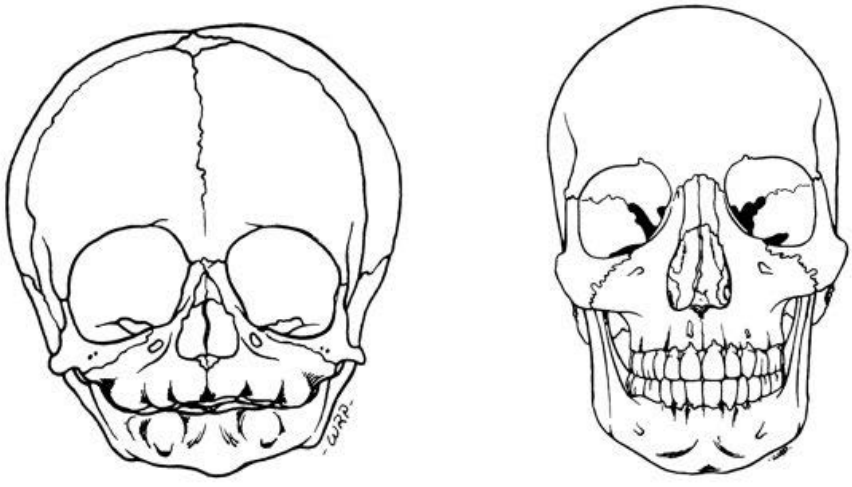
The Changing Features of the Growing Face

The "baby face" has large-appearing eyes, dainty jaws, a tiny pug nose, puffy cheeks with buccal and labial fat pads, a high intellectual-like forehead without coarse eyebrow ridges, a low nasal bridge, a small mouth, velvety skin, and overall wide and short proportions. It is a cute face. It warms the cockles of parental hearts. A parent can worry, though, because the otherwise great little face "has no chin," or "the jaw is much too small," or "the eyes are too far apart." However, these and many of the other features of the baby's face gradually undergo marked changes as the face grows and develops through the years. The chin develops, jaw size catches up, and the eyes appear less wide-set. From the many possible variations that can exist among different individuals, a person's own facial characteristics take on, month by month, definitive adult form. The general features of any fully grown face can be quite different from those of the same individual as an infant and young child. Trying to decide which parent the infant "looks like" or which uncle it "takes after" is a fun game, but usually more or less futile. There is little in the general shape and proportions of the infantile face, at least topographically, to give a hint as to what form it will take in later years. Unless, of course, the adult happens to have a euryprosopic face with pudgy cheeks, wide-set eyes, pug nose, etc., all childlike features.

In general, the baby's face grows out from under the brain. Structures must grow proportionally more and for a longer period of time the further they are from the neurocranium. Therefore, growth of the mandible begins later and continues longer than midfacial or orbital development. Growth is not merely a process of size increases. Rather, progressive facial enlargement is a "differential" developmental process in which each of the many component parts matures earlier or later than the others, to different extents in different facial regions, in a multitude of different directions, and at different rates. It is a gradual maturational process involving a complex of different but functionally interrelated organs, parts, and tissues. The growth process also involves a bewildering succession of regional changes in proportions and requires countless localized, ongoing "adjustments" to achieve proper fitting and function among all of the parts.

The child's face is not merely a miniature of the adult, as dramatically illustrated in Figure 8-12 which shows a neonatal skull enlarged to the same size as a fully grown one. The baby's face appears diminutive relative to the larger, more precocious cranium above and behind it (Fig. 8-13). The respective proportions change significantly, however. The growth of the brain slows considerably after about the third or fourth year of childhood, but the facial bones continue to enlarge markedly for many more years to accommodate airway and masticatory growth and functions.

FIGURE 8-12



The eyes, which are precocious along with the brain, thus can appear large in the young child. As facial growth continues, however, the nasal and jaw regions later develop disproportionately to the earlier-maturing orbit and its soft tissues. As a result, the eyes of the adult appear smaller in proportion.

The ears of the infant and child appear to be low; in the adult they are much higher with respect to the face. Do the ears actually rise? No; they in fact move downward during continued development. However, the face enlarges inferiorly even farther, so that the **relative** position of the ears seems to rise. In an infant,

note that the body of the mandible is in near alignment with the auditory meatus, thus reflecting their commonality of embryonic origin. Later, the corpus descends as the midface and ramus lengthen vertically, and this relationship becomes more obscure.

The young child's precocious forehead is upright and bulbous. The forehead of the adult, however, becomes much more sloping (the amount of slope is related to sex and headform, as already explained). The forehead region of the child seems very large and high because the face beneath it is still relatively small. The child's forehead continues to enlarge during the early years, but the face enlarges much more, so that the proportionate size of the forehead becomes reduced.

FIGURE 8-13.
(Courtesy of William L. Brudon. From Enlow, D. H.: *The Human Face*. New York, Harper & Row, 1968, with permission.)

The child's face appears broad because the brain and basicranium develop earlier and faster than the facial composite, as already explained. As development continues, **vertical** facial growth (enlarging airway, dentition) then comes to bypass expansion in width to a marked extent, so that a much more narrow facial proportion characterizes the adult, especially in dolichocephalics and dinarics.

The nasal bridge is quite low in the child. It rises (to a greater or lesser extent in different facial types) to become much more prominent in adults.

The eyes of the infant can seem quite wide-set, with a broad-appearing nasal bridge between them. This is because the nasal bridge is so low, and also because

much of the width of the bridge has already been attained in the infant. With continued growth, the eyes spread farther laterally, but only to a relatively small extent. Actually, the eyes of the adult face are not much farther apart than those in the child. Because of the larger nose, higher nasal bridge, increase in the vertical facial dimension, and the widening of the cheekbones, the eyes of the adult thus **appear** much closer together.

The infant and young child have much more of a pug nose than the adult. It protrudes very little and is vertically quite short. The shape and size of the infantile nose, however, give little indication of what will happen to it during subsequent growth. The extent of proboscis enlargement can be considerable. The lower part of the nose in the adult is proportionately much wider and a great deal more prominent.

The whole nasal region of the infant is vertically shallow. The level of the nasal floor lies close to the inferior orbital rim. In the adult, the midface becomes greatly expanded, and the nasal floor has descended well below the orbital floor. This change is quite marked because of the enormous enlargement of the nasal chambers. Note the close proximity of the young child's maxillary arch to the orbit, in contrast to their positions in the adult.

The superior and inferior orbital rims of the young child are in an approximately vertical line or inclined behind (see Figs. 5-17 and 2-12). Because of frontal sinus development and supraorbital protrusion in the adult, however the upper orbital rim noticeably overhangs the lower. The orbital opening and lateral orbital rim become inclined obliquely forward. Supraorbital and glabellar protrusion is particularly marked in the adult male because of the larger nose needed to accommodate larger lungs.

Below the orbit, the nasal chambers in the adult face expand laterally nearly halfway across the orbital floor. In the infant, the breadth of the nasal cavity inferiorly scarcely exceeds the width of the nasal bridge and interorbital space (Fig. 2-25). During subsequent growth, the inferior portion of the nose expands laterally much more than the superior part.

The tip of the infant's nasal bone protrudes very little beyond the inferior orbital rim. The area **between** the nasal tip and the inferior rim of the orbit (i.e., the lateral bony wall of the nose) is characteristically narrow and shallow. In the adult, this area becomes markedly expanded. The divergent directions of orbital, nasal, cheekbone, and maxillary arch growth "draw out" the contours among them. The facial parts become spread apart and much deeper.

The nasal region of a growing child's midface is, almost literally, a keystone of facial architecture, that is, a key part upon which other surrounding parts, and the multiple anatomic arches formed by them, are dependent for placement and stability. If this keystone is malformed for any reason, other facial parts are affected during growth, and facial dysplasia or malocclusion can occur. The facial airway, therefore, is an exceedingly significant component involved in normal versus abnormal facial morphogenesis.

The orbits and the cheekbones in the child are more forward appearing because the whole face is still relatively flat and wide. In the infant, the protrusive appearance of the cheekbones is augmented by the characteristic infantile buccal

fat pad in the overlying hypodermis. Adults tending to have a relatively wide and short (thus more childlike) type of face typically show an even greater "cherubic" appearance if they are overweight; the buccal region contains adipose tissue resembling the buccal fat pad of infancy.

In Figures 8-14 to 8-17, note the following topographic facial variations: 1, the tarsal part of the upper eyelid exposed; 2, the eye laterally covered by an eyelid fold; 3, the iris covered by the upper eyelid; 4, most of the iris exposed; 5, the lateral corner of eye higher than the medial corner; 6, the lateral eye corner lower than the medial corner; 7, the top of the nasal bridge (root) markedly indented; 8, a high nasal root (so-called "Greek nose"); 9, a narrow nasal root; 10, a broad nasal root; 11, a narrow nasal slope; 12, a broad nasal slope; 13, a concave nasal profile; 14, a straight nasal profile; 15, a convex nasal profile; 16, inconspicuous nasal wings; 17, prominent nasal wings; 18, V-shaped nasal wings; 19, rounded nasal wings; 20, arched nasal wings; 21, straight nasal wings; 22, a narrow nasal tip; 23, a broad, flattened nasal tip; 24, a thick, fleshy nasal wing; 25, a thin nasal wing; 26, asymmetric nasal openings; 27, symmetric openings; 28, posterolaterally directed openings, 29, laterally directed openings; 30, narrow, elongate openings;

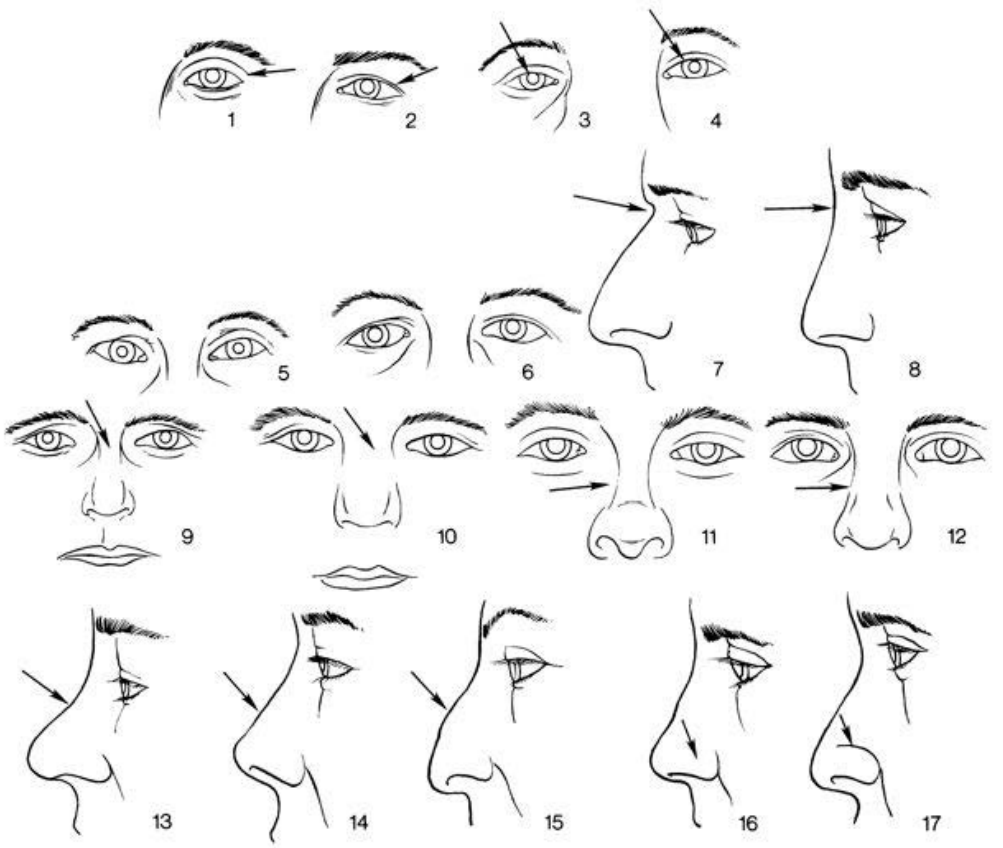


FIGURE 8-14.
(Modified from Hulanicka, B.: Nadbitka Z Nru 86, Materialow 1 Prac antropologicznych Wroclaw, 115, 1973, with permission.)

31, rounded nasal opening ; 32, an upward nasal inclination; 33, a straight lower nasal border; 34, a downward inclined nasal border; 35, a vertically short upper lip; 36, a long upper lip (check also to see whether the upper lip profile is straight or concave); 37, the upper lip without a midline "Cupid's bow"; 38, a deep midline notch in the upper lip (look also for a more conspicuous philtrum above the upper lip, and check for thinness or thickness of the red part of both the upper and lower lips); 39, an acutely curved lower border (concavity) below the lower lip; 40, lesser concavity between lower lip and chin and a greater distance between the lip and mentolabial sulcus; 41, the lower lip retrusive; 42, the lips equally protruding; 43,

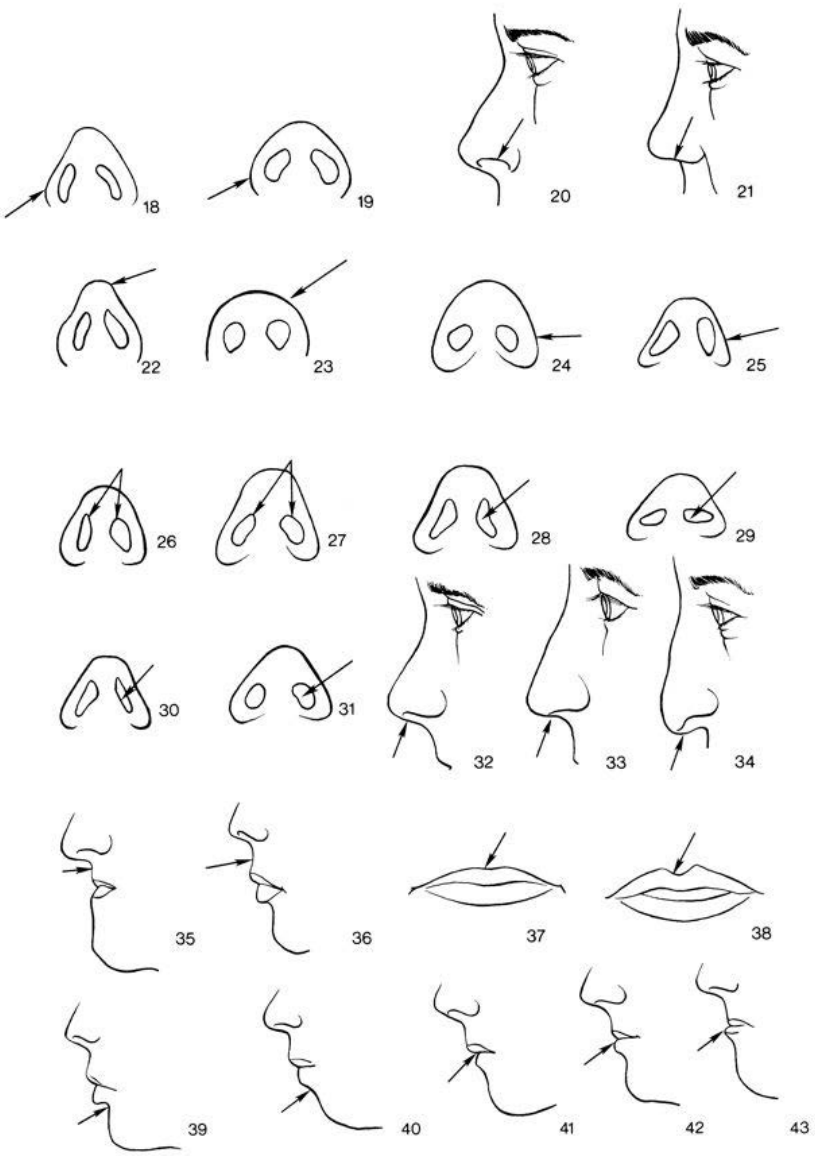


FIGURE 8-15.
(Modified from Hulanicka, B.: Nadbitka Z Nru 86, Materialow Pracantropologicznych Wroclaw, 115, 1973, with permission.)

the lower lip protrusive; 44, a pointed mandible; 45, a squared mandible; 46, no chin cleft; 47, a bifid chin; 48, a retrusive mandible (and chin); 49, a prominent chin; 50, slight rolling of the upper border of ear helix; 51, pronounced helix rolling; 52, a flat, shallow ear scapha; 53, a pronounced, deep groove below the scapha; 54, slight rolling of the middle part of the helix; 55, pronounced middle helix rolling; 56, a short, low crus; 57, a prominent, long crus; 58, a dangling ear lobe; 59, an ear lobe fused with facial skin; 60, slight ear protrusion; 61, marked ear protrusion; 62, a diamond-shaped face; 63, a long, narrow face; 64, a round, short face; 65, an oval face; 66, a square face; 67, an egg haped face. Although the cheekbone is prominent in early childhood, it is nonetheless quite diminutive and fragile, compared with that of the adult. The malar process and the inferior part of the zygoma enlarge considerably during childhood growth, even though they actually remodel in a **backward** direction until definitive arch length is achieved. Because of the differential extents and directions of growth in other parts of the face, these growth increases by the zygoma are often masked. The **protrusive** modes of supraorbital and nasal remodeling and displacement cause the adult forehead and nose to appear progressively more prominent relative to the

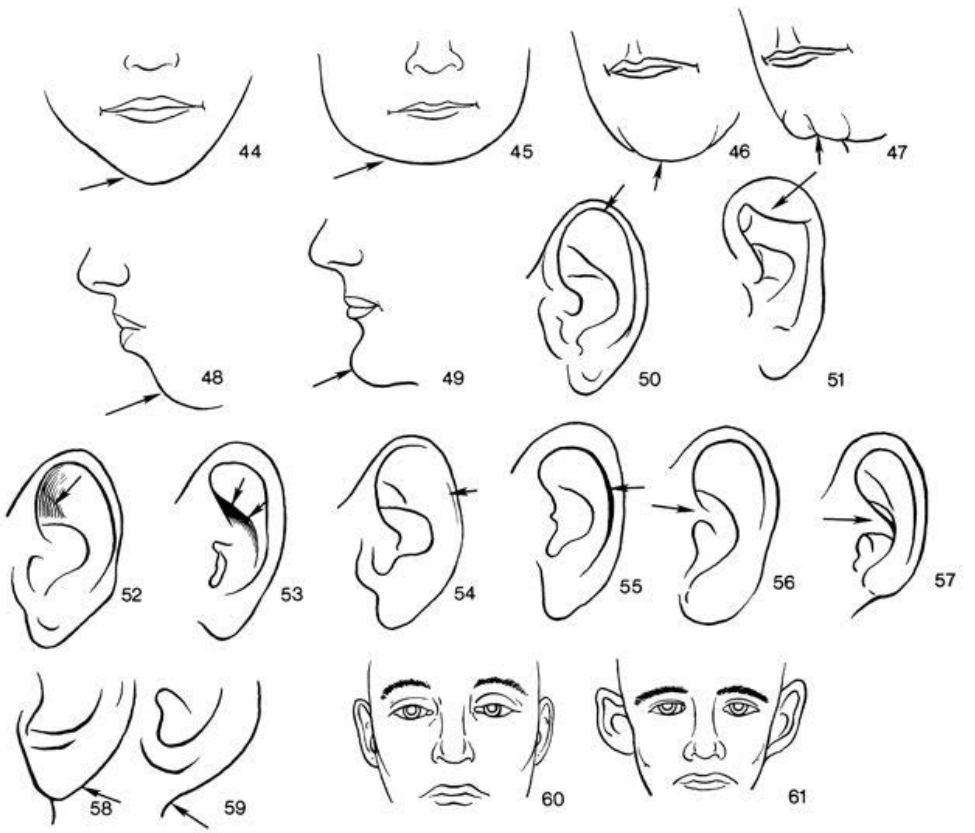
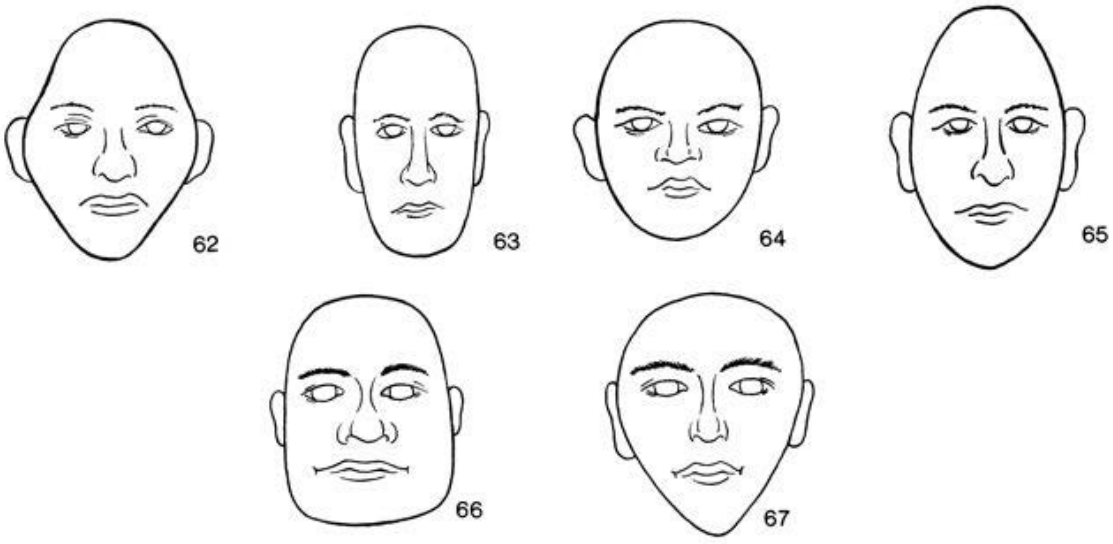


FIGURE 8-16.
(Modified from Hulanicka, B.: Nadbitka Z Nru 86, Materialow 1 Prac antropologicznych Wroclaw, 115, 1973, with permission.)

FIGURE 8-17



retrusively remodeling cheekbones and lateral orbital rims, thus drawing out the depth of the face due to regional developmental divergence in these contiguous facial regions. This feature is more noticeable in the male.

The entire face of the adult is thus much deeper anteroposteriorly, and the whole face is **drawn out** in many directions. The adult face has much bolder topographic features, and it is much less "flat." As the whole face expands, the frontal, maxillary, and ethmoidal *sinuses* enlarge to occupy spaces not otherwise functionally utilized. Architecturally, the sinuses are leftover "dead" (unused) spaces (see page 179). They were not created especially to provide "resonance to the voice," nasal drip, air warming, or other special functions, although they have become secondarily involved in such roles.

The mandible of the young child appears quite small and "underdeveloped" relative to the upper jaw and the face in general. It is small not only in actual size but also proportionately, and it is retrusively placed as well. The child's anterior cranial fossae directly overlie the nasomaxillary complex suspended from them. **Because the anterior cranial fossae are developmentally precocious, the nasomaxillary complex is thereby carried to a more protrusive position than the mandible**, which articulates on the ectocranial side of the middle endocranial fossae located more posteriorly. Much of the basicranial expansion affecting forward nasomaxillary displacement thus does not affect the mandible early on. Only later does the mandible "catch up" as its ramus (together with the attached, growing muscles of mastication) matches or exceeds the development of the overlying, later-growing middle cranial fossae. Because of this, it is sometimes difficult to predict during early childhood possible skeletal malocclusions that might or might not become fully expressed during later development.

The chin is incompletely formed in the infant; indeed, it hardly exists at all. Because of remodeling changes that gradually take place, however, the chin becomes more prominent year by year. A "cleft" is sometimes formed in the **fleshy**

part of the chin (not usually in the bone itself) when the two sides of the lower jaw fuse during early postnatal development. The cleft deepens when the soft tissues of the two sides then continue to expand. For some reason, this facial feature has become adopted in our society as a symbol of masculinity when it is present in the male. Its presence in the female has no social significance one way or the other.

The young child's mandible appears to be pointed. This is because it is wide, short, and more V-shaped. In the adult, the entire lower jaw becomes "squared." With the development of the chin, together with massive growth in the lateral areas of the trihedral eminence, eruption of the permanent dentition, enlargement of each ramus, expansion of the masticatory musculature, and flaring of the gonial regions, the whole lower face takes on a more U-shaped configuration, resulting in a **considerably** more full facial appearance (Fig. 2-25).

In the infant and young child, the gonial region lies well inside (medial to) the cheekbone. In the adult, the posteroinferior corner of the mandible extends laterally out to the cheekbone, or nearly so. This gives the posterior part of the jaw a square appearance.

The ramus of the adult mandible is much longer vertically (Fig. 2-12). It is also more upright (this refers to the ramus as a whole and not to the misleading "gonial angle"). The **sizeable** elongation of the ramus accommodates the massive vertical expansion of the nasal region and the eruption of the deciduous and then the permanent teeth along with masticatory muscle development.

The premaxillary region normally protrudes beyond the mandible in the infant and young child, and it lies in line with or forward of the bony tip of the nose (Fig. 2-12). This gives a prominent appearance to the upper jaw and lip. In subsequent facial development, however, the nose becomes much more protrusive, and the tip of the nasal bone comes to lie well ahead of the basal bone of the premaxilla.

The forward surface of the bony maxillary arch in the infant, with its yet unerupted dentition, has a vertically convex topography. This is in contrast to the characteristically later concave contour of this region in the adult. The alveolar bone in this area of the adult face is noticeably more protrusive and proportionately much more massive (in conjunction with the permanent dentition) (Fig. 2-12).

The whole face, vertically, is a great deal longer and more obliquely sloping as a result of the many changes outlined above (Fig. 5-17).

The quite small mastoid process of the infant later develops into the sizable protuberance of the adult. A bony styloid process is also lacking in the newborn. The ring-shaped bone around the external acoustic meatus faces downward in the infant, but is later rotated during growth into a more vertical position.

At birth, the overall length of the basicranium is approximately 60 to 65 per cent complete, and it increases rapidly. By 5 to 7 years, it reaches about 90 per cent of its full size. Also, about 85 per cent of the adult width of the cranium is attained by the second to third year.

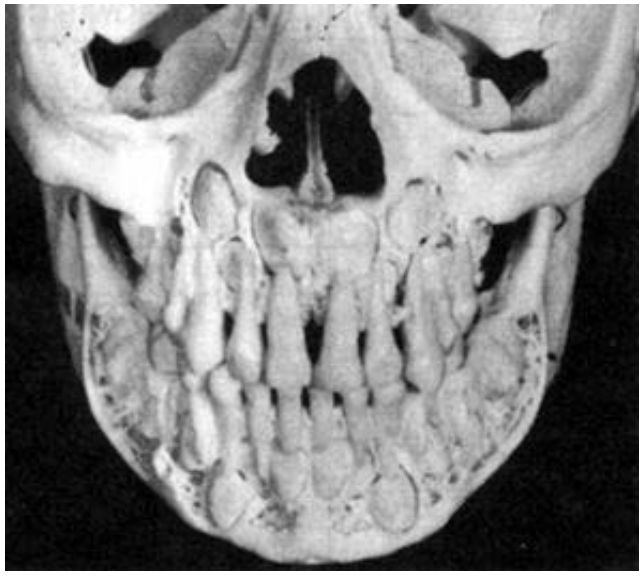
In the newborn, six fontanelles ("soft spots") are present among the bones of the skull roof. They cover over at different times, but all have been reduced to sutures by the eighteenth month. The sutures of the cranial vault are relatively nonjagged in the baby, and the outer surface of the bone is smooth. A much rougher bone texture characterizes the surface of the adult calvaria, and the suture lines

become noticeably much more dentate and interlocking (Fig. 2-12). The metopic suture (separating the right and left halves of the frontal bone) usually fuses by the second year and the premaxillary-maxillary suture is mostly fused by the first to second year, with only a trace sometimes remaining. By the third year, the principal cranial and facial suture systems still intact are the coronal, lambdoidal, and circumaxillary. Subsequent closure then begins around the twenty-fifth to thirtieth year, usually in the sequence of sagittal, coronal, and lambdoidal, with those bounding the temporal bone following. The latter can remain partially open even in the aged skull. Traces of the facial sutures can often remain through advanced old age.

In the child, the slender neck below a relatively large cranium gives a characteristic "boyish" appearance to the whole head. This gradually disappears until about puberty, when the expansion of the neck muscles and other soft tissues causes a proportionate decrease in the prominence of the head relative to enlarging neck circumference. This is less noticeable in the female.

The external appearance of the baby's face does not reveal the truly striking enormity of the dental battery developing within it (Fig. 8-18). The teeth are a dominant part of the infant's face as a whole, yet they are not even seen. The parent does not usually realize they are already even there at all, much less suspect the massiveness of their extent. In this illustration, one is almost overwhelmed by the remarkable extent of **teeth** all over the midfacial region. The average person does not appreciate that the mouth of the little child is bounded by a virtual palisade of multitiered primary and permanent teeth in many stages of development. When a crown tip first protrudes through the gingiva as it erupts, the parent naturally

FIGURE 8-18



believes that the process is just beginning, and that the welcome new tooth is only a tiny but newsworthy addition to the pink mouth. It is not realized that the whole midface is occupied by a vast magazine of unerupted teeth hidden to the eyes. The thin covering and supporting **bone** of the jaws is a much less commanding feature of the young face.

Facial Variations

The spectrum of topographic facial variations is virtually a whole field unto itself and most interesting indeed. As pointed out earlier, relatively small features can have demanding impact on the character of a person's face. A catalog of some of these features commonly encountered is pictured in Figures 8-14 to 8-17. The spread of combinations is endless.

9

The Plan of the Human Face

The human face is certainly different from that of other mammals. The long, narrow, functional muzzle that slopes gracefully onto the streamlined cranium of a typical mammal is in marked contrast to the muzzleless, broad, vertical, flattened human face, enveloped by an enormous balloon-shaped cranium with a bulbous forehead overhanging tiny, retrusive jaws, a small mouth, a chin, and the curious vestige of a narrow fleshy snout with an owl-eyed and wide face showing changing expressions. Although somehow beautiful to our eyes, this has to be, in the extreme, an "odd" design by ordinary mammalian standards.

Our upright posture involves a great many anatomic and functional adaptations throughout every part of the body, and no one of these would work without all the others. We have "feet," and the human foot stands by itself, as it were, as a unique human anatomic feature. The designs of the toes, foot bones, arch of the foot, ankle, leg bones, pelvis, and vertebral column all interrelate in the anatomic composite that provides upright body stance. The head is in a balanced position on an upright spine. The arms and hands have become freed. The manipulation of food and other objects, defense, offense, and so forth, utilize primarily the hands, rather than the jaws.

The enormous enlargement and the resultant configuration of the brain have caused a "flexure" (bending) of the human basicranium (Fig. 9-1). This relates to two key features. First, the spinal cord is now aligned vertically, a change that permits upright, bipedal body stance allowing free arms and hands. Second, the orbits have undergone a rotation in conjunction with frontal cerebral lobe expansion. This aligns them so that they point in the forward direction of upright (bipedal) body movement. The body has become vertical, but the neutral visual axis is thereby still horizontal, as in other mammals, which is the functional position. (Note: The muzzle of a typical animal points obliquely downward in its "neutral" position, not straight forward. This aligns the orbital axis approximately parallel with the ground and toward the direction of body movement. The cranial base of the typical mammal is flat, in contrast to the flexed human cranium, and the spinal cord passes into a horizontally directed vertebral column.)

Which particular anatomic or functional change "came first" in this evolutionary chain has long been argued. Upright stance? Brachiation? Enlarged brain? Downward-rotated and decreasingly prognathic dental arches and jaws? Basicranial flexure? Development of hands and binocular vision? An important concept, however, is that the multitude of these changes are all functionally interrelated. They have developed and evolved as a phylogenetic "package,"

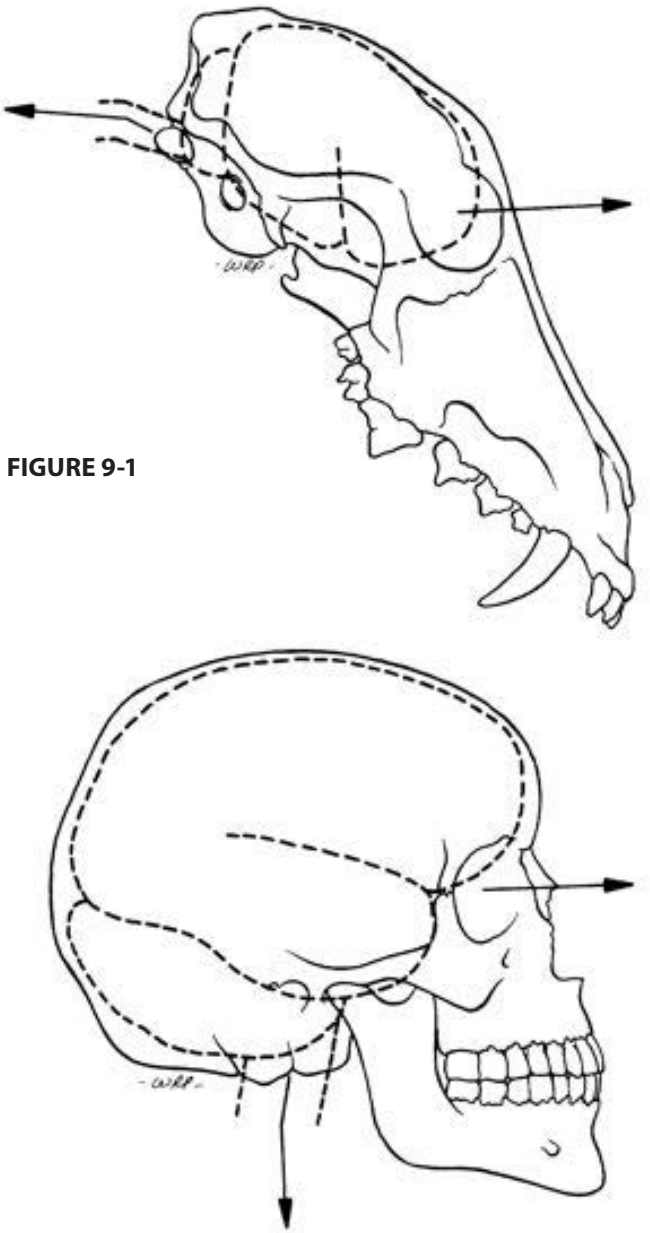


FIGURE 9-1

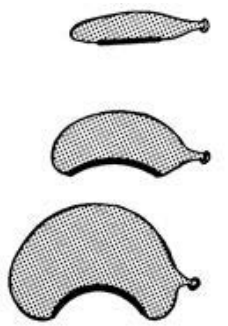
regardless of which one (or combination) actually led off as a primary step in evolution.

Although the human face is topographically "different" from the faces of other mammals, no special violations of the general mammalian plan for facial construction seem to have occurred. The face of man conforms to the same basic morphologic and morphogenetic rules complied with by most mammals in general. Differences have to do mostly with proportionate sizes of component parts and their rotational placements as related to body stance, head posture, and brain size and configuration, but not with any basic departures from the standard morphologic guidelines.

BRAIN ENLARGEMENT, BASICRANIAL FLEXURE, AND FACIAL ROTATIONS

If a short piece of adhesive tape is affixed to a rubber balloon and the balloon then inflated, it will expand in a curved manner (Fig. 9-2). The balloon bends because it enlarges around the nonexpanding basal segment. The enormous human cerebrum similarly expands around a much smaller and lesser-enlarging midventral segment (the medulla, pons, hypothalamus, diencephalon, optic chiasma). This causes a bending of the whole underside of the brain. The **flexure** of the basicranium results. The foramen magnum in the typical mammalian skull is located at the posterior aspect of the cranium (Fig. 9-3). In man, it is in the midventral part of the expanded cranial floor at an approximate mechanical balance point for upright head support on a vertical spine (Fig. 9-4).

FIGURE 9-2



The expansion of the frontal cerebral lobes displaces the frontal bone upward and outward (Figs. 9-3 and 9-4). This results in the distinctive, bulbous, upright "forehead" of the human face, although it is really part of the neurocranium and not the face (viscero—or sphlanchno—cranium) proper. The frontal lobes also relate to a developmental rotation of the human orbits into new positions. As the forehead becomes developmentally rotated into a vertical plane by the enlarging brain behind it, the superior orbital rims are carried with it. The eyes now point at a right angle to the spinal cord. The spine is vertical, but the human orbital axis is still thus horizontal. Vision is directed toward forward body movement.*

The expansion of the frontal and, particularly, the temporal lobes of the cerebrum also, importantly, underlies a rotation of the orbits toward the midline (Figs. 9-5, 9-6, and 9-7). The eyes are moved closer together. **Two** separate axes of orbital rotation are thus associated with the massive expansion of the cerebrum. One displaces the orbits vertically, and the other carries them horizontally in medial directions into a full binocular position. Different extents of these two separate rotational movements are seen among different primate species. In the monkey, for example, the extent of upright orbital alignment and frontal bossing is much less than in man, as determined by the relative sizes of their frontal lobes.

* In some anthropoids, such as the gorilla, the massive supraorbital ridges may also rotate vertically independent of the frontal lobe. In the human face, however, the orbits **must** rotate into a vertical alignment because of the expanded size of the frontal lobes.

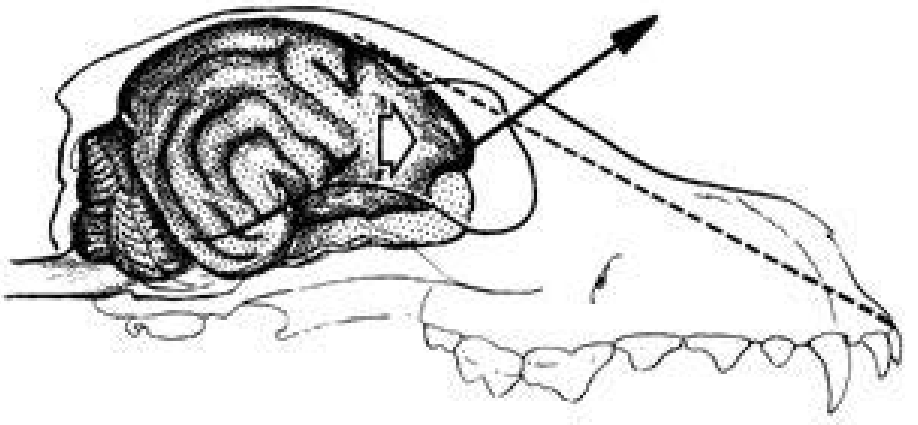


FIGURE 9-3.
(From Enlow, D. H., and J. McNamara: The neurocranial basis for facial form and pattern. Angle Orthod., 43:256, 1973, with permission.)

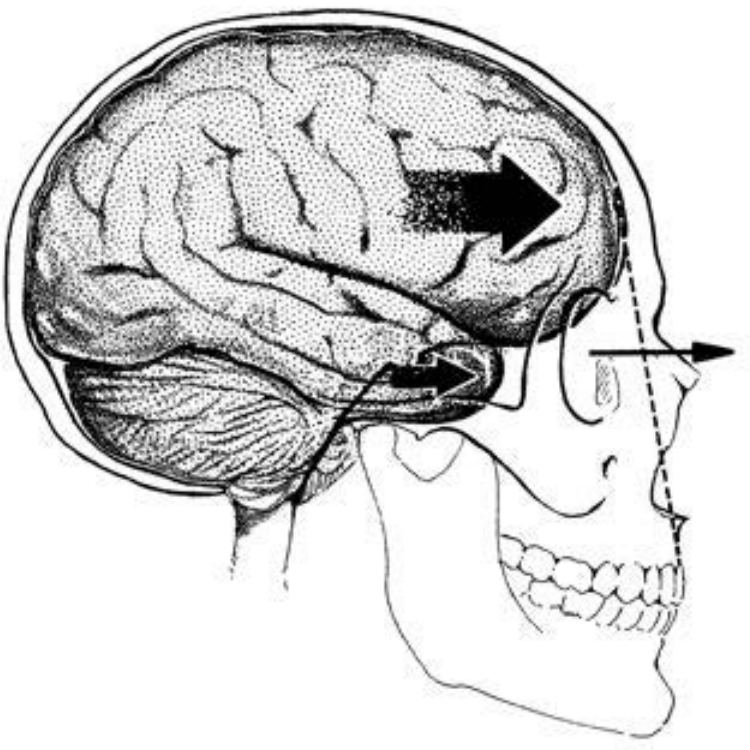


FIGURE 9-4.
(From Enlow, D. H., and J. McNamara: The neurocranial basis for facial form and pattern. Angle Orthod., 43:256, 1973, with permission.)

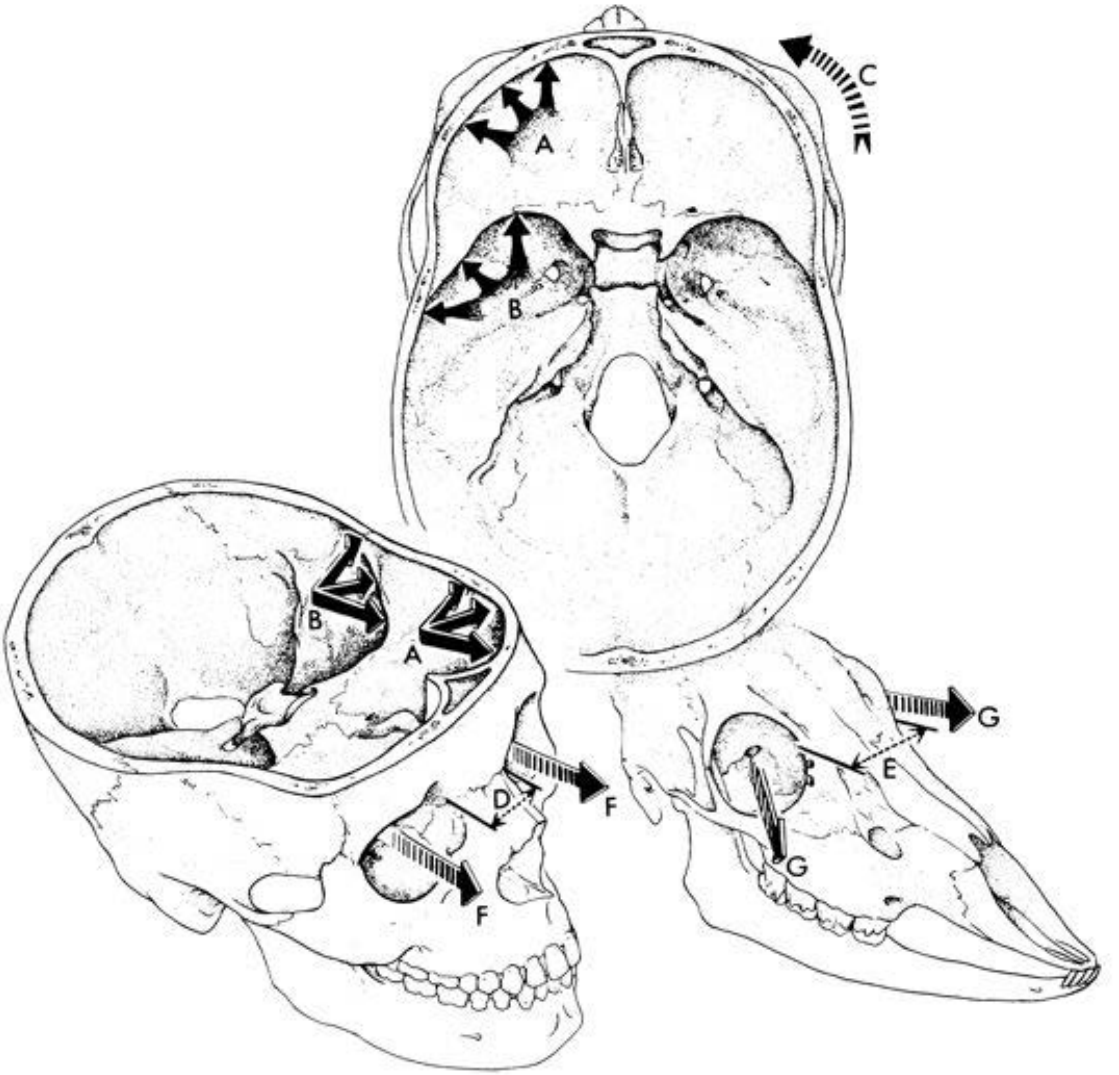


FIGURE 9-5.
Facial features related to enlarged human brain and bipedal posture compared to a smaller-brained mammal. The enlarged frontal lobes and anterior endocranial fossae (A), together with the large temporal lobes and middle cranial fossae (B), produce a wide, flat face with squared cheekbones (C), orbital rotation toward the midline, a reduced proportionate transverse size of the airway and nasal base (D), forward-pointing orbits (F), and a rotational placement of the facial composite beneath the cranial floor. In a typical mammal (deer), note the divergent orbital axes (G), the proportionately larger interorbital and nasal space (E), the more narrow and angular face, and the more protrusive snout and muzzle projecting forward rather than beneath the anterior cranial floor. (From Enlow. D. H.: *The Human Face*. New York, Harper & Row, 1968, p. 190, with permission.)

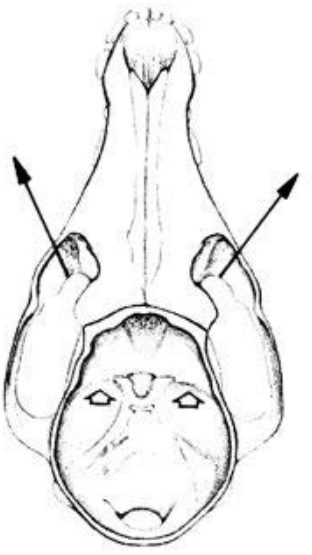


FIGURE 9-6.
(From Enlow, D. H., and J. McNamara: The neurocranial basis for facial form and pattern. Angle Orthod., 43:256, 1973, with permission.)

The simian orbits are quite close-set, however, and this relates to the proportionate sizes of the temporal lobes.

The binocular arrangement of the orbits is a feature complementing finger-controlled manipulation of food, tools, weapons, and so forth. The absence of a long, protrusive muzzle allows close-up vision of hand-held objects. The human **mind** directs the **free hands** that can work with **three-dimensional** perspective in an **upright** stance on feet. The enormous size of the human brain and the human basicranial flexure are key factors, but **all** these changes are required for full human expression, and they are all mutually interdependent developmentally as well as functionally.

Orbital rotation toward the midline, importantly, significantly reduces the dimension of the interorbital space (Figs. 9-7 and 9-8). This is one of two basic factors that underlie reduction in the extent of snout protrusion in man and some other (but not all) primates. Because the interorbital segment is the root of the nasal region, a decrease in this dimension reduces the structural (and also the physiologic) base of the bony nose. A wide nasal base can support a proportionately longer snout. A narrow nasal base, however, reduces the architectural limit to which the bony part of the nose can protrude, and the snout is thereby shorter. The second basic factor involved in the extent of reduction of nasal protrusion deals with the rotation of the olfactory bulbs (see below).

The olfactory sense in *Homo* has become a much less dominant factor in environmental awareness and is far exceeded by many other mammalian groups. In addition to proportionate downsizing of the human nasal and olfactory mucosae, olfactory receptors in the frontal sinuses are also lacking, which is in contrast to other forms more dependent on aromatic sensations for food-getting or protection.

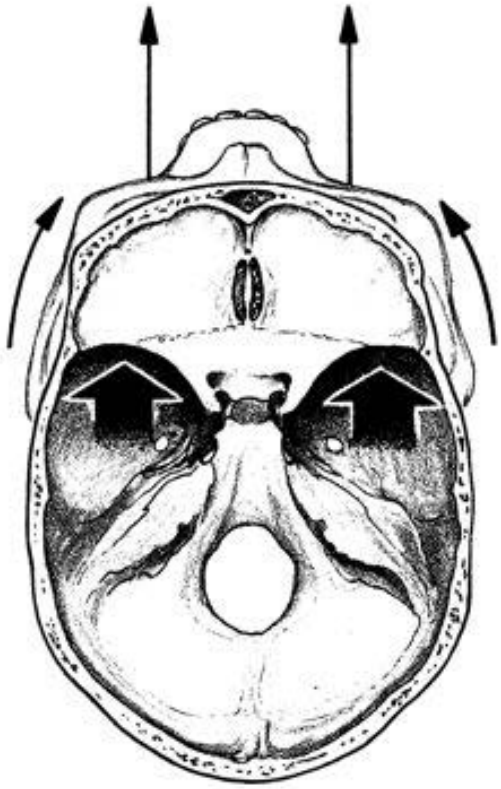
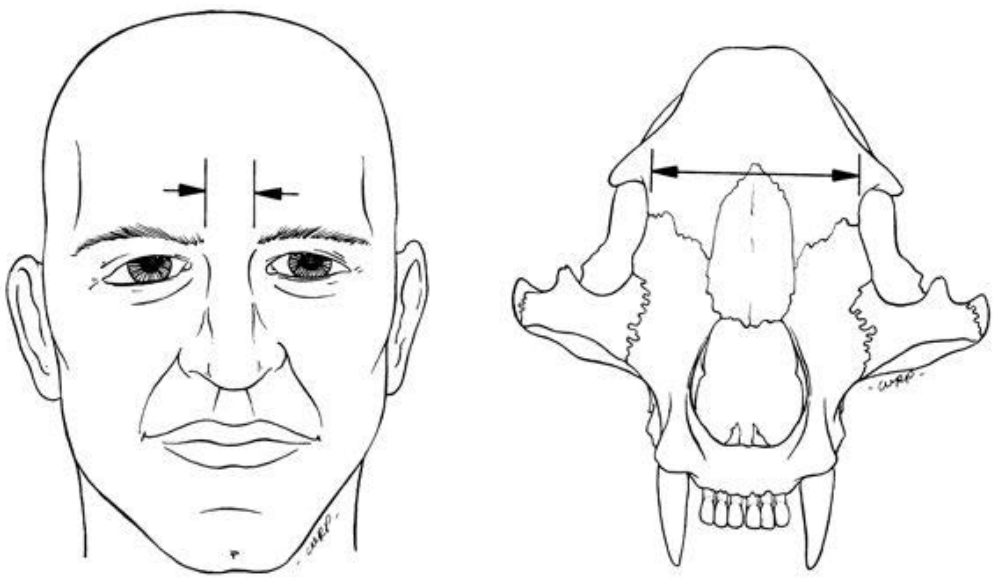


FIGURE 9-7.
(From Enlow, D. H., and J. McNamara: The neurocranial basis for facial form and pattern. Angle Orthod., 43:256, 1973, with permission.)

FIGURE 9-8



The nasal region above and the oral region below are two sides of the same coin, that is, the palate (Fig. 9-9). Reduction in nasal protrusion is accompanied by a more or less equivalent reduction of the upper jaw. The whole face in any mammal necessarily becomes reduced in length as a result. However, the human face has also been **rotated** into a nearly **vertical** alignment related to the massive enlargement of the brain and flexure of the basicranium. The downward rotation of the **olfactory bulbs** and the whole **anterior cranial floor** by the enlarged frontal lobes of the cerebrum has caused a corresponding downward rotation of the nasomaxillary complex (Fig. 9-10).

The nasal mucosa is ordinarily an active tissue involved in temperature regulation in most mammals. Vasoconstriction and vasodilation of the vessels in the massive mucosal spread covering the turbinates control the amount of heat retention or loss. Because of marked nasal reduction in man, however, this important function has been largely taken over by the relatively hairless and sweat gland-loaded human integument. Control of blood flow in the dermis, combined with sweat gland activity, provides the equivalent for nasal thermoregulation. This is possible in man (and in a very few other species, such as the pig) because of a near-naked skin. In thick-furred animals, thermoregulation is carried out by regulating heat transfers in the nasal mucosa, panting control to release or conserve body

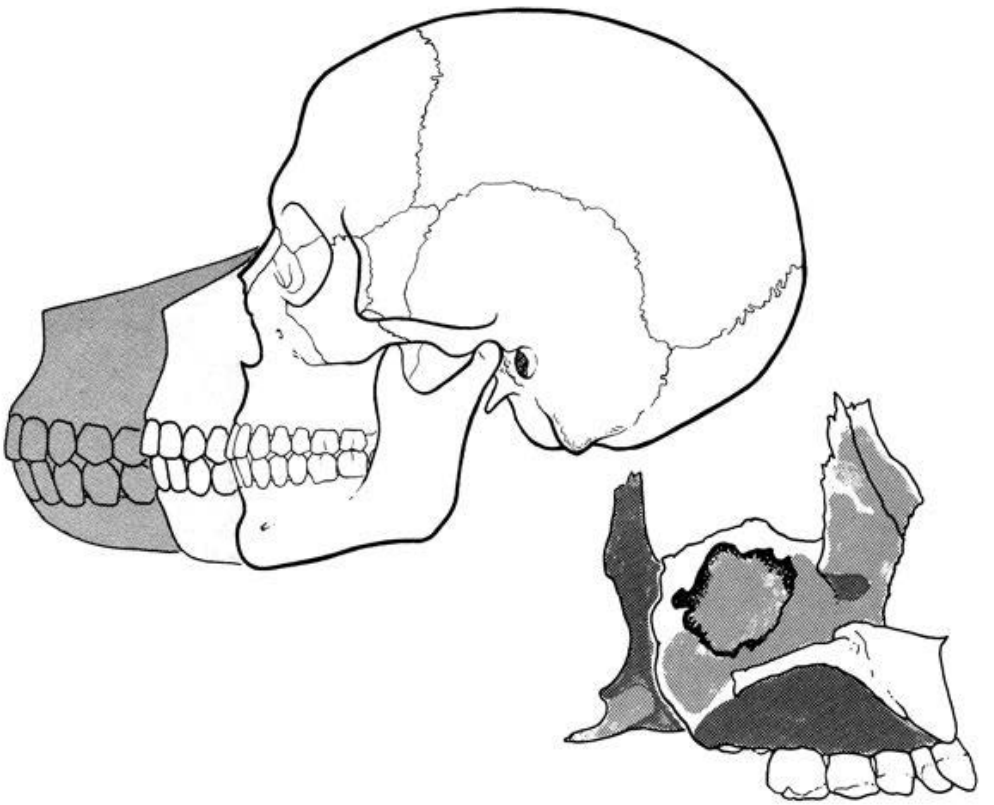


FIGURE 9-9.
(Lower figure from Enlow, D. H., and S. Bang: Growth and remodeling of the human maxilla. Am. J. Orthod. 51:446, 1965, with permission.)

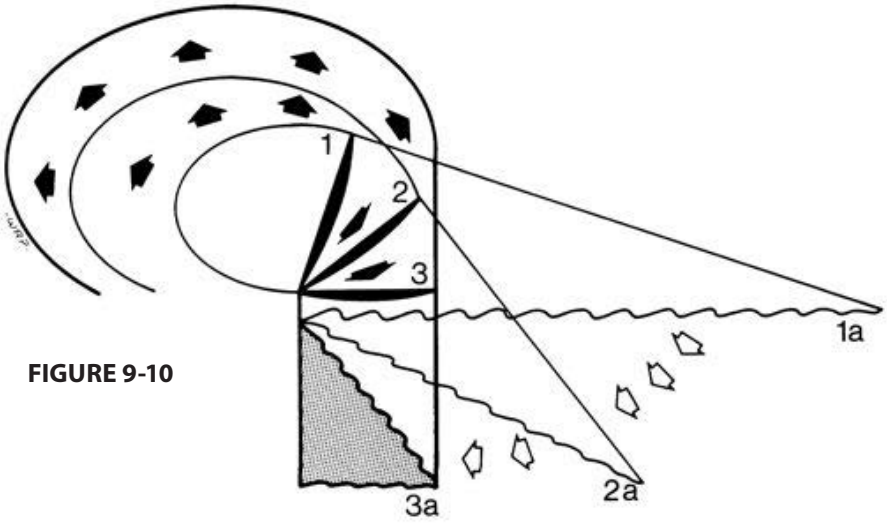


FIGURE 9-10

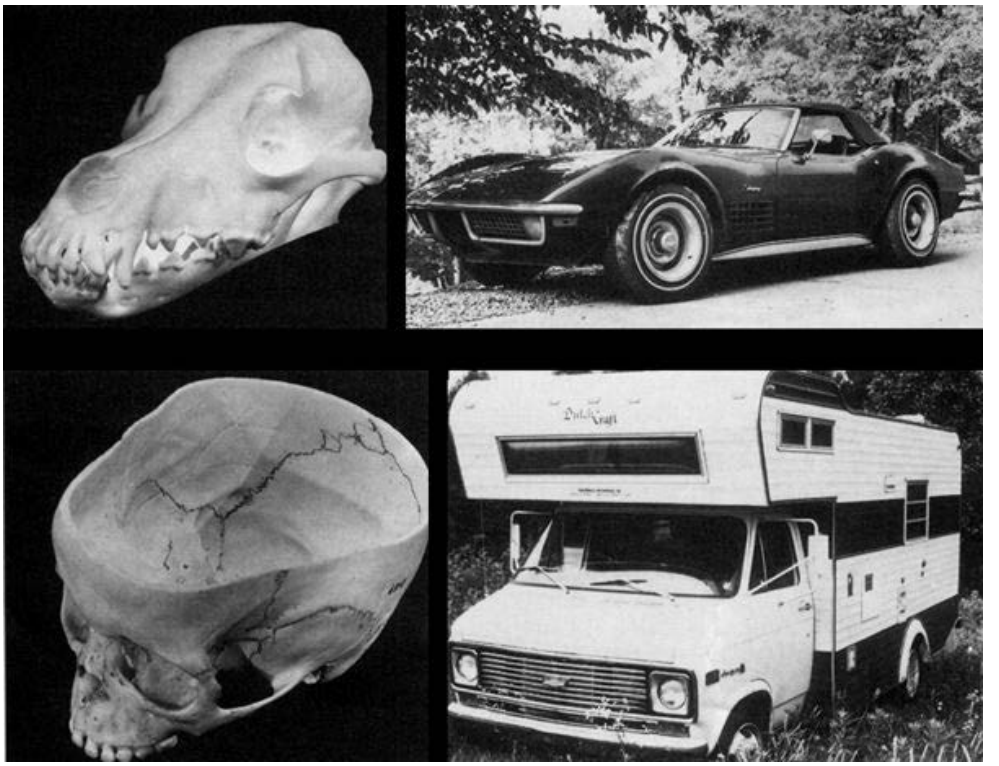
heat, limited perspiration in hairless areas (such as pads of the paws), and a fluffing of the fur to increase dead air insulation. The latter also makes the animal look menacingly larger to prospective enemies, and it increases the nonvital part of his anatomy for them to bite. We have only the atavistic holdover: goose bumps.

All mammalian forms have bony reinforcement "pillars" built into the architectonic design of the craniofacial complex. These pillars are parts of bones that provide a buttress for structural support and biomechanical stress resistance that balances the physical properties of the skull against the composite of forces acting within it. This includes the forces of growth itself. Although customarily described with reference to tooth positions, the nature of support goes well beyond just accommodation to masticatory forces. In the human face, one of these pillars is the "key ridge," which is a vertical column of thickened maxillary bone approximately centered above the functionally important area around the upper first molar. The mechanical support column then continues upward from this ridge into and through the lateral orbital rim and on to the supraorbital-reinforced frontal bone. The second maxillary molar is reinforced by a vertical sheet of bone, the posterolateral orbital wall, which extends directly above this tooth. Except for the very thin bone enclosing the posterior part of the large maxillary sinus, the third molar is situated behind the orbit, and it has no further bony support above this. It has thus become effectively disfranchised mechanically and phylogenetically. The incisors are supported by an arch, the bony rim of the overlying nasal opening, with which it shares a common embryonic development, and also by the vertical nasal septum. Each canine tooth is reinforced by a marked thickening of the lateral nasal wall, toward which the cuspid root points, and thence on to and through the thickened frontal process of the maxilla into the glabellar thickening of the forehead.

The human face is exceptionally wide because the brain and cranial floor are wide. However, the face has been almost engulfed by the massive brain behind and above it (Fig. 9-11). Note the colossal size of the human cranium, in comparison with that of the typical mammal. The expanded frontal lobes of the human brain are located **above** the eyes (and above almost the whole remainder of the face)

rather than being located **behind** the eyes, and a big forehead has thus been added. This also underlies rotation of the orbits into vertical, forward-facing positions as well as to the rotation of the face as a whole into a unique downward-backward position.

FIGURE 9-11



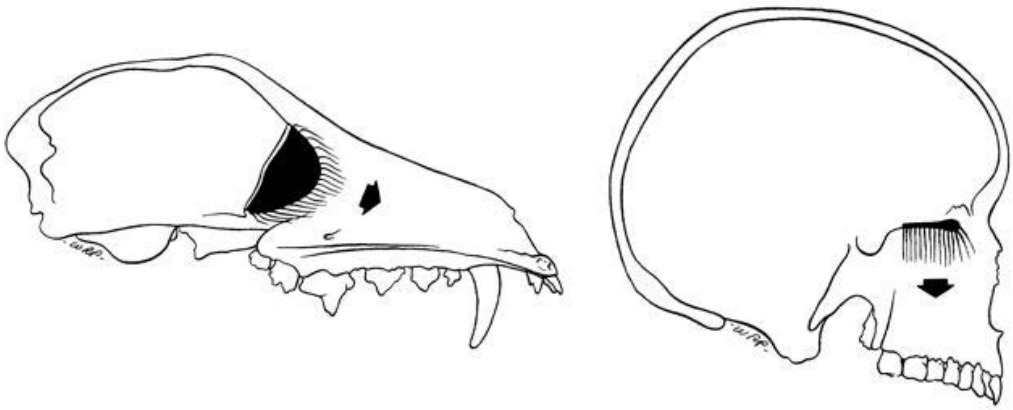
Herbivores have orbits widely set to each facial side, thus providing a much greater peripheral range of vision to detect some approaching carnivore. The carnivore, on the other hand, can rotate its eyeballs into more forward-looking positions, even though the orbits are aligned obliquely laterally, thus favoring a stereoscopic chase. Carnivores generally tend to have a much shorter muzzle and snout because of the greater degree of medial rotation of the orbits, producing a narrower interorbital nasal base. Herbivores generally tend to have greater snout and muzzle protrusion, because their orbits are much more wide-set, with a broader interorbital nasal root.

Note that the enlarged human cerebrum has caused a downward rotational displacement of the olfactory bulbs (Fig. 9-12). In all other mammals, the bulbs and cribriform plates are nearly upright or obliquely aligned, depending on the size and configuration of the frontal lobes. In man, the bulbs have been rotated into **horizontal** positions by the cerebrum. This is a significant factor in the basic design of the human face.

The olfactory bulbs relate directly to the alignment and the direction of growth of the adjacent nasal region (Fig. 9-12). The long axis of the snout in most

mammals is constructed so that it necessarily points in the general direction of the sensory olfactory nerves within it. The plane of the naso-maxillary region is thereby approximately **perpendicular to the plane of the olfactory bulbs**. This is a major anatomic and functional relationship that underlies the direction of nasomaxillary development in the face of virtually any mammal. As the bulbs in the human brain became rotated progressively from a vertical position to horizontal because of increases in brain size or because of its shape (*1, 2*, and *3* in Fig. 9-10), the whole face has been similarly rotated from a horizontal to a vertically downward plane (*1a, 2a*, and *3a*). Or, stated another way, the face has become rotated down by the expanded anterior cranial floor as the floor rotates downward as a result of the enlargement of the frontal lobes.

FIGURE 9-12



Nasomaxillary Configuration

The maxilla of most mammals has a triangular configuration. In man, it is uniquely rectangular (Fig. 9-10). This was caused by a rotation of the occlusion into a horizontal plane adapting to the vertical rotation of the whole midface. The occlusal plane in most mammals, including man, is approximately parallel to the Frankfort plane (a plane from the top of the auditory meatus to the inferior rim of the orbit). This aligns the jaws in a functional position relative to the visual, olfactory, and hearing senses. In the human maxilla, the design change that allowed for this resulted in the creation of a new arch-positioning facial region, the unique **suborbital** compartment. Most of this phylogenetically expanded area is occupied by the otherwise nonfunctional maxillary sinus (uses such as air warming, nasal drip, and voice resonance are secondary). No prior genetic or epigenetic program for this unprecedented new area existed, no new real function caused a new tissue or organ invention, and the result, therefore, is nothing i.e. a space. An **orbital floor** developed in conjunction with this added facial region to provide for orbital soft tissue support. This is a special feature relating to the new maxillary configuration. The middle and lower parts of the face now lie **beneath the eyes** rather than in front of them. Compare also with Figure 9-5.

The nasal region is thus **vertically** disposed in the human face (Fig. 9-12). The neutral axis of the spread of the sensory olfactory nerves is vertical, and the resultant vertical vector of nasomaxillary growth has become a major feature of

human facial development. The characteristic vertical human facial profile is a composite result of (1) a bulbous forehead, (2) rotation of the whole nasal region into essentially a vertical plane, (3) reduction of snout protrusion in conjunction with medial orbital convergence, (4) rotation of the orbits into upright positions, (5) rotation of the maxillary arch downward and backward, and squaring of the nasomaxillary complex, (7) leveling of the horizontal palate and maxillary arch, (8) creation of the maxillary sinuses, (9) addition of an orbital floor and lateral orbital wall, and (10) bimaxillary reduction in the extent of prognathism matching nasal reduction. The face also became markedly widened because of the increased breadth of the brain and cranial floor and because the orbits and cheekbones are rotated into forward-facing positions. The face of man now lies **beneath** the frontal lobes of the brain; in other mammals the face is largely in front of the cerebrum. The nasal chambers are housed largely **within** the face, between and below the orbits, rather than projecting forward within a protrusive muzzle. The projecting human snout itself houses very little of the mucosal part of the nasal chambers back within the face. The whole face has been "reduced" to a quite flat topographic configuration as a combined result of these multiple alterations.

Reduction of the nasal region associated with orbital convergence and olfactory and anterior cranial fossa rotation must necessarily also be accompanied by a more or less equal reduction in maxillary arch length, as pointed out above. Only a relatively slight degree of horizontal divergence between the two can exist. If either one becomes reduced in length, so must the other. This refers only to the bony part of the nasal region; some species have a fleshy proboscis protruding beyond the jaws and palate (such as man and the elephant).

Why does the human face have an overhanging, fleshy "nose"? The protrusion of the cartilaginous and soft tissue portion of the nasal complex has a design alignment that provides for **downward**-directed external nares (Fig. 9-13). It serves to aim the inflow of air obliquely upward toward sensory nerve endings into the olfactory bulbs located in the **ceiling** of the nasal chambers. This is in contrast to the external nasal apertures of other mammals taking air into more horizontal nasal chambers having the cribriform plates and nerve endings located within the posterior wall. Thus, the human face has a fleshy, protuberant "nose" as a functional adaptation to the unique vertical disposition of its nasomaxillary complex.

The rotation of the whole face downward and **backward** has resulted in a facial placement within the recess (the "facial pocket") created by the basicranial flexure. What will happen if the brain **continues** to enlarge phylogenetically and thereby produce an even further extent of backward rotation? There is virtually no more room remaining to "rotate into," at least given the present design and arrangement of all the various soft tissue and skeletal parts involved. Already the face has almost reached the airway because of rotation. The posterior cranial floor, the vertebral column, and the face, as in a closing vise, are all coming together; there are important parts in between. What phylogenetic facial adjustments have occurred? See page 87 for an evaluation. (The porpoise skull shows how evolutionary craniofacial adaptations also involving an enlarged brain have taken place in yet another way that is unconventional among mammals.)

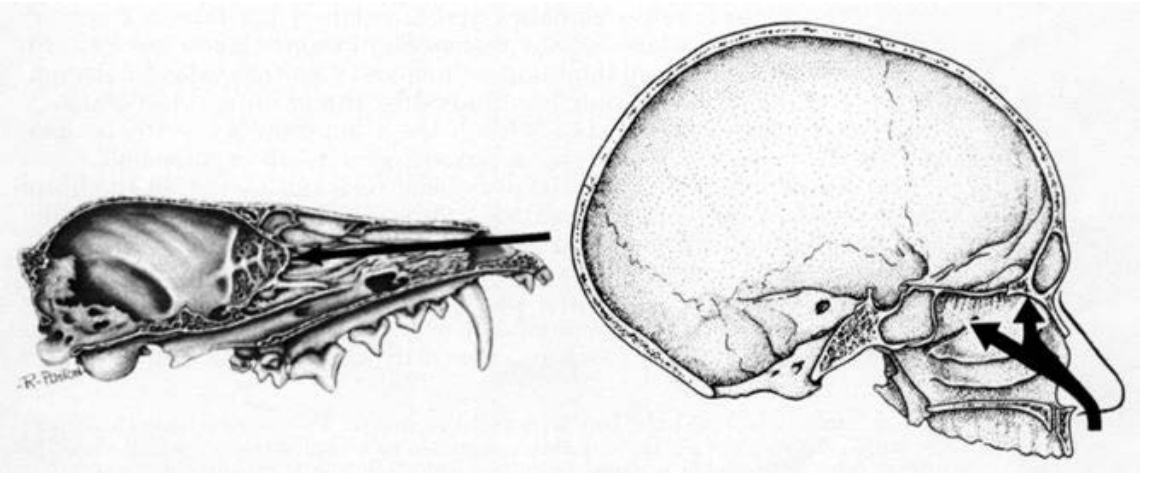


FIGURE 9-13.
(Figure at right from Enlow, D. H.: *The Human Face*. New York, Harper & Row, 1968, p. 188, with permission.)

Growth Field Boundaries

The development of each part of the face involves two basic considerations. The first is the **amount** of growth, and the second is the **direction** of growth. These two factors constitute the growth "vector."

Growth proceeds according to a mosaic of regional developmental "fields." Each field as well as groups of fields have prescribed boundaries. These boundaries establish regional growth perimeters, and there is a maximum and minimum growth capacity within each. Growth does not ordinarily exceed each perimeter if a developmental equilibrium is to be sustained. However, clinical manipulation of the growing face can result in violation of these maximum and minimum boundaries. The consequences of such violations are either physiologic rebound or pathologic degeneration. Physiologic rebound occurs when the natural forces of the boundary reclaim their territory. An example of this type of rebound is the lingual collapse of lower cuspids that have been pushed laterally, past the equilibrium boundary of the modiolus and orbicularis oris musculature. An example of pathologic degeneration is encountered when a retainer is used to hold maxillary molars outside the physiologic boundary. Instead of lingual collapse, fenestration of the root surface and breakdown of periodontal supporting structures occurs. Since neither physiologic rebound nor pathologic degeneration is a desirable outcome, the goal of dentofacial therapeutic procedures must be to improve esthetics and function of the face in harmony with the growth boundaries of the region. Significantly, there are forward, downward, backward, and lateral growth boundaries that exist for the major parts of the face, and they are **shared** by the brain and basicranium. The perimeters for the regional fields related to brain growth and the perimeters for the major facial regional fields have become established in common.

The reason for this basicranial and facial relationship is that the brain has evolved in conjunction with the cranial floor. The form, size, endocranial topographic features, and angular characteristics of one conform to the other. The floor of the cranium, in turn, is the **template** upon which the face is built. The junctional part of the face cannot be significantly **wider**, for example, than the maximal width of the cranium. There would be nothing to which it could be attached.† Similarly, the **length** and **height** of specific parts of the cranial floor are projected as equivalent dimensions for the face, as described later.

The face is structurally or developmentally dependent on the basicranium.‡ This is an important concept, because a number of normal and abnormal variations in facial form relate, at least in part, to underlying circumstances present in the cranial floor (see Chapter 10).

The floor of the cranium has developed in **phylogenetic** association with the brain. Whatever "independent" genetic control actually exists in the basicranium itself (this is historically controversial), the shape and size of the floor of the cranium have become established because its genes were especially adopted by the natural selection process to accommodate an **interdependent** association with the developing brain. Thus established, the basicranium presumably has a measure of genetic independence, since it can continue to partially develop even though the overlying brain atrophies or is removed. The face similarly develops **in conjunction with** the cranial floor (and the brain), and the control of facial development has become established in a way that accommodates its functional association with the neurocranium. In all areas, however, a developmental **latitude** exists that provides potential for **adjustments** during growth to accommodate developmental variations in one part or another. This is involved in the operation of the "functional matrix," and it is the factor that allows for a unified coexistence of the many separate, developing parts all growing and functioning in relation to one another. There are regional differences, however, in the **capacity** for such developmental adjustment. Some areas, such as alveolar bone and the tooth sockets, are extremely labile and responsive to variable and changing circumstances. Other areas, such as the basicranium, are much less sensitive and adaptable. The "intrinsic programming" for the latter is presumed to be greater than for the former because of their different levels of developmental independence and whatever the different factors are that determine and control them. The basicranium is, nonetheless, developmentally responsive and able to adjust to extrinsic factors. Head position related to sleeping habits, for example, can have a marked effect on basicranial configuration which, in turn, impacts directly on facial form and pattern. (See Chapter 8.)

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† Hence, there is a physiologic limit to midfacial expansion. The theoretic lateral maximum lies within the width of the basicranium, or between pace-setting cranial nerves placed by it, from which the midfacial complex is suspended. Clinically, it should be expanded past the boundary and then allowed to rebound ("develop") to the practical if not theoretical limit for jaw width.

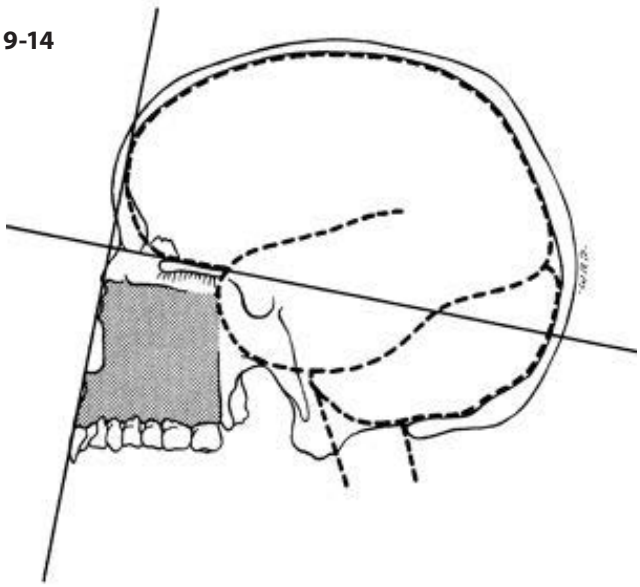
‡ The whole face has often been described, in years past, as a genetically and developmentally separate region, the only tie between them being that they happen to be placed in juxtaposition, as a picture hangs on a wall. No cause-and-effect relationships were believed by some earlier workers to exist between the neurocranium and the size or shape of the face. This is certainly not the case.

While the endocranial side of the cranial floor is adapted to the configuration and topographic contours of the ventral surface of the brain, the topography of the ectocranial side of the basicranium is structurally adapted to the composite of the facial, pharyngeal, and cervical components. A measure of morphogenic divergence thus occurs between these contralateral sides of the cranial floor.

The forward **boundary** of the brain is shared by the forward border of the nasomaxillary complex. The **course** of growth by the nasal part of the face relates, appropriately, to the olfactory bulbs and the sensory olfactory nerves. These two factors underlie the "vector" of midfacial growth, that is, the amount and the direction. To show this, a line is drawn from the forward edge of the brain down to the anterior-most, inferior-most point of the nasomaxillary complex (superior prosthion; Figs. 9-14 and 9-15). This represents the **midfacial** plane.§ Note that the midfacial plane is perpendicular to the **olfactory bulb** (or the cribriform plate, as seen in lateral headfilms). In the human face, this plane also frequently touches the anterior nasal spine. The development of the long axis of the nasal region thus proceeds in the same general direction as the neutral axis of its sensory nerve spread. The amount of growth is established by the prescribed perimeter of its growth field. **The nasomaxillary complex develops within a growth field out to the edge of the brain and its basicranium in a direction perpendicular to the olfactory bulbs.**

§ "Nasion" is often used in cephalometric studies as a point for drawing the facial plane, but this can give misleading results because nasion is so variable in relation to distinct male/female and headform differences, which are seldom taken into account. Moreover, the purpose of the **midfacial** plane described above is to show the relationship between the **brain** and the **nasomaxillary complex**. Thus, the edge of the brain is used, rather than nasion. Also, the above descriptions presume that the alignment of the nerves is the lead factor that determines the direction of midfacial growth. Of course, it may be the converse. Whichever, the important point is that they are established **together** in a constant relationship to the olfactory bulbs, which, in turn, are placed according to the size and shape of the brain.

FIGURE 9-14



The olfactory bulb and nasomaxillary alignment relationship exists among mammals in general. In species or groups having a smaller brain and, as a result, a more upright olfactory bulb, the snout and muzzle tend to be correspondingly more horizontal and much more protrusive (Fig. 9-15). As the olfactory bulbs become rotated downward in different mammalian groups because of increasing brain size (or shape, as in more round-headed species such as the bulldog), the muzzle correspondingly rotates down with them and becomes less protrusive. In the human situation, the olfactory bulbs have become virtually horizontal because of the massive growth of the frontal lobes. The nasal part of the human face thus becomes **vertically** aligned during development in conjunction with the neutral vertical axis of olfactory nerve distribution. (Figs. 9-12 and 9-14). This is a distinctive developmental and anatomic feature. In other mammals, development of the facial airway and olfactory nerve alignment is much more horizontally or obliquely disposed.

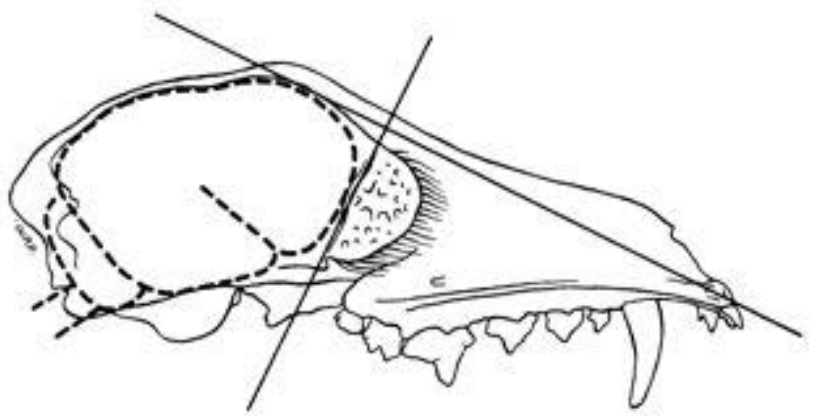


FIGURE 9-15.
(From Enlow, D. H., and M. Azuma: Functional growth boundaries in the human and mammalian face. In: *Morphogenesis and Malformations of the Face and Brain*. Ed. by D. Bergsma. Birth Defects Orig. Art. Ser., Vol. XI; No. 7. New York, Alan R. Liss, Inc. for The National Foundation—March of Dimes, White Plains, New York, with permission.)

The nasomaxillary complex, as mentioned earlier, is specifically associated with the anterior cranial fossae. The posterior boundary of these paired fossae establishes the corresponding posterior boundary for the midface. This is essentially a nonvariable anatomic relationship. The **direction** of growth in this region is established by the particular special sense located in this part of the face, which is the visual sense. The posterior maxillary tuberosity is located just beneath the floor of the orbit, and the orbital floor is the roof of the maxillary tuberosity and the sinus within it. The tuberosity is aligned approximately perpendicular to the neutral geometric axis of the orbit (Fig. 9-16). **The posterior plane of the midface extends from the junction between the anterior and middle cranial fossae (e.g., the inferior junction between the frontal and temporal lobes and the anterior-most edge of the great wings of the sphenoid), downward in a direction perpendicular to the neutral axis of the orbit.** This vertical plane passes along the posterior surface of the maxillary tuberosity.

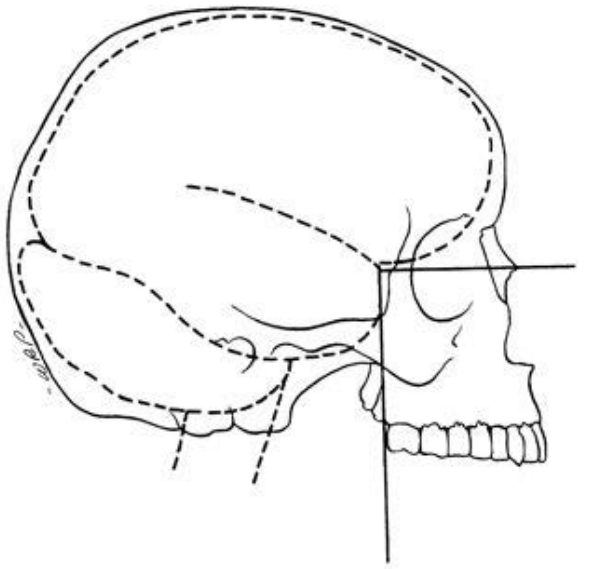


FIGURE 9-16.
(From Enlow, D. H., and M. Azuma: Functional growth boundaries in the human and mammalian face. In: *Morphogenesis and Malformations of the Face and Brain*. Ed. by D. Bergsma. Birth Defects Orig. Art. Ser., Vol. XI, No. 7. New York, Alan R. Liss, Inc. for the National Foundation—March of Dimes, White Plains, New York, with permission.)

The boundary just described represents one of the key anatomic planes in the face. This is the posterior maxillary (PM) plane (Fig. 9-17). There are many "cephalometric planes" in the face and cranium. Most of these, however, do not represent (and are not so intended) (1) key sites of growth and remodeling or (2) functional relationships among the various parts of the skull, including soft tissue associations. Most conventional cephalometric planes, such as sella-nasion, unfortunately, bypass the really important key sites of development without recognizing them. Sella itself, for example, is a "landmark of convenience" because it can be readily and reliably located. But trying to use it to determine real morphogenetic relationships would be like looking for lost keys under a lamp post because that's where the light is. The vertical PM boundary, in contrast, is a **natural anatomic and morphogenic** plane that relates directly to the factors that establish the basic design of the face. It is one of the most important developmental and structural planes in the face and cranium.

The **PM** plane delineates naturally the various anatomic **counterparts** of the craniofacial complex. The frontal lobe, the anterior cranial fossa, the upper part of the ethmomaxillary complex, the palate, and the maxillary arch are all mutual counterparts lying anterior to the PM line (Fig. 9-18, *a, b*, and *c*). All these parts have posterior boundaries that are placed along this vertical plane. Similarly, the temporal lobe, the middle cranial fossa, and the posterior oropharyngeal space with the bridging ramus are mutual counterparts located behind the PM plane (*d, e*, and *f*). The anterior boundaries of these parts are precisely positioned along this vertical line. The PM plane is a **developmental interface** between the vertical series of counterparts in front of and behind it. This key plane retains these basic relationships throughout the growth process.

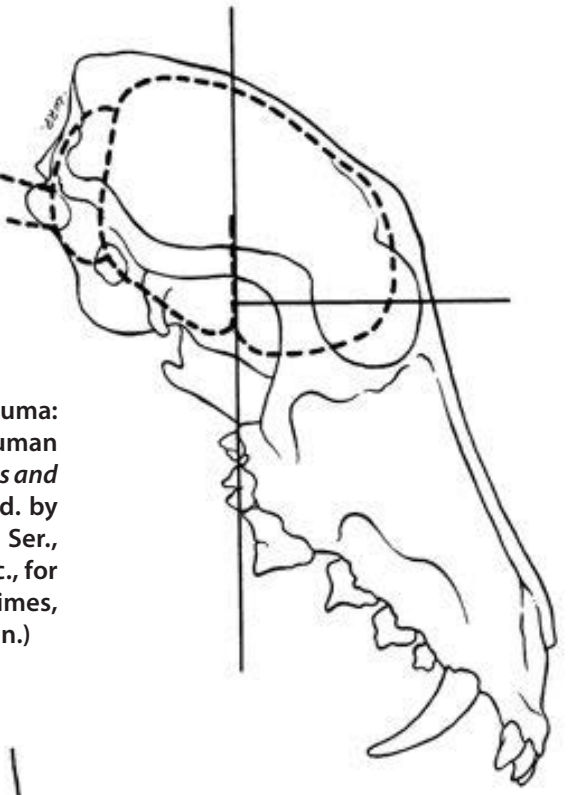


FIGURE 9-17.
(From Enlow, D. H., and M. Azuma: Functional growth boundaries in the human and mammalian face. In: *Morphogenesis and Malformations of the Face and Brain*. Ed. by D. Bergsma. Birth Defects Orig. Art. Ser., Vol. XI, No. 7. New York, Alan R. Liss, Inc., for The National Foundation—March of Dimes, White Plains, New York, with permission.)

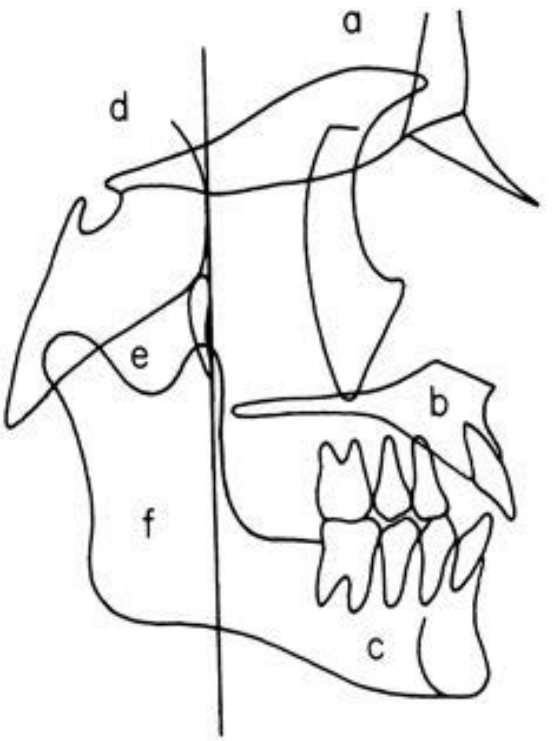


FIGURE 9-18.
(From Enlow, D. H.: Postnatal growth and development of the face and cranium. In: *Scientific Foundations of Dentistry.* Ed. by B. Cohen and I. R. H. Kramer. London, Heinemann, 1975, with permission.)

The positional relationships between the frontal lobes of the cerebrum (anterior cranial fossae) and facial components, and also between part of the middle cranial fossae and the pharynx, are established early in embryonic development. In Figure 13-3, note that the cephalic flexure places the maxillary and mandibular arches in direct juxtaposition with what will become the frontal lobes and the anterior cranial fossae.

The **corpus** of the mandible is a counterpart to those parts lying in front of the PM plane. The **ramus** is a counterpart of the parts behind the PM plane. The placement of the mandible and the size of its parts, however, are more independently variable than those of the ethmomaxillary complex. The posterior boundary of the corpus **should** lie on the PM line. This is the "lingual tuberosity," which is the direct mandibular equivalent of the maxillary tuberosity. The forward boundary of the ramus, where it joins the lingual tuberosity, should also lie on the PM line. (Note: The anterior edge of the obliquely aligned ramus overlaps the lingual tuberosity, but this edge does not represent the actual forward point of the **effective** ramus dimension; the lingual tuberosity itself is the functional junction between the corpus and the ramus.) Because the mandible is a separate bone not attached directly to the cranium by sutures, its latitude for structural variation is not subject to the same degree of developmental and structural communality that occurs between the growth fields shared by the cranial floor and the maxilla. Also, ramus development relates directly to the muscles of mastication, and this requires interplay adjustments. Independent variations can thus exist in the dimensions and the placement of both the ramus and the corpus. The ramus, for example, may fall short of the PM plane, or it may protrude well forward of it. This variability in features is often **compensatory**, as described in Chapter 10. Furthermore, whole-mandible rotations are common, and these developmental "displacement" movements shift the mandible into many variable positions.

Passing from the basicranium, the maxillary nerve crosses the pterygopalatine fossa and then into the inferior orbital fissure. This segment of the nerve, prior to its downward turn through the infraorbital canal and out through the suborbital foramen, closely parallels the plane of the palate. An embryonic relationship exists, and the alignment of the nerve is usually accompanied by a corresponding upward or downward rotational alignment of the palate.

Just as other facial boundaries coincide with basicranial boundaries, the inferior nasomaxillary boundary is established, when growth is complete, by the inferior surface of the brain and basicranium (Figs. 9-19 and 9-20).¶

If Class II and Class III headfilm tracings are superimposed on the cribriform plates (representing the olfactory bulbs), it is apparent that the anterior plane of

¶ Some simians and anthropoids have an **established vertical hypoplasia** in the anterior part of the maxillary arch. In the rhesus monkey, for example, the premaxillary region is "high," or, at least, the posterior part of the nasomaxillary complex is vertically "long." A differentially greater extent of downward displacement takes place in the posterior part of the arch as compared with the anterior part. This in effect causes an "upward" rotation of the anterior region, and direct downward bone growth by this area does not move it fully to the inferior level attained by the posterior part of the arch. Resultant anterior open bites are quite frequent, much more so than in the human face. A similar arch rotation can, in fact, occur in the human maxilla, but the anterior part of the arch develops inferiorly to an extent that fully offsets it. (See also Chapter 10.)

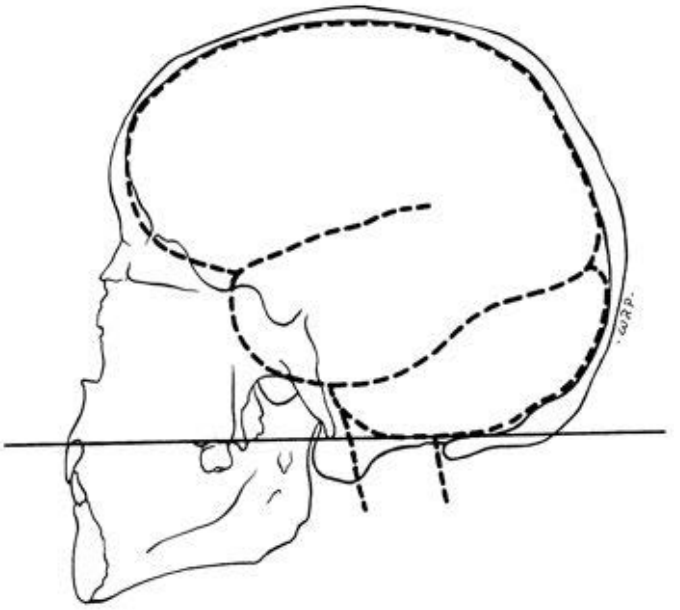


FIGURE 9-19.
(From Enlow, D.H., and M. Azuma: Functional growth boundaries in the human and mammalian face. In: *Morphogenesis and Malformations of the Face and Brain*. Ed. by D. Bergsma. Birth Defects Orig. Art. Ser., Vol. XI. No. 7. New York, Alan R. Liss, Inc., for The National Foundation—March of Dimes, White Plains, New York, with permission.)

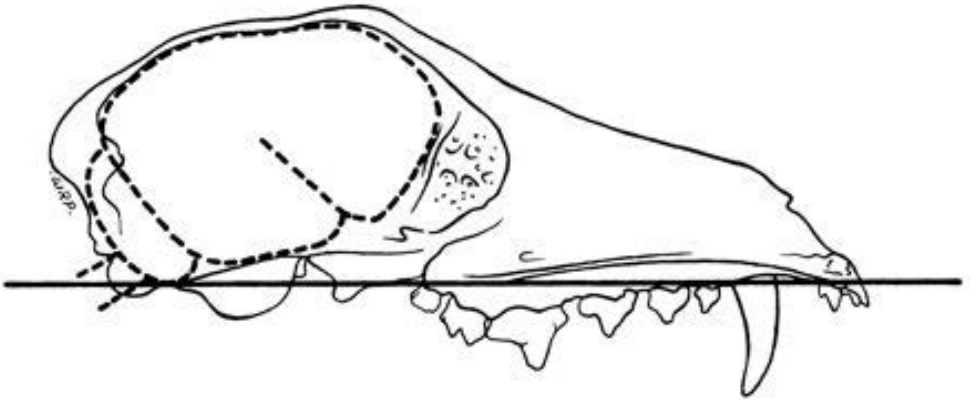


FIGURE 9-20.
(From Enlow, D. H., and M. Azuma: Functional growth boundaries in the human and mammalian face. In: *Morphogenesis and Malformations of the Face and Brain*. Ed. by D. Bergsma. Birth Defects Orig. Art. Ser., Vol. XI, No. 7. New York, Alan R. Liss, Inc., for The National Foundation—March of Dimes, White Plains, New York, with permission.)

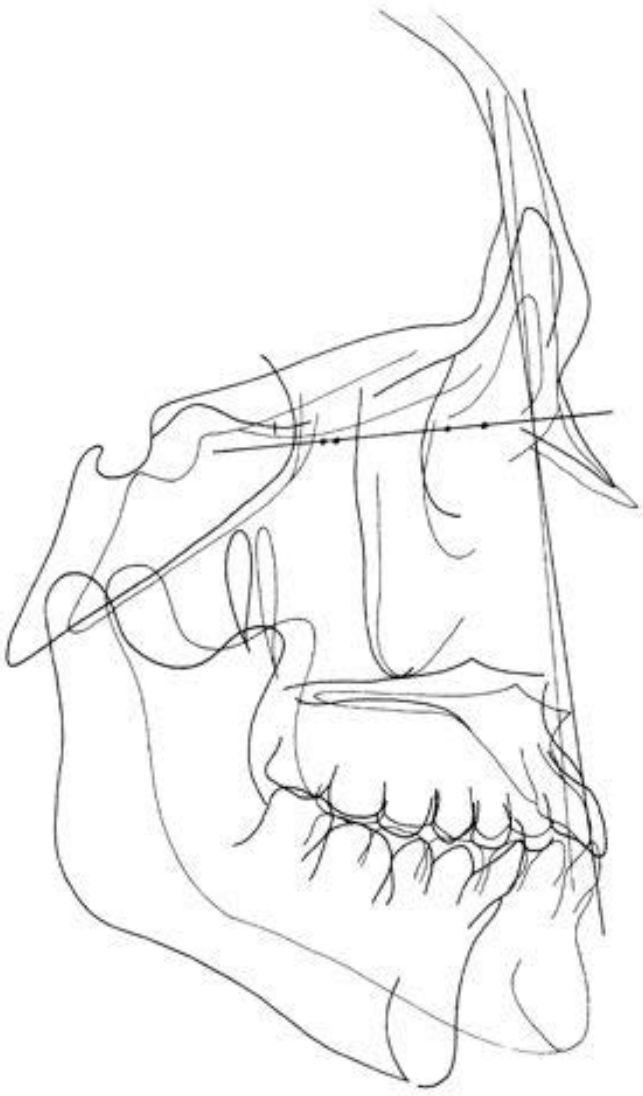


FIGURE 9-21.
(From Enlow, D. H., and J. McNamara: The neurocranial basis for facial form and pattern. Angle Orthod., 43: 256, 1973, with permission.)

the nasomaxillary region in both malocclusion categories conforms closely to the normal, perpendicular olfactory relationship. Note the similarity of the midfacial plane alignments (Fig. 9-21). In this particular Class II individual (and most others as well), it is not the basal bone of the maxilla itself that "protrudes" (relative to the basicranium); rather, it is the **mandible** that is actually **retrusive**. In the Class III individual, it is not always the maxilla that is retrusive; the **mandible** is **protrusive**. In both individuals, the nasomaxillary complex is located where it is supposed to be, and its horizontal dimensions are not out of line as they relate to the brain and anterior cranial fossae.

In summary, development in each region of the face involves two basic factors: (1) the amount of growth by any given part and (2) the direction of growth by that part. The brain establishes (or at least shares) the various **boundaries** that determine the maximum and minimum **amounts of facial growth**. This is because the floor of the cranium is the template upon which the face is constructed. The **directions** of regional remodeling among the different parts of the face are inseparably associated with the special sense organs housed within the face. These two factors establish a prescribed growth perimeter that defines the borders of the growth compartment occupied by the nasomaxillary complex (Figs. 9-22 and 9-23). All the many components that constitute the midface, including the bones, muscles, mucosae, connective tissues, cartilage, nerves, vessels, tongue, teeth, and so on, participate and actively interrelate in the composite expression of growth, the sum of which can produce enlargement up to a given individual's maximum, as determined by the midfacial growth boundaries. The growth of the **midface** is not limitless, and it is not independently and randomly determined entirely within itself.

Superior prosthion thus comes to lie in a predetermined position that has been programmed by the brain-cranial base-sense organ-soft tissue composite of developmental factors. Superior prosthion is composed of alveolar bone, which is a highly labile and responsive type of bone tissue. Traditionally, this area of bone is regarded as quite unstable and subject to a wide range of variations according to the many forces that act on it. This is quite true, as will be seen below. However, prosthion has a specific target location that it will occupy if the growth process is not disturbed by intrinsic or extrinsic imbalances (e.g., thumb sucking). The target point is not programmed within prosthion itself, or even just within the maxilla. It is determined, rather, by the composite of all the growth-establishing factors mentioned above. In most cases, prosthion has settled in, when growth is complete, right on or very close to its target point.

In the headfilm tracing shown in Figure 9-24, it is seen that prosthion falls short of the predetermined midfacial plane; growth is incomplete, however. In the same individual, when facial development has become largely completed, prosthion will have arrived at its place on the perpendicular adult (dashed) midfacial line. In Figure 9-25, the two headfilms are superimposed on the cribriform plane to show the "before" and "after" growth stages.

Can the brain-sense organ relationship within the face be violated? Of course; it frequently happens. For example, thumb sucking (mentioned above), tongue thrust, and various developmental defects can move the teeth and alveolar bone to places that are out of bounds with respect to the normal growth process (Fig. 9-26). The forces and factors of ordinary growth become overridden by extrinsic forces,

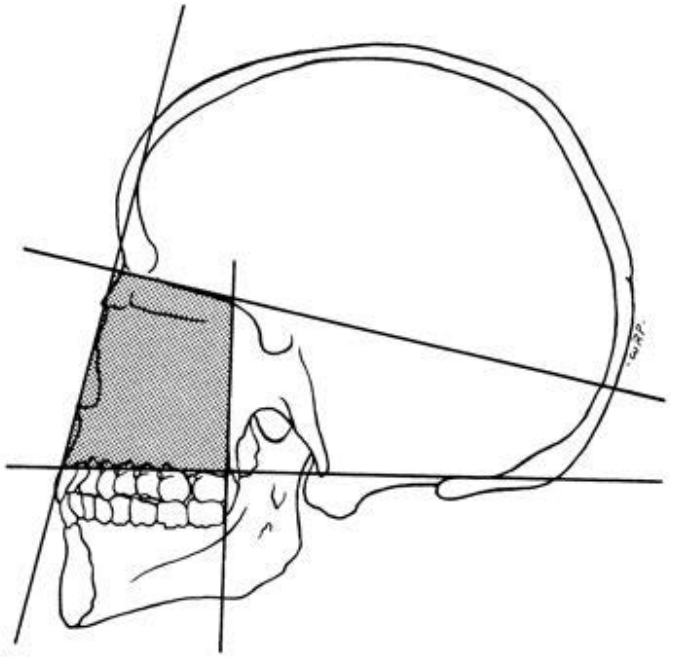


FIGURE 9-22.
(From Enlow, D. H., and M. Azuma: Functional growth boundaries in the human and mammalian face. In: *Morphogenesis and Malformations of the Face and Brain*. Ed. by D. Bergsma. Birth Defects Orig. Art. Ser., Vol. XI, No. 7. New York, Alan R. Liss, Inc., for The National Foundation—March of Dimes, White Plains, New York, with permission.)

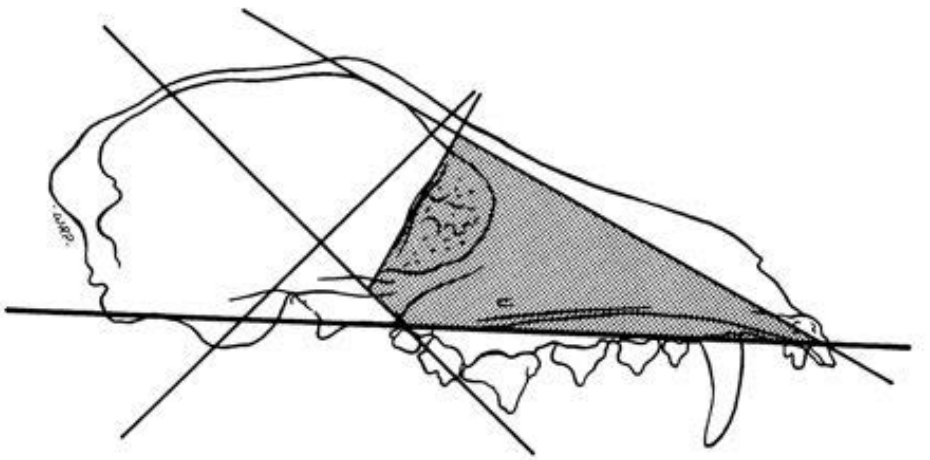


FIGURE 9-23.
(From Enlow, D. H., and M. Azuma: Functional growth boundaries in the human and mammalian face. In: *Morphogenesis and Malformations of the Face and Brain*. Ed. by D. Bergsma. Birth Defects Orig. Art. Ser., Vol. XI, No. 7. New York, Alan R. Liss, Inc., for The National Foundation—March of Dimes, White Plains, New York, with permission.)

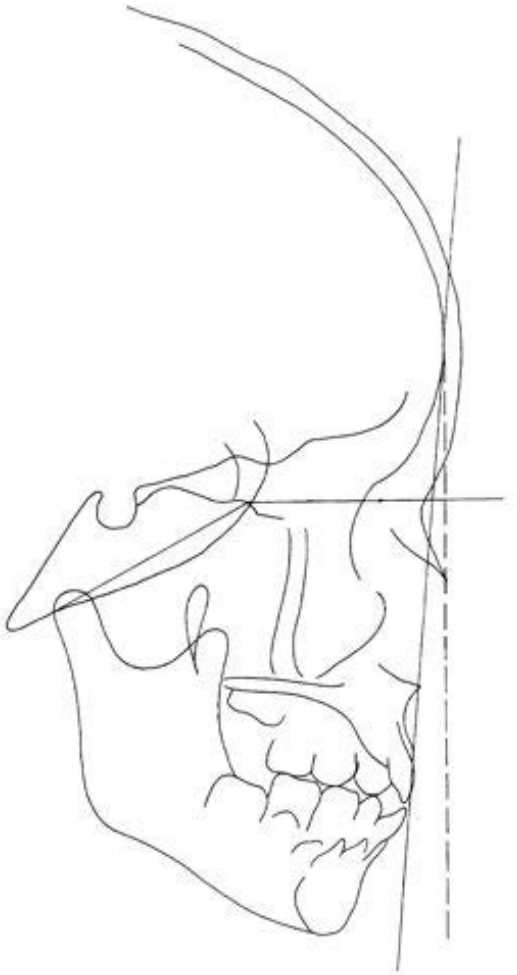


FIGURE 9-24.
(From Enlow, D. H., and J. McNamara: The neurocranial basis for facial form and pattern. Angle Orthod., 43:256, 1973, with permission.)

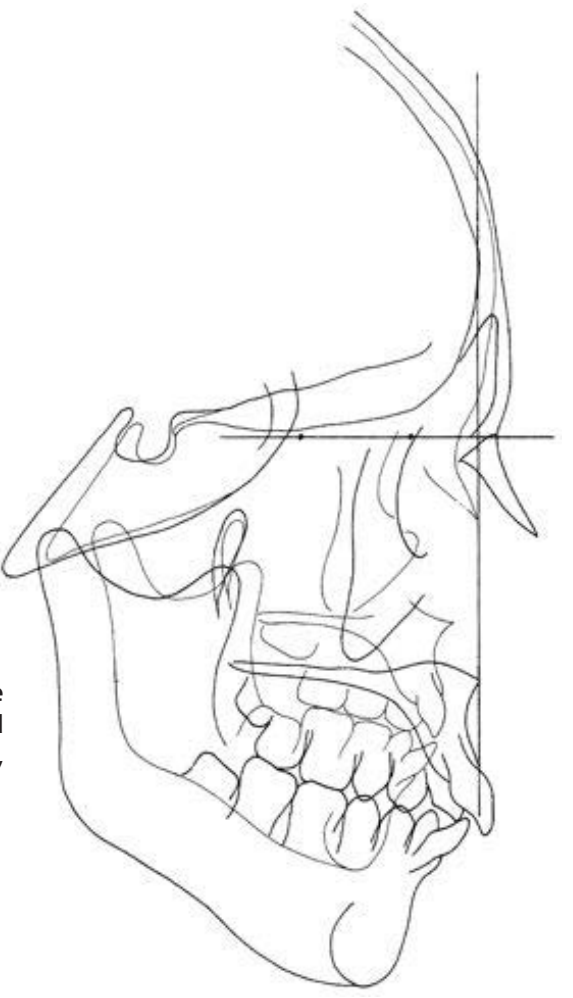
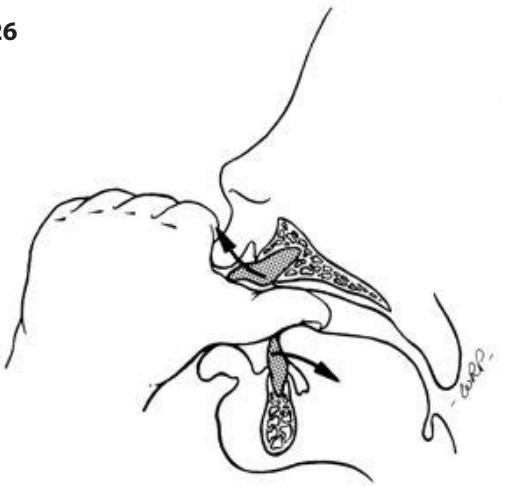


FIGURE 9-25.
(From Enlow, D. H., and J. McNamara: The neurocranial basis for facial form and pattern. Angle Orthod., 43:256, 1973, with permission.)

FIGURE 9-26



and the prescribed boundary and the usual limit of growth are thereby overrun. However, this produces a structural and functional imbalance. If the overriding ectopic factors are removed, the normal balance of functional intrinsic relationships work toward a greater or lesser return to the normal position, conforming with the natural anatomic boundary of the growth field. This is the reason most children who suck their thumb during early childhood, but later stop the habit, do not have the typical "thumb sucker's" malocclusion. Once the abnormal force has been removed, physiologic rebound seeks equilibrium (Chapter 1), and subsequent development returns the component parts to a balanced relationship.

Because many anatomic boundaries, large and small, exist throughout the face and cranium, the factor of boundary "security" is a major and important consideration to the clinician. If one given facial growth field is made to overrun the boundary of another field, either by clinical intervention or because of a developmental abnormality, one or the other will necessarily become compromised. A competition for the same space by the two overlapping growth fields occurs, and one field will necessarily become subordinate. This has great meaning with regard to the **stability** of a region and the functional "equilibrium" among different structural parts. If, for example, a given treatment procedure causes a violation of some growth boundary, will hard-earned treatment results subsequently be lost because functional stability and balance have been disturbed? Or, perhaps, will results be lost because the activity of a growth field that has been imposed upon subsequently causes a return ("rebound") toward the original structural pattern when treatment is stopped? Another similar question is whether a treatment procedure that targets form rather than function can actually **change** the long-term growth program. If normal physiologic function does not result from the normalization of form, subsequent growth, after treatment is ceased, can erase the treatment results, because growth then proceeds along its original unaffected course. This may be one of the reasons for "relapse" of early arch expansion during subsequent periods of rapid growth. Periods of rapid change can be useful to the clinician "working with growth" if more growth is beneficial to treatment. For example, in the case of mandibular retrognathia, additional mandibular growth

would be highly desirable. In contrast, in cases of mandibular prognathism additional mandibular growth would be anathema. Clinicians must also consider that physiologic factors involved in rebound must also be biologically active during periods of rapid change. Therefore, the clinician contemplating early treatment must be aware that interventions that address "cause" will be more effective during periods of rapid growth, and interventions that address "effect" are likely to be more successful during periods of slower growth and adaptation. This leads to the clinical axiom, "Treat cause early and effect late". The rate of facial growth is often overlooked when evaluating the stability of surgical manipulations of facial bones. It is likely that the stable surgical results observed in adults are due to decreased biologic activity thus slowing physiologic rebound. A better understanding of growth boundaries will help address fundamental clinical questions such as when can the maxilla be successfully expanded. Two dimensional cephalometric radiographs have limited our ability to identify biologically relevant structures such as the cribiform plate and the lingual tuberosity. In the future, the use of three dimensional imaging will allow visualization of these and other biologically important anatomic structures and allow new tools for craniofacial diagnosis and treatment planning to be developed.

There are many theoretically possible alternatives with regard to stability versus rebound/relapse as summarized in Figure 1-1. Some alternatives seem more feasible than others, and still more possibilities probably exist that are not included. Some may hold true for one given clinical or growth circumstance, and others for different circumstances. All, however, must be included as considerations in the "big picture".

10

Normal Variations in Facial Form and the Anatomic Basis for Malocclusions

Variation is a basic law of biology. The pool of structural, functional, and genetic-based variations always present within a population of any species provides the capacity for adaptation to a changing environment. This increases the probability of survival for those individuals having features most suitable for the needs of the time. The human face certainly has its share of variations. Indeed, there are probably more basic, divergent kinds of facial patterns among humans than among the faces of most other species. This is because unusual facial and cranial adaptations have occurred in relation to human brain expansion. A great range of facial differences exists because the brain, proportionately, is so large and so variable in configuration. There is also a much greater likelihood for different kinds of malocclusions in the human face than in the faces of most other species for the same reasons. In fact, actual tendencies toward malocclusions are **built into** the basic design of our faces because of the unusual relationships inherent in their design.

A comprehensive taxonomic system for cataloging and naming facial types based on developmental variations does not presently exist. However, three general classification categories are in common use. One relates to headform type (see Chapter 8), another deals with malocclusions (see below), and the third is based on topographic profile (Fig. 10-7). All three systems are directly interrelated with regard to underlying and predisposing morphogenic and morphologic characteristics.

Headform and Malocclusion Tendencies

In individuals (or whole populations) having a **dolichocephalic** headform, the brain is horizontally long and relatively narrow (see Fig. 10-1). This sets up a basicranium that is somewhat more flat; that is, the flexure between the middle and the anterior parts of the cranial floor is more open (Figs. 10-1, 10-2 and 10-18). It is also horizontally longer. These factors have several basic consequences for the pattern of the face. First, the whole nasomaxillary complex is placed in a more **protrusive** position relative to the mandible because of the forward basicranial

FIGURE 10-1

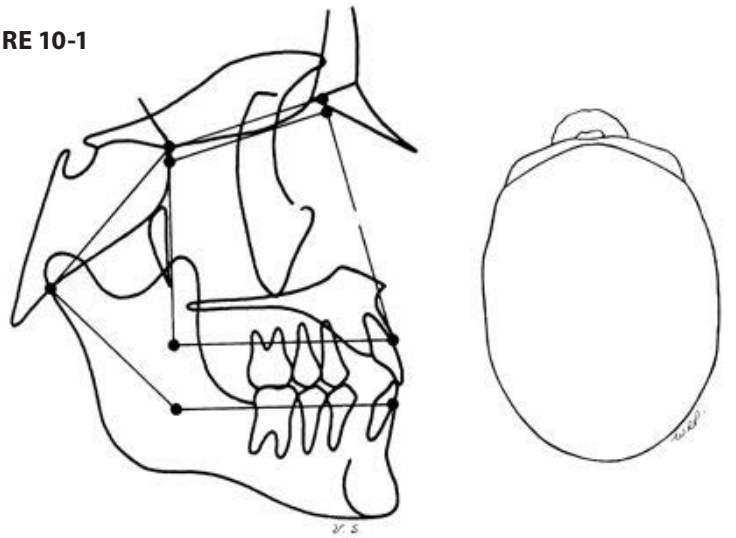
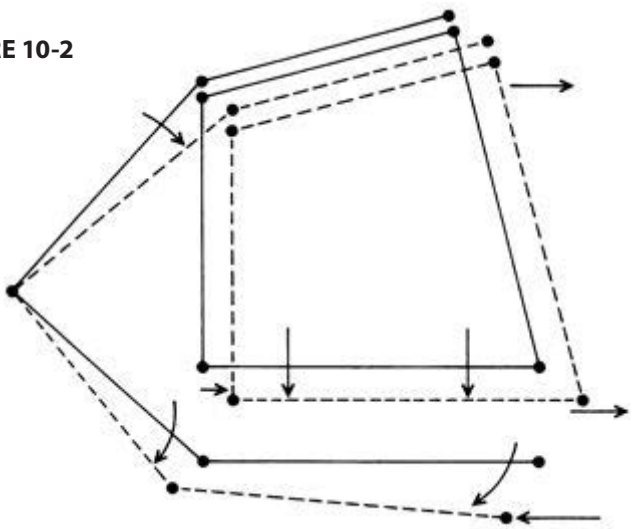
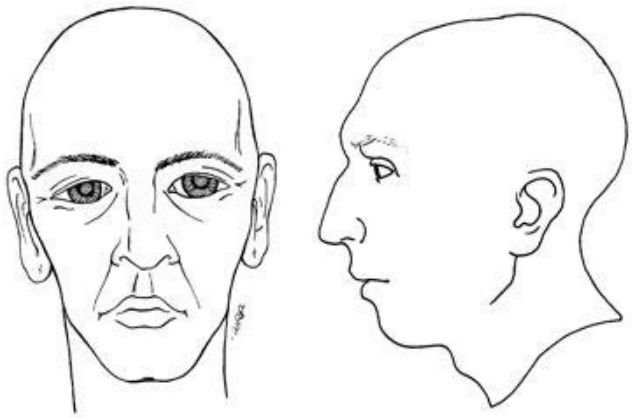


FIGURE 10-2



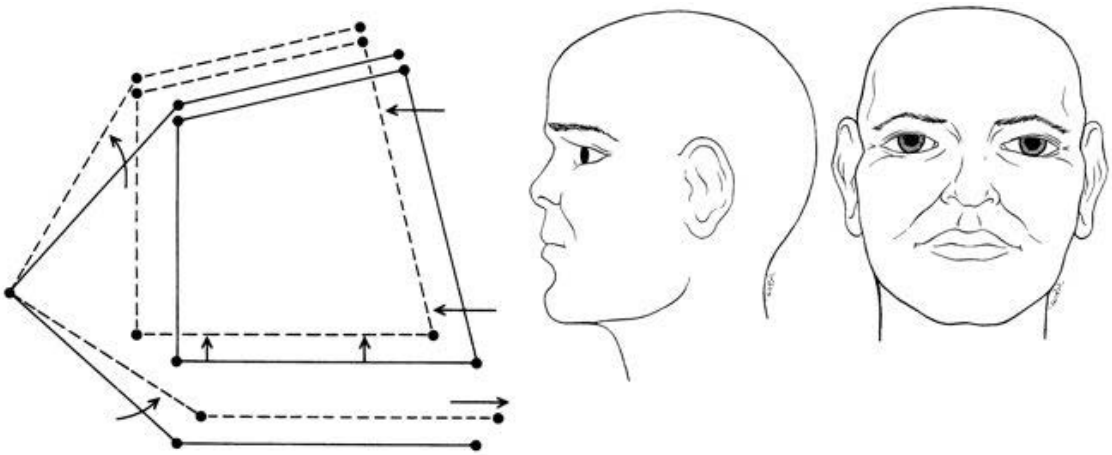
rotation and, also, the horizontally longer anterior and middle segments of the cranial floor. Second, the whole nasomaxillary complex is lowered relative to the mandibular condyle. This causes a downward and **backward** rotation of the entire mandible. The vertically long face of the dolichocephalic adds to this, as described later. Third, the occlusal plane becomes rotated into a downward-inclined alignment. The **two-way** forward placement of the maxilla and backward placement of the mandibular corpus results in a tendency toward mandibular retrusion, and the placement of the molars results in a Class II position. The resultant profile is retrognathic (Figs. 10-3 and 10-7). However, compensatory changes are usually operative, as explained later. Because of the more open cranial base angle and the more oblique trajectory of the spinal cord into the cervical region, this type of face is associated with individuals having a greater tendency toward a somewhat stooped posture and anterior inclination of the head and neck.

FIGURE 10-3



Individuals or ethnic groups with a **brachycephalic** headform have a rounder, wider brain. This sets up a basicranial floor that is more upright and has a more closed flexure, which decreases the effective anteroposterior dimension of the middle cranial fossa (Figs. 10-4 and 10-5). The facial result is a more posterior placement of the maxilla. Furthermore, the horizontal length of the nasomaxillary complex is also relatively short. Because the brachycephalized basicranium is wider but less elongate in the anteroposterior dimension, the middle and anterior cranial fossae are correspondingly foreshortened (not shown in the schematic diagram). The anterior cranial fossa sets up the template for the horizontal length and bilateral width of the nasomaxillary complex, which is thereby also shorter, but wider. The composite result is a relative retrusion of the nasomaxillary complex and a more forward relative placement of the entire mandible. This causes a greater tendency toward a prognathic profile and a Class III molar relationship. The occlusal plane may be aligned upward, but various compensatory processes usually result in either a perpendicular or a downward-inclined occlusal plane. Other compensatory changes are also operative, as explained next, and these tend to counteract the built-in Class III tendencies. Because of the more upright middle

FIGURE 10-4



cranial fossa and the more vertical trajectory of the spinal cord, individuals with all these various facial features also have a tendency for a more erect posture with the head in a more "military" (at braced attention) position.

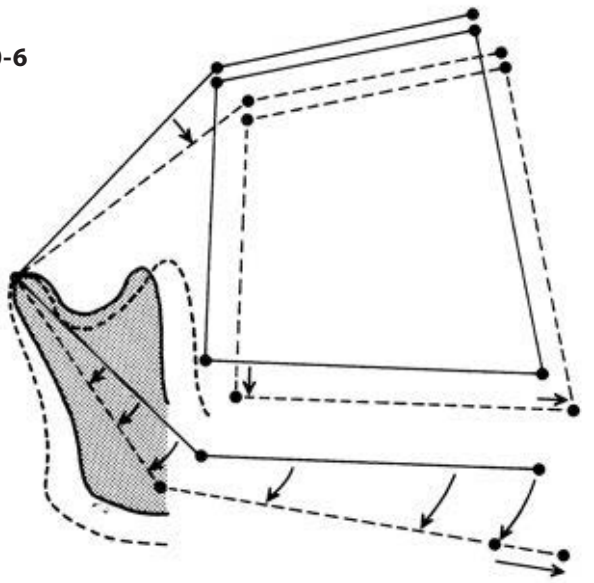
FIGURE 10-5

The basic nature of interrelationships among (1) brain form, (2) facial profile, and (3) occlusal type predisposes characteristic facial types and malocclusions among different types of populations. Some Caucasian groups with a tendency for a dolichocephalic headform have a corresponding **tendency** toward Class II malocclusions and a retrognathic profile. Far-Eastern populations, having mostly a brachycephalic headform, have a correspondingly greater tendency toward Class III malocclusions or bimaxillary protrusion and a prognathic profile. These respective tendencies are built into the basic plan of facial construction. However, most of us also have intrinsic structural features that have compensated for these tendencies (the growth process itself working toward balance, Chapter 1). **If** we have such compensatory features, the built-in tendencies are offset, to a greater or lesser extent, and we thereby have a Class I occlusion, even though the reasons for the underlying tendencies are still present. **If** these compensatory features are less than complete, however, the built-in tendencies then become more fully expressed, and we have a malocclusion but less severe than the tendencies otherwise could produce. The existence of anatomic compensations is the main reason that the total variation in the occlusion of the upper and lower first molar teeth across all human beings is only 6 millimeters. This is remarkable considering the range of facial appearance among human populations. In fact, treatment planning with regard to the human face is often a matter of deciding which anatomic compensations to keep or augment and which to eliminate or reduce.

How does a face undergo intrinsic compensations during its development? One example that is very common is shown here. In the situation described above, the mandible was placed in a retrusive (retrognathic) position owning to its downward and backward rotation resulting from the more open type of cranial base flexure (and/or a vertically long nasomaxillary complex). The mandibular **ramus**, however, can compensate by an increase in its horizontal dimension (Fig. 10-6). This places the whole mandibular arch more anteriorly beneath the maxilla, and it positions the teeth in a "normal" or a Class I type of molar relationship. The extent of mandibular retrusion that would otherwise be present thus becomes partially or completely eliminated, and a profile in which the chin lies on or close

to the orthognathic profile line results. The downward placement of the dental arch, caused by the downward-backward mandibular rotation described above is offset by an upward drift of the anterior mandibular teeth and a downward drift of the anterior maxillary teeth. This causes a curved occlusal plane, the curve of Spee (see following pages).

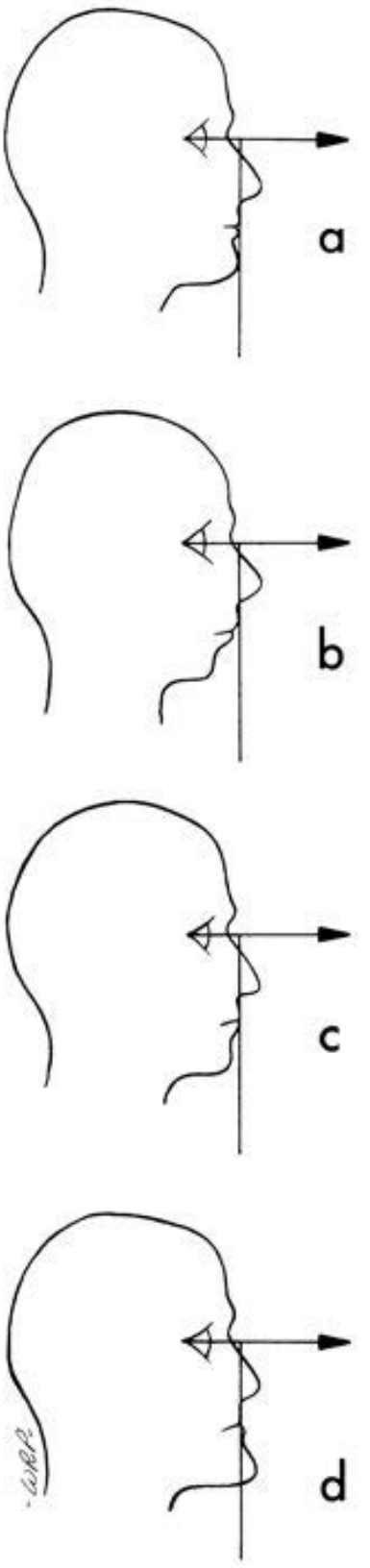
FIGURE 10-6



The face on each of us, virtually without exception, is the composite of a great many regional "imbalances." Some of these offset and partially or completely counteract the effects of the others. The wide ramus cited above, for example, is actually an imbalance, but it serves to reduce, as a normal adjustment process, the effects of some other angular or dimensional imbalances caused by the built-in tendencies toward malocclusions. The particular feature of a wide ramus is very common among the dolichocephalic Caucasians. When this and other compensatory factors are present, the underlying stacked deck toward retrognathia and a Class II malocclusion is removed or made less severe. Thus, many of us have a slightly retrognathic profile and a little anterior tooth crowding.

Three general types of facial profile exist: **orthognathic, retrognathic,** and **prognathic** (Fig. 10-7). The orthognathic ("straight-jawed") form is the everyday standard for a good profile, and it is the type common to most Hollywood and television big names. It is easy to "eyeball" a person's face, without actual need for headfilms or precision anthropometric instruments, to see what his or her profile type is. Simply visualize a line extending from the center of the orbit looking straight forward (*a*). Now visualize a **vertical** line **perpendicular** to the orbital line extending down along the surface of the upper lip. This line will just touch the lower lip and the tip of the chin in a person with an orthognathic profile. Time otherwise thrown away waiting around air terminals, sitting in classes, or standing on line can be put to interesting use quietly studying people's profiles and the facial patterns described herein.

FIGURE 10-7



The retrognathic face has a characteristic convex-appearing profile. The tip of the chin lies somewhere behind the vertical line, and the lower lip is retrusive. The chin may be 2 or 3 cm behind the line in a severely retrognathic face (*b*). Among many Caucasians, however, it is common to have about a half centimeter or so of chin retrusion (*c*). The profile is retrognathic, but the extent is reduced because the growth process itself has provided a number of adaptive adjustments that partially offset "built-in" tendencies that can exist toward mandibular retrusion. "Facial development" is one's own personal orthodontist.

The "Effective Dimension"

In this section, specific cause-and-effect relationships underlying differences in facial pattern are explained. Each regional area throughout the face and cranium is considered separately. To evaluate the structural and developmental situation for each given region, a simple test is used: that region is compared with other regions with which it must "fit." If they have a variance of respective fit, the result is appraised by noting whether it causes (1) a mandibular retrusive or (2) mandibular protrusive effect. As will be seen, imbalances in many parts of the head are passed on, region by region, and in turn affect the placement of the jaws and the resultant nature of the occlusion.

Two basic factors must be considered for each region. The first is the **dimension** of a particular part. Is it "long" or is it "short" with regard to its assembly with other parts?

Great care must be used to evaluate only that particular span or dimension of a bone specifically involved in actual, direct fitting. This is the **effective dimension**. The second fundamental consideration is the **alignment** of any given part. This must also be included (although many cephalometric studies do not), because any rotational change either increases or decreases the **expression** of a dimension.

The relationship between effective mandibular length and vertical maxillary dimension is very important to the clinician because patients often present with combined anteroposterior and vertical facial disharmony. For example, individuals with mandibular prognathia can also have increased lower vertical facial height due to increased maxillary vertical dimension. This increased vertical masks the extent of

the prognathism. When the vertical height is corrected surgically, or with the use of TADs in the growing patient, the degree of prognathism is unmasked. Therefore, to harmonize the facial components, patients with orthognathic profiles and long faces need a treatment plan to decrease mandibular length after correction of the vertical excess. This is a critical factor for the clinician to consider because it dramatically affects the treatment plan.

The Dimensional Factor of Alignment

To illustrate the important effects of **alignment** as a basic factor involved in determining facial pattern, in Figure 10-18 the alignment of the middle cranial fossa in a Class I child was changed (on paper) to a less upright position. All the other facial regions, including the mandible, maxilla, and the anterior cranial fossa, were then reassembled around the realigned middle cranial fossa. **No** changes in the actual dimensions of any parts were made. The horizontal and vertical **expression** of the middle cranial fossa dimension, however, resulted in a change from the Class I pattern into a Class II pattern, even though all the individual bones were exactly the same size.

In Figures 10-8 and 10-9, if the horizontal dimension of the mandibular corpus (*b*) is short relative to its counterpart, the bony maxillary arch (*a*), the effect is, of course, mandibular retrusion (probably with anterior crowding of the teeth). Note that this does not necessarily cause a Class II molar relationship, because the **posterior** parts of the upper and lower bony arches can still be properly positioned. It is emphasized that these are **relative** comparisons between two contiguous parts within the **same** individual. The mandible is not being compared with a norm or an average value derived from a population sample. Whatever the actual value of this mandibular dimension happens to be in millimeters, or regardless of how it compares with some statistical mean, it is short when compared with the dimensional value that really matters—its counterpart, the horizontal dimension of the maxillary body in that particular individual.*

DIMENSIONAL AND ALIGNMENT PATTERN COMBINATIONS

The remainder of this chapter describes, first, **regional** anatomic features that have a (1) mandibular retrusive or (2) mandibular protrusive effect. Throughout the face and neurocranium, each local region, one by one, is considered. The "counterpart" principle is often applied (Chapters 3 and 9). Second, regional **combinations** as they affect mandibular retrusion or protrusion are described. Third, how built-in malocclusion tendencies can be partially or almost totally compensated by the growth process itself are outlined. Fourth, the typical

* See also Figure 9-18. Evaluation of headfilms utilizing this concept is the "counterpart analysis," a procedure that determines morphologic and developmental features **within** a given individual and which does not require tracing superimpositions or comparisons with population norms. See Martone et al., 1992 for references.

anatomic composite patterns underlying malocclusions are explained. Finally, the continuous spectrum of facial and malocclusion types involving all these morphologic and morphogenic features is highlighted.

FIGURE 10-8

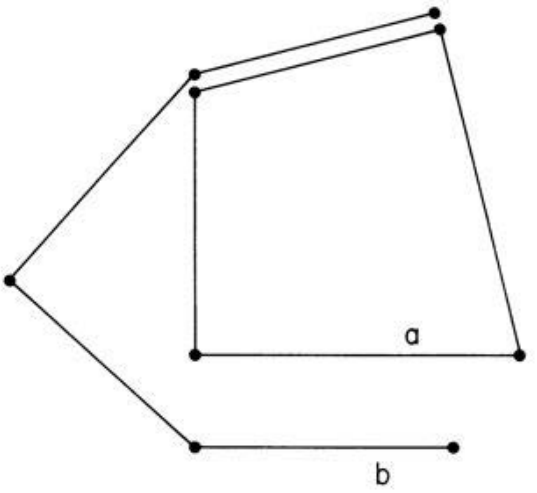
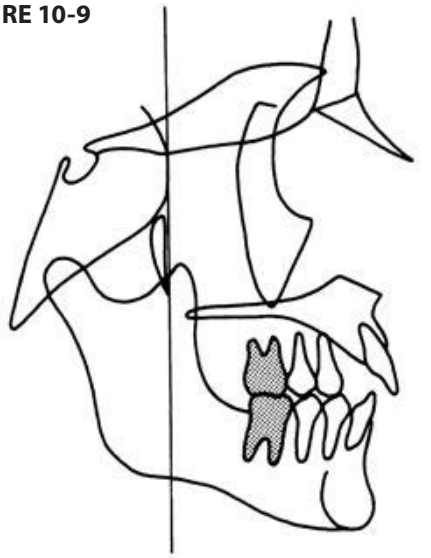


FIGURE 10-9



If the mandibular corpus is dimensionally long, the effect, of course, is mandibular protrusion. A horizontally short maxillary arch has the same effect. (There are anatomic ways to tell which is long and which is short, as explained in Chapter 9). Whether or not a long corpus produces a Class III molar relationship depends on whether it is long mesial or distal to the first molars.

In Figure 10-10, the upper part of the nasomaxillary complex is horizontally long **relative** to its counterparts, the anterior cranial fossa, the palate, and the maxillary and mandibular arches. Note that this has no effect on the occlusion. The individual can **appear** retrognathic, but this is a result of the protrusive nature of the upper part of the face and not the jaws themselves. Because the superior part of the ethmomaxillary region is protrusive, the outer table of the frontal bone remodels with it. The result is a sizeable frontal sinus, heavy eyebrow ridges and glabella, sloping forehead, high nasal bridge, and long nose. The cheekbone area appears retrusive because of the prominent nasal region and forehead, and the eyes are deep set.

If this upper part of the nasomaxillary complex is quite protrusive, as it usually is in the dolichocephalic face, the slope of the nose will often be curved or bent into a classic aquiline (eagle beak), Roman nose, or Dick Tracy configuration **if** the nose is **also** vertically long (Fig. 10-11). The longer the vertical size of the nose, the more its slope must bend. This nasal shape is quite common in leptoprosopic males, and it typically has a rather narrow and sharp configuration. The ventral edge surrounding the nares may be horizontal, but often has a tendency to tip downward. This is in contrast to the vertically and protrusively shorter type of nose in which the lower margin can angle upward. In another type of nasal bending, the **middle** part of the nasal region may be quite protrusive; this produces

a characteristic and gracefully recurved (sigmoid) configuration of the nasal slope as the lower portion grades and curves back onto the less protrusive upper part. The cheekbone area in this type of face is often notably prominent because this entire level of the midface also tends to be prominent.

The above facial features, in general, characterize the long, narrow-faced, dolichocephalic headform found among many (but not all) Caucasian groups and also the dinaric type of headform. These features affect characteristics such as the extent of frontal sinus expansion and the slope of the forehead, and are thus sex and age related.

FIGURE 10-10

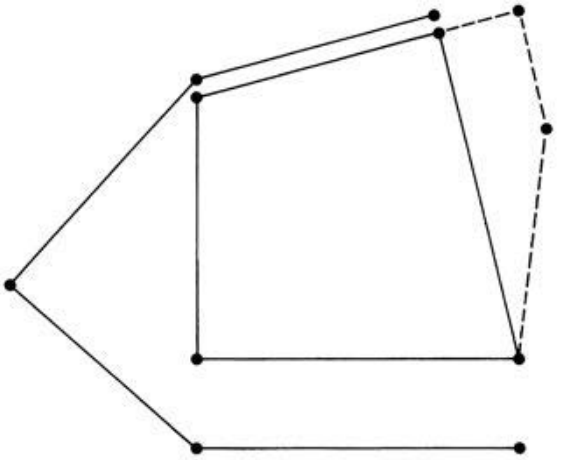
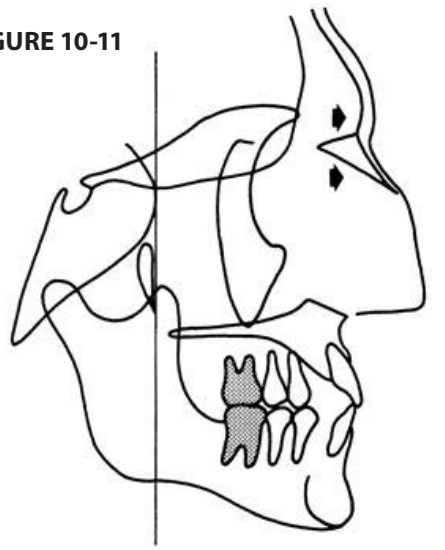


FIGURE 10-11



If the upper part of the nasomaxillary complex is **not** protrusive, so that its anteroposterior size more nearly matches counterpart dimensions in the anterior cranial fossa, palate, and maxillary and mandibular arches, quite a different facial pattern results. The frontal sinuses are comparatively smaller, the forehead is more upright, the eyebrow ridges and glabella are not as prominent, the nose is not nearly as protrusive, and the nasal bridge is much lower. The jaws appear more prominent because the upper nasal region is less protrusive. The cheekbones also appear more prominent for the same reason. The whole face is much flatter and wider appearing. This composite of facial features is typically found in the broad-faced, brachycephalic type of headform that characterizes many Far-Eastern individuals. Some Caucasian populations are also broad faced, with a shorter nose, more prominent mandible, lower nasal bridge, and so forth, including, for example, many individuals having a facial heritage from middle regions of Europe, parts of southern Ireland, and a scattering of geographic locations elsewhere in the world. It has become a common type of Caucasian face in North America. A shorter, but wider nose and nasal chambers provide approximately equivalent airway capacity in comparison with the narrower but longer and more protrusive nose of the dolichocephalic type of headform.

If the effective anteroposterior (not oblique) breadth of the ramus is narrow relative to its counterpart, which is the effective horizontal (not oblique) dimension

of the middle cranial fossa, a mandibular retrusive effect is produced (Fig. 10-12). Note that the mandibular arch lies in a resultant **offset position** relative to its counterpart, the maxillary arch. Even though the upper and lower arches themselves are, in this example, actually matched in dimensions, the profile is retrognathic. The arches are in offset positions because the parts **behind** them are "imbalanced." Note that the posterior part of the maxillary arch lies well anterior (mesial) to the posterior part of the mandibular arch. This is one (of several) of the basic skeletal causes that underlie a **Class II molar relationship.** Remember, the "real" anatomic junction between the ramus and corpus is the lingual tuberosity, rather than the oblique "anterior border" where it overlaps the corpus because of muscle attachment. Because the lingual tuberosity cannot be directly visualized on cephalometic radiographs, it is not represented here. However, it is located distal to the vertical reference line because of the narrow ramus in this individual.

In Figure 10-13, the effective horizontal (not oblique) dimension of the ramus is broad relative to the middle cranial fossa. Or, the cranial fossa is horizontally narrow relative to the ramus (either way because this is a relative comparison). The effect is mandibular protrusion due to the resultant offset positions between the upper and lower arches, even though the horizontal dimensions of the arches themselves can match. This is one (of several) of the basic skeletal causes for a **Class III molar relationship.** The lingual tuberosity (not shown) is mesial to the vertical reference line.

FIGURE 10-12

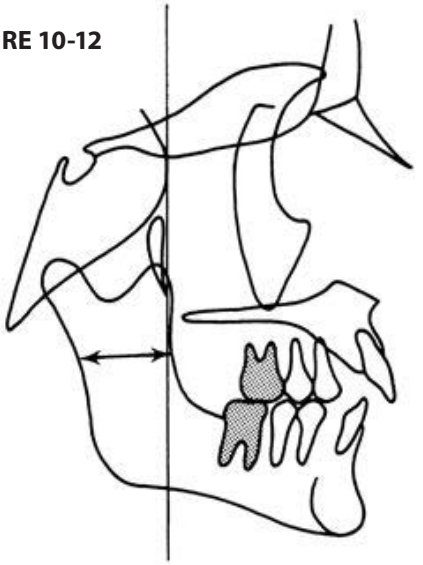
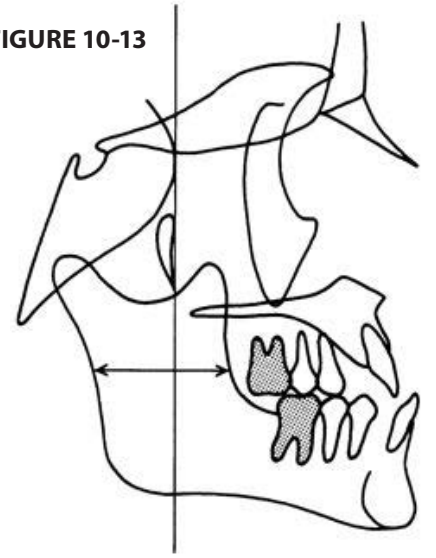


FIGURE 10-13



If the mandible as a whole has a downward-backward alignment (as a result, for example, of a vertically long nasomaxillary region), the effect is mandibular retrusion (Fig. 10-14). While this increases the expression of its vertical ramus dimension, the horizontal is necessarily decreased at the same time. The mandible is rotated downward and **backward**. As a result, the mandibular arch becomes offset relative to the upper arch. The profile is retrognathic, and the offset placement of the arches causes a Class II molar relationship. Note that the mandibular corpus

is rotated downward, causing a downward-inclined mandibular occlusal plane (see page 213 for an explanation of dental compensations).

If the mandible has a more forward and upward inclined alignment (as a result of a vertically short midface), the effect is mandibular protrusion (Fig. 10-15). The arches are offset, and the molars have a resultant Class III relationship. The occlusal plane has an upward inclination relative to the neutral orbital axis or to the vertical posterior maxillary (PM) line. The posterior maxillary teeth can drift inferiorly and/or the gonial angle can open (compensatory adjustments) to provide proper occlusal fit. Otherwise, a posterior open bite can result.

FIGURE 10-14

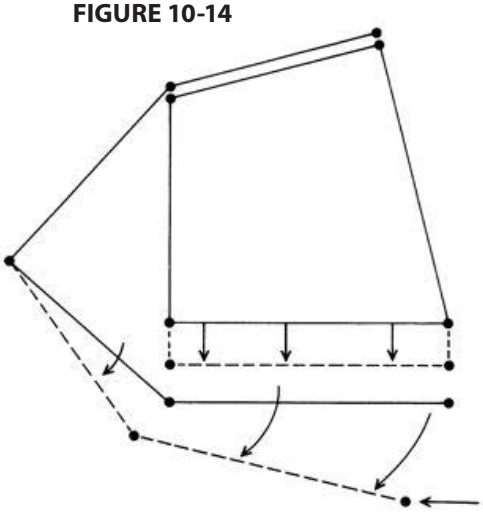
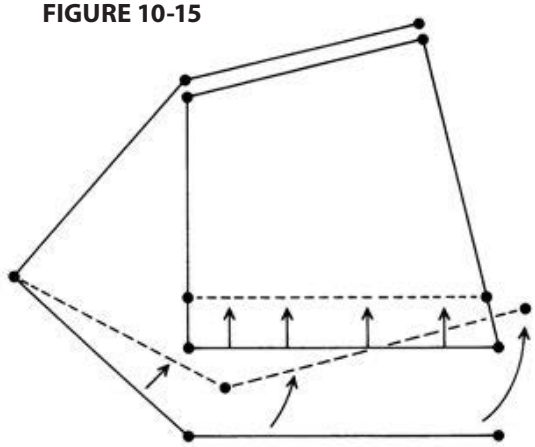


FIGURE 10-15



If the ramus has a closed alignment with the corpus (i.e., a closed "gonial angle"), a mandibular **retrusive** effect is produced. A more open alignment ramus-to-corpus relationship produces a mandibular protrusive effect. These various alignment relationships can be misunderstood and the whole subject of mandibular "rotations" has been perplexing because there are two basic and separate kinds of mandibular skeletal rotations (exclusive of dental arch rotations, which will be described separately).

1. The alignment position of the **whole mandible** can be up or down at the condylar pivot. The primary reason that this kind of developmental rotation takes place is to adjust to whatever vertical size exists for the midface **and** the alignment of the middle cranial fossa. The mandible rotates forward and upward to meet a short midface and/or a closed basicranial flexure (Fig. 10-16), and it rotates down and back (Figs. 10-14, 10-17 and 10-18) to accommodate a vertically long midface and/or a more open basicranial flexure. These are a **displacement** type of rotation (see page 39).

2. The angle between the ramus and the corpus also can become increased or decreased as a separate kind of rotation (Fig. 10-19). This does not refer merely to the conventional "gonial angle" but, rather, to the alignment between the

whole of the ramus and the corpus. This is a **remodeling** type of rotation, in contrast to the displacement type (page 39). The oblique axis of the ramus can thus be more upright, with the ramus-corpus angular relationship thereby "closed." (See also Fig. 10-20.) Or the converse can occur by an opening of the ramus-corpus angle. In either case, the corpus thereby becomes positioned up or down **relative** to the ramus. While the corpus and its dental arch can participate to a limited extent in the opening and closing of its angle with the ramus, it is necessarily the **ramus** that carries out most of developmental remodeling involved. It would not be possible, for example, for the entire corpus (not merely the dentoalveolar portion) to rotate upward by its own remodeling to close the gonial angle.

FIGURE 10-16

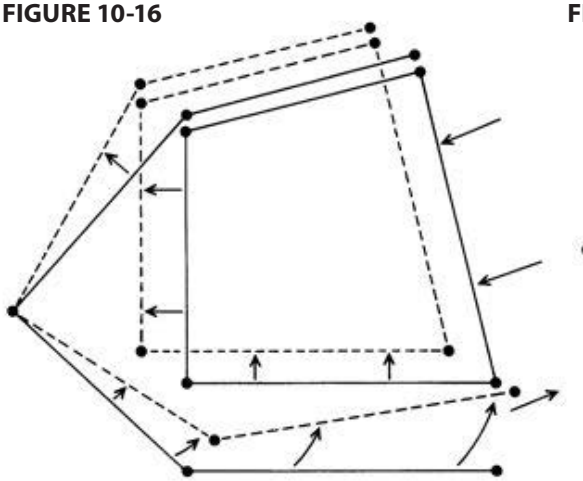


FIGURE 10-17

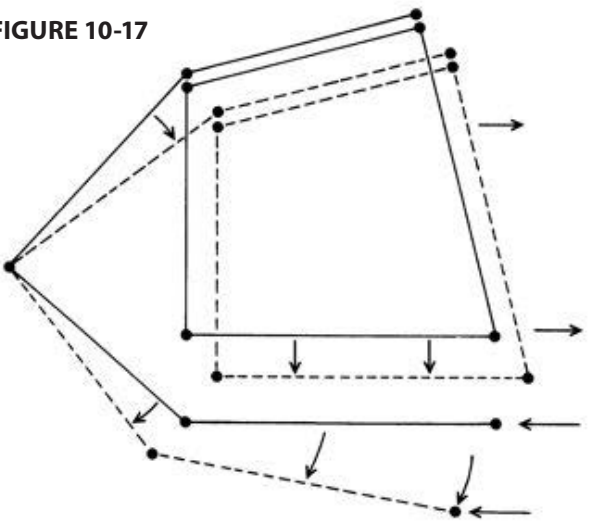
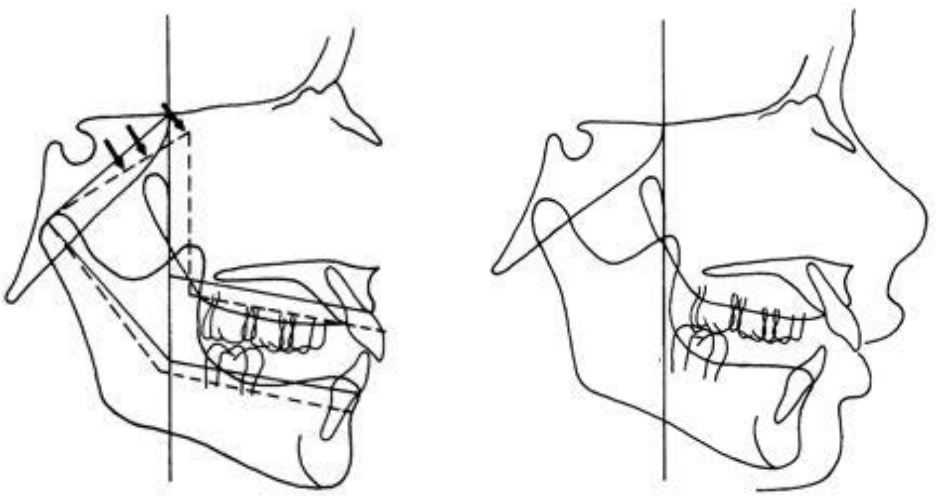


FIGURE 10-18



There are two basic reasons ramus-corpus remodeling rotations occur. The **first** was described on page 83 and deals with the need for a progressively more upright ramus to accommodate a vertically lengthening midface. The remodeling changes that carry this out were also outlined.

The result is a ramus-corpus alignment that naturally and normally becomes more closed as the midface grows. The **second** reason is to accommodate the results of whole-mandible (displacement) rotation. When the entire mandible rotates forward and upward, the mandibular corpus is normally rotated downward by ramus remodeling to some extent in order to compensate. This helps to keep the mandibular dental arch in a constant functional relationship. In addition, the posterior maxillary teeth may drift inferiorly. The occlusal plane can be brought to a perpendicular position relative to the PM plane, or it may still have slight upward inclination. When the ramus (and whole mandible) is rotated backward and downward by displacement, the ramus-to-corpus angle can be closed by ramus remodeling, thereby compensating. The respective amounts of these counteracting rotations are not always equal, however. If they are equal, or if no rotations at all occur, the occlusal plane will be almost exactly perpendicular to the vertical **PM** plane. Often, however, the occlusal plane has a noticeable downward angulation because the amount of ramus remodeling realignment falls short of the downward displacement rotation of the whole mandible. One can "eyeball" how much downward occlusal plane rotation exists by visualizing it relative to the neutral horizontal axis of the orbit. If the two are parallel, the occlusal plane is perpendicular to the **PM** plane. In many individuals the occlusal plane angles downward, to a greater or lesser extent, and in a few it will angle upward. Persons with a vertically shorter nasal region tend to have a perpendicular or an upward occlusal plane alignment, or at least a much lesser amount of downward rotation. The occlusal plane in long-faced and long-nosed individuals tends to be downward-rotated to a greater extent. Failure to recognize occlusal plane compensations can be a major source of surgical treatment planning error in "short-faced" patients with relative mandibular prognathia. If the surgeon shortens the mandible when the patient really needs an occlusal plane rotation the resulting facial appearance is un-natural. Bimaxillary surgery is needed to increase facial height thus correcting the relative mandibular prognathia by introducing a ramus corpus rotation at the mandibular osteotomy site.

A closed alignment between the ramus and the corpus **shortens** overall mandibular length and thereby has a mandibular retrusive effect (Fig. 10-19*a*). An open alignment increases it and has a protrusive effect (Fig. 10-19*b*). There are two ways to illustrate why this occurs. First, the straight-line dimension (overall mandibular length) from *a* to *c* is decreased; the dimension from *b* to *c* is increased. Second, if the upper and lower arches *M* and *N* in Figure 10-20 are aligned upward, *M* protrudes beyond *N* by the distance *x* relative to the **occlusal plane** (not the vertical facial profile). When aligned downward, *N* protrudes by the distance *y* relative to the downward-inclined occlusal plane.

If the ramus-corpus angle is opened, the prominence of the antegonial notch is increased. This is caused by the downward angulation of the mandibular body at its junction with the ramus. If the ramus-corpus angle is **closed**, the size of

the antegonial notch can be reduced or obliterated entirely because of the upward alignment of the corpus relative to the ramus. (See Fig. 4-20.)

Note especially that the effects of whole-mandible rotations and ramus-to-corpus rotations are **opposite**. This is why the subject of mandibular "rotations" can be confusing. When the entire mandible is aligned downward (rotated clockwise), a mandibular retrusive effect is produced; but when just the corpus is aligned downward (rotated clockwise) relative to the ramus, a mandibular protrusive effect results (Fig. 10-21). An upward whole-mandible alignment (counter clockwise rotation) is mandibular protrusive, and an upward alignment of the corpus only is mandibular retrusive.

An individual can have a retrognathic profile and **not** have a Class II malocclusion, even though many of the underlying skeletal factors are the same for both. This is because different planes of reference relate separately to the profile and to malocclusions.

A forward-inclined middle cranial fossa has a two-way maxillary protrusive and a mandibular retrusive effect (Figs. 10-17 and 10-18). Because the expression of the effective horizontal (not oblique) dimension of the middle fossa is increased, the maxilla becomes offset anteriorly with respect to the mandibular corpus. The midface is also lowered, and this causes the whole mandible to rock down and back. The maxilla thus is carried forward, and the mandible is rotated backward in this composite, two-way movement. Mandibular retrusion results, even though the arch lengths of the upper and lower jaws can have equivalent dimensions, as shown here. These changes in skeletal pattern cause a Class II molar relationship because the lower bony arch is posteriorly offset.

A backward-inclined middle cranial fossa† has a mandibular protrusive effect. This contributes to a Class III type of molar relationship. The maxilla is placed backward, and the mandible rotates forward into a protrusive position. Note that the mandibular occlusal plane is rotated into an upward-inclined position. To compensate, as mentioned above, the posterior maxillary teeth can descend (drift

† Note this important point. The conventional way to represent the "cranial base angle" is by a line from basion to sella to nasion. Although useful in conventional cephalometrics, this is not the anatomically meaningful way to do it. The **real** relationship (so far as the face is concerned) involves the contact between the condyle and the cranial floor (thus not basion), and the junction corner between the cranial floor and the nasomaxillary complex (thus not sella). **This** is the relationship that **directly** determines the anatomic effects of the three-point contact among the cranial floor, the mandible, and the maxillary tuberosity. Basion-sella-nasion only indirectly reflects this and these three traditional landmarks have nothing to do with the actual anatomic fitting of the key junctions involved. They are removed **midline** structures that do not relate directly to the **lateral** positions of the upper and lower arches, the lateral contacts between the mandibular condyles and the cranial floor, and the lateral effects of the angle between the lateral parts of the floor of the middle and anterior cranial fossae relative to the maxillary tuberosities. Sella, basion, and nasion themselves can be almost **anywhere** among the midline axis, within normal variation limits, and not affect the "angle" that really counts: the angle from the temporomandible joint (TMJ) articulation to the point of junction between the middle and anterior cranial fossae, that is, the point where the nasomaxillary complex joints the cranial floor.

inferiorly) or the ramus-corpus angle is opened, or both.

It was pointed out above that if the nasomaxillary region is vertically long relative to the ramus and middle cranial fossa, the result is a downward and backward placement of the whole mandible to varying degrees in different faces (Figs. 10-14 and 10-22). Note the resultant mandibular retrusive effect, the retrognathic profile, and the skeletal basis for a Class II molar relationship. A forward alignment of the middle cranial fossa also causes a similar kind of mandibular rotation. If **both** occur in the same individual, the total extent of mandibular rotation is the sum of the two. (Dental changes can preclude an anterior open bite; see discussion of curve of Spee on page 89.)

FIGURE 10-19

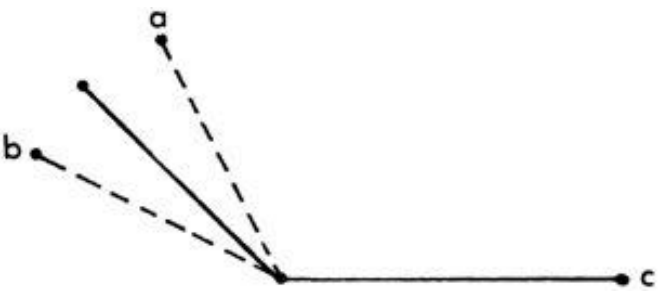


FIGURE 10-20

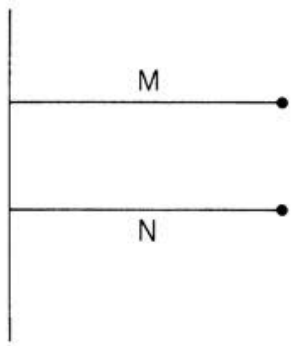
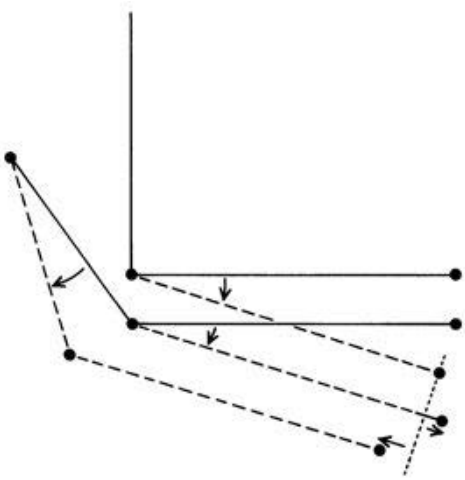
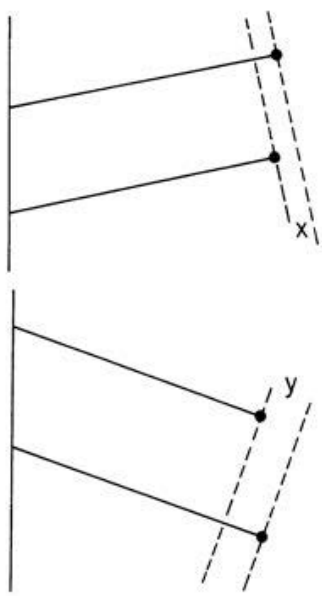


FIGURE 10-21





If the nasomaxillary region is vertically short, as noted earlier, a mandibular protrusive effect is produced (Figs. 10-15 and 10-23). The mandible rotates forward and upward, and the resultant offset positions between the maxillary and mandibular arches can contribute to a Class III type of molar relationship.

Note that a **vertical** imbalance has resulted in a **horizontal** structural effect.‡ It is, of course, incorrect to assume that malocclusions are based, essentially, only on horizontal dysplasias. A closed basicranial relationship is also mandibular protrusive and adds to the extent if involved together with a short midface.

All the above relationships illustrate the various effects of changes in the dimensions or the alignment of any **one** given region, as for the ramus, middle cranial fossa, maxillary arch, and so on. The skull of any given individual, however, is a composite of many combinations of such relationships among **all** the regional parts. Outlined below are examples of several different combinations of various regional dimensional and alignment imbalances and balances.

FIGURE 10-22

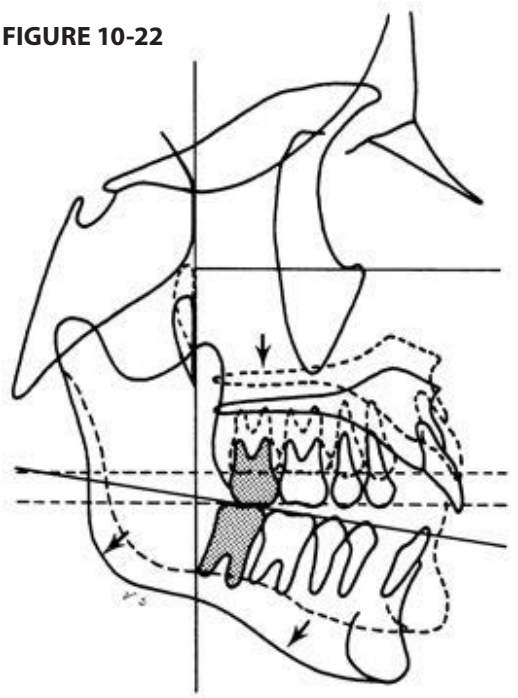
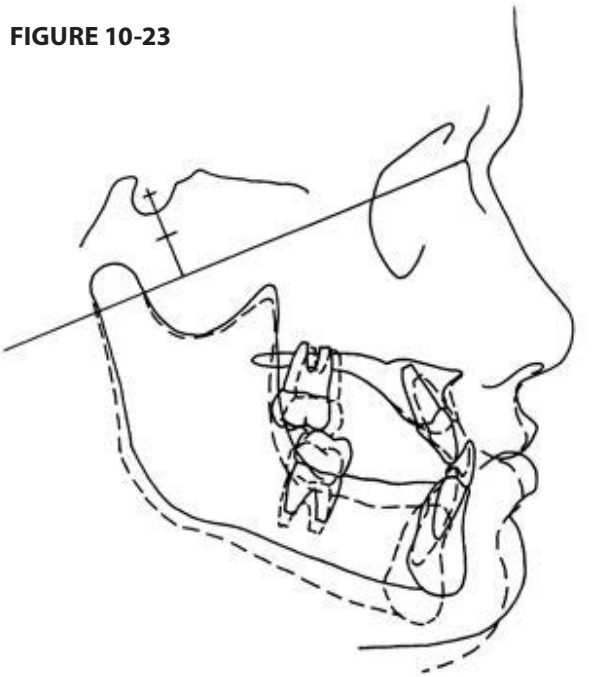


FIGURE 10-23



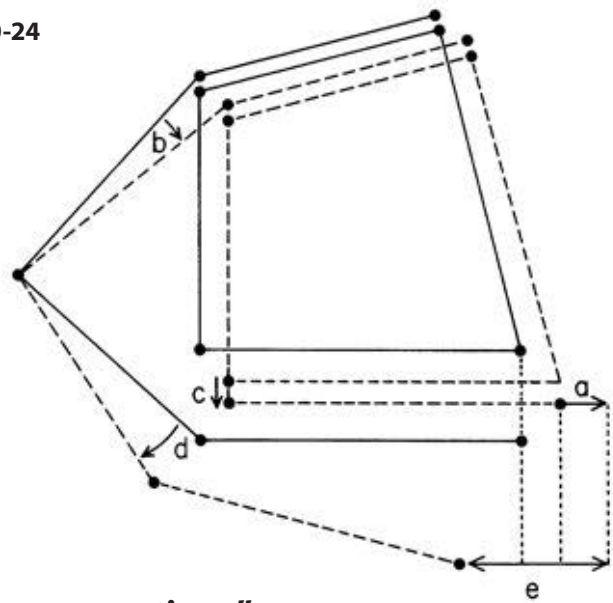
In the combination shown in Figure 10-24, the horizontal dimension of the maxillary arch exceeds that of the mandibular arch (*a*). The middle cranial fossa has a forward-inclined alignment (*b*), and the midface (*c*) is also vertically long. The mandible is rotated downward and backward (*d*). **All** of these features have mandibular retrusive effects, and their combined sum (*e*) results in a severe Class II malocclusion and severe retrognathia. Idealized treatment for this individual would require a treatment plan that addressed each imbalance. The development of a problem list (imbalance list) and intervention to address each problem is the essence of orthodontic diagnosis and treatment planning. For example, in the case shown in Figure 10-25, in a young child, the protrusion and vertical excess of the maxilla might be addressed using a high pull headgear and Temporary Anchorage

‡ This is important clinically because ideally the vertical **imbalance** must be addressed, not the horizontal **effect**. (See Fig. 10-23.)

Devices (TADs) to limit vertical drift of the dentition (*b*). The headgear would be designed to redirect maxillary forward development and the TADs used to modify vertical facial relationships. As the vertical imbalance improves, treatment may then address the mandibular shape using a vertical chin cup or Frankel FR IV appliance (*c*). In the adult, a treatment plan might include extraction of upper first bicuspids (to address the maxillary protrusion) followed by maxillary LeForte I surgery (to address the vertical) with autorotation of the mandible (to address displacement rotation of the mandible). A vertical reduction and advancement genioplasty (to address the remodeling rotation of the mandible) may also improve the final aesthetic result.

The combination schematized in Figure 10-26 illustrates a horizontally short mandibular corpus (relative to the individual's maxillary arch) in combination with a backward-rotated middle cranial fossa, a forward-rotated mandible, and an opened ramus-corpus angle. The composite result is an individual with a Class II type of lower arch, a Class III molar relationship, a Class III type of basicranial alignment, and a Class I (orthognathic) type of profile because of the contrasting retrusive/protrusive combination.

FIGURE 10-24



Growth "Compensations"

A more biologic heading for this intrinsic growth process is "developmental adjustments working toward balance," as outlined in Chapter 1. The factor of morphologic adjustment during facial development is a basic and important biologic concept. The compensatory process involves latitudes of morphogenetic give and take among the various regional parts as all grow in close interrelationship. The composite result is a state of functional and structural equilibrium. Indeed, growth is a constant, ongoing, compensatory process striving toward ultimate homeostasis as a bone grows in relation to its developing muscles, as connective tissue grows in relation to both bone and muscle, and as blood vessels, nerves,

FIGURE 10-25

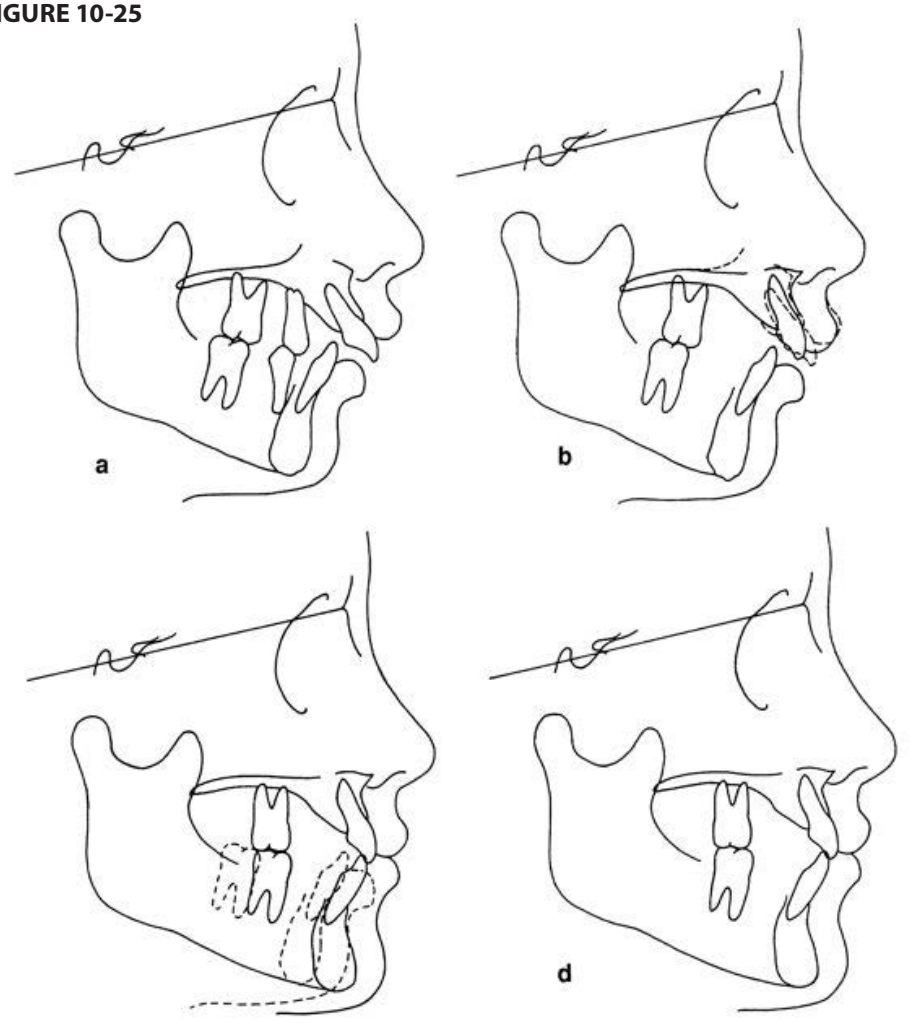
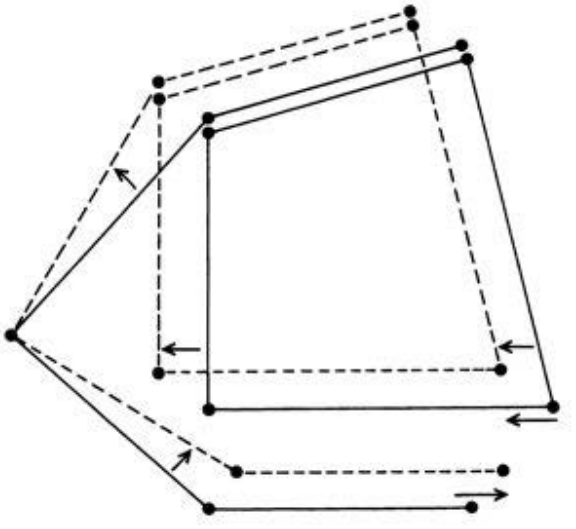


FIGURE 10-26



epithelia, and so forth all develop in relation to everything. When the growth process is "complete," a state of compromise equilibrium has been achieved, even though a malocclusion or some other dysplasia may exist. There nearly always exist a number of regional morphologic imbalances to greater or lesser degrees of severity, but the aggregate construction of the craniofacial composite as a whole is functional, albeit with multiple regional variations, some of which likely depart from a population mean.

A frequently encountered compensatory combination involves the ramus of the mandible. When the nasomaxillary complex is vertically "long" and/or the middle cranial fossa has a forward-downward rotational alignment, the whole mandible consequently becomes rotated into a downward-backward placement. As described previously, these are significant factors that underlie the skeletal basis for a retrusion of the mandible and a Class II molar relationship. However, developmental processes can respond by a widening of the horizontal breadth of the ramus. This compensatory adjustment places the mandibular arch more protrusively, thereby partially or totally counteracting the extent of its backward rotation. What would have been a Class II malocclusion and a retrognathic profile have been converted into a Class I occlusion, and the severity of the potential malocclusion has been reduced. Should compensation fail entirely, the malocclusion becomes fully expressed.

Understand that, in carrying out this compensatory role, the ramus does not itself respond as though it has a brain of its own and somehow elects to do something good. As pointed out earlier, growth is a prolonged process striving toward functional and structural equilibrium. The skeletal response by the ramus is a result of continuous remodeling actions by its "genic" tissues receiving instruction signals paced by the growth and function of the masticatory muscles; the airway; the pharyngeal muscles, and mucosa, tonsils, tongue, lips, cheeks, connective tissue, and so on, all of which develop in a composite, interrelated manner that has a latitude for adjustment to the growth and morphology of other, contiguous regions (e.g., the basicranium, nasal region, and oral complex). If such latitude is not exceeded, at least a partial compensatory relationship can be achieved during the growth period. When growth has become completed, the capacity for compensation by parts remodeling to adjust to other parts, as a component of growth, becomes diminished. The potential in the adult, thus, is less (Fig. 1-1).

Other examples of compensatory developmental adjustments, including palatal rotations, anterior crowding, gonial angle remodeling, and occlusal plane rotations, are described elsewhere in this and other chapters. It is apparent that a Class II malocclusion is **not** merely caused by a "long maxillary arch" or a short mandible. Malocclusions are quite multifactorial because of the complex architectonics involved.

Dentoalveolar Compensations

During the development and establishment of the occlusion, ongoing and intensive adjustments occur involving dentoalveolar remodeling as well. The functional placement of the teeth is very much a part of growth. The mobility of

the teeth allows responses to the many skeletal and soft tissue growth processes taking place throughout the face and cranium. A basic point to keep in mind is that, unlike bone, a tooth is not self-mobile by its own remodeling process. It must be moved by forces extrinsic to it. Figures 10-27 to 10-31 explain some common changes involved.

In the first diagram (Fig. 10-27), the vertical and horizontal dimensions among the various skeletal parts and counterparts are in balance. The alignments of all the parts also are in "neutral" positions. That is, the nature of the alignments is such that neither protrusion nor retrusion of the upper or lower jaws is produced; the angular relationships are balanced so as not to increase or decrease the "expression" of any of the various key dimensions. Note that the occlusal plane is perpendicular to the vertical reference line (the PM plane) and parallel to the neutral orbital axis (shown here below the orbit, rather than within its geometric center).

The nasomaxillary complex in the next stage (Fig. 10-28) has become lengthened vertically to a greater extent. This is common, as described earlier. The amount of midfacial growth has **exceeded** the vertical growth of the ramus-middle cranial fossa composite. The result is downward-backward alignment of the whole mandible to accommodate the longer nasomaxillary complex. A vertical "imbalance" has thus been introduced, and the expression of the vertical ramus height has been increased to match it by a downward mandibular rotation. (This same effect on the mandible can also be caused or augmented by a proclination of the middle cranial fossa, as previously described.) Note especially that the mandibular corpus, and with it the lower teeth, now has a consequent downward inclination relative to the vertical **PM** line. This "opens" the anterior bite; only the second molars are in occlusal contact. The amount of occlusal separation increases toward the incisors.

Note also the retrusion of the mandible, overjet, and the Class II molar relationship caused by the mandible's rotation. The upper teeth "drift" (**not** simply erupt) inferiorly until each comes into contact with its antagonist (Fig. 10-29). The last molars were already in contact; the second premolar must drift downward only a short distance. The first premolar drifts inferiorly even more because of the greater gap involved. The central incisors move down the greatest distance. As a final result, full arch-length occlusal contact is attained. The occlusal plane is **straight** (not curved, as in other variations described below). The occlusal plane bisects the upper and lower incisor overlap, just as it did in the first, "balanced" stage. The occlusal plane, however, is now inclined obliquely downward.

Another common adjustive combination may occur. The upper teeth drift inferiorly, but the canines and incisors do not move down to the full extent needed to completely close the occlusion, only to about the same extent that the premolars drift inferiorly (Fig. 10-30).

The anterior **mandibular** teeth, however, drift superiorly until full arch occlusal contact is reached (Fig. 10-31). The lower incisors must move upward much more, however, than the cuspids and premolars. Note that the roots of the anterior teeth have become realigned as all of the teeth are repackaged, and that the cusps of the lower incisors and canines are **noticeably much higher** than the premolars and molars.

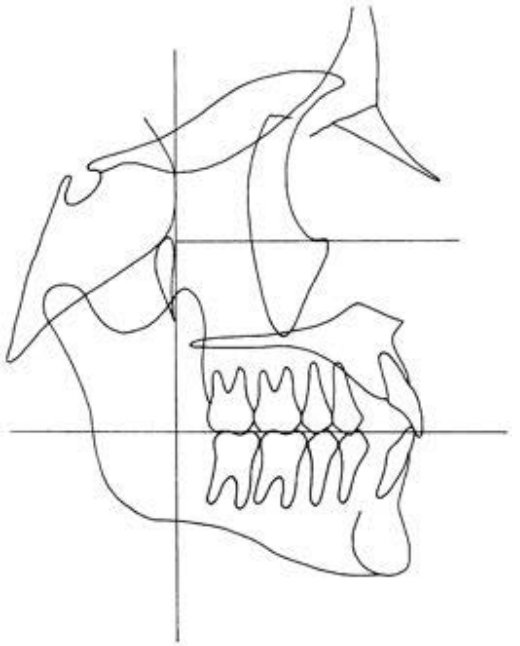


FIGURE 10-27.
(From Enlow, D. H., T. Kuroda, and A. B. Lewis: The morphological and morphogenetic basis for craniofacial form and pattern. Angle Orthod. 41:161, 1971, with permission.)

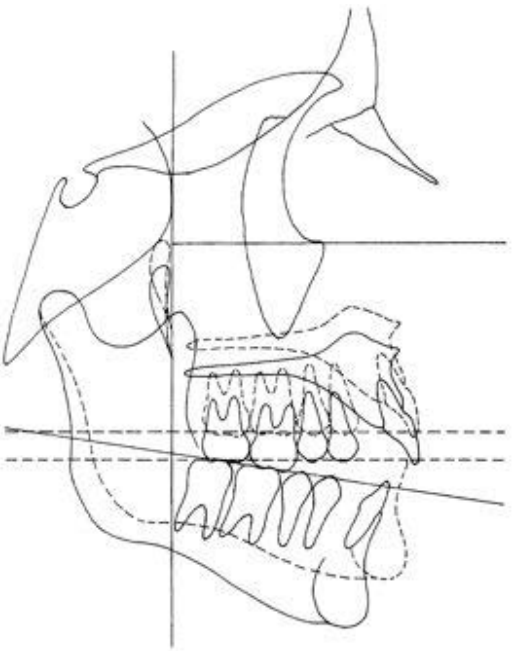


FIGURE 10-28.
(From Enlow, D. H., T. Kuroda, and A. B. Lewis: The morphological and morphogenetic basis to craniofacial form and pattern. Angle Orthod., 41:161, 1971, with permission.)

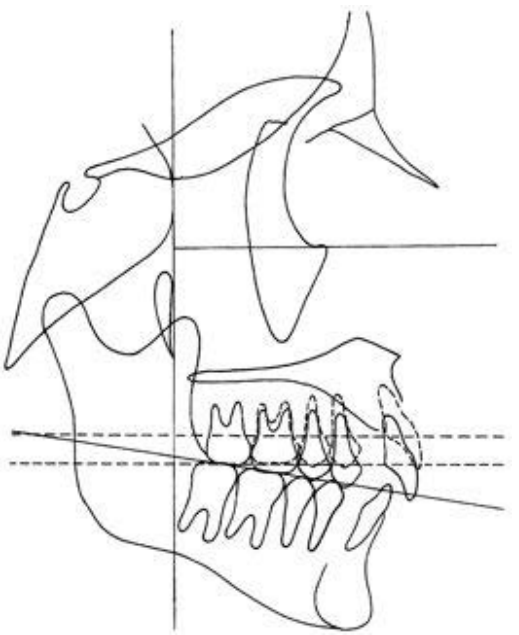


FIGURE 10-29.
(From Enlow, D. H., T. Kuroda, and A. B. Lewis. The morphological and morphogenetic basis for craniofacial form and pattern. Angle Orthod., 41: 161, 1971, with permission.)

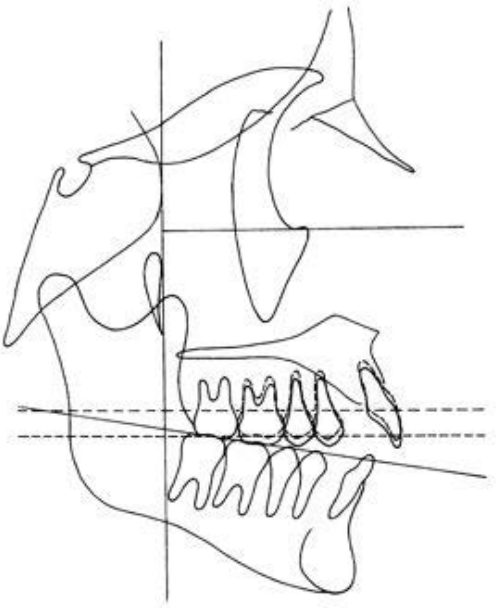


FIGURE 10-30.
(From Enlow, D. H., T. Kuroda, and A. B. Lewis. The morphological and morphogenetic basis for craniofacial form and pattern. Angle Orthod., 41: 161, 1971, with permission.)

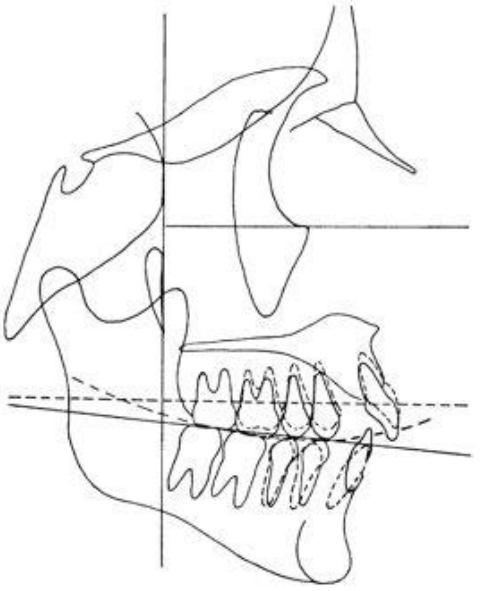


FIGURE 10-31.
(From Enlow, D. H., T. Kuroda, and A. B. Lewis: The morphological and morphogenetiuc basis for craniofacial form and pattern. Angle orthod., 41:161, 1971, with permission.)

Dentoalveolar Curve (of Spee)

There are two ways to represent the **occlusal plane**. The traditional method is to draw a line along the contact points of all the teeth to the midpoint of the overlap between the upper and lower incisors. In the first two examples cited above, this line is straight. In the last, however, note how the line is curved as it exactly bisects the overlap of the upper and lower incisors. This is called the **curve of Spee** or the dentoalveolar curve, and the reason for its development was just outlined above. A second way to represent the occlusal plane is to run a line from the posterior-most molar contact point straight to the anterior-most premolar contact point. The incisors are not considered. This is termed the "functional occlusal plane," and it is always a straight line whether or not a curve of Spee exists.

In the first and second examples of occlusal development (Figs. 10-28 and 10-29), a curve of Spee did not develop, and the two methods for representing the

occlusal plane result in the same line. In the last example, however, the curved occlusal plane bisecting the incisor overlap and the straight functional occlusal plane are divergent. Note how the mandibular incisors rise considerably above the level of the functional occlusal plane. The maxillary incisors, however, fall well short and do not even touch this straight-line functional occlusal plane. In individuals having a marked curve of Spee, the alveolar region of the mandible just above the chin is characteristically more elongate because the incisors have drifted superiorly for several millimeters or more.

The dentoalveolar curve (of Spee) is a common developmental adjustment that provides intrinsic compensation for an **anterior open bite**. A combination of several factors underlies the skeletal tendency for this type of malocclusion. If (1) normal long-faced development or, also, if airway (or other) problems lead to an opening of the ramus-corpus (gonial) angle; (2) if the whole mandible is displacement-rotated down and back as explained previously; and/or (3) if a counterclockwise rotational alignment of the palate and maxillary arch occurs because of displacement by the anterior cranial fossa, the conditions predisposing an anterior open bite can converge to cause this developmental variation. The vertical drifting (not simply eruption) of the anterior mandibular teeth can then close what would otherwise be a skeletal (not merely dental) gap. Should this intrinsic process fail, the open bite thereby expresses itself fully. Relapse is a frequent problem because these predisposing conditions are not fully eliminated by treatment, and the resultant imbalance activates the growth process to return to a state of balance. Conversely, if a **deep overbite** occurs, it is often in patients who have a horizontally short mandible in conjunction with a closed ramus-corpus angle, a clockwise rotational alignment of the palate and maxillary arch, and a deep curve of Spee. It is important for clinicians to understand the compensatory role of the curve of Spee and to make a thoughtful decision to either eliminate or keep this compensation. "Knee-jerk" leveling of the curve of Spee is a recipe for iatrogenic open bite.

Another kind of dental compensation also commonly occurs. It was underscored earlier that the teeth have only a very limited capacity for remodeling (particularly after they have become fully formed). That is, a tooth cannot become markedly reshaped by selective remodeling resorption and deposition of dentin and enamel throughout its various areas to accommodate spatial and functional relationships; only a relatively limited extent of root resorption, deposition of cementum, trajectory of root growth, and crown wear is possible in this regard. This means that most adaptive adjustments for a tooth must be carried out by the "displacement" process. While extensive resorptive and depository remodeling is a basic growth function for the housing alveolar bone, it is not a factor for the tooth itself. If, however, the capacity for this bone remodeling is exceeded, as for example, by an alveolar arch that is too small for the teeth it must support, the developmental and functional recourse then is displacement of some of the teeth. Thus, **anterior crowding** is, in effect, a compensatory means by which the teeth are housed beyond the limit (growth field) provided by the available bone and its growth and remodeling potential. The treatment options for alignment of crowded lower teeth are (1) to increase the available alveolar bone for tooth alignment or

(2) to reduce the number of teeth to compensate for the lack of alveolar bone. Importantly, since the anterior mandibular alveolar surface is resorptive and its growth field boundary constrained, option 2 is often the biologically sound treatment decision.

SUMMARY OF CLASS II AND CLASS III SKELETAL FEATURES

In the Class II individual (Fig. 10-32), note that the mandibular arch is short relative to the maxillary arch. The mandibular arch in the Class III individual (Fig. 10-33), conversely, is horizontally long relative to the maxillary arch, its counterpart.

The middle cranial fossa in the Class II individual has a forward and downward-inclined alignment. In the Class III individual the middle cranial fossa is aligned backward and upward. The nasomaxillary complex is thereby placed more retrusively in the Class III individual and more protrusively in the Class II individual. Rotations of the mandible are also involved (see below).

The nasomaxillary complex in the Class II individual is vertically long relative to the vertical dimension of the ramus (or the ramus is short relative to the maxilla). This long midface, together with the downward-forward alignment of the middle cranial fossa, causes a downward-backward rotational alignment of the whole mandible in the Class II individual.§ The Class III mandible is rotated forward in conjunction with an upward-backward middle cranial fossa rotation and a vertically short nasal region. The midface is short relative to the vertical dimension of the ramus (or the ramus is long relative to the maxilla; either way, since it is a relative comparison). Although the face of the Class III individual "looks" quite long (Fig. 10-33), it is the lower face (mandible), not the nasal region, that causes this.

In Class II individuals, the headform type is often dolichocephalic or mesocephalic. The anterior cranial fossa is thereby relatively long and narrow; and because it is the template for the nasomaxillary complex, the palate and maxillary arch are correspondingly elongate and narrow. In Class III individuals, conversely, both the anterior and middle cranial fossae tend to be wider and shorter (brachycephalic), and this thereby establishes a foreshortened, but wider palate, maxillary arch, and pharynx.

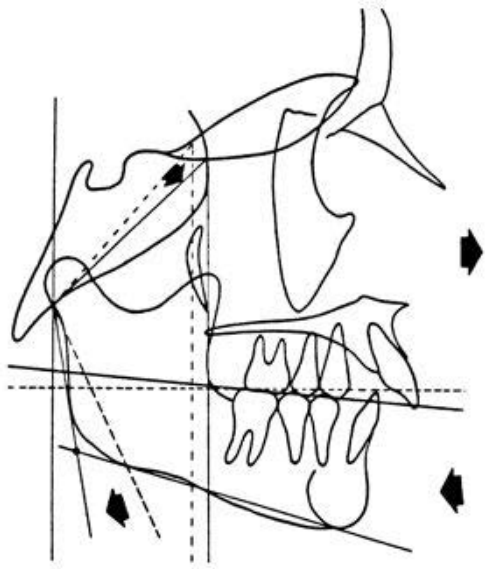
The ramus-corpus (gonial) angle is more closed in the Class II, but open in the Class III face, thereby shortening and lengthening, respectively, overall mandibular length. In the Class III face this produces the characteristic steeply angled alignment of the mandibular corpus.¶ Note that the anterior mandibular teeth in the Class III individual have drifted upward to a considerable extent

§ The dashed lines in the figures represent "neutral" alignment positions. See Enlow et al., 1971a, for the biologic rationale involved.

¶ Note this significant point. The Class II face can have a steep mandibular plane angle, thereby appearing similar to the downward-inclined mandibular corpus of the Class III face, which also is a "steep plane" (Fig. 10-31). The underlying reasons, however, are different and should not be confused. In the Class II, it is a **whole-mandible displacement** rotation. In the Class III, it is a **ramus-to-corpus remodeling** rotation. The former relates to a **long** midface, whereas the latter relates to a **short** midface, a significant distinction often overlooked!

(a compensatory adjustment to close the open ramus-corpus angle), so that the occlusal plane itself is not angled as sharply downward. This causes the characteristically elongate, high alveolar region above a prominent-appearing chin observed in many Class III faces.** It is interesting to note that, although the anterior mandibular teeth drift markedly superiorly in the Class III, they do not rise above the functional occlusal plane to form a dentoalveolar curve. A marked extent of compensatory downward drifting of the maxillary dentition may also occur in many cases (as seen in Figure 10-33) to compensate for the vertically short nasal region. In the Class II face, conversely, it is the **nasal region** that appears relatively elongate vertically, with a much shorter appearing vertical depth in the region of the chin. Note that the Class II maxillary apical base is **much closer** to the palate compared to the **more downward-drifted** maxillary teeth in the Class III. However, some Class II individuals may show a steeply inclined mandibular plane causing a lengthened, but still retrusive, appearance of the lower face. This results when the downward-backward alignment of the whole mandible (a displacement rotation produced by the Class II long face/short ramus/open middle cranial fossa relationships described earlier) is not accompanied by closure of the gonial angle. A deeper compensatory curve of Spee often develops.

FIGURE 10-32



To date, the combination of these multiple features contributes to the composite skeletal basis for mandibular retrusion in the Class II individual and mandibular protrusion in the Class III individual. However, note that the Class II ramus is horizontally broad and that the Class III ramus is narrow. These are,

** An interesting and not uncommon Class III variation occurs in the dolichocephalic, rather than the usual brachycephalic headform. It is related to Class I individuals having a strong Class III tendency, except that underlying mandibular protrusive features dominate over the long, narrow (leptoprosopic) maxillary protrusive features also present. The result is an elongate, narrow head with a long, pointed nose, but also with a contrasting protrusive, rather than retrusive, mandible (as in the classic "wicked witch"). See Martone et al., 1992.

as explained earlier, compensatory features that partially counteract the other characteristics that combine to cause Class II mandibular retrusion and Class III protrusion, respectively. The resultant malocclusions are thereby less severe than they would have been had the ramus in each been of "normal" dimension. Had the ramus actually been narrow in the Class II individual and wide in the Class III individual, they would, of course, have added to (rather than subtracting from) the composite basis for the malocclusion.

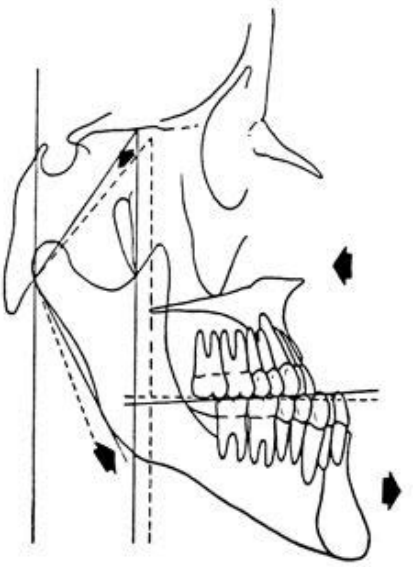


FIGURE 10-33.
(From Enlow, D. H., T. Kuroda, and A. B. Lewis: The morphological and morphogenetiuc basis for craniofacial form and pattern. Angle Orthod., 41: 161, 1971, with permission.)

Most Class II individuals thus have an anterioposteriorly short mandibular corpus, a vertically long nasomaxillary complex, a whole maxillary dental arch that drifts inferiorly much less than in Class III but in which the anterior teeth drift down more than the posterior teeth, a downward-and backward-aligned mandible, a forward and downward middle cranial fossa alignment, a closed ramus-corpus (gonial) angle, and (in severe malocclusions) a narrow ramus and a long fore-and-aft middle cranial fossa.

The **converse** of all these regional relationships characterizes the Class III malocclusion. Each such feature occurs in about 70 per cent or more of Class II and III individuals respectively. What about the other 30 or so per cent? This is where "offsetting penalties" come into play. Instead of a forward-inclined, dolichocephalic type of middle cranial fossa and/or a long midface causing mandibular retrusion, for example, a given individual can have a **backward**-aligned, brachycephalic type of fossa and a more pug-like nasal region. These features may then combine with one or more other regional mandibular **protrusive** features, such as, perhaps, a broad ramus, a long corpus, or an open gonial angle, to partially counteract the various mandibular retrusive factors also present. In any given individual, the sum of the dimensional values for all the mandibular protrusive features weighs against

the sum of the values for all the mandibular retrusive features. Either they come into a effective balance that zero's out, or one or the other wins. If the mandibular retrusive features dominate, the **severity** of the resultant Class II malocclusion and retrognathic type of face depends, first, on how much (in millimeters) the total of these retrusive features amounts to, and second, how much the counteracting features subtract from this total.

Each of us has a natural, normal predisposition toward either mandibular retrusion (Class II) or protrusion (Class III). A fundamental concept is that there is no such thing as a "separate" Class I facial category. A pervasive misconception is that the Class I craniofacial composite is essentially "all normal and balanced except for minor irregularities." All Class I individuals, however, have a predominant **tendency** one way or the other toward a composite retrusive or protrusive malocclusion. Most narrow- and long-faced Class I individuals have the **same** underlying facial and cranial features that are present in the long-faced Class II individuals. The same percentage of the various mandibular retrusive relationships described above occur in both. This is why a Class II predisposition usually exists to a greater or lesser extent. The difference between the Class I and II malocclusions, however, is the extent and magnitude of the imbalances and the number and extent of counteracting features. If the compensating characteristics are adequate, a more or less balance of imbalances in a Class I face results. If they partially or totally fail, marginal to severe malocclusion and facial disproportion results. A person having an attractive, well-proportioned face, with an orthognathic (or nearly so) **profile** and only relatively minor occlusal irregularities **also** has, unsuspected by him or her and deep within the face and cranium, the **same** underlying characteristics that caused a cousin to have a noticeably retrognathic profile and a Class II malocclusion. Our hero, however, has a particularly broad ramus and some other happy offsetting characteristics that are winners for him as an individual. Most of us have at least a reasonable-appearing face, although somewhat short of perfect, for the same reasons.

A fundamental principle to keep always in mind is that the growth process is continuously creating imbalances as the muscles and the airway, for example, continue to develop. At the same time, however, the growth process is **working toward** an aggregate state of composite functional and developmental equilibrium among all the separate multitudes of parts. (See Chapter 1.)

THE FACIAL SPECTRUM

The conventional perception of pattern variation is that there are essentially three principal facial types, each associated with one of the three chief malocclusion categories. These are, simply, mandibular protrusion (Class III), mandibular retrusion (Class II), and normal or nearly so (Class I). Even as a generalization, however, this perception bypasses significant morphologic and key developmental points and precludes a much more basic biologic awareness of the factors underlying facial form and pattern. It is not possible, for example, **not** to have underlying malocclusion tendencies in the Class I, whether or not fully masked by the growth process, considering our unique craniofacial character.

As already described in the other chapters and in the pages above describing facial component combinations, the multiple structural reasons for the wide variations in facial form are cataloged. How the nature of the anatomic combinations leads to a **spectrum** of facial patterns is now outlined.

The Continuous Facial Sequence and Developmental Intergrades

If **all** of the regional anatomic conditions (1) having a mandibular **protrusive** effect should exist in a given person, and (2) each regional effect is **severe** within the bounds of workable function, and (3) no significant compensatory adjustments occur, **then** the aggregate result would be a severe pattern of mandibular protrusion and an extreme Class III malocclusion. This is one end of a continuous morphologic spectrum. It extends on to the other (opposite) anatomic end representing retrognathia and severe Class II malocclusion in which all of the opposite regional features exist and without significant developmental compensatory adjustments. Between these opposite extremes lies an infinite spectrum of **mixes** that grade from each end toward the middle. Facial taxonomists have divided the whole into the three general groups, I, II, and III. An important point is that the middle span (Class I) **is not** comprised of individuals in which no retrusive or protrusive regional features exist at all—that is, everything is actually quite neutral and nearly perfectly constructed with only minor misfits among parts. Quite the contrary, as described next.

Working toward a retrusive/protrusive overlap in the middle of the spectrum, two factors are in play that create **gradations**. First, the severities at both ends decrease for some (not necessarily all) of the regional "trusive-causing" relationships. Second, a contrasting **mix** of relationships, with compensations added, occurs in a progressive direction toward the middle overlap. The result is a gradation away from "severe" until both ends meet at the transition crossover from one side to the other. This point is different in every individual person because of the infinite variability of the magnitudes and character of the mix.

In all cases, it is important to understand that the built-in, underlying protrusive/retrusive tendencies relating to headform variations (Chapters 1, 8, and 10) **are still present**, but are masked because of the offsetting combinations of contrasting local features. For example, palatal and ramus remodeling can help offset a maxillary displacement; or, upward dentoalveolar remodeling (curve of Spee) offsets a downward-backward mandibular alignment relating to an elongate nasomaxillary complex.

Just where the central crossover point exists is subject to a taxonomist's definition and a value judgment. Then, an arbitrary boundary is designated on either side of the point as definition of the Class I span. The serious misconception arises, however, that no significant malocclusion-causing factors now exist within this middle span because everything is virtually neutral or nearly so and within the bounds of our definition of normal. What, in fact, does indeed exist within this middle span is a composite of regional variations that are protrusive or retrusive

causing but, in combination, balance each other out.†† This is a point that needs really serious consideration.

The Class I category, thus, is not in itself an anatomically discrete group. It is, rather, a blend of contrasting features that more or less nullify each other to an extent that is intermediate between the groups on either side having blends weighted either more toward retrusion versus protrusion. An important point, as a bottom line, is that **Class I is not a homogeneous grouping**. To regard it as so is to mask significant variations within it. Most individuals are either on the Class II or the Class III **side**, depending on their personal mix of regional features. Thus, some persons are Class I with an underlying Class III tendency, others with a Class II tendency. Each will likely respond quite differently to treatment procedures (Enlow et al., 1988). Interestingly, a Class I more on the Class II side is actually more closely related morphologically to a Class II than to a Class I on the Class III side. To regard all Class Is, thus, simply as a single, structurally neutral and homogenous group, without taking these contrasting anatomic factors fully into account, is most regrettable. It disguises significant underlying morphology and developmental tendencies. This is a clinically **most** relevant point because divergent vectors of growth are involved. Since the clinical objective is to "work with growth" (i.e., to understand what is happening in order to manage it), the contrasting conditions involved are obviously fundamental factors.

The increased use of digital records makes it feasible to create large databases of clinical information. With a large sample it may be possible to conduct research studies **that take into account** these anatomic and developmental distinctions. In most studies of orthodontic treatment outcomes the within group variation is often greater than or equal to the between group variation. This leads to nondiscriminating findings because results are mixed into a common pot and all the subgroups simply cancel each other out. Careful analysis of pretreatment variation is a most important consideration and has almost never been a factor in most clinical and research studies.

A final and significant point is drawn from the spectrum of these morphogenic and facial assembly patterns. It is significant because of the great potential for clinical fine-tuning. The point is that, because of the structural spectrum involving mixes of the retrusive and protrusive original combinations, Class I, II, and III *subgroups* can be distinguished. Although not yet named or formally designated, their existence is real. That subgroupings demonstrate different developmental lines is known (Enlow et al., 1988; Martone et al., 1992, Choi, 1993). The degree to which these subgroups vary in their morphogenic responses to treatment procedures would be important knowledge to be gained in the future.

†† This factor might help "explain" the clinical case that "appeared" to be a simple Class I and during treatment "turned into" a Class II or III. The Class II or III components were there all along, and they became more fully expressed during treatment.

11

The Structural Basis for Ethnic Variations in Facial Form

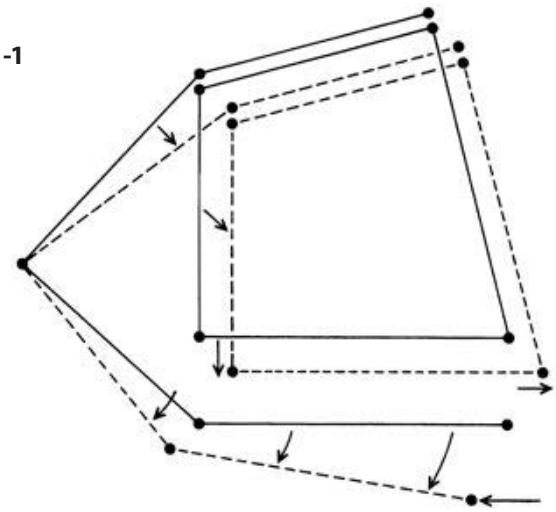
Age, sex, and population differences in the pattern of facial structure have been pointed out in the preceding chapters. The purpose of this section is to summarize this information briefly and add to it as a separate topic. Although this is an interesting subject in its own right, it is quite important for the clinician to realize that population norms derived from a given sample are not necessarily valid or accurate for other samples or groups, especially if ethnic and geographic variations are involved.

The phylogenetic basis for the unique construction of the human face was outlined in Chapter 9. It will be recalled that both the shape and size of the brain are key factors relating to the structure of the face. Because the basicranium is the bridge between them, and because the floor of the cranium is the template upon which the face is constructed, variations in the shape of the brain in **any** species are associated with corresponding variations in the form of the face. For example, the junctional part of the midface can only be as wide as the floor of the cranium. It cannot be wider because there is nothing to attach it to. Thus, narrow-brain species or subgroups are correspondingly narrow faced. Compare the face of the long, narrow-brained collie dog with that of the short, round-brained boxer or bulldog. Proportionately, man has an exceptionally wide face, in comparison with the typical mammal, because of the colossal size and the shape of the brain. The various rotations of the olfactory bulbs, orbits, and so forth (caused by the brain's characteristics) combine with the boundaries of the brain to establish, in all species, the amount and the principal directions of facial growth. Because of these factors, the shape and size of the brain are involved, also, in the variations of facial pattern **within** any given species or population group, as well as between them. There are, however, other factors that come into play, as will be seen.

Human population groups having a dolichocephalic headform naturally have a proportionately more narrow and longer face than those with a brachycephalic type of headform. The wider brain (with no special difference in overall volume) has the proportionately wider face. It has been claimed that there is an evolutionary (secular) trend toward the brachycephalic type among human groups. If this is happening, there will also be, as well, related long-term population distribution changes in facial structure, the nature of built-in tendencies toward malocclusions, and profile features.

The more open ("flat") cranial base flexure that usually characterizes the dolichocephalic headform in many Caucasian groups sets up a more protrusive upper face and a more retrusive lower face (Figs. 11-1, 11-2, and 11-3, bottom). The whole nasomaxillary complex is placed in a more forward position, and it is lowered relative to the mandibular condyle. Because the midface is relatively long, there is the tendency for a downward and backward rotation of the whole mandible. The posteroanterior dimension of the pharynx is large because of the longer and more horizontally aligned middle cranial fossae. Because the anterior cranial fossae are elongate and narrow, the palate and maxillary arch are correspondingly long and narrow. The extent of nasal protrusion is quite marked, and the outer cortical table of the forehead remodels anteriorly contiguous with a high nasal bridge. A large frontal sinus is thereby formed between the inner and outer tables. The forehead is much more sloping as a result, and the glabella becomes noticeably protrusive. The eyeballs are deep-set. The cheekbones often appear less prominent and more "hollow" because the remainder of the upper and the middle face are so protrusive. Because the mandible is rotated posteriorly, it tends to be retrusive, and the whole profile takes on a characteristic convexity for all these reasons. A Class II tendency (i.e., maxillary protrusion and/or mandibular retrusion) is **built in**. There is also a high incidence of a broad ramus to compensate, at least in part, for the tendency toward mandibular retrusion.

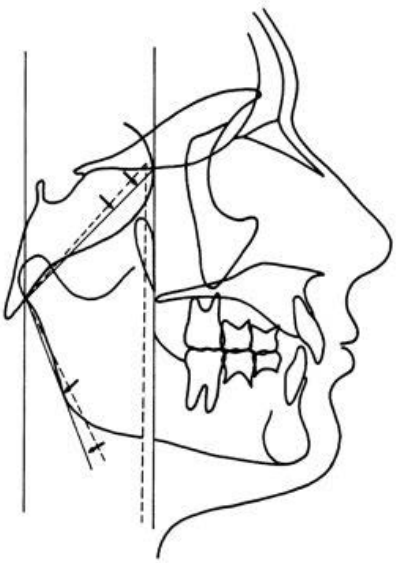
FIGURE 11-1



The more closed, upright basicranial flexure that usually characterizes the brachycephalic head sets up a correspondingly wider, flatter, more upright type of face (Figs. 11-3, top, and 11-4). The rounder, horizontally shorter brain and correspondingly foreshortened anterior cranial fossa establish a wider but anteroposteriorly shorter upper and midfacial region. The palate and dental arches are thereby also foreshortened, but relatively wide. The whole upper and midfacial region is also placed less protrusively because of the more upright middle cranial fossa. The middle cranial fossa, and, therefore, the pharyngeal region, is anteroposteriorly shorter for the same reasons. This further decreases the relative extent of the upper and midfacial protrusion. In addition, the upper part of the

ethmomaxillary complex does not expand anteriorly to nearly the same extent described for the previous facial type. The wider, shorter nasal and pharyngeal airway is approximately equivalent in capacity to those of other facial types having a much greater extent of nasal and maxillary protrusion but with a narrower passageway. The composite result is a more upright and bulbous forehead, less protrusion of the glabella and eyebrow ridges, a thinner frontal sinus, a much lower nasal bridge, a shorter pug-type nose, more shallow orbits and less deep-set eyeballs, and a tendency for a forward rotation of the entire mandible (unless offset by a vertical lengthening of the midface, and vertical drifting of the dentoalveolar arch, which is a feature in some, but not all, individuals.) The face appears flatter, broader, and squared. The cheekbones are more prominent appearing because the remainder of the upper and middle face is not as protrusive. There is a greater likelihood for an orthognathic (straight) profile, and the chin along with the entire mandible appear prominent. A greater tendency for bimaxillary protrusion with a Class III type of malocclusion and a prognathic mandible exists. In the brachycephalic (euryprosopic) face, the eyes can "look" wide-set because the nasal bridge is low. In some sub-groups, the nasomaxillary complex can be relatively long vertically, and the mandible can thus rotate downward and backward (Fig. 11-5) rather than anterosuperiorly (Fig. 11-4). The mandibular corpus tends to be shorter relative to the maxillary arch in some groups, and these mandibular features contribute to a compensation for the built-in tendency toward prognathism and bimaxillary protrusion.

FIGURE 11-2



It is important to realize that any predominantly brachycephalic population embodies a great range of variation from the "typical," as outlined above, to a mix of underlying facial features that grade toward the dolichocephalic/leptoprosopic form. The Far-Eastern (Oriental) populations, for example, do not represent a single, homogeneous grouping, but, rather, a composite assemblage of many geographically, environmentally, and morphologically diverse subgroups that

have evolved into quite distinctive and variably dissimilar craniofacial types. In contrast to the very round and flat-faced pattern, a more leptoprosopic, angular, long, and thin-nosed form also exists. The extent to which such facial variation relates to different anatomic types of malocclusions, and responses to different clinical interventions, is not well catalogued. Some anatomic and morphogenetic descriptions of these variations as they relate to upper respiratory behavior have been published (Hans et al. 2001, Cakirer et al. 2001).

FIGURE 11-3

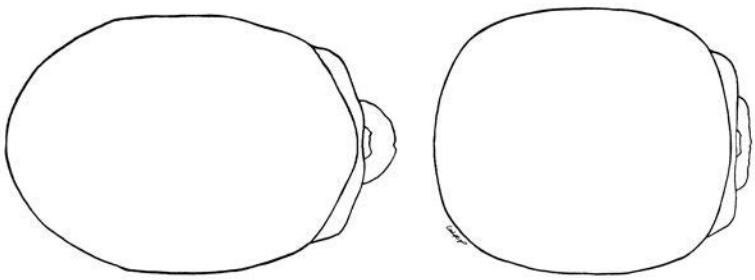
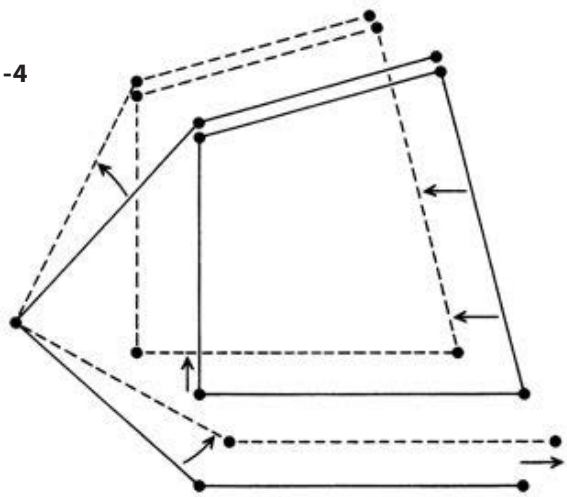


FIGURE 11-4

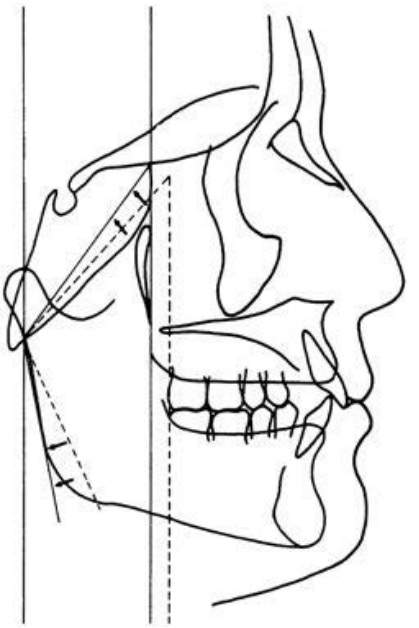


The above features characterize the Oriental face,* as well as certain Caucasian groups that also have a rounder brachycephalic ("Alpine") type of headform with many of these same facial features. (This does not include the dinaric headform, which is a fundamentally separate brachycephalic category.) The brachycephalic Caucasian type of face, like many Oriental faces, is wider, the nasal bridge lower, the nose flatter and shorter, the midface variably shorter, the forehead more upright, and the mandible more prominent. There are fewer underlying Class II tendencies in this basically different type of Caucasian face. Class I individuals having this

* The information in this section is based on investigations carried out by the author in collaboration with Dr. Takayuki Kuroda of the Tokyo Medical and Dental University.

composite facial structure tend toward a more orthognathic type of profile. When a Class II malocclusion does develop, however, it is a different kind (see Enlow et al., 1988). Care must be taken by the orthodontist because there are often stronger mandibular protrusive factors operating within this face. In this type of class II, the use of Class II elastic traction to establish a Class I molar relationship should be delayed until mandibular growth has been more fully expressed. It would also be acceptable to leave a greater discrepancy between the ligamentous position of the mandibular condyle (sometimes called Centric Relation) and the position of the condyle dictated by maximum intercuspation of the teeth to allow for greater mandibular growth during the retention phase of treatment.

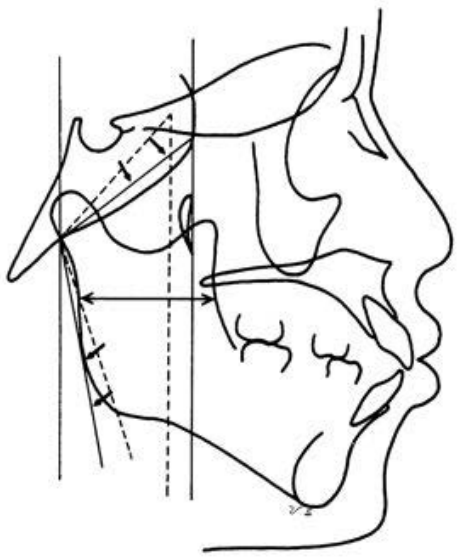
FIGURE 11-5



Black individuals, as with some Caucasians, tend to have an elongate, dolichocephalic headform, although there also occur wider faced individuals, just as among some Caucasian individuals and subgroups. The middle cranial fossa has an anteriorly inclined (open) alignment, even more so than in Caucasians. This factor causes the whole mandible to rotate markedly down and back (Fig. 11-6). The mandibular corpus tends to be horizontally long relative to the bony (not dental) maxillary arch. Unlike the typical "long-headed" Caucasian facial type, the upper part of the face in the black expands much less and is, therefore, not nearly so protrusive. In this respect, the face of the black corresponds to that of the Oriental. The forehead is more upright and bulbous than in most Caucasians, the frontal sinus proportionately less expanded; the nasal bridge lower; the nose flatter, wider, and less protrusive; and the cheekbones more prominent. Although the upper part of the nasal region in narrow-faced black individuals tends to be correspondingly narrow, approximately equivalent airway capacity is achieved by a wider dimension in the more inferior part of the nasal passageway in conjunction with a flaring of the nasal alae.

One special feature characterizes the black face; **the mandibular ramus is quite broad** proportionate to the middle cranial fossa. In a previous chapter, it was pointed out that the horizontal dimension of the ramus is a site that commonly participates in compensations for structural imbalances in other parts of the face and cranium. The forward inclination of the middle cranial fossa that characterizes many Caucasian groups, for example, is partially or completely counteracted by the development of a wider ramus, thereby offsetting or reducing the intrinsic tendency for mandibular retrusion and a Class II malocclusion. The mandible of the black also has this feature, but the amount is characteristically **much** greater. The very broad ramus places the mandibular corpus (which can also be long relative to the bony maxillary arch) in a resultant protrusive position. This, in turn, causes the maxillary incisors to tip labially, and a **bimaxillary** protrusion is thereby produced. This is an advanced feature that for the dolichocephalic black often precludes severe Class II malocclusions. If present at all, they are usually of the Class II "B" type. That is, mandibular B point lies well ahead of maxillary A point, in contrast to the more severe Class II "A" type, in which A point is the more protrusive relative to the occlusal plane (see Enlow et al., 1971a). Class I variations can also be problematic, especially when mandibular retrognathia is associated with a Class I molar occlusion. These "tooth/face" discrepancies require careful consideration of the treatment alternatives to achieve the optimal compromise between esthetics, function, and stability. Surgical augmentation of the bony chin is often a necessary procedure if a more prominent chin is needed for optimum aesthetic balance.

FIGURE 11-6



The anatomic basis for the Class III type of malocclusion in blacks has a different structural pattern compared to other population groups. The basicranium of the black Class III (or the related bimaxillary protrusive tendency) does not usually have a posterosuperior alignment of the middle cranial fossa in contrast to the brachycephalic Oriental and Caucasian Class III (and bimaxillary protrusion). Rather, the black Class III malocclusion tends to have an actual forward-downward

rotated middle cranial fossa, and the ramus is aligned backward, not forward. This reduces the extent of the mandibular protrusive features. The nasomaxillary complex is thereby placed more anteriorly, not posteriorly. The basicranium, thus, is not a principal factor among blacks that contributes directly to the protrusive placement of the mandible in Class III malocclusions, as it does in the other population groups. The basicranium, rather, is a counteracting feature. As pointed out above, the **wider ramus** of the black is a key anatomic compensatory feature that effectively offsets and largely precludes Class II malocclusion tendencies that otherwise relate to an anteriorly inclined middle cranial fossa. However, the same broad nature of the ramus **also** exists in most black Class III individuals as well as in the Class I. In Orientals and Caucasians, the Class III ramus is often "narrow" and reduces the extent of, and thereby partially compensates for, lower jaw protrusion. In the black Class III individual, conversely, not only is the ramus noncompensatory, its broad relative dimension adds to rather than subtracts from the extent of mandibular prognathism. Thus, the mandibular ramus of the black is an effective feature that minimizes one type of malocclusion, but that tends to aggravate another type. (See Enlow et al., 1982; Martone et al., 1992 for additional descriptive information.) Nonetheless, it is an advanced craniofacial factor that has eliminated a real threat in human evolution, which is the entrapment of the lower jaw caused by brain expansion and upright, bipedal body posture. (See Chapters 8 and 9.)

The combination of the tendency for bimaxillary protrusion and wide mandibular ramus often makes treatment planning for the black patient with anterior dental crossbite a challenge for the clinician. Two points are worthy of consideration in the treatment planning process. First, bimaxillary protrusion allows the clinician a greater range of dental compensation in treating anterior crossbite. Because the vertical facial relationships are often favorable, individuals with bimaxillary protrusion can often be dentally compensated by removal of upper second and lower first bicuspids. This often produces an aesthetically acceptable dental compromise, because the length of the mandibular corpus and position of the bony chin are acceptable. The second factor that needs to be considered concerns the individual with skeletal mandibular prognathia, secondary to an increase in ramus width.

In this group the clinician should consider surgical reduction in overall mandibular length by narrowing the ramus rather than the corpus. Procedures such as the internal vertical sliding osteotomy may be more effective in reducing mandibular prognathism in an individual with a wide ramus. However, the impact of malocclusion tendencies on treatment response is an area that desperately needs further study.

12

Control Processes in Facial Growth

There was a time, not long ago, when attempts to understand how facial growth is regulated were at a much simpler level. Most of the discussions seemed to settle on intrinsic "genetic" control versus everything else, such as biomechanical forces and hormones, and the ability of intramembranous versus the presumed preprogrammed (i.e., genetic) control of endochondral growth. A common approach was the naming of some special part or process that has the master power to control growth and thereby explain what can't be explained; a kind of theological disclaimer. From primitive civilization to today, if some particular phenomenon is not understood, a "deity" can be contrived to account for it, that is, a graven image, a fanciful invention of the mind. Thus, we have "condylar growth," and there has been abiding faith. Genetics itself has been such a deity, often misused to cover our insightful shortfall and to delude ourselves into believing that we understand what we fully do not. A common variation is the giving of descriptive titles, quite legitimate as far as they are intended to go, but which can be misused to try to explain the "how" when only the "what" is partially revealed (e.g., the functional matrix and Wolff's law of bone transformation). With regard to genetics, the old and compelling idea that there exist specific genes for virtually every structural detail throughout the craniofacial complex is simply not true. The completion of the Human Genome Project has raised more questions than answers and the role of genetics in growth and development is infinitely more complex than anyone expected. (See Chapter 20 for more details) Furthermore, how selective gene activations are in **response** (effect, rather than cause) to extracellular signals is a direction receiving increasing understanding and emphasis.

The explanations of the growth control process that prevailed until just a few years ago were straightforward, easy to understand, and so plausible that they were adopted and used for many years as the basis for a number of clinical concepts. Most seemed to center on control of **bone** growth, probably because the bones are what are seen and measured using cephalometric radiographs and because any basic clinical change in the face requires a reshaping and resizing of the underlying bones. The entire process of growth control seemed no particular puzzle and readily explainable. First, the growth of bone tissue by cartilage growth plates was presumed to be regulated entirely and directly by the intrinsic genetic programming within the cartilage cells. Intramembranous bone growth,

however, was believed to have a different source of control. This latter mode of the osteogenetic process was known to be particularly sensitive to biomechanical stress and strain and responds to tension and pressure by either bone deposition or resorption. Tension, as traditionally believed, specifically induces bone formation. Pressure, if it exceeds a relatively sensitive threshold limit, specifically triggers resorption. When tension is exerted on a bone, as at places of muscle attachment, the bone grows locally in response. Thus, sites of muscle insertion are usually marked by tuberosities, tubercles, and crests that form because of direct, localized fields of muscle traction. Because many muscles attach near the ends of a bone, rather than on its shaft, the epiphyses are much larger than the diaphysis, because this is where the muscles apply the most tension and where the bone thereby expands. As long as a muscle continues to grow, the bone is also stimulated to grow. This is because of the continuing biomechanical imbalance between them due to the expansion in muscle mass and resultant increasing force. The growing muscle would exceed the capacity of the bone to support it, and the osteoblasts are thereby triggered to form new bone in response. When muscle and overall body growth is complete, the bones attain biomechanical equilibrium with the muscles (and body weight, posture, and so forth). The forces of the muscles are then in balance with the physical properties of the bone. This turns off osteoblastic activity, and skeletal growth ceases. If any future circumstances cause departure from this sensitive state of bone—soft tissue equilibrium, such as major changes in body weight, loss of teeth, or the fracture of a bone, the process is revived until once again mechanical equilibrium subsequently becomes attained.

It is easy to understand why such up-front and reasonable explanations were attractive and almost universally adopted by earlier workers. For one thing, there is much basic truth in some of these concepts, as far as they go. They served to explain almost everything then known about bone and its growth. As more genes associated with craniofacial dysmorphias have been identified (See Chapter 20), the realization that a number of shortcomings exist led to a reevaluation of the whole process of growth control. The subject is a "new" frontier in facial biology. However, even past microscopic examination of bony change raised questions about this simple explanation for growth control.

First, there is no one-to-one correlation between places of muscle attachment and the pattern of distribution of resorptive and depository fields (Fig. 12-1); remodeling control is more biologically complex. Moreover, it is also known that there is no direct one-to-one correlation between tension deposition and pressure resorption (this old pressure versus tension concept is greatly oversimplified; see pages 130 and 135). In addition, it is clear from recent work by Tsolakis and Spyropoulos (see Chapter 19) that experimental forces applied to bone trigger remodeling responses at remote sites as well as at the point of force application. **Microscopic examination reveals that about half of all craniofacial bone surfaces to which muscles attach are actually resorptive**, not depository. Many muscles have widespread attachments, and within these surface areas, some growth fields are resorptive and others are depository. Yet these contrasting remodeling surfaces are subject to the same pull by the same muscle, supplied by the same blood vessels, and innervated by the same nerves. The temporalis muscle, for example, inserts

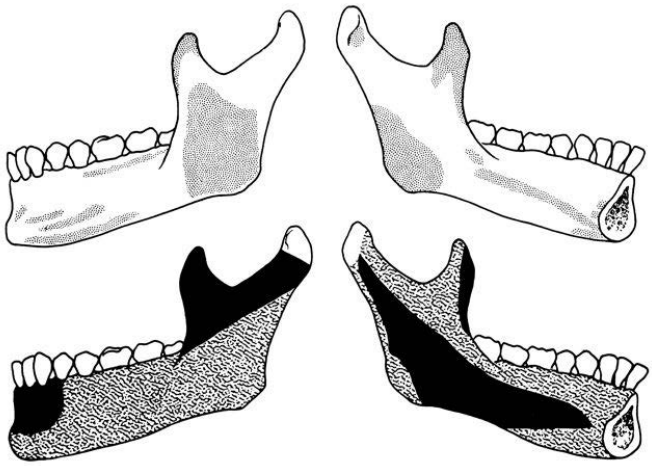


FIGURE 12-1.
The top figures show the distribution of muscle attachments on the buccal and lingual sides of the mandible. The bottom figures illustrate the pattern of surface resorptive (dark) and depository (light) growth and remodeling fields. Note that there is no one-to-one correlation between these respective patterns. As described in the text, this does not mean that muscle forces are not involved in growth control; it does show, however, that the old "muscle-tension—direct bone deposition" concept is invalid. (From Enlow, D. H.: Wolff's law and the factor of architectonic circumstance. Am. J. Orthod., 54:803, 1968, with permission.)

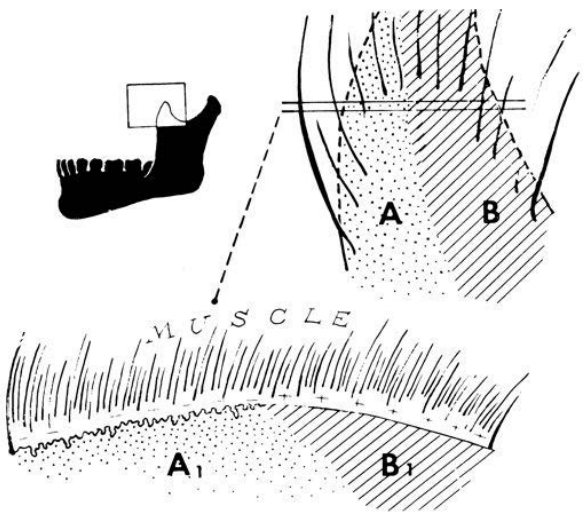


FIGURE 12-2.
The temporalis muscle attaches to surface A and B on the lingual side of the coronoid process. In microscopic sections, it is seen that the attachment on A_1 involves a resorptive bone surface; the same muscle is also inserted on surface B_1, which is depository. (From Enlow, D. H.: Wolff's law and the factor of architectonic circumstance. Am. J. Orthod., 54:803, 1968, with permission.)

onto the coronoid process of the mandible (Fig. 12-2). As shown in Chapter 4, parts of this mandibular region have external surfaces that are resorptive. The muscle exerts tension, but the bone to which it directly attaches undergoes resorption. Other surfaces of temporalis muscle attachment are characteristically depository.

Furthermore, some muscles pull in one direction, but the bone surfaces into which they insert grow in other directions. The pterygoid muscle, for example, attaches onto the posterior part of the ramus. The muscle pulls anteriorly, but this part of the bone remodels posteriorly.

Growth control involves a cascade of graded feedback chains from the systemic down to the local tissue, cellular, and molecular levels and back again. The problems at hand deal with the local control process in all the regional parts everywhere. How each local area responds to the local activating signals involved in the local anatomy with local functions, and how each local region grows in concert with all other regions—this is the complex biologic holy grail. Learning to better control all this is still the ultimate clinical objective.

SYNOPSIS OF CRANIOFACIAL GROWTH CONTROL THEORIES

Several alternative explanations that attempt to address the questions surrounding the ultimate basis of growth control, or some of its component aspects, have historically dominated the attention and thinking of leading biologic theoreticians. Although each such working theory is separate, a trend has always been to merge some of them selectively into a composite scheme in order to help account for the baffling array of poorly understood issues.

The Genetic Blueprint

Always at the forefront of any growth control discussion is the old and perplexing question of the real extent of "genetic" control. The role of genetic preprogramming has long been presumed to have a fundamental and perhaps overriding influence in establishing the basic facial pattern and the features upon which the internal and external "environment" then begins to play at some yet-to-be-understood levels. Contemporary researchers, however, have not been able to accept the idea that, simply stated, genes are the exclusive determinants for all growth parameters, including regional growth amounts, velocities, and minute details of regional configuration. Fully realized, of course, is the understanding that genes are indeed a basic participant in the operation of any given cell's organelles leading to the expression of that cell's particular function. For example, an osteoclast, a prechondroblast, or a contractile fibroblast each does its cellular function when activated, and it then ceases when signals deactivate it. Its own internal genes are not the actual "starter and stopper." In fact, the DNA content of all cells in the body is identical. It is the RNA being expressed by each cell type that determines the cell's intracellular and extracellular proteins and ultimately the functions of that cell line. At issue is the mechanism by which intercellular conditions **activate** intracellular processes, and just how the complex array of many

different cell types and tissue combinations can manage to interact as a composite whole. **Selective** and **regulated** activation of specific genes within a cell's full genetic complement from without, however, is presumed to be one answer. A key factor is the recognition that **epigenetic** regulation can determine, to a substantial extent, the behavioral growth activities of "genic" tissue types. This means that these developmental "genic" tissues do not actually govern their own functions; rather, their role in growth is controlled by epigenetic influences from **other** tissue groups and their functional, structural, and developmental input signals.

A major complicating factor in the search for the genetic plan for facial growth is the flawed concept that a single gene or group of genes controls growth. Although researchers have been able to identify specific gene mutations that result in dysmorphic syndromes, the effect of the mutation is variable. A single genotype gives rise to many phenotypes and a given phenotype can be associated with many genotypes. The lack of a one-to-one correspondence between genotype and phenotype is attributed to gene-to-gene and gene-to-environment interactions. The difficulties encountered in identifying the growth control processes affected in severely dysmorphic conditions increases logarithmically when researchers begin to search for genes controlling "normal" growth. (See Chapter 20)

Biomechanical Forces

A powerful line of reasoning has historically focused on the play of physical forces acting on a bone to regulate its development, morphologic configuration, histologic structure, and physical properties. Wolff's law of bone transformation, introduced in the late 1800s, quickly became a leading and most useful working concept, and is still quite valid **if** it is not overextended. Essentially, an application of the old and trusted idea that form interrelates with and is inseparable from function, this cornerstone principle states the biologic truism that a bone grows and develops in such a manner that the composite of physiologic forces exerted on it are accommodated by the bone's developmental process, thereby adapting structure to the complex of functions. This descriptive perspective, however, has often been overstated; it has been presumed that the actual biologic process of developmental control is explained, that is, **how** control of development is carried out, rather than simply a description of what is happening. One principal omission (and a major flaw) in many old attempts to apply Wolff's law has been a lack of distinction between physical forces acting on a bone (i.e., its hard part) and forces acting on the osteogenic connective tissues (periosteum, growth cartilages, sutures, etc.) that actually produce and remodel the bone.

Many experiments have been carried out in which muscles were severed, or the soft tissues otherwise altered, and in which artificial mechanical forces were experimentally exerted on a living bone. Because such procedures always produce some kind of response with resultant changes in the form of the bone, it has often been concluded that stress is, therefore, the principal factor controlling bone growth. Such experiments, however, do not "prove" such a role for the mechanical forces, since certain critical variables necessarily exist that cannot be controlled in the experimental design. These include vascular and neural interruption, temperature

changes, alterations in pH and oxygen tension, and so on, all of which are known to affect bone growth. The fundamental question must then be asked: Do extrinsic or unusual factors that can affect the course of a bone's development also necessarily represent the same intrinsic factors that actually carry out the direct, primary control of the basic histogenic processes of growth and differentiation? That key question is simply not addressed. Nonetheless, there can be no doubt whatsoever that mechanical forces, indeed, represent one (of many) of the "messengers" (see below) involved in the activation of osteogenic connective tissue. What **regulates** the complex **balance** of "genic" activities among all the multitude of cell and tissue participants is the key issue.

Sutures, Condyles, and Synchondroses

In the 1920s, a then new model for growth control began to emerge that flourished through the 40s and 50s, with some holdover yet even today. Many of the groundbreaking ideas within these pioneering explanations have since been set aside and replaced by more biologically tuned and complete understandings. They did, nonetheless, generate a number of testable hypotheses that could be answered using the scientific method and history was served.

It was presumed, quite reasonably at the time, that the growth, form, and dimensions of a bone are governed by intrinsic genetic programming residing within that bone's own bone-producing cells of the periosteum, sutures, and bone-related cartilages. While influences such as hormones and muscle actions could augment these gene-dominant growth determinants, bones such as the mandible or maxilla, and all of their morphologic features, were held to be largely self-generated products. The displacements of bones as they enlarge were also attributed to the expansive forces residing within their osteogenic sutures and cartilages and a "thrust" by the new bone tissues they produce. The idea expanded to include a concept of growth "centers" that were presumed to provide inclusive growth regulation for each of the whole bones they serve. Today, most front-line researchers discount the notion of such "master growth centers," replacing it with a concept of regional "sites" of growth, each of which is a localized area having its own regional circumstances and conditions and which operates under its own regional process of growth control. A feedback system allows reciprocal growth interactions and developmental adaptations with the other sites.

The Nasal Septum

It became understood (albeit slowly, historically) that "centers," such as the facial sutures, cannot actually **drive** the nasomaxillary complex into downward and forward displacement. This is because a suture is a traction-adapted (not a "pushing" and pressure-adapted) type of tissue. To try to resolve the dilemma that then arose, James Scott, a well-known Irish anatomist, reasoned that the cartilaginous nasal septum has features and occupies a strategic position that might answer the question of what "motor" causes the midface to displace anteriorly and inferiorly as it grows in size. Because cartilage is a more pressure-tolerant

tissue than the vascular-sensitive sutures, it presumably has the developmental capacity to expansively **push** the whole nasomaxillary complex downward and forward. With this thought, Scott's famous nasal septum theory was born. Latham (1970) proposed an interesting modification of Scott's theory when he suggested that the actual physical force for the maxillary displacement movement may be, at least in part, a pulling action of the septopremaxillary ligament resulting from septal enlargement, rather than a pushing action. Such an effect can be noted in a bilateral palatal cleft: the embryonic nasomedial process ("premaxilla") is displaced protrusively, but without maxillary-to-premaxillary sutural attachment, the maxillae are not drawn forward and are left behind.

The laboratory testing of the nasal septum theory has been a concerted target of many researchers for many years since, but the idea has encountered formidable laboratory obstacles because of difficulties in controlling the multiple developmental variables involved. This has led to differing interpretations of the experimental results. One problem is that most animal experiments of the kind here relevant involve conditions in which functions normally carried out by some given anatomic structure, when that anatomic part is altered or excised, can be compensated to some extent by other structures. Or, it cannot be presumed that, if some structural part is experimentally removed, what happens as a consequence, therefore, reflects the actual function of that part in situ. Then, too, importantly, it cannot be simply assumed that any given structure's function is the same when conditions have been experimentally altered as when they existed in an undisturbed (nonexperimental) state. Other concerns are outlined in Chapter 1.

Whether or not the nasal septum operates as the essential pacemaker for maxillary displacement, it is important for normal midfacial growth. (Hans et al. 1996). And the septum is a component of the "functional matrix," and it thereby contributes its own share of developmental participation in combination with all the other components necessarily also involved.

An important and fundamental point is that growth control is multifactorial. The old notion that a single, presiding agent, such as the nasal septum or condyle, has sole responsibility for pacesetting the growth process is not true. This is highlighted later as well as discussions in several previous chapters.

The Functional Matrix

Basic form/function principles proposed by van der Klaauw were greatly elaborated by Melvin Moss and evolved into a landmark concept having great impact among practicing clinicians and craniofacial theoreticians alike. Although some of the deeper issues involved were heatedly controversial for a time, one very valued outcome was a most intensive and productive debate on important questions and a great deal of new research.

Simply stated and omitting some details, the "functional matrix" concept deals primarily with the ultimate source of osteogenic regulation. Although many aspects were clouded historically by operational uncertainties, i.e. "**how**" it operates, the core of the idea is straightforward and not in itself controversial.

The role of genes in cellular organelle functioning (e.g., production of

specific tissue protein types, enzymes) in response to extracellular messengers that activate a given cell's physiologic role in the grand scheme is not an issue. Stimuli emanating from the **growth** and the **actions** of all the multiple sources within the growing head and body (the functional matrix), directly or indirectly, function to turn on or turn off cellular organelle activity in each and all of the "genic" tissues. This yields growing, changing, custom-fitted bones having regional dimensions and changing configurations that update constantly to accommodate the changing developmental conditions and biomechanical circumstances in each localized region of each separate bone and the aggregate of all in an interrelated system. Each bone becomes **continuously** and precisely adapted to these multiple developmental conditions because it is the composite of these conditions that regulate a bone's ongoing configuration, size, fitting, and the timing involved.

The functional matrix concept is not intended to explain **how** the actual morphogenic process works, but, rather, describes **what** happens to achieve the combination of actions, reactions, and feedback interplay that occurs. This is important. The nature of the signals involved and how they operate are separate but quite significant issues dealt with later.

A basic consideration, also, is that the term "functional matrix" can be misleading, because it connotes primarily the function of a soft tissue part (e.g., muscle contraction). **Growth enlargements** are also directly involved in giving the signals that activate osteogenic connective tissues, and this is an equally significant factor (see Fig. 1-7). Also, the functional matrix concept was developed primarily for bone growth; the biologic principles involved can be extended effectively to soft tissues as well.

Composite Explanations

Many experimental studies, together with observations of certain congenital craniofacial dysplasias ("nature's experiments") and much theoretical reasoning, led to combinations of various growth control theories attempting to account for the complexities of development. Some were grouped by van Limborgh, for example, into a model that distinguishes factors influencing chondrocranial versus desmocranial (intramembranous) craniofacial development. With the chondrocranium serving as an early but ongoing pacer, intrinsic genetic cell multiplication capability, general epigenetic influence (e.g., hormones), and general environmental factors (food and oxygen supply, etc.) were all proposed as agents within an interplay scheme for the endochondral part of basicranial developmental control. Desmocranial development, separately, was described as a morphogenic response to some balance among most of these factors, but with local epigenetic and local environmental factors (mechanical forces) playing a dominant regulatory role.

The elegant studies of Petrovic and more recently by Tsolakis and his colleagues have challenged the thinking of contemporary craniofacial biologists. Emerging from this experimental work have been elaborate cybernetic models that illustrate many of the complex developmental interrelationships among almost all of the multiple cellular and tissue elements involved in growth control. Professional

students going beyond the present chapter's introduction on "control" will need to utilize their insight as a springboard.

Control Messengers

Growth control is essentially a localized developmental process working with local function as it responds to multiple developmental interplay with other growing parts. It is complemented all the while by systemic support. Growth is carried out by specific, restricted, regional **fields**, each of which has differing growth activity in amounts, directions, velocities, and timing (described in earlier chapters). The diverse cell populations within each of these fields respond to activating intracellular or extracellular signals. "First messengers" are extracellular activators for which specific cell-surface receptors are selectively sensitive. They include biomechanical, bioelectric, hormonal, enzymatic, oxygen, carbon dioxide, etc., factors. A reception signal then fires a cascade of "second messengers" within a given cell that results in the function of that cell and its organelles, such as fiber production and proteoglycan production, calcification, acid or alkaline phosphatase secretion, and rate and duration of mitotic cell divisions. Adenyl cyclase and cyclic adenosine monophosphate (cAMP) are second messengers leading to cytoplasmic and nuclear DNA-RNA transfers.

In the **immediate** environment enclosing an osteoblast or osteoclast, a first-messenger hormone or enzyme, a bioelectric potential change, or a pressure/tension factor acting on the cell's outer sensory membrane receptors can activate a second messenger (membrane-bound adenyl cyclase), which in turn accelerates the transformation of adenosine triphosphate (ATP) to cAMP within the cytoplasm, which then activates the synthesis of other specific enzymes relating specifically to bone deposition or resorption. Ionic calcium is mobilized from mitochondrion storage, and inner and outer membrane permeability is altered that selectively controls the flux of other ions in the synthesis and discharge of the products secreted by the cell.

During bone formation, the osteoblast takes in amino acids, glucose, and sulfate for the synthesis of the glycoproteins and collagen in the formative organic part of the bone matrix. The cytoplasmic organelles within the osteoblast participate in the formation, storage, and secretion of tropocollagen, the mucopolysaccharide ground substance, and also ions that form the inorganic (hydroxyapatite) phase of the bone matrix. Alkaline glycerophosphatase is related to bone formation (in contrast to acid phosphatase, which relates to resorption) and is associated with the collagen fibril as it is released from the osteoblast. High levels of alkaline phosphatase are also involved in the formation of the hydroxyapatite. The citric acid cycle and glycolytic enzymes provide generalized energy sources for all these activities.

The osteoclast contains an abundance of mitochondria in addition to lysosomes and an extensive endoplasmic smooth membrane system. The osteoclast produces, stores, and secretes enzymes (such as collagenase) and acids that relate to the breakdown of both the organic and inorganic components of bone. The lysosomes are involved in acid phosphatase storage and transport. First messengers,

such as parathyroid hormone or bioelectric charges, stimulate receptor sites on the cell membrane. This activates adenyl cyclase, which in turn causes increases in cytoplasmic AMP. The latter then increases the permeability of the lysosomal membrane. By an exocytosis of the lysosomal contents, the resorption of both the organic and inorganic parts of the bone is carried out through the activity of the acid hydrolases, lactates, and citrates. The endoplasmic smooth membrane system is also involved in this process of enzyme transport and release.

Bioelectric Signals

The piezo factor has been one of the great bone growth-control hopes since the mid-1960s and has promised to clarify just how muscle and other biomechanical force actions can be translated into precisely regulated bone remodeling responses. The idea, in brief, is that distortions of the collagen crystals in bone, caused by minute (ultramicroscopic) deformations of the bone matrix due to mechanical strains, generate bioelectric charges in the immediate area of deformation (i.e, the piezo effect). These altered electrical potentials appear to relate, either directly or indirectly, to the triggering of osteoblastic and osteoclastic responses (see page 137).

To put this factor in perspective, one key point must be understood. There are two separate **target categories** for the mechanical actions of muscles, and also the effect of muscle and soft tissue growth enlargements, gravity, and all other such physical sources. One target is the cellular component of the osteogenic **connective tissues** that cover a bone. The outer surfaces of these cells are loaded with receptors that are sensitive to the **direct** effects of first messenger agents and forces. The second basic target category is the calcified part of the bone itself, the matrix, in contrast to the covering connective tissues just mentioned. Mechanical forces produced both by growth and by function acting on the calcified matrix cause minute distortions that generate positive and negative polarities. While response thresholds of regional force levels are still poorly understood, a minute **concavity** under active distortion is known to emanate a negative (-) bioelectric charge, and a **convexity** generates a positive (+) charge. (Figure 7-9 shows a schematic "bone" responding to a force, producing a convex side and a concave side, and the resulting bioelectric potentials created.) Negative charges then transmit to the osteogenic cells within the connective tissue on the concave side firing osteoblasts into depository activity. A positive charge on the convex side activates an osteoclastic response. The result is **coordinated regional** remodeling, inside and outside surfaces alike, that shapes the bone and enlarges its overall size. When mechanical equilibrium is achieved between the bone and the composite of growth and functional forces playing on it, the polarities are neutralized, and the remodeling activities are turned off.

This scheme appears to explain nicely the actual basis for remodeling **coordination** between the periosteal side and the contralateral endosteal surface in a given region of a bone; that is, one side can be convex, and the other concave, with the common forces acting within this region thus resulting in complementary deposition/resorption responses. Also, note that the pressure/tension and

deposition/resorption responses characterizing the osteogenic connective tissue membrane versus the bone matrix itself are opposites. (See pages 137 to 138 for further discussion.) While the piezo electric effect has been found to be a good model for long-bone remodeling, recent studies suggest that other factors may be involved in tooth movement and alveolar bone remodeling (Tuncay et al., 1990, 1994).

There is a key question, however, that contemporary researchers appear not yet to have asked, at least to date: an explanation of the nature of the **balance and interplay** between growth-affecting mechanical forces acting directly on a bone's **osteogenic membranes** (the first category) versus forces acting on the **bone matrix itself** (second category). Since the respective force-causing responses are actually reversed, a threshold sensitivity or some synergistic means for selectivity must be operative. There is a basic need for resolving this key question, yet the question itself has yet to be asked by researchers.

Other Factors of Growth Control

The **neurotropic factor** involves the network of nerves (all kinds, motor as well as sensory) as links for feedback interrelationships among all the soft tissues and bone. Researchers are increasingly interested in the supporting cells of the nervous system (e.g., Schwann, glial); the potential role of these cells in the growth process is mostly unexplored. The nerves are believed to provide pathways for stimuli that presumably can trigger certain bone and soft tissue remodeling responses. It is not believed, however, that this process is carried out by actual nervous impulses. Rather, it appears to function by transport of neurosecretory material along nerve tracts (analogous perhaps to the neurohumoral flow from the hypothalamus to the neurohypophysis along tracts in the infundibulum) or by an exoplasmic streaming within the neuron. In this way, feedback information is passed, for example, from the connective tissue stroma of a muscle to the osteogenic periosteum of the bone associated with that muscle. The "functional matrix" thereby operates to govern the bone's development. It is an interesting but yet incomplete hypothesis in need of more study.

A great many laboratory investigations are now being conducted in attempts to clarify relationships between, for example, AMP and biomechanical forces. Other independent studies have been underway, most not even dealing with craniofacial biology per se, on the role of important substances such as the cascade of prostaglandins, somatomedins, osteonectines, leukotrienes, possible neurotropic balancing agents, and intercellular communications involving "G" proteins. Still other work is ongoing seeking out possible chalone-like agents in bone (i.e., localized tissue-level, hormone-like substances believed to affect the magnitude of cell divisions). Day by day, new information and new links are being added. A real problem is that such frontier research is proceeding quite separate from our own craniofacial mainstream, and with very little input. However, to help place all these old and new factors in some kind of working perspective, one can use the following relative simple "test" to classify their respective roles:

1. Is a given factor held to be the sole, primary agent that is directly responsible for the master control of growth? Of course, such a single, uniquitous agent does not exist. Historically, bone investigators have searched for such a master control factor, perhaps a special "hormone" or special "inductor," that does it all. However, it is now realized that the control process must necessarily be multifactorial and control involves a multidirectional chain of regulatory links. Not all of the individual links participate in all types of growth changes. Rather, selected directional combinations exist for different specific control pathways that follow many objective routes and involve many different agents.

2. Does a given factor function as a "trigger" that induces or turns on other specific, selected agents that then launch the process of control response? Is it the first link in the lengthy chain? Is it the initial agent in the process of "induction"? Is it a "first messenger"? Biomechanical forces presumably represent such a trigger. It is still justifiably believed that pressures and tensions are indeed among the basic agents involved in eventual growth control (although not following the traditional but over-simplified historical explanations). Different kinds of bone, however, appear to have variable thresholds of response to physical forces (e.g., basal bone versus alveolar bone in the mandible and maxilla, which are relatively nonsensitive versus quite labile, respectively, to physical strain). It is also evident that biomechanical forces are not the sole agents participating in control. Even when involved, many other links are also required as second and third level messengers. Oxygen tension at the intercellular level may be another example, since this factor is known to be involved in "genic" cell differentiation.

3. Is a given factor, in effect, the **title** for some biologic process without accounting for the actual operational mechanism involved? This is an important category. **Such a title describes what happens, but not how it happens.** It does not explain the implementation and mechanics of the control process it is presumed to represent. It is just, in effect, a synonym for "control process" without explaining how it actually works. The reason an awareness of this category is so important is that we all often tend to use such titles as though they do indeed explain the mechanism involved. With continued use, we delude ourselves into believing that we actually understand the real basis for the control process itself. "Wolff's law," "functional matrix," "genetics," "induction," and even "development" itself are all such descriptive labels for biologic control systems. While each has a legitimate and valid place in describing the events that occur during growth, they do not, of course, explain how the system works (each was never intended for this ultimate purpose, even though all of us have often misused them as such). Be ever alert to this common and deceptive conceptual pitfall. (See also the "graven image" analogy used to explain what we do not fully understand, page 231.)

4. Does a given factor function essentially in a supportive role or as a catalyst? Many nutrients would be examples of this category.

5. Does a given factor accompany the control process, but not actually take part in a determinative way? It has been necessary for laboratory researchers studying the piezo effect, for example, to establish whether bioelectric potentials are actually first messengers or whether they simply "occur" in a by-product and non—remodeling-activating role.

6. Is a given factor an actual cause, rather than an inert effect, in the growth control process? The piezo factor also serves as an example of this category. Do bioelectric charges in bone directly trigger remodeling responses, or are the responses merely the incidental result? Is there an intermediary role for this factor? Whatever future research answers this question, the replacement ideas will be subject to the same tests.

One fundamental feature of the control process is now clear. No given tissue, such as bone, grows and differentiates in an isolated, independent manner by a wholly intrinsic regulatory process. Control is essentially a system of complex intercellular feedback pathways, informational interchanges, and reciprocal responses. Tissues, organs and parts of organs all necessarily develop as packages, all differentiating in close interplay. A given bone and all its muscles, nerves, blood vessels, connective tissues, and epithelia is an interdependent, developmental composite. Bones have specific mechanisms for increasing in length (e.g., an epiphyseal plate, synchondrosis, condyle), and they have another specific mechanism for increasing in width (subperiosteal intramembranous remodeling). Correspondingly, muscles also have a specific growth mechanism for increases in length and another, separately, for increases in width. Both of these conjoint muscle growth processes proceed in concert with respective linear and diametrical growth mechanisms for the bone. There are reciprocal feedback interrelationships between the muscle and bone, as well as the various other tissues, and they all enlarge together, not as "separate," independent, and self-contained units. For example, input to the sensory nerve endings in the periodontal membrane can trigger alveolar remodeling responses to occlusal signals from the teeth. These same signals can be passed on through an arc to motor nerves supplying the muscles of mastication. In conjunction with muscle adaptations to the individualized nature of the occlusion, then, the bones of the face undergo widespread, regional remodeling in association with the muscles and soft tissue matrix. The old concept that a "growth cartilage," simply, serves as the primary self-contained regulator for the overall development of the facial and mandibular musculoskeletal composite is now regarded as an unacceptable explanation. This is because many input factors are now known to be involved. However, we still have a long way to go in understanding the whole of the growth control system. History will probably judge this as one of the great problems of our time.

THE ARCHITECTONICS OF GROWTH CONTROL: A SUMMARY

Previous chapters outline a diverse collection of factors that need to be taken into account whenever dealing with facial "growth control". These are presented below as a series of separate points and issues with emphasis on the "architectonics" of growth control—simply, the dynamics of the developmental interrelationships among all of the growing parts, soft and hard tissues alike, and how this enters into the Big Picture.

1. Growth is a **differential** process of progressive maturation. Different parts have different schedules in which changing growth velocities occur at different times, by different regional amounts, and in different regional directions. For example, vertical facial development has a quite different morphogenic timetable than the transverse because bilateral basicranial width is precocious, compared with facial airway enlargement and tooth eruption. The airway relates to whole body and lung development, whereas the bicondylar and bizygomatic dimensions relate to the earlier transverse maturation of the cerebral temporal and frontal lobes with their basicranial fossae. This creates many developmental complexities. For example, the architectonics of mandibular growth control must thereby provide for a differential timing of whole-ramus (not just condylar) development to adapt to differences in vertical versus anteroposterior maturation of the pharyngeal compartment as well as, at the same time, differential anteroposterior versus vertical growth enlargements, remodeling, and displacement of the nasomaxillary complex, including its airway and erupting dental components. This indeed requires fancy developmental footwork on the part of the growing ramus if precision of dimensional changes, fitting of separate parts, and proper timing are to be collectively achieved. The developmental complexities here cannot be overstated.

2. **Development is a process working toward an ongoing state of aggregate, composite structural and functional equilibrium.** Growth, of course, involves constant changes in size, shape, and relationships among all of the separate parts and the regional components of each part. Any change in any given part must be proportionately matched by appropriate growth changes and adjustments in **many** other parts, nearby as well as distant, to sustain and progressively achieve functional and structural balance of the whole. In short, growth anywhere in any region, local area, or part, is **not isolated.** This seems quite obvious; yet the complex of **interrelations** that exist is often bypassed in our thinking and in the literature. "Balance" is a developmental aggregate involving close interplay throughout. For example, the shape and size of one's external nose and facial airway are not determined solely by blueprint (or any other kind of control) just within these parts themselves, since many other parts elsewhere establish rigid developmental conditions. Interorbital width and nasomaxillary boundaries presented by the basicranium, for example,

require reciprocal compliance of any genetic, epigenetic, or soft tissue growth determinants acting on the separate "genic" tissues that produce the growing nasal region.

Certain regional **imbalances** exist in everyone that are natural and architectonically inescapable (Chapters 1, 9, and 10). Individual or population differences in headform configuration, for example, establish corresponding differences in the basicranial template for many facial dimensions, growth field boundaries, and component alignments. These variations set up many different kinds of facial configurations within which some "imbalances" have necessarily been introduced because of architectural complexity. However, growth and development work toward a state of aggregate architectonic equilibrium, so that emerging from the process of development are certain other regional "imbalances" that are offsetting and compensating. Groups of localized imbalances thus can balance other groups of imbalances as a normal part of the growth control process, the composite of which is more or less in functional equilibrium. Nonetheless, virtually limitless morphologic variations have been introduced, most of which we regard as more or less normal. For some others, the latitude for developmental adjustment is exceeded, and a malocclusion or some other structural dysplasia results, even though "balanced" in itself. In a sense, the process of growth and development is nature's own clinician, and it usually works quite well, even though various malocclusion **tendencies** exist in all of us because of established, normal headform type variations.

In addition to different phylogenic lines (e.g., headform variations) leading to developmental facial variations, ontogenic factors are involved as well. Mouth breathing is an example. A broken lip seal requires different muscle actions for mandibular posturing, and an open-jaw swallow similarly requires different muscular combinations. These factors produce different signals to the osteogenic, chondrogenic, myogenic, and fibrogenic components that revise the course of development, thus leading to adjustive morphologic variations to create a developmental balance among parts that had become morphogenetically imbalanced. (See Fig. 1-7.)

3. The goodness of fit among contiguous anatomic parts and groups of parts is remarkable. Consider the extreme precision of shape and size fitting between, for example, the temporalis muscle and its bony coronoid process; or the fit between a tooth's root and its alveolar socket; or one bone in sutural articulation with another. Developmental interplay establishes this, while all the time sustaining continuous growth and function. This requires a kind of "servo" system to create the architectonic exchange of signals that turn on or off, up or down, the cytogenic responses that drive the remodeling progression. Consider a cranial nerve and its basicranial foramen. The size and shape of the foramen must **precisely** match its nerve and perineural connective and vascular tissues. As the latter grow and develop, so must the foramen with no mismatch whatever. Then, as the nerve **moves** with the developing brain, the foramen must likewise remodel in **precise** lockstep. This is one example of the

many delicate, very significant kinds of developmental relationships that all too often have gone unsung in our appreciation of the Big Picture.

4. If we consider only bony components, we see that each bone and all of its regional parts participate directly and actively. Most of us readily acknowledge this. Why, then, do we persist in highlighting just a mandibular condyle, for example, while largely ignoring the rest of the ramus, which is just as significant and developmentally noteworthy in the Big Picture. It is the **whole** ramus that is a respondent to the massive growth-influencing muscles of mastication, and it is the development of the **whole** ramus and all of its parts, in teamwork, that places the mandibular arch, and establishes mandibular fitting with the maxilla on one side and the basicranium on the other.

   It is important to understand that there is no common, overriding, **centralized** control force that regulates the developmental and anatomic details for each individual region throughout any given whole bone. Rather, the different regional areas have different **local** developmental, functional, and structural conditions and circumstances that generate appropriate regional signals activating all local osteogenic connective tissues (periosteum, endosteum, cartilages, sutures, periodontal membranes) for precision operation of their responses as a part of the overall growth control system. This provides the **regional** balances and goodness-of-fit among parts, as highlighted herein.

5. The nature and capacity for interrelated growth adjustments among separate parts vary among different tissue types. Bone, for example, has a wide latitude for responsive remodeling adaptations through intrinsic manipulation of the osteogenic connective tissues to produce size and shape conformation in precision fitting. A tooth, in contrast, has a much lower potential for adjustive remodeling and, hence, teeth undergo posteruptive growth movements and location changes mostly by a displacement process. An **occlusal curve** is another such intrinsic developmental adjustment of the dentition and its alveolar bone to developmental conditions imposed on the dental arches from without. **Anterior crowding** is another and, while a "malocclusion," is actually an adjustive compensation to provide fitting of the dentition within a prescribed growth field as established by other, multiple architectonic and morphogenic relationships. One imbalance has balanced others to achieve a kind of aggregate structural equilibrium. This concept of dental crowding as a biologically necessary compensatory mechanism to allow adjustment to changes in regional and global growth fields has important implications for the orthodontic clinician. Currently, lifelong retention of dental alignment is advocated by many clinicians. This recommendation is often thought to have no associated risk. However, if changes in dental alignment occur to maintain a healthy occlusion in a changing environment, preventing those changes may lead to pathologic degeneration of the teeth or supporting structures. This theoretical problem may indeed become a real concern as lifetime retention gains increasing popularity in the orthodontically treated population.

The capacity for remodeling of cartilage, either interstitially or by its chondrogenic connective tissue, is also much more constrained than for bone. **Condylar** cartilage can, however, undergo alterations in growth direction and magnitude by differential turning on and turning off of prechondroblastic proliferation around its periphery, thus producing adaptive growth vectors in response to changing architectonic conditions. This is in contrast, significantly, to the "primary" cartilages of the basicranium and long bones.

6. The two principal catagories of growth movement, **displacement** and **remodeling**, is one of the most fundamental concepts of growth. Yet to this day, this all-important facet, more often than not, is totally disregarded when trying to account for how a given appliance or other clinical procedure is presumed to work. The significance of this point cannot be overemphasized. Both types of movement are usually clumped together simply as "growth" without distinguishing between them. The reason distinction is very important is that each represents a separate and distinct target in the intrinsic control process utilized for different clinical procedures. Headgear, for example, manipulates directions and magnitudes of the displacement type of movement (whole-bone and soft tissue movements), with bony and soft tissue remodeling then providing adjustments to altered whole-part placements. Periodontal connective tissue responses to fixed appliances activate alveolar remodeling adjustments in response to tooth displacements. Functional appliances presumably activate altered combinations of displacement and remodeling up and down the line. Some orthognathic procedures involve the surgical moving of bones or their parts followed by osteogenic transplants, thus paralleling that which natural growth did not fully achieve—displacement (surgical movements) and remodeling (sizing and shaping by transplants). This also highlights the reason bony articulations, including sutures, movable joints, synchondroses, and tooth junctions, need a full share of attention in understanding the growth process and its control. They are the places from which displacement movements emanate as a given bone simultaneously undergoes its remodeling by the enclosing osteogenic and chondrogenic connective tissues. Displacement moves whole bones away from each other at their joint contacts, thus complementing their enlargements. Remodeling, at the same time, produces the enlargement, constructs the configuration and its progressive changes, provides precision fitting with contiguous soft tissues and with other bones, and creates the ongoing "compensations" or adjustments leading to a composite working equilibrium among all the separate parts.

   Another example of the displacement/remodeling combination is the placement and development of the incisor part of the maxilla. By far, most of the actual downward-forward "growth" of the premaxillary region is by whole-maxilla movement caused by enlargements of all the other craniofacial parts above and behind, not just intrinsic remodeling growth within that localized premaxillary region itself. Localized remodeling produces the size and shape of that regional area, but most of its considerable extent of growth movement over the years is a product of secondary displacement.

7. Variation in headform, as previously mentioned, is an important factor. The reason is that the dolicho, brachy, or dinaric types establish quite different basicranial templates for facial development. Whatever growth control resides within the mandibular and ethmomaxillary components themselves must necessarily yield and conform to a higher level of predetermination in a number of respects. For example, facial shape and proportions are projections of the anterior cranial fossa and can incorporate any basicranial asymmetries that exist. The apical base of the maxillary dentition, in turn, is established by the basicranial-determined configuration and size of the palatal perimeter. For another example, the middle cranial fossa establishes the anteroposterior placement of the maxilla relative to the mandible. Because it is known that (1) variations in headform set up corresponding variations in facial type and pattern, (2) headform variations predispose specific, corresponding malocclusion tendencies, (3) headform and resultant facial variations involving different anatomic combinations respond differently to different treatment procedures, and (4) that different rebound tendencies exist in different pattern combinations, **much** closer attention should be given to headform consideration than at present. A Class I dinaric has a different anatomic combination than a Class I brachycephalic, and both are basically different than a Class I dolichocephalic. The intrinsic control of facial growth, therefore, is strongly influenced by factors external to the face itself, and this must be taken into account in our Big Architectonic Picture. Predispositions for variations in mandibular retrusive versus protrusive tendencies are **built into** the phylogenetic heritage of different population groups.

   Beginning students are inclined to perceived the Class I category as an anatomically separate and distinct type having good overall balance for each regional part with only minor departures from an ideal. Not so. In the assembly of all the multitude of craniofacial parts, **all** of us have a multitude of regional phylogenic and headform-established imbalances throughout the craniofacial complex, as briefly summarized above. Some of these are mandibular retrusive and others protrusive in their regional effects. If the aggregate is offsetting (i.e., a balancing of imbalances), a Class I results, but with a "tendency" one way or the other because the underlying features that are retrusive-causing or protrusive-causing still exist, but are partially nullified by the compensations. The Class I is in the middle span between the extremes of this spectrum involving multiple composites of different architectonic combinations but with two sides grading away in opposite directions.

8. With regard to our phylogenic facial heritage, the factor of bipedal body posture interrelating with our enormous human brain and marked basicranial flexure have led to an inferoposterior rotational placement of the nasomaxillary complex. The midface has come to lie **below** the anterior cranial fossa, rather than protruding largely forward from it. This has caught the mandible in a closing vise between the midface above and the pharyngeal airway, gullet, and cervical column behind. Overjet, overbite, anterior crossbite, and an unprecedented developmental problem situation for the human temporomandibular joint

(TMJ) are some consequences. The adaptive remodeling capacity of the ramus, with its condyle, is especially noteworthy in adjusting to these severe conditions. The wonder is not that so much TMJ distress as a common clinical result exists, but rather, that there is not much more.

Furthermore, the human basicranial suture growth system, inherited from smaller brained ancestors, cannot provide for the full range needed to accommodate our grossly enlarged brain. Thus, an unusual basicranial remodeling pattern, not found in any other known mammal including anthropoids, has been added to the human growth package. Still another evolutionary factor is that of orbital convergence toward the midline in conjunction with enlarged temporal lobe expansion. Together with facial rotation, nasal configuration and placement are affected, since the interorbital (nasal) compartment has become significantly reduced as well as moved into a vertical position. Human nasomaxillary development has adjusted, in many regional ways, to these major phylogenic and ontogenic conditions.

9. With respect to growth rotations, this timely subject has justifiably become of great interest. The classification of rotation types is simple: some are "displacement" types, and some are "remodeling" rotations. (The literature is needlessly confusing on this subject.) Remodeling rotations represent one of the developmental adjustments ("compensations") emphasized above. For example, basicranial growth can predispose a naso-maxillary "displacement rotation" that would "unbalance" the alignment of the palate. By differential "remodeling rotation" of the anterior versus posterior parts of the palate, however, the palate as a whole can be progressively leveled into a functional position as the basicranium grows. Similar mandibular rotations exist in relation to variable basicranial proportions as well as midfacial size, configuration, rotations, and alignment variations. The architectonic factor of developmental rotations thus enters prominently into our Big Picture of selective, regional growth control factors.

10. Considering architectonic feedback communication and give-and-take regional adaptations in the interrelationships involved in growth control, three examples stand out in which component parts each play a particularly significant role. One is the mandibular ramus; the second is the periodontal connective tissue membrane; and the third is the insignificant-appearing little lacrimal bone.

In a typical gross anatomy course for freshmen, the ramus is usually dismissed as just a handle (the word itself means "branch") for masticatory muscle attachments, which is surely important enough. But how much more dynamic for the student if its **other** very interesting and essential (developmental) functions were also dramatized. Consider first, that the ramus bridges the pharyngeal space to functionally position the lower arch in changing occlusion with the upper. The enlargement of this changing pharyngeal space is progressively established by its ceiling, which is formed by the enlarging middle cranial fossae and their growing temporal lobes, a morphogenic process continuing long into childhood. The anteroposterior

breadth of the ramus must **match** this basicranial developmental progression, with multiple and diverse basicranial, maxillary, and mandibular rotations also taken into account, by equivalent growth amounts and with corresponding timing—an elaborate architectonic interrelationship of many separate parts. Otherwise, either excessive anterior crossbite or mandibular retrusion would ensue. Furthermore, the vertical height of the ramus must match the changing vertical increases of the nasal and dental parts of the ethmomaxillary complex, taking into account the vertical lengthening of the middle cranial fossae as well and, importantly, marked differences in vertical versus horizontal timing within the naso-maxillary complex and basicranium. Too much or too little and too early or too late sets up an anterior deep or open bite, and the latitude for mismatch is very slight. All of this requires a precision and coordination of signals given to the osteogenic connective tissues that enclose the ramus, which respond by enlarging, relocating, rotating, shaping, and constantly adjusting the whole ramus (and its condyle). This is a most remarkable complex and histogenically interplay among the separate parts involved.

The periodontal membrane (PDM) is a "genic" connective tissue that shapes, sizes, and constantly remodels and relocates the alveolar bone to match its moving, resident teeth and which also carries the teeth in vertical and horizontal drifting movements in addition to their initial eruption. The PDM is a dynamic, complex connective tissue membrane with many and diverse component elements (often demeaned as merely a connecting "ligament") responsive to the multiple signals that activate its multiple cell types to carry out these architectonic growth functions. In addition, the PDM contributes to tooth formation and provides vascular pathways and proprioceptive and other sensory and vasomotor innervations. Consider the high degree of precision required of the intrinsic control process in coordinating tooth movement and alveolar remodeling. The direction, amount, and timing must be absolutely precise, with virtually no divergence. It is the wondrous PDM that carries all this out. Clinically, this elaborately coordinated growth process is manipulated by substituting clinical control to override the intrinsic control. But the histogenic process itself is the same. The remarkable PDM and its symphony of movements are a workhorse for the orthodontist.

The tiny, thin flake of a lacrimal bone receives little, if any, attention in a standard gross anatomy course and given no special highlight at all. Yet phylogenetically this seemingly insignificant little bone has survived as a discrete part even as many of the other much more robust cranial bones have lost their individual identity through multiple mergers and fusions. The reason for its evolutionary retention is that it has a special and essential architectonic role in facial development. It is an island of bone surrounded by osteogenic (remodeling-capable) sutural connective tissue responsive to growth control signals emanating from all around it. It is strategically situated with the ethmoid, the nasal part of the maxilla, the frontal bone, the orbitosphenoid, the alisphenoid, and the orbital part of the maxilla, all of which are growing in different directions, at different times, by different amounts, and with different functional relationships. A "slide" of bones along their sutural interfaces is

involved, and this is achieved through an elaborate process of relinkages by the sutural connective tissue fibers. It was pointed out that precision of fitting is an essential part of growth control; by virtue of the lacrimal bone's **adjustive suture system**, all of these separate parts can undergo their differential displacements and their own enlargements, directional relocations, and remodeling, yet continuously fit with one another as they all develop and function. Without this adaptive system, the face simply could not "grow" at all. With it, the human (and mammalian) face has successfully survived in the long course of evolution. (Refer to pages 98 and 109.)

There should be marble and bronze monuments glorifying the ramus, the PDM, and the lacrimal bone, all prominently displayed in the atrium of every dental school; and students should be expected to doff their caps in solemn reverence each morning when passing!

11. Clinical intervention into the growth process and its control is by either one of two approaches, both of which are analogous to the intrinsic growth process itself. The first approach is by surgical substitutions for the natural displacement and remodeling processes that were incomplete or derailed. The second approach is by overriding intrinsic control signals with clinically induced (e.g., orthodontic) signals that overwhelm the intrinsic regulation of osteogenic, chondrogenic, myogenic, neurogenic, and fibrogenic systems. Then, the same actual biologic operations of these systems proceed, but now under control-revised directions. However, in all cases, if the same conditions that created the original intrinsic signals still persist after treatment, then architectonic *rebound growth* naturally adjusts back to the former, balanced pattern. Interestingly, these two forms of clinical intervention are conceptually different. Orthodontic intervention attempts to augment natural compensatory changes to achieve an improved aesthetic and functional balance among facial components. For example, for patients with mandibular retrognathia, an orthodontist will often accentuate the degree of mandibular dental protrusion (a natural anatomic compensation for mandibular retrognathia) by using Class II elastic traction of a bionator-type removable appliance. In contrast, surgical interventions require that dental compensations be removed (usually by presurgical orthodontic tooth movement) prior to surgical correction of the skeletal imbalance.[**]

Removal of compensation allows the surgical team to maximize skeletal balance and to improve postsurgical occlusal stability. Although conceptually different, these two clinical intervention strategies must necessarily "work with" the same set of biologic rules. This fact is often overlooked in debates concerning the clinical efficacy of surgery and/or orthodontic correction of malocclusion. Clearly, the degree and amount of change that occurs with

* It is interesting to note that by removing dental compensations prior to surgery, the clinician is creating a facial morphology that is capable of postsurgical compensation (sometimes referred to incorrectly as surgical relapse). Such postsurgical compensation is probably responsible for the occlusal stability seen with orthognathic procedures compared to gnathic procedures.

facial development between 9 and 14 years of age far exceeds the limits of surgical manipulation of facial bones. However, the potential for posttreatment physiologic rebound is also far greater.

12. There is another fundamental clinical consideration that, more often than not, is conceptually bypassed. When the muscles of facial expression contract (*function*), the mechanical effect is an **upward** and **backward** retrusive force exerted on the maxilla. Yet everyone knows that the maxilla "grows forward and downward." Does **this** not contradict the *functional matrix* principle? Similarly, when the masticatory muscles function, the net mechanical effect on the mandible is also upward and backward, not downward and forward. Does **this**, thereby, not also violate belief that "function" of the functional matrix is the basic driver for growth control? However, two basic factors are omitted in presenting these comments. First, the important distinction between the **displacement** type of growth movement versus **remodeling** growth movement was not made. Second, importantly, the **growth enlargements** of the respective muscles were not included, only their contractile functions.

    With respect to displacement movements, the connective tissue stroma of each muscle is directly or indirectly continuous with fibers attaching to the bones, and **enlargements in diameter** of mandibular muscles such as the masseter and temporalis have an anteriorly displacing effect on the whole mandible. Their **enlargements in length** have an inferiorly displacing and mandibular-carrying effect. As the facial expression muscles, oropharyngeal soft tissues, and facial integument all undergo **outward** growth expansion, there is an outward and downward carrying movement of all the nasomaxillary and mandibular parts.

    At the same time, **functioning** of all the muscles (contractions) and all other soft tissue components is proceeding. The "genic" connective tissues (condylar cartilages and the sutural, periosteal, endosteal, and periodontal membranes) respond to the signals produced by the functioning, growing systems everywhere around the mandible and maxilla. This activates remodeling to adapt regional sizes, progressive regional configurations, and ongoing adjustments involved throughout all regional parts of each whole bone and its contiguous soft tissues. The maxilla and mandible "separate" (displacement) at their sutures and at the TMJ, and this is simultaneously accompanied by overall enlargement of each bone into the "spaces" created. The coronoid process, the gonial region, the lingual tuberosity, and so forth, are all formed and continuously enlarged to **precisely fit** with the muscles and other soft tissues they serve. They fit because of feedback control among them involving the turning on and off of the regional osteogenic connective tissues. Tooth roots fit their sockets for the same reason. Bony edges at the interdigitating sutures merge and mesh precisely. Nerves and vessels to and from a bone exactly fit their foramina in size, shape, and constantly changing locations. The condylar cartilage continues to fit its displacement-moving and remodeling fossa. And so on. To do this, (1) the growing configurations and size changes of the muscles and other soft tissues, (2) the displacements of

the bones, (3) the functions of all the multitude of soft tissues, and (4) the complex bony remodeling processes everywhere, are all developmentally inseparable. All are required as an architectonic package. They are isolated here descriptively so that we can better perceive their respective roles. In real life they simply cannot be biologically separated. One of the reasons many animal experiments intended to "prove or disprove," for example, the "functional matrix" or the "condyle as a master growth center," have always been much less than fully successful, is that these four factors were not **each** recognized and taken into account. Indeed, such experiments play against a stacked deck because of the actual inseparability of these factors as independent and controllable experimental variables.

Finally, refer to Figure 1-7 for a generalized overview of the dynamic, exceedingly precise architectonic interplay among regional parts as they all develop and as all continue to function as they do so. A most remarkable developmental system indeed.

PUTTING IT ALL TOGETHER

The Facial Growth Process follows a precisely organized Plan. The principal elements of this Plan are pure Basic Biology. It has nothing to do with growth averages or normative values. It is not based on midline radiographic points or non-biologic cephalometric planes such as Sella Nasion. This whole biologic Plan is logical and understandable, and every aspect of it is involved in orthodontic intervention into this Biology during facial growth. A major point is that this biology is the very same biology that underlies growth into malocclusions, and it is the very same in all ways in the biology that is the basis for orthodontic treatment. Clinical treatment, in virtually every way, is the intervention into the CONTROL SYSTEM that regulates this biology.

"Growth" requires that all the multitude of parts in a growing face become separated from each other so that growth has spaces for new growth additions. How everything becomes moved apart has historically been a very contentious issue. The effective resolution, as we now view it, is not problematic. All the anatomic parts are moved away from each other by their own growth additions which, at the same time, fill in these additions into the spaces they simultaneously create. How that works is that EVERYTHING is emeshed in connective tissue (CT) which, as the title connotes, connects everything directly or indirectly to everything else. Collagenous fibers are the operative structures because they are strongly resistant to stretching and thus can be used for "pulling". As all the parts, together with enclosing organ CT grading down to intercellular "pulling tissue units", enlarge by their own growth increases, the enclosing collagenous fibers are acted on by traction forces that play among them. These CT tension forces PULL everything away from everything else, with the growing parts filling in the spaces simultaneously as the spaces are formed. This is what is happening as the involved players collectively produce Growth. The movements of everything apart is "Displacement", and the growth enlargement process of the cells and tissue components is "Remodeling". There are different kinds of "remodeling", and all

utilize the same common resorptive and depository system, but the remodeling involved in Growth is genuine REmodeling, and not simply "modeling" as some proponents of biomechanical theory advocate. This is because everything is replaced and entirely remodeled during the growth process.

In summary, the Remodeling process (1) SIZES and (2) SHAPES all of the given anatomic parts. The Displacement Process, as it moves everything away from everything else, is thus creating the spaces for the growth enlargement by Remodeling. All of the parts are progressively moved into their successive functional POSITIONS, and remain fixed there, all by the Displacement process implemented by the Remodeling Process. The functional Positions place everything in a PRECISELY FITTED MOSAIC by FINE TUNING the individual configurations of each component by the Remodeling Process. This remodeling movement arranges each of them at the same time by the Displacement Process into constant "GOODNESS OF FIT" relationships. Wow! This is the basic Plan for the growth process.

The important principle to understand is that Displacement and Remodeling, together, constitute the GROWTH TEAM that is the functional working tool that does everything in growth. It is the basic operational engine that drives "growth". This is how it works without treatment. It is the very same biology utilized by clinical intervention into the growth Control System. In the present book, this GROWTH TEAM PLAY is seen countless times when explaining how virtually every detail of growth WORKS. Importantly, the TEAM is the operational tool used by all orthodontists to clinically modify the course of craniofacial development. You will not find this account of how the biology of growth actually WORKS, at least at present, in any orthodontic (or any other clinical) textbook. Yet, "working with growth" has long a basic tenet of orthodontics.

13

Prenatal Facial Growth and Development*

A 1-month-old embryo has no real face. But the key primordia have already begun to gather, and these slight early swellings, depressions, and thickenings are rapidly to undergo a series of mergers, rearrangements, and enlargements that will transform them, as if by sleight of hand, from a cluster of separate masses into a **face**.

The head of a 4-week-old human embryo is mostly just a brain covered by a thin sheet of ectoderm and mesoderm. Where the mouth will later be is marked by a tiny depression, the **stomodeum** (Fig. 13-1). The eyes have already begun to form by a thickening of the surface ectoderm (the future lens), which meets an outpouching from the brain (the future retina). The eyes are still located at the sides of the head, however, as in a fish. As the brain continues to grow and expand, the eyes become rotated toward each other by the rapidly enlarging brain and toward the midline of what is soon to become a face. Does this not greatly reduce the intervening span between the right and left eyes? Yes, but in a relative sense. **Everything** is increasing in size, including the interorbital dimension. The eyes are actually moving farther apart; but because other parts of the head are enlarging even more, the proportionate size of the interorbital area is becoming decreased. When illustrating the process of facial growth, it has always been traditional to show all of the stages as about equal in size. Keep in mind, however, that there is actually considerable overall enlargement as the process progresses. These changes are continuous and proceed very swiftly.

The mammalian pharynx is the homologue of the ancestral region that develops into the branchial chamber and gill system of fishes. The human pharyngeal pouches and clefts, however, did not "evolve from gills." More correctly, the embryonic primordia that developed into the fish's branchial system were phylogenetically converted to develop into **other** structures instead of gills. This is where many of the parts of the face come in.

As the whole developing head markedly expands, the membrane that covers the stomodeum does not keep pace with it. This thin sheet (the buccopharyngeal or oral membrane) quickly breaks through, and the **pharynx** becomes opened to

* This brief chapter presents a digest of the basics of facial embryology, not an extended account for advanced-level study. The objective is an introductory overview or an outline intended for a refresher review.

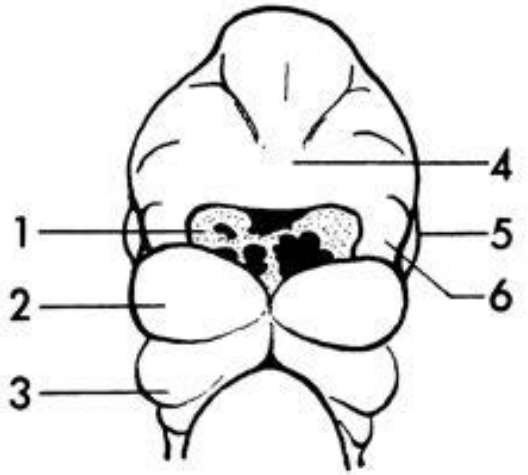


FIGURE 13-1.
Human face of about 4 weeks. 1, Stomodeal plate (buccopharyngeal membrane). 2, Mandibular arch (swelling or process). 3, Hyoid arch. 4, Frontal eminence (or prominence). 5, Optic vesicle. 6, Region where the maxillary process (or "swelling") of the first arch is just beginning to form. (Modified from Patten, B. M.: *Human Embryology*, 3rd Ed. New York, McGraw-Hill, 1968, with permission.)

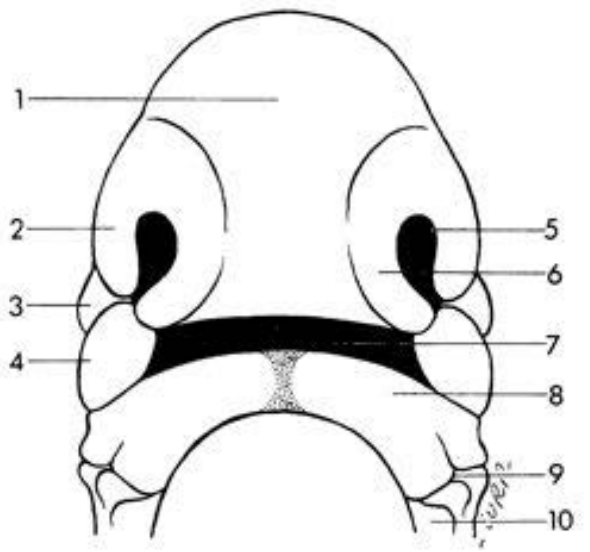


FIGURE 13-2.
Face at about 5 weeks. 1, Frontal prominence. 2, Lateral nasal swelling. 3, Eye. 4, Maxilliary swelling. 5, Nasal pit. 6, Medial nasal swelling. 7, Stomodeum. 8, Mandibular swelling. 9, Hyomandibular cleft. 10, Hyoid arch.

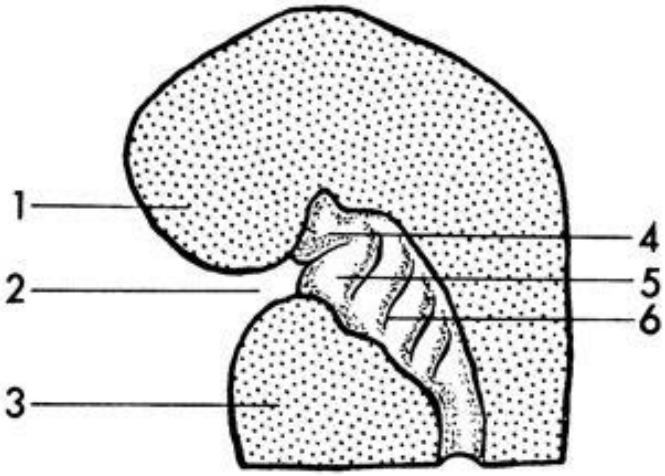


FIGURE 13-3.
Internal view of pharyngeal region. 1, Forebrain. 2, Stomodeum, 3, Cardiac prominence. 4, Maxillary process. 5, Mandibular process. 6, Pouch between second and third arches. (Modified from Langman, J.: *Medical Embryology*. Baltimore, Williams & Wilkins, 1969, with permission.)

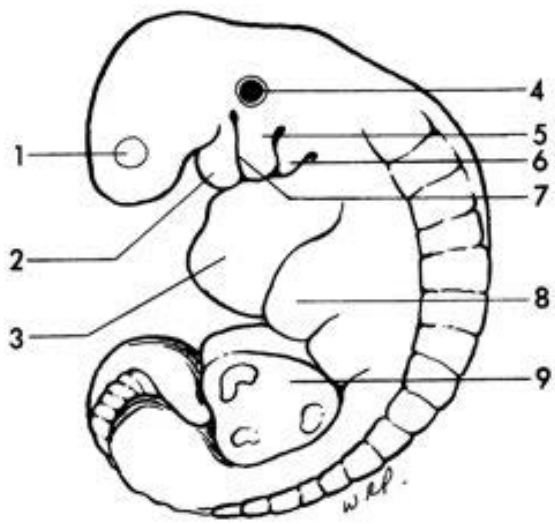


FIGURE 13-4.
Human embryo at about 4 weeks. 1, Optic vesicle. 2, Mandibular arch (process or swelling). 3, Cardiac prominence. 4, Auditory (otic) vesicle. 5, Hyoid arch. 6, Third arch. 7, Hyomandibular cleft. 8, Hepatic prominence. 9, Primitive umbilical cord. (Modified from Patten, B. M.: *Human Embryology*. 3rd Ed. New York, McGraw-Hill, 1968, with permission.)

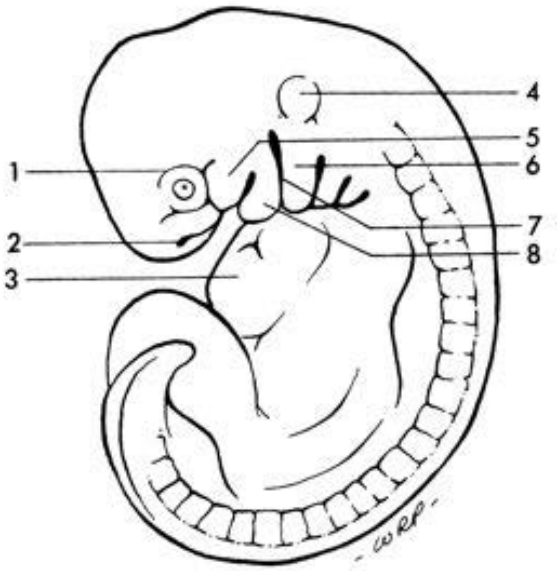


FIGURE 13-5.
Human embryo at about 5 weeks. 1, Eye. 2, Nasal pit. 3, Cardiac prominence. 4, Auditory vesicle. 5, Maxillary process. 6, Hyoid arch. 7, Hyomandibular cleft. 8, Mandibular arch. (Modified from Patten, B. M.: *Human Embryology,* 3rd Ed. New York, McGraw-Hill, 1968, with permission.)

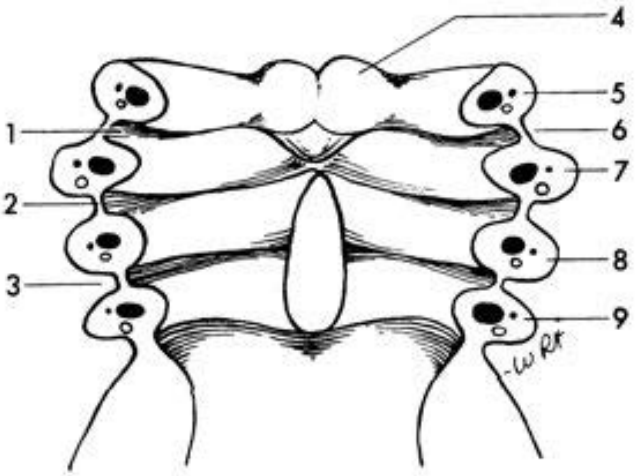


FIGURE 13-6.
Internal view of pharyngeal floor and cut arches. 1, First pharyngeal pouch between first and second arches (to become middle ear chamber). 2, Branchial membrane. 3, Pharyngeal cleft. 4, Region that will develop into the anterior two thirds (body) of tongue. 5, First (mandibular) arch containing its specific cartilage, cranial nerve, and aortic arch. The pharyngeal arch is also filled with branchiomeric mesenchyme. 6, First pharyngeal cleft (hyomandibular) to become external ear canal. 7, Second (hyoid) pharyngeal arch. 8, Third pharyngeal arch with its own cartilage, aortic arch, cranial nerve, and branchiomeric mesenchyme. 9, Fourth pharyngeal arch. (Modified from Moore, K. L.: *Before We Are Born: Basic Embryology and Birth Defects*. Philadelphia, W. B. Saunders, 1974, with permission.)

the outside (Fig. 13-2). Everything in front will become the face, and **this** is what is now about to develop. To appreciate how much facial growth is going to occur, realize that the location of the buccopharyngeal membrane in the 1-month-old embryo is at the level of the tonsils in the adult. An enormous amount of facial expansion thus will proceed in front of the stomodeum. On the internal side of this early cornerstone opening is the endodermally lined pharyngeal region. The pharynx is that part of the foregut characterized by the **pharyngeal** (visceral, branchial) **arches** (Fig. 13-3). Within the pharynx, a **pharyngeal pouch** lies between the arches, and on the outside a **pharyngeal cleft** occurs between the arches (Fig. 13-4). The ectoderm-endoderm contact between each cleft and pouch is the **branchial membrane** (Fig. 13-6).

All these various pharyngeal parts are major participants in the subsequent formation of many component structures in the head and neck.†

Each right and left pharyngeal arch has a specific cranial nerve, a specific artery (aortic arch), and programmed mesenchyme that develops into the particular muscles and specific embryonic cartilages that identify with that pharyngeal arch (Figs. 13-5 and 13-6). Specific facial bones then develop within specific pharyngeal arches. This is a basic and important concept because, if one can understand the simple embryonic relationships involved, understanding the exceedingly complex adult anatomy is so much easier. The **muscles** that develop in relation to each arch associate directly with the **bones** forming in that arch and are innervated by the resident **cranial nerve** of the same arch as well as supplied by the corresponding artery. Embryonic pharyngeal pouches and clefts also give rise to developing parts that extend to adult derivatives. All of this has a logical, systematic, readily recognizable developmental rationale in the embryo. Remembering these specific prenatal relationships, the far less fathomable plan for the seemingly garbled adult morphology makes sense.

In the human embryo, there are five bilateral pairs of pharyngeal arches. The first is the right and left **mandibular** arch. A bud develops from each first arch to form the paired **maxillary processes**. Both the mandibular and maxillary primordia are thus of first arch origin. The second pharyngeal arch is the **hyoid** arch (Fig. 13-5). The remaining arches are identified by respective numbers only.

The cartilage of the first pharyngeal arch is **Meckel's** cartilage, right and left (Figs. 13-1, 13-6, and 13-7). It occupies a location that becomes the core of the mandibular corpus, which forms around it. The bony mandible itself develops, independently, directly from the embryonic connective tissue that surrounds Meckel's cartilage. Most of this cartilage actually disappears, but parts of it give rise to the anlagen for two ear ossicles (the malleus and incus), and the perichondrium of Meckel's cartilage forms the beginning of the sphenomandibular ligament.

The cartilage of the hyoid (second) arch is **Reichert's** cartilage. It forms the third of the three ear ossicles on each side, the stapes. The remainder gives rise to the styloid process of the cranium, the stylohyoid ligament, the lesser horn of the hyoid bone, and a portion of the hyoid body (Fig. 13-7).

Muscles form from the mesenchyme of the arches. This mesenchyme is

† Migrating cranial neural crest cells contribute extensively to the early primordia of many tissues developing in the face and pharyngeal region (see Johnston et al., 1973).

termed **branchiomeric** (Gr. *branchia*, gills; Gr. *meros*, segment), in contrast to mesenchyme of somite origin elsewhere in the body. From the branchiomeric mesenchyme of the first arch, the muscles of mastication, the anterior belly of the digastric, the tensor palatini, the mylohyoid, and the tensor tympani muscles all develop. From the branchiomeric mesenchyme of the second arch develop the muscles of facial expression, the stylohyoid, the stapedius, the posterior belly of the digastric, and the auricular muscles.

The specific cranial nerves that supply the first arch are the mandibular and maxillary branches of the trigeminal nerve (V). The specific cranial nerve for the second arch is the facial nerve (VII). Thus, the muscles of the first arch (muscles of mastication, and so forth) are innervated by the mandibular division of V, regardless of the anatomic position in which each muscle finally becomes located later in development. The muscles of facial expression formed by the branchiomeric mesenchyme of the second pharyngeal arch correspondingly are all innervated by the facial nerve.

Note the gathering of many structures related to the formative ear region in and around the first and second pharyngeal arches (Figs. 13-8 and 13-9). The **auditory placode** differentiates early as a surface thickening of the ectoderm just above and behind the first pharyngeal cleft. This placode rapidly invaginates to form the auditory (otic) vesicle (Figs. 13-4 and 13-5), which then differentiates into the structures of the inner ear (semicircular canals, cochlea). The first pharyngeal **cleft** (between the first and second arches) forms the external auditory meatus and outer ear canal, and the branchial membrane between the cleft and pouch undergoes remodeling to participate in the formation of the tympanic membrane (Fig. 13-10). The first pharyngeal **pouch** becomes expanded into the middle ear chamber leading into the pharynx. The ear ossicles, developing from the cartilages of the first and second arches, conveniently abut this area and soon become enveloped within the expanding first pharyngeal pouch (middle ear chamber). They function as the bridge between the tympanic membrane and the inner ear. The auricle of each external ear develops from the surface swellings around the first pharyngeal cleft, and the bumps already present on these embryonic primordia form the characteristic hillocks of the adult ear lobe (Fig. 13-9).

The cartilage of the **third** pharyngeal arch is the precursor for the greater horn of the hyoid bone and part of the body (Fig. 13-7). The single muscle that develops from the third arch branchiomeric mesenchyme is the stylopharyngeus. The specific cranial nerve entering the third arch is the **glossopharyngeal**. It, therefore, supplies the muscle that develops from this arch. The cartilages in the remainder of the arches form into the thyroid, cricoid, and arytenoid components of the larynx. From fourth arch branchiomeric mesenchyme, the cricothyroid and pharyngeal constrictor muscles are formed. The specific nerve of the fourth pharyngeal arch is the superior laryngeal branch of the **vagus**. The intrinsic laryngeal muscles develop from the sixth arch and are innervated by that arch's resident nerve, which is the recurrent laryngeal branch of the vagus.

In each second pharyngeal pouch, the lining endoderm and underlying mesenchyme proliferate to form the paired **palatine tonsils**. From the lining of the third pouch develops **parathyroid III** (so called because of its third arch

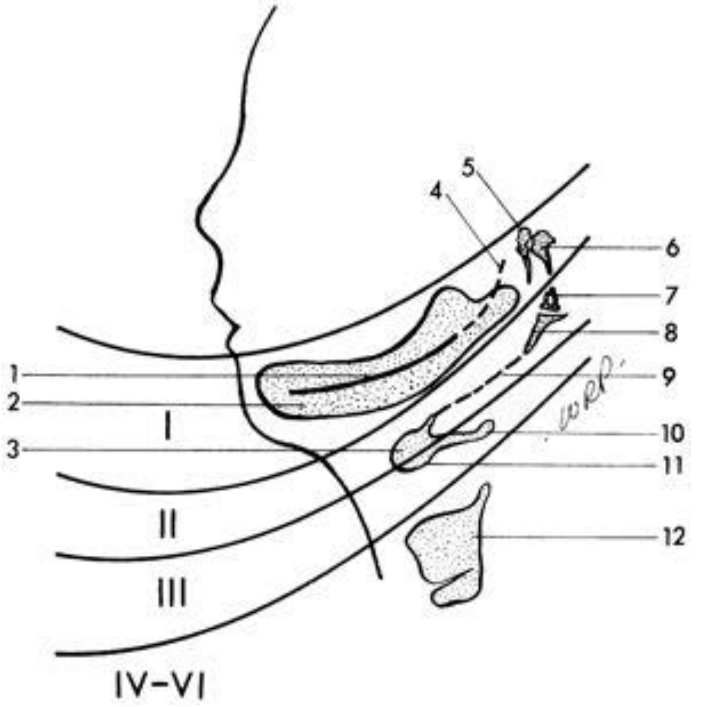


FIGURE 13-7.
Pharyngeal arch derivatives (I to VI). 1, Meckel's cartilage. 2, Intramembranous bone developing around Meckel's cartilage. 3, Superior part of body and lesser horn of hyoid. 4, Sphenomandibular ligament. 5, Malleus. 6, Incus. 7, Stapes. 8, Styloid process. 9, Stylohyoid ligament. 10, Greater horn of hyoid bone. 11, Inferior part of hyoid body. 12, Laryngeal cartilages.

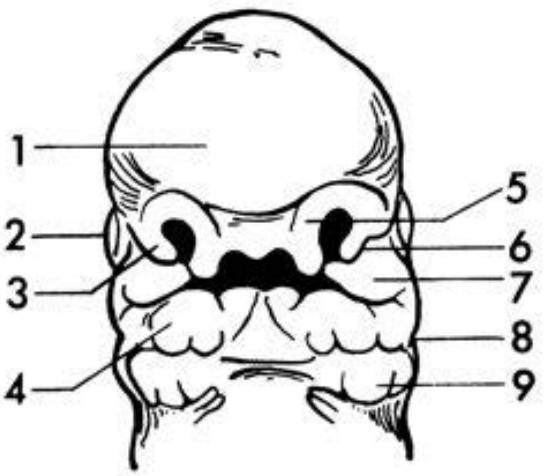


FIGURE 13-8.
Facial region at about 5½ weeks. 1, Forebrain. 2, Optic vesicle. 3, Lateral nasal swelling. 4, Mandibular process. 5, Medial nasal swelling. 6, Nasolacrimal groove. 7, Maxillary process. 8, Hyomandibular cleft. 9, Hyoid arch. (Modified from Patten. B. M.: *Human Embryology,* 3rd Ed. New York, McGraw-Hill. 1968, with permission.)

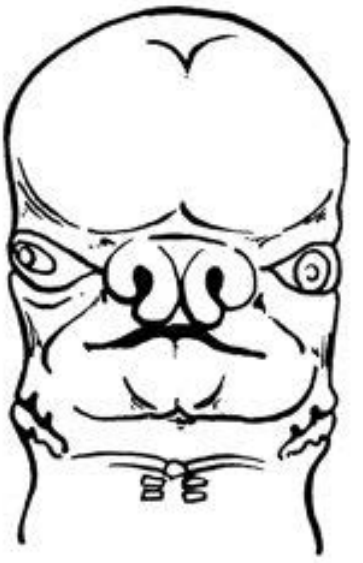


FIGURE 13-9.
Face at about 7 weeks.

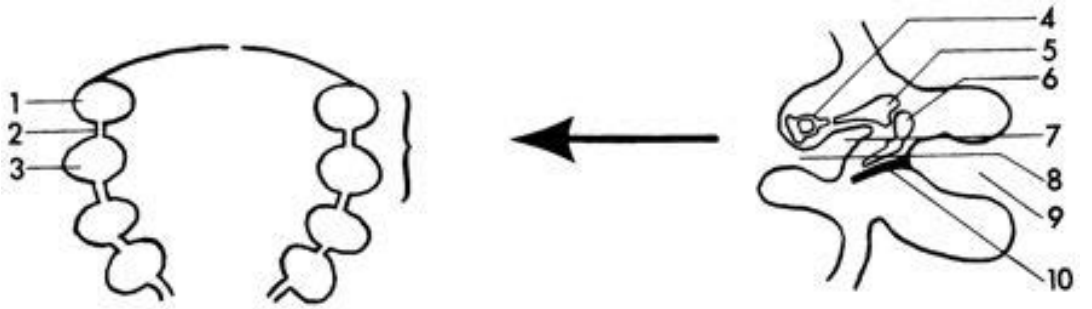


FIGURE 13-10.
Developing ear region. 1, Mandibular arch. 2, Branchial membrane between the cleft on the outside and pouch on the inside of the pharynx. 3, Hyoid arch. 4, Stapes. 5, Incus. 6, Malleus. 7, Middle ear chamber to expand as tympanic cavity surrounding auditory ossicles. 8, Auditory (eustachian) tube. 9, External ear canal. 10, Aniage for the tympanic membrane.

origin). This will form the "inferior" parathyroid because it later descends to a level below parathyroid IV. The thymus also develops from the lining of the third pharyngeal arch. Parathyroid IV (the "superior" parathyroid) develops from the fourth pouch.

In the floor of the pharynx, the first (mandibular) arches rapidly give rise to growing **lingual swellings** (Fig. 13-11). A smaller midline swelling, the **tuberculum impar**, is also present, and these three structures develop into the mucosal covering for the anterior two thirds, or body, of the tongue. Since the mandibular nerve supplies first arch tissue, it therefore provides the sensory (tactile) innervation for

the mucosa of the body of the tongue. The chorda tympani, which is a branch of VII that diverges from the second to the first arch by crossing through the branchial (tympanic) membrane to join the mandibular nerve (lingual branch), provides gustatory innervation for the tongue's mucosa.

At the root of the midventral parts of the second, third, and fourth pharyngeal arches, another prominent swelling occurs, the **copula**. This general region develops into the posterior third (root) of the tongue. The cranial nerves supplying the third and fourth arches are the glossopharyngeal and vagus, and these are thus the sensory nerves that innervate the mucosa of the root of the tongue. The core of the tongue is occupied by its "intrinsic" muscles. These originate from a more caudal region (probably from occipital somatic mesoderm) and grow into the expanding mucosal covering for the tongue being formed by the floor of the pharynx (described above). The motor innervation to these muscles is thus provided by the paired hypoglossal (XII) nerves. They are carried along with the intrinsic muscles as they migrate anteriorly into the formative body of the tongue.

Anatomically, the body of the tongue is separated from the root by a V-shaped sulcus (the **sulcus terminalis**). This marks the approximate line between the derivatives of first arch origin and those from the arches behind the first arch. At the midline in this developing groove, between the tuberculum impar and the copula, the thyroid primordium develops as an epithelial diverticulum into the pharyngeal floor (Fig. 13-12). It then separates from the mucosal lining and migrates caudally. The point of invagination, however, remains as a permanent pit termed the **foramen caecum** (Fig. 13-11). It is located at the apex of the V and is a landmark that identifies the adult position of the embryonic boundary between the first and second arches. As for most glandular tissues, the thyroid is thus of epithelial origin; and because the primordium develops from the pharyngeal lining, it is of endodermal derivation.

By the time an embryo is about 5 weeks old, the first pharyngeal arch has formed recognizable **maxillary** and **mandibular swellings**. Just above the

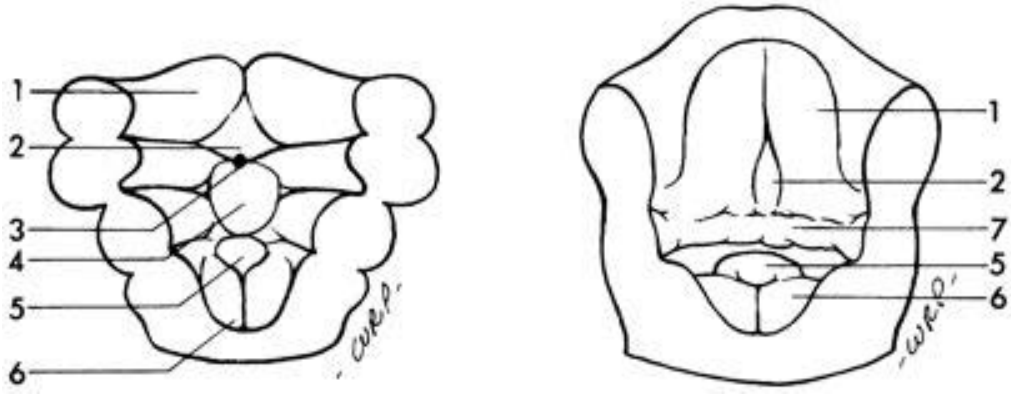


FIGURE 13-11.
Developing tongue at 6 and 8 weeks. 1, Lateral lingual swelling. 2, Tuberculum impar. 3, Foramen caecum. 4, Copula. 5, Epiglottis. 6, Arytenoid swellings. 7, Root of tongue.

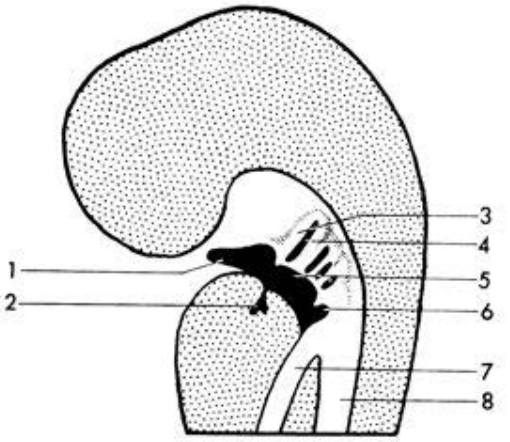


FIGURE 13-12.
Body of tongue (lateral lingual swellings and tuberculum impar). 2, Thyroid diverticulum. 3, Mandibular arch. 4, Pouch between first and second arches. 5, Root of tongue (copula). 6, Arytenoid swellings. 7, Trachea. 8, Esophagus.

stomodeum, the paired, laterally located **nasal placodes** have already differentiated by thickenings of the surface ectoderm. Horseshoelike ridges (**nasal swellings**) have developed around them to form deepening **nasal pits**. The floor of each pit is termed the oronasal membrane, but it is a transient structure that soon breaks through, thus opening the nasal pits directly into the oral cavity. At the same time, the semicircular nasal swellings continue to enlarge. Each swelling is composed of a lateral and medial limb. The expanding **medial** limbs merge at the midline to form the primordium that will differentiate into the middle part of the nose, the philtrum (cupid's bow) of the lip, the "incisor" part of the maxilla (premaxilla), and the small primary palate. (See Figs. 13-1 and 13-2.)

The rapidly growing **lateral** limbs of each nasal swelling form the alae of the nose (Figs. 13-8 and 13-9. While these changes occur, the maxillary swellings are also enlarging, and they subsequently merge with the medial limbs of the nasal swellings. The furrow between them (not a complete cleft in normal development) disappears, and a closed, U-shaped arch is thereby formed. The medial limbs develop into the middle span of both the maxillary arch and the upper lip as mentioned above. The cuspid, premolar, molar, and lateral lip parts of the upper arch develop from the paired maxillary processes. (See Brin et al., 1990, for an evaluation of prenatal nasal and cuspid relationships.) **These** are all some of the lines of merger that can be involved in cleft lip and jaw. Sometimes, developmental variations are encountered in which the blastema of a tooth is caught on the "wrong" side of a cleft; this always causes excitement because that is not the way it is supposed to be.

An oblique groove is present between the maxillary swelling and the lateral limb of the nasal swelling (Fig. 13-8). This is the **nasolacrimal groove**, which will soon close, but the line of merger establishes a developmental pathway for the later formation of the nasolacrimal duct. If this merger fails, a permanent facial cleft or fissure results.

The superficial tissues in lateral areas of the maxillary process fuse with the mandibular process to form the cheek. **Epithelial pearls** often occur along such lines of mucosal and cutaneous fusion. These are small islands of epithelial cells that were "programmed" to form, but were caught up in the fusion process. **Fordyce's spots**, which are remnants of cutaneous sebaceous glands, can similarly be found in the adult buccal mucosa, for the same reason, along lines of fusion.

The growing right and left mandibular swellings join at the midline to form the lower jaw and lip. A cartilaginous interface forms at this junction.‡

The **frontal prominence** forms the forehead and a vertical zone of developing tissue between the merging right and left medial nasal swellings. Here, the midline **nasal septum** is formed, which was historically believed by some to function as a pacemaker in later fetal development when its core becomes cartilaginous (see Chapter 5).

To date, these multiple, regional facial changes are all occurring at about the same time and have proceeded **rapidly** from about the fourth to the sixth week of embryonic development. The paired **palatal shelves** are now forming from each side of the maxillary arch (Figs. 13-13 and 13-14). The oral cavity is still relatively small, however, and the sizable tongue remains interposed between the right and left shelves. The early shelves necessarily enlarge downward in an obliquely vertical manner because of this. However, the inferior expansion of the whole lower part of the face carries the tongue downward. The oral cavity increases greatly in size. The paired nasal chambers are still continuous with the oral cavity. (Right and left nasal cavities are present because the nasal septum, developing inferiorly from the frontal prominence, has sustained the original paired nature of the nasal primordia.) The oral and nasal cavities at this stage are separated from each other only in the anterior-most region by the tiny, unpaired **primary palate** (median palatine process). The latter was formed by the fusion of the nasomedial ("premaxillary") processes. The whole lower part of the developing face, including the tongue and the floor of the oral cavity, now becomes displaced inferiorly to a greater extent than the enlarging palatal shelves are descending, so that the newly formed shelves of the maxilla are free to expand **medially** as well. They come together and soon fuse along the midline (the palatal raphe). The shelves "swing upward" in order to contact each other, but some of this apparent upward rotation is produced by differential growth. The shelves are growing and, especially, are becoming displaced inferiorly. By differential growth, the shelves expand downward as well as toward each other, but the **entire** nasal chamber on each side also expands laterally and inferiorly. Some of this apparent "upward swing" is relative and is a consequence of actual inferomedial growth. This is a process in which the different parts in the two nasal chambers and the oral cavity

‡ Although a limited amount of endochondral ossification will later occur here, the two halves of the mandible fuse completely after birth, unlike the permanently separate lower jaw halves of nonprimate mammals. Except in the "secondary cartilage" of the mandibular condyle (and to a much lesser extent in the mandibular symphysis and a small cartilage on the coronoid process), the greater part of the mandibular ossification is by the intermembranous mechanism. Meckel's cartilage does not participate in the endochondral ossification process (except for random spots here and there) and disappears except for its contribution to the ear ossicles and ligaments. The maxilla is entirely of intramembranous origin.

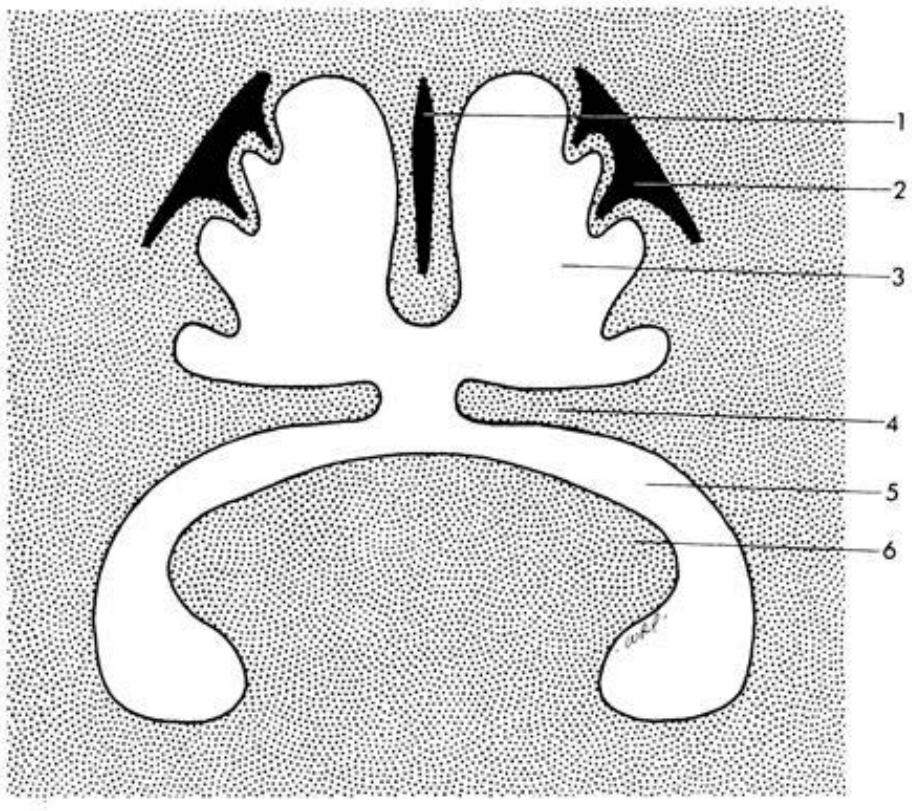


FIGURE 13-13.
Frontal section through the oronasal region in a 7½-week embryo. 1, Cartilage of the nasal septum. 2, Cartilage of the nasal conchae. 3, Nasal chamber. 4, Palatal shelf. 5, Oral cavity. 6, Tongue. (Adapted from Langman, J.: *Medical Embryology*. Baltimore, Williams & Wilkins, 1969, with permission.)

all grow at differential rates and to different extents as the whole midfacial region rapidly increases in size. While the schematic illustrations in Figures 13-13 and 13-14 are shown here as the same in size, in reality, a considerable enlargement is occurring.

The merger of the right and left palatal shelves forms the **secondary palate**. Bone tissue soon appears within it. This part of the palate is a direct extension of the maxilla from which it develops. The original primary palate, formed from the nasomedial (premaxillary) processes, is retained as a small median, unpaired, triangular segment of the palatal complex in the anterior region just ahead of the incisive foramen, a land-mark that identifies the midline boundary between the primary and secondary parts of the palate (Fig. 13-15). The separate palatine bones and their posterior contribution to the palatal complex do not develop until somewhat later. In the meantime, the nasal septum has merged with the superior surface of the palate. The two nasal chambers are now completely compartmented, and both have been closed off from the oral cavity along the length of the palate (Figs. 13-14 and 13-16).

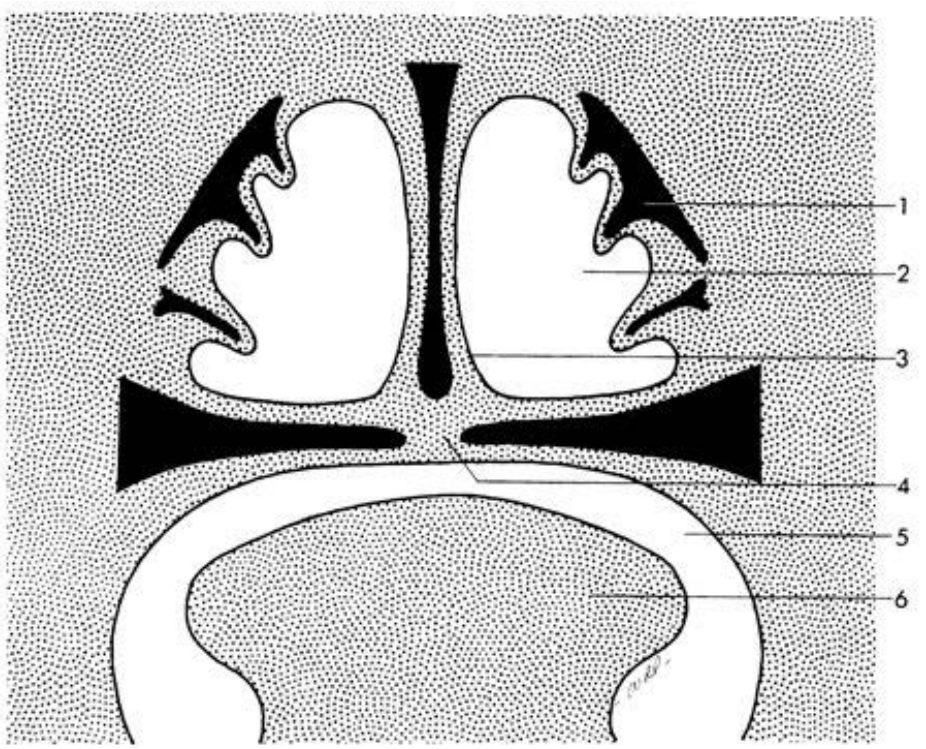


FIGURE 13-14.
Frontal section through the oronasal region of a 10-week embryo. 1, Nasal conchae. 2, Nasal chamber. 3, Nasal septum. 4, Palatal shelves, fused at midline and fused with nasal septum. The intramembranous bone of the palatal shelves (from the maxilla) is beginning to form. 5, Oral cavity. 6, Tongue. (Adapted from Langman, J.: *Medical Embryology.* Baltimore, Williams & Wilkins, 1969, with permission.)

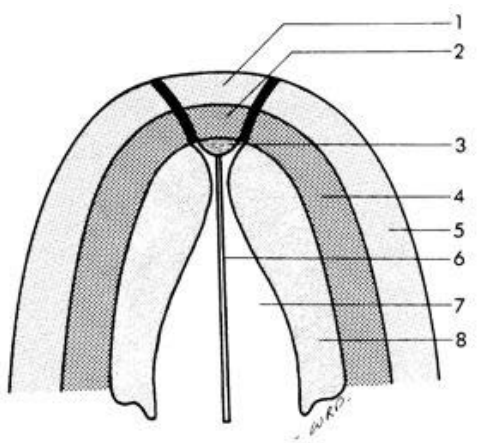


FIGURE 13-15.
Oral view of the palatal shelves in a 7½-week embryo. 1, Philtrum of upper lip. 2, "Premaxillary" segment from medial nasal process. 3, Primary palate. 4, Upper arch (part derived from maxillary swelling. 5, Cheek. 6, Nasal septum. 7, Open oral and nasal cavities. 8, Palatal shelves. In this stage, the philtrum and premaxillary segment have already merged with the maxillary swellings.

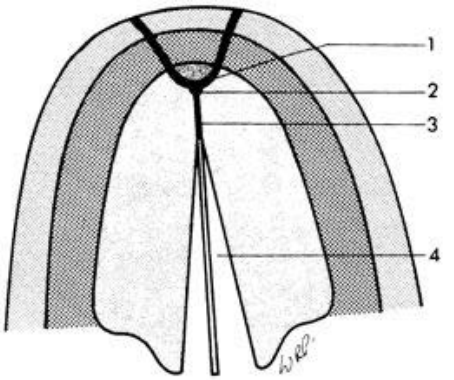


FIGURE 13-16.
Oral view of palate showing beginning of fusion. 1, Merger of midline primary palate with bilateral secondary palatal shelves. 2, Incisive foramen. 3, Palatal raphe (midline fusion). 4, Open nasal and oral chambers.

REMODELING IN THE FETAL FACE

The process of "remodeling," involving differential, regional "fields" of periosteal and endosteal resorptive and depository surfaces, first begins in the fetus at about 10 weeks in two principal locations: on lining surfaces of the bone around tooth buds and on the endocranial surface of the frontal bone. The major remodeling throughout the remainder of the early facial skeleton begins at about 14 weeks. Before this time the bones enlarge in all directions from their respective ossification centers. Remodeling, as a process that accompanies growth, starts when the definitive form of each of the individual bones of the face and cranium is attained (Fig. 13-17). As the officiation centers appear and begin to grow (Fig. 13-18), the **remodeling** process also begins and serves to progressively shape the individual bones as it simultaneously enlarges them.

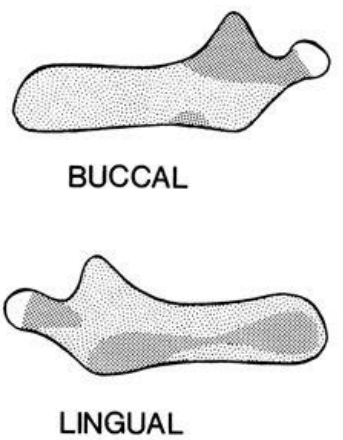


FIGURE 13-17.
Mandible in the last trimester of fetal development. Dark stippling represents resorptive fields, and light stippling indicates depository fields.

Nasomaxillary Complex

The anterior part of the maxilla in both the fetus and the child is depository on lingual surfaces and resorptive on nasal lining surfaces. A major difference exists, however, on the anterior-most (labial) surface. Here it is depository in the fetus, but characteristically becomes resorptive after the first few years following birth when definitive arch length has been attained. During the fetal period, the exterior surface of the entire maxilla, including its anterior part,§ remains depository to provide for increasing arch length in conjunction with the development of the tooth buds and their subsequent enlargement. Resorption occurs on the alveolar lining surfaces surrounding each of the tooth buds. The fetal maxillary arch thus lengthens horizontally in both posterior **and** anterior directions, in contrast to the largely posterior mode of elongation in the later periods of childhood development. In the postnatal face, after the primary teeth have erupted and are being shed to make way for the permanent arch, the anterior (labial) surface of the maxillary arch begins to become resorptive (Kurihara and Enlow, 1980a). This is part of the growth and remodeling process that continues to produce the **downward** growth movement of the maxillary arch and palate.

The posterior and infraorbital surfaces of the maxilla proper are depository in both prenatal and postnatal life. The process of posterior deposition on the maxillary tuberosity progressively increases the maxilla in horizontal length. Deposition on the orbital floor in the fetal skull keeps it in a constant positional relationship with the eyeball, just as in the growing child. The eyeball enlarges in volume at a decreasing rate after the fourth to fifth fetal months. Its volume increases by over 100 per cent before the fifth month, by 50 per cent during the sixth and seventh months, and only by 23 to 30 per cent in the eighth and ninth months. Remodeling of the orbital floor takes place because the entire maxilla, including the orbit, is displaced in a progressively inferior direction in relation to continuing new bone growth at the frontomaxillary suture. At the same time, deposition on the orbital floor serves to carry it superiorly, thereby maintaining it in a constant position relative to the eyeball. The infraorbital canal is also compensating by resorption superior to and deposition inferior to the infraorbital nerve. This maintains a constant relationship between the nerve and the orbital floor, along which the infraorbital nerve passes before entering the infraorbital canal.

The external surface of the frontal process of the maxilla is depository during both prenatal and postnatal facial development. The contralateral nasal side is mostly depository, with some resorption at older fetal ages, but it is entirely resorptive postnatally. This area in the rapidly growing young child is characterized by a massive lateral expansion of the lateral nasal walls, including the etmoidal plates and sinuses. The latter part of fetal life appears to be a transitory period in which these surfaces are just beginning a major lateral expansive movement.

In both the fetal and postnatal periods, the nasal side of the palate (including

§ An old game among facial researchers is arguing whether or not a premaxilla actually exists as a separate bone in man and how many ontogenic ossification centers are involved. Phylogenically, there is no question.

FIGURE 13-18.
Human skull at about 3 months. Intramembranous bones are shown in black. Cartilage is represented by light stippling, and bones developing by endochondral ossification are indicated by darker stippling. Approximate time of appearance for each bone is indicated in parenthesis. 1, Parietal bone (10 weeks). 2, Interparietal bone (8 weeks). 3, Supraoccipital (8 weeks). 4, Dorsum sellae (still cartilaginous). 5, Temporal wing of sphenoid (2 to 3 months; the basisphenoid appears at 12 to 13 weeks, orbitosphenoid of 12 weeks and presphenoid at 5 months). 6, Squamous part of temporal bone (2 to 3 months). 7, Basioccipital (2 to 3 months). 8, Hyoid (still cartilaginous). 9, Thyroid (still cartilaginous). 10, Cricoid (still cartilaginous). 11, Frontal bone (7½ weeks). 12, Crista galli, still cartilaginous (inferiorly, the middle concha begins ossification at 16 weeks, the superior and inferior conchae at 18 weeks, the perpendicular plate of ethmoid begins ossification during the first postnatal year, the cribriform plate during the second postnatal year, the vomer at 8 total weeks). 13, Nasal bone (8 weeks). 14, Lacrimal bone (8½ weeks). 15, Malar (8 weeks). 16, Maxilla (end of 6th week; premaxilla, 7 weeks). 17, Mandible (6 to 8 weeks). 18, Tympanic ring (begins at 9 weeks, with complete ring at 12 weeks); petrous bone, (5 to 6 months). 19, Styloid process, (still cartilaginous). (Modified from Patten, B. M.: *Human Embryology,* 3rd Ed. New York, McGraw-Hill, 1968, with permission.)

the palatine bone) is resorptive except along the midline, and the oral surface is depository. This provides for an inferior remodeling relocation of the palate and a vertical enlargement of the nasal chambers and also, significantly, provides a key means that sustains functional alignment of the palate as the entire maxilla undergoes variable displacement rotations.

The mucosal surface of each vertical plate of the palatine bone is resorptive, and the opposite surface on the lateral nasal wall is depository in both the prenatal and postnatal periods. This provides for the bilateral expansion of this part of the nasal chamber in width.

The Mandible

The beginning fetal mandible, as in the earliest growth stages of the other bones of the skull, initially has outside surfaces that are entirely depository in character. At about 10 weeks, however, resorption begins around the rapidly expanding tooth buds and is present thereafter. By 13 weeks, distinct resorptive fields are becoming established on the buccal side of the coronoid process, on the lingual side of the ramus, and on the lingual side of the posterior part of the corpus. The anterior edge of the ramus is already resorptive, and the posterior border is depository. In some specimens, however, the anterior margin along the tip of the coronoid process shows deposition, suggesting a "rotation" to a more upright position (see Chapter 4). By 26 weeks, the basic growth and remodeling pattern that continues on into postnatal development is seen except, notably, in the incisor region (Fig. 13-17). In the fetal and early postnatal mandible, the entire labial side of the anterior part of the corpus is still depository. As in the fetal and young postnatal maxilla, the fetal mandibular corpus grows and lengthens **mesially** as well as distally in conjunction with the establishment of the primary dentition. The lingual side of the fetal corpus in the incisor region is resorptive after about the fifteenth week in most (but not all) mandibles. This contributes to a forward remodeling movement of the entire incisor region of the corpus. Subsequent to the deciduous dentition period of childhood growth, however, the alveolar bone on the labial side in the forward part of the arch undergoes a reversal to become **resorptive**, and the opposite lingual side becomes uniformly depository. This change occurs in conjunction with the unique lingual direction of incisor movement in the child's mandible. From this time, the chin begins to take on a progressively more prominent form; the mental protuberance continues to remodel anteriorly, while the alveolar bone above it remodels posteriorly until the lower permanent incisors reach their definitive positions. (See Kurihara and Enlow, 1980a, for more details.)

A DEVELOPMENTAL TIMETABLE

A "priority plan" exists during the prenatal growth and development of the face and the body in general. Some specific organs and anatomic parts are given priority status in earlier timing and/or growth rate; some other parts receive partially deferred attention. This is determined, in general, by the urgency of a given part's functional role in the early physiologic relationships of the developing fetus. Certain developing anatomic components, such as the cardiovascular system and many parts of the nervous system, are essential to the maturation of all other parts and to fetal life itself. Components such as the lungs and the nasal and oral parts of the face and the urogenital and digestive systems are essentially nonfunctional during the fetal period. Their respective roles are carried out by placental interchanges. Although their prenatal maturation is incomplete, these growth-delayed parts must nonetheless achieve readiness for immediate and full newborn-level function at the instant of birth. This is "prospective developmental differentiation." That is, structure develops even though function has yet to

exert its ontogenic influence on structural design and differentiation. This is an extension, not an exception, of the "structure and function interplay" principle of biology, with the phylogeny of function establishing a provisional developmental program. Airway size must be prepared and adequate to accommodate newborn lung size, which, in turn, must be sufficient to meet the functional needs for the size of the body at the time. Neonatal oral functions must also be ready to respond, virtually instantaneously, upon birth. Thereafter, as described throughout earlier chapters, differential extents and rates of developmental maturation occur among the multitude of different body (and facial) regions and parts. The neonatal brain, calvaria, basicranium, and eyes are relatively large in comparison with the proportionately much shorter face. However, as general body size progressively increases, the lungs enlarge to match, and the nasal part of the face (not just the external nose) correspondingly begins to increase significantly in height and length. The dentition begins to emerge, chewing begins to replace suckling, neural reflexes change, swallowing patterns change, and the oral part of the face, with its rapidly enlarging masticatory muscles and growing and developing jaw bones, keeps pace. All of the multitude of regional parts proceed developmentally within a general field of facial growth, which has a perimeter prescribed by the precocious basicranial template.

14

Bone and Cartilage

Cartilage provides three basic functions. It gives **flexible** support in appropriate anatomic places (the nasal tip, ear lobe, thoracic cage, tracheal rings); it is a **pressure-tolerant** tissue located in specific skeletal areas where direct pressure occurs (articular cartilage); and it functions as a **"growth cartilage"** in conjunction with certain enlarging bones (synchondrosis, condylar cartilage, epiphyseal plate). Cartilage is a nonvascular connective tissue and it is ordinarily noncalcified. Both vascularization and calcification, however, are involved as steps in the replacement process by bone tissue.

Several distinctive structural features relate to cartilage. First these are listed, and then the nature of their interrelationships is explained in terms of the various fundamental functions of cartilage.

Cartilage is a special type of connective tissue that has a stiff, firm, but **not hard** intercellular matrix. It provides rigid support, but it is so soft that it can be cut with a fingernail. This feature is based on the exceptionally high content of water-bound ground substance. The rich amount of chondroitin sulfate (Gr. *chondros*, cartilage) in the cartilage matrix is associated with a noncollagenous protein, and this combination has the special property of marked hydrophilia. This gives the turgid, firm character to the matrix. Cartilage thus develops in a variety of body locations where flexible (not brittle) support is appropriate. Cartilage has an enclosing vascular chondrogenic membrane, but it can have surfaces without one. Cartilage can grow appositionally and/or interstitially.

Because the matrix is noncalcified, the matrix is also able to be nonvascular. Nutrients and metabolic wastes diffuse directly through the soft matrix to and from cells. Thus, blood vessels are not required within cartilage as they usually are in bone, with its hard, impervious matrix.

Because the matrix is nonvascular, cartilage is pressure tolerant. There are no vessels near the surface to mash closed by compression, thereby allowing cartilage to operate metabolically because there are no pathways of supply to occlude. The water-bound, noncompressible matrix, furthermore, is not badly distorted by the force, and its turgid nature protects the cells within it from surface pressures.

Unlike bone, cartilage can function without a covering membrane because its nonvascular matrix is thus not dependent upon **surface** blood vessels in an enclosing **surface** membrane.

Because cartilage can function without a covering membrane, it is especially adaptable to sites involving pressure, as on long-bone articular surfaces and the surfaces of epiphyseal plates. If a soft connective tissue membrane were present,

its vessels would be closed off by the compression, and the cells would be subject to anoxia as well as damage by the direct pressure itself. Furthermore, a delicate perichondrial membrane could not withstand abrasive articular movements. In conjunction with synovial secretions, however, the naked surface of the articular cartilage provides relatively frictionless movements while bearing great weight under severe pressure. (Note: The "secondary" cartilage of the mandibular condyle involves a specialized tissue system. Another tissue type having a characteristic capacity for pressure tolerance is dense non-vascular and relatively acellular collagenous connective tissue. The secondary condylar cartilage has a unique "capsule" composed of such tissue.)

Because cartilage has an interstitial as well as a membrane-dependent appositional mode of growth, it can thereby still grow in those pressure areas where an enclosing connective tissue membrane is absent. This includes articular surfaces, synchondroses, and epiphyseal plates. (Note again, the "secondary" nature of the condylar cartilage has a specialized system, as described in Chapter 4.)

Because ordinary cartilage matrix is not calcified, cell divisions can take place—which is not the case in bone—thus providing interstitial growth.

One can readily see how each and all of the above features interrelate and are directly interdependent. To perform the functions of a **growth cartilage** in endochondral sites of bone growth, no one of these features could work without all the others. Cartilage exists as a separate, special tissue type because of these special features, and its multiple functions could not be carried out by **any** of the other soft or hard tissues.

BONE

Bone provides the specialized feature of **hardness**. Because of this, it has several unique developmental characteristics. A bone, of course, cannot enlarge by interstitial growth because its cells are locked into a non-expandable matrix. It is thus dependent upon covering vascular membranes (periosteum and endosteum) providing the osteogenic capacity for an appositional system of growth. This is also why bone is necessarily a **traction** (tension) -adapted kind of tissue and why "bone" is said to be "pressure-sensitive."* The covering soft tissue membrane is sensitive to direct compression, because any undue amount would occlude its blood vessels, interfere with osteoblastic deposition of new bone, and cause avascular necrosis.

* The whole subject of pressure and tension is greatly oversimplified in most routine descriptions, as it is also in the present discussion. The actual nature of the forces that act on a bone is quite complex and can seldom be designated purely as either tension or pressure. A bundle of periosteal fibers, for example, can exert tension on a bone surface, but that same surface can also be under a compressive influence from other sources, such as a flexure pressure effect on a concave curvature. Pressure effects can also be exerted by intercellular fluids in a region otherwise classified as under tension. An osteogenic cell located between collagenous fibers under tension actually receives a direct pressure effect. Moreover, compressive effects on the hard part of the bone itself can have an osteoblastic trigger effect, while compressive effects on blood vessels can have an osteoclastic effect. The old, greatly oversimplified, and inaccurate concept that one-to-one tension-deposition and pressure-resorption relationships exist is no longer acceptable, as described in Chapter 12. The overall growth control system is much more complex, and is incompletely understood at present.

Actually, it is the periosteal **membrane** and not the hard part of the bone itself that is pressure sensitive (see page 240). The magnitude of compression is such that it cannot exceed capillary pressure, which is a "light" force of about 25 gm/cm^2.

The degree of vascular flow is affected by the amount and type of mechanical force acting on a soft tissue, and this is directly involved in initiating either chondrogenesis or osteogenesis. More extreme levels of hypoxia caused by higher levels of pressure are known to stimulate formation of chondroblasts leading to nonvascular cartilage, rather than osteoblasts, from undifferentiated connective tissue cells. For all of these various reasons, two basic modes of bone growth thus exist, one (intramembranous) is adapted to a localized environment of tension (or at least levels of pressure less than that of capillary pressure), and the other (endochondral) to more extreme compressive forces.

Soft tissues, in general, grow (1) by increasing the **number** of cells (as in epithelia); (2) by increasing the **size** of cells (as in skeletal muscle); or (3) by increasing the amount of **matrix** between cells (as in loose connective tissue). Many tissue types combine two or all three of these different modes of growth (as in cartilage). All are interstitial systems of growth because they involve expansive changes of tissue components already present. Bone, because it is calcified, must necessarily grow by a process of adding new cells and new matrix onto the existing **surfaces** of previously formed generations of bone tissue. It cannot, of course, expand interstitially by division and proliferation of osteocytes because the cells have no place to divide to; they and their genes are locked into their calcified, nonexpanding matrix. Bone **must**, therefore, grow in relationship to a covering or lining membrane. It is the bone surface, either periosteal or endosteal, that is the site of growth activity. This type of growth is termed "appositional," in contrast to "interstitial" expansion. All bone that is fully calcified grows in this manner regardless of its mode of osteogenesis (endochondral or intramembranous).

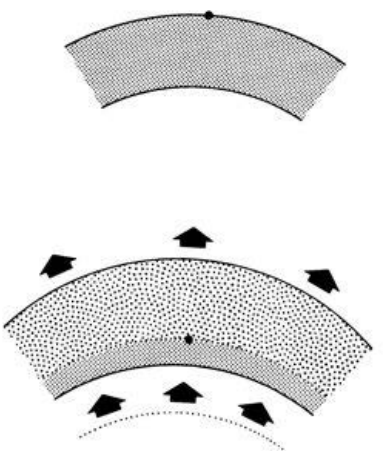
Where **compression** is involved that exceeds the connective tissue membrane's threshold level of capillary pressure, as mentioned above, the intramembranous growth mechanism (which is dependent upon vascular membranes, as the name indicates) does not have the capacity to function. Thus, a growth cartilage grows **toward** the site of compression. Epiphyseal plates, synchondroses, and other "growth cartilages" provide for the linear enlargement of bones that have pressure contacts at their ends. The cartilage grows interstitially and/or by apposition on one side as the older part of the cartilage on the other side is removed and replaced by bone. The cartilage functions essentially as a kind of advance ram that shields the sensitive endosteal bone membrane beneath it and, importantly, **also provides for the growth elongation of the bone at the same time**. The **other** areas of the bone grow intramembranously.

During active growth, a depository bone surface is constantly changing because of new additions; any given point within the compact substance of bone, however deep, **used** to be an actual exposed surface, either periosteal or endosteal. If a **metallic implant marker** or **vital dye** (such as alizarin, or tetracycline, or the procion dyes) is used in a living bone, growth changes that occur after the marker is implanted or the dye is administered can be determined. Such markers and the lines formed by the vital dyes become covered over wherever subsequent surface

deposition occurs. (Note: Vital dyes stain only that bone actively being laid down during the period in which the dye is in the bloodstream. Thus, one injection forms a thin, colored line on all the active surfaces of the bone. Subsequently formed bone deposits are not colored.) Metallic implants (tiny tantalum bits) can be injected into the cortex of a growing bone by means of a special "gun." These radiopaque markers can then be seen in x-ray films taken days, weeks, or even years later in order to determine where and how much a bone has been **remodeled** by deposition and resorption relative to the markers. **Displacement** movements of whole bones can also be determined by noting the directions and amounts of separation among implants previously inserted into two or more separate bones.

Bone **deposition** is only part of the overall process of bone enlargement; it is one phase of a multiphase growth system. **Resorption** is another part, and this is just as important and necessary as deposition (Fig. 16-1). Ordinary resorption associated with growth is not "pathologic," although beginning students sometimes mistakenly view it as evil because resorption is a destructive process or because it can also be involved in some diseases. Resorption **must** accompany deposition, and deposition on one side of a cortical plate (or cancellous trabecula) with resorption from the other brings about the growth movement ("relocation") of the part. Thus, deposition on the periosteal side with resorption on the endosteal surface of a cortex moves the whole cortex outward and at the same

FIGURE 16-1



time proportionately increases its thickness. Note, that a surface implant (Fig. 16-1) becomes translocated from one side to the other by this process. The implant itself does not move; rather, the remodeling bone moves around the implant. As pointed out in Chapter 2, **remodeling** also takes place everywhere throughout an entire bone because the bone does not simply expand by generalized external deposition and internal resorption.

Remodeling (Fig. 16-2) involves various combinations of deposition (*1* and *3*) and resorption (*2* and *4*) on different periosteal and endosteal surfaces in order to **move** (relocate) a part of a growing bone into a new location (see Chapter 2). The process of relocation shapes and sizes a bone continuously as a basic part of the growth mechanism.

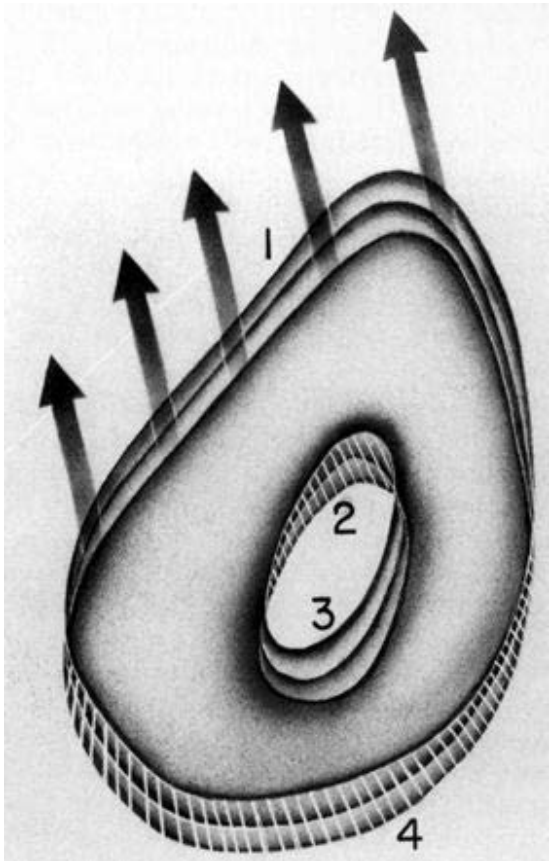


FIGURE 16-2.
(From Enlow, D. H.: *Principles of Bone Remodeling*. Springfield, III., Charles C Thomas, 1963, with permission.)

Surface depository activity is by the osteoblasts of the innermost cellular layer of the thick periosteum or the very thin endosteum. The latter is only about one cell thick and lacks a thick, fibrous layer because muscles, tendons, and other such force-adapted tissues are not attached to it. Vessels enclosed within endosteal circumferential layers of bone characteristically enter at a right angle because the endosteum is not under tension and its vessels, therefore, are not drawn out toward the long axis of the bone. Periosteal "slippage" over a bone surface is involved in the periosteum in relation to the forces acting on it as it grows, and this causes the periosteal vessels to become enclosed at much more acute angles. The old notion of perpendicular "Volkmann" canals entering the bone from the outer periosteum needs to be forgotten. (See Enlow, 1963, for an in-depth, early historical account of bone as a tissue.)

As new bone is progressively laid down, the covering periosteal membrane **grows** in an outward direction. The outer membrane grows inward if the periosteal surface is resorptive. However, these membranes do not simply "move" as they bring about the cortical movement. They are not merely pushed or pulled or somehow migrate into their new positions. Rather, the membranes each **grow** from the one

location to the other. It is the growth movement of the outer and inner membranes that produces the growth movement (remodeling) of the bony cortex located between these "genic" membranes. The membrane has its own internal, interstitial growth process. Just as the bone **remodels** during growth, the periosteum also undergoes its own internal remodeling process. Remember that the membrane itself paces the bone changes, and that the "fields" of growth activity (described in Chapter 2) reside in this membrane and the other soft tissues, rather than within or on the bone itself.

As collagenous fibers and ground substances are laid down by the osteoblasts (*g* in Fig. 16-3), this new layer of **osteoid** almost simultaneously undergoes calcification to become bone tissue (*x* in Fig. 16-4). Some of the osteoblasts are enclosed to become new osteocytes, and some of the periosteal blood vessels contiguous with the bone surface are also incorporated. The anastomosing vessels then lie within a network of **vascular canals** as bone is formed around them. Note that the fibers of attachment (Sharpeys fibers) become more deeply embedded as new bone is formed around the collagenous fibers in the innermost layer of the periosteum (*d*). The periosteal fibers now, themselves, **grow** and lengthen by their own remodeling outward. Fiber segment *d* thus lengthens on the outside while it is being enclosed by new bone on the inside (e^1 in Fig. 16-4). It does this by **conversion** of segment *c* into a new addition onto *d*, which elongates by this process. Segment *c* is a special **precollagenous** fibril. It is a very thin fibril that requires special staining methods (see Kraw and Enlow, 1967). Many such fibrils (i.e., "linkage" fibrils) form a distinctive zone within the periosteal membrane. Under the control of the rich population of fibroblasts, these slender fibrils become remodeled by coalescence into the thick collagenous fibers that form the lengthening segments of *d*. This is done by binding with ground substance (proteoglycans).

FIGURE 16-3

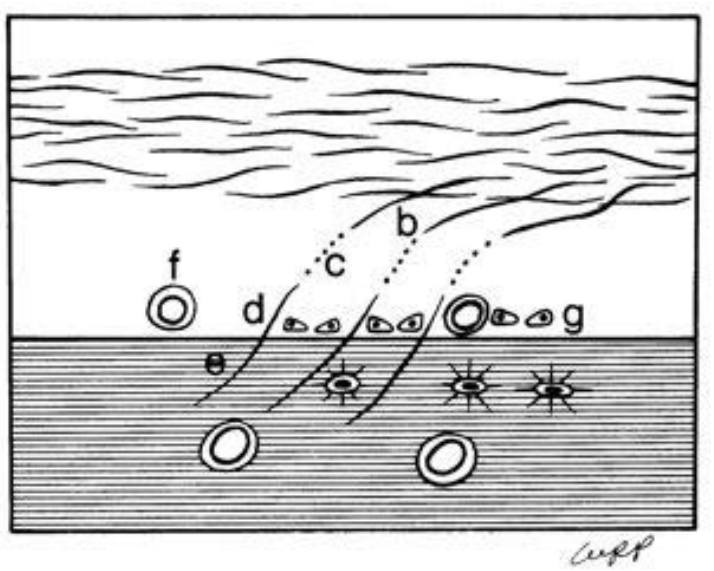
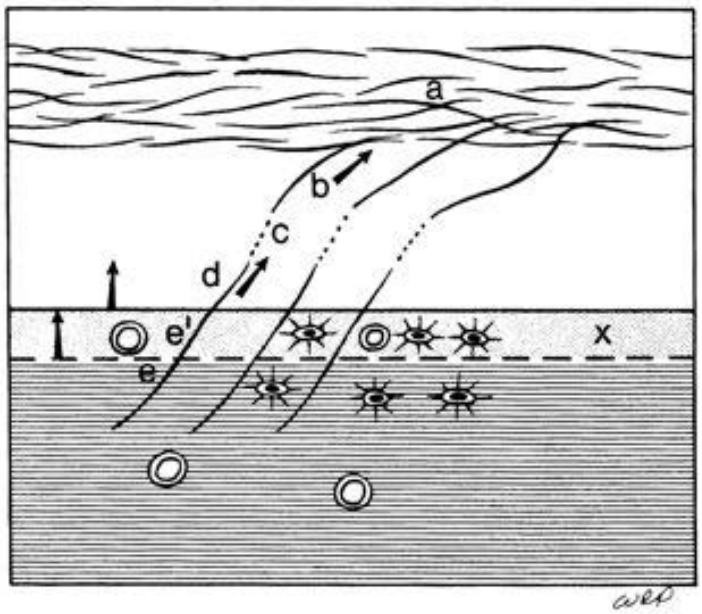


FIGURE 16-4



Segment *c* now lengthens in a direction away from the bone surface. It is not presently known whether this is done by a remodeling conversion from segment *b* through enzymatic removal of the binding ground substance to release its numerous, slender precollagenous fibrils, or whether new precollagenous lengths are added directly to *c* by the fibroblasts in this zone. As these changes occur, however, new *b* segments are being formed by fibroblastic activity as they join the expanding outer, dense "fibrous" layer of the periosteum (*a*). The entire periosteum thus **relocates** outward as the bone surface correspondingly drifts in the same direction. If the periosteal surface is **resorptive**, rather than depository, the sequence of operations is the same, but the direction is reversed. That is, the periosteum and its fibers develop **toward**, rather than away from, the bone surface, which is moving inward, rather than outward.

How does a muscle or tendon maintain continuous attachment onto a bone surface that is **resorptive**? Moreover, how does a muscle **migrate** over a bone surface (whether resorptive or depository) as the bone lengthens? For example, the muscle shown in Figures 16-5 and 16-6 **moves** its insertion. It must also sustain constant attachment on a mixed remodeling surface, part of which is undergoing progressive resorption and part deposition. The other muscle shown in Figure 16-6 attaches entirely onto a resorptive surface. Bone resorption is customarily regarded as a process that results in total destruction of the bone tissue, including as well its anchoring fibers of attachment In many non—stress-bearing locations, this can be true. In some (not all) areas involving muscle, tendon, ligament, and periodontal attachments, however, there are several histogenic means by which fibrous attachment is sustained. First, although not common, the process of fiber destruction is not necessarily complete. Some of the fibers in the ordinary bone

matrix are not removed by the resorptive process, especially when aligned in the direction of strain. These fibers become uncovered as the remainder of the bone matrix around them is resorbed, and they are then freed to function as fibers of the **periosteum** while retaining continuity with the fibers in the bone of which they were once a part. A second and much more commonly seen histogenetic mechanism involves an "adhesive" mode of attachment in which certain fibroblast-like cells secrete proteoglycans onto a naked resorptive surface (Kurihara and Enlow, 1980b and c). New precollagenous fibrils are then formed as the adhesive secretion of proteoglycans continues. The new fibrils then link to the more mature fibers in the periosteum, the proteoglycans serving as the binding agent (Fig. 16-7). If new bone is subsequently deposited by the periosteum, the calcified adhesive interface then becomes a "reversal line." (Note: A comparable process also occurs in periodontal attachments on remodeling alveolar bone.) This cycles over and over as the process continues.

In other stress-bearing locations, another separate mechanism is seen that provides for continuous fibrous attachments on resorptive, remodeling, and relocating bone surfaces. This involves a reconstruction of the bone **deep**

FIGURE 16-5

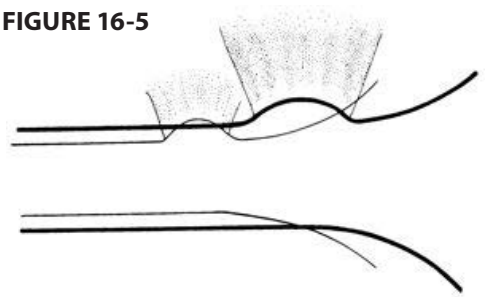


FIGURE 16-6

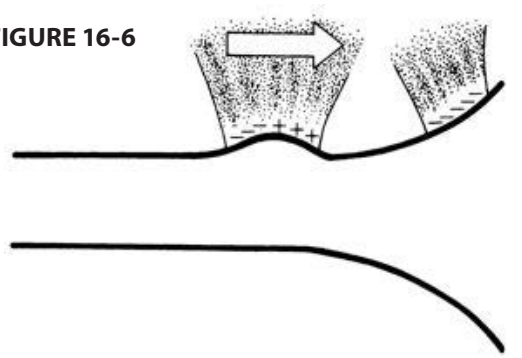


FIGURE 16-7

to the resorptive surface in order to provide uninterrupted fibrous anchorage. "Undermining" resorption takes place, in which resorption canals are formed well below the surface of the inward-advancing resorptive periosteal front. New bone is then laid down with these deeper, protected spaces, and it is this attachment that provides additional fibrous anchorage while the outside bone surface itself is undergoing resorptive removal. Thus, as periosteal surface is resorbed, a large number of resorptive spaces are formed (in histologic sections, many appear as cut-off canals). These spaces anastomose with each other. The fibers in the new bone subsequently deposited in them are continuous by relinking with the remodeling fibers of the periosteum, and anchorage is thereby maintained (Fig. 16-8). The structural result is a generation of haversian systems (secondary osteons) **deep** to the surface. The fibrous matrix of each osteon and its connection by labile linkage fibrils to the inward-moving resorptive periosteum are protected from resorption until the resorptive front reaches them. However, new waves of haversian systems are constantly being formed in advance of the resorptive front, so that new deeper osteons replace, in turn, those that become exposed as they reach the resorptive periosteal surface. Moreover, a muscle moves and migrates along a bone surface by this same process of haversian formation, as well as by lateral reconnections of the labile linkage fibrils (*x*) in the intermediate part of the periosteum (or equivalent areas for direct tendon insertions). Thus, the precollagenous linkage fibrils connecting with fiber *a* in the outer part of the periosteum (Figs. 16-9 and 16-10) become recombined with the precollagenous fibrils of fiber b^1 in the inner part of the periosteum, and so on. This progression moves the entire muscle along the bone surface to keep pace with the elongation of the entire growing bone. Separations of fiber bundles by enzymatic removal of the ground substance binding, together with precollagenous fibril regoupings by new ground substance formation, are believed to carry this out. See also Figure 16-20.

Sutures have an osteogenic process comparable to periosteal bone growth. The suture is an inward reflection of the periosteal membrane, and the fibrous, linkage, and osteoblastic zones are directly continuous from one to the other (Figs. 16-11 and 16-12). As a new layer of bone is added (*x*), inner collagenous fibers *d* become embedded to form new attachment fibers (e^1) in the bone matrix. The *d* fibers, however, lengthen by conversion from the labile linkage fibers (*c*) just as previously described for the periosteum, and *d* fibers converted, in turn, into lengthening *c* fibers (or fibroblastic activity may bring about direct lengthening of *c*, as in the periosteum). As new bone is added to the sutural contact surfaces, the bones are simultaneously displaced away from one another (see Chapter 2 for a discussion of the physical forces that cause this displacement). Many sutures have three basic layers (on each side), as indicated here (Fig. 16-13). Some sutural types, however, have another layer of loosely arranged fibers located within the center of the dense-fibered capsular layer dividing the two sides. The basic plan of growth and remodeling, however, is the same. When the process of growth ceases, the suture becomes essentially a mature ligament, and the precollagenous linkage fibrils are no longer present.

Importantly, the source of the propulsive force that produces the "downward and forward" displacement movement of the nasomaxillary complex at its various

FIGURE 16-8

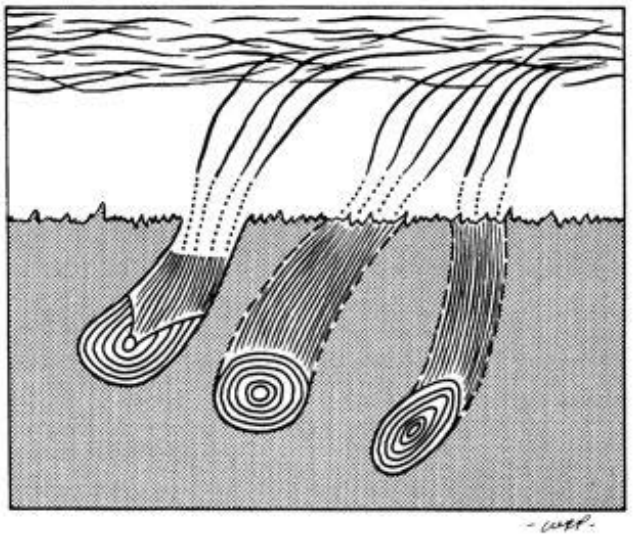


FIGURE 16-9

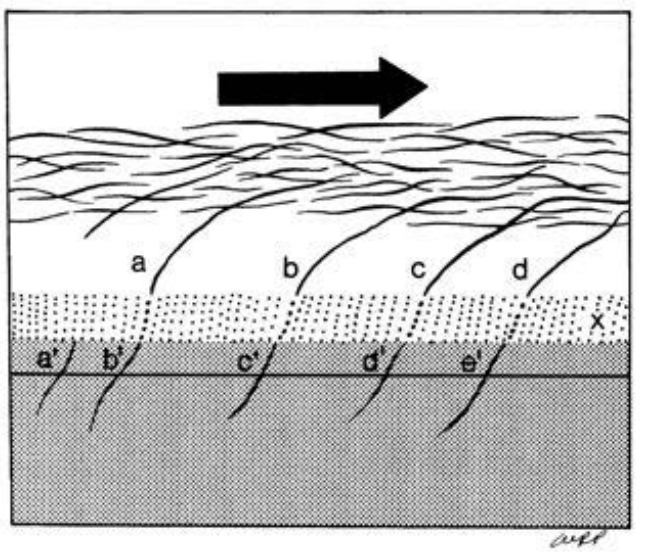


FIGURE 16-10

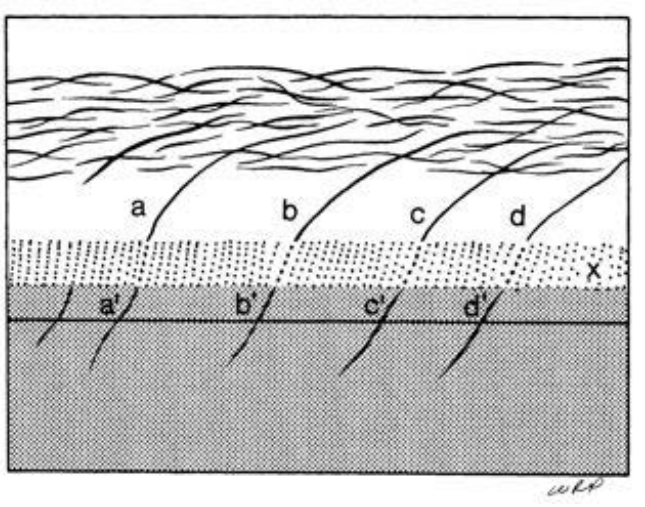


FIGURE 16-11

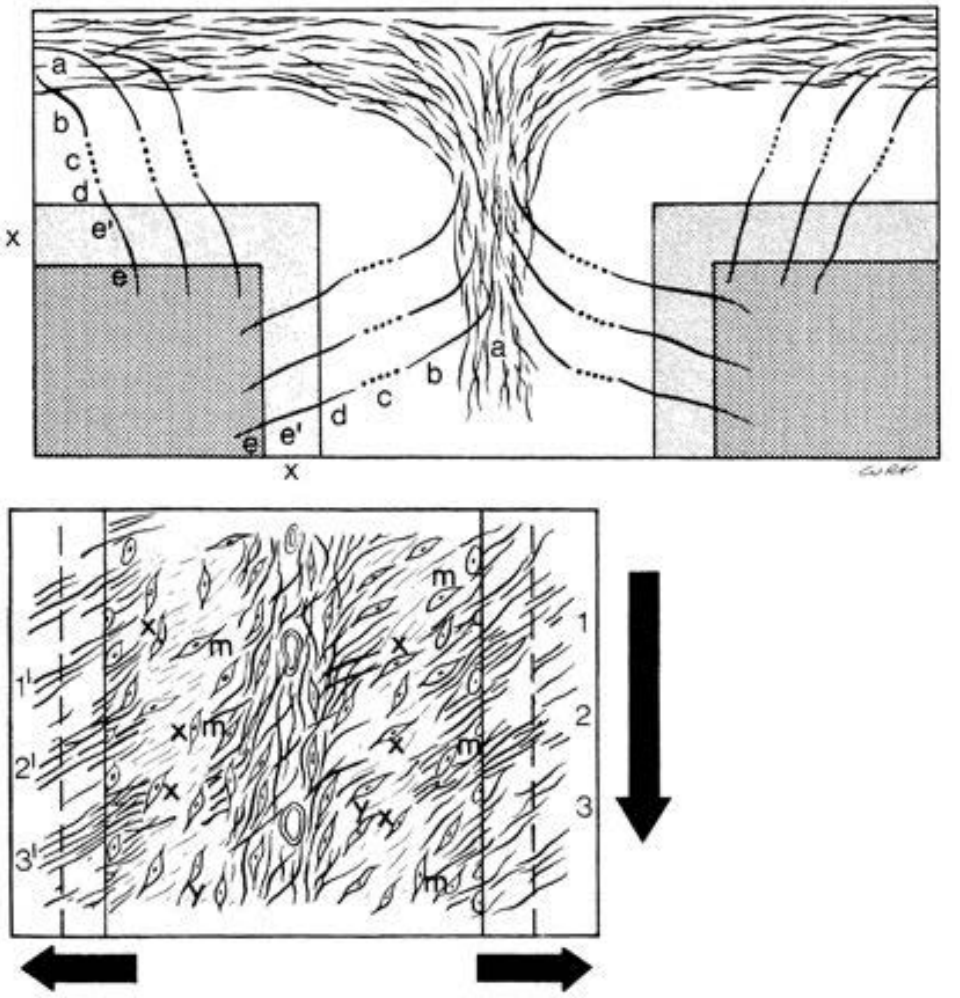
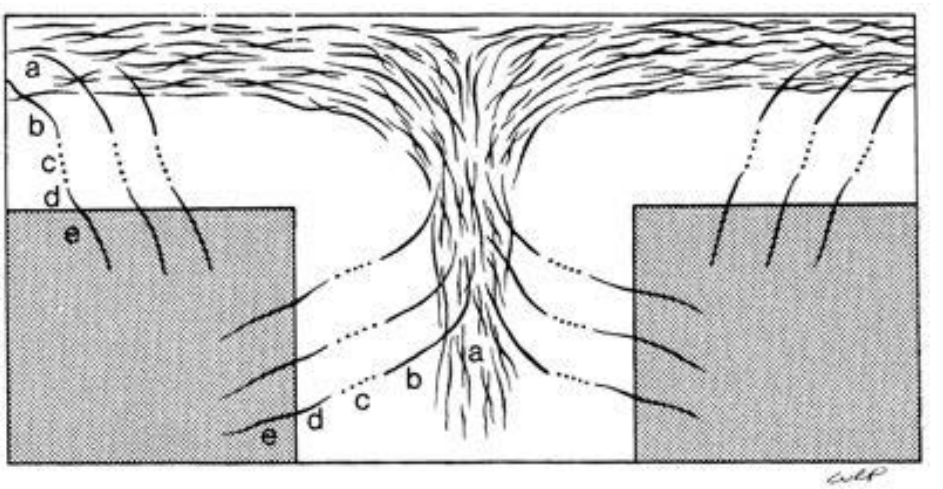


FIGURE 16-12



sutures has long been a subject of controversy. It has recently been suggested that the abundant population of actively contractile fibroblasts ("myofibroblasts," *m* cells in Fig. 16-12) within the linkage zone of the sutural membrane provide, at least in part, a contractile force that exerts tension on the fibrous framework. This, in turn, presumably pulls one bone along its sutural interface with another bone or at least contributes to the moving, relinking placements of the fibers as some other propulsive force causes the bone movements. The bone thus "slides" along the suture as new bone tissue, at the same time, is laid down on the sutural edges. The midface is thereby **pulled** forward and downward along its multiple sutural surfaces. Special collagen-degrading and collagen-producing fibroblasts (*x* and *y*) provide the fiber remodeling and ground substance relinkage changes also involved. Fibers at level *1*, which were formerly linked with fibers at level 1^1, have become relinked with fibers at 2^1, and so on. (See Azuma et al., 1975.) Refer to pages 98 and 106.

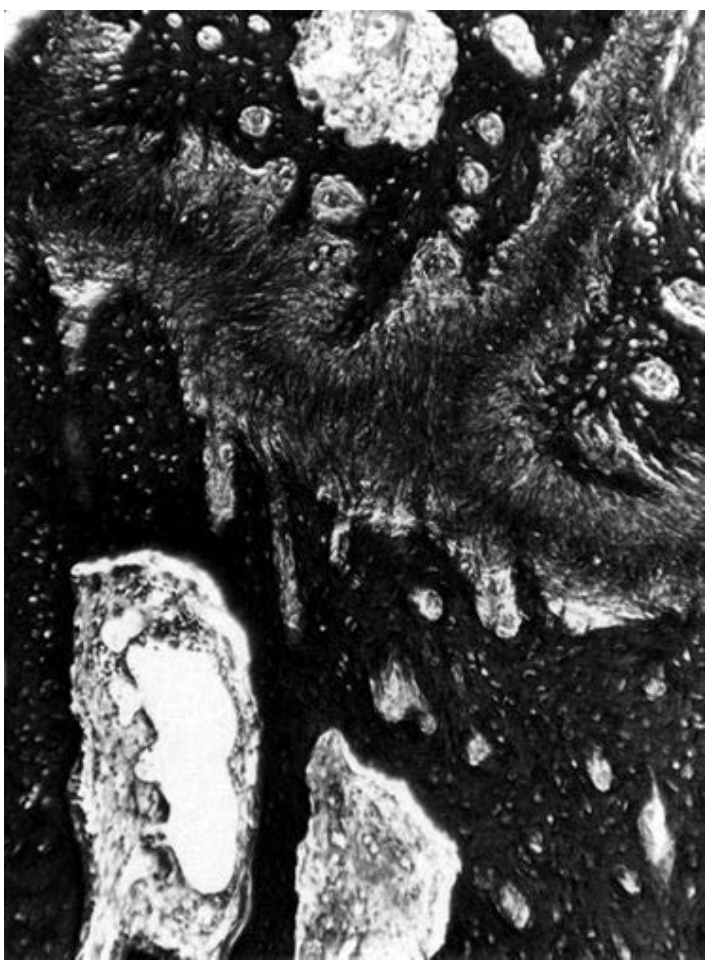


FIGURE 16-13.
(From Enlow, D. H.: *The Human Face.* New York, Harper & Row, 1968, p. 96, with permission.)

BONE TISSUE TYPES RELATED TO DEVELOPMENT AND FUNCTION

Histology textbooks teach that the haversian system (secondary osteon) is the structural "unit" of bone. This is quite incorrect. In the bone of the young, growing child, the haversian system is **not** a major structural feature. (Most vertebrate groups and species lack haversian systems altogether. See Enlow, 1963.) This old haversian system notion not only has misled students of dentistry and medicine, but also has concealed an important concept. There are other, much more widespread kinds of bone in the child's growing skeleton. The concept is this: different functional and developmental circumstances and conditions exist, and there is a specific histologic type of bone tissue for each. Some bone types are fast growing, others slow growing. Some bone types grow inward, others outward. Some are associated with muscles, tendon, or periodontal attachments; others are not. Some bone types form a thick cortex; others, a thin cortex. Some relate to a dense vascular supply; others, to scant vascularization—and so on. "Haversian systems" could not do all this. These are important points because a basic feature of bone is its developmental versatility and adaptability as a tissue.

Primary vascular bone tissue (Figs. 16-14 and 16-17) is the principal type of periosteal cortical bone in the growing skeleton of the child. The vessels are enclosed within canals as new bone is laid down around each vessel in the osteogenic part of the periosteum. These canals are not derived from precursor secondary resorption spaces. If the bone is fast growing, many vessels and their canals characteristically become enclosed. If it is slower growing, fewer or even no canals are incorporated

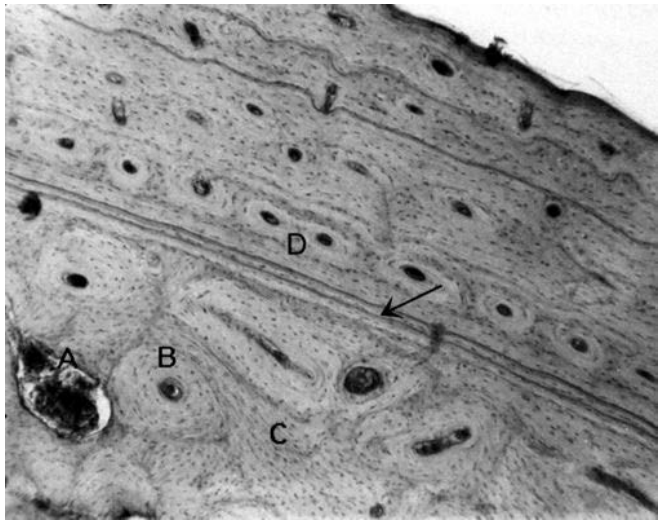


FIGURE 16-14.
The lower half of this cortical section shows an endosteal layer replaced by secondary osteons. The upper half is a periosteal layer composed of a layered mix of lamellar and non-lamellar bone containing primary vascular canals and primary osteons. (From Enlow, D. H.: *Principles of Bone Remodeling.* Springfield, Ill, Charles C Thomas, 1963, with permission.)

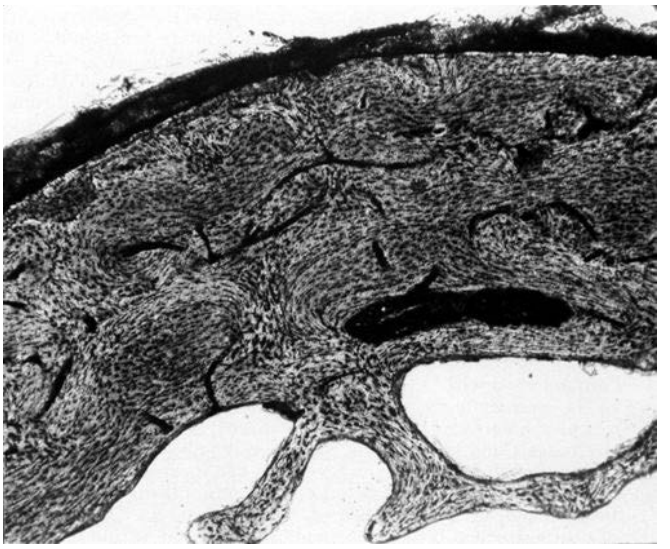


FIGURE 16-15.
This section was taken from an inward-growing region of the cortex. It is compacted cancellous bone produced by endosteal deposition and periosteal resorption. The large spaces between the coarse cancellous trabeculae have been filled with lamellar bone. (From Enlow, D. H.: A study of the postnatal growth and remodeling of bone. Am. J. Anat., 110:79, 1962, with permission.)

within the compact bone substance. **Compacted coarse cancellous** bone (Fig. 16-15) is the principal cortical type formed by the endosteum. One half to two thirds of all the cortical bone in the body is composed of this important, distinctive structural type. It is formed by the inward growth of the cortex into the medulla (i.e., periosteal resorption and endosteal deposition). Medullary cancellous bone is converted into cortical compact bone by filling the spaces until they are reduced to vascular canal size (Figs. 16-16, and 16-17). Although a major and widespread bone tissue type, standard histology texts have yet to recognize it, just as with the primary vascular type just described.

Fine cancellous bone (Fig. 16-18) is one of the fastest growing types. It is formed throughout the fetal skeleton and also occurs in rapidly enlarging parts of all postnatal bones. This type of cortical bone tissue is characterized by spaces that are larger than ordinary vascular canals, but smaller than the coarse cancelli of the medulla. **Nonlamellar** (also called "fibrous") bone is also a rapid-growing type, and it occurs in conjunction with fine cancellous bone formation, although "compact" areas of a fast growing cortex may also be nonlamellar. (See Enlow, 1963 and 1990 for further details.)

Bundle bone is characterized by dense inclusions of attachment fibers from the periodontal membrane (see Fig. 7-3). This bone type is formed only on the depository sides of the alveolar socket. The resorptive side is usually composed of compacted coarse cancellous (endosteal) bone or, if the alveolar plate is very thin, of bundle bone formed on the depository side, but translated over to the resorptive side as alveolar drift proceeds. **Chondroid** bone is found at the apex of the alveolar

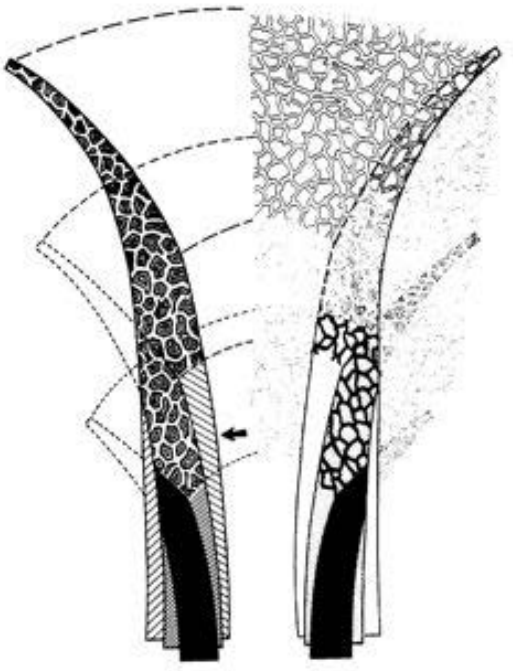


FIGURE 16-16.
The wide end of a bone grows in a longitudinal direction by deposition on the endosteal side and resorption from the periosteal side. This is because the inside surface actually faces toward the direction of growth. Medullary bone is converted into cortical bone by cancellous compaction. In areas where cancellous trabeculae are no longer present, however, inward growth involves deposition of inner circumferential bone (arrow). The inward mode of growth also serves to reduce the wide part into the more narrow part (diaphysis) as the whole bone lengthens. In the diaphysis, the direction of growth reverses, and periosteal bone is laid down. (From Enlow, D. H.: *Principles of Bone Remodeling*. Springfield, III. Charles C Thomas, 1963, with permission.)

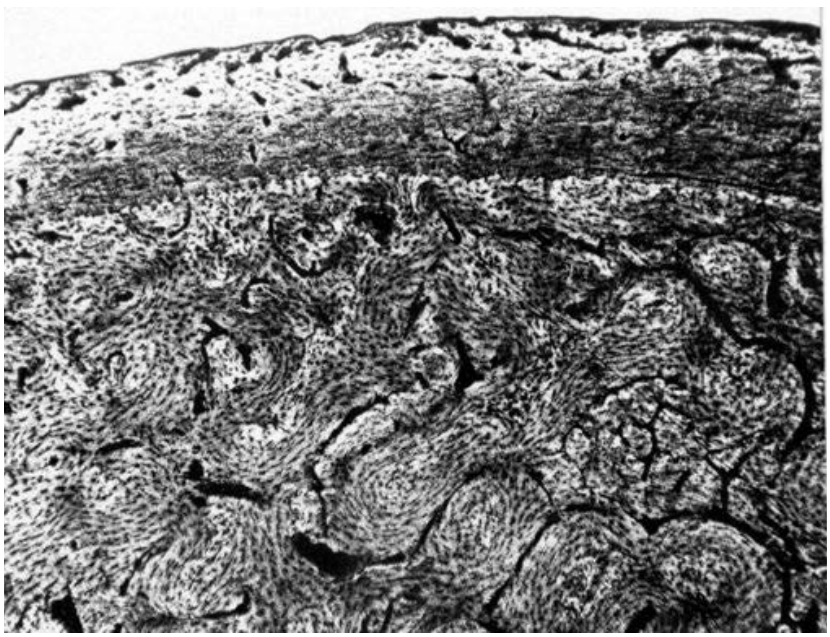


FIGURE 16-17.
The inner layer in this transverse section of cortical bone was produced during a period of inward (endosteal) growth and was formed by the process of cancellous compaction. After outward reversal, a periosteal layer of primary vascular bone was subsequently laid down. Note the reversal front between these two zones. (From Enlow. D. H.: A study of the postnatal growth and remodeling of bone. Am. J. Anat., 110:79, 1962. with permission.)

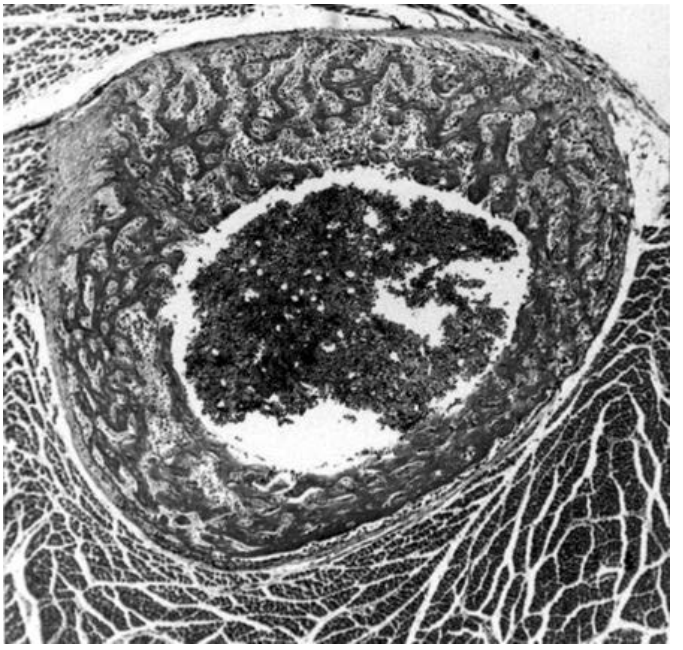


FIGURE 16-18.
The cortices in fetal bones are composed of fine cancellous, nonlamellar bone tissue. Note the relatively small connective tissue-filled spaces. Areas of very fast growth in postnatal bones can also be fine cancellous in structure. (From Enlow, D. H.: A study of the postnatal growth and remodeling of bone. Am. J. Anat. 110:79, 1962, with permission.)

rim and other rapidly forming areas throughout the skeleton (such as the apex of growing tuberosities where tendons attach). This bone tissue type resembles cartilage because of the large, rounded appearance of its crowded osteocytes surrounded by a nonlamellar, basophilic matrix. Because it undergoes internal metaplasia into **other** bone tissues types, chondroid bone is perhaps the only kind of bone tissue that actually has what might be regarded as an interstitial mode of growth.

Haversian Systems

When haversian (secondary) replacement of bone occurs, several functional reasons are involved, in addition to that already described, and relate to conditions that develop in the older skeleton. First, during childhood growth, generalized remodeling during growth provides a constant **turnover** of bone tissue so long as growth continues. The bone is not present long enough for any marked extent of osteocyte aging and necrosis to develop. (Bone cells have a finite life period.) Moreover, the histologic types of childhood bone are more highly vascular because they are mostly rapid forming, as just seen. This favors osteocyte survival. As a child matures and growth slows, however, slower forming bone types become more widespread, and these tend to be less dense in vascular distribution, which

promotes earlier onset of osteocyte necrosis. Because "growth remodeling" no longer replaces bone in the adult, "haversian remodeling" then becomes operative to provide vital, new bone by internal cortical reconstruction. Resorption canals followed by concentric deposition of Haversian lamellae within them thus replaces old, dying, or dead cortical bone, and the result is a secondary osteon. This reconstruction process **also** contributes to mineral homeostasis in older individuals, since more aged bone is less labile in surface ion exchanges. Another function of haversian reconstruction is to provide replacement for bone that has experienced extensive structural fatigue involving extensive "microfracture" accumulation over time.

These are important factors leading to osseointegration of dental implants. The implant is placed by the surgeon and the bone adjacent to the implant undergoes necrosis. Cutting and filling cones (See Fig. 16-19) then sequentially replace the bone contiguous with the implant, resulting in an implant that is "part" of the bone (osseointegrated).

In summary, thus, the continuous renewal of childhood bone by the growth (remodeling) process becomes largely discontinued in the adult. But, a different form of the same remodeling mechanisms (resorption and deposition) takes over in a way that does not change the size and shape of the skeleton; that is, haversian (secondary) reconstruction within the existing bone.

An interesting point is that the haversian system is a visible example of a basic biologic concept, which is the principle of the "tissue cylinder." **All** vascular tissues, soft as well as hard, involve a relationship which states, simply, that there is a cylinder of tissue supplied by blood vessels centrally positioned within that

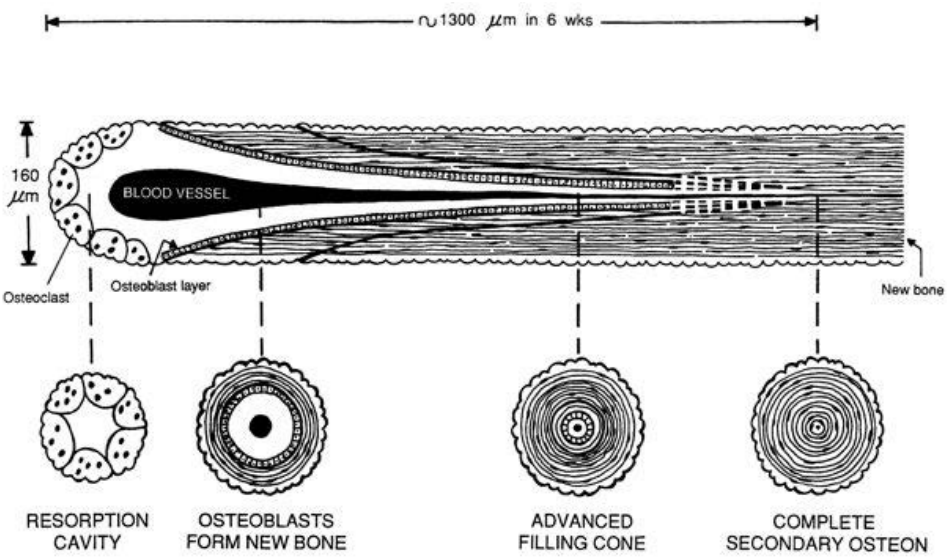


FIGURE 16-19.
Schematic diagram of a cutting and filling cone. (Adapted from Roberts. W. E., P. K. Turley, N. Brezniak, and P. J. Fielder: Bone physiology and metabolism. Calif. Dent. Assoc. J., 15(10):54-61, 1987, with permission.)

cylinder. The radius of the cylinder is the extent to which physiologic transfers can occur from the center to and from the cells resident within the cylinder. In bone, this universal tissue relationship is quite graphically demonstrated, since the secondary osteon is, itself, a functional tissue cylinder.

In conjunction with muscle migrations and continuous reattachments along a growing bone's surface (see above), a limited distribution of **haversian systems** is formed in some areas of muscle attachment on resorptive or remodeling surfaces of a bone in the child (Fig. 16-20), but otherwise, secondary osteons are not a principal feature of the young skeleton. Most haversian systems develop and accumulate much later in life and are concerned with the secondary reconstruction of the original primary cortical bone.

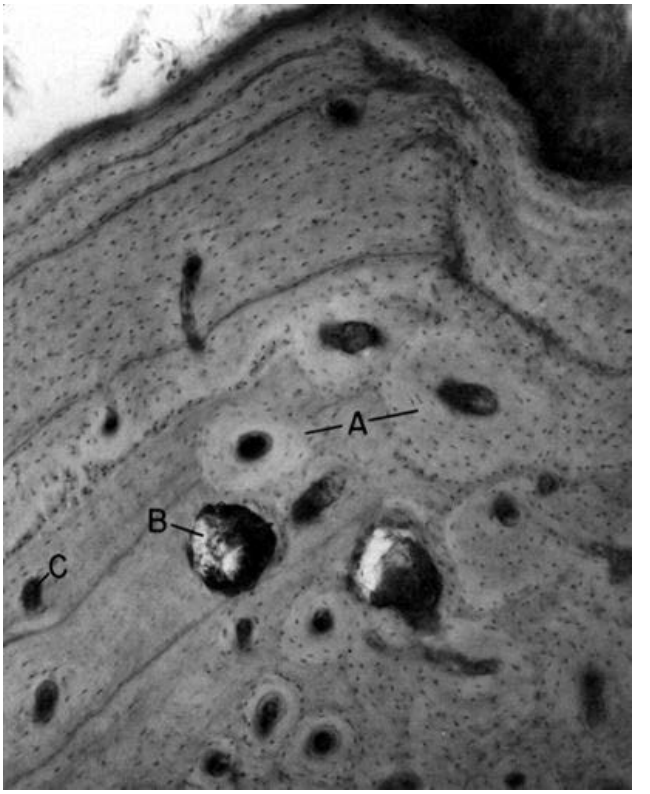


FIGURE 16-20.
These secondary osteons are located in a tuberosity that is undegoing a remodeling movement. The shifting of the attached muscle involves the continued formation of haversian systems. A primary vascular canal (C) is enlarged into a resorption canal (B), and concentric lamellae subsequently deposited within the resorption spaces result in fully formed secondary osteons (A). (From Enlow, D. H.: Functions of the haversian system. Am. J. Anat., 110:269, 1962, with permission.)

Lamellar bone is a slower growing type found throughout most parts of the adult's skeleton and slower forming areas of the child's skeleton. Depending on location, they may be termed periosteal circumferential, endosteal circumferential, cortical, coarse-cancellous, or haversian lamellae. **Primary osteons** (in contrast to the secondary osteon) are relative small structures in which concentric lamellae are laid down in the fine-cancellous spaces of non-lamellar bone, as seen in Figure 16-14.

Part 2

More Essentials of Facial Growth

15

Adult Craniofacial Growth

Rolf G. Behrents, D.D.S., M.S., Ph.D.

The changing face of the human clearly reflects time. From the initiating morphogenesis of prenatal development, through the growth years of infancy, childhood and adolescence, into the mature period of adulthood, and then continuing on into old age, the altering contours characterizing the aging process of human facial form are consistently demonstrated without exception. Many studies have been conducted to describe and analyze the external changes of the face, and more studies have been conducted to characterize the underlying osseous changes that provide the template for the surface alterations.

Fascination with this continuing example of biologic alteration has traditionally focused on the periods of life when the most rapid and obvious changes were taking place. This is practical and logical, for the information gained is deemed important in understanding the growth process and effecting treatment.

Based on many years of study most of the contemporary textbooks describing the growth process suggest that postnatal growth peaks in mid adolescence and slows dramatically in late adolescence, and that no growth occurs in adulthood. Common dates of cessation revolve around 14 years of age in females and 16 in males. This is considered the norm, but some variation is acknowledged, such that an occasional "late maturer" is a recognized phenomenon.

As a result of these deeply rooted notions about growth cessation, the adult craniofacial skeleton is viewed as a stable, static entity in terms of size and shape change; and later, in life, degenerative changes are often described in characterizing old age. Facial wrinkles, sagging tissues, resorbed alveolar bone, loss of teeth and decreased vertical dimensions of the lower third of the face are often used to caricature old age.

Conclusions drawn from this information, while seemingly logical and satisfactory to the casual observer, however, are not founded on actual adult data but, rather, are based on extrapolations of the adolescent growth pattern. Simply stated, growth is assumed to cease in adulthood because it slows in adolescence. Thus, our common understanding of growth termination is based on incomplete information. Also when the literature is scrutinized, little evidence is found to verify that progressive facial changes noted in adulthood have no underlying skeletal basis. Furthermore, and more important, the literature provides no substantiation for the belief that biologic alteration is impossible and that growth ceases in the

adult years. The purpose of this chapter is to convince the reader that the concept of a termination of craniofacial growth in adolescence is an erroneous belief.

BIOLOGIC PROCESSES IN ADULTHOOD

Contrary to what might be thought in some disciplines, biologic processes are not dormant during adulthood. Substantial literature is available suggesting that biologic activity occurs or, rather, continues into and during adulthood and that these processes have a direct bearing on alteration of the craniofacial skeleton. Although all tissues are subject to change during adulthood, the present discussion will focus on some of the morphologic changes that occur in bone. A more complete picture of the manifest changes that occur during aging at the cell, tissues, and organ levels may be seen in the works of Andrew (1971), Finch and Hayflick (1977), Sinclair (1978), Kohn (1978), and others.

CHANGES IN BONE

Although in the earliest concepts bone was regarded as static in nature during adulthood, it is now believed that various dynamic changes occur throughout life. Bone, with regard to age, continues to alter qualitatively and quantitatively. In addition to changes in cellular morphologic features, in old age there is a decrease in bone water content, an increase in apatite crystal size, an increase in the volume but a decrease in the weight of the skeleton, a decrease in bone mass, and a decrease in physical density.

With regard to bone turnover, bone remodeling appears to be slower, but not absent, with age. The literature describes a high bone turnover rate in childhood, deposition of new bone being more prevalent than resorption. In young adult bone, the turnover rate is slower and markedly less bone formation and deposition occurs, but nonetheless both processes still continue. In old bone the delicate balance between deposition and resorption is found to be disturbed, with a gradual increase in resorptive activity, particularly on endosteal bone surfaces. However, little evidence of substantial bone formation changes is evident (i.e., little evidence of a reduction in bone formation with age). This accounts for the more-than-occasional report of active bone formation (even involving the facial bones) and even *increased bone formation* during and after middle age in spite of the fact that more resorptive activity is also apparently taking place.

Although this appears to be the general human condition, considerable individual variation also appears to be the rule. Depending on the age and the bone studied, new lamellar bone may be found in some specimens well into the ninth decade of life, whereas in other specimens such as activity is not easily discernible beyond adolescence. In terms of localization, Enlow (1982) points out that there may be some areas of primary (nonhaversian) periosteal or endosteal bone encountered in old bone, and that these deposits may represent remodeling alterations in response to morphologic skeletal changes associated with changes in weight, posture, loss of teeth, and other factors.

Thus, it appears logical to assume that some degree of bone turnover

continues well into adulthood and, further, that the phenomenon of appositional growth and functional adaptation may well continue into senility. Such situations, if general for man, would be expected to result in alterations of bones throughout aging that are measurable with common analytic tools. This is indeed the case.

In addition to the continuing replacement of cancellous and cortical bone evident on the microscopic level, gross changes are also noted with age. Enlow (1982) points to a change (decrease) in the width of the cortex in advanced age. This is noted in the literature with respect to many of the tubular bones of the appendicular skeleton such as the rib, metacarpal, humerus, and femur. Some trabecular bone loss is also noted, especially in women. As a result, there is an increase in the various canals and cancellous spaces of the aged and cortical "thinning," a condition often termed osteoporosis. A decrease in cortical size has also been implicated in the increased incidence of "spontaneous" fractures noted in the aged. However, while the width of the cortex is decreasing, the external dimensions of the bone are increasing.

This aspect of bone development or bone redistribution involving gross bone changes on both the periosteal and endosteal surfaces has been extensively studied by Garn (1970) and others. Their studies have demonstrated that up through the fourth decade, continuing bone gain in both sexes is accomplished by both subperiosteal and endosteal deposition, resulting in an increase in cortical thickness and a maximum bone mass between 30 and 40 years of age. Beyond that age, subperiosteal apposition continues throughout life, resulting in increased external dimensions (more for women) and, because of endosteal resorption, an expansion of the medullary cavity (Fig. 15-1). These two effects result in a decrease in cortical thickness with advanced age. This phenomenon is apparently a general human condition and occurs regardless of genetic, socioeconomic, nutritional, or environmental factors.

Because such bone "expansion" has been shown to occur in a variety of situations, including the bones of the hand and arm, leg and foot, and vertebral

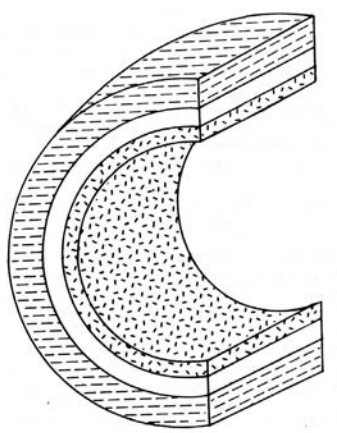


FIGURE 15-1.
Schematic drawing of a tubular bone. Bone is deposited subperiosteally (parallel dashes) and endosteally (random dashes) until 30-40 years of age. Thereafter, bone is deposited subperiosteally and resorbed endosteally. External bone diameters increase during this process.

column, it might be expect that complex physical assessments that collectively evaluate many bones would show increases during adulthood even though the changes noted per bone were small. This has been found to be the case in several important situations. For example, reports of stature assessment are often quite varied in their findings; with growth cessation (maximum height) reported to occur at anywhere from 17 to 45 years of age (which is long after epiphyseal union). Given that height is a gross measure of all the tissue alterations that occur from the top of the head to the bottoms of the feet, and given that increases in dimensions have been reported for the bones in between, it is not surprising that a large variation in age of attained maximum height has been reported. With he bones growing and with (some) tissues being variously compressed, age of maximum height attainment becomes more a varied individual attainment than a specific human event.

Another example of complex osseous change involves the bones of the hand. It has been shown that during adulthood some of the bones gain in dimension while others show a diminution in size (Aksharanugraha, Harris, and Behrents: 1987; Behrents and Harris: 1987). The phalanges gain in length while the metacarpals lose. However, on the whole, the overall dimensions of each finger (ray) remain approximately the same, with increases canceled by the negative changes. This differential situation, once unrecognized, is another example of the complex osseous changes that may occur during adulthood.

ADULT CRANIOFACIAL GROWTH

More to the point, alterations of the craniofacial skeleton have been reported during adulthood with consistency. As might be predicted, studies conducted before the routine use of roentgenographic cephalometrics techniques were only successful in *suggesting* that alteration of the craniofacial skeleton continued into adulthood. Early studies often used cross-sectional material – measurements were performed on dry skulls by the use of craniometric techniques or the external surfaces of living individuals with various anthropometric assessments – or depended on casual observation. As a result, group means usual pointed inconclusively to differences between younger and older groups of individuals. However, several important observations were made. For example, results of early studies suggested that during adulthood the cranium became thicker and the depth, width, and height of the face became greater by several millimeters. Similar alterations of soft tissue structures were also suggested: nose height and breadth increased significantly into later adulthood, as did ear and lip height. On the other hand, contrary views were prevalent questioning whether changes occurred in the craniofacial skeleton at all. This conflict was understandable because sample limitations, inadequate study designs, and uncontrolled variables (such as the lost of teeth) easily confounded findings and fueled differences of opinion.

With the advent of the cephalometer, precisely controlled longitudinal studies became possible on living, growing individuals. Several decades of investigation using this approach have shown quite conclusively that the craniofacial skeleton continues to "grow" during adulthood. In general, the suggestions pointed to using

the cross-sectional approach were corroborated, extended, and detailed with the longitudinal approach and the cephalometrics technique. Credit should be given to Buchi (1950), Thompson and Kendrick (1964), Carlsson and Persson (1967), Tallgren (1974), Israel (1968; 1973a; 1973b; 1977), Forsberg (1979), Susanne (1978), Sarnas and Solow (1980), and Lewis and Roche (1988) for their contributions in documenting adult craniofacial change. More recently, more studies have provided more details regarding adult craniofacial growth (Bishara, Teder, and Jackobsen: 1994; Cretot: 1997; Formby, Nanda, and Currier: 1994; Forsberg, Eliasson, and Westergren: 1991; Love, Murray, and Mamandras: 1990, Noverraz and Van der Linden: 1991).

A major contribution was provided by Israel in a series of studies. With lateral skull x-rays and cephalograms of adult men and women, his findings were clear. All of the craniofacial measures used demonstrated size increases. Dimensional increases were noted for cranial thickness, upper facial height, sinus size, and other aspects of the craniofacial skeleton. He concluded that his results infer a virtual symmetrical magnification enlargement process that generally amounts to approximately a 4 to 5 per cent increase in size. Regional assessments demonstrated differential change. In this regard, he suggested that the upper face increased by 6 per cent, the frontal sinuses by 9 to 14 per cent, and the mandible by 5 to 7 per cent. Although criticized on technical grounds, the works of Israel clearly demonstrate that an enlargement of the craniofacial skeleton occurs, but the amount of change was not as clear.

The investigators who followed Israel have to a great extent validated his findings. However, because of the difficulties attendant to the conduct of longitudinal investigations, many adult studies conducted have suffered in application because of difficulties involving the short age spans studied, abnormal status of the dentition, small samples, and technical limitations. For example, numerous papers describing craniofacial change in the adult are based on longitudinal observations made on fewer than 100 individuals.

A study by the author (Behrents, 1985; 1986) however, appears to have largely overcome the previous limitations and perhaps provides new insight into the nature of the specific morphologic changes within the craniofacial complex associated with adulthood. The sample was drawn from those individuals who had previously participated in the Bolton Study in Cleveland, Ohio. The Bolton Study was an extensive longitudinal investigation initiated in the 1930s and 1940s and was conducted to document and describe craniofacial changes that occurred in healthy, normal children and adolescents. The most unusual feature of the Bolton Study was a new technique developed by the director of the study, Dr. B. Holly Broadbent, Sr.: roentgenographic cephalometrics. Fortunately, the original Bolton Study continued for several decades, and many of the participants were studied continuously until they reached young adulthood. Behrents then recalled, in the early 1980s, 113 of the original participants for new data collection. Together with existing records in the Bolton Study Collection, 163 cases spanning the ages 17 to 83 years were used for determining the nature and extent of the craniofacial changes that occurred between young adulthood and later adulthood. Upon recall, the participants were in good health, and most had a full complement of teeth.

The facial changes noted by direct observation during the recall examinations revealed changes consistent with expectations. Typically, faces appeared somewhat larger, with increased size noted, especially for the nose and the ears. Size increases were apparent for ear length, ear breadth, and thickening of the lobe. The nose appeared to be broader and longer and had a more down-turned tip. The lower third of the face showed evidence of increased dimensions, although lip prominence had lessened (Fig. 15-2).

Consistent with the visual change, radiographic examinations revealed that continuing bone alteration was the norm. This affected not only external osseous architecture but also internal architecture. Sinus size and shape alterations were a consistent finding when the frontal, sphenoidal, and maxillary sinus cavities were assessed by comparing x-rays from young adulthood with those collected during older adulthood (Fig. 15-3). Obliteration of the radiographic images of the sutures, thinning of the parietal bones, and calcification of the falx cerebri were also seen.

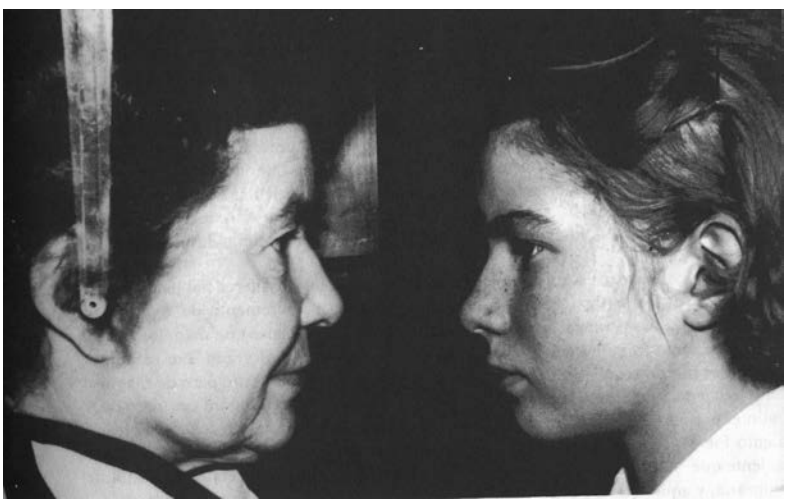


FIGURE 15-2.
Facial changes in a female from age 14 to age 60 (from Behrents, 1985).

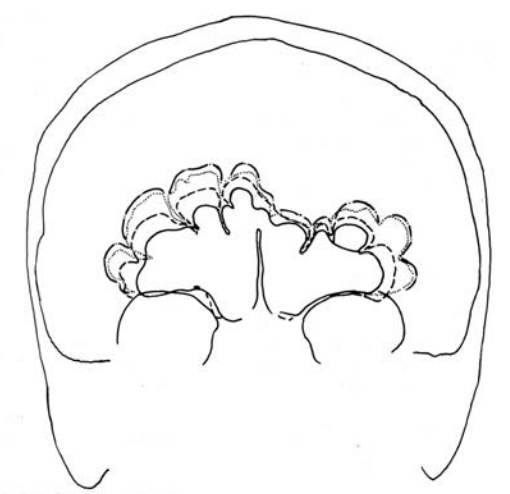


FIGURE 15-3.
An example of enlargement of the frontal sinus during adulthood. Solid line indicates the sinus outline at age 17 with the dashed, dotted, and dash-dot lines indicating the change in the sinus outline occurring over progressively older ages (21, 36, and 52 years of age) (from Behrents, 1985).

Extensive cephalometrics data, adjusted for magnification, revealed continuing growth of the craniofacial complex at all age levels. For both male and female subject, most of the distance measures and some of the angular values demonstrated significant change (Fig. 15-4 and 15-5). Presence of growth during adulthood was the norm; it was not unusual for 95 per cent of the sample to show a dimensional increase for a particular measure during adulthood. It is thus clear that the designation of "late maturer" is really a misnomer, for continuing maturation of the craniofacial skeleton during adulthood is the norm.

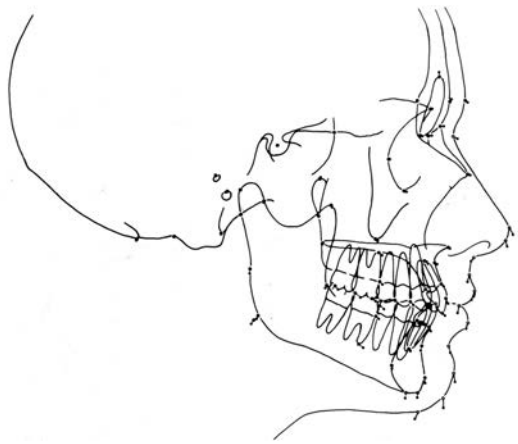


FIGURE 15-4.
Mean change in the female during adulthood for skeletal, dental, and soft tissue landmarks. The open circles indicate the mean location of the landmarks in young adulthood while the solid circles denote the mean landmark locations in older adulthood. The background tracing is based on young adulthood anatomy (from Behrents, 1985).

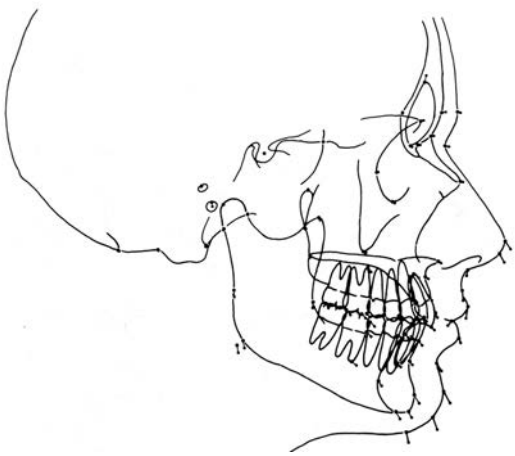


FIGURE 15-5.
Mean landmark change in the male during adulthood. Constructed as in figure 4, the mean amount and direction of change of each landmark can be visualized (from Behrents, 1985).

Overall, both size and differential shape changes were noted for various areas of the face (Figs. 15-6 to 15-9). A 2 to 10 per cent increase was the rule, the bones of the cranial base altering least; the facial bones a moderate amount, the frontal sinus more, and the soft tissues most. The changes seen were similar to typical adolescent alterations but of a lesser magnitude and rate. The typical direction of growth varied to a certain extent, depending on the period of adulthood studied. In young adulthood the direction of growth was specific to an individual's growth pattern. In other words, a "horizontal grower" grew horizontally in young adulthood, and a "vertical grower" grew vertically. Later in adulthood, however, vertical dimensional change appears to predominate. Soft tissue changes were more dramatic than skeletal change but remained related and patterned by the skeletal alteration.

FIGURE 15-6.
Superimposed tracing based on registration on Sella and orientation along Sella-Nasion. Male at age 28 (dashed line) and age 79 (solid line).

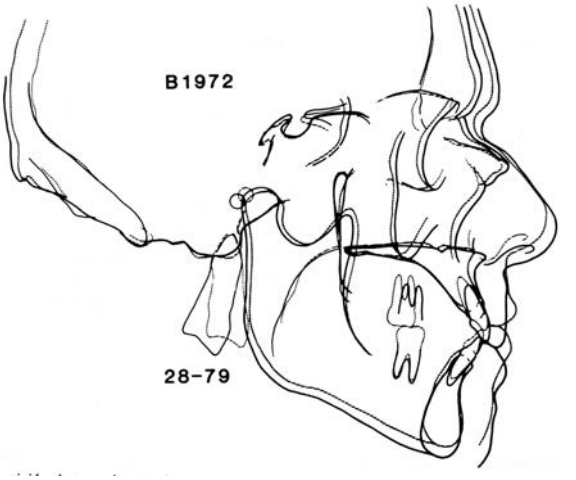


FIGURE 15-7.
Superimposition of a male at ages 23 and 67. This individual had also undergone a rhinoplasty in the intervening years.

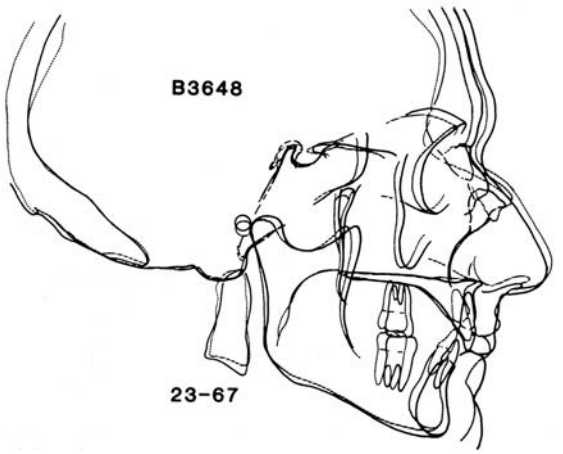


FIGURE 15-8.
Superimposition of a female at ages 17 and 58. Considerable vertical change is seen.

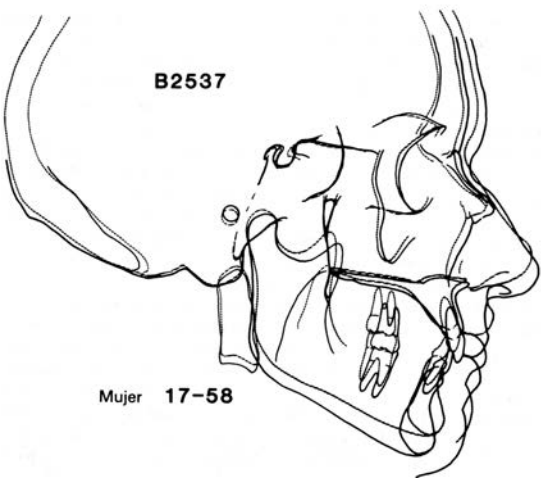
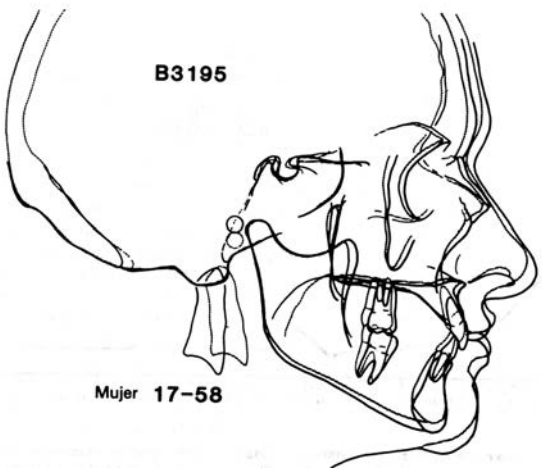


FIGURE 15-9.
Superimposition of a female at ages 17 and 58 (from Behrents, 1989).



Definite differences in the nature and extent of some changes were found when comparing males and females. Typically, females were smaller than males at comparable ages in young adulthood, and during adulthood females grew less; males were generally 5 to 9 per cent larger. Although the anterior length of the face was increasing in both sexes, vertical change was more characteristic of the female. It appears that this lengthening of the face is occurring by two different means, depending on sex. A forward rotation of the mandible was seen in the male, and a converse rotation in females was common. Regardless of the rotation, vertical dimensions of the face increased with time. Beyond this, dimorphic features characterizing the sexes were especially prominent in the upper face. Differences were noted in the orbital region, the orbit being more upright in the female; the area of glabella, the male glabellar area being more robust; and the nose, with the male nose being larger and longer.

The data further suggest that females undergo a generalized growth deceleration in their teens, but with a reacceleration later (Fig. 15-10). This cyclic process may be related to the fact that most of the females in their 20s and 30s were bearing children. It has been demonstrated that pregnancy does affect bone turnover. During pregnancy periosteal bone formation rates are elevated; and as a result, the cross-sectional area of the medullary cavity and the endosteal and periosteal perimeters increase. It is thus quite conceivable that the external dimensions of the facial bones may be affected during the process. Males, on the other hand, show a very regular, gradually decelerating pattern of growth over the adult years (Fig. 15-11).

Regional considerations demonstrated that little significant change occurs in the cranial base region except at its extreme extensions. The occipital condylar area tends to be displaced downward and forward with time; and as might be expected, the area around nasion tends to develop in an anterior direction. The endocranial surface of the frontal bone appears to be fairly stable; however, the ectocranial surface of the outer table continues to develop significantly through

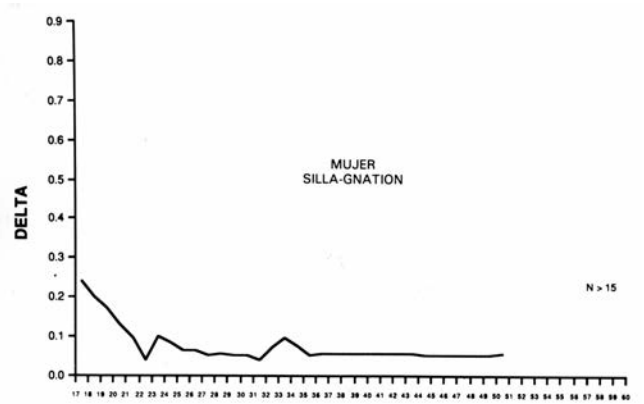


FIGURE 15-10.
Growth curve for the distance measure Sella-Gnathion in the female based on partitioned growth increments (from Behrents, 1989).

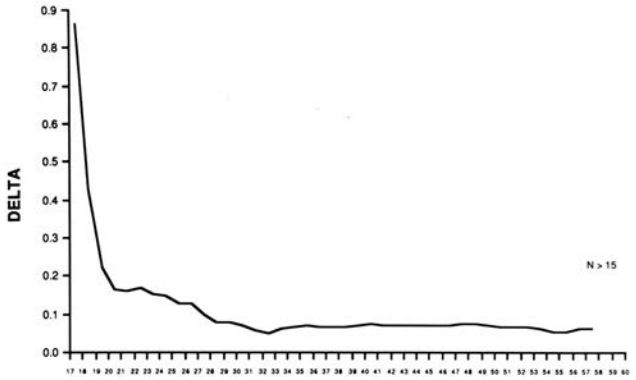


FIGURE 15-11.
Growth curve for Sella-Gnathion in the male (from Behrents, 1989).

time (9 per cent enlargement). As might be expected, the frontal sinus increases substantially in size. Positionally, the upper and lower extremes of the sinus move forward and apart during adulthood. As a result of all these changes, the usual measurements designed to measure the length of the cranial base indicate that a small but significant increase occurs over time. Furthermore, the area including the external aspects of porion tended to shift downward relative to the remainder of the cranial base. This might well affect measures using Frankfort horizontal reference schemes. Anatomic porion, on the other hand, remains quite stable with regard to other cranial base structures.

In the midface, differing amounts of activity occur when the anterior and posterior aspect are studied. In the posterior region activity is slight, with only a few subtle changes noted; little substantive change is seen in the region of the pterygomaxillary fissure. However, definite change occurs in the palatal region. Apparently, the palatal structures continue to relocate posteriorly and inferiorly. Some differences were noted with regard to sexual dimorphism; males showed a greater and more inferior development of this region than females. This latter change is consistent with a general observation characteristic of the male where a consistent posterior counterclockwise development and rotation of structures is seen (Fig. 15-12).

The anterior aspects of the midface showed consistent change in both sexes. Most development was expressed anteriorly, even though vertical displacement was also apparent. As mentioned above, the nasal region continued to develop anteriorly. Thus, the position of nasion and the tip of the nasal bone were located in an anterior position with time. In the female there was a tendency for the tip of the nasal bone to elevate. Likewise, the superior, lateral, and inferior aspects of the orbit moved consistently forward with time. Such a change increases the size of the orbital cavity. Such remodeling and displacement, of course, also involves the zygomatic processes. The anterior aspects of the palate move forward, but also substantially downward during adulthood. The alveolus also increases in size, vertically. Apparently, in the absence of periodontal disease continued development of the alveolus is possible.

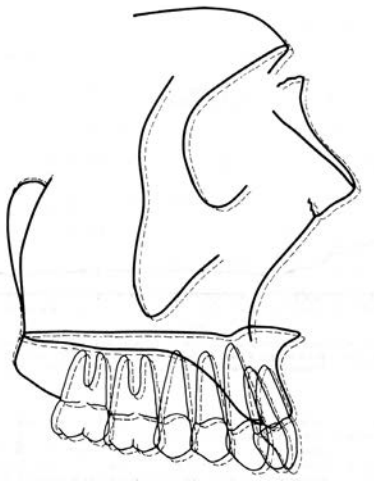


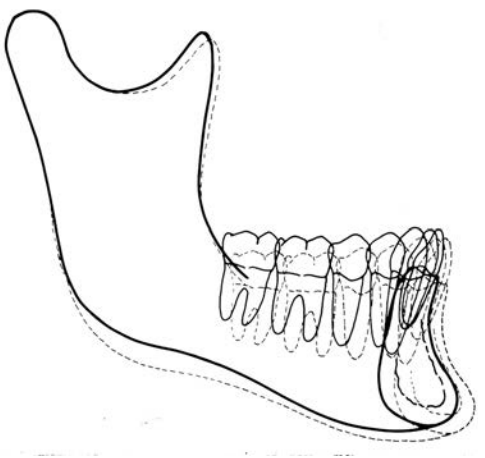
FIGURE 15-12.
Schematic diagram indicating the generalized changes occuring in the midface (Sella/Sella-Nasion orientation). Slight differences are noted between males and females but are not represented here (from Behrents, 1985).

It might be expected that the changes seen in the cranial base and midface have an additive effect on the mandible such that considerable change in size and position should occur (Figs. 15-13 and 15-14). This is the case. The chin continues to be displaced in an anterior direction during all ages, but much more of this activity is seen in the male. In the female the mandible comes forward, but not to the extent seen in the upper and lower anterior regions of the midface. Thus there is a tendency for the female mandible to appear more retruded with age, even though the chin is coming forward. Significant vertical translation of the chin also occurs in both sexes. Thus, the anterior facial dimensions consistently increase. The mean increase for total face height (nasion to menton) was 2.8 mm during adulthood, but in individual cases change on the order of 10 mm (1 cm!) did occur. Compared with changes seen in the upper face, the amount of lower facial change was double (0.9mm, compared with 1.9mm).

Of separate, but related interest is the rotation of the mandible. Although this rotation is not extensive, it is apparent the mandible continues to rotate in a counterclockwise fashion in the male and in a clockwise direction in the female. Although both movements are subtle, they tend to effect an elongation of the face, although by differing means. Growth also affects the position of gonion, with that structure being relocated inferiorly and anteriorly in the male and inferiorly and posteriorly in the female. More general posterior development of the mandible is seen in the male consistent with what is occurring in the midface.

Although it might be suggested that many of the changes noted for the mandible are due to growth effects produced elsewhere in the craniofacial skeleton, it is clear that the mandible itself is growing (Figs. 15-15 and 15-16). Distances intended to describe the growth of the regions of the mandible show that the overall length of the mandible, the body, and ramus, and the alveolar regions are all increasing in size. Furthermore, in the male, the angle between the body and the ramus becomes more acute by a small amount with age. The anterior border of

FIGURE 15-13.
Schematic diagram of the change noted for the female mandible in relation to a Sella/Sella-Nasion orientation. Vertical and clockwise rotations are seen (from Behrents, 1985).



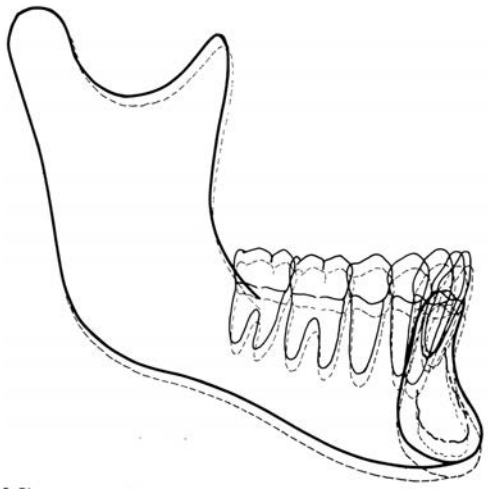
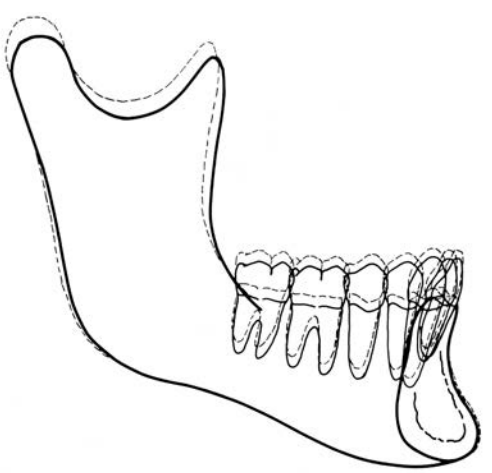


FIGURE 15-14.
Schematic diagram of the change noted for the male mandible in relation to a Sella/Sella-Nasion orientation. Counterclockwise and anterior movement is characteristic of the male (from Behrents, 1985).

FIGURE 15-15.
Schematic diagram generalizing the size and shape change seen in the female mandible during adulthood. According to this regional superimposition there is an apparent increase in the height of the alveolar process. A slight amount of resorption (anterior) and deposition (posterior) is seen in the ramus (from Behrents, 1985).



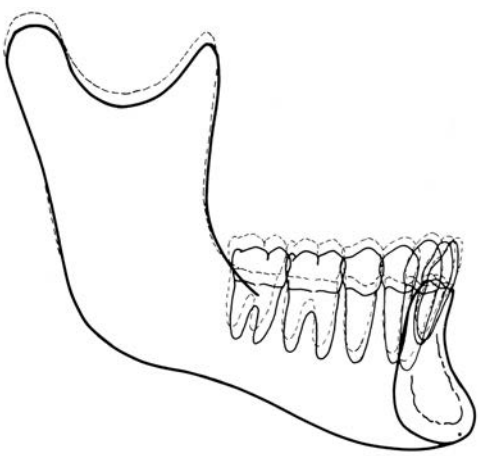


FIGURE 15-16.
Schematic diagram generalizing the size and shape change seen in the male mandible during adulthood. Compared to the female, similar and greater changes are noted in the male (from Behrents, 1985).

the ramus continues to relocate posteriorly with time. This suggests that resorption of the anterior border of the ramus continues in adulthood much like that seen in adolescence. Such activity during adulthood might have some effect on the ability of the third molars to erupt later in life. The posterior border of the ramus appears to be stationary in the female and moving anteriorly in the male. These two effects produce a decrease in the width of the ramus with time.

As might be expect, the dentition reacts to the osseous changes that occur during adulthood (Figs. 15-17 and 15-18). Consistently, for both sexes, the maxillary anterior teeth become more vertically upright during adulthood. The lower anterior teeth, however, appear quite stable in their orientation, with only a tendency for proclination in the female. The posterior teeth apparently change their inclination in response to the altered position of the mandible. The axis of the molars shows a significant uprighting in the male and a tendency for being more distally inclined in the female. The lower molar movements complement those of the upper molars: in the male the molar becomes more upright, and in the female there is a tendency for a more mesial inclination. By virtue of these changes, it might be expected that the dentition would appear less prominent in the older adult. With regard to the relationship of the teeth to the bony profile, this is not the case. However, with

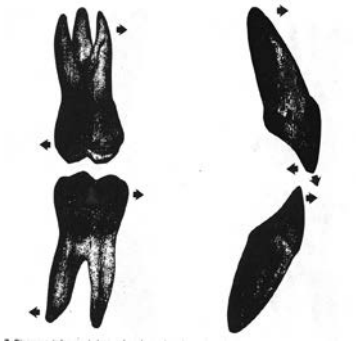


FIGURE 15-17.
Diagram of the tooth movements seen in the female during adulthood. Uprighting of the upper anterior teeth is consistently seen and there is a tendency for the lower anterior teeth to tip forward (after Behrents, 1985).

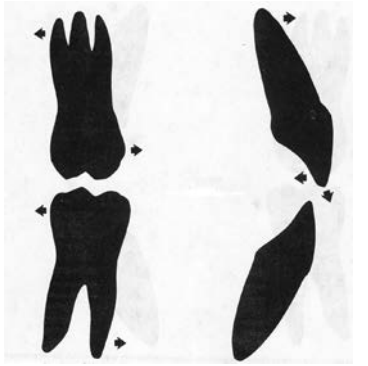


FIGURE 15-18.
Diagram of the tooth movements seen in the male during adulthood. Uprighting of the upper anterior teeth is also consistently seen (after Behrents, 1985).

regard to the relationship of the teeth to the soft tissue profile, the teeth do appear less prominent. Curiously, overbite did not increase with time. However, when it was realized that attrition was commonplace, it is reasonable to surmise that overbite did increase but it was decreased by incisal wear.

The soft tissue mask surrounding the osseous structures undergoes notable alterations with age. The changes are more extensive than those seen within and among the osseous structures, but still the soft tissue changes remain related to the underlying osseous change. Anterior movement of the soft tissues over the glabella, the nasal region, the midface, and the chin are seen. Likewise, the soft tissues also reflect vertical osseous changes as seen over the nasal region, the midface and the chin. But some differences are noted. The nose grows a great deal in size, and the tip becomes more angled and downturned. The height of the upper lip follows this same developmental course and lengthens to the same extent that the nose grows. Furthermore, because of the dental changes together with the growth of the nose and the anterior movement of the chin, the teeth appear less prominent, the lip area flattened, and the lips located more inferiorly, almost completely covering the upper incisors. Thus, overall, with age there is a straightening and elongation of the profile. Such findings mirror those found in other studies.

In addition to the healthy, dentate, orthodontically untreated sample, small subsets of individuals who had undergone various treatments (orthodontics, multiple extractions, rhinoplasty) were also studied. In these subjects continuing adult growth was demonstrated, but the nature and amount of the alteration were different from those of their untreated counterparts. In general, in young adulthood the group of individuals who had previously received orthodontic treatment were indeed different in craniofacial configuration when compared with untreated individuals. During adulthood they also grew poorly, as they had in the past. For example, persons with a retruded mandible retained this characteristic during adulthood, and in some instances it worsened. Persons who had experienced the lost of many teeth also grew in adulthood, but their growth was again different from that of either the treated or untreated individuals. Typically, these cases grew less in adulthood; this was particularly evident in the anterior portions of the face. Such cases also demonstrated loss of vertical dimension with time; almost all of the persons in the untreated sample (most of whom had complete dentition) showed the opposite effect. The loss of teeth obviously alters the configuration of the adult craniofacial skeleton.

EXPLANATORY MECHANISMS

Given that continued craniofacial growth is a demonstrated reality, it might be helpful to understand which mechanisms active in adolescence are also present in adulthood. Unfortunately, in terms of mechanisms that can serve to account for the change, we are pressed by the available literature to conclude that few mechanisms remain after late adolescence. For example, it is difficult to imagine that the translation mechanisms conceptualized by Moss and Scott are active during adulthood (with regard to the nasal septum, brain growth, orbit, etc.), and little literature really exists on the topic. In adulthood, the brain apparently becomes

smaller; the septal cartilage may continue to grow until the 30s but mainly at its anterior free end. Further, although it has been shown that a condylar contribution to mandibular growth can occur into the early 20s, weak evidence (acromegaly) is present to suggest that the condylar cartilage retains the potential for substantial change during later adulthood. Although the major translatory mechanisms supposedly active during adolescence do not appear to be plausible with regard to adult growth, some other mechanisms present during adolescence may continue into the adult period. For example, considerable literature is available with regard to the patency of sutures during adulthood.

Estimates of suture closure vary widely depending on the suture studied, the manner of study, and the specimens under review. Some studies suggest that the sutures of the cranium close in later adolescence; and those of the face, soon after. However, there is no clear consensus on these issues, and some investigators suggest that cranial and facial sutures remain patent past age 18 and clearly into the 20s and perhaps even into the 30s. Also, there appears to be considerable variation within each region. For example, it has been suggested that the palatine and intermaxillary sutures remain unossified into the 30s and that the frontozygomatic suture remains open until the eighth decade of life. Furthermore, there may be individual variation as well. Therefore, on the basis of the available literature, it is possible that the sutures may in some ways and in some areas participate in the growth activity seen in the adult. Their principal post adolescent contribution, however, occurs early in adulthood.

Remodeling of the facial bones to effect the growth changes seen in adulthood, however, seems a rationale approach to the explanation of adult alteration. Although the amounts and rates of change differ for adolescence and adulthood, remodeling continues throughout life and is the most plausible explanatory mechanism relating to bone surface activity. However, one might question why such activity would be *expected* to result in a change in morphology. In this regard, Enlow (1986) believes that once basic adult form is obtained, unless there is an intrinsic environmental alteration (change in function, change in biomechanical circumstance, and loss of teeth) gross (not histologic) remodeling is basically static in effect. Environmental changes, however, are quite likely—indeed, certain. Therefore, a morphologic change is logical, and remodeling seems the probable explanation.

SUMMARY

What is evident from this discourse is that growth is apparently operative at wider age spans than previously thought, and there may be no cessation of growth at all. Recent reports (Lewis and Roche, 1988) support the view that active growth of the craniofacial complex continues into adulthood, but perhaps only into the 30s. Whether or not growth ceases in middle age or continues is probably and unanswerable question, given the sensitivity of the available recording techniques. Regardless, that adults grow has been confirmed; growth of the craniofacial complex does not cease in adolescence. The amount of growth occurring during adulthood is small, especially when the amount per year is quantified. Nonetheless, the cumulative increments of growth over time cause a modest amount of

differential alteration of the craniofacial skeleton. Generally a 2 to 10 per cent enlargement occurs. Characterization of the growth in adulthood suggests that it is an extension of the adolescent change except that in the later ages, a generalized vertical elongation of the facial structures predominates.

CONCLUSIONS AND APPLICATIONS

It is clear that tissues in general and the skeleton in particular are far from quiescent during adulthood and aging. Distinct, sometimes complementary and sometimes contradictory (different from activity during childhood and adolescence) processes are involved. Furthermore, such processes may exert influences that alter a skeletal structure so as to decrease or increase its prominence. Thus, it follows that such change could be noted during anthropometric, craniometric, or cephalometrics study.

On the basis of the findings presented here, it must be recognized that growth and development of the craniofacial skeleton is a continuing, long-term process that apparently has its periods of exuberance and relative quiescence, but the biologic mechanisms that incite or regulate the changes remain intact and never really terminate. We know that bone is continuously remodeled irrespective of physiologic demands and aging, we know that bone responds to injury and manipulation at all ages, and now we know that the process of growth apparently does not terminate as was once thought and that changes in morphology may continue long after puberty-adults grow.

Considerable discussion has resulted, since the aspects of adult change have been documented and accepted, over what to call such change. Arguments have suggested "adaptation," "maturation," something other than "growth." This issue, although mainly semantic, cannot be resolved. The difficulty lies in our definitions of growth, which are imperfect because of our lack of understanding of its mechanisms and its temporal nuances. For now, the adult alteration noted here is indeed "growth," for it is not unique to the adult (although it has some unique features); it is merely an extension of the changes seen during earlier years. The nature of the differences is best described in terms of degree, not in terms of kind.

Many of the technical procedures that have been developed to improve facial form in the adolescent have been based on our understanding of the nature of man, including growth. One would expect, therefore, that the present information about growth during adulthood might come to influence the clinician's diagnostic and treatment planning activities on patients within that category. So, too, might the present knowledge find application and even misapplication in providing a basis for some treatments. Perhaps more important, present knowledge should direct the clinician to understand that osseous change will continue long after supposedly definitive treatments are rendered; rigid stability of the craniofacial skeleton is an untenable concept.

Of greatest significance, however, is acceptance of the concepts that adults retain the capacity to change and may do so. Given that concept, ultimately prediction and control of the processes of growth will become possible.

16

Introduction to the Temporomandibular Joint

J. M. H. Dibbets, D.D.S., Ph.D.

The temporomandibular joint (TMJ or the TM joint) is a bilateral synovial diarthrosis. This learned expression means that we have one freely movable joint on each side, left and right, surrounded by a capsule whose internal lining produces a viscid synovial fluid. The joint permits the mouth to be opened and closed, and the jaw to be protruded, retruded and shifted laterally. During these movements the joint capsule, together with the lateral and the spheno-mandibular ligaments, provides structural stability. The stylomandibular ligament is considered of minor importance in this respect and therefore accessory.

Because the dynamic performance of all joints normally covers the functional demands placed upon them, it is not logical to assign to the TM joint additional structural/functional complexities, as some authors do. Any joint of the body may be regarded as an intricately interrelated functional structure as well as a natural adaptation to environmental needs and constraints.

The TM joint has received much attention in the literature during recent years. What makes this joint so interesting? Large-scale research has revealed that this articulation often generates signs and symptoms that may indicate a dysfunction of the system. Clicking, snapping, crepitation, locking, pain, and instability are reported with high frequency. Numerous, too, are the etiologic factors assigned to dysfunction. Whether or not the resulting therapeutic regimens are oriented to the cause or to the symptom is a question still open to debate. However, from information available at this moment, obtained from conscientiously conducted studies, it appears that simple mechanical explanations for dysfunction, such as occlusal irregularities, do not hold.

In this chapter developmental aspects of the TM joint will be briefly recapitulated. Emphasis is placed on those aspects that are unique for the temporomandibular articulation. Among these are the "secondary" character of the joint and the specific analysis of the processes and mechanisms involved in the growth of the components.

DEVELOPMENTAL ASPECTS

In the sixth week of intrauterine life, a condensation of mesenchyme develops lateral to Meckel's cartilage. The development of this condensation into a lower jaw proceeds rapidly. Within 1 week a complete membranous bony plate, albeit fragile, is formed, paralleling and locally enveloping the bilateral Meckel's cartilaginous rods. At 10 weeks the bony mandible has recognizable form, and Meckel's cartilage starts to be resorbed. This branchiomer cartilage does not contribute to the newly formed mandible. During the same period condylar fields develop at the cranial ends of the mandible. Within 2 weeks the condylar processes are clearly recognizable, and (secondary) cartilage production will have begun. By another 2 weeks, during the fourteenth week, endochondral ossification of this new cartilage will start centrally in the ramus, proceeding upward. From the twentieth week on there occurs an equilibrium between the production of cartilage and subsequent replacement by bone, creating the typical picture of a growing mandibular condyle.

At 10 weeks the lateral pterygoid muscle is already formative, and its two heads may be distinguished. One head attaches to the condyle and one to the formative disk. The disk emerges from a mesenchyme field that develops between the developing condyle, the temporal squama, and Meckel's cartilage. Some authors assume continuity as a single system, extending from the lateral pterygoid muscle to the malleus.

The cartilage of both the condyle and the tubercle consists of cartilage cells and of matrix, which is composed of a network of collagen fibers and hydrophilic proteoglycans, binding water. The collagen fibers mechanically prevent a continuous swelling, resulting from osmotic absorption of water by the proteoglycans, and thus allow pressure to be generated within the network. This pressure counteracts functional loading of the joint. When this loading exceeds the pressure, liquid is expelled into the interstitium. This fluid provides lubrication -"weeping lubrication"- and metabolic support; unloading makes the fluid return into the cartilage matrix.

The temporal component of the joint does not acquire its characteristic sigmoid shape until after birth, as may be seen in Figure 16-1. An 8-month old human fetus still has a straight zygomatic arch. The mandible can slide forward and backward horizontally without being displaced vertically. This situation will change quickly once tooth eruption has started.

At 4 years of age the temporomandibular articulation has achieved many of its adult characteristics. A tubercle has formed, and the condyle process and mandible have clearly progressed beyond the neonatal shape. The external meatus still occupies a low position relative to the condyle, but with further maturation this position will change vertically. (See Figure 16-2)

In the adult situation, as shown in Figure 16-3, the slope of the temporal tubercle is more vertical. The external meatus occupies a vertical position at the same level as the condyle. This change is not due to remodeling of the structures of the middle ear and their bony housing, but can be accounted for by downward remodeling of the temporal part of the joint.

FIGURE 16-1

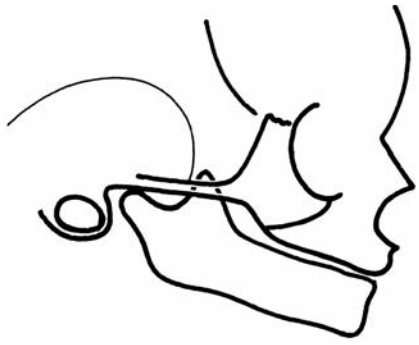


FIGURE 16-2

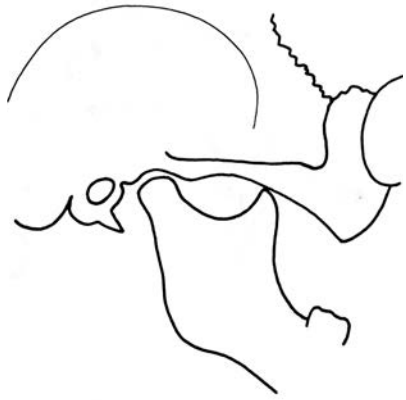
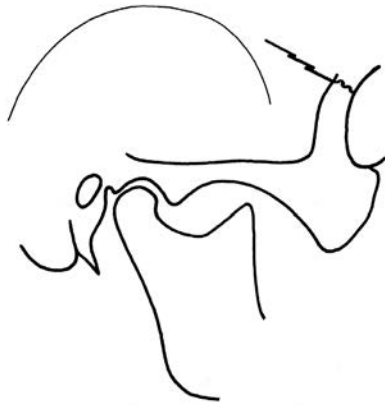


FIGURE 16-3



Between the tubercle and the condyle a connective tissue disk is positioned that divides the joint cavity into an upper and a lower chamber. These cavities are filled with synovial fluid. Centrally the disk is composed of dense avascular tissue, with fibers oriented in a sagittal direction. Above and in front of the condyle are the so-called posterior and anterior bands. These bands are part of a continuous system and may be compared to a stretched ring. This ellipsoid ring, viewed from above, runs superiorly along the long axis of the ovoid condyle. The ellipsoid ring, incorporating both the anterior and posterior bands, is completely integrated within the disk. Dorsally, the posterior band continues into a bilaminar zone.

The upper zone consists of highly elastic tissue, permitting displacement of the disk during opening and closing. The lower zone is much less elastic and assures positional stability between disk and condyle.

Between the two zones, and in particular near the posterior capsule, there is loose connective tissue with a rich blood supply. During forward shift of the condyle these vessels significantly enlarge their cross-section. The articular capsule and ligaments run from the temporal mandibular fossa and tubercle to the neck of the condyle. The capsule and disk are continuous at the dorsal, medial, and lateral aspects. As a result, the disk in reality is a three-dimensional structure, similar to a hat capping the condylar head. The anterior band is attached to the superior head of the lateral pterygoid muscle; the inferior head of the muscle connects with the condyle itself.

SECONDARY JOINT

The vertebrate temporomandibular joint is known as a secondary joint. The adjective "secondary" refers to several separate properties of the joint that are not original; that is, there were "primary" characteristics that have been replaced by "secondary" ones. Let us find out which was first and what came thereafter.

In Chapters 4 and 13 it is explained that a new joint evolved during phylogeny, replacing the original, or primary, articulation. This original joint developed within the branchial arch system at the junction of the primary palate and the first gill arch in primitive fishes. In our phylogenetic history this was the primary mandibular joint. Through many evolutionary stages and continuing time this articulation even at one time-point combined the functions of the jaw joint and hearing apparatus in some Amphibians. In front of this original joint a new, secondary, articulation between the skull and a tooth bearing structure, the dentary bone, came into being. "Secondary," therefore, applies to the joint as a later or secondary development in our phylogenetic history.

During our ontogeny some features of our phylogenetic history are still recognizable. This is e.g. true for the original articulation between Meckel's cartilage (the primary mandible) and the cranium that is replaced during ontogeny by a new joint. This new articulation developed after the other joints had already formed, and the new articulation is indicated as a secondary joint. "Secondary," therefore, applies to the TM-joint because it develops late in our ontogenetic development.

There is a third and again important reason to attach to the TM joint the adjective "secondary." It refers to the "second" appearance of the cartilaginous components of the joint. After all true primary cartilages had formed, then, within the mesenchyme blastema of what will be the future mandible, a new cartilage formation begins as a secondary event in four regions: the condylar process, the coronoid process, the symphysis, and the gonial region. The latter three will have disappeared around birth. Condylar secondary cartilage, however, remains for the rest of our life and is maintained within the membranous components of both the squamous portion of the temporal bone and the condylar process of the mandible. "Secondary," therefore, applies to the new cartilage tissue that only came to exist after the primary cartilages had differentiated.

A fourth reason to attach the label "secondary" to the TM joint may be found in the origin of the cartilaginous tissue. Being late in ontogeny, this new cartilage develops within a mesenchyme blastema, as explained in the previous paragraph. The secondarily induced differentiation of mesenchyme into cartilage will testify its inheritance for the rest of its life cycle by the connective tissue covering. Primary cartilages are covered by a thin perichondrium. Secondary cartilage, in contrast, is covered by a fully developed, however thin, mesenchyme tissue layer. The source from which condylar cartilage is derived is found within this mesenchyme covering. First there are mesenchyme cells, and then these cells differentiate into cartilage as a secondary event. "Secondary," therefore, applies to the late differentiation of the mesenchyme tissue from which the cartilage originates.

A fifth and final reason to attach to the TM joint the adjective "secondary" The mesenchyme covering of the condyle contributes to a fundamental characteristic of secondary condylar cartilage. Primary epiphyseal cartilage reacts during development primarily to overall systemic growth stimuli such as hormones. In contrast to this, condylar cartilage only secondarily follows these overall stimuli after additional modulation by local factors. This is substantiated by numerous laboratory experiments. Condylar cartilage cannot be cultured in vitro as easily as can primary cartilages. "Secondary," therefore, applies to the characteristic secondary response of the condyle during growth.

TISSUE PROLIFERATION

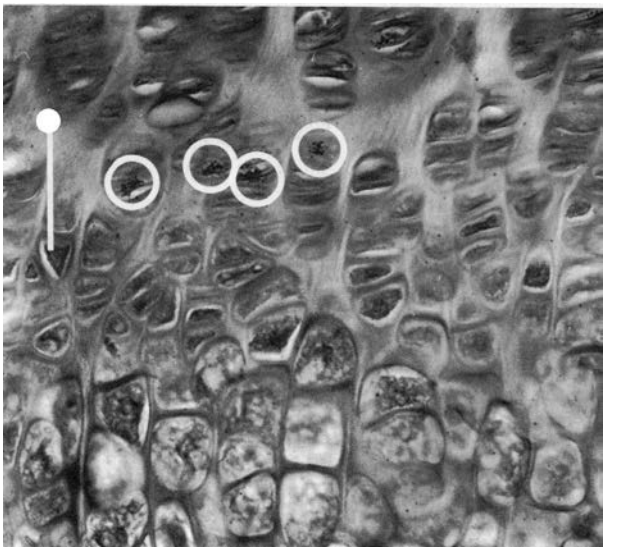
The notion of a mesenchyme-like covering of condylar cartilage is fundamental for understanding the growth mechanism of the condyle. The condylar growth process is different from epiphyseal growth and is a unique feature of secondary cartilage.

Primary cartilage growth is presented in a highly schematized fashion in Figure 16-4. The large left circle represents a cartilage cell within the central layer of a growing epiphyseal plate. The arrow indicates the transition to the subsequent developmental condition, a normal mitosis, which is represented by the slash. As a result of the mitosis two daughter cells will originate, together containing the total amount of organic substance from the original mother cell. Each will inherit half of the duplicated chromosomes, and each cell will initially be smaller than the original cell. The next phase during epiphyseal growth is enlargement of the two daughters, each to full size. At this stage both now mature cells produce and secrete extracellular matrix that will make these cells to drift apart. One cell may remain within the germinative layer and probably be a new mother; the other may drift away and subsequently be eroded and replaced by bone. This very schematic series highlights one of the essential elements of primary cartilage growth: cleavage of previously differentiated mature cartilage cells. The translation of the diagram to reality is facilitated by the autoradiogram in Figure 16-5. The vertical bar equals 50 μ. Autoradiography is a technique of exposing a photographic emulsion to a radioactive source that is incorporated within a tissue. In this case the radioactive labeled nucleotide thymidine was injected into a 49-day-old rat 2 hours before sacrifice. The thymidine incorporated rapidly into cells preparing for division. Its

radioactivity causes black spots in the photographic emulsion. The circles locate their positions. Apparently cell divisions are taking place in the middle part of an epiphyseal plate of the rat tibia. This interstitial mode sharply contrasts with secondary mode of cartilage growth.

FIGURE 16-4

FIGURE 16-5



The next diagram, Figure 16-6, shows a highly schematized cycle of secondary condylar cartilage growth. The left double contour indicates the mesenchyme-like tissue, covering of the mandibular condyle. The large circle in the proximity of the covering membrane represents differentiated condylar cartilage. The arrow indicates the transition to the subsequent developmental condition, the birth of a new cartilage cell. The place of labor is the undifferentiated soft tissue layer, in which one small cell splits itself into two even smaller new cells. This very special event can be seen in Figure 16-7, showing a 49-day-old rat condyle. The technique of autoradiography has been used again, and the vertical bar equals 50 μ. Here, within the soft tissue covering, and surrounded by undifferentiated mesenchyme, mitosis is about to occur: the cell preparing for division is identified by the black spots above the nucleus. The circle locates this happy moment. Let us return to schematic Figure 16-8, second step from left. After mitosis resulted in 2 mesenchyme cells, these cells will come to full size, and of one of them will migrate out of the covering membrane in the direction of the condyle's interior. This is schematized by the black dot directly underneath the soft tissue covering, but still within the

FIGURE 16-6

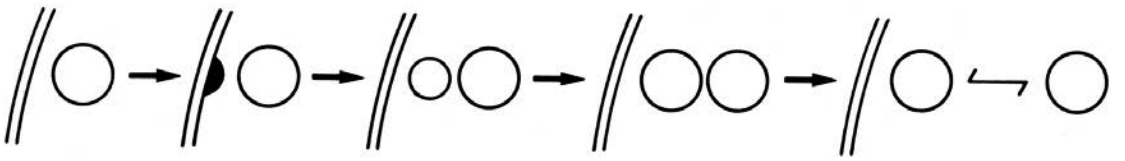
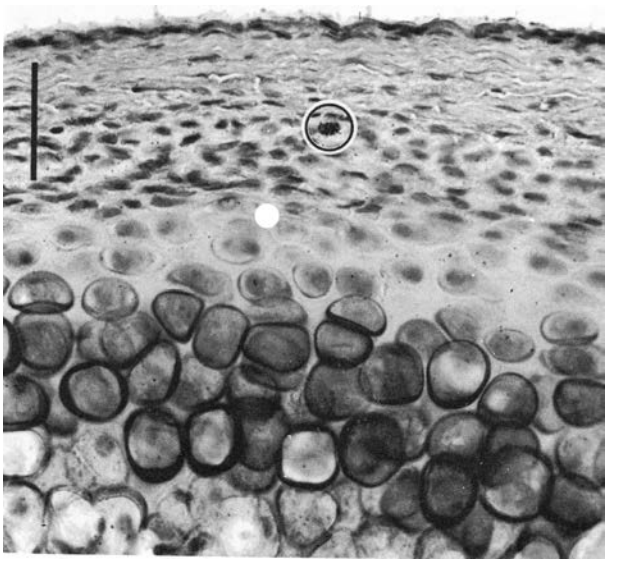


FIGURE 16-7



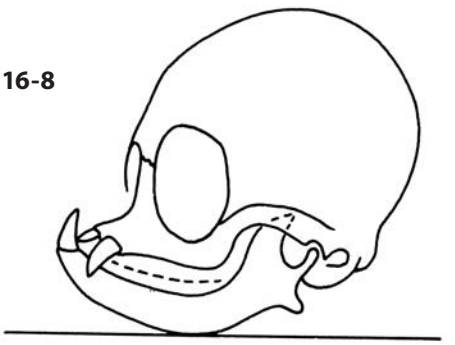
condylar membrane. Here, at this specific moment, a differentiation takes place and the mesenchyme-like cell becomes an immature cartilage cell. This immature cartilage is depicted by the smaller circle positioned between the membrane and the larger, mature cartilage cell in the third step. A new member of the cartilage family has been added without the mitosis of an existing cartilage mother cell, but through mitosis of an undifferentiated mesenchyme cell. The far-reaching consequences of this will be discussed later on. To complete the present sequence we have to note that the new cartilage cell will expand to mature size and start the production of extracellular matrix. The amount of matrix, though, may be small compared to primary growth cartilage. The cells will drift apart, and the process of endochondral bone formation will finish the life cycle. The mode of growth in which new cells are added from the exterior is called appositional growth.

One of the properties of secondary cartilage growth visualized in the diagrams 16-4 and 16-6 is the quantitatively equivalent result, compared with primary cartilage. As can be seen in the schematic figures, there is an equal capacity for producing new cartilage tissue. Both schematic series occur within equal time spans, and both sequences produce equal mitotic rates during growth.

Another, and most vital, property depicted in the diagram is related to the source from which new cells are derived. There exists an inherited dysplasia that prevents mitosis of differentiated cartilage cells, called achondroplasia. Because

interstitial proliferation of cartilage cells within epiphyseal growth plates is inhibited, this dysplasia results in dwarfism. However, since the cartilage in the mandibular condyle originates from the cells of the soft tissue covering, condylar chondrogenesis is not affected. In achondroplasia the mandible has a normal growth tendency. This is evident in some dog breeds, as in bulldogs and the King Charles spaniel (see Fig. 16-8). Here the cartilaginous development of the cranium is inhibited at the synchondroses (a primary, not secondary, cartilage), which leaves the mandible unaffected. The resulting profile is characteristic for the differences between the modes of interstitial cartilage growth and appositional growth. The cranium has become dome-shaped because of a failing response of the synchondroses within the cranial floor to accommodate the expanding brain; the desmocranium, in contrast, reacted quickly and made the skull look like an inflated balloon. The mandible, on the other hand, has developed normally. This once again demonstrates the unique character of secondary, compared with primary cartilage.

FIGURE 16-8



GROWTH OF THE TUBERCLE

At birth the temporal component of the human TM joint is essentially flat or shallow. This early developmental stage of its anatomy thus facilitates horizontal mandibular excursions during breast-feeding. The capacity for horizontal excursions then remains to some extent for the rest of the life. Unlike most other diarthrotic joints, the TM joint has a considerable degree of translational freedom. On opening, the condylar component rotates relative to its temporal counter structure and also translates forward and downward.

Depicted in Figure 16-9 are the postnatal changes of the tubercle as seen on tracings, superimposed along the Frankfort horizontal and registered on the external acoustic meatus. The zygomatic process of temporal bone anterior to the condyle is progressively lowered relative to the posterior part. At birth the surface is practically horizontal, and a slope hardly exists. When the primary teeth are present, permitting the first forceful chewing actions, this slope has become steeper and has already attained more than 40 per cent of its adult inclination. At the time of the first transitional period, when the first molars and front teeth have emerged, the slope has reached 70 per cent of its adult value. When the transition of the premolars starts, 90 per cent of adult angulation is attained. The total change postnatally amounts to about 40 degrees.

A thin layer of secondary cartilage covers the tubercle. This cartilage is derived from cell divisions in the mesenchyme covering, with subsequent differentiation, and thus is analogous to the cartilage of the condyle. In this way the articulating areas of the temporal bone, and consequently of the tubercle, are products of endochondral bone formation. In contrast to this, the region posterior and anterior to it is subject to processes of intramembranous bone formation and remodeling.

FIGURE 16-9

GROWTH OF THE CONDYLE

One of the very important functions of the TM joint postnatally is to provide the amount, direction, and timing of its own regional growth responses in relationship to the ongoing and widespread changes in the surrounding craniofacial regions. It is demonstrated throughout this book that remodeling is capable of maintaining form and proportions while it simultaneously provides changing size. The growing mandible as a whole is dependent for the bulk of its substance on the process of intramembranous bone formation and remodeling. The endochondral contribution of the condyle in the actual amount of new bone tissue produced is, by far, of lesser magnitude.

One aspect of TM joint growth consists of interrelated enlargements of its various components, in addition to the developmental interrelationships of the facial and cranial parts. The condyle enlarges in harmony with the disk and the glenoid fossa as a tubercle undergoes development at the temporal part. These changes involve both intramembranous and endochondral bone formation and continuous reattachments of the connective tissues of the associated ligaments and the capsule. The fossa simultaneously enlarges by means of anterior remodeling relocation and a vertical development of the tubercle. The condyle simultaneously expands by appositional growth. The capsular ligaments and disk also enlarge and grow over the bony surfaces with new attachment locations. With these changes, growth proceeds in a more or less comparable fashion for other joints in the body. They all grow larger, whether knee, ringer, or chewing joints; and they all continue to function as they grow and develop postnatally.

In Figure 16-10 the ramus is seen to remodel and relocate in a posterior direction. It does so by posteriorly directed divisions of the condylar mesenchyme. The whole ramus at the same time remodels and relocates backward by posterior

deposition and anterior resorption. If the ramus were to grow only vertically, on the other hand, without any contribution to widening of the ramus, as illustrated in Figure 16-11, the endochondral mode of bone growth would create a track of new bone one condyle wide. The bulk of the vertically lengthening ramus, again, is formed by intramembranous bone production. These extremes of horizontal and vertical growth can have infinite intermediate combinations, as seen in Figure 16-12, for example. Please note in Figure 16-12 that resorption occurs underneath the gonial angle. This is a normal phenomenon and can be observed in every forward rotating mandible. Resorptive shortening of the ramus implies that part of the vertical contribution of condylar growth and concomitant vertical relocation of the upper part of the ramus will not be expressed in a vertically longer ramus. Whatever the combination, the condyle becomes progressively relocated by appositional cell divisions, differentiation into cartilage, and expansive endochondral growth. That the condyle grows, and not is displaced, from a small mandible all the way to adult size is a notion of utmost importance, as will be explained.

Because of this sizable trajectory of growth of the condyle all surrounding structures attached to the condylar neck and elsewhere have to relocate in

FIGURE 16-10

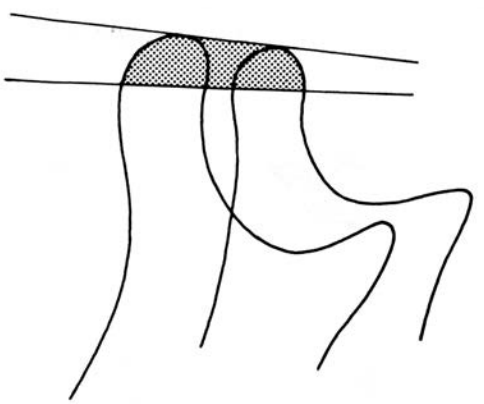


FIGURE 16-11

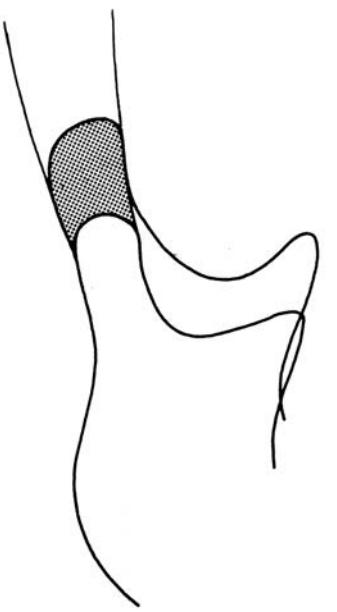


FIGURE 16-12

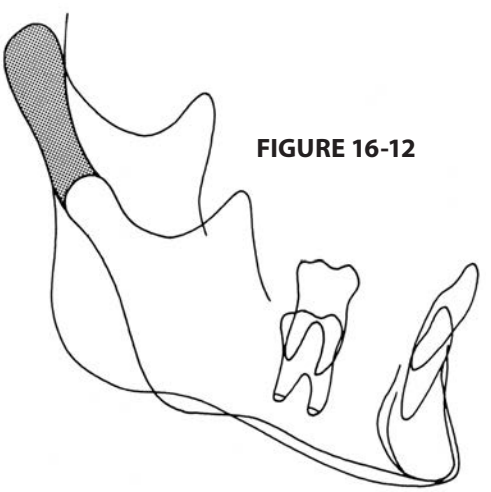
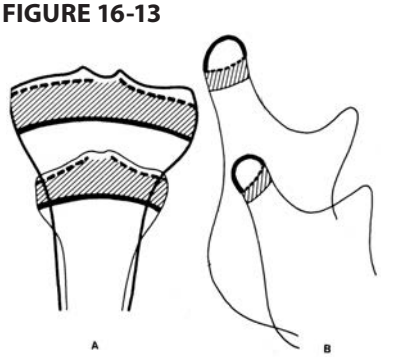


FIGURE 16-13



proportionate amounts. Capsular ligaments, rigid and strong in their function of stabilization against disarticulation, relocate continuously by detachments and reattachments. This capsular relocation can be done with substantial speed since condylar growth undergoes spurts every now and then. The process certainly requires a very sophisticated mechanism in order to continue to provide firm attachments and a changing interface simultaneously, often on a resorptive surface of cortical bone (see Chapter 14).

By reason of two conditions -instantaneous, versatile condylar directional response and intracapsular appositional endochondral growth- TM joint development occupies a special position among joints. Unique, indeed, are the impressive trajectory of intracapsular growth that the condyle has to achieve, and the ongoing and imperative reorientation of the stabilizing elements of this joint. This intracapsular growth of the TM condyle contrasts sharply with other joints in which the cartilage proliferation occurs in epiphyseal growth plates. These plates are situated outside the attachments of the capsules and ligaments. As a consequence, the TM joint needs a more extensive relocation of the capsule and ligaments, suggesting a greater chance on damage during development than the other joints of the body. This is exemplified in Figure 16-13, comparing a growing tibia (A left) with growth of the condyle (B right). The articular surface boundary is indicated by the broken line, the growth zone by the bold line, while the area for attachment of the capsule and ligaments (hatched) is located between the broken and the bold line. It is apparent that for the tibia there exists a considerable epiphyseal area for attachment of ligaments between the broken and the bold lines (hatched). This hatched area in the tibia is not affected by growth in the epiphyseal plate, and thus there is no need for major relocation of the capsule and ligaments. For the TM joint the situation is quite different. As all condylar growth takes place within the capsule, there exists a considerable need for relocation by detachment and reattachment.

To accommodate the complex conditions that exist with respect to changing vertical versus horizontal middle cranial fossa enlargement, progressive horizontal and vertical adaptations of mandibular form and position are necessary in order to place the lower arch in correct juxtaposition with the upper arch. These complex conditions, among others, include pharyngeal and nasal expansion, displacements of the palate and maxillary arch, remodeling adjustments of the palate and upper alveolar structure, drifting of primary and permanent dentitions (more specifically the vertical drift component), basicranial angular changes, concomitant secondary ethmomaxillary displacement, and nasomaxillary primary rotations. In addition, there are to be noted significant facial and cranial variations related to head form and morphologic and morphogenetic differences-during ascending ages. Obviously the condyles (and whole rami) must have an exceedingly versatile capacity for composite adjustments and adaptations for all of these multiple conditions. It is essential that throughout all of this, the mandibular arch is continuously positioned in functional occlusion with the maxillary arch and that a functional articulation in the TM joint is sustained, all simultaneously and without developmental interruption.

These essential relationships are sustained by appropriate responses of the

condyles to biologic signals in the amount, direction, and timing of its growth in conjunction with corresponding remodeling of the rami by their osteogenic connective tissues. It is primarily the mandibular rami, not the corpus, that provide for these mandibular adjustments (Fig. 16-12). The mandibular dentition drifts vertically to provide for these adjustments. Primary and secondary rotational and positional adjustments accompany these remodeling adaptations as the whole mandible simultaneously enlarges, as described in Chapter 4.

Acknowledgment

The author is indebted to Dr. H.W.B. Jansen, University of Groningen, for providing Figures 16-5 and 16-7.

17

Bone Growth Remodeling of The Early Human Face

Dr. Timothy G. Bromage
Dr. Alan Boyde

It is through an analysis of early human craniofacial growth and development that we gain several insights on the plan of the face. First is the link between variations of bone growth remodeling during our own ontogeny with those variations occurring over long stretches of evolutionary time. The second is a clearer understanding of the objective that remodeling has in generating the architecture of the face. Third, but not the least important, is that through an analysis of bone growth remodeling from fossil bone, aspects of the life histories of early hominids can be observed at the tissue and cellular level. These insights help to create enhanced understanding, which is useful to a variety of scientific disciplines concerned with hard tissue biology and the craniofacial skeleton.

BRIEF COMMENTS ON HOW WE CAN STUDY FACE AND JAW GROWTH TODAY

In studying the evolution from modern human infant to adult, we can benefit from the ability to follow living individuals during growth by observation, photography, measurement, facial casting, 3D computer measurement of external facial form, the standard cephalometric x-ray projections widely used for a long time by orthodontists and other standard diagnostic 'dental' x-ray procedures. There are very large numbers of dead individuals to be examined in anatomical, anthropological and forensic collections, and this material has been studied by sectioning and histology, notably by Enlow. Conclusions based on morphological study alone can be confirmed by 'labelling' procedures employing bone-mineralizing-front-seeking fluorophores such as the tetracycline antibiotics, calcein and alizarin in relevant animal species. Scanning electron microscopy (SEM) can be employed to study bone surface activity states, and thus to map bone addition and removal during growth and drift. We can profit from comparisons of contrasting species.

Possibilities for examining fossil remains are, in contrast, exceedingly limited, but general approaches involving non-destructive microscopical methodologies can, do and have contributed to our understanding of facial growth. These are (1) replica techniques for SEM, (2) direct inspection of both surface topography by through-focus mapping using confocal scanning optical reflection microscopy (CSOM) which to an extent duplicates the data obtained by SEM – but also amplifies it – and (3) sub-surface reflection CSOM, which permits the study of lamellar architecture deep to both natural and fractured bone facets. In the last case, we can especially profit from the parallel orientation of osteocyte lacunae with the collagen lamella in which they lie, since the former are larger and more obvious features, often well preserved in fossils. The possibilities of x-ray microtomography are superficially appealing, but the resolution of laboratory based systems which can handle large intact samples is too poor for histological analysis, even in modern human material – and this currently lies in the realms of imagination for unbroken fossil samples.

We know a great deal about facial growth and remodelling in our own species. We can mix and match data from gross growth studies with LM and SEM histology and it all 'makes sense'. Further, we can make it make more sense by making comparative studies in extant hominid species. In approaching fossil material, therefore, we can have confidence in amplifying and cross correlating gross anatomical growth observations with what can be winnowed from microscopical chaff – and that is the approach we have taken and review in this chapter.

A BRIEF REVIEW OF HUMAN EVOLUTION

The temporal scale of human evolution over the last 6 million years can be seen in Figure 17-1. In this chapter we will mainly concern ourselves with the portion of human prehistory straddling the Pliocene and Pleistocene epochs, roughly 3.0-1.5 million years ago (Ma). From putative ancestors, the earliest humans, or "hominids" in the common vernacular, spread throughout Africa along the river forests and lakes some 3.5 Ma when the climate was relatively humid. These were the several species of "gracile" australopithecines, so called because of the delicate form of their facial skeletons in comparison to later "robust" australopithecines. Some 2.8 Ma there was a general phase of global cooling and increasing aridity, culminating about 2.5 Ma at which time the *Australopithcus* lineage split into two major lines, one leading to *Homo sapiens* and the other to the "robust" australopithecines *(*genus *Paranthropus)* that became extinct about 1.0 Ma. Cranial capacities in these australopithecines species ranged from about 400-550 cubic centimetres (cc).

Paranthropus had a robust masticatory musculature and large teeth while *Homo rudolfensis* proved to be more flexible, with its capacity to adapt to climatic change with the development of a larger and more inquiring brain. The first tools evolved contemporaneously with the genus *Homo*, which enabled food preparation to take place outside of the body. Their incipient tool culture compensated for the effects of climate change long enough to enable *Homo rudolfensis* better to utilize alternative sources of food than had been the case with any previous hominid

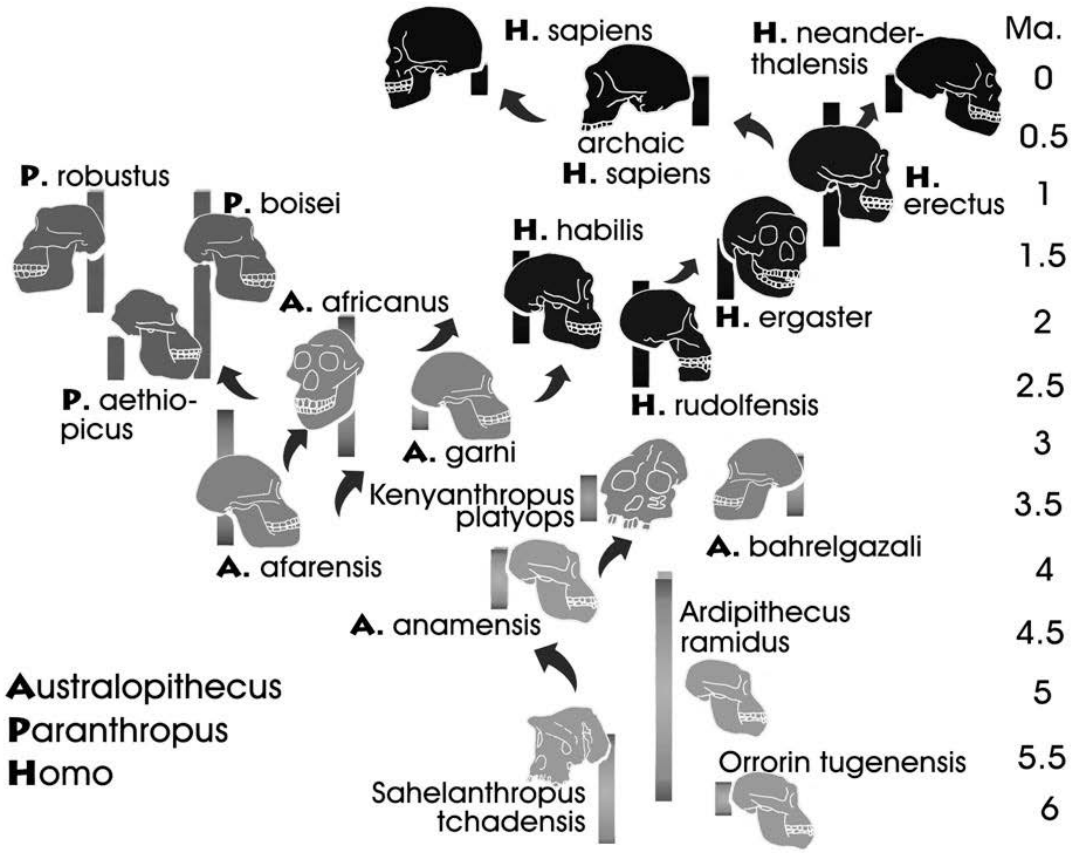


FIGURE 17-1.
Timescale of human evolution. Putative earliest hominids give rise to basal Australopithecus, which branches into Paranthropus and Homo lineages.

species. From earliest *Homo*, the species *Homo habilis* evolved, and, through the development of tools, was able to reap advantages not only in the provision of food but also in the ability to adapt to environmental change. These early *Homo* species had brain sizes around 550-700 cc.

By some 2-1.8 Ma *Homo ergaster* evolved in Africa and early representatives of *Homo* spread from Africa into Eurasia. Eventually, *Homo erectus* arose, these early humans spreading in Africa and well into Asia and Europe. The oldest skull, barely 2 million years old, has a brain size of about 800–900 cc, but by a million years ago individuals had reached about 900-1000 cc, and by 0.5 Ma it exceeded 1100 to 1200 cc.

Approximately 500,000 years ago there developed in Africa an archaic version of modern humans, and later the Neanderthals of Eurasia, which were a side-branch of humanity. Biologically modern *Homo sapiens* evolved in Africa about 200,000 years ago and subsequently dispersed throughout the Old World. The modern human average brain size is around 1350 cc.

THE STUDY OF FOSSIL HOMINID BONE GROWTH REMODELING

This chapter is a description of the bone growth remodeling patterns of *Australopithecus*, *Paranthropus*, and early *Homo*, which is useful for interpreting their craniofacial growth and development. Bone growth remodeling is a fundamental mechanism responsible for skeletal morphogenesis that involves coordinated surface patterns of bone resorption and deposition activity (described in detail in Chapter 2). Indeed, it is the key mechanism by which species specific craniofacial growth patterns are achieved and reflects, for instance, the differences between modern human and other non-human primate patterns of facial growth.

Evaluation of bone growth remodeling from histological thin sections (e.g. 10-100 μm thick) cut or sliced from developing bones has been the method of choice and also that method used for nearly all of the research leading to the knowledge contained in this book. Histology has been and will continue to be the 'gold' standard, particularly because the stratigraphy with depth in a bone contains many microanatomical characteristics indicative of how the bone grew (see Chapter 14 for details). Presently the only way of accessing this depth at sufficient resolution is by histological sectioning. It goes without saying, however, that no curator of unique fossil hominid bone will allow specimens to be sectioned, which is considered destructive sampling. Of course, it must be emphasized that it is actually "constructive" sampling, providing valuable information, but the integrity of rare or unique specimens can be compromised and we feel that it is our responsibility to search for ways of acquiring this information non-destructively.

The challenge we face for the non-destructive examination of fossil hominid bone is to obtain research-grade images of microanatomical features relating to bone growth remodeling and, even in field settings, from surfaces *and* below the surface. To achieve this, new approaches to the study of early hominid mineralized tissue biology have been developed, making it now possible to investigate actual mechanisms accountable for growth and development. Prior to the development of these approaches, hominid growth studies were studies of apparent growth. There were no studies of hominid growth mechanisms and therefore no study of actual growth processes characterizing early hominids. Recently, however, human evolution researchers, or paleoanthropologists, have focused upon non-destructive methods of analyzing the secretory activities of bone and tooth forming cells, giving developmental dynamics for fossil hominid bones and teeth. Dynamic features of skeletal morphogenesis in ontogeny and phylogeny can thus be described.

Bone growth remodeling takes place at periosteal and endosteal surfaces (see Chapter 2) and any internal space maintaining its spatial relations during growth. Because it is a surface phenomenon, techniques for the study of bone growth remodeling have been adopted by human paleontologists to study the processes of early hominid growth and development. In the main these studies are distinguished by their utilization of scanning electron and confocal optical microscopes to characterize the once forming (and mineralizing) and resorbing (or resorbed) fronts of fossil periosteal bone surfaces.

A **Scanning electron microscope (SEM)** study of developing cortical bone

surfaces reveals the characteristic microscopic surface features of bone formation and. Bone forming cells (osteoblasts) elaborate layers of an organic matrix containing highly oriented collagen which subsequently mineralizes. In fresh bone the mineralizing front is characterized by incompletely mineralized collagen fiber bundles in which spindle-shaped mineral clusters align in tandem along their lengths. In anything but the fastest bone deposition, arrays of preferentially oriented collagen fiber bundles are laid down. Canalicular spaces and osteocyte lacunae help to define the bundles (Figure 17-2a-b). In some cases, bundles are oriented parallel with the associated vasculature, forming a much larger feature called intervascular ridging bone (IVR) formed as a result of a capillary bed being incorporated into the developing bone. This cortical surface pattern is often useful because, whereas incompletely mineralized fiber bundles are extremely delicate, easily losing their mineral clusters when the non-mineralized organic matrix is removed, the IVR level of bone organisation is relatively resistant to destruction by abrasion, making it possible to identify forming bone surfaces on abraded fossil hominid bone (Figure 17-2b). Bone resorption is also an integral part of the bone growth remodeling process that permits resizing, reshaping and repositioning of bone during growth. The coordinated activity of bone matrix resorbing cells, osteoclasts, results in fields of anisotropic resorption bays, or Howship's lacunae, which are readily visible by SEM on resorbing bone surfaces (Figure 17-2c-d).

It should be noted that fossil hominid craniofacial remains are not normally examined directly by the SEM and a high-resolution replication technique is employed on original hominid specimens. A silicone-based dental impression material is used to make the negative and an epoxy resin forms the positive surface replica for imaging. The smallest features we need to resolve in order to distinguish forming and resorbing surfaces are about 5 μm, but the replica technique retains detail of less than 1 μm in size.

Portable Confocal Scanning Optical Microscope (PCSOM) examination of bone growth remodeling features on original fossil hominid specimens provides excellent surface reflection images comparable in detail to low-magnification SEM images. However, useful too is the capability with such a microscope to image non-destructively *below* the intact surface by as much as 50-100 μm. This is not very deep, but from such images it is possible to reconstruct some microanatomical elements at depth, such as collagen fiber orientation and osteocyte distributions, which can inform us about the nature of the bone's formation. For instance, in Figure 17-3a, spindle-shaped osteocyte lacunae observed deep to the periosteal surface of a gracile australopithecine femur shaft (*Australopithecus afarensis*, specimen AL 288-1a-p; "Lucy") show a preferred orientation, indicative of parallel-fibered circumferential lamellar bone deposition at this surface [to distinguish it from bone with 'crossed' lamellae]. Figure 17-3b shows osteocyte lacunae without a preferred orientation, which exists below the periosteal surface of a robust australopithecine mandible (*Paranthropus robustus*, specimen SK 64) previously characterized by SEM as resorptive in this location. This relatively non-oriented lacunar distribution is characteristic of bone formation at endosteal surfaces, but this lacunar pattern is observed at the periosteal surface, which means this bone volume was relocated by cortical drift indicative of contralateral bone surface

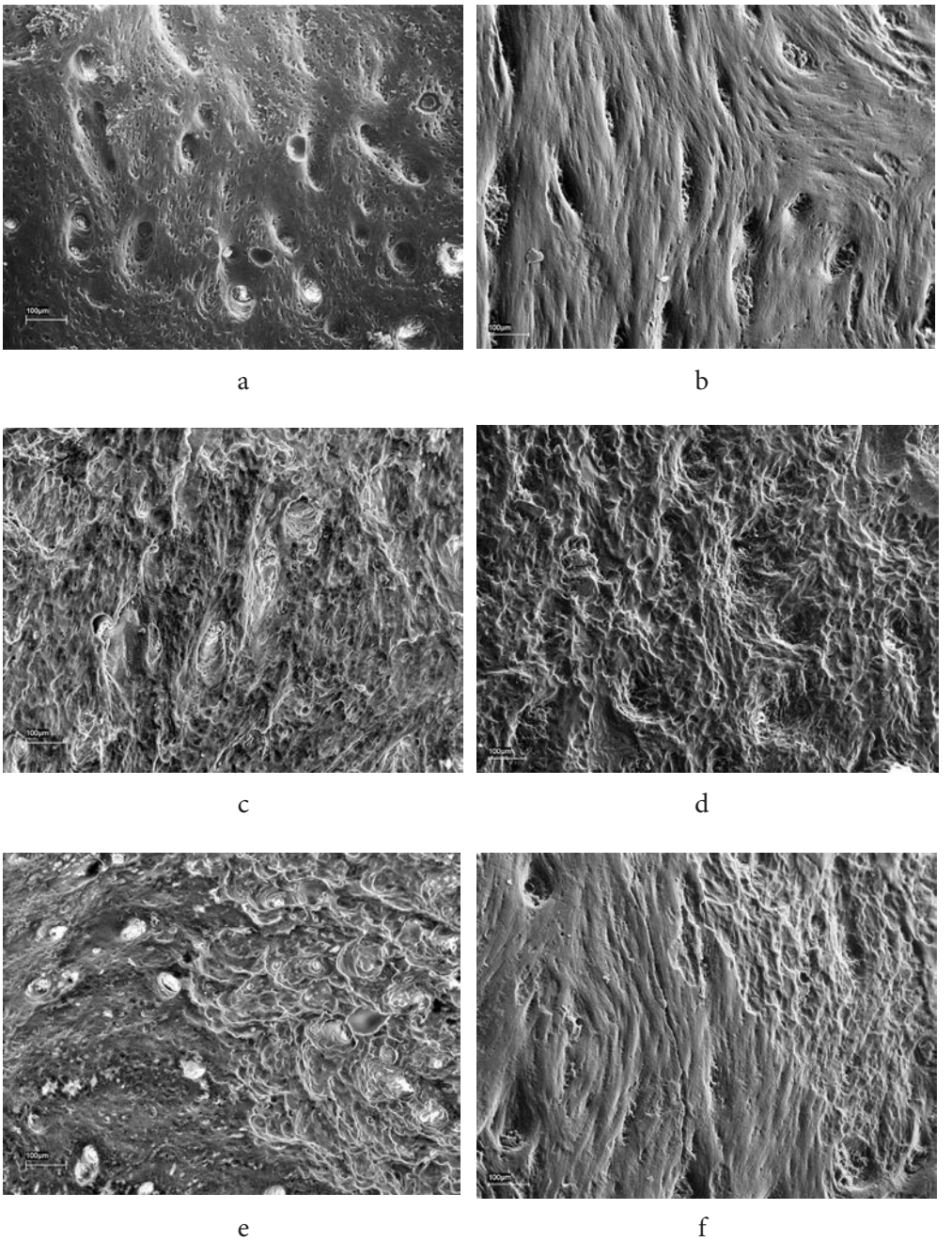
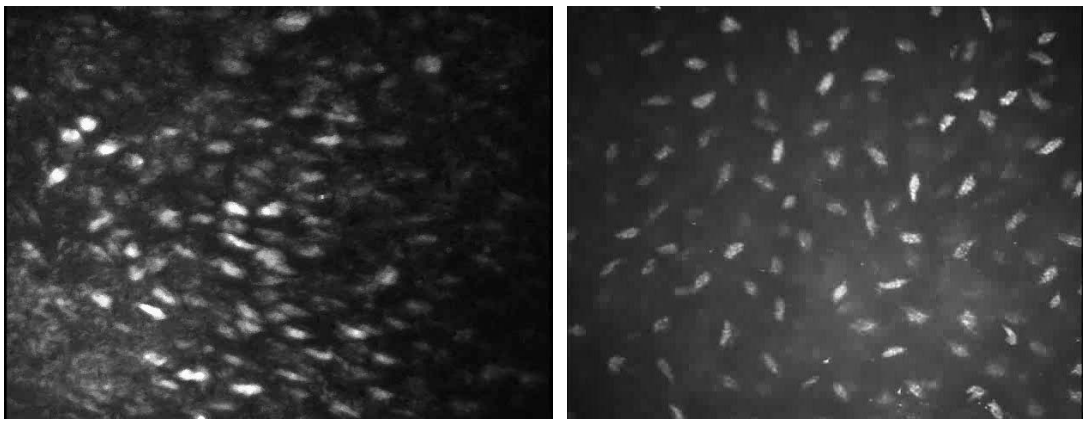


FIGURE 17-2.
Replicas of juvenile macaque (*M. mulatta;* left column) and fossil hominid (*P. robustus;* right column) facial bone growth remodeling surfaces. a-b, Forming surfaces; c-d, resorbing surfaces; e-f, remodeling reversal between fields of Howship's lacunae and forming bone. Thin sections of the macaque facial material permitted a comparison of traditional histological interpretations with SEM of the remodeling surfaces.



e f

FIGURE 17-3.
a, Oriented osteocyte lacunae characteristic of rapid, parallel-fibered periosteal bone formation. Note weak reflection from the bone surface at lower left, which was included in the initial image before acquiring below surface lacunae. b, Osteocyte lacunae without a preferred orientation characteristic of endosteally relocated bone beneath a region of periosteal bone resorption. Lacunae parallel the collagen bundle orientation in the lamellae in which they lie, and this pattern arises from intercepting several successive lamellae in depth, indicating 'crossed' lamellar organisation. Images a and b were recorded under the same conditions: the size of the osteocyte lacunae is greater in a and their concentration, i.e. volume fraction, is much higher, both trends indicating more immature and more rapid bone formation.

resorption (see Chapters 2 and 14). However, because non-oriented lacunar distributions may also characterize rapid bone formation rates at any surface, it is always useful to observe other features, such as osteocyte shape and distribution, which provide information on bone formation rate, as well as surface topography for evidence of Howship's lacunae.

Information gathered from SEM and PCSOM sources about the distributions of bone growth remodeling activities on fossil hominid bone may be used to create descriptive "maps" that contribute to interpretations of facial growth and development. However, there are two caveats that should be noted. First, these maps are invariably patchy in nature due to the vagaries of fossilization. For the purpose of forming morphogenetic interpretations, we must rely on the generalized field nature of surface bone growth remodeling, whilst recognizing that some spatial and temporal heterogeneity of activity may occur within a field. In a few cases where no remodeling data for a specified location exists, yet we desire a morphogenetic interpretation, the term "possible" will be used when speculating on the remodeling characteristics. The second caveat is that, because of abrasion, we cannot know if bone growth remodeling surfaces were active at the time of death or whether the surface was "resting", forming or resorbing; that is, the surface characteristics area indicative of a bone growth remodeling circumstance, but the cellular activity has ceased. Thus all we can say is that the *last* bone growth *activity* state was forming or resorbing.

THE STUDY OF FOSSIL HOMINID FACIAL GROWTH

The morphogenetic interpretations presented here take into account concepts and principles of craniofacial growth and remodeling described in other chapters of this book. The concepts of cortical drift and the "V" principle are particularly important to an understanding of bone growth remodeling (see Chapter 2 for details). Cortical drift requires that bone deposits accrue in the direction of growth while bone resorption occurs on the contralateral cortical surface that faces away from the direction of growth, thus causing the drift of bone through morphological space. This cortex may constitute the entire thickness of a bone and be very thin (e.g. bones of the orbital and nasal cavities) or it may belong to a bone that has an endosteal surface facing a diploic cavity, which is also involved in the remodeling process. Endosteal surfaces facing the direction of growth will receive deposits and endosteal surfaces facing away from the growth direction will undergo resorption. For such a bone to be simply moving through tissue space, remodeling activities on the endosteal and periosteal surfaces of a cortex typically have an opposite remodeling configuration on what is said to be the corresponding contralateral cortex. For the most part, SEM and PCSOM access is limited to periosteal surfaces in the fossil hominids.

Enlow (1963) noted that many bones of the craniofacial region were shaped like a "V" and had unique cortical drift properties. He named the "V" principle which accounted for the enlargement of bone even when its outer surfaces were resorptive and its inner surfaces were depository. This seemingly paradoxical situation is explained by realizing that the inner surface is situated such that it faces the growth direction. Thus enlargement is toward its own wide end during continuous sequential adjustments in that direction. The inner surface receives bone deposits while the outer surface is progressively removed because it faces away from the growth direction. At the same time the entire bone also moves in a direction away from the narrow end, sequentially adjusting, or relocating, various parts of the "V" to new levels. Wide areas of the "V" are relocated into narrow parts while moving the "V" simultaneously forward. For example, most surfaces facing the orbital cavity have been found to be '*depository*' in humans and macaques. Consequently the bony orbital framework drifts anteriorly because the inner aspect of the "cone" (visualized as a three-dimensional representation of a "V") faces the growth direction. It is also important to stress that the contralateral surfaces are largely '*resorptive*', "pinching out" the bony orbit posteriorly and that enlargement is dependent upon deposition at the free edge of the orbital rim, which is oriented in line with the outward projection of the "V".

Interpretations of early hominid facial growth below will also be provided for sutural remodeling (see Chapter 14). Whereas microscopic features relating to sutural remodeling have not been observed on fossil hominids, they can be inferred from the nature of periosteal remodeling patterns, vectors, and principles.

EARLY HOMINID FACIAL GROWTH AND REMODELING

Australopithecus

Taken together, an *Australopithecus* sample of 10 fragmentary facial remains provides a consistent pattern of facial remodeling (Figure 17-4, Table 17-1).

The orbital region (see Chapter 5). Inner surfaces were depository. The surfaces have an anterior component to their orientation and hence deposits of bone over portions of the inner surface combined with contralateral resorption cause the orbit to drift anteriorly according to the "V" principle. At the same time, expansion of the orbit can be accommodated by sutural growth in the vertical dimension at the frontomaxillary and frontozygomatic sutures, whereas sutural contributions to increases in orbital width come principally from the external and inner (orbital floor) aspects of the zygomaticomaxillary suture. Sutural contributions on the floor and sides of the orbit, from the articulations of the frontal, zygomatic, maxilla, lacrimal, ethmoid and sphenoid bones would help to maintain the orbit in anatomical relation to the posteriorly located cranial floor and the expanding ethmoid sinus and nasal cavity during growth. The roof of the orbital cavity harbors no sutures and so surface deposition combined with a possible resorptive or intermittent resorptive contralateral surface on the floor of the anterior cranial fossa would serve to relocate the orbit in an anteroinferior direction.

Whereas deposits on the superior orbital wall keep pace with downward vectors of growth due to the expanding and posterosuperiorly overlying frontal lobes of the brain, deposits on the inferior aspect relocate the orbital floor upward, compensating for downward growth influences from the developing nasal capsule and palate. This maintains the orbit in the same relative position during the growth period. Deposits on the superior, inferior, superolateral, inferolateral and medial orbital rims would permit these "free" edges of the orbit to drift outward and anteriorly, permitting an increase in orbital volume.

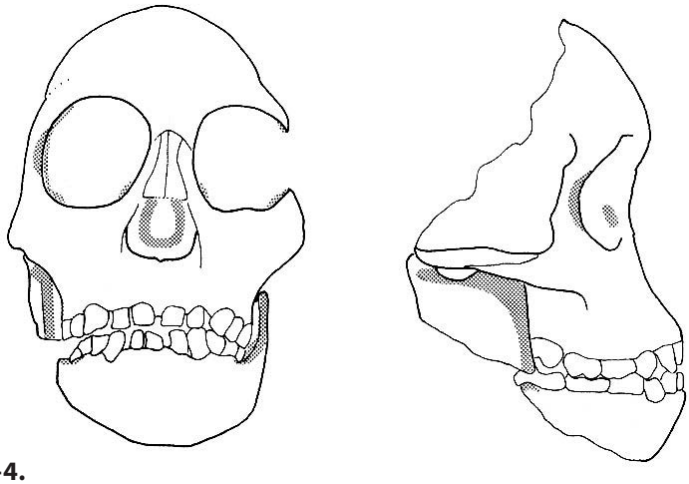


FIGURE 17-4.
Frontal and lateral views of *Australopithecus* facial remodeling, superimposed onto the Taung child (*A. africanus*) about 3·3 years old (based on an ape-like life history). Shaded bone represents resorption while non-shaded regions represent deposition.

Specimens

Individual #	Most recently erup ed teeth
Pan	
1	$M^1 + M_1$
2	$M^1 + M_1$
3	$M^1 + M_1$
4	$M^1 + M_1$
5	$M^1 + M_1$
6	$M^1 + M_1$
Homo	
1	$M^1 + M_1$
2	$M^1 l_1$
3	$M^1 + M_1$
4	$M_1 l^1$
5	$M^1 + M_1$
6	$M_1 l^1$

Results

	Pan						modern *Homo*					
Anatomical Reg on	1	2	3	4	5	6	1	2	3	4	5	6
Anterior temporal fossa	-	+	+	+	+	+	+	+	+	+	+	+
Lateral orbital rim	+	+	+	+	+	+	+	+	+	+	+	-
Inferomedial to infraorbital foramen	+	+	+	+	+	+	+	-	+	+	+	+
Nasoalveolar clivusclivus	+	+	+	+	+	+	-	-	+	-	-	-
Labial incisor alveolus (mandible)	+	+	+	+	+	+	-	-	+	-	-	-
Lingual incisor alveolus (mandible)	+	+	+	+	+	-	+	+	+	+	+	-

Table 17-1.
Three early hominid taxa have been investigated for their bone growth remodeling. Chronological ages, when estimated, and element (M = mandibular fragment, FS = facial skeleton fragment) are given for each specimen.

The resorptive field in the vicinity of the superomedial part of the lacrimal would permit this corner of the orbit to relocate inferomedially, keeping pace with descent of the nasal capsule (see below). The bone in the vicinity of the inferior part of the lacrimal is depository and would relocate superolaterally to accommodate the rapidly expanding ethmoidal sinuses below. Resorption at the lateral anteroinferior corner of the orbital floor relocates this area inferolaterally, outward and forward. This resorptive field might possibly have been continuous with one over the central portion of the anteriorly-facing aspect of the lateral orbital rim. This area could not be mapped, but this field may have existed given the notched configuration of the rim as seen in lateral view. The posteriorly-facing cortex of the lateral orbital rim (the contralateral cortex) on the anterior wall of the temporal fossa is depository. This provides evidence for a posteriorly directed

mode of growth of the lateral orbital rim.

The nasal and frontal region (see Chapter 5). The forward-facing periosteal surfaces of this region showed bone deposits. This feature contributes to the maintenance of a facial depth and profile congruent with the anteriorly drifting orbits. Resorption within the inner recesses of the nasal capsule signifies outward modes of growth, laterally and inferiorly. Whereas the orbital floor is depository in order to maintain the orbits in a superior position, i.e., above the jaws, resorption on the nasal floor serves to relocate the floor inferiorly, increasing the nasal capsule's vertical separation from the orbits during growth. The expanding orbital and nasal functional matrices require this separation because of medial encroachment of the orbit during growth onto the root of the nose. The roof of the nasal capsule was possibly depository to some degree, with contralateral resorption on the meningeal aspect (anteroinferior to the frontal lobes), so that this region could participate in the downward mode of growth. Deposits at the frontomaxillary suture would also accommodate and act in concert with increases in height of the nasal capsule.

The posterolateral wall of the nasal capsule is characterized by bone deposits. The nasal capsule retreats somewhat posteriorly and its posterior boundaries may face obliquely forward, backward, downward or upward depending upon the position along the wall and the state of the developing dentition. The forward facing aspect of the lateral wall is depository, serving to relocate the capsule anteriorly. The anterolateral wall shows localized deposits, particularly where the piriform aperture grades onto the external bone table. However, resorption occurs just behind the aperture.

The premaxillary, maxillary and zygomatic region (see Chapter 5). All external periosteal surfaces are depository. Deposits on the lateral-facing aspects along with mid-palatal sutural growth and deposits on the anteriorly-facing aspects combined with sutural growth in the coronal plane contribute to lateral and anterior components of growth in this region. The persistence of premaxillary sutures in young *Australopithecus* supports this growth sequence, which assists in the providing of space for sizeable incisors that are developing in this area and promoting the physiological spacing between deciduous incisors. Resorption on the floor signifies a depository palatal surface (the contralateral cortex) and a downward component of growth of the nasomaxillary complex. Thus deposits on the other inferiorly directed surfaces of this region, that is, zygomaticoalveolar crest, zygomatic and alveolar arches, also participate with sutures in the transverse plane, in facial height increase.

The posteroinferior aspect of the zygomatic bone on the anterior wall of the temporal fossa is depository, parallel with but on the opposite side to the lateral orbital wall, where the zygomatic turns sharply medially and posteriorly. This suggests that the inferolateral and superolateral distribution of resorptive activity on the inner orbital aspect was relatively larger than that of modern *Homo* and more similar in proportion to the condition observed in the rhesus macaque. Bone resorption occurs on the posteroinferior portion of the zygomatic just behind the zygomaticoalveolar crest. Combined with deposits on the anteriorly-facing aspect of the zygomatic bone, this remodeling pattern would have the effect of relocating the root of the zygomaticoalveolar crest anteriorly.

The mandible (see Chapter 4). The preserved retromolar space and the anterior root of the ascending ramus are resorptive and characterize the remodeling conversion of ramus into corpus, thereby lengthening the corpus to accommodate the developing permanent teeth. The coronoid process in immature *Australopithecus*, like that also described for early *Homo* and *Paranthropus* (below) is oriented such that its medial surface faces slightly posterosuperiorly. The medial surface is depository, which would serve to lengthen the process vertically, and posteromedially relocate the coronoid process into posteromedial alignment during growth by cortical drift. Therefore, this remodeling feature is combined with resorption on the superolateral aspect of the coronoid process (the contralateral cortex) because this surface would be facing away from the growth direction.

Resorption extends along the anterior and medial surfaces of the condylar neck. This remodeling feature permits the growing, wide end of the condyle to successively relocate into the narrower neck while the mandible is being displaced inferiorly and anteriorly, commensurate with its posterosuperior growth and articulation with the cranial base. Resorption also occurs in the region where the lingual tuberosity grades into the narrower and more laterally positioned ramus thus permitting this conversion to take place and providing for the posterolateral widening of the ramus. Deposits on the posterior aspect of the ramus confirm the deduction of the mandible's posterior drift (and anterior displacement) and go together with the growth and remodeling interpretations above.

The labial and buccal surfaces of the corpus are principally depository, which would contribute to anterior and lateral modes of growth. The only evidence for a deviation from this pattern is the bilateral expression of resorption lacuna concentrations beneath the permanent lower lateral incisors of one specimen examined. In apes, these teeth begin to erupt just prior to eruption of the permanent second molars (which were in this case in the process of erupting) and so resorption around these teeth probably represents minor alveolar adjustments in tooth position.

The lingual surfaces of the mandibular corpus are depository. Deposits on this aspect of the mandible face obliquely backward and downward and so would have contributed to the posterior direction of growth as well as to an increase in the vertical dimension of the corpus into wider projections of the basal arch according to the "V" principle, particularly during growth increments in width of the condylar articulation with the cranial base. In macaques, the alveolar planum is resorptive and this, together with labial deposition, contributes to the anteriorly directed drift of the muzzle of this prognathic monkey.

Early *Homo*

Only two mid-facial remains and three mandibles of early *Homo* have been investigated for their bone growth remodeling characteristics, (Table 17-1), but certain morphogenetic interpretations can be drawn from the remodeling maps, particularly for the mandibles.

The maxillary region. The preserved maxillary remains indicate bone deposits on the posteriorly-facing aspect of the maxillary tuberosity, the buccal aspects as far forward as the distal surface of the canine, the oral side of the

palate and the lateral-facing surface of the anterior aspect of the medial wall of the temporal fossa. There is nothing in these limited results concerning the lower portions of the midface that diverges from the remodeling patterns characteristic of the *Australopithecus* sample. Given the similarity of some *H. habilis* specimens with *Australopithecus*, it is no surprise to find that the remodeling patterns corresponding with the development of the relatively prognathic face of the latter group are also exemplified in the face of some early *Homo* specimens. However, the emphasis on forward growth of the face cannot be demonstrated by deposits on the anterior-facing aspects of the zygomaticomaxillary region, because these parts are not represented in the study sample.

It is nevertheless fortuitous that the pterygoid fossa of one preserved juvenile shows evidence of resorptive activity during growth because this is an indication that the fossa was relocating by cortical drift in an anteroinferior direction (Figure 17-5). This remodeling feature has been demonstrated for macaques and contrasts with the depository nature of the fossa in the development of the characteristic orthognathic human face. Thus deposits on the fossa's contralateral cortices, combined with deposits on the maxillary tuberosity (displacing the maxilla anteriorly), would have served to emphasize the anterior component of growth in the early *Homo* face. It could therefore be predicted that the nasoalveolar clivus of at least some early *Homo* specimens would be depository, as noted for *Australopithecus*.

An additional remodeling feature, not available for study in the *Australopithecus* sample, is the resorptive field located on the inner posterolateral

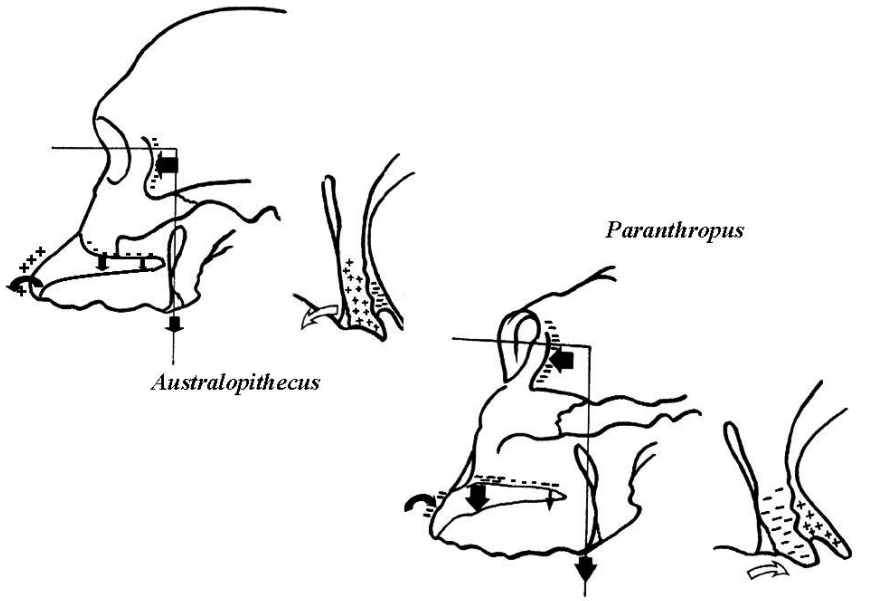


FIGURE 17-5.
Lateral views of *Australopithecus* and *Paranthropus* bone growth remodeling together with enlarged tracings of their respective pterygoid regions. Bone deposition (++) and resorption (--) are shown together with closed arrows denoting cortical drift. Open arrows indicate cortical drift and growth direction of the pterygoid apparatus and maxillary tuberosity.

aspect of the palate above the last tooth to erupt posteriorly. Modern humans are characterised by deposition on this surface and shown an inward turning of the maxillary tuberosity. *Pan* shows a beveled surface outward in this vicinity, but no remodeling data is available to account for this. Resorptive remodeling in this position may be in part responsible for widening of the palate.

The mandible. There is nothing in the remodeling features of early *Homo* mandibles that distinguishes them from *Australopithecus* (Figure 17-4). The preserved retromolar space and the anterior root of the ramus of the latter are resorptive and characterize the remodeling conversion of ramus into corpus, thereby lengthening the corpus to accommodate the developing posterior permanent teeth. In addition, the anterior portions of the ramus are depository on their medial and lateral surfaces. Coronoid process orientations suggest that superolateral deposition on the medial surface, together with contralateral resorption, characterize the posteromedial relocation of the process as it occurs in all other primate forms studied (i.e., modern *Homo*, *Macaca*, and *Australopithecus*).

The labial, buccal and lingual surfaces of the mandibular corpora are mostly depository accounting for the growth in width, height and length, as described for *Australopithecus*. However, some specimens show resorption on the alveolar plane which contributes to the anterior mode of mandibular growth and ventral rotation of the plane relative to its posterior border.

Glimpses of resorptive activity are recognized around the posterior margin of the mandibular foramen indicating that during anterior displacements of the mandible, the foramen must have drifted posteriorly to maintain its relative position on the ramus during growth movements of the whole mandible in the anterior direction. Another specimen exhibits resorption on the posteroinferior borders of its mental foramen suggesting that this structure also maintained relative vertical position on the corpus by relocating inferiorly during increases in corpus height.

Paranthropus

Paranthropus has a pattern of facial remodeling characterized by resorption on some anterior and anterolateral surfaces of the maxilla and mandible (Figure 17-6, Table 17-1). These resorptive surfaces are reminiscent of modern human remodeling patterns with the result that both taxa have relatively orthognathic facial skeletons.

The orbital region. The inner orbital aspect is characterised by deposits on the [preserved] inferior, lateral and superior walls. Thus, similar to *Australopithecus*, and as explained for humans and macaques, these surfaces have an anterior component to their orientation and the orbit hence drifted anteriorly according to the "V" principle. However, the nature of this drift takes on different proportions in *Paranthropus*.

Resorption occurs on the inner superolateral depression, largely hidden in frontal view by the overhanging supraorbital margin. Resorption on this surface combined with deposits on the anteriorly-facing aspect of the supraorbital margin would relocate the orbit and superior orbital margin anterolaterally.

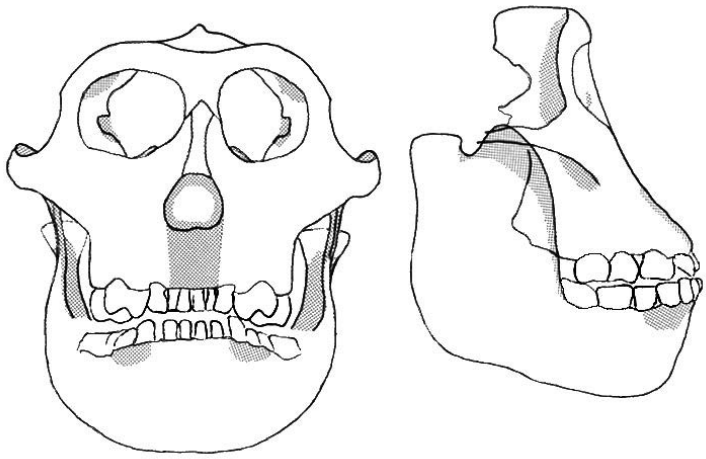


FIGURE 17-6.
Frontal and lateral views of *Paranthropus* facial remodeling, superimposed on a construct based largely on two *P. robustus* specimens, the midface data coming from an individual aged 11.3 years old (based on an ape-like life history). No younger specimen was available for this figure to enable direct comparison of absolute proportions with Figure DD-DD. Shaded bone represents resorption, while non-shaded regions represent deposition.

Resorption also occurs over the inner posterolateral orbital wall, reflecting the conversion of bone located in the region of "minimum frontal breadth" into the wider braincase behind (where the cranial cavity impinges on the orbit). Here the drift was outward, to compensate for anteriorward relocation and separation of the orbits from the cranial cavity, and is verified by deposits on the anteromedial wall of the temporal fossa. The inner lateral orbital wall (slightly anterior to the posterolateral resorptive field noted above) could not be mapped, nor was the contralateral surface interpretable, but the inner orbital aspect would likely have been depository according to the "V" principle and is verified to a certain extent by the observed resorptive character of the anterior wall of the temporal fossa.

Expansion of the orbit is also complemented by sutural growth at the frontozygomatic, frontomaxillary and zygomaticomaxillary sutures. Additions at the zygomaticomaxillary suture of *Paranthropus* must have been extraordinary - as determined by the markedly inferior position of the infraorbital foramen relative to the suture. In all living apes and humans the foramen is within a few millimeters of the suture early in postnatal life (and so likely was that of *Paranthropus*) and their increasing separation reflects increments of growth on the maxillary side of the suture. The reality of a differential increase along the suture notwithstanding, the foramen is sequentially displaced inferiorly as a result of this growth and generally maintains its position (albeit with some variation) lateral to the vertical center of the nasal capsule and below the center of the orbital cavity or the apex of the zygomaticomaxillary suture where it meets the inferior orbital margin. This architectural relationship is a constant during craniofacial growth that is maintained during vertical descent of the nasal capsule and palate and horizontal growth occurring with increasingly wider projections of the midface.

Compensatory remodeling upward - i.e., resorption on the roof of the infraorbital canal and deposition on its floor - probably occurred very early in *Paranthropus*' postnatal life when increases in orbital mass probably exceeded palatal descent, but this would have been greatly overshadowed during later ontogenetic stages.

Growth at the zygomaticomaxillary suture also results in an increase in the transverse dimension of the suborbital region. This contributes, in part, to the lateral expansion of the peripheral face in the zygomaticomaxillary region. The frontozygomatic suture is situated obliquely on the superolateral orbital rim to maintain relations of the zygomatic process of the frontal bone to the frontal process of the zygomatic bone (which was being displaced laterally due to growth increments at the zygomaticomaxillary suture). This sutural configuration can be observed on many living ape (including hominid) specimens. Together with inner orbital superolateral resorptive remodeling, this growth pattern increases the vertical height and width of the orbital cavity.

The nasal and frontal region. Increases in height of the medial orbital margin were abetted by deposits at the frontomaxillary suture. Sutural increments were oblique to the transverse plane and closely related to vertical and horizontal compensatory adjustments made necessary during inferolateral growth and displacement of the maxilla. The frontomaxillary suture is normally inclined inferolaterally away from the frontonasal contact in order to maintain its distal relation to the middle third of the medial orbital rim: a spatial relationship observed for all living apes and humans. The obliquity of the frontomaxillary suture, combined with an anteriorly-facing frontal process of the maxilla, widened the *Paranthropus* interorbital dimension considerably.

The anteriorly-facing periosteal surfaces of the nasal region are depository. This would provide for the forward growth of this region keeping pace with the anteriorly drifting orbits. Resorption is indicated within the preserved upper regions of the nasoalveolar "gutter" on some specimens, which signified an outward and inferior direction of growth. Deposits at the frontomaxillary suture would act in concert with inferior growth of the nasomaxillary complex as described for *Australopithecus*. Similarly, descent of the nasomaxillary complex was compensated for by deposits on the orbital floor thus increasing the vertical separation of the palate from the orbits.

The premaxillary, maxillary and zygomatic region. This region shows marked remodeling differences from the *Australopithecus* examples which can be explained as a result of pronounced vertical hyperplasia of the *Paranthropus* posterior face (i.e., the development of a vertically long posterior facial height). The vertical dimension of the posterior face of human children increases more than the anterior face resulting in an upward rotation of the nasomaxillary complex (i.e., clockwise when viewed in profile from the left side). This relative hyperplasia of the posterior maxilla was observed to be compensated by differential resorption over the nasal floor such that the anterior portions drifted further inferiorly thus matching the disproportionate displacement superiorly. All extant hominoids are characterised by a vertical hyperplasia of the posterior face, offsetting otherwise empirical architectural relationships. In *Paranthropus*, relative vertical hyperplasia of the posterior face was relatively extreme.

Paranthropus posterior face hyperplasia is indicated by an especially deep (superoinferiorly) posterior palate. This resulted in a considerable amount of upward growth rotation of the anterior face and would have been compensated for by differentially greater resorptive activity over the anterior nasal floor. Increased inferior drift of the anterior palate "re-establishes" and maintains the occlusal plane in the downward and forward growth of the face and results in a relatively shallow anterior palate. This palatal configuration is one of the uniquely derived characters defining *Paranthropus*. Comparatively, eastern African *Paranthropus boisei* exhibits a deep anterior palate than *Paranthropus robustus*, which indicates a relatively greater alveolar contribution to anterior facial height, but a gradient does still characterize increasing palatal alveolar depth posteriorly.

Unrestrained upward rotation of the anterior face would have the undesired effect of shifting the upper face and orbital axes out of balance with the harmonious down and forward growth of the face. Upward rotation of the orbital functional matrices results in their confluence with the neural functional matrix above and behind, requiring the orbits to displace and drift anteriorly. Therefore, because of the marked posterior hyperplasia and the upward rotation of the anterior face that this would cause, the orbital functional matrices would likewise have to compromise their position and relocate markedly anterior. This anterior relocation, resulting in a relatively long and broad anterior temporal fossa, together with the marked increases in facial width described above, results in the postorbital constriction characteristic of *Paranthropus*. This was accompanied by resorption over the greater part of the anterior wall (posteriorly-facing) of the temporal fossa. This, together with deposits over much of the anterolateral and lateral anteroinferior aspects of the inner orbital walls, and deposits on the anteriorly-facing aspects of the maxillary and zygomatic bones, relocated the upper *Paranthropus* face in an anterior and lateral direction. Anterior relocation involves the entire upper face and is indicated by resorption high up and behind the supraorbital margin as well. Furthermore, the anterior aspect of the pronounced concavity behind the supraorbital margin faces away from the growth direction and is consequently resorptive, and together with deposits on the anterior aspects of the supraorbital margin, relocated the superior orbital margin anteriorly in concert with the rest of the upper face.

According to Rak (1983, 1985) the structural basis for relative facial orthognathism in *Paranthropus* is accounted for by two "antagonistic" processes: (1) the anterior positioning of the upper face and (2) the posterior positioning of the dental arch relative to the cranial base. The facial growth and remodeling described for the *Paranthropus* upper face (above) explains the ontogenetic pattern of events that determine the first of these morphological observations. As regards the posterior positioning of the *Paranthropus* dental arch, the distance between the third molar and the articular eminence is relatively short compared with other hominids and apes. Thus while the dental arch was certainly no shorter than that of other hominids it nevertheless retruded underneath the face providing a musculoskeletal adaptation emphasizing an anteriorly positioned masticatory musculature. This was possible given the accentuated protrusion of the upper face, and whereas the midface in modern *Homo* lay beneath the anterior cranial fossa,

the midface of *Paranthropus* descends largely beneath an anteriorly positioned upper face.

Evidence for maxillary retrusion relative to the upper face, independent of the absolute length and position of the dental arch, is the resorptive character of the external aspect of the pterygoid lamina (Figure 17-5). The pterygoid fossa was depository and this region drifted posteriorly during growth. Enlow (1975) noted that the protrusive nasomaxillary complex of the rhesus monkey was associated with resorptive pterygoid fossae and contralateral depository surfaces on the external aspects of the pterygoid laminae. This remodeling pattern was explained to account for a downward and forward growth direction of the pterygoid region and, combined with deposits at the maxillary tuberosity, served to emphasize the anterior component of growth. This pattern characterizes early *Homo* and possibly *Australopithecus* but does not characterize *Paranthropus*. Modern *Homo* has depository pterygoid fossae and a resorptive external aspect of the laminae, which reflects the downward human facial growth vector. *Paranthropus* also exhibits this pattern as represented by the resorptive aspect of the anteromedial and medial aspects of the medial pterygoid lamina. Thus anterior displacement of the midface was not emphasized by an anterior drift of the pterygoid region. The *Paranthropus* pterygoid region was, instead, drifting inferiorly and may, furthermore, have permitted relocation of the arch posteriorly thus compensating for a small proportion of the anterior displacement of the dental arch.

Sequential retrusion of the *Paranthropus* maxillary arch during ontogeny was also made possible by resorption over the nasoalveolar clivus, a mechanism characteristic of modern *Homo*. This remodeling pattern accompanies a facial growth vector that emphasizes a downward growth of the midface versus the forward growth of this region in *Australopithecus* (characterised by deposits). The resorptive nasoalveolar clivus in *Paranthropus* and modern *Homo* is oriented obliquely upward and hence is facing significantly away from its downward growth direction. This resorptive field sequentially relocated the nasoalveolar clivus posteroinferiorly and mitigated anterior growth displacement of the maxilla originating at the maxillary tuberosity. Upward growth rotation was also compensated downward and, combined with the depository character of the surfaces lateral to the piriform aperture, a sunken clivus or "gutter" was formed. Thus whereas Rak (1983) interprets no morphological basis for comparison of like degrees of facial orthognathism in *Paranthropus* and modern *Homo*, it is evident that the ontogenetic mechanisms are remarkably similar (i.e., resorptive remodeling over the nasoalveolar clivus and external aspect of the pterygoid laminae) and that they can be directly compared.

Resorption is also variably expressed on the buccal surfaces of the maxillary corpus, according to the "V" principle, which encouraged the downward mode of growth of the maxillary arch. Thus deposits on the palatal surfaces relocated this structure inferiorly while additions at the "free" alveolar edges, which are oriented slightly obliquely outward, served to shift the arch into wider projections of the "V". Resorption far back on the buccal maxillary corpus may have been compensatory to posterior arch widening influences thereby relocating the maxillary tuberosities inward.

The mandible. The *Paranthropus* mandible is the best represented element in the fossil hominid sample examined. The retromolar space and the anterior root of the ascending ramus, as in all taxa studied to date (i.e., *Australopithecus* and early *Homo* in the present work, and modern *Homo* and Macaca studied by Enlow and his coworkers) is resorptive and was responsible for the conversion of ramus into the posteriorly elongating corpus. The exceptionally broad retromolar space, which is characteristic for the taxon, extended back to the pronounced endocoronoid buttress. The broad and deep retromolar space has surfaces facing anteriorly and medially, indicating a posterior and lateral drift of the anteroinferior aspect of the ramus during growth. Indeed, lateral cortical drift of the anterior root of the ascending ramus would have been a necessary ontogenetic sequence relocating the ramus into line with the pronounced buccal swelling at its base: a feature characteristic of robustly constructed jaws. Lingual resorption beneath the first-second permanent molar region might also be related to the lateral drift resulting in the buccal swelling. Posterior drift of the *Paranthropus* lingual tuberosity was a necessary concomitant of the addition of large molar teeth to the posterior arch - which was, in turn, directly related to the necessity of a broad retromolar space (i.e., a posteriorly drifting surface scaled to the dentition that it must accommodate). Deposits at the lingual tuberosity also complemented displacement of the mandibular arch anteriorly, as described for the maxilla.

The posterolateral drift of the anteroinferior region of the ascending ramus was matched by a similar growth vector at the gonial region. Additions on the posterior aspects of the rami displaced the mandible anteriorly, resulting in growth at the mandibular condyles. Resorption in a concavity of the medial gonial region sequentially relocated the gonial margin into a more lateral position behind the lingual tuberosity. The medial aspect of the inferior gonial margin, however, is depository and accounted for an inversion of the margin. This inversion may also be verified to a limited extent by resorptive activity on the lateral aspect. The posterolateral wall of the mandibular foramen is resorptive and likewise relocated in an outward direction in order to keep pace with ramus growth and to maintain a position posterior to the developing teeth.

It can be seen that whereas the lateral anteroinferior aspect of the anterior root of the ascending ramus is depository, commensurate with its lateral mode of growth, the superolateral surface of the coronoid process is resorptive. Its contralateral surface on the medial aspect of the process is depository and faces posteriorly (the back of the endocoronoid buttress), medially and superiorly. Thus the growth direction of the process reflected anterior and inferior displacement of the mandible and medial compensation for increases in width of the ramus involved in maintaining the condylar articulation with the widening cranial base. Resorption over the neck of the condyle reflected the conversion of the growing wide end of the condyle into the narrower neck during inferior displacement.

The labiobuccal remodeling of the *Paranthropus* mandible is consistent with the ontogenetic pattern associated with a retruded midface. Resorption is variably expressed on the anterolateral corner of the mandible which reflected compensatory increments of growth inward, consistent with the downward component of facial growth in *Paranthropus*. Resorption over the labiobuccal surfaces of the mandible

is characteristic for modern humans, indicating that the resorptive character of the *Paranthropus* anterior corpus posteromedially relocates an anteriorly displaced dental arch in order to maintain occlusal relations between the two jaws. There would be, of course, a certain amount of variation in this respect, which reflects the variable extent to which posterior relocation would be necessary.

Indeed, it should be stressed that *Paranthropus* is relatively orthognathic only when it is compared, for instance, to *Australopithecus*. When *Paranthropus* is compared to modern *Homo*, it can be seen that the human face is relatively less protruding, which relates to resorption around the labial alveolus anteriorly to the midline in the lower jaw. In *Paranthropus*, resorptive activity is situated around the canine-premolar region which kept pace with increments of growth inward in this region of the maxilla, but deposits over the upper portions of the maxilla and zygomatic as well as over the anterior pillars resulted in a relatively more anteriorly situated and prognathic profile. Nevertheless, the characteristic development of a high and vertical ramus was in keeping with the development of a relatively orthognathic profile in *Paranthropus* The high ramus reflects the relative increase in posterior facial height discussed above. The verticality of the ramus in this case reflects the downward versus the forward emphasis of facial growth. Thus it may be that resorptive compensation in a posterior direction of the mandibular incisor alveolus (as in modern *Homo*), relative to the degree of maxillary protrusion, was not necessary.

The remaining labial, buccal and lingual surfaces of the mandibular corpus are depository. Deposits over the whole vertical extent of the symphyseal region precluded the development of a chin as it is expressed in modern humans (accounted for by resorptive remodeling in this vicinity: Enlow & Harris, 1964). Buccal increments increased mandibular proportions while lingual accumulations, combined with deposits on the inferolateral margin, also served to relocate the mandible into wider projections according to the "V" principle. In addition, displacement of the *Paranthropus* mandible was compensated by resorption within the posterior rim of the mental foramen. Thus cortical drift posteriorly maintained the foramen in the premolar region throughout the growth period. Finally, it should be noted that addition of bone characterizes the lingual symphyseal surface. This reflected the posterosuperior mode of growth of this region - contrary to early *Homo* and possibly *Australopithecus*.

SUMMARY

The principal objective of this chapter has been to characterize the facial bone growth remodeling processes of early hominids and to apply these results to morphogenetic interpretations of facial growth. New juvenile specimens have come to light since evaluation of specimens listed in Table 17-1, which deserve study, but until then, Figures 17-4 and 17-5 summarize our knowledge of the remodeling patterns characterizing the face of *Australopithecus* and *Paranthropus*.

The pattern of *Australopithecus* facial remodeling, superimposed on facial profiles of the Taung child (Figure 17-4), agrees with preliminary data available for Pan (Table 17-2) and the results of remodeling studies on the growing macaque face.

The study sample of immature early *Homo* craniofacial material is not sufficient to warrant a complete portrayal of their facial remodeling. However, with the available evidence, there is nothing about their remodeling pattern that distinguishes them from *Australopithecus* (Figure 17-4). However, it should be noted that no upper midfacial remodeling results were represented in the study sample.

It may be determined that *Australopithecus* and early *Homo* shared the primitive facial remodeling pattern, as also represented by the extant chimpanzee. Both *Australopithecus* and early *Homo* facial growth were characterised by bone deposits on forward-facing aspects of the face which served to emphasize the forward mode of growth. This was combined with an anteriorly-drifting pterygoid complex, thus allowing the full complement of deposits at the maxillary tuberosity to provide for anterior displacement of the midface. Alterations in the rates of bone displacement and depositional remodeling must have been responsible for their morphological differences, however, as well as accounting for the variable extent to which members of these taxa exhibited a prognathic facial profile (however, see below for a discussion on the possible resorptive remodeling over the early Homo zygomaticomaxillary region). Resorption over the forward-facing aspect of the lateral orbital rim may have been responsible for the notched configuration of the rim as seen in lateral view (Figure 17-4). However, in a preliminary study, only one out of six modern human faces examined illustrated resorption in this region (Table 17-2). This human sample contained only individuals in mid-childhood, thus we might suspect that differential rates of deposition over the supraorbital and infraorbital regions may contribute, together with the possibility of some early childhood or intermittent adolescent resorption, for the retruded lateral orbital rim.

Paranthropus differs from *Australopithecus* and early *Homo* by the occurrence of resorptive fields consistently observed on the anterior wall of the temporal fossa (extending superiorly to include the ophyronic groove), the nasoalveolar clivus and the anterolateral corners of the mandibular corpus during ontogeny (Figure 17-6). The latter two features correspond to remodeling patterns typically associated with modern *Homo* (Table 17-2) and emphasize a downward facial growth vector contributing to relative orthognathy in *Paranthropus*. These remodeling features were combined with marked increases in posterior facial height, a posteroinferiorly-drifting pterygoid complex, a relatively deep posterior palate and an upward rotation of the upper face compensated by an anterior relocation of the upper face above the jaws. An inferiorly-directed facial growth vector was the result and, combined with posterior relocation of the jaws, determined an ontogenetic sequence related to an anteriorly positioned but vertically disposed masticatory system. Resorption over the inferomedial aspect of the orbital wall is indicated to suggest that the downward component of *Paranthropus* midfacial growth might possibly have resulted in medial compensation of the interorbital region. It could also be suggested that the large interorbital width characteristic of *Paranthropus* was an indication that medial relocation did not occur.

The foregoing illustrates how the study of actual growth mechanisms and processes can be used to provide a more complete portrayal of early hominid craniofacial biology. To the extent that phylogeny may be defined as shifts in ontogeny through time, this descriptive ontogenetic data may then also be

Australopithecus		Early *Homo*		*Paranthropus*	
Specimen	Age; Element	Specimen	Age; Element	Specimen	Age; Element
LH 2	3.25yr; M	ER 820	5.3yr; M	SK 438	1.0yr; M
Taung	3.3yr; FS+M	SK 27	5.5-6.0yr; FS	SK 841a	2.0yr; M
Sts 24 + 24a	3.3yr; FS	OH 13	10.9yr; FS+M	SK 64	2.5yr; M
Sts 57	3.4yr; FS	OH 7	6.0-8.0; M	SK 3978	2.5yr; M
Sts 2	3.5yr; FS			ER 1820	2.7yr; M
MLD 2	6.6yr; M			SK 62	3.25yr; M
Sts 52	11.3yr; FS+M			SK 61	3.3yr; M
AL 333-105	FS			SK 63	3.3yr; M
LH 21	FS			SK 66	3.5yr; FS
Stw 59	FS			SK 25	6.6yr; M
				SK 55b	6.6yr; M
				SK 47	7.5yr; FS
				SK 843	7.5yr; M
				SK 13/14	8.0yr; FS
				SK 52	11.3yr; FS
				SK 54	FS

TABLE 17-2.
Six juvenile wild-shot Pan troglodytes and 6 juvenile Protohistoric Arikara people from Mobridge, South Dakota, were evaluated for the last active remodeling state in 6 anatomical regions. A resorption (-) or deposition (+) code is provided for each of the six specimens, within each taxon and in the order of occurrence given above.

incorporated into hominid systematics and phylogenetic reconstructions. Indeed, ontogenetic transformations are data and should support and complement one's views of phylogeny. Further to this it should be stressed that ontogenetic data may test transformation hypotheses and the credibility of claimed synapomorphic (uniquely derived and shared by two or more species of a lineage, reflecting shared ancestry) and symplesiomorphic (shared by two or more species, but representing a primitive condition also shared with their common ancestor) characters. Clearly, the scientific validity of a proposed phylogenetic relationship may be endorsed by its testability.

Considerable morphological evidence has been noted indicating that *Australopithecus africanus* shares many derived character states with *Paranthropus* and that *Australopithecus afarensis* shares many primitive character states with *Homo*. These morphological observations have been used to support the view that *A. afarensis* currently represents the best last common ancestor of all later hominids. because *A. africanus* is already derived in the direction of *Paranthropus*,

One of the many morphological characters that can now be evaluated ontogenetically in these taxa is the exposure of the nasocanine and nasoalveolar

contours of the face in *norma lateralis* (this is of course one aspect of a trait complex). *A. afarensis* has been considered to share this "primitive" feature with *Homo* (e.g., Kimbel et al., 1984). On the basis of the remodeling data gleaned to date and the extreme likelihood that *A. afarensis* facial remodeling resembles that of the extant chimpanzee, we can now propose that this feature in *A. afarensis* is the result of bone deposition over forward-facing aspects of the face to emphasize the anteriorward growth vector of the midface. However, in *Homo sapiens*, we know that bone resorption over much or most of the anteriorly-facing aspects of the midface serves to emphasize the downward growth vector of the face.

Utterly different ontogenetic mechanisms explain the apparent morphological similarity between *A. afarensis* and modern *Homo*. The ontogeny of the modern *Homo* midface may thus be interpreted to represent the uniquely derived character state while designating the morphological feature as a homoplasy; that is, as a trait shared not because of common ancestry, but shared for some other reason. This homoplasy would be due either to the reacquisition of the ancestral condition or the maintenance of the ancestral trait attended by new underlying principles.

It is unfortunate that no early *Homo* specimen has yet been evaluated for its upper midfacial bone growth remodeling. The nasoalveolar region and buccal alveolar bone of the early *Homo* specimens examined were characterised by bone deposits, as were those of *Australopithecus*. This would emphasize an anteriorward lower midfacial growth trajectory. For the midfacial contours to be visible from the side, however, the suborbital region would either have to have been very limited in its forward development relative to the nasoalveolar clivus, while still retaining the depository remodeling pattern (at very low rates), or the suborbital region would have to have been resorptive during growth, this region remaining relatively posterior during successive relocation of the clivus anteriorly. The second alternative would indicate the beginnings of the definitive modern *Homo* facial remodeling pattern.

Of the mature early *Homo*, some may be interpreted to have had such a resorptive zygomaticomaxillary region. Normally, if one has some notion of the species-specific remodeling pattern and variability, it is possible to correlate local remodeling activity with local variations in the bone surface topography (cf. Kurihara et al., 1980). Whilst speculative, the broad hollow observed beneath some early *Homo* suborbital regions may have been the result of a resorptive field occupying this relative location during growth. Other early *Homo* specimens, such as those attributed to *H. rudolfensis*, are equivocal in this regard (although some specimens may conform in this and other ways to the modern *Homo* remodeling pattern).

More than one narrative can be explored, however. For instance, there is a counter argument to the proposal that midfacial ontogenetic differences between *A. afarensis* and *Homo* contradicts the claimed symplesiomorphic morphology. If one could demonstrate, for instance, that certain functional or *Homo*-lineage architectural parameters of the *A. afarensis* and *Homo* midface were the same, then it could be argued that a change in ontogenetic pattern was required to maintain this function and/or empirical architectural relationship (and morphological similarity) and that this ontogenetic change represents an appropriate transformation series (see Chapter 9 for details on facial architectural constraints during development).

Insofar as some enamel microanatomical characters, which adhere to strict genetic controls over tooth development, and that some characters related to mandibular tooth root morphology and the corpus are shared by *A. afarensis* and *H. rudolfensis*, we may suggest that these taxa are linked in an ancestor-descendant relationship. Therefore, alterations in remodeling over the course of human evolution are surmised to have maintained craniofacial architecture in the face of concomitant changes in function, morphology, and development.

18

Craniofacial Imaging

J. Martin Palomo D.D.S., M.S.D.
B. Holly Broadbent Jr., D.D.S.

HISTORICAL PERSPECTIVE AND THE 3D DIGITAL AGE

At the beginning of the 20th century, plaster was the primary material used to capture craniofacial morphology. Almost all practitioners used plaster to make casts of the teeth and alveolar bone. These dental casts, along with a careful clinical examination of the patient, formed the database for craniofacial diagnosis and treatment planning. One particularly ambitious practitioner, Calvin Case, advocated the use of plaster facial moulages to record facial changes before and after treatment (Figure 18-1). Although we tend to think that craniofacial records have steadily improved over the years, one factor that often is not appreciated is that these early records captured a patient's craniofacial morphology in three dimensions (3D). Technical difficulties in obtaining facial moulages and the practical problems of storage prevented most practitioners from adopting such technique. Advances in photography and radiography changed the way practitioners recorded facial morphology. By the end of the 20th century, the combination of two dimensional (2D) radiographs and photographs, along with three dimensional (3D) dental casts was the most common method used to document the patient's morphology.

Over the last 80 years, these basic records have steadily improved in quality, and recently, these records have moved from analog to digital format. This analog to digital conversion of records has not progressed at the same rate for all record types. For example, in January 2004, Eastman Kodak Co. announced that it would stop selling traditional film cameras in the United States, Canada and Western Europe. This was a significant landmark in the transition between film and digital photography. According to estimates by InfoTrends Research Group, global film camera shipments in 2004 shrank to 36 million units from about 48 million in 2003, while digital camera shipments rose to 53 million from 41 million units. A similar trend in digital radiography has also occurred, but at a slower rate. Digital x-ray machines are not mass market items and fewer users results in higher prices. In addition to higher prices, the availability of low radiation dose computed tomography has also slowed the adoption of 2D digital radiography. Dental casts, the oldest 3D record, also have a digital version. Since incorporation in the market

about 10 years ago, 3D digital casts have gained increasing acceptance. However, there still is some resistance to using digital models, especially from practitioners accustomed to the "touch and feel" of plaster. In addition, most of the current options available to generate digital study casts still require dental impressions.

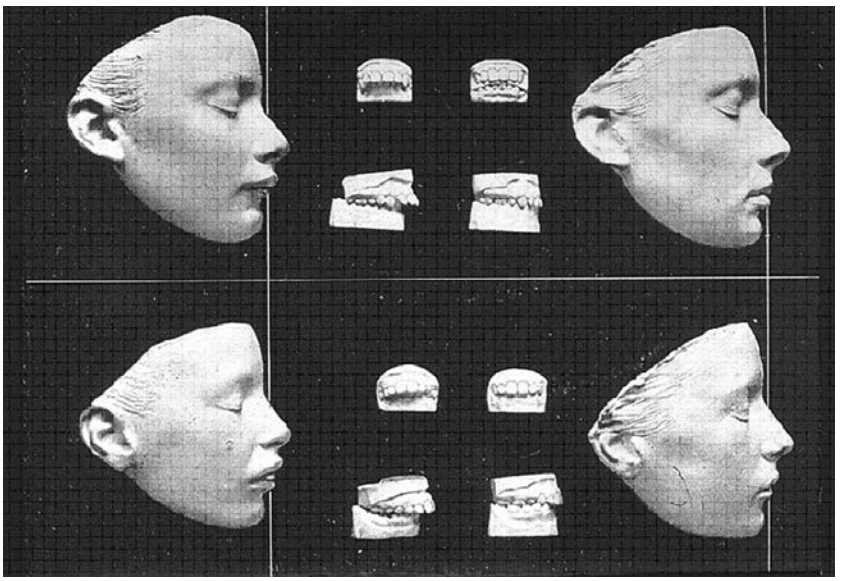


Figure 18-1.
Calvin Case's facial moulages.

All digital records require less physical storage, improve access, conservation, communication, and duplication capabilities, but only 3D images provide additional diagnostic information. Some might ask, "Why do we need a three-dimensional record?" The short answer to this question is that our patients are 3D and, therefore, it takes three dimensions to accurately represent their morphology. In traditional cephalometry, 3D craniofacial structures are projected onto a 2D radiographic film. This process creates cephalometric structures and landmarks that do not exist in the patient. Examples of such structures are the mandibular symphysis, articulare, the pterygoid fossa, and the "key ridge." Although clinicians around the world constantly refer to these structures as anatomic landmarks, they are in fact, artifacts of the cephalometric technique, and cannot be identified on a dry skull. Another problem arises when bilateral structures are averaged to create a unified anatomic outline. An example of this process is the averaging of the right and left inferior borders of the mandible to create the "mandibular plane." Such averaging of bilateral structures creates two problems. First, the "plane" that is created is really a line that is an abstraction based on the anatomy of the patient. Second, averaging the structures results in a loss of parasagittal information, and any true asymmetry of the patient is lost. It is difficult to determine how important this lost information is to diagnosis and treatment planning.

A digital image uses a file extension to identify the file format and allow the software to read the data. Different file formats can be used for a 3D image.

The Digital Imaging and Communications in Medicine (DICOM) standard was created in 1995 by the American College of Radiology and the National Electrical Manufacturers Association (ACR-NEMA) to facilitate the viewing and distribution of medical images, such as CT scans, MRIs, and ultrasound. The DICOM standard has a file extension "dcm", equivalent to the format of a digital picture with extensions such as "tiff", "jpeg", etc. The DICOM standard allows software companies developing imaging applications to concentrate on processing image data rather than on reading a wide variety of proprietary data formats. DICOM allows communication and transfer of images among centers, clinicians, and hospitals. DICOM is the standard for 3D image data. Although some orthodontic imaging software manufacturers use a compressed proprietary file format for some operations, like superimposition, they also provide a conversion or export utility to produce DICOM files.

In this chapter we will divide three dimensional digital orthodontic images into three types: (A) soft tissue face, (B) craniofacial skeleton, and (C) dentition. Importantly, all of these images can be stored, manipulated and transferred as DICOM images.

The Three Dimensional Soft Tissue Face

The most popular methods to capture 3D facial information are 1) the structured light method, and 2) the laser scanning method. The light-based method, also known as the stereo photogrammetric method, is based on one of the primary ways humans perceive shape (Figure 18-2). It uses digital cameras mounted at different angles to provide different views of a subject. All cameras are activated simultaneously, creating views that are combined into a 3D image. Sometimes only one or two of the cameras used is a color camera, and the image from that camera is the color source for the entire 3D image.

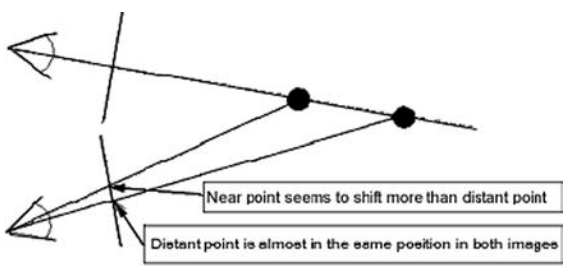


Figure 18-2.
Principle of Sterephotogrammetry.

The number of cameras used in this scanner varies according to manufacturers. For example, the Virtuoso® Shape 3D Camera System (Visual Interfaces, Inc, Pittsburgh, PA) has 6 black and white and 1 color camera, the Face Camera from the same manufacturer has 8 black and white and 2 color cameras, whereas the ShapeWare (Eyetronics, Belgium) has only one camera and one flash device (Figure 18-3). A vertical stripe or a grid pattern is projected on the subject

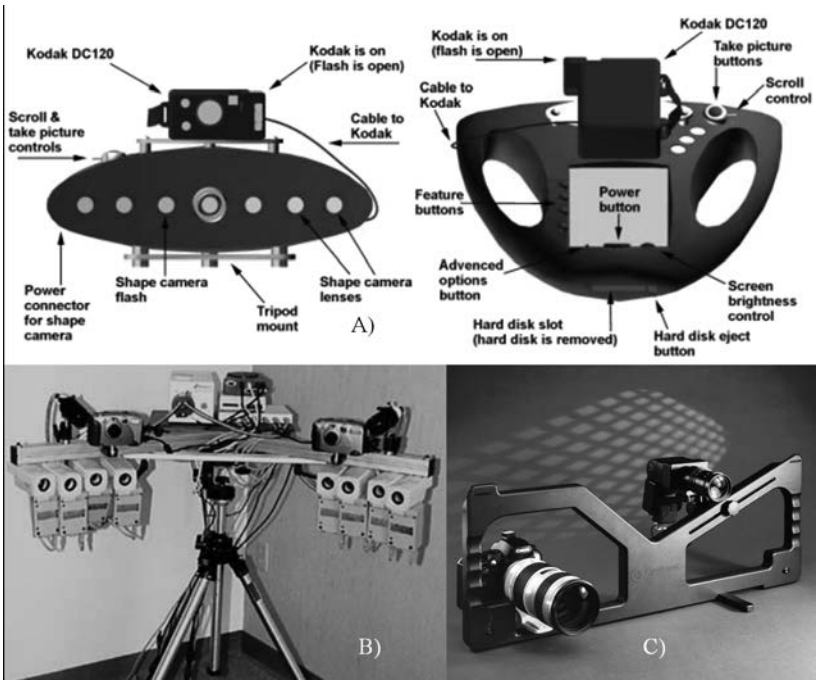


Figure 18-3.
A) Virtuoso® Shape 3D Camera System (courtesy of Visual Interfaces, Inc, Pittsburgh, PA); B) Face Camera (courtesy of Visual Interfaces, Inc, Pittsburgh, PA); ShapeWare (courtesy of Eyetronics, Belgium).

at the moment the image is captured (Figure 18-4). The distortion in this pattern is captured by the shape lenses. These distortions are interpreted as 3D information by the computer software.

Multiple views are then obtained by using multiple cameras systems, or by taking a sequence of pictures when using single camera systems. The multiple views are then manually, semi-automatically, or automatically stitched together to produce a 3D facial model. The alignment is done by designating three or more correspondent landmarks on overlapping images (Figure 18-5). After the alignment, a computer program merges the images, discards duplicate data, smooths the model's edges, blends the colors evenly, and fills holes that may have occurred due to shadows or reflection (Figure 18-6).

The second category of soft tissue face 3D images uses the same principles as light-based systems, however, instead of a light pattern being distorted, a laser pattern is used. And distortions in the laser pattern are interpreted as 3D information. As with the light-based systems, some laser-based systems capture one image at a time while others mount the laser on a revolving arm and capture a series of images in a panoramic fashion (Figure 18-7). All systems usually use an additional camera to obtain color information and texture maps. Since the laser beam is a straight line, laser scanners cannot image undercut and apposed surfaces. Therefore, areas in the shadow of other structures, or other places where the laser beam could not reach, are not captured.

Figure 18-4.
Projection of stripe or grid pattern for shape capture using the Face Camera (courtesy of Visual Interfaces, Inc, Pittsburgh, PA)

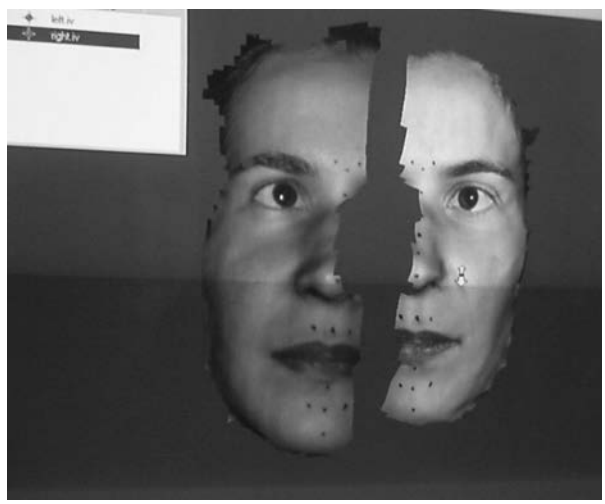


Figure 18-5.
Alignment of 3D facial images taken from different angles. Coincident landmarks seen in both images are used to create a combination of these images. (courtesy of Orametrix, Inc, Dallas, TX USA)

Acquiring dimensionally accurate facial images using either structured light or laser approaches is challenging. Problems include tissue reflectance, interference of hair and eyebrows, change of posture between different takes (when more than one 2D image is combined to create the final 3D image) and movement during imaging (more so with lasers because of longer exposure times). Certain structures like the eyes and ears do not image well due to extreme reflectance and/or undercuts where visible and laser light cannot enter. Image processing software reduces these problems producing relatively accurate images

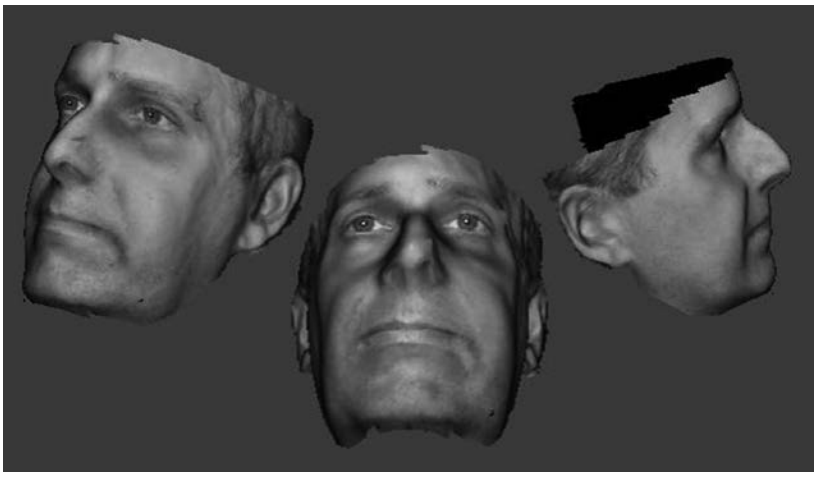


Figure 18-6.
3D facial image taken using the structured light method. The image seen here is a combination of 2 images taken from different angles. A) Image taken with the Shape Camera, B) Image taken with the ShapeWare (courtesy of Eyetronics, Belgium).

The 3D face can be analyzed by using linear measurements, area, perimeter, volumetric and symmetry analysis, all of which can be used for diagnosis and treatment planning, as well as for outcome assessment. Outcome assessments for soft tissue changes, when using photographs, are usually done in the profile view because of radiographic and photo limitations. A 3D model of the face allows a complete evaluation of the treatment outcome, adding valuable information about treatment changes.

To illustrate the capabilities of facial outcome assessment, we used a light-based system to capture 3D facial information simulating a pre- and a post-surgical mandibular advancement procedure. Two images were taken, one with neutral occlusion, and another with protrusive occlusion. Facial landmarks were identified in both images, in order to achieve accurate alignment. The landmarks were chosen

Figure 18-7.
A) The Minolta Vivid Portable laser based scanner; and B) Panoramic fashion laser based scanner (courtesy of Cyberware, Inc –Monterey – CA, USA).

around the orbit, around the nose, and forehead, where changes would not occur. After superimposing both images, two different analyses were performed. In the first one, the protrusive image was converted into a see-through wire frame so we could visualize the changes (Figure 18-8). The second analysis compares both images, turning them into a single image with different color intensity. The further apart the pixels are in the two superimposed images (i.e. the greater the anatomic change) the darker the shade of blue (Figure 18-9). This analysis is called surface metric distance and it shows that the biggest differences between these two images occurred in the anterior midline region. This technique is a simple yet effective way to illustrate differences between images.

Another option available to capture the 3D face is to use a digital photograph of the patient's face to add color to the soft tissue information obtained from a 3D CBCT scan of the patient. This technique makes the image appear more life-like and does not require the purchase of additional hardware (Figure 18-10). Using a 2D digital photo to colorize the 3D image may not provide all capabilities of a true 3D face image, but it does offer a low-cost solution for clinicians who want more visually attractive computed tomography facial images. However, since CT uses ionizing radiation to create the image it is not the best choice for clinicians who only need 3D images of the soft tissue face.

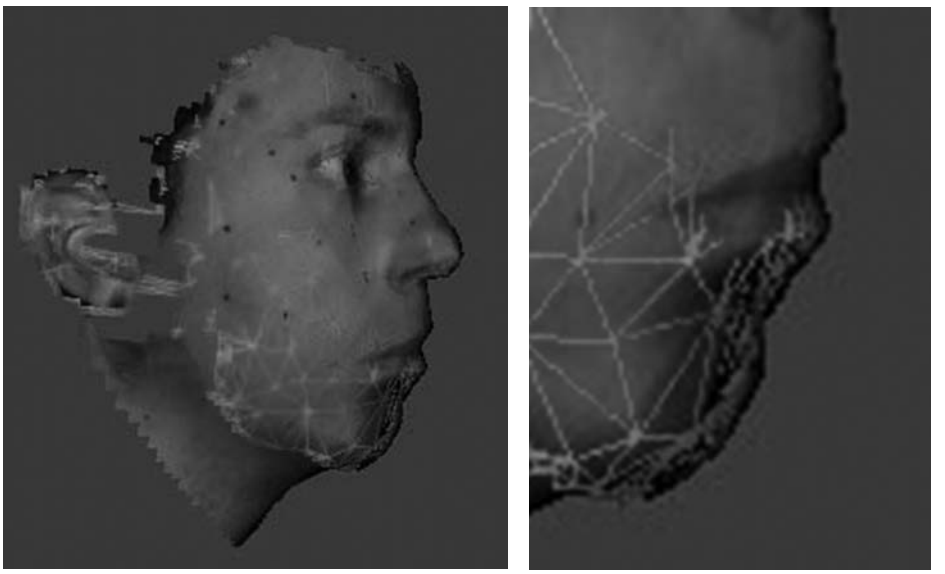


Figure 18-8.
Superimposing of Pre and Post mandibular protrusion. A) The protrusive image was converted into a see through wire frame. B) Close up on the chin area showing difference in mandibular position.

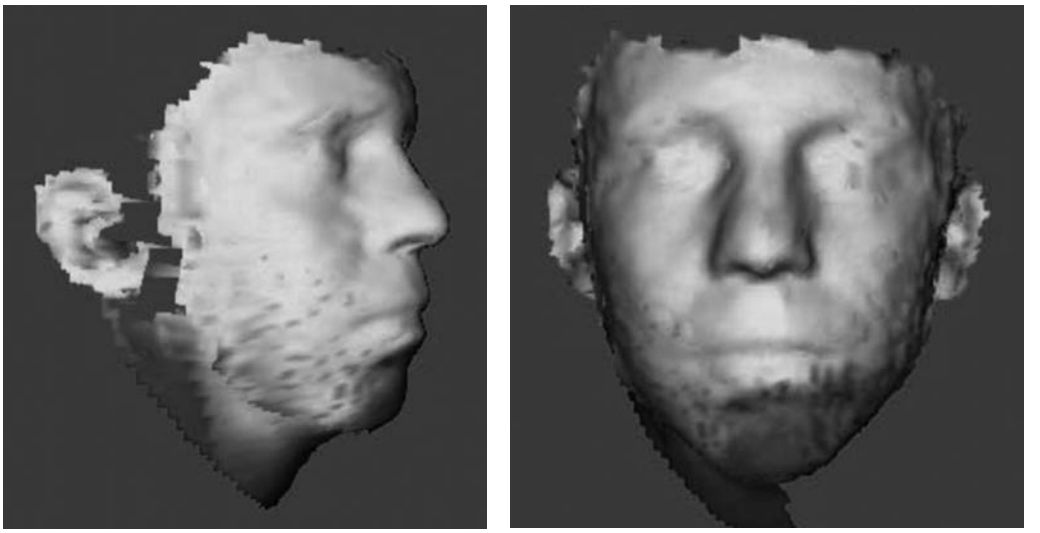


Figure 18-9.
Surface metric distance analysis comparing a Pre and Post 3D facial images. A) Profile view of a superimposition of pre and post mandibular protrusion. The darker the shade of blue, the further apart are the pixels, hence the more change is noted. B) Frontal view of the same superimposition. C) Superimposition of pre and post orthodontic treatment which featured extraction of four first bicuspids. Note the profile assessment available with this particular program (courtesy of 3dMD – Atlanta, GA).

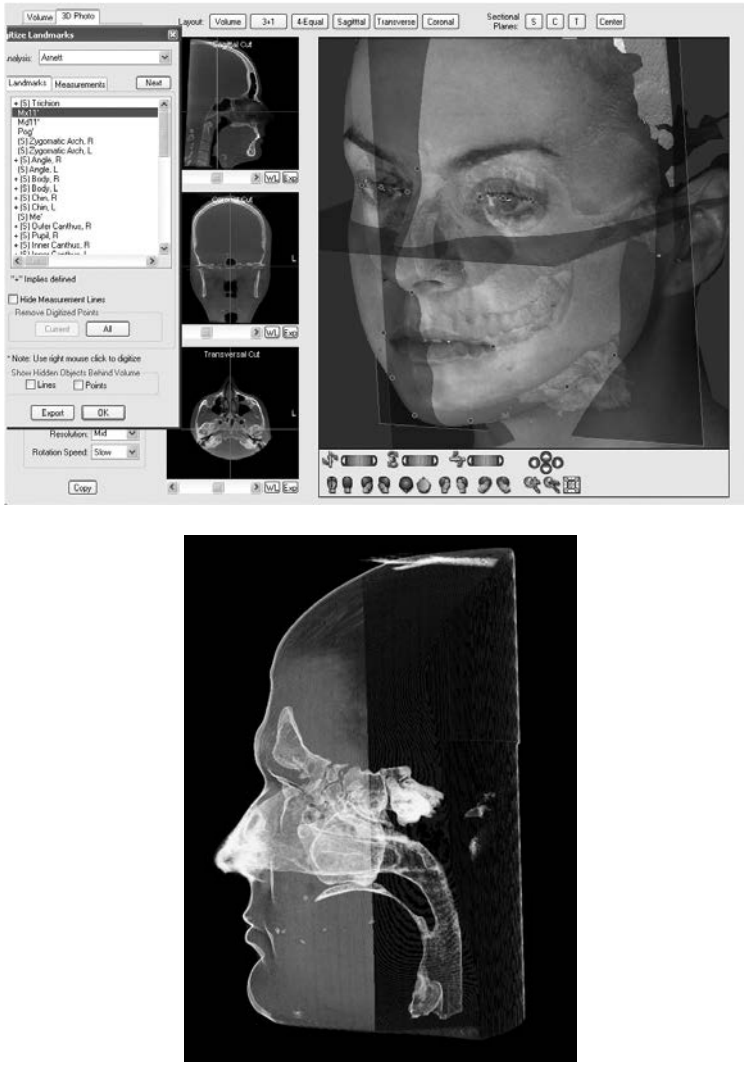


Figure 18-10.
A) Incorporation of a true 3D scan of the face to the CBCT data (courtesy of Dolphin Imaging – Chatsworth, CA). B) Combination of an extra-oral frontal photograph to a CBCT image. The extra oral photograph is converted to a 3D mode using proprietary technology. This combination adds the extra-oral color information to the CBCT volume and allows both images to be saved as a single record (courtesy of Anatomage Inc. – San Jose, CA).

Current technology is available for the collection of 3D representations of the human face. Different methods and systems are available, and all seem to do a fairly accurate job recording the face as a time record. Diagnosis, treatment planning and outcome assessment analyses are possible by using the 3D face. Furthermore, the use of 3D images would give additional information beyond what is currently used in the clinical environment. At this point, the cost of the hardware and software, as well as the lack of evidence of clinical utility, are the major obstacles preventing widespread use of this technology in patient care.

The Three Dimensional Craniofacial Skeleton

Since 1931, lateral and frontal cephalometric films have been used to study longitudinal changes in the craniofacial skeleton. Limitations of this technique include loss of parasagital information, projection enlargement and superimposition of bilateral anatomic structures.. The need for three-dimensional cephalometry was recognized in the 1960s by Savara, and has been confirmed by many in the literature. Altobelli specifically called attention to the lack of three-dimensional standards for pediatric and adult craniofacial patients. Dean and Hans noted that normative 3D cephalometric data would be an important tool in the study of craniofacial variation, as well as diagnosis, treatment planning, stereotactic treatment, prosthetic and appliance design, and outcome assessment. Various manual techniques for abstracting three-dimensional coordinate data from biorthogonal head films have been developed. Until recently, this work remained impractical because of the time-consuming nature of pencil tracing of films and computer mouse-based landmark identification from tracings. The use of computers and digital radiographs makes the creation of a 3D image from 2 different 2D views of the same subject less labor intensive, but still not sufficiently practical and user-friendly for routine clinical use. The importance of computer-based cephalometry was long ago recognized by Ricketts, who wrote: "Cephalometrics, when computerized, becomes the most powerful tool of information yet devised for the practicing clinician".

Indirect Methods

Both indirect and direct methods can be used to generate 3D representations of the craniofacial skeleton. Indirect methods use the principles of stereometry and combine two radiographs with different views of the same object to create a 3D image. To create an accurate image using this method requires: 1) the availability of homologous landmarks, 2) the knowledge of the enlargement used, 3) no movement of the patient's head while taking of both radiographs, and 4) knowledge of distances between x-ray cassettes and the head. The Bolton cephalometer uses two x-ray heads to allow the lateral and frontal headfilms to be taken without moving the patient's head. Most other cephalometers have only one x-ray head and require repositioning of the patients head to take the lateral and frontal radiographs. For this reason, most lateral and frontal cephalometric pairs cannot be used to generate three dimensional data.

In 1975, Broadbent et al. introduced the Broadbent Orientator. The Orientator uses the information obtained from biorthogonal plane film radiographs to create 3D data points. In order to use the Orientator to acquire 3D data, one must assume that the beams of the posterior and lateral tube heads orthogonally intersect in the center of the head (Figure 18-11). Manual use of the Broadbent Orientator for 3D data collection is cumbersome and, 3D data collection although possible, was not routinely attempted. Recent advances in computer graphics allow easier collection and interpretation of 3D data, using a computerized version of the original Broadbent Orientator. The Broadbent cephalometer is equipped to

adjust to all necessary requirements, and it has been used on several occasions to create 3D images, but almost exclusively for research purposes (Figure 18-12).

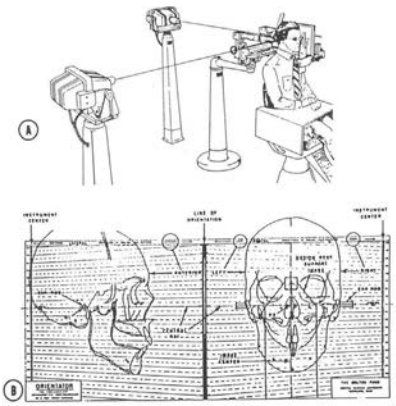


Figure 18-11.
A) Broadbent-Bolton cephalometer, B) Broadbent Bolton Orientator. Illustrations © Bolton-Brush Growth Study Center, reprinted with permission.

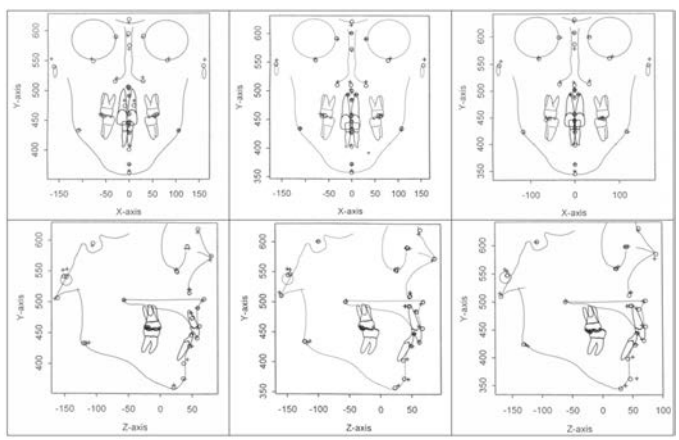


Figure 18-12.
Procrustes Fit of landmarks comparing Class II ("o") to Class I ("+") for ages: 06, 11, and 15 in the frontal and the lateral view.

Direct Methods-MRI and CT

Three dimensional images of the craniofacial skeleton can be directly acquired using either Magnetic Resonance Imaging (MRI) or Computed Tomography (CT). Magnetic Resonance Imaging (MRI) has been available for many years. However, the high cost and large size of the units usually means that they have to be located in hospitals or medical imaging centers. Since most dental images are taken in the dental office, the use of MRI in dental medicine has been limited to the temporomandibular joint and airway. A big advantage of MRI over CT is that it does not use ionizing radiation yet allows for dynamic 3D imaging.

With increasing concern about the effects of ionizing radiation on human health, MRI may play a larger role in craniofacial imaging in the future.

Computed tomography (CT) was developed by Nobel Prize winner Sir Godfrey Hounsfield. Since its invention, the CT scanner has had over 6 significant changes in the way the data is captured and the amount of radiation necessary for the scan. The Cone Beam Computed Tomography (CBCT) or Cone Beam Volumetric Tomography (CBVT) method is the latest generation of CT technology. Craniofacial CBCT was developed to counter some of the limitations of earlier generations of CT scanning devices and to make 3D technology practical for dental medicine. The radiation source consists of a conventional, low-radiation x-ray tube, and the resultant beam is projected onto a panel detector, producing a more focused beam and considerably less scatter radiation compared to the helical CT devices. The total radiation is approximately 20% of that of a helical CT and can be equivalent to the exposure dose received during a full-mouth periapical series.

The innovations mentioned above allow the CBCT unit to be less expensive and smaller in size than a traditional CT machine. When compared to earlier generation CT scanners, CBCT is more sensitive and more accurate, requires less radiation, captures the maxilla and mandible in a single rotation of the x-ray source, and is more cost-effective for patients. Another advantage of the CBCT technology over earlier generations of CT scanners such as helical CT is the low level of metal artifacts in the image. An image taken with helical CT of an area close to a metallic restoration, a crown, or an implant is very difficult to analyze and diagnose because of the artifacts and distortions that the presence of the metal creates. This is a major limitation in the use of helical CT images, since many patients have metal present in their mouths. With CBCT technology, the area around the metal presence is usually of diagnostic quality. CBCT offers surface as well as radiographic view modes. The latter are similar to traditional radiographs familiar to dental practitioners.

Currently, there are several commercially available CBCT scanners designed for imaging craniofacial and dental structures. These scanners differ in image receptor type (Charge Coupled Device or Amorphous Flat Panel), field of view, scan time, and overall scanner size/weight (Table 18-1). A CBCT provides more diagnostic information than a panoramic x-ray, full mouth periapical series, lateral and frontal cephalograms, and occlusal radiographs combined. And all of the above mentioned views can be generated from a single ten second scan. Clinicians are also able to generate additional views that are impossible to obtain with traditional radiography (Figure 18-13).

For all above reasons, CBCT is becoming increasingly popular. The resulting images are user-friendly and provide far more information than conventional 2D radiographs. Both skeletal and soft tissue anatomy is captured and can be displayed together or separately (Figure 18-14). Axial, sagittal, and coronal "slice-by- slice" images are also created along with reference lines that make location of these slices less complicated. For example, even when observing only the coronal view or a small segment of a complete image, lines in the sagittal slice view indicate the height and position of the slice or object being analyzed (Figure 18-15).

CBCT is digital by nature and uses a computer program to construct a 3D volume from a series of 250 to 300 2D images. With the 3D format, new terminology is used. For instance, voxel (volume element) is used instead of pixel (picture element), since it is referring to volume and not to a 2D space. Other terms used for 3D images are "region of interest", abbreviated as ROI, and "field of view" abbreviated as FOV. The ROI is the 3D region that the clinician wants to evaluate. For example, when asking for a periapical radiograph of the mandibular incisors, the ROI is that incisor area. The FOV is the area captured during the scanning session. The resolution of an image is related to the size of the field of view (FOV), which is the resulting size of the image (Figure 18-16). For example, if the clinician wants to visualize a cyst in the mandibular incisor area, and a large FOV that includes the entire head is used, the observer must zoom to the ROI, and the image quality will not be as good as a FOV that was focused only in that incisor area. This concept is similar to what occurs for digital photography. If the clinician wants to see a central incisor in detail, a good starting point would be an intraoral picture of the target area, not the full smile. In the latter scenario, zooming in would make the incisor appear fuzzy, indicating poor resolution. Although the technical resolution in CBCT images ranges from 0.1 to 0.5 mm, in clinical use it is difficult to resolve two structures of similar density unless they are separated by 0.6-1.0 mm. (Ballrick et. al. 2008, Palomo et. al 2007).

Table 18-1.
Specification of the currently available cone beam CT machines approved for use in dentistry.

Trade Name	NewTom	i-CAT	CB MercuRay	Accuitomo	ILUMA	Galileos	ProMax 3D	Dental CBCT
Manufacturer	Quantitative Radiology, Verona, Italy	Imaging Sciences, Hatfield PA, USA	Hitachi Medical Co. Tokyo, Japan	J Morita Kyoto, Japan	IMTEC Ardmore OK, USA	Sirona Dental Systems Bensheim, Germany	Planmeca Oy Helsinki, Finland	Yoshida Dental Co. Tokyo, Japan
Weight	1058 lb	425 lb	2094 lb	882 lb	770 lb	308 lb		
Tube Voltage	110 kVP	120 kVP	60-120 kVP	60-80 kVP	120 kVp	85 kVp	50 – 84 kVp	90 kVp
Tube Current	2-6 mA	3-8 mA	10-15 mA	1-10 mA	4 mA	5-7 mA	0.5 – 16 mA	4 mA
Scan Time *	36	10-40	9.6	18	10-40	14	18	19-37
Detector Type	CCD	Flat panel	CCD	CCD	Flat panel	CCD	Flat Panel	Flat Panel
Grayscale	12 bit	14 bit	12 bit	12 bit	14 bit		12 bit	
Field of View (H x D)	6" 9" 12"	6.7 x 5" (regular) 8.6" x 5" (expanded)	6" 9" 12"	1.2 x 1.6"	OrthoCAT: 7.5"x7.5" DentalCAT: 4"x 6.7"	6" x 6"	3 x 3" 2 x 3" 2 x 1.5"	3 x 3"
Voxel Size	0.2 – 0.4 mm	0.2 – 0.4 mm	0.18-0.38 mm	0.13 mm	0.1 – 0.4 mm	0.15-0.3 mm	0.16 mm	0.1-03 mm

* Scan time is how long the machine takes to take an image (in seconds), and does not represent exposure time. For example, in the NewTom even though the scan time is 36 seconds, the actual exposure time is 5.4 seconds.

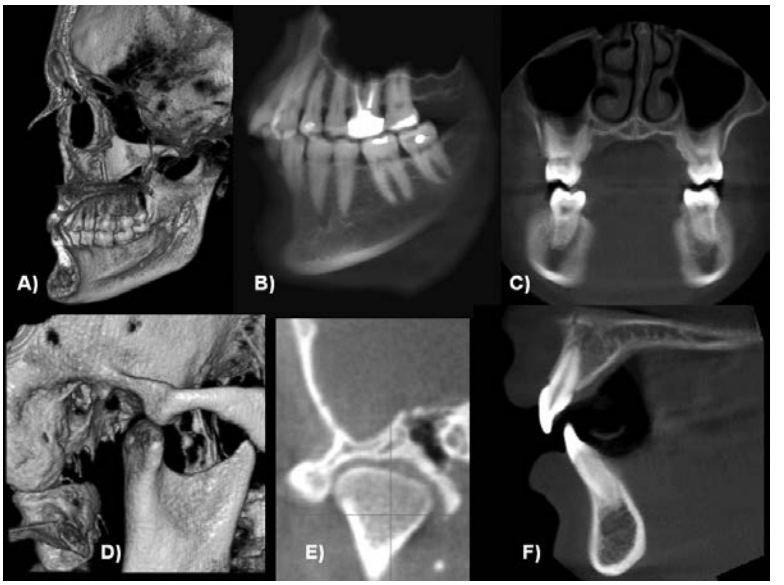


Figure 18-13.
Images taken with a CBCT scanner. A) Surface view of the inside of a subject's right side; B) Slice view of the dentition, providing a more complete periapical view; C) Coronal view of the molars in occlusion, showing the Curve of Wilson; D) Surface view of the TMJ complex with the mouth opened; E) Slice view of the TMJ complex, showing the condyle; and F) Sagittal view of the overjet in the slice mode.

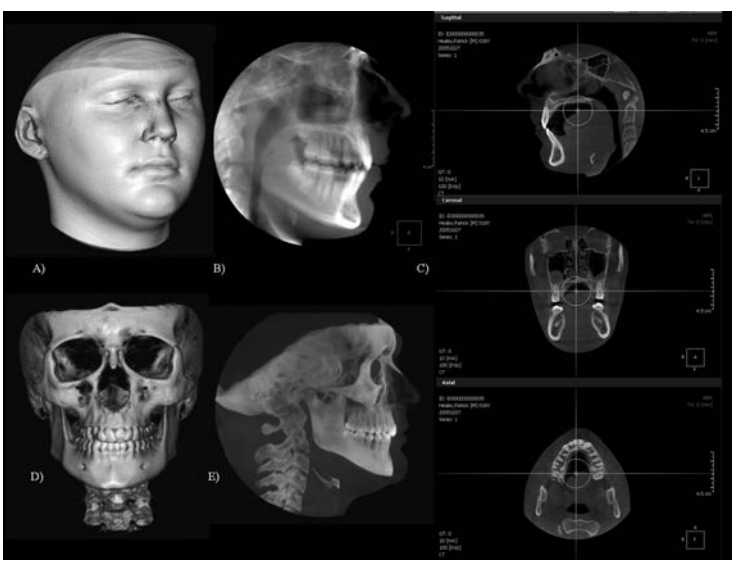
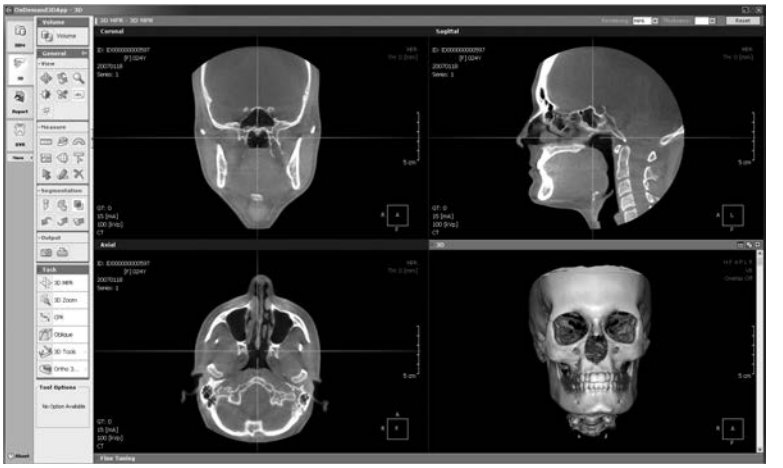


Figure 18-14.
There are several possible ways to visualize a CBCT image. A) soft tissue view using the surface mode, B) radiographic mode, in which the 3D image is turned into 2D as in a traditional radiography, C) slice view, divided by sagittal, coronal, and axial view, D) skeletal rendering using the surface mode, and E) maximum intensity projection (MIP) mode which provides a 3D radiographic view.



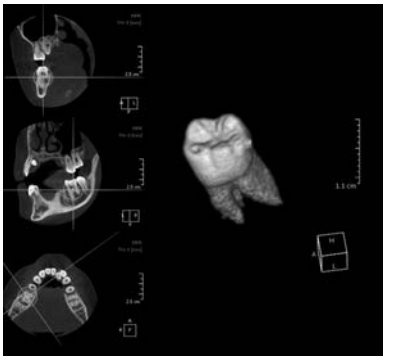


Figure 18-15.
A) Regular interface of a DICOM viewer program showing both slice view and 3D surface mode (OnDemand – Cybermed Inc – Torrance, CA). B) By using a combination of sculpting and segmentation we are able to view a single tooth and study surface that cannot be viewed either clinically or with traditional radiography, since there would be adjacent teeth present. Note the reference lines showing the area being analyzed.

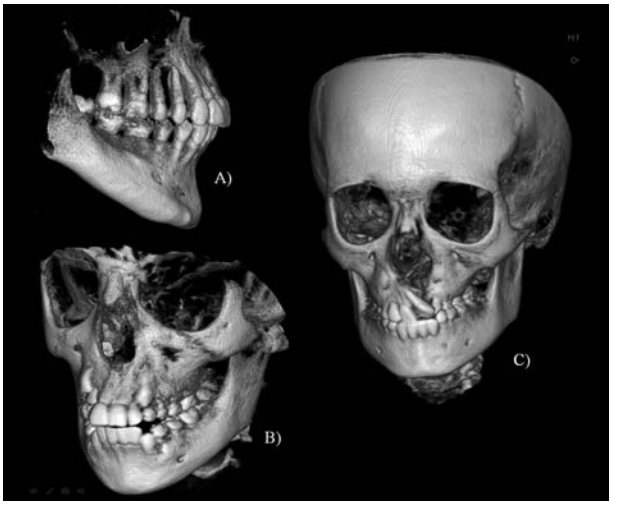


Figure 18-16.
The 3 field of views available for the Hitachi CB MercuRay scanner (Hitachi Medical Systems – Twinsburg, OH). A) The small field of view (6") usually show the whole dentition of both arches, and sometimes the condyles; B) the medium field of view (9") consistently shows the condyles and most of the mid and lower face; and C) the large field of view shows most of the craniofacial structures.

Clinical Applications of CBCT in Dental Medicine

The ultimate goal of craniofacial imaging is to answer clinical questions. CBCT image data can be processed with software tools to produce diagnostic quality periapical radiographs, panoramic radiographs, cephalograms, occlusal radiographs, and TMJ images. In addition, CBCT image data can also genereate views that cannot be produced with regular radiographic machines such as axial and cross-sectional views.

Following are some of the possible clinical uses of the CBCT technology:

Impacted Teeth

Impacted maxillary cuspids have been reported to be distributed as 85% palatal and 15% buccal. The tube shift method has traditionally been used to locate the position of these cuspids and provides an approximation of the level of difficulty associated with the management of these teeth. This method is labor intensive. The use of CBCT has proven useful in the management of patients with impacted teeth (Figure 18-17). The CBCT allows for a more precise analysis of the extent of the pathology related to the ectopic tooth. Clinical reports using 3D imaging have shown that the incidence of root resorption of teeth adjacent to impacted teeth is greater than previously thought. CBCT images can be used to locate the precise position of ectopic cuspids and to design treatment strategies that result in less invasive surgical procedures. CBCT has been shown to outperform conventional radiography in diagnostic accuracy when used by both orthodontists and radiologists. This increased accuracy allows for less invasive surgery, smaller incisions/more conservative flap design, better prognosis, and overall reduced morbidity associated with the surgery (Figure 18-18).

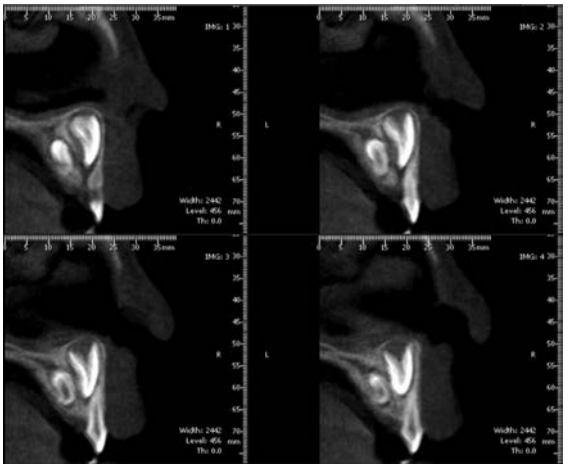


Figure 18-17.
CBCT images of a patient with an impacted supranumerary tooth. A) Anterior view of the maxilla in the radiographic mode, B) View of the right haft of the maxilla in the radiographic mode, C) Surface view of the anterior right segment of the maxilla, D) Anterior view of the maxilla in the surface mode, and E) Occlusal view of the maxilla in the radiographic mode.

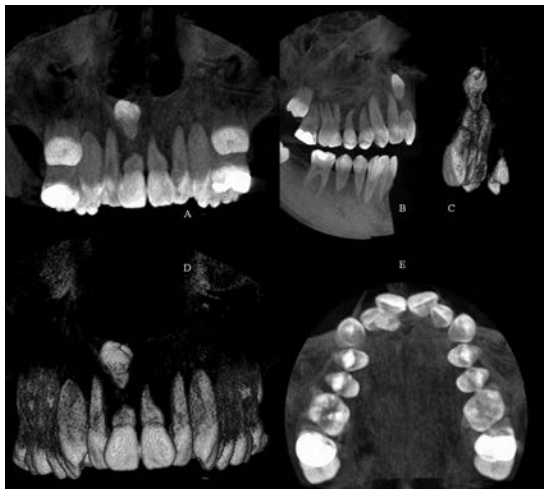


Figure 18-18.
Sagittal views using CBCT technology of a patient presenting with supranumerary teeth in upper central area. The image clearly shows lack of contact and damage from part of the supranumerary teeth. The image also shows that access through the palate would probably be an efficient approach without the need for removal of the deciduous teeth.

Airway Analysis

CBCT technology provides a major improvement for evaluation of the airway, allowing for 3D and volumetric determinations (Figure 18-19). Airway analysis has conventionally been performed using lateral cephalograms. Three-dimensional airway analysis is useful for the understanding of more complex conditions such as obstructive sleep apnea (OSA) and enlarged adenoids. CBCT has demonstrated significant differences in airway volume and the anteroposterior dimension of the oropharyngeal airway between OSA patients and gender-matched controls.

Implant Planning and Bone Quality Assessment

Implantologists have long appreciated the value of 3D imaging. Conventional CT scans are used to assess the osseous dimensions, bone density, and alveolar height, especially when multiple implants are planned (Figure 18-20). Locating landmarks and anatomy such as the inferior alveolar canal, maxillary sinus, and mental foramen occurs more accurately with a CT scan. The use of the third dimension has improved the clinical success of implants and their associated prostheses, and led to more accurate and esthetic outcomes.

With CBCT technology both the cost and effective radiation dose can be reduced. CBCT has been in use in implant therapy and may be employed for the clinical assessment of bone graft quality following alveolar surgery in patients with cleft lip and palate. The images produced provide more precise evaluation of the alveolus. This technology can help the clinician determine if the patient should be restored or if teeth should be moved orthodontically into the repaired alveolus.

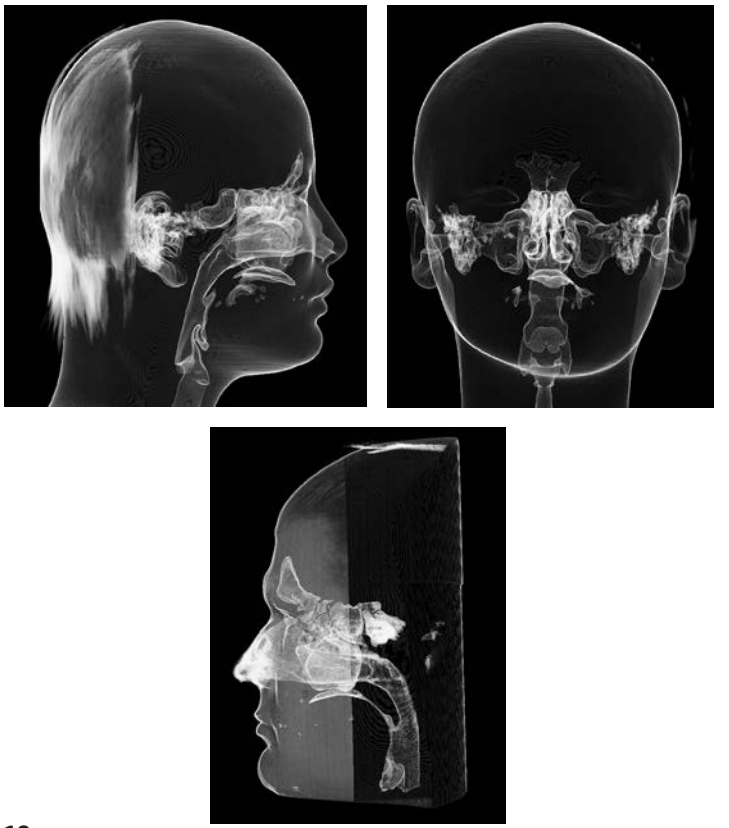


Figure 18-19.
Analyzing the airway with only a lateral film would not be able to identify a possible airway lateral constriction abnormality. A) Lateral and B) Frontal view of the airway by using CBCT. The airway can be segmented and analyzed volumetrically in three dimensions. C) Close-up of the airway in a surface mode (Courtesy of Anatomage Inc. – San Jose, CA).

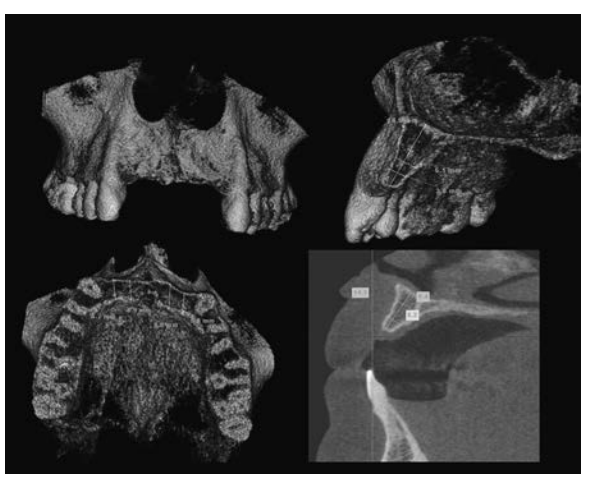


Figure 18-20.
Alveolar height and width assessment for the placing of dental implants. The CBCT image gives a true 1:1 three dimensional representation of the patient.

Location of Anatomic Structures

Anatomic structures such as the inferior alveolar nerve, maxillary sinus, mental foramen, and adjacent roots are easily visible using CBCT (Figure 18-21). The CBCT image also allows for precise measurement of distance, area, and volume. Using these features, clinicians can feel confident in the treatment planning for sinus lifts, ridge augmentations, extractions, and implant placements.

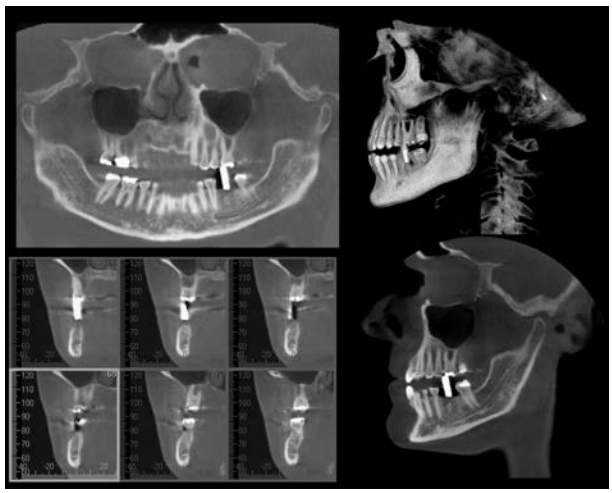


Figure 18-21.
Images produced from a single exposure for the purpose of dental implant planning. The images selected here are panoramic and cross sectional views with the mandibular nerve marked, as well as a surface and radiographic (maximum intensity projection) view with the stent in place Image courtesy of Hitachi Medical Systems of America Co.– Twinsburg, OH).

Temporomandibular Joint (TMJ) Morphology

CBCT imaging of the temporomandibular joint has been evaluated and compared to other methods. Results indicate better imaging with CBCT compared to traditional radiography and helical CT. The CBCT showed greater sensitivity and accuracy than the helical CT in the identification of mandibular condyle abnormalities. Condylar resorption is reported to occur in 5% to 10% of patients who undergo orthognathic surgery. CBCT images provide high diagnostic quality (Figure 18-22) with lower patient radiation exposure as compared to conventional CT techniques, therefore, CBCT should be considered as the imaging technique of choice when investigating bony changes of the TMJ.

Radiation Exposure

sCone beam uses up to 4 times less radiation than a conventional CT to produce 3D volumetric images. The effective radiation dose depends on the settings used (kVp and mA), collimation, and the exposure time. The use of lower mA and/or collimation are some of the ways to reduce the amount of radiation, but reducing

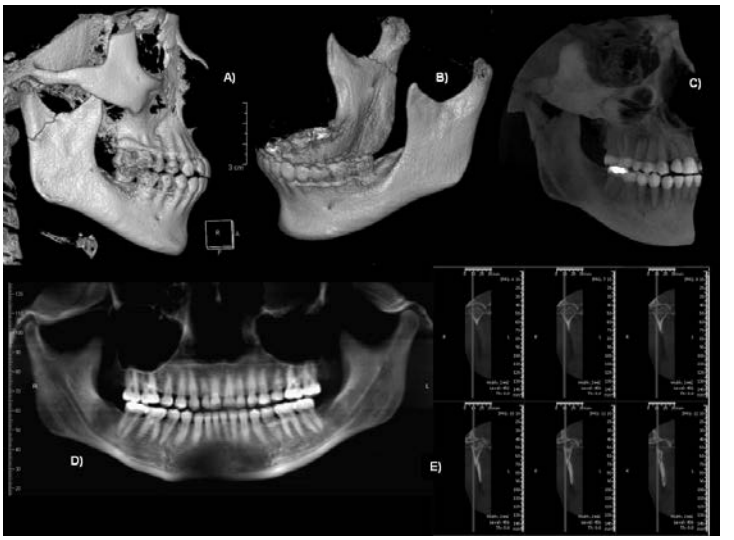


Figure 18-22.
Different possible views of the TMJ complex by using CBCT. A) and B) surface mode, C) Radiographic mode, D) close up of the radiographic view, and E) cross sectional in the radiographic mode.

radiation dose may affect the image quality. Radiation exposure from a typical full head CBCT scan has been reported to be as low as 45 μSv (micro-sievert, SI unit for ionizing radiation) to as high as 650 μSv. For comparison, radiation exposure from a full-mouth series of analog radiographs has been reported to be 150 μSv. and an analog panoramic radiograph exposes patients to about 54 μSv . It is important to remember that there are non-clinical sources of x-ray exposure. For example, a roundtrip airplane flight from Paris to Tokyo exposes each passenger to an effective dose of 139 μSv. In 2001, a report associating the use of conventional CT in children to radiation-induced cancer resulted in CTs being adjusted downward to have an effective dose ranging from 2,600 μSv to 6,000 μSv. Even at the highest settings possible, none of the CBCT units will provide anything near that dose.

The ADA (American Dental Association) Council on Scientific Affairs recommends the use of techniques that would reduce the amount of radiation received during dental radiography. Known as the As Low As Reasonably Achievable (ALARA) principle, this includes taking radiographs based on the patient's needs (as determined by a clinical examination), using the fastest film compatible with the diagnostic task, collimating the beam to a size as close to that of the film as feasible, and using leaded aprons and thyroid shields. An accepted ratio between exposure and image quality can be reached in order to apply the ALARA principle, as well as a good match between region of interest (ROI) and field of view (FOV).

CBCT is capable of imaging hard-tissue and most soft-tissue structures, however, this technology cannot precisely map muscles and their attachments, or show tendons and vessels. These structures would have to be imaged using magnetic resonance imaging technology.

THE THREE DIMENSIONAL DENTITION

Digital study casts have not been as widely accepted from its beginning as the other digital records. The patient's record representing the dentition has traditionally been the only 3D record used. The use of a digital representation of the dentition currently gives as much information as a plaster dental cast would, with some added benefits, but some clinicians do not want to give up the ability to "feel" the occlusion and maneuver the dental casts in different positions.

Invisalign® (Align Technology, Inc. Santa Clara – CA, USA) probably played a significant role in familiarizing clinicians with digital dental casts. In order to offer Invisalign treatment, the clinician has to work with digital dental casts. Align Technologies, in a sense, helped make the transition easier.

Converting the dental casts to a 3D computerized image yields no proven additional diagnostic and treatment planning information, but there are several advantages of a computerized 3D dental cast. Superimposition of pre- and post-treatment lateral cephalograms is an accepted and currently used method of outcome assessment. A superimposition of pre- and post-treatment casts can only be performed by using digital 3D images. Using the ClinCheck® with Invisalign® models, it is possible to use superimpositions of pre and post treatment images, in order to aid in treatment planning decisions (Figure 18-23).

In the workshop report by Hans in 1993, it was agreed that the space required to store models was a universal problem for all clinicians. The digitization of the dental cast allows for easy storage, fast access, and conservation of the dental casts. This saves physical space, time, conserves the records in an intact form, and for longer time, since old records would not need to be trashed due to storage needs. The digitization of the dental casts allows measuring, dental cast analyses, and provides views that would not be possible without destruction of the dental cast (Figure 18-24).

A digital image of the dentition can also be "printed" creating a "physical" dental cast. These options give the clinicians the advantages of both digital and analog worlds. Digital 3D dental casts can be produced either indirectly or directly. The indirect method requires an accurate dental impression with alginate or polyvinyl siloxane. The 3D digital dental cast can be produced by scanning the impression, or scanning the poured cast resulted from the impression. The scanning of the dental cast can be either destructive or non-destructive. Destructive methods involve the removal of a thin layer of material, alternating with image capture to generate a stack of images that are rendered in 3D. Non-destructive methods involve the use of a laser based system with a multi axis robot to obtain several perspectives of the plaster model that are combined to form a complete 3D model. Another approach to non-destructive methods includes the use of Micro CT to image the dental cast or impression.

A direct method of producing 3D digital images of the dentition is made possible by using a scanner to capture both dental shape and information. Orametrix (Orametrix, Inc – Dallas, TX) uses a structured light intraoral scanner (Figure 18-25) to directly produce a 3D image of the dentition. After isolating the dentition and application of an opaquing agent, small images of the dentition are

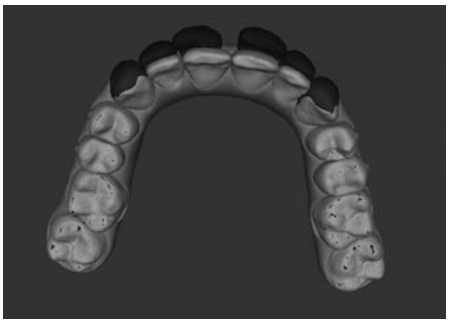


Figure 18-23.
Superimposition of digital 3D dental casts using Align Technology's ClinCheck®.

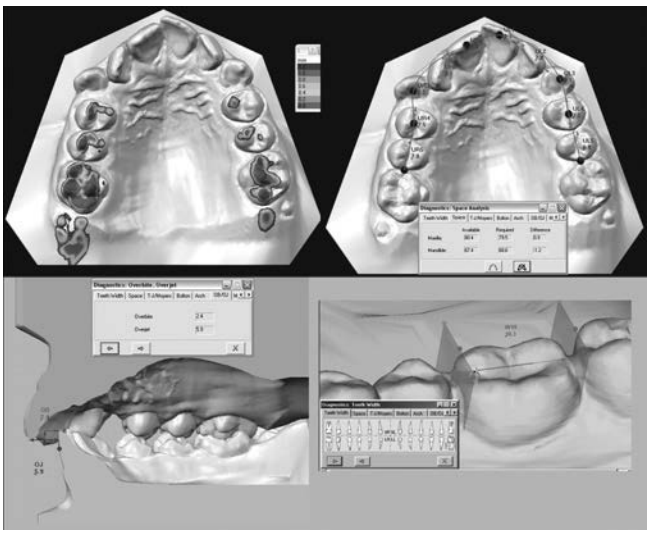


Figure 18-24.
Analysis and measurements performed in a digital dental cast including analyses of contact points, cross section views, and measurements on a digital dental cast (Courtesy of OrthoCad/Cadent– Carlstadt – NJ, USA).

Figure 18-25.
The Orametrix intra oral scanner (courtesy of Orametrix, Inc Dallas – TX, USA.

taken with a video camera while a light pattern is projected onto the teeth. The images are streamed to a computer where they are registered. The complete dental arch is imaged in approximately 90 seconds. A clear advantage of this method is the elimination of the impression and pouring/trimming needs. Nevertheless, according to published reports, the contact points between teeth do not image well, and segmenting the teeth can be challenging.

A more recent method for direct capture of the dentition in 3D is by extracting the dental information out of a CBCT scan. The process is a service provided by a company (Anatomage Inc. – San Jose, CA) which by using segmentation of tissues with different density values, is able to isolate the teeth. The teeth can then be saved as a separate 3D image, which can be seen and moved like a digital dental cast. One important difference from all the other available methods is that it also provides root information (Figure 18-26). The ability to see the root morphology and location is a major diagnostic addition to the dental record. Never before, either in plaster or digital format, was this information available. Another advantage is that by using this method, the radiographic and dental records are taken at the same time, in less than 5 minutes, without the need for a lab, impressions, or anything to be inserted inside the patient's mouth.

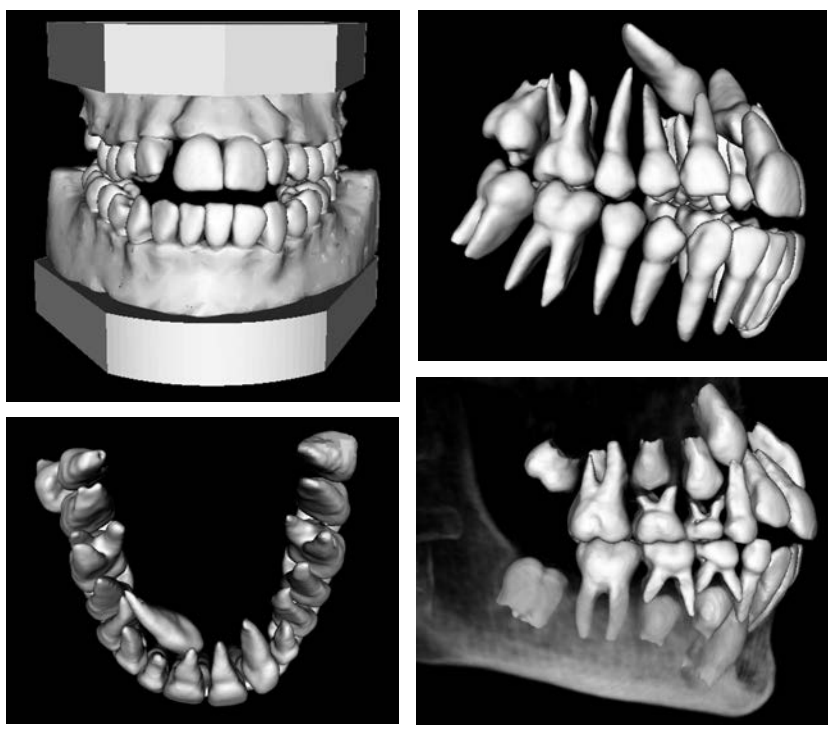


Figure 18-26.
An electronic dental cast can be generated from the CBCT scan, with the advantage of having root information and location. Different views are possible, and even the A) traditional plaster cast look can be achieved; B) Lateral view of dentition only; C) View of the dentition from the top; and D) View of the dentition in conjunction with the skeletal tissue (courtesy of Anatomage Inc. – San Jose, CA).

Printed Models and Holograms

As mentioned before, a 3D image of a 3D object is clearly the most accurate representation possible. Using a computer screen gives the operator the ability to rotate the image and see it from different angles, but it can be considered as a two-dimensional way of visualizing a 3D image. The depth information of the object can be captured by measurements, but not visually. The possible ways of seeing a 3D object in 3D space are either printing the actual image in 3D, or as a hologram.

A direct digital image can be printed in 3D by using stereolithography. Stereolithography is a process for creating three-dimensional objects using a computer-controlled laser to build up the required structure, layer by layer, from a liquid photopolymer that solidifies. This technology allows the creation of 3D models out of CT based images (Figure 18-27). These models can be useful for treatment planning and surgical simulations, and provide a 3D representation of the patient that can be held and seen in 3D space. The same technology also allows the creation of appliances like retainers, functional appliances, aligners, etc based on the CBCT information, without the need for an impression or a patient visit.

Another way of seeing a 3D image in 3D space is by using holographic technology (Figure 18-28). This true holographic perspective may allow surgeons to be more efficient, more precise, and more confident during pre-surgical planning, in the operating room, and for post-surgical assessment and follow-up.

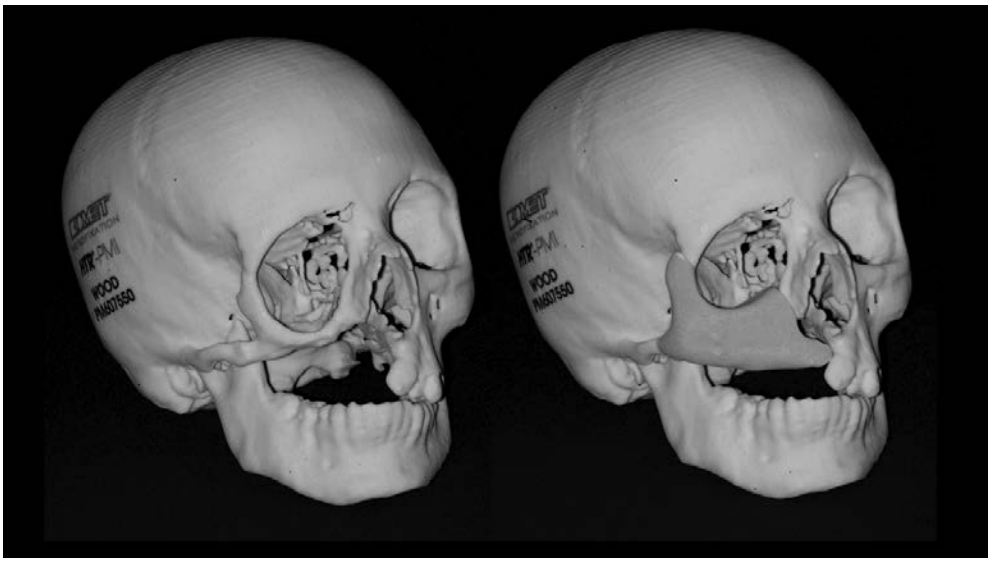


Figure 18-27.
Based on a CBCT scan, a stereolithographic model of the patient's skull was created. In addition to the skull, a facial prosthesis segment was also designed on top of the CBCT image, achieving precise fit and anatomic contour in order to not only achieve optimal esthetics, but also serve as a base for future dental implants. This figure shows the same skull with and without the prosthesis.

Figure 18-28.
Hologram showing a CT image of a pelvic bone in 3D space. A Voxbox® is necessary for visualization (courtesy of Voxel, Inc – Provo – UT, USA).

A Complete 3D Patient Record

The ideal patient record situation would be a complete 3D craniofacial record in which there would be individual as well as conjunctive access to soft tissue of the face, skeleton, and dentition. The only way we can have such a record is in a digital format. Several attempts have been made to create a complete 3D craniofacial record. Most of the attempts involve the collection of separate images for face, craniofacial skeleton, and dentition, and then combining them into a single image (Figure 18-29). This process may not be very accurate since the records are taken at different times with the patient in different positions. The methodology may also not be user friendly or practical in a clinical environment.

The dentition can currently be generated out of a CBCT scan, which not only eliminates a step in the records taking, but also eliminates the problem of different patient positioning, since both radiographic and dental volumes are collected simultaneously. With this possibility, the CBCT technology provides an almost complete record of the patient, with the exception of soft tissue color information. Nevertheless, such information can be added to the scan, completing the patient's record (Figure 18-30).

Outcome assessments in 3D will also be necessary in order to better assess the additional information present, and possibly have a deeper understanding

of the effects of different treatment techniques and choices. This additional information is properly analyzed, may allow us to have an evidence based approach to our treatment options. Superimpositions of 3D images has been possible for a long time, but has recently been made more user friendly, and can now be easily integrated into the clinical environment (Figure 18-31).

The Cone Beam Computed Tomography scanners show strong potential in becoming the source of a complete patient's record. If this eventuates, a records appointment could end up taking less than 10 minutes. With a new record's format, new analysis will need to be created in order to fully analyze the additional information collected. One of the places where new analyses are being developed is the Craniofacial Imaging Center at the Case Western Reserve University School of Dental Medicine, and the Bolton-Brush Growth Study Center.

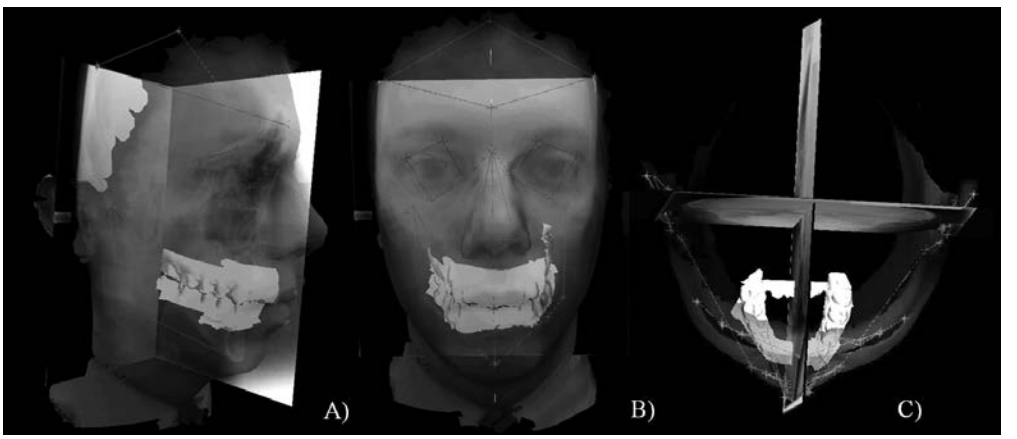


Figure 18-29.
Constructed complete 3D patient record using frontal and lateral cephalograms, digital 3D dental casts, and a 3D stereophotogrammetric image of the face. A) Lateral view, B) frontal view, and C) view from the top.

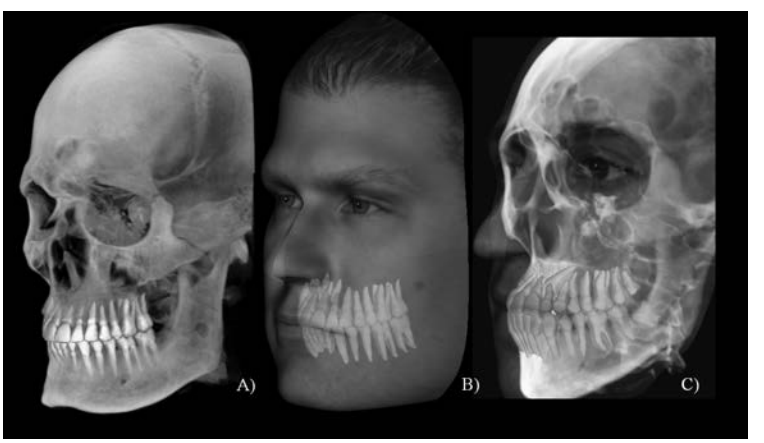


Figure 18-30.
Mix and match of soft tissue, skeletal, and dental images, combined as a single 3D recor by using the InVivo program (courtesy of Anatomage Inc. – San Jose, CA).

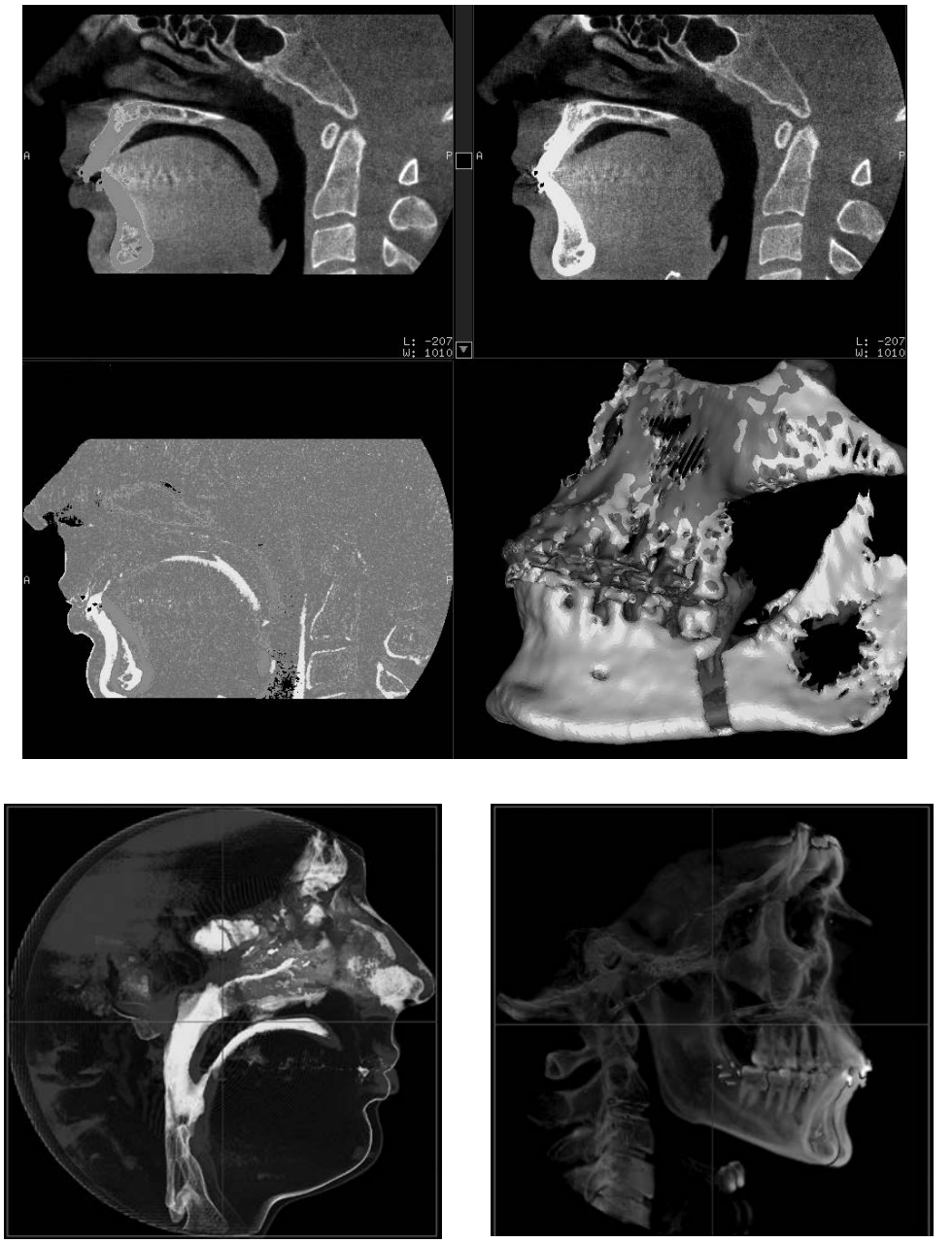


Figure 18-31.
A) Custom program showing the superimposition of pre and post CBCT images of a mandibular advancement surgery procedure. B) Superimposition of the same patient using a commercially available user-friendly program that can be easily integrated into the clinical environment (InVivo - Anatomage Inc. – San Jose, CA), and C) Same program allows the evaluation of airway and soft tissue changes.

THE BOLTON STANDARDS

Orthodontic diagnosis of growing children and outcome assessment often involves comparison of patient's radiographs with the clinician's perception of normality. A subjective endeavor by definition, the variation between practitioners often leads to the desire for a second opinion. While the large scale pattern of craniofacial growth and development generally are known, there still is a need for a tangible, material, and statistical way of visualizing normality.

Modern statistics owes its origin largely to attempts to measure and describe craniofacial anatomy. The history of craniometry in growth studies can be divided into four stages, earliest to latest: first, craniometry by caliper measurements (dry skull only) that was cross sectional (different individuals in each cohort) but which could be represented three dimensionally; second, soft tissue cephalometry (on live human heads) that was longitudinal but also could be represented three dimensionally; third, roentgenographic cephalometry (projection X-ray head films) that is longitudinal but in practice is two dimensional; and most recently digital tomographic cephalometry (plain film X-ray, CT, CBCT, MRI) that is longitudinal and, once again, three dimensional.

The earliest interest in radiographically based cephalometry was taken by B.H. Broadbent, Sr. He became interested in craniofacial growth and development as a student of T. Wingate Todd. Working with Todd in 1924, Broadbent added a metric scale to Todd's craniostat (an instrument used to hold a dry skull in a fixed position), and turned it into a craniometer, which permitted direct measurements of craniofacial structures. In 1925, Broadbent added an X-ray film holder to the craniometer, converting it into a roentgenographic craniometer, and making possible the standardized collection of dry skull cranial X-rays.

In 1926, Broadbent developed the roentgenographic cephalometer (Figure 18-32). This device holds the head of a living subject in a fixed position, while precise and reproducible lateral and posteroanterior (frontal) radiographs are taken. The Broadbent-Bolton cephalometer became the primary data collection device for several craniofacial growth studies (Table 18-2). In later studies, Broadbent fully developed and suggested a standard protocol by which frontal and lateral head radiographs could be taken clinically.

Broadbent began longitudinal data collection for the "Bolton Study of the Development of the Face of the Growing Child" in 1927. After completing data collection in 1959, he selected what he would call the "Bolton Faces". The following criteria of "normality" were established as the framework for the Bolton Face selection process: (1) Excellence of static occlusion as viewed on dental study casts and cephalometric radiographs, (2) A good health history, which precluded those with significant debilitating diseases from becoming part of the group, (3) Faces that conformed favorably to the statistically derived mean of craniofacial measurements, (4) Esthetically favorable faces, as chosen arbitrarily by the Bolton Study Board of Managers, and (5) Availability of long-term records, i.e., the individual case was recorded annually from 1 year to 18 years of age.

The published Bolton Standards included thirty-two people (16 males, 16 females). One operator, William H. Golden, produced all the lateral and

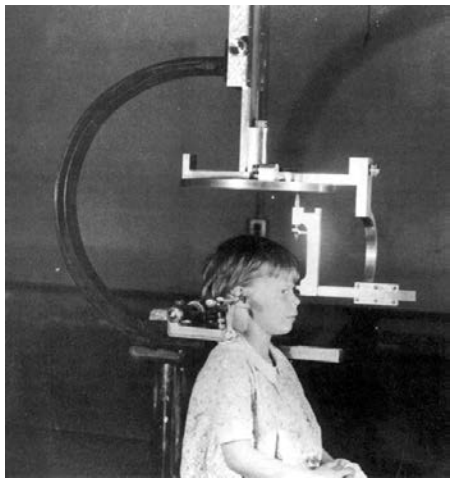


Figure 18-32.
First Broadbent cephalometer, designed to hold the living head in a manner similar to that used by the radiographic craniometer. (From Broadbent, B. H., Sr., B. H. Broadbent, Jr., and W. Golden: Bolton Standards of Dentofacial Developmental Growth. St. Louis, C. V. Mosby, 1975, with permission).

Table 18-2.
Longitudinal Growth Studies that used the Broadbent-Bolton Cephalometer.

Study	Reference
The Bolton Study	(Broadbent et al. 1975)
The Burlington Growth Study	(Popovich, Thompson 1977; Saunders et al. 1980)
The Michigan Growth Study	(Primack 1978; Ackerman 1979)
The Belfast Growth Study	(Kerr 1979)
The Philadelphia Growth Study	(Krogman, Sassouni 1957)
The Denver Growth Study	(Sherman et al. 1988)
The Fels Research Institute Study	(Lewis et al. 1985)
The Forsyth Twin Study	(Medicus et al. 1971)
The Iowa Child Welfare Study	(Bishara et al. 1985)
The Matthews Imphant Collection	(Baumrind et al. 1987)
The Meharry Growth Study	(Richardson 1991)
The Montreal Growth Study	(Buschang, Tanguay 1989)
The Oregon Growth Study	(Buck, Brown 1987)

posteroanterior radiograph tracings. Golden condensed all bilateral (right and left) structures seen in the lateral view into a single central outline, assuming right and left symmetry. After that, all the tracings were divided into mixed-sex age groups. Next, pairs of tracings within each age group were manually averaged, resulting in a third tracing. This third tracing then was positioned near the third tracing produced from the other pair to produce a new average, and so on until the entire age series had been "averaged" (Figure 18-33). At the end, Golden produced a single mixed-sex "average" tracing, representing frontal and lateral views for each age group. The published Bolton Standards present two average tracings (frontal and lateral) for each age group, as the male and female average tracings were averaged together (Figure 18-34).

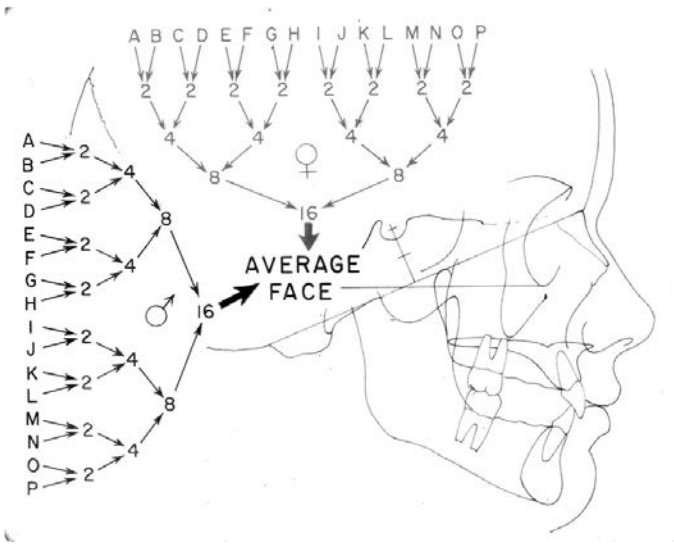


Figure 18-33.
Illustration showing averaging of male and females tracings, which are ultimately combined in to a single average for each age.

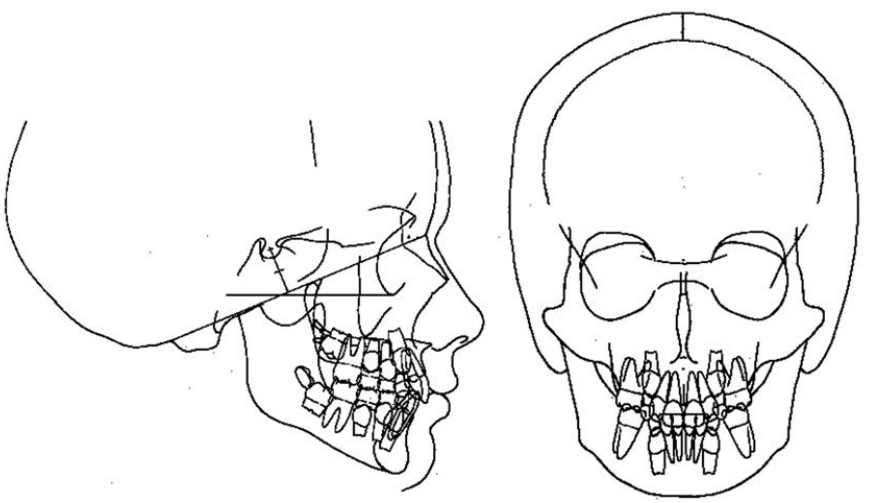


Figure 18-34.
The lateral and frontal Bolton Standards of Dentofacial Developmental Growth. The lateral standards are available from age 1 to 18, while the frontal standards are available from age 3 to 18, since no lateral radiographs were taken prior to age 3.

It should be noted that the frontal radiographs were not taken routinely during the first two years of age because of the difficulty of positioning the infant, coupled with the long exposure time then necessary to obtain radiographs. Thus, the frontal Bolton Study films began at age 3, and were sampled yearly up until age 18, whereas the lateral tracings trace birth through age 18.

Using the Bolton Standards

The use of the Bolton Standards by the clinician, researcher, or teacher is limited only by the individual's imagination. These applications may be simple when used as a comparative standpoint or complex as the use of a baseline for describing involved morphologic patterns, paths of dental eruption, or the correlation of an infinite number of specified linear or angular measurements.

The Bolton Standards are related to general concepts of normality because they are not artificial gauges, but rather have been derived from actual cases that present a so-called "normal condition" of craniofacial morphology as well as arch alignment. It should be pointed out, however, that rather than their being a statistical mean drawn at random from the population, they are instead a representation of the "optimum" or, as stated by the Merriam-Webster medical dictionary, "most favorable condition under given circumstances".

The Bolton Standards as a measuring device may be used in a manner analogous to other accepted measuring devices to provide comparative sizes and morphologic patterns, as well as dental eruption stages. Different methods for using the Bolton Standards have been described. Here are some suggested methods for Bolton Standard analysis:

Using the Lateral View

1. Superimpose the appropriate chronologic Bolton Standard on the tracing (or radiograph) and compare in a cranial base plane. The Bolton-Nasion plane is the recommended choice to compare cranial bases. The Bolton point is defined as the center of the foramen magnum, located on the lateral cephalometric radiograph as the highest point in the profile image of the postcondylar notches of the occipital bone. The Bolton point may be obscured by the mastoid process during the teen years. The Bolton point is selected rather than the Basion because of the desire to get an impression of the total length of the cranial base. This does not mean, however, that the Basion-Nasion plane should not be used and interpreted by those who find it more desirable. The Bolton Standards clearly indicate both Bolton and Basion among several other landmarks.

2. Assess the component skeletal parts individually with the Bolton Standard that best approximates the skeletal area under appraisal and assign a Bolton age to the Cranial Base, Maxilla, Mandible, and Soft tissue Profile (Figure 18-35). To assess the Cranial Base, the Bolton-Nasion plane is suggested. The maxillary Bolton correlation can be performed by superimposing the maxillary planes and comparing the linear dimensions between the posterior nasal spine (PNS) and both A point and the anterior nasal spine (ANS). The mandibular Bolton correlation can be performed by superimposing on a line created from Articulare to Gnathion denoting the "effective length". At the end of the Bolton Standard Correlation analysis, one should have an age for the cranial base, maxilla, mandible and soft tissue profile separately (Figure 18-36).

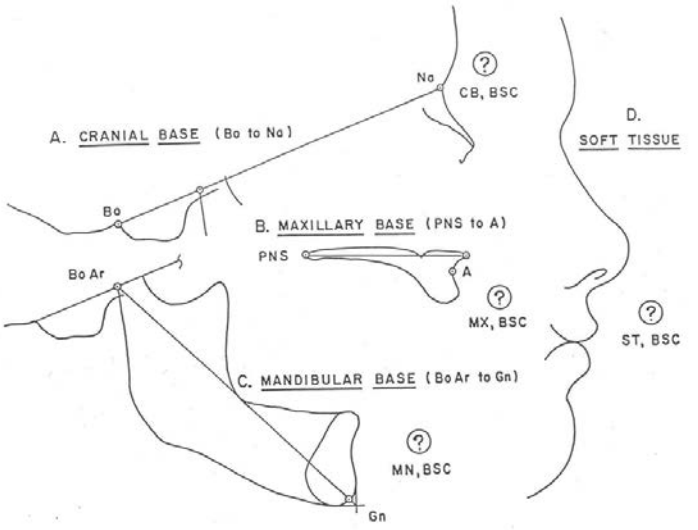


Figure 18-35.
Example of Bolton Standard Correlation (BSC) use. A) Tracing of a 8 year and 11 month old patient; B) Superimposition of Standard age 9 on Bo-Na shows that the patient's Cranial Base is longer that his chronological age. The standard age that fit the best was age 15; C) Superimposition of age 9 on PNS-A shows a good fit, assigning the age 9 to the maxilla; and D) Superimposition of Standard 9 on Ar-Gn appears to be slightly larger. The age 8 had the best mandibular fit. The final conclusion is that the patient has a long cranial base for this age.

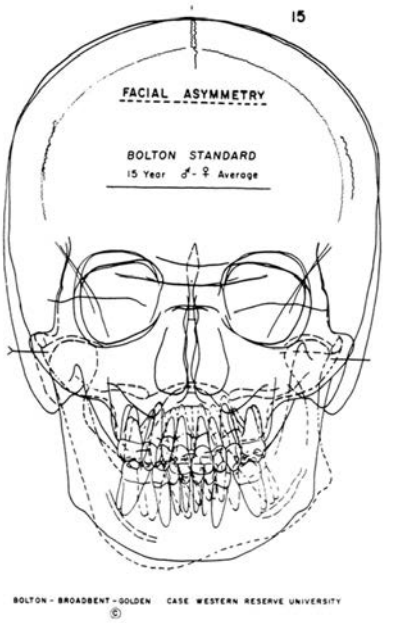


Figure 18-36.
Fifteen-year-old frontal Bolton Standard (superimposed on the midsagittal plane and orbits) to indicate facial asymmetry in a typical case. (From the Bolton Study, Bolton-Brush Growth Study Center. Case Western Reserve University, Cleveland, Ohio, with permission.)

3. Analyze by superimposing with a "best fit" method on the soft tissue profile, starting with the forehead and nose. In this method, the position of the maxilla, mandible, dentition, and vertical dimension can be analyzed. This simple and fast method is very useful not only for diagnosis and treatment planning procedures,b ut also for consultation and patient communication as well.

Using the Frontal View

1. Superimpose the appropriate chronologic Bolton Standard by coordinating the cranial, midfacial, and mandibular outlines as closely as possible in relation to the midsagittal plane and orbital outlines.

2. Assess the component parts from the standpoint of morphology, size, symmetry, and individual variation, both skeletally and dentally (Figure 18-37).

3. Observe anomalous positions of individual dental units, both erupted and unerupted.

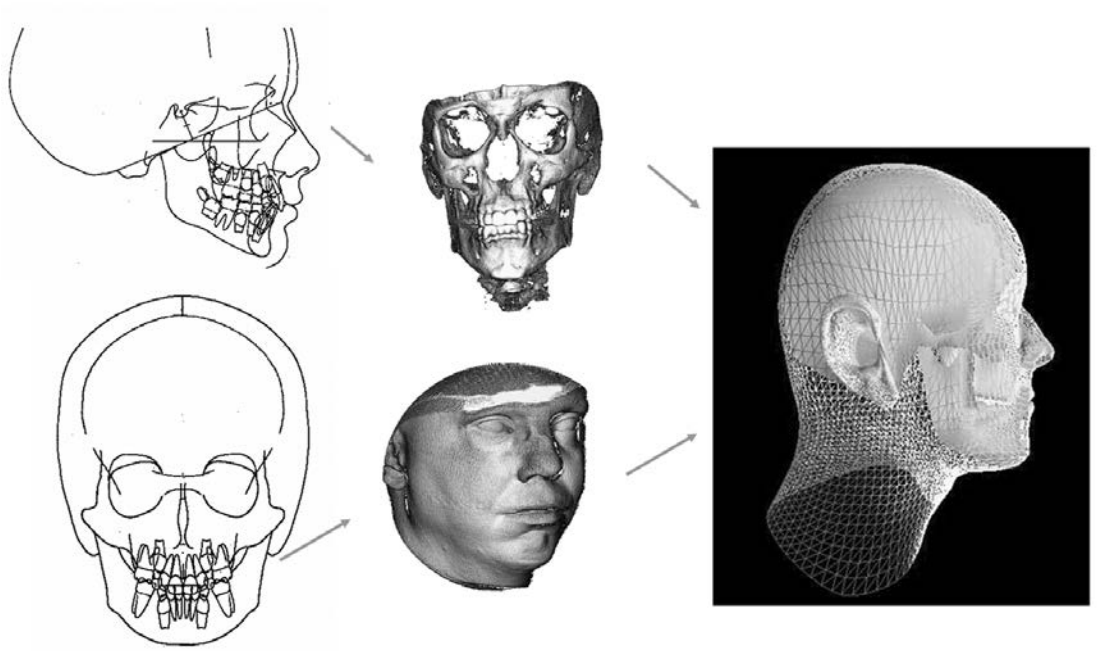


Figure 18-37.
The 3D standards are created from the inherent 3D skeletal and soft tissue information present in the traditional Bolton Standards, with the addition of volumetric data.

3D Bolton Standards-The Future of Craniofacial Treatment Planning

From digital radiography, the next evolutionary step is the use of true and direct 3D representations of the patient. To use true 3D information, we will shift from landmarks and lines to surfaces, and from linear measurements and angles to volumes and areas.

The simple and effective visualization that the Bolton Standards offers is now in the process of being converted to 3D. This conversion only is possible due to the care employed when taking the original radiographs used to create the standards, and the lack of patient movement when taking the lateral and frontal radiographs. The new 3D Standards will be created from the inherent 3D skeletal and soft tissue information present in the traditional Bolton Standards, with the addition of volumetric data from matched subjects (Figure 18-38 and 18-39). The superimposition of a standard to the patient's 3D image can be performed by making the standard's image transparent, or by combining both images and using a surface metric distance method.

The 3D standards currently are under development and will provide a way to better visualize, analyze, and communicate when using 3D volumetric images.

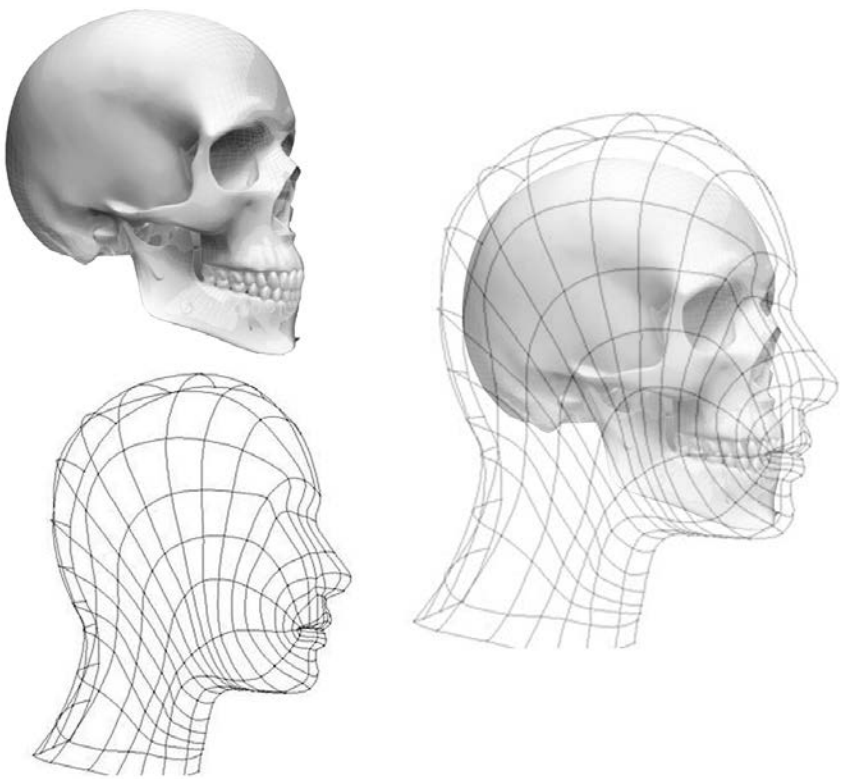


Figure 18-38.
The 3D Standards can be seen as soft tissue, skeletal tissue, and a see-through combination of soft tissue and skeletal data.

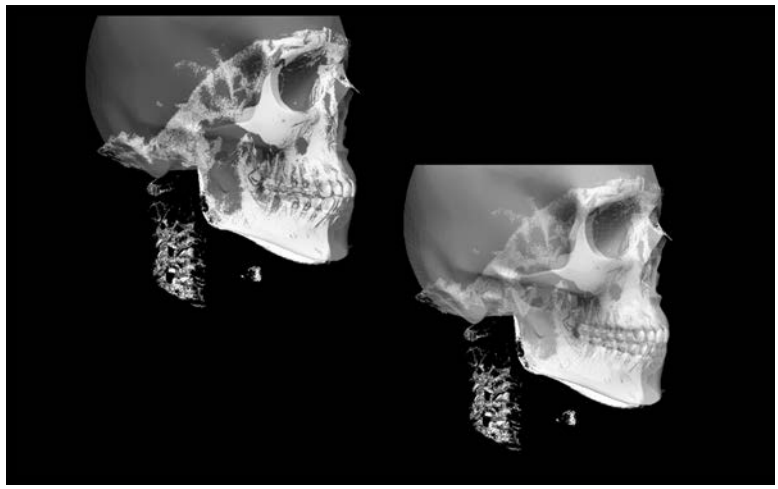


Figure 18-39.
The 3D Standards allow for separate skeletal and soft tissue comparisons. The superimposition can be done by transparency of the standard.

COUNTERPART ANALYSIS (OF ENLOW)

This is a method in which the various facial and cranial parts are compared with each other to see, simply, how they fit. The individual is measured against himself, rather than compared with population standards and norms. Most conventional methods of analysis and cephalometric growth studies are intended essentially to determine what a particular growth or form pattern is. This procedure was developed to explain how such a pattern was produced in any given person. The ANB angle, for example, tells one the nature of the positional relationship between the anterior part of the upper and lower arches and provides an index with which one can gauge the extent of malocclusions. The counterpart procedure is intended to account for the composite of the anatomic and morphogenetic factors that produced the particular ANB angle (and other measurements) found in a given person.

Most conventional cephalometric planes and angles are not intended to coincide with or indicate actual sites and fields of growth and remodeling, and they are thus not appropriate for the essentially anatomic purposes just described. Because most standard planes and angles do not represent the patterns and distribution of growth fields, comparisons of the individual with population standards are required; there is usually no other basis for interpretation, owing to the nature of the planes themselves. However, if planes are constructed so that the activities of the growth and remodeling fields are in fact directly represented, a built-in and morphologically natural set of "standards" is identifiable that allows meaningful evaluation of overall craniofacial form and pattern without population comparisons.

The analysis is based on the counterpart principle. This is the actual design basis upon which the face is constructed and which underlies the plan of its intrinsic growth process. The counterpart concept was described in previous

chapters, and it was used as the working basis for explaining how the face grows. The counterpart analysis is, in effect, the same. It shows where imbalances exist, how much is involved, and what the effects are.

In Figures 18-40 and 18-41 construction lines have been drawn on a headfilm tracing to represent several key fields and sites of growth. These include the maxillary tuberosity, the mandibular condyle (using articulare for convenience, rather condylion), the ramus-corpus junction, the posterior border of the ramus, the anterior surfaces of both the maxillary and mandibular bony arches, the occlusal plane, and the junction between the middle and anterior cranial fossae (the anterior-most extent of the great wings of the sphenoid where they cross the cranial floor). Other planes may be added to represent other major growth areas, if desired, such as the zygomatic arch, the palate, the olfactory plane, and the anterior-vertical plane of the midface.

Note that the PM vertical plane is represented. This is the important boundary that separates the anterior cranial fossa and nasomaxillary complex from the middle cranial fossa and pharynx. The ramus relates to the latter and the corpus to the former.

Two basic factors are important in evaluating the role of any bone or part of a bone in a composite assembly of several different bones. The first is the bone's size (horizontal and vertical), and the second is its alignment (rotational position). In this analysis, both must be considered. The reason is that the nature of the alignment of any bone affects the expression of its various dimensions. The determination of a bone's dimension alone is not enough (and can be misleading); its alignment must also be known for one to see just how this factor affects its actual dimensions. In the counterpart analysis, both are determined for all the various bony parts and counterparts.

The rationale, in brief, is that the vertical and/or horizontal size of one given part is compared with that of its specific counterpart(s). If they exactly match, or nearly so, a dimensional "balance" exists between them. If one or the other is long or short, however, the resulting imbalance can cause either **protrusion** or **retrusion** of the part of the face involved and thereby affect the profile, either directly or indirectly. The various parts and counterparts are then checked for their alignment to see if each, independently, has a protrusive or a retrusive effect, regardless of the nature of the dimensions. Then all the regional part-counterpart relationships are added up to see how the sum of all underlies the face of any given individual. This may be done on a single headfilm tracing at any age, or serial headfilms can be used for determining the progressive effects of age changes or of treatment results.

Figure 18-40 shows a Class II individual in whom major variations and imbalances are present for the different horizontal and vertical dimensions and for the alignment relationships (compare with the Class III individual described in the next paragraph). Note that (1) the mandibular corpus is short **relative** to its counterpart, the maxillary bony arch (both skeletally and dentally in this individual); (2) the corpus is aligned (rotated) upward (i.e, the "gonial angle" is more closed); (3) the middle cranial fossa is aligned obliquely more forward (the dashed lines represent "neutral" alignment positions); (4) the ramus is aligned

more backward; and (5) the nasomaxillary complex is vertically long (resulting in a downward and backward ramus rotation). **All** these features are either mandibular retrusive or maxillary protrusive, and they have combined to produce the multifactorial basis for a Class II malocclusion and retrognathic profile. Note, however, that the horizontal breadth of the ramus **exceeds** its counterpart, the horizontal (not oblique) dimension of the middle cranial fossa. This is a compensatory feature that has partially offset the aggregate effects produced by other features and thereby reduced the severity of the malocclusion. If desired, the actual amounts of each and all of these effects can be measured.

Figure 18-41 shows an individual in whom the dimensions and alignments combine to produce the composite, multifactorial basis for a Class III malocclusion. Note that (1) the dental **and** skeletal dimensions of the mandibular corpus in this individual exceed maxillary arch length; (2) the corpus is aligned (rotated) markedly downward; (3) the middle cranial fossa is aligned backward; and (4) the ramus is aligned forward. These relationships are all mandibular protrusive or maxillary retrusive, and they combine to produce the prognathic face and Class III malocclusion. The horizontal breadth of the ramus, however, is less than that of its counterpart, the middle cranial fossa. This is a compensatory feature that, in this particular individual, has partially offset the composite effects of the other relationships and thereby reduced the extent of the malocclusion.

For a more detailed description of the construction lines used, how to determine the actual dimensions, and how to establish the "neutral" alignment planes for any given individual, see Enlow et al. (1971).

The "counterpart analysis" is not intended as a routine clinical tool for everyday office use in diagnosis and treatment planning. It is not needed for this, because the rationale for treatment procedures, at least today, is not usually

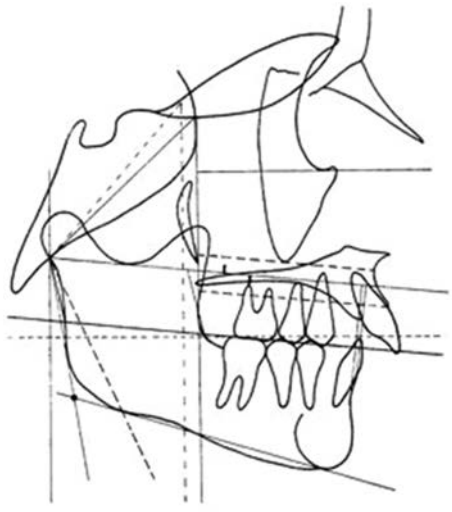


Figure 18-40.
Headfilm tracing of a Class II patient. Construction lines have been added for the counterpart analysis. (From Enlow, D. H., T. Kuroda, and A. B. Lewis: The morphological and morphogenetic basis for craniofacial form and pattern. Angle Orthod., 41:161, 1971, with permission).

based on corrections of the actual underlying causes of mal-occlusions and other kinds of facial and cranial dysplasias. The counterpart analysis is useful, however, in determining what treatment has done in terms of the specific anatomic and developmental changes that have been brought about, more so than most types of analyses, because the others deal more with correlative geometry than with morphologic and morphogenetic relationships. Actually, the immediate payoff for the counterpart analysis has already been largely achieved. It has pointed out more clearly the multifactorial basis for malocclusions and just what some of the specific anatomic and developmental factors are. It has shown how a number of compensatory features participate. It has explained how and why population groups have either Class II or Class III tendencies. Except for such specific types of research studies, however, the counterpart analysis is inappropriate as a routine clinical method. When the intrinsic control processes of facial growth become better understood, when the control processes themselves can be controlled, and when treatment procedures can **then** become based on the real causative factors that underlie structural imbalances, then cephalometric analyses utilizing genuine anatomic and developmental relationships will become increasingly more relevant. The counterpart analysis itself, of course, is far from complete and is only a beginning, but it is a concept. It is also very useful in understanding the rationale for the basic plan of normal facial construction as well as malocclusions and in explaining and teaching this complex subject to students in a way that is relatively easy to understand.

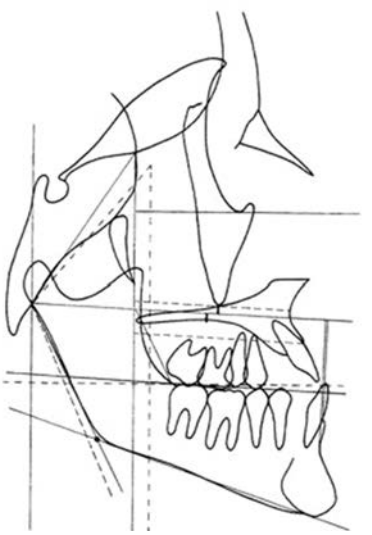


Figure 18-41.
Headfilm tracing of a Class III patient. Compare with Figure 18-44. (From Enlow, D. H., T. Kuroda, and A. B. Lewis. The morphological and morphogenetic basis for craniofacial form and pattern. Angle Orthod., 41:161, 1971, with permission).

Glossary of Terms

The terms to be defined are primarily related to landmarks used in roentgenographic cephalometry. The definitions used are those most commonly found in the craniometric and orthodontic literature.

A:
See Subspinale

Antegonion:
The highest point of the notch or concavity of the lower border of the ramus where it joins the body of the mandible.

ANS, Anterior nasal spine:
A sharp median process formed by the forward prolongation of the two maxillae at the lower margin of the anterior aperture of the nose.

Anteroposterior (AP) or frontal growth axis of the head and face:
A transverse zone delineated by a plane through the coronal suture above, passing down through the pterygomaxillary fissure near the posterior termination of the hard palate along the anterior border of the ascending rami, and through the junction of the horizontal and vertical components of the mandible. It marks the division of the anterior from the posterior component of craniofacial developmental growth when lateral tracings are oriented in Bolton relation.

Ar, Articulare:
Bjork—The intersection of the image of the posterior border of the ramus with the base of the occipital bone. Bolton—The point of intersection, in lateral aspect, of the posterior border of the condyle of the mandible with the Bolton plane.

B:
See Supramentale.

Ba, Basion:
The point where the median sagittal plane of the skull intersects the lowest point on the anterior margin of the foramen magnum.

Bolton plane:
A line joining the Bolton point and nasion on the lateral cephalogram.

Bo, Bolton point:
A point in space about the center of the foramen magnum that is located on the lateral cephalogram by the highest point in the profile image of the postcondylare notches of the occipital bone.

BSC, Bolton standard correlation:

CB, Bolton cranial base:
A line from Bolton articulare to nasion.

MX, Bolton maxillary base:
A line from PNS to ANS.

MN, Bolton mandibular base:
A line from Bolton articulare to gnathion.

Bregma:
The point on the skull corresponding to the junction of the coronal and sagittal sutures.

Cephalogram or Radiograph:
A generally accepted term describing a standardized roentgenographic (x-ray) picture of the head.

Cephalometer (roentgenographic cephalometer):
In craniometry, an instrument for measuring the head. A cephalometer (device for holding the head) combined with roentgenographic equipment for the production of standardized complementary lateral and frontal radiographs used for measuring developmental growth of the dentition, face, and head.

Cd, Condylion:
The most superior point on the head of the condyle.

Convexity, angle of:
The angle formed by a line nasion to A and a projection of a line pogonion to A.

Coronal suture:
The transverse union of the frontal with the parietal bones.

Craniostat:
A device for holding the head for craniometric study.

Dacryon:
A point on the inner wall of the orbit at the junction of the frontal and lacrimal bones and maxilla.

Facial angle:
The angle formed by the junction of a line connecting nasion and pogonion, FP (facial plane), with the horizontal plane of the head, FH (Frankfort plane).

Facial height:

Total:
The distance between nasion and gnathion when projected on a frontal plane.

Lower face:
The distance between ANS and gnathion when projected on a frontal plane.

Upper face:
The distance between ANS and nasion when projected on a frontal plane.

FP, Facial plane:
The line connecting nasion and pogonion on the lateral cephalogram.

FH, Frankfort horizontal plane:
A horizontal plane determined by the two poria and left orbitale. It approximates closely the position in which the head is carried during life and is established on the lateral cephalogram by a line joining orbitale with porion as indicated by the top of the ear rod.

FMA angle:
The angle formed by the mandibular plane and the Frankfort horizontal plane.

Foramen rotundum:
A round opening in the greater wing of the sphenoid bone for the passage of the superior division of the fifth nerve.

FOP, Functional occlusal plane:
A horizontal line from the posterior-most occlusal contact of the last fully erupted maxillomandibular molars extending anteriorly to the anterior-most occlusal contact of the fully erupted premolars.

Frontotemporale:
A point near the root of the zygomatic process of the frontal bone at the anterior-most point along the curvature of the temporal line.

Gl, Glabella:
The most anterior point on the frontal bone.

Gn, Gnathion:
The lowest, most anterior midline point on the symphysis of the mandible.

Go, Gonion:
The external angle of the mandible, located on the later cephalogram by bisecting the angle formed by tangents to the posterior border of the ramus and the inferior

border of the mandible.

Ii, Incisor inferius:
The tip of the crown of the most anterior mandibular central incisor.

Is, Incisor superius:
The tip of the crown of the most anterior maxillary central incisor.

Id, Infradentale:
The most anterior point of the tip of the alveolar process between the mandibular central incisors.

I, Inion:
The apex of the external occipital protuberance.

Interincisal angle:
The angle formed by the long axis of the lower incisor and the long axis of the upper incisor.

Internal angle of the mandible:
Located on the lateral cephalogram by bisecting the angle formed by tangents to the anterior border of the ramus and the superior border (alveolar crests) of the mandible. Note: A line joining the internal angle and the antegonion marks the junction of the ramus with the body of the mandible.

Key ridge:
The prominent ridge, formed by the malar process, which divides the canine fossa from the infratemporal fossa on the lateral surface of the maxillary bone.

Lateral growth axis:
The division between the right and left lateral components of growth (see median sagittal plane).

MP, Mandibular planes:
Variations of definitions include:

A tangent to the lower border of the mandible.

A line joining gonion and gnathion.

A line joining gonion and menton.

A line from mention tangent to the posteroinferior border of the mandible.

Maxillary plane:
See Palatal plane.

Median sagittal plane:
See Lateral growth axis. The anteroposterior median plane of the cranium and face.

Me, Menton:
The most inferior point on the symphysis of the mandible in the median plane. Seen on the lateral cephalogram as the most inferior point on the symphyseal outline.

N, Na, Nasion:
The craniometric point where the midsagittal plane intersects the most anterior point of the nasofrontal suture. (The anterior termination of the Bolton plane.)

Normal face:
By normal face, we do not mean a face of certain dimensions or particular form (features), but a well-grown face, harmoniously developed, skeletally and dentally, and consistent in developmental progress with its years (Bolton).

O point:
Center for convergence area of horizontal planes used in Sassouni's analysis.

Occ, Occlusal plane:
A line passing through one-half of the cusp heights of the first permanent molars and one half of the overbite of the incisors.

Op, Opisthion:
The most posterior point of the foramen magnum.

Orbital plane:
The frontal (transverse) plane of the head passing through the left orbital point.

Or, Orbitale:
In craniometry, the lowest point on the inferior margin of the orbit. The left orbital point is used in conjunction with the poria to orient the skull on the Frankfort horizontal plane.

Pal, Palatal plane, Maxillary plane:
A line connecting the tip of the anterior nasal spine with the tip of the posterior nasal spine as recorded in the lateral cephalogram.

PM, Posterior maxillary plane:
A vertical line from the averaged intersections of the great wings of the sphenoid and the anterior cranial floor, extending inferiorly to the averaged lower-most points of PTM.

Po, Pog, Pogonion:
The most anterior point on the symphysis of the mandible in the median plane.

Points:

A:
See Subspinale.

B:
See Supramentale.

D:
The center of the cross section of the body of the symphysis. It is established by visual inspection.

R:
Bolton registration point. The center of the Bolton cranial base; a point midway on the perpendicular erected from the Bolton plane to the center of the sella turcica (S).

P, Porion:
Anatomic porion is the outer upper margin of the external auditory canal; machine porion is the uppermost point on the outline of the ear rods of the cephalometer.

Porionic axis:
A line drawn between the two poria.

PNS, Posterior nasal spine:
A process formed by the united, projecting median ends of the posterior borders of the two palatine bones.

Pr, Prosthion:
The most anterior point of the alveolar portion of the premaxilla, between the upper central incisors.

PTM, Pterygomaxillary fissure:
In the lateral cephalogram, an inverted, elongated, teardrop shaped area formed by the divergence of the maxilla from the pterygoid process of the sphenoid. The posterior nasal spine and staphylion are generally located beneath the lower pointed end of this area.

Pt-vertical:
A vertical line tangent to the posterior contour of PTM perpendicular to FH.

S, Sella turcica (Turkish saddle):
The hypophyseal or pituitary fossa of the sphenoid bone lodging the pituitary body. The landmark S is the center of sella as seen in the lateral cephalogram and located by inspection.

SE, Sphenoethmoidal suture:
The most superior point of the suture.

Si:
The most inferior point on the lower contour of the sella turcica.

SN, Sella-nasion plane:
The plane formed by connecting a line from sella to nasion.

Skeletal age:
The maturational age of an individual as determined by the analysis of bone age indicated by the hand-wrist x-ray (biologic age).

SO, Spheno-occipital synchondrosis:
The most superior point of the junction between the sphenoid and occipital bones.

Sp:
The most posterior point on the posterior contour of the sella turcica.

Supraorbital plane:
A line tangent to the anterior clinoid process and the most superior point on the roof of the orbit, as seen on the lateral cephalogram.

Sta, Staphylion:
The point in the medial line (interpalatal suture) of the posterior part of the hard palate where it is crossed by a line drawn tangent to the curves of the posterior margins of the palate. (In the lateral cephalogram, the posterior curved margins of the hard palate frequently may be seen more clearly than the posterior nasal spine).

SOr, Supraorbitale:
The uppermost point of the orbital ridge on the lateral cephalogram, it can be located at the junction of the roof of the orbit and the lateral contour of the orbital ridge.

Subspinale (Point A):
That point in the median sagittal plane where the lower front edge of the anterior nasal spine meets the front wall of the maxillary alveolar process (Downs' point A).

Supramentale (Point B):
The deepest midline point on the mandible between infradentale and pogonion (Downs' point B).

Te, Temporale:
The intersection of the shadows of the ethmoid and the anterior wall of the infratemporal fossa.

Vertex:
The most superior point on the cranial vault.

Y-axis:
The line joining the sella turcica (S) center and gnathion.

Zygion:
The point on the zygoma on either side, at the extremity of the bizygomatic diameter.

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Mechanobiological Perspectives of Orthodonic Tooth Movement Related to Bone Physiology

Meropi N. Spyropoulos, D.D.S., M.S., Ph.D.

Apostolos I. Tsolakis, D.D.S., M.S.D., Ph.D.

MECHANOBIOLOGICAL PERSPECTIVES OF ORTHODONTIC TOOTH MOVEMENT RELATED TO BONE PHYSIOLOGY

Different theories have been proposed for the explanation of the tooth movement phenomenon, including the pressure tension hypothesis and the bone bending mechanism. Following orthodontic tooth movement, tissue reactions result in alveolar bone turnover due to remodeling processes. During tooth movement the alveolar bone undergoes both resorption and deposition that are dependent upon the nature of the applied force, the biological state of the involved tissues and the general health status of the treated individual.

Mechanical and metabolic control of alveolar bone remodeling may influence the extent, the rate, and the quality of tooth movement, through various biochemical responses. Various locally produced cytokines have been identified as regulatory molecules for the recruitment and activation of osteoclasts during orthodontic tooth movement, but there is still a lack of knowledge concerning the mechanism that coordinates all different cells and tissues involved.

Aging seems to be a crucial factor for orthodontic tooth movement, since not only the quality of the alveolar bone is different, but also bone turnover mechanisms are disturbed and locally produced regulatory molecules are less responsive to orthodontic forces in adults as compared to juveniles.

In the light of skeletal physiology, bone remodeling patterns may be recognized not only at the site of the moving tooth, but also in adjacent and remote areas. As a result we may distinguish: 1. Distal and remote alveolar bone responses (RAR), 2. Regional acceleratory phenomena (RAP) and 3. Bone distraction like phenomena (DLP).

Pressure Tension Hypothesis

In the biology of tooth movement, the pressure and tension hypothesis, strictly related to the periodontal ligament (PDL) area, was used to explain changes around the orthodontically moving tooth (Reitan 1985, Masella and Meister 2006, Meikle 2006). Following the loading of a tooth, alveolar bone formation occurs on the side where tension of the ligament is exerted, and resorption is observed on the pressure side (King et al 1991, Spyropoulos 2006).

The stress/strain distribution in the periodontal tissues results in the differentiation of the PDL progenitor cells into compression-associated osteoclasts and tension-associated osteoblasts. As a result, bone is resorbed in the compression area and bone is formed at the tension area.

Frontal alveolar bone resorption is a result of light force application that preserves cellular and vascular integrity, while undermining alveolar bone resorption is associated with heavy forces causing hyalinization effects in the periodontal ligament tissues. Undermining resorption is considered to be the result of fatigue-failure mechanism in means of biomechanics of skeletal adaptation (Roberts et al 2004). Alveolar bone surface resorption may be an atrophic response to suboptimal loading or fatigue-failure result due to excessive tensile strain. Although the PDL is an extension of the periosteum, it possesses a unique physiology due to the tooth supporting mechanism (Picton and Wills 1978) and to the existence of an osteogenic vascular network in its center (Roberts et al. 1987). Another major difference between the PDL and the periosteum resides in the fact that tensile stress is increased within the PDL instead of at the bone surface.

Despite of the fact that the pressure-tension hypothesis directs our conception of tooth movement mechanism, Meikle (2006) addresses questions related to the ability of the PDL to generate tension and to transmit further differential pressures within the periodontium. Therefore, it seems that the remodeling responses of the alveolar bone during tooth movement constitute independent mechanisms from the PDL response.

Bone Loading and Bending Mechanism

Loading of a tooth during orthodontic treatment is responsible for the surrounding tissue response that results to tooth movement. The magnitude of the applied loads is essential for the osseous response and dictates bone remodeling patterns. Frost, in 1987, developed the "mechanostat theory" that relates mechanical loading to bone reaction, and describes a relationship between different strain values and modeling or remodeling activities. Strain is defined as "mechanical deformation per unit length" and it is expressed as the change in length divided by the original length of the relevant tissues. For example if a bone of 100mm in length is shortened by 2mm, the respective strain is expressed as 2% strain, 0.02 strain or 20,000 microstrain ($\mu\varepsilon$). The ultimate strength of bone is ~ 25,000 $\mu\varepsilon$ and the physiologic range of bone loading is ~ 200 to 2500 $\mu\varepsilon$. Under the normal range of ~ 200 to 2500 $\mu\varepsilon$, the accumulated fatigue damage is repaired by remodeling, so that the bone mass and its structural integrity remain intact. Strains exceeding

2.500 $\mu\varepsilon$ result in bone formation due to subperiosteal hypertrophy, in order to reduce surface strain. Strains ranging between 50 and 200 $\mu\varepsilon$ result in bone surface resorption due to disuse atrophy. Strains overexceeding the 4.000$\mu\varepsilon$ result in stress fractures or bone raptures because the repair process fails to compensate for the fatigue damage.

As for all postnatal mammalian load-bearing bones a strength-safety factor (SSF) exists, an analogous SSF may exist for alveolar bone. The SSF is defined as the ultimate strength of a load-bearing skeletal organ, divided by the typical peak stress (force per unit area) caused by a subject's voluntary physical activities expressed by muscle forces. According to Frost (2003b), the SSM in properly-adapted young adult load-bearing mammalian cortical bones would equal 6. In this way, such bones are about six times stronger than the minimum required to keep voluntary (muscular) loads from breaking them. Thus an analogous SSF for the alveolar bone may exist and it may be taken into account for the exerted orthodontic forces.

In the light of the mechanostat theory, tooth movement can be perceived as a coordinated expression of remodeling activity. Bone surface resorption during sustained tooth movement could be an atrophic response to suboptimal loading, or a fatigue-failure reaction to excessive tensile strain. The initial PDL osteoclastic response to tooth movement appears to be related to hypertrophic and fatigue-failure mechanisms.

During orthodontic tooth movement, exerted loads result in strains that produce mechanical bending of the alveolar bone. Bone deformation was first recognized by Angle (1907) and brought back to focus, in the orthodontic literature, by Baumrind (1969).

Mechanical deformation, the result of the application of an external load to a bone, is detected by osteocytes, that are sensitive to stress exerted on intact bone, functioning as mechanosensory cells. Signals generated by mechanical loading are transmitted between bone cells through gap junctions and between cells and extracellular matrix through hemichannels. Both gap junctions and hemichannels provide important signaling pathways for osteocytes, osteoblasts and osteoclasts and play crucial regulatory roles during the various phases of bone remodeling processes (Jiang et al 2007). Furthermore, gap junctions and hemichannels play important role in the regulation of mechanotransduction in bone, following orthodontic tooth movement (Gluhak – Heinrich et al 2006).

REGULATORY MOLECULES IN TOOTH MOVEMENT

Extensive biologic and molecular research on proteins and processes that signal cell and tissue reactions, following orthodontic tooth movement, has been carried out during the last thirty years. Various protein molecules are involved, namely cytokines and genes that control mainly bone remodeling processes.

Prostaglandins (PGs) and Leucotrienes (LTs)

Prostaglandins (PGs) are important molecular mediators of mechanical stress and they are involved in the transduction of mechanical information into biologic response during the different phases of the tooth movement process

(Harell et al. 1977). PGE_1 and PGE_2 act directly on osteoclasts and stimulate bone resorption, by increasing the number and the capacity of bone resorbing cells. Also leukotrienes (LTs) play a role in tooth movement as stimulators of bone resorption. It has been shown that local prostaglandin injections may increase the rate of tooth movement (Yamasaki et al 1980) and that disturbance of the LT pathway causes a reduction of tooth movement (Mohammed et al. 1989).

Cytokines

Cytokines are low-molecular weight extracellular proteins that mediate immunological responses and affect bone metabolism, acting in an autocrine or paracrine fashion. Cytokines involved in tooth movement include the interleukins (Ils), growth factors (GFs), tumor necrosis factors (TNFs), interferons (IFNs), and colony-stimulating factors (CSFs).

IL-1 mediates in attracting leukocytes, osteoclasts and osteoblasts and promotes bone resorption (Heath 1985, Jager et al 2005). TNFa stimulates directly the differentiation of osteoclast precursors into osteoclasts in the presence of macrophage colony-stimulating factors (M-CSF) and advances bone resorption (Davidovitch et al 1988). Transforming growth factors (TGF_B) enhance synthesis of collagen and noncollagenous proteins after force application (Davidovitch 1995). IFNγ causes bone resorption by apoptosis of effector T cells (Alhashimi et al. 2000).

The rather recently discovered RANKL/RANK/Osteoprotegerin (OPG) system (Simonet et al 1997, Lacey et al. 1998) and its role in osteoclast differentiation and activation, during bone remodeling, seems to enhance a new insight into bone physiology. The discovery of RANKL, RANK and OPG provide the final common pathway to the molecular and physiological mechanisms that control osteoclast differentiation and activation, and, hence, may actually influence orthodontic tooth movement.

RANK and RANKL are new members of the TNF receptor ligand family. RANK is the receptor activator of nuclear factor-κ B, also called osteoclast differentiation factor (ODF), TNF-related induced cytokine (TRANCE), or osteoprotegerin ligand (OPGL). RANKL is the RANK ligand, a membrane-bound TNF-related factor, that is expressed by osteoblast-stromal cells. RANKL is required for the differentiation of preosteoclasts into mature osteoclasts and for osteoclastic activity. Osteoprotegerin (OPG), a secreted TNF receptor, and RANKL are expressed by osteoblast-stromal cells and the ratio of these proteins may modulate the ability of these cells to stimulate osteoclast differentiation and the rate of bone resorption.

OPG functions as a soluble decoy receptor for RANKL and it competes with RANK. The biological effects of OPG are opposite of the RANKL-induced processes, because OPG prevents RANKL interaction with its receptor RANK. Therefore, an excessive amount of OPG results in osteopetrosis (Simonet et al 1997), while an absence of OPG, due to genetic factors, results in osteoporosis (Bucay et al. 1998).

It seems that there is a genetic mechanism that controls the coupling of the bone resorption and formation and this is expressed by the system RANK/

RANKL/OPG. Roberts and coworkers (2006) propose the following sequence of biologic events:

1. Bone and dentine microdamage results in release of inflammatory cytokines and exposure of mineralized collagen to extracellular fluid. 2. T-cells produce the ligand RANKL, which induces osteoclast histogenesis 3. Preosteoclasts have RANK receptors which are activated by the RANKL to form osteoclasts 4. During bone resorption, growth factors are released and stimulate preosteoclasts to produce OPG (a decoy receptor that binds RANKL) 5. Mononuclear cells move in and coat the resorbed bony surface with cementing substance 6. Perivascular osteogenic cells differentiate to preosteoblasts 7. As a last stage, osteoblasts form new bone and fill the resorption cavity.

There is some evidence of a role for RANKL and OPG in osteoclast activation during orthodontic tooth movement. Shiotani and coworkers (2001) reported RANKL protein in osteoblasts, osteocytes, fibroblasts and osteoclasts during application of orthodontic forces. According to Oshiro and coworkers (2002) there is no difference in RANKL expression between OPG-deficient and normal mice, after application of orthodontic forces, although there is severe alveolar bone resorption in OPG-deficient animals. The above findings support the idea that the relative expression of RANKL and OPG contributes to osteoclast activity.

Kanzaki and coworkers (2004) reported that local OPG transfer to periodontal tissue inhibited RANKL mediated osteoclastogenesis and inhibited experimental tooth movement. Furthermore Kanzaki and coworkers (2006) state that local RANKL gene transfer might be a useful tool, not only for shortening orthodontic treatment time but also for moving ankylosed teeth.

Low and coworkers (2005) concluded that RANKL and OPG levels were seen to increase in the environment during root resorption, following the application of heavy forces. Nonetheless, when RANKL was detected, it was only in association with orthodontic forces. According to these researchers (2005) further investigations are needed in order to determine the precise localization of OPG and RANKL expression in the rat jaw, following the application of heavy orthodontic forces.

Nevertheless, it seems that the RANK/RANKL/OPG system gives new insight into the biology of tooth movement and may play a crucial role in future orthodontic treatment. Overall, the influence of local administration of RANKL, RANK and OPG on the rate and quality of orthodontic tooth movement needs further thorough study.

TOOTH MOVEMENT: INFLAMMATORY PROCESS OR EXAGGERATED PHYSIOLOGIC RESPONSE?

The question that arises from the biologic and molecular study of orthodontic tooth movement is whether the tooth movement is a result of an inflammatory process or an exaggerated physiologic tissue response. According to Meikle (2006) tooth movement is a sterile process and the extent of any damage to the tissue will depend upon the magnitude of the applied force. Furthermore, "tooth movement does not meet the four classical criteria for inflammation (redness,

swelling, heat and pain) perhaps apart from the pain". On the other hand, Roberts and coworkers (2006) state that orthodontic tooth movement is a mechanically mediated inflammatory process.

Regardless of what it really is, tooth movement occurs as the result of therapeutic strains and loads that are used during orthodontic tooth movement. It is clear that orthodontists treat disorders which, according to Frost (2003a), are healthy departures from normal means, but not departures that impair an organ's health or represent diseases. It is also clear that almost all molecular genes that are involved in the inflammatory process are present during the various stages of orthodontic tooth movement. Also, inflammations are distinguished into those of microbial and non-microbial origin, and it is known that different types of arthritis are due to typical inflammatory processes of non-microbial origin. The existence of a therapeutic load that does not impair health or function causes the alveolar process to adapt in size, position and architecture, and the bone remodeling processes are involved in the osseous adaptation to this therapeutic intervention. It is thought then, that orthodontic tooth movement is a loading phenomenon that deviates from normal averages and it is mediated through nephron equivalent mechanisms (Frost 2003a) that stimulate inflammatory pathways.

AGING AND TOOTH MOVEMENT

Orthodontic tooth movement is dependent on the magnitude, direction and duration of the force applied. Also, it is dependent on the patient's general health situation as well as on the patient's local health conditions or on drug intake (Tyrovola and Spyropoulos 2001). Age is one of the factors that differentiates the periodontal tissues cell population and vascularization (Stahl et al. 1969). Bone density is less prominent in young versus mature animals (Burnell et al. 1980) and, under the same conditions, a greater amount and rate of tooth movement in younger rats may be expected (Bridges et al 1988). Furthermore Tanne and coworkers (1998) suggest that there is a delay in tooth movement in adult humans due to a reduction in the biological response of the PDL. Kabasawa and coworkers (1996) reported that bone formative activity of osteoblasts and bone resorptive activity of osteoclasts declined with age in the normal rat alveolar bone surrounding the maxillary molars. However, no difference in number, size and activity of osteoclasts and osteoblasts, following mechanical stress of the rat alveolar bone, was found among different rat ages. Also, Ren and coworkers (2003) state that besides a delay in the onset of tooth movement in adult animals, tooth movement could be equally efficient in adults once it had started. This may be due to a decrease in RANKL/OPG ratio in the gingival crevicular fluid during the early stages of orthodontic tooth movement in adult individuals (Kawasaki et al 2006).

However, whilst in the early adulthood minerals and cells are in a greater concentration into the bone tissue, in the later years of life due to the age related loss of bone (Wronski et al 1989) senile osteopenia is apparent. Also, in women over fifty years of age postmenauposal osteoporosis is established due to estrogen deficiency. Therefore, orthodontists must be aware of the fact that, in older patients, osteocytes are fewer and the bone is less dense. Also it has been found that in

2-years-old animals the total volume of stromal cells, part of which constitutes the stem cell compartment of the osteogenic lineage, is 1/4 of that found in 1-month-old animals and 1/3 of that found in 6-months- old animals. Thus, it has been concluded that the main factor causing bone loss with age is a diminished maturation of pre-osteoblasts into osteoblasts (Roholl et al 1994).

In one of our rat experiments, where orthodontic tooth movement was achieved following ovariectomy, we concluded that osteoporosis affected both the rate and the quality of tooth movement. Ovariectomized rats (i.e. osteoporotic) showed greater orthodontic movement of the loaded teeth as compared to controls and disturbed lamellar structure was found in the alveolar bone of the experimental osteoporotic group (Tsolakis 2002, Tsolakis et al. 2003).

HISTOMORPHOLOGICAL FINDINGS IN AREAS ADJACENT TO AND REMOTE FROM THE LOADED TOOTH

Recent studies have suggested that, when a bone is loaded, osteocytes sense this load through the interstitial bone fluid in the canaliculae and osteocyte lacunae, and generate signals that may influence cells near or on a distant bone surface. Transmission of the afore-mentioned signals is through the functional syncytium of osteocytes, bone lining cells and stromal cells, and the gap junctions and hemichannels between them, which play important roles in the regulation of mechano-transduction in bone (Marotti 2000, Frost 2004, Gluhak – Heinrich et al. 2006). This syncytium of cells, that detects and transmits information, is considered to play a role in the control of remodeling (Marotti 2000). Hence, strains on a certain bone area may influence neighboring structures of the same bone.

In order to investigate this possibility, we carried out different rat experiments, and we found: 1 Distal and remote alveolar bone responses (RAR), 2. Regional acceleratory phenomena (RAP) and 3. Distraction like phenomena (DLP), in adjacent and remote areas of the bone.

In these experiments, the following experimental procedure was used. Orthodontic rat molar movement was achieved by the application of a closed coil spring extending from the upper right first molar to the upper right central incisor. No orthodontic force was applied to the upper left first molars which were used as control teeth. The coil spring was 1cm in length, and its activation for 0.25cm produced a force 60gr that is considered a heavy orthodontic force. The orthodontic force lasted for 14 days. Histologic examinations of the interradicular areas of the upper right and left first, second and third molars were performed. Also, the cortical bone of the maxilla, mesial to the upper right and left first molar, as well as the cortical bone of the maxilla, distal to the upper and left third molars, were examined histologically for all animals.

DISTAL AND REMOTE ALVEOLAR BONE RESPONSES (RAR)

It has been well established that, following the loading of a tooth, alveolar bone formation occurs on the tension side of the root, and resorption is observed

on the pressure side, but with differentiation according to other micro sub-tension and sub-strain areas (Spyropoulos 2006). Although this remodeling process is the basis of clinical orthodontic practice, the impact of applied forces on the teeth adjacent and distal to the loaded ones, has not been thoroughly studied. One of our investigations was undertaken to examine the effect of the application of orthodontic forces on the alveolar bone of the teeth adjacent and distal to the loaded ones, in normal adult female rats (Tsolakis et al 2008a).

The following histological findings were observed in the paradental tissues of the non-loaded teeth. On the side of the rat maxilla, where the 60gr orthodontic forces were applied, the alveolar bone in the interdental space between the 2nd and 3rd molars revealed remodeling activity. Extensive resorption was noticed in the direction of movement (Fig. 19-1), whereas bone formation appeared on the rear side of the interdental space and was less prominent. The osseous tissue of the interradicular area of the right 2nd molar showed internal resorption with preservation of the alveolar bone architecture. On the left side of the rat maxilla, where no orthodontic forces were applied, the alveolar bone in the interdental space between the 2nd and 3rd molars was denser and did not show any activity. Nevertheless, the osseous tissue of the interradicular area of the 2nd left molar also revealed internal resorption.

To our knowledge, based on the available literature, changes occurring at the teeth and supportive bone, adjacent or distal to the loaded tooth , have not been studied. The study mentioned above examined this area and the changes therein.

The applied force of 60gr* is considered a heavy orthodontic force and it was purposely designed in order to influence bone remodeling activities. According to Frost (1995, 1997), the bone strength index as well as the minimum effective

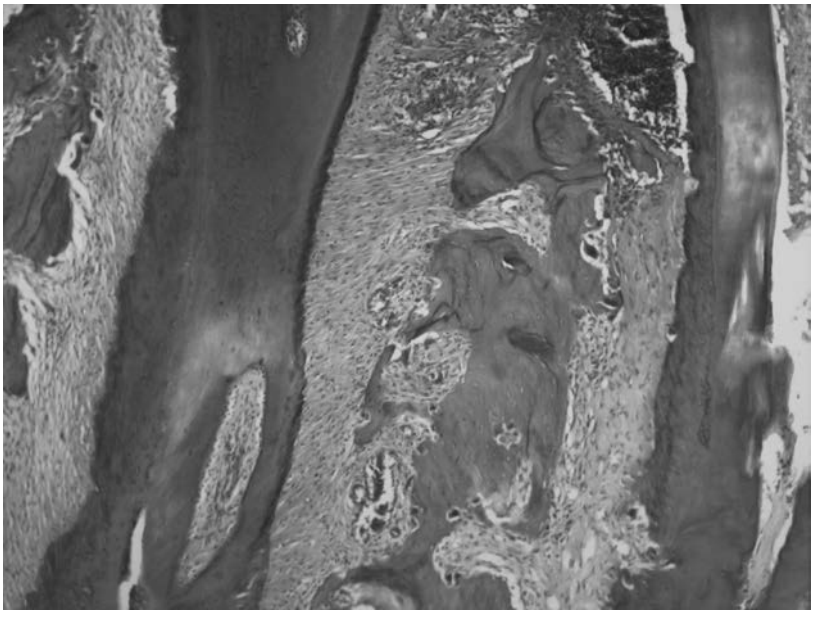

Figure 19-1.
External resorption in the interdental area

strain, vary for the same organs in different species and the strain magnitudes in skeletal organs depend on the relationship between their strength and the size of the loads exerted on them. Taking into account the above mentioned feature of the Utah paradigm, the different anatomic and functional characteristics of the two species, human and rat, as well as the different forces generated from the masseter muscles and applied to the molar teeth of the two species, a force of 60gr* applied to the rat molar may be comparable to a force of 480gr* applied to the human molar tooth. These forces may be compared to the orthopaedic forces applied to human molar teeth through application of extraoral appliances.

In the biology of tooth movement, the pressure and tension hypothesis, strictly related to the PDL area, was used to explain changes around the orthodontically moved tooth (Rygh 1972; Reitan 1985; Masella and Meister 2006). Although it is thought that forces applied to individual teeth may affect the alveolar bone of the proximal teeth, as well as the relative cortical bone, there has been no attempt to study possible changes in areas neighboring to the loaded ones. Our findings support the conclusion that the changes in the alveolar bone in areas distal and remote to the mechanically loaded ones are similar to the changes observed in the alveolar bone of the loaded tooth. Theses results confirm that bone bending and remodeling of the periodontal tissues (Epker and Frost 1965; Meikle 2006) are interrelated in orthodontic tooth movement.

REGIONAL ACCELERATORY PHENOMENA (RAP)

A Regional Acceleratory Phenomenon (RAP) may be produced by traumatic and stress fractures, lacerations, as well as by infectious and non-infectious inflammations, and by orthodontic forces (Frost 1983, 2004). During a RAP, all ongoing regional processes are accelerated and it seems to represent a biologic "SOS" mechanism, during serious noxious stimuli (Jee and Yao 2001). Moreover, RAP normally improves the body's ability to resist and manage established infections, as well as the resulting healing, in all hard and soft tissues (Frost 2004).

Recent concepts interrelating bone biology and skeletal bio-mechanics consider microdamages of bone tissue as events that initiate bone remodeling (Martin 2003, Parfitt 2002). Also, it seems that the initial PDL response to orthodontic loading is related to hypertrophic and fatigue mechanisms (Katona et al 1995, Roberts 2000). Furthermore, Verna and coworkers (2004) suggest a role for microcracks, in the initiation of bone remodeling, following the application of orthodontic forces.

Our hypothesis states that, if orthodontic forces exerted on bone are perceived as traumatic or non-infectious stimuli by the local tissues, followed by microdamage, RAP phenomena may potentially appear in the surrounding bone tissue. Thus, the purpose of one of our studies was to determine any RAP phenomena following the application of heavy orthodontic forces on rat molars (Tsolakis et al 2008b).

The following histological findings were observed. Cortical bone of normal rats, on the right side, where the orthodontic forces were applied mesially to the right first molar, showed distortion of bone structure and woven bone formation (Fig. 19-2). In comparison, in the left side, where no orthodontic forces were

applied, the bone ahead of the left first molar was lamellar and well oriented. The cortical bone thickness, of both sides, remained unaffected.

The Regional Acceleratory Phenomenon (RAP) was first recognized in the orthodontic and dentofacial orthopedic literature by Melsen (2001). In her study, woven bone formation was observed ahead of the alveolus, i.e. in the direction of the displacement in normal rats, and was interpreted as a RAP. In our study, the application of heavy forces evoked accelerated changes, as shown by histological sections in normal and mature animals. It seems that the strains exerted on the surrounding bone tissues, ahead of the loaded teeth, affected remodeling processes in the bone.

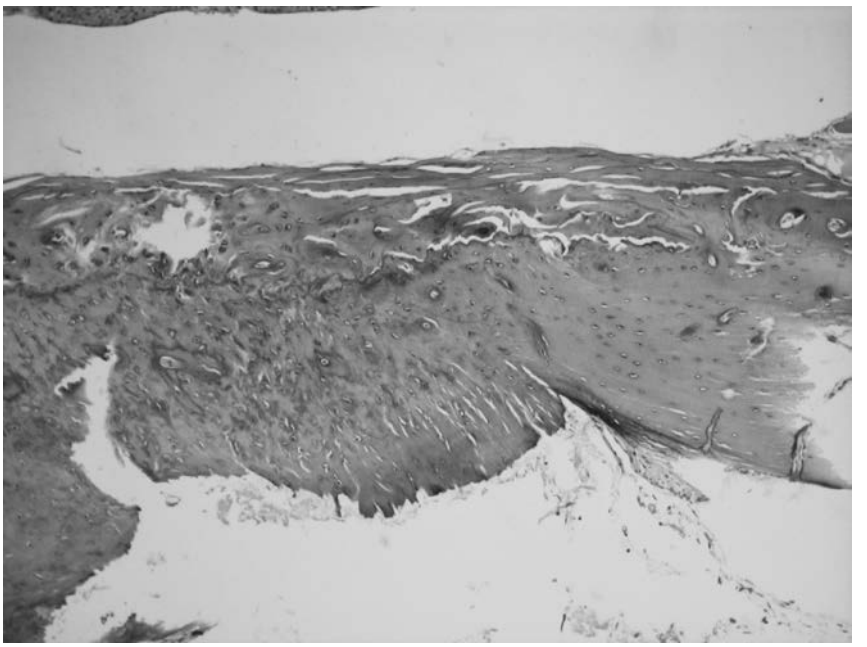

Figure 19-2.
Woven bone formation

In conclusion, the application of orthodontic tooth forces on normal mature rats, in our study, created a RAP ahead of the loaded teeth of normal rats, which was demonstrated histologically, as seen in Fig. 19-2. The RAP was expressed with distorted bone structure and woven bone production at the area coinciding with the direction of the displacement of the tooth.

DISTRACTION LIKE PHENOMENA (DLP)

So far, clinical changes observed in the anterior maxillary region of orthodontic patients, following the application of a distal headgear to the upper molars are not documented in the light of contemporary bone biology. Also, the effects of the application of facial mask or reverse headgear on the maxillary bone, to be interpreted, demand a biologic scheme. Our hypothesis considers mechanical

bone deformation, distal to the loading area, as a possible mechanism for bone generation due to the applied forces. Since spongiosa provides mechanical support for teeth and it transfers loads from teeth to cortical bone and vice versa (Frost 2004), heavy forces applied to the dentoalveolar complex may influence adjacent and distal cortical bone. In this way strains applied to teeth act as a mechanical stimulus to the underlying cortical bone and they are converted to thresholds that influence bone / or remodeling patterns.

In one of our studies the purpose was to test our aforementioned hypothesis, and to investigate the morphological changes in cortical maxillary bone, following the application of heavy orthodontic forces, in mature male rats (Tsolakis et al. 2008c).

The following histological findings were observed. The cortical bone, on the right side, the site of retraction, revealed extensive structural distortion, without, however, obvious discontinuation. Bone hypertrophy and subperiosteal callus formation was noticed (Fig. 19-3). The callus was observed along the cortical bone up to the level of the third molar and along the curvature of the upper jaw. Vascular nutrition was remarkable.

In comparison, on the left non-retracted side, the cortical bone at the level of the apices showed no activity from the first molar on to the third molar.

It has been considered that orthodontic tooth movement may be initiating a form of "distraction osteogenesis" in the PDL (Liou and Huang 1998). Also, in the case of rapid orthodontic tooth movement, the process of osteogenesis on the tension side is similar to a distraction osteogenesis response, aiming at avoiding the formation of an infrabony defect on this side.

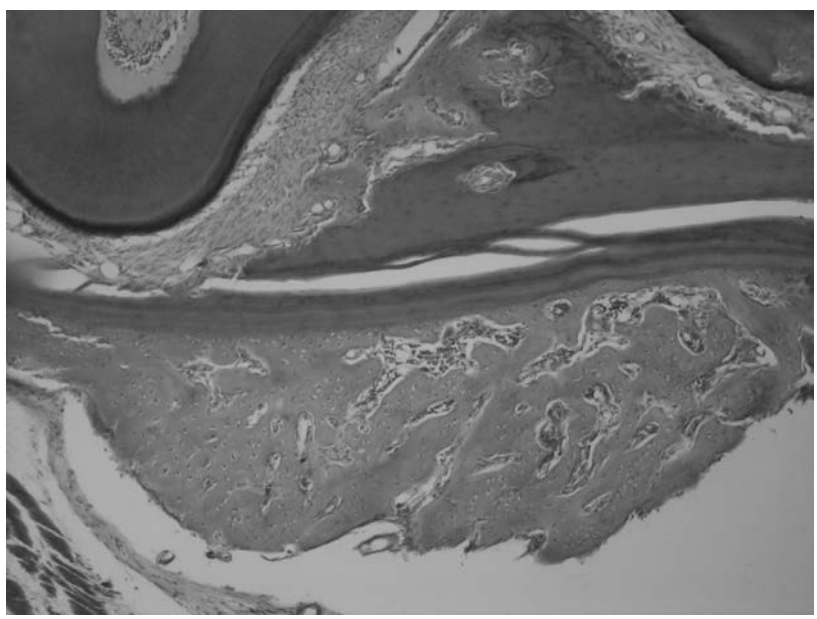

Figure 19-3.
Subperiosteal callus

In our experiment, the applied orthodontic force of 60gr* which is considered as a heavy force (its application exceeds 4000*με)*, distracts the PDL, the adjacent bone, but also influences the cortical bone distal to the point of application up to the curvature of the rat maxilla. The osteogenic reaction to these forces, observed in our experimental rats, is similar to a bone distraction phenomenon, expressed by the formation of subperiosteal callus on the outer bony side, offering a cushioning and splint effect at the affected site. The forming callus consists of fiber-osseous tissue and follows an intramembranous ossification process rather than a cartilaginous and vascular invasion.

Reitan and Kvam (1971), in their experimental rats, after loading with 4, 12, 30gr* of force, observed and described, without further explanation, thick bone deposition, at a certain distance from the undermining bone resorption site, along the curvature of the upper jaw. Glickman and Smulow (1965) referred to the rapid formation of compensatory bone layers following increasing pressure, during tooth movement, as a "buttressing bone formation" phenomenon, due to the alteration of occlusal contacts. From all the above findings, it can be deduced that a distraction type callus and distraction bone type develops during the application of heavy orthodontic forces in rats. The bone tissue resembles that of distracted bones and one may refer to this process as bone DLP, which, apparently, constitutes a protective mechanism and bone formation in response to disruptive mechanical stresses applied to a certain bone region during orthodontic movement.

EPILOGUE

The concept that will probably change in the coming years will be based on the increasing insight into the biology of bone formation and resorption, via interventions in the signaling pathways and molecules by genomic and hormonal regulation, and this will probably be followed by related breakthroughs. Firstly, pathological situations of general bony health conditions will be resolved, while craniofacial growth and development, as well as orthodontic tooth movement will be influenced in respective individuals. By establishing normal bone physiology, in patients with hormonal and proteome's disturbances, we may alter the rate and quality of orthodontic tooth movement resulting in faster and more stable results.

Orthodontic tooth movement is a local loading phenomenon mediated by hundreds of genes that present interplaying and antagonistic features, through genetic and epigenetic signaling. Mechano-biologic response to orthodontic loading depends on interpatient variations in bone and connective tissue cell populations, genes and signaling patterns. In non-syndromic individuals presenting with normal bone remodeling pathways, alteration of the local environment, namely the relationship of the tooth in occlusion, may be mostly a local tissue loading intervention that triggers normal patho-physiological processes.

It is however obvious that, still the magnitude and the nature of the applied forces is a crucial factor for orthodontic tooth movement in average normal individuals. The "mechanostat" theory points out the importance of strain levels and their influence on bone-remodeling homeostasis. Intervention in molecular

mechanisms may influence orthodontic tooth movement reactions in terms of aging and general health conditions that deviate from normal. Also, patterns of alteration of secreted signaling molecules in blood and/or crevicular fluid may influence the nature of the applied mechanics and the magnitude and duration of the therapeutic forces applied.

Consequently, there is a need for categorization of the loading strains in alveolar and cortical bone, as well as the distinction between orthodontic and orthopedic forces, in the light of contemporary mechanobiology. All functioning gene mechanisms, following orthodontic tooth movement, are secondary to applied strains in bone and there is still a lack of knowledge regarding the significance of magnitude of the applied forces. Also, a gap of knowledge exists as regards the end tissue reactions like distal and Remote Alveolar bone Responses (RAR), Regional Acceleratory Phenomena (RAP) and Distraction Like Phenomena (DLP) following orthodontic treatment in normal individuals. As a conclusion, it can be supported that, any future relevant research must be oriented not only to the molecular level, but also to the identification and categorization of the various strain levels, as well as to the local tissue bio-adaptability and reaction. The latter will hopefully lead to reductions in treatment time as well as improving the quality of orthodontic results.

20

Genetics of the Craniofacial Complex

Richard J. Sherwood
Dana L. Duren

The past two decades have seen a significant transition in the biological sciences largely due to the advances in genomic research. Craniofacial research, most notably research into craniofacial anomalies, has moved from categorization of syndromes based on phenotypic patterns to the identification of specific genetic mutations responsible for these syndromes. One cannot help but be impressed by the wealth of detailed genetic information that is rapidly becoming available for human dental and cranial disorders (Cohen, 2002b; Shieh et al., 2006; Riise et al., 2002; e.g., Brennan and Pauli, 2001; Mulliken, 2002). It is also clear, however, that advances in the genetics of craniofacial disorders have not provided unambiguous answers to important questions regarding the genetic architecture of normal and abnormal craniofacial variation. This chapter will explore some of the recent advances in, and approaches to, elucidation of the genetic underpinnings of the anatomy of the human craniofacial complex.

Much of the current knowledge regarding the effects of genes on human craniofacial variation comes from the examination of dysmorphic syndromes. Numerous craniofacial syndromes have been identified and classified, and the identification of genetic defects associated with these syndromes is becoming common. As more and more studies identifying a genetic role in craniofacial syndromes are reported, the importance of understanding the role of individual genes, interactions between genes, and interactions between genes and the environment becomes critical.

As noted, searching for the genetics underlying disease states has provided inspiration for a wide variety of research. The popular press frequently portrays this as a investigation by the wiliest of detectives searching for an elusive criminal, one who inflicts great hardship on their innocent victims. Despite claims in the national news that "*the*" gene for obesity or some other condition has been discovered, one may ask why the cure for these conditions doesn't follow quickly from these discoveries. The simple answer to this question is that there is no simple answer. We are somewhat programmed to believe that a condition has a single cause, malaria

is caused by a *Plasmodium* parasite, tuberculosis by a *Mycobacterium*, influenza by an *influenza* virus, therefore, genetic disorders should be caused by "*a*" mutation in "*a*" gene. The truth, however, is that there are at least four different *Plasmodium* parasites, several *Mycobacterium* species, new influenza variants every year, and that genetic disorders may be caused by mutations in one or several genes.

To understand the genetics of craniofacial anomalies it is important to understand the variety of ways genetic disorders may present themselves, and the range of additional factors that may lead to the wide variety of phenotypic manifestations. To do this we will examine three examples of craniofacial disorders; cleft lip and palate, holoprosencephaly, and craniosynostotic disorders. Each of these conditions presents a set of complications when searching for the underlying genetic etiology, and these complications are as important to understand as are the underlying genes themselves.

Following the presentation of these three disorders, a brief discussion of current approaches in genetic epidemiology will demonstrate how the search for genes responsible for normal variation in craniofacial traits is possible.

Cleft Lip and Palate

Cleft lip with or without cleft palate is among the most common congenital deformities found in humans. That cleft lip and palate can have a significant genetic component is without question, but there can also be a significant environmental (i.e., non-genetic) component. It is frequently noted that clefting tends to cluster in families but that there is a high discordance rate in monozygotic twins (Arosarena, 2007) indicating a multifactorial mode of inheritance. Orofacial clefting can present itself as a variable phenotype with clefting restricted solely to the palate (often designated CPO – cleft palate only), or can involve the lip and the palate (designated CL/P). The phenotype varies based on the relative involvement of the lip and palate, and ranges from unilateral to bilateral, and from incomplete to complete. Clefts are also designated as non-syndromic when restricted to the lip and palate (accounting for up to 70% of cases; Ghassibe et al., 2006), or syndromic if other anomalies occur with the cleft. Syndromes with accompanying CL/P are numerous and may include postcranial anomalies such as short stature (Sonoda and Kouno, 2000; Mathieu et al., 1993; Zelante and Ruscitto, 2003), syndactyly (Richieri-Costa et al., 1985) or hypospadias (Joss et al., 2002; Schilbach and Rott, 1988). Because of the varied nature of these syndromes, identifying a specific genetic etiology for the CL/P in each case is difficult.

Because CL/P is a relatively common condition, with a clear familial history, there have been numerous studies searching for its genetic underpinnings. Table 1 describes some of the genes identified as causative factors in CL/P. While these genes are generally described as the cause of non-syndromic forms of CL/P in some individuals the clefting defect is associated with additional defects. The additional defects are often minor enough that these cases are still considered as non-syndromic. For instance, patients with IRF6 mutations frequently show pitting of the lower lip in addition to CL/P; this combination is generally diagnostic of Van der Woude syndrome. However, individuals with IRF6 mutations may

also possess more significant syndromic symptoms such as cutaneous or genital anomalies diagnostic of popliteal pterygium syndrome. Phenotypic variability such as that found with IRF6 mutations is an important consideration and will be discussed further below.

To understand the genetic etiology of CL/P, the growth and development of normal palate/lip formation must be understood. (See Chapter 13) Proper palate formation relies on adequate growth of the components, the palatal processes, and a mechanism to fuse those components. Failure to achieve adequate growth of the palatal processes will automatically result in a failure to fuse as the processes will not be approximated. Once the palatal processes are in approximation, fusion must then occur. This involves a degeneration of the epithelium overlying the palatal processes and the descending nasal septum, followed by a subsequent coalescence of the underlying mesenchyme (Sperber, 1989). Failure of any of these processes will result in the clefting phenotype.

Given that palatogenesis is a complex phenomenon it is not surprising that multiple genes are integral to proper palatal formation. As can be seen in Table 1, specific genes have been identified as controlling specific aspects of palate formation. For example, early palatal growth is influenced by FGF10 prior to elevation of the shelves, while mutations in GLI2 are linked to a disruption in the elevation of the palatal shelf. SPRY2 and MSX1 are important in growth of the palate once they have elevated and SATB2 and IRF6 are important for fusion of the palatal elements. The interactions of these genes are also important and several pathways have been described that incorporate the genes described as well as additional genes such as some of the bone morphogenetic proteins (BMPs) or the homeobox Sonic Hedgehog (SHH). (Murray and Schutte, 2004; Zhang et al., 2002.)

In addition to genetic influences, several environmental factors have been implicated in the etiology of CL/P. A number of teratogenic substances that cause a variety of embryologic disorders have also been implicated in CL/P. Murray

Table 1. Genes known to affect palate development.

Gene	*Chromosome*	*Developmental aspect affected*	*Reference*
SATB2	2q32-q33	Shelf elevation or adhesion	(FitzPatrick et al., 2003)
IRF6	1q32-q41	Palatal fusion	(Ghassibe et al., 2006; Ghassibe et al., 2005)
GLI2	2q14.1-21	Disrupted palatal shelf elevation	(Mo et al., 1997)
MSX1	4p16.1	Palatal shelves elevate normally but don't make contact or fuse. Regulates expression of BMP4.	(Zhang et al., 2002)
SKI	1p36.3		(Lu et al., 2005)
SPRY2	13q31.1	Medial directed growth	(Welsh et al., 2007)
FGF10/FGFR2B	5p12	Disruption to early palatogenesis prior to elevation	(Rice et al., 2004)
TGFβ3	1p33-p32	Defective fusion	(Welsh et al., 2007)

and Schutte (2004) describe maternal smoking, pharmaceutical use including benzodiazepines or phenytoin, or the pesticide dioxin (see also Romitti et al., 2007), as important risk factors for CL/P. Murray and Schutte also suggest that infection or the immune response to infection, as well as nutrients and cholesterol metabolism are also important environmental influences that may increase the risk of CL/P, although there is less evidence to support these ideas. Finally, the mechanical environment may also play a role during craniofacial morphogenesis. The effects of compressive and shear forces on chondrogenesis and osteogenesis have been documented (Carter et al., 1987a; Carter et al., 1987b; Carter, 1987) and have been suggested as critical in prenatal craniofacial development (Radlanski and Renz, 2006). While it can be difficult to incorporate into morphogenetic models, it is important to consider the range of effects introduced by environmental influences.

Holoprosencephaly

One of the most dramatic of craniofacial anomalies is holoprosencephaly. Holoprosencephaly is defined by the lack of cleavage of the prosencephalon (forebrain) resulting in a monoventricular cerebrum (Cohen, Jr., 2006). Along with brain malformations, the most severe forms of holoprosencephaly include a suite of craniofacial abnormalities characterized by CL/P, cyclopia, and formation of a proboscis above the single eye. Less severe facial forms of holoprosencephaly may be characterized by levels of hypotelorism or a single maxillary central incisor. Richieri-Costa and Ribeiro (2006) describe a holoprosencephaly-like phenotype in which some of the milder facial forms, including the single incisor, flattened nose, and a poorly developed filtrum, are found in the absence of neural anomalies.

As with CL/P, a number of genes have been implicated in holoprosencephaly and these are presented in Table 2. Numerous loci (designated HPE1, HPE2, etc.) have been identified as etiologic factors in holoprosencephaly and the affected genes have been identified. A common factor in many cases of holoprosencephaly

Table 2. Genes with known relationships to holoprosencephaly.

Gene	*Chromosome*	*Region affected / Regulatory action*	*Reference*
HPE1	21q22.3		OMIM 236100
HPE2 (SIX3)	2p21	Development of anterior neural plate	(Wallis et al., 1999)
HPE3 (SHH)	7q36	Expressed in floorplate of neural tube	(Roessler et al., 1996)
HPE4 (TGIF)	18p11.3	Bifurcation of brain, establishment of ventral midline structures	(Gripp et al., 2000)
HPE5 (ZIC2)	13q32	Expressed in dorsal neural tube	(Brown et al., 1998)
HPE6	2q37.1		(Lehman et al., 2001)
HPE7 (PTCH1)	9q22.3	Decreases SHH signaling	(Ming et al., 2002)
HPE8	14q13		(Kamnasaran et al., 2005)
HPE9 (GLI2)	2q14	Mediator of SHH signaling	(Roessler et al., 2003)

is a disruption in the sonic hedgehog (SHH) signaling pathway. SHH is a powerful morphogen and is critical in many aspects of embryonic patterning including expression in the floorplate of the neural tube and notochord. Reduced expression of SHH disrupts brain formation and can lead to holoprosencephaly. Patched (PTCH1) is an SHH receptor, and GLI2 is a mediator of SHH (Ming and Muenke, 2002). Not surprisingly, mutations in PTCH1 or GLI2 have also been shown to lead to holoprosencephaly.

As noted, there is considerable phenotypic variation across holoprosencephaly cases. Ming and Muenke (2002) have speculated that a contributory factor to the extent of this variation is the interaction of two or more genes. They identify mother-offspring cases where both demonstrate a SHH mutation but the child has an additional mutation. In these cases the mother is phenotypically normal but the child presents with holoprosencephaly. The importance of such gene interactions has been demonstrated in animal models. For instance, Seppala et al. (2007) has identified an interaction between GAS1 and SHH in mice. In this case mice with a deletion of GAS1 exhibit a mild form of holoprosencephaly whereas those with the deletion in addition to the loss of a single SHH allele exhibited a more severe phenotype.

A number of environmental factors have also been shown to increase the risk of holoprosencephaly. Maternal diabetes, ethanol exposure, retinoic acid exposure, and defects in cholesterol metabolism during gestation have been shown to increase the likelihood of holoprosencephaly in humans and/or animal models (Ming and Muenke, 2002). Many of these environmental factors may affect forebrain development in the same manner as some of the genes described above, via an alteration of the SHH signaling pathway. Given that any single case may have multiple factors acting, the sum total of these gene-gene and/or gene-environment interactions may explain the extent of phenotypic variation in holoprosencephalic cases.

Craniosynostotic Disorders

Another common craniofacial disorder is premature fusion of the cranial sutures (craniosynostosis). Craniosynostoses can occur in one or more sutures arresting cranial growth at that site. Because sutures are the primary site for growth in cranial bones, if craniosynostosis is left unchecked the cranium will become misshapen as underlying structures (e.g., the brain) continue to grow (Kimonis et al., 2007; Cohen, Jr., 2005). For instance, in synostosis of the sagittal suture mediolateral growth is compromised and the skull takes on a long narrow shape (scaphocephaly). When paired sutures such as the coronal or lambdoid sutures become synostotic they are frequently unilaterally affected resulting in an asymmetric head shape (plagiocephaly).

Craniosynostosis may occur alone (non-syndromic) or in concert with other symptoms (syndromic). Kimonis et al. (2007) report that over 180 syndromes involving craniosynostosis have been identified. A small sample of these are presented in Table 3. Along with the deformation of the cranium based on the pattern of suture closure, a number of craniofacial features, such as a beaked nose or

hypertelorism, are common to craniosynostic disorders. Because of developmental similarities of connective tissues throughout the body, it is not surprising that postcranial deformities are often associated with craniosynostotic syndromes. These postcranial deformities include shortened digits (brachydactyly) in Crouzon or Pfeiffer syndrome, skin anomalies in Beare-Stevenson Cutis Gyrata, syndactyly in Apert, Carpenter, or Saethre-Chotzen syndromes, or urogenital disorders in Apert, Baller-Gerold, or Antley-Bixler syndromes.

Table 3. Genes known to cause craniosynostotic disorders.

Disorder	*Gene*	*Sutures affected*	*Reference*
Muenke Syndrome	FGFR3	Coronal	(Kimonis et al., 2007)
Saethre-Chotzen Syndrome	TWIST1	Coronal, metopic, sagittal	(Kimonis et al., 2007)
Crouzon Syndrome	FGFR2	Coronal	(Kimonis et al., 2007)
Beare-Stevenson Cutis Gyrata	FGFR2	Coronal, lamboid	(Kimonis et al., 2007)
Pfeiffer Syndrome	FGFR2, FGFR1	Multiple	(Kimonis et al., 2007)
Apert Syndrome	FGFR2	Coronal fusion with agenesis of sagittal and metopic sutures	(Cohen, Jr., 2005)
Craniofrontonasal Dysplasia	EFNB1	Coronal	(Kimonis et al., 2007)
Baller-Gerold Syndrome	RECQL4, TWIST1	Coronal	(Kimonis et al., 2007)
Antley-Bixler Syndrome	POR	Multiple	(Kimonis et al., 2007)
Boston-Type Craniosynostosis	MSX2	Coronal	(Kimonis et al., 2007)

The majority of craniosynostotic syndromes have been linked to mutations in genes related to fibroblast growth factor signaling pathways (Kimonis et al., 2007), specifically fibroblast growth factor receptor (FGFR) genes. Each FGFR contain 3 immunoglobulin-like domains, one transmembrane domain, and two tyrosine kinase domains (Kimonis et al., 2007; Cohen, 2002a). A limited number of mutations on the FGFR1 and FGFR3 are known to result in synostotic symptoms. The majority of mutations resulting in craniosynostosis are found on FGFR2 clustered on the IgIII domain (Figure 20-1).

It is interesting to note that there is not always a one-to-one relationship between a given mutation and the resultant phenotype. For example, the same mutation in FGFR2 (Cys278Phe) may result in an individual with either Crouzon or Pfeiffer syndrome. Conversely, a given syndrome, such as Pfeiffer syndrome, may result from a mutation in FGFR1 or a mutation in FGFR2. Furthermore, over 40 different TWIST mutations have been identified for Saethre-Chotzen syndrome (Cohen, 2002b). This heterogeneity has made some suggest that, instead of numerous individual distinct syndromes, there are only a handful of syndromes each with considerable variation along a continuum. This idea has been rejected as syndromes do tend to present a definable set of symptoms that breed true in

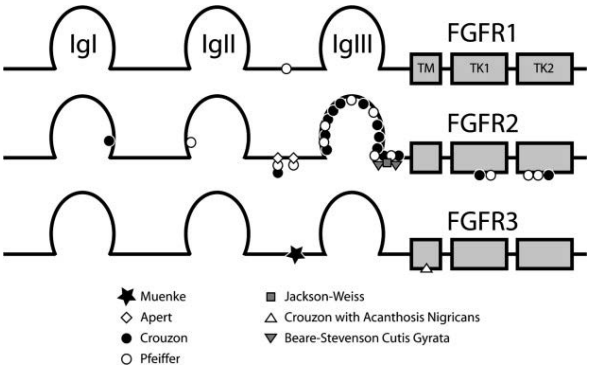

Figure 20-1.
Location of mutations on fibroblast growth factor receptors known to result in craniosynostosis. Note the majority occur in the IgIII domain of FGFR2. (From Kimonis et al. 2007).

families. Cohen and MacLean (1999) suggest several ways to integrate phenotypic and genotypic nomenclature that are likely to become standard practice as we continue to elucidate the these relationships. While their system may be a bit cumbersome, e.g., the simple Crouzon syndrome would be replaced by "Crouzon syndrome, FGFR2, Cys278Phe", such a system may become necessary for clarity.

Genetics of Craniofacial Anomalies

The three anomalies discussed above, cleft lip and palate, holoprosencephaly, and craniosynostotic disorders, provide excellent examples regarding the current state of knowledge of the genetic etiology of craniofacial disorders. It is important to note that in all three conditions a common phenotypic suite may have multiple genetic causes. In some cases the reasons for this are obvious. For instance, a perturbation in any of the developmental processes of palate formation may result in the phenotype of cleft palate. As characterization of the genes responsible for specific aspects of growth, elevation, and fusion of the palatal processes becomes clear, the relationship between multiple genes and a single phenotype is obvious.

Whereas specific genes have been linked to specific developmental processes in CL/P, the relationship between genotype and phenotype in holoprosencephaly is not that clear. In this case a single gene, SHH, seems to play a primary role in proper formation of the early brain and related structures. Rather than individual genes affecting specific aspects of brain development, genes associated with holoprosencephaly largely affect the expression of SHH which, in turn, leads to the dysmorphology. Similar to holoprosencephaly, craniosynostotic disorders seem to be strongly related to the affects of one primary gene, FGFR2. Multiple mutations have been identified on FGFR2 and these are responsible for a wide spectrum of syndromes.

Interactions between genes and between genes and the environment are clearly important in determining the phenotypic manifestation as well. As noted in the introduction, there still persists an expectation (or hope) that a given mutation

will produce a singular outcome. Even if the (non-genetic) environment were held constant, this expectation would not be warranted. The cumulative pleiotropic effects of genes and gene- gene interactions would be expected to produce a wide-range of phenotypes proportional to the number of genes involved. In other words, variability among normal genes would be expected to produce variable phenotypes when acting in concert with a mutated gene. When a diverse environment is introduced, the range of phenotypic expression is multiplied.

Genetic Epidemiology

It is clear from the previous section that substantial information regarding the genetic influence on the human craniofacial complex has been gained from investigation of the genetics of dysmorphic syndromes. While the advances made by examining the genetic etiology of craniofacial pathogenesis are significant, they do not provide an adequate characterization of the genetic background to normal craniofacial development and morphology. In animal models the study of normal development is accomplished via the careful study and manipulation of growing embryos. This approach is not possible when considering human development and, so, other techniques must be applied. Modern day quantitative genetics provides such an approach.

Quantitative Genetics of Craniofacial Form

The field of quantitative genetics has blossomed in the past decade. This is due in part to advancements in human genomics including rapid DNA sequencing (Ziebolz and Droege, 2007), the human genome project (Collins et al., 2003; Collins and Mansoura, 2001; Collins, 1997), and the HapMap Project (The international HapMap Consortium, 2003; Schmidt, 2003) . The field has also seen great advancements thanks to increased computing power and availability of powerful statistical software able to handle large data sets from large extended pedigrees (see for example, Almasy and Warren, 2005).

The term quantitative genetics refers to the analytical methods for decomposing phenotypic variation into its constituent genetic and non-genetic (referred to as environmental) components. That is, any quantitative trait, such as flexion of the basicranium (measured as the angle from basion to sella to nasion) is expected to vary between individuals. That variation is expected to be due, in part, to the influence of genes, but it is also expected that some proportion of the variance is due to non-genetic influences. These environmental influences may be numerous and are often hard to measure. Quantitative genetics works under the basic assumption that the degree to which a trait is controlled by genes can be determined by examining the distribution of that trait among and between families. Some basic concepts and terminology used are provided here.

Heritability is the proportion of variance in a trait attributable to genetic factors. There are two levels of heritability. There is broad-sense heritability, which takes into account all possible genetic contributions to the variance in the trait. Broad-sense heritability is expressed as $H^2 =$, where σ_G^2 is the total genetic variance

and σ_P^2 is the phenotypic variance. Most of the time, portions of these genetic contributions cannot be readily estimated or measured (such as the effects of dominant genes), and thus broad-sense heritability is less useful to the craniofacial quantitative geneticist. Narrow-sense heritability, on the other hand, considers only the additive genetic variance of a trait, σ_A^2. Narrow-sense heritability is expressed as $h^2 = \frac{\sigma_A^2}{\sigma_P^2}$. Population-based quantitative genetic methods are aimed at estimating the narrow-sense heritability of a trait.

Once it has been demonstrated that a trait is under genetic control the next question would be, do two traits exhibit the shared effects of a gene or set of genes? In quantitative genetics the measure of shared genetic effects (or pleiotropy) is measured as the genetic correlation (symbolized as ρ_G), which, like other measures of correlation, will vary between -1.0 and 1.0. In this measure, the value of the correlation will give an indication of the extent of shared genetic contribution between the traits. A value of 0 would indicate genetic independence and a value of 1 or –1 would indicate complete pleiotropy. The sign will indicate the nature of the relationship much as in phenotypic correlations. That is, for positive genetic correlations both traits would be expected to increase or decrease concurrently. With negative genetic correlations as the value of one trait increases the value of the other would expect to decrease.

It should be pointed out that the phenotypic correlation or ρ_P, commonly measured as a pearson product moment correlation, is related to the genetic and environmental correlations as $\rho_P = \rho_G\left(\sqrt{h_1^2}\sqrt{h_2^2}\right) + \rho_E\left(\sqrt{1-h_1^2}\sqrt{1-h_2^2}\right)$. That is, the genetic and environmental causes of correlation combine to give the phenotypic correlation. Heritability also plays a role in the determination of the phenotypic correlation in that, if both traits have low heritabilities, the phenotypic correlation is largely determined by the environmental correlation. If both traits show high heritability, then genetic correlation is the primary determinant.

Linkage Analysis

Of course, one of the most important questions that can be asked is: what genes are responsible for the variation in a trait? One popular approach is genome-wide linkage analysis. We (Sherwood and colleagues) are currently using this approach to examine the genetic underpinnings of craniofacial variation in participants of the Fels Longitudinal Study. This study, in part, consists of a large archive of lateral cranial radiographs which have been phenotyped for 75 different quantitative traits. To conduct linkage analyses using these traits, individuals in the study are genotyped for ~400 highly polymorphic autosomal genetic markers spaced evenly across the genome. The key to these methods is determining that genetic similarities between individuals are due to identity by descent (IBD). Alleles at a marker locus are IBD if they have been inherited from a common ancestor. For example, siblings that share alleles both inherited from their mother are IBD at that locus.

Similar to the way that variation is partitioned into genetic and phenotypic variance in heritability analysis, it is possible to partition the phenotypic variance across the genetic markers. In this way a quantitative trait locus (QTL; the

chromosomal region influencing variation in a trait) can be identified. Figures 2 and 3 graphically demonstrate the results of linkage analysis for a given trait. A string plot (Figure 20-2) graphically depicts each chromosome (excluding the X and Y chromosomes) as a straight line. The strength of the linkage between the trait in question and the chromosomal regions is indicated by the curve adjacent to each chromosomal line. This curve represents the LOD (Log-Odds) score at any given chromosomal region. A linkage is determined to be statistically significant if the LOD score is above 3.0 (Lange and Boehnke, 1983). In figure 2 there are two significant linkages for cranial height, one on chromosome 3 and one on chromosome 12.

A LOD plot (Figure 20-3) provides the details of linkage for a single chromosome, in this case the linkage details for cranial height on chromosome 3 are portrayed. The X-axis identifies the position (in centiMorgans, cM) of the linkage, the Y-axis identifies the strength of the linkage. At the top of the graph, the names of the genetic markers used are given. It is important to note that this type of analysis does not identify genes but merely chromosomal regions. Regions under a LOD peak typically contain many genes. There are a number of techniques

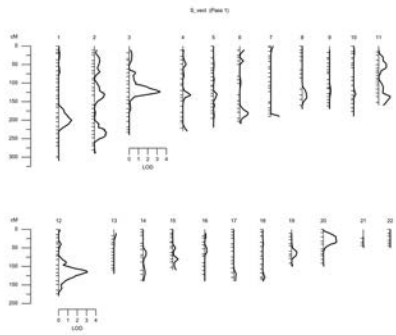

Figure 20-2.
String plot for cranial height (sella – vertex). Chromosomes are identified as numbered straight lines, curves indicate strength of linkage signal. Statistically significant linkages (LOD score > 3.0) are found on chromosomes 3 and 12.

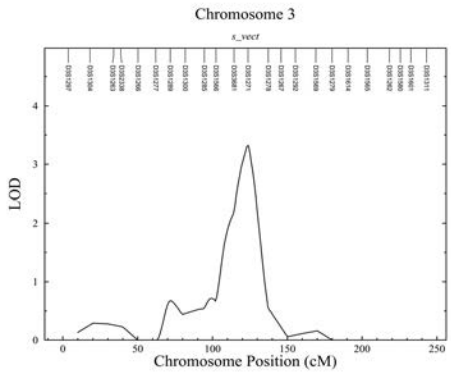

Figure 20-3.
Lod plot showing the linkage results for cranial height (sella – vertex) on chromosome 3.

that can be used to search the regions identified via linkage analysis for the genes responsible for variation in the trait. It is possible to examine these regions using the UCSC Genome Browser (http://genome.ucsc.edu) or to use the growing number of sophisticated bioinformatic techniques to prioritize genes relative to the phenotypes under examination.

The Future of Craniofacial Genetics

This chapter has touched briefly on the current state of knowledge of human craniofacial genetics. It should be clear that there is presently a wealth of information available regarding the genetic regulation of craniofacial growth and development. It should be equally clear that much more remains to be learned. In the early 1980s Slavkin (1983) described the "genetic paradigm," as forming the basis for research into birth defects. He defined this paradigm as recognizing the interaction between the gene and the environment in producing a phenotype. Not surprisingly, with the rapid growth in genetic data, the perceived role of the environment began to diminish. By the late 1990s Moss (1997) identified the "genomic thesis" as the dominant paradigm suggesting that the role of the environment (epigenetic factors in his terminology) was being overlooked in favor of genetic deterministic models. As we have repeatedly stated throughout this chapter the anatomy of the craniofacial complex results from the interplay between numerous genes and the ever-changing non-genetic environment.

Perhaps one of the most exciting avenues for the future of craniofacial genetics is the growing field of gene therapy and tissue engineering. The craniofacial complex, most notably the jaws and dentition, is one of the primary foci for research into these areas. There are practical reasons for this; 1) discreet elements such as the teeth provide an easily managed object for manipulation, and 2) the "normality" of the engineered structures is easy to assess. The clinical reasons for this focus are also numerous; even small craniofacial defects (whether congenital or acquired) can impact multiple aspects of physical and mental health. Current approaches to regenerative dentistry are examining the potential of restoring specific tissues in the pulp chamber of teeth (Murray et al., 2007; Nakashima et al., 2002; Nakashima et al., 2003; Nakashima et al., 2004; Nakashima, 2005), periodontal ligaments (Prabhu and Mehta, 2003; Jin et al., 2004; Nakahara, 2006), complete teeth (Hu et al., 2006; Young et al., 2002; Young et al., 2005b; Duailibi et al., 2006), and/or the supporting bone (Dunn et al., 2005; Young et al., 2005a). Gene therapy has even been investigated as a means to accelerate orthodontic treatment (Kanzaki et al., 2006). Improved characterization of the genetic architecture of the human craniofacial complex will clearly facilitate the application of gene therapy and tissue engineering approaches. While the clinician's focus is often on an individual patient, the variation in form and function that made that patient unique has a rich genetic and environmental history that can be elucidated through quantitative genetics and family-based study designs. As the field of craniofacial genetics continues to develop, new techniques and applications in both clinical and basic research will be discovered thus enhancing our ability to diagnose and treat craniofacial disorders.

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Intrinsic Biological Basis for Accurate Prediction of Facial Growth*

Eeman Dajani, B.D.S., M.S.

Introduction

Craniofacial growth is the fundamental process that produces either a normal facial assembly or some variation of the normal resulting in a malocclusion or other kinds of facial dysplasia. Understanding how the facial growth process works is essential for the selection of proper treatment modalities, and it is the most basic of requirements for understanding proper diagnosis and treatment planning. Needed also is a reliable means for forecasting growth effects on facial construction with advancing childhood age. Over the years there have been many efforts, with mixed results, to create a workable, usable and accurate prediction system. Orthodontists, pediatric dentists, orofacial surgeons and many other health specialists could be able to perform the best possible treatment if they knew precisely the biological growth potential of that patient's biological growth characteristics. That is, growth prediction based on the patient's own personal biological facial growth plan. This requires that the intrinsic biology of facial growth within an individual child be utilized rather than comparisons with "standards" of facial growth norms based on averages of values in mixed population samples.

One of the important factors in achieving an optimal treatment outcome is the timing of orthodontic treatment. Clinicians could be better able to time the orthognathic procedure if properly based on an individual's biological potential and when particular growth periods occur during childhood facial growth. Interest in three dimensional (3-D) imaging devices has increased substantially during the recent past. The third dimension helps to better evaluate, diagnose, plan the treatment, and educate the patient (Kau CH et al. 2005). However, there

* Note: All figures, tables and other illustrations are located in the Appendix. Refer to Chapter 9 for background information and figures helpful for the full understanding of the information in the present chapter.

is a notable lack of reference studies, except for masses of cephalometric data, having little relevant actual morphogenic value. They provide quantitative values for two dimensional (2-D) growth changes occurring in the very important, growth-pacing parts of the basicranium and key anatomical facial parts through childhood (Sgouros et al. 1999). The postnatal growth of the component parts of the nasomaxillary complex, further, goes through two different kinds of regulated morphogenic changes that interrelate with each other. The first is the growing size and configuration of each component, and second is the progressively changing position of each growing component in its real 3-D location and its relation to surrounding structures. The developmental system of biological displacement guides the growth movements of all the facial components to achieve their final functional positions. Thus, the unique, functional combination of both remodeling and displacement are the two essential growth tools that, working together, represent the basic facial developmental system (Enlow DH and Hans MG 1996) McMahon *et al.* (1999), in their study of such relationships, demonstrated strongly that cribriform plate and olfactory bulb orientation relates directly to nasomaxillary growth direction and position by establishing a maxillary growth vector. This produces the precise degree of whole face developmental rotation seen in the many mammalian species examined. The literature, however, lacks studies that have yet to recognize and evaluate this growth vector, it's timing, and developmental relationships in the human face and neurocranium.

Background

Balanced facial growth is an interrelating process involving circummaxillary, mandibular, dental and basicranial growth. Growth of the maxilla itself is a result of the complex movements of displacement, rotation and the shape and size produced by remodeling as well as the growth of skeletal and soft tissue components surrounding it (Precious D et al. 1987). Significantly, the basic growth combination of displacement and remodeling will produce a 3-D maxillary product, and the understanding of this is becoming more essential in craniofacial biology. The remodeling and displacement process will result in progressive changes in the 3-D size, shape and relative displacement position of the nasomaxillary components and the mandible. Growth rates also vary at different times during childhood development. The processes of facial growth and changes in the dental arches continue to a much later age than had previously been realized. Even though our knowledge of the craniofacial growth process itself has significantly increased, growth changes of both the dentition and all of craniofacial development are still incomplete with regard to understanding its regulation

In a thorough comparative study by Turchetta *et al.* (2007); three different methodologies for facial growth prediction were evaluated, Rickettes' methodology for estimating future skeletal changes was based on chronologic age, the Johnston methodology used a grid system for incremental skeletal projections that are also based on chronologic age, and the Fishman system of maturation assessment which is based on maturational timing and period. In a study comparing the traditional 2-D cephalometry and a 3-D approach to human dry skulls, Adams *et al.* (2007)

concluded that there is an inherent problem in representing a linear measurement occupying a 3-D space with a 2-D image. Evaluating distances in 3-D space with a 2-D image grossly exaggerates the true measure and offers a distorted view of craniofacial growth. Cavalcanti *et al.* (2004) studied the precision and accuracy of anthropometric measurements using 3-D computed tomography for craniofacial clinical applications. They also compared craniometric landmarks using bone and soft tissue protocol and concluded that there is no difference between 3-D imaging and physical measurement.

The cephalogram is the standard means used by clinicians to assess skeletal, dental and soft tissue relationships. This approach, however, has been based on two-dimensional 2-D views for analysis of 3-D objects. As pointed out above, evaluating distances in 3-D space with a 2-D image grossly exaggerates the true measure and offers a distorted view of craniofacial growth. Baumrind et al. (1976) concluded that the measurement errors in tracing a superimposition are a consequential factor affecting confidence in head film comparisons, particularly in individual cases and when growth prediction and evaluation of treatment effects are considered.

Farkas *et al.* (2003) *studied* basicranial growth during childhood by using advanced 3-D visualization techniques for MRI scans. Thirty four landmark points were utilized for linear and angular measurements of 600 normal children aged 1 to 15 years The anterior cranial fossae showed a rapid growth rate. Two main growth periods were observed before and after the first 5 years of age. Roussouw *et al* (1991) studied the relation between the frontal sinus size and maxillary and mandibular horizontal lengths, and showed that the frontal sinus can possibly be used as an additional indicator when one is predicting mandibular growth.

Procedure

An unfulfilled challenge in craiofacial biology has been the meaningful prediction of facial development in any given individual child based on his or her own intrinsic biological growth characteristics. The goal of the present study has been to anticipate individual growth potential based on biological information rather than statistical population averages and norms. All previous studies have been based on such population values.

The present study (Dajani E. 2006) applies basic biological concepts, as suggested in Chapter 9 of this text, for the determination of growth trajectories and developmental boundaries of the nasomaxillary complex in relation to the growing basicranium. This allows prediction of their composite growth potential, The key factor is that it utilizes the biological potential within any given individual child. This enables forecasting both the magnitudes and the multiple directions of nasomaxillary growth, thus defining the nasomaxillary growth vectors. It is, most importantly, an intrinsic study of an individual's facial growth pattern based entirely on that individual's own biological growth determinants and details of operation of the facial growth process. Population and normative standards in themselves have little actual biological basis. Many of the cephalometric procedures still being used, both in 2-D and now 3-D, are strongly contradicted by this biological concept.

The human face appears to be quite different than in other mammals, yet it adheres to virtually all of the basic "rules" which govern the facial growth process. Our elongated, narrow, functional muzzle has become altered during evolution into the broad, reduced jaws and flattened human face. (Please see Chapter 9). The head is in a balanced position on an upright spine, and the human face has also become rotated in concert with our cranial flexure and massively enlarged brain into a nearly vertical alignment. Prior work has shown that the degree of basicranial flexion among primates is determined by relative brain size (and other factors), with anatomically modern humans possibly having a less flexed basicranium than would be expected for their relative brain size. Basicranial flexion has also been suggested to be adaptive in that the head has an approximate spheroid brain shape, thereby minimizing connections between different parts of the brain. Consideration of recently published ontogenetic data of adults suggests that much of the variance in basicranial flexion can still be explained as a mechanical consequence of brain enlargement relative to basicranial structure and length (Ross et al. 2004).

The face has become rotated inferiorly by an expansive carry of the floor of the anterior endocranial fossae as the result of brain enlargement. The human face is now located beneath the cerebrum rather than in front of it. It has been rotated simultaneously both downward and backward. In contrast to other mammals, the human head is less dependant on aromatic sensations for food procurement and for protection. The olfactory bulbs in human form thus have become a much less dominant factor contributing, among other key factors discussed below, to the size reduction of the olfactory mucosae and the olfactory receptors in the frontal sinuses. (Chapter 9) The long axis of the snout in all mammals thus far examined, is aligned in alliance with the neutral direction of the sensory olfactory nerves within it. The growth plane of the face, setting nasomaxillary region within the face, is thereby perpendicular (the neutral line) to the axis of the plane of the olfactory bulbs and underlying cribiform plates.

As the alignment determining olfactory bulbs on the enlarging human brain became rotated progressively from a vertical alignment to horizontal, the facial plane has thereby been similarly rotated downward with it from a horizontal to the distinctive vertically oriented human plane. The palate is the common connection in all mammals and some reptiles between the oral and the nasal regions. Any reduction on the nasal side must necessarily be accompanied by reduction on the opposite side, which is the entire oral complex. The basicranial downward rotation displaces the entire upper jaw, nasal region, mandible, and all associated parts into a more confined, reduced newly created space. This requires altered alignments for some major facial growth boundaries, and that in turn requires major growth adjustments for the olfactory, visual, and auditory regions. A reduction in the protrusive extent of both jaws as well as the overlying snout is the result of their displacement into a new, simultaneously developing, reduced relative space. Moreover, available space for the whole human face has become confined by the downward swing of the nasomaxillary complex. The remodeling and displacement processes must necessarily also produce altered mandibular as well as nasal and maxillary reductions in extents of their protrusions, this is required by the newly

established growth boundaries. The outcome of this combination of olfactory, maxillary, orbital, auditory, and whole facial rotations by the alignment of the changing basicranial, olfactory and other growth templates thus underlies the unusual configuration of the human craniofacial complex. All of the standard mammalian developmental rules seem to have been carried over with no out of bounds innovations introduced. (Chapter 9).

Figure 9-9 to 9-22 in Chapter 9 summarizes the anatomical boundaries of the nasomaxillary complex:

- The Superior Boundary, which is the Olfactory plane.
- The Posterior Boundary, which is demarcated by the junction between the middle and anterior cranial fossea superiorly, and the maxillary tuberosity inferiorly.
- The Inferior Boundary is demarcated by a line connecting the most inferior point of the brain posteriorly, and superior prosthion (SP) as the anterior target passing on or near the maxillary tuberosity.
- The Anterior Growth Boundary (AGB) is demarcated by a line that runs from the most anterior point of the frontal lobe (or the corresponding, adjacent endocranial point on the frontal bone) inferiorly to and through the cribriform plate down the mature position of Superior Prosthion (SP).

The ethmoidal complex runs along the mid-sagittal plane and provides partial connection of the neurocranial complex to the facial skeleton. It consists of various plates and paired projections, and a superior and single projection, the crista galli, protruding into the endocranium. Lateral projections from the crista galli are the left and right cribriform plates. In life they cradle the olfactory bulbs and the first cranial sensory nerves entering them. Bundles of axons brachiate through porosities of these plates from the underlying nasal cavity. Please know that the ethmoid complex is an extremely significant intermediate, multifunctional bone and soft tissue system located between the face and brain. It contains neural elements which target relays to and from the face and brain.

Based on morphological relationships of the nasomaxillary complex, Enlow theorized that major facial areas develop along major pathways determined by the outreaching facial special senses (olfactory, visual, auditory). Regional growth continues along these tracks until reaching their mature positions at respective anatomic growth boundaries. For the olfactory sense, the development of the whole nasomaxillary complex follows this olfactory-established growth pathway, having a principal axis located in a mid-position within the incoming spread of sensory axon distribution. This mid-axis is a neutral growth plane oriented perpendicular to the cribriform plate (plane) and olfactory bulbs. The floor of the anterior endocranial fossa and the olfactory plane, and especially the cribriform plane, is a compound template giving developmental direction to the mid-axis

of the spread of olfactory nerves coming from the terminal olfactory mucosae, on each side of the face. The general target for the development of the olfactory growth plane, as described below, is to position the Anterior Border of the face. The moving relocation of the growing development of the Anterior Border continues progressively toward it's final mature location. After the olfactory growth plane reaches AGB and they both no longer continue to develop along the plane of growth movement, no further anterior nasomaxillary growth occurs, unless a cranial dysplasia exists. This growth relationship, now, defines one of the growth vectors assumed by nasomaxillary development involving the three outreach special senses. That is, it identifies (1) the nasomaxillary growth direction and (2) it's growth magnitude. McMahon *et al.* (1999) provided extensive statistical support in a detailed study of these relationships.

Growth of the major, established biological boundaries of the face are not ordinarily bypassed after a developmental equilibrium is attained unless a growth abnormality intervenes. The endocranial floor of the neurocranium is the template for whole face construction, and many structural features and dimensions of the face are based on a combined brain and facial relationship. Yet to be studied are other facial growth vectors that relate to structural relationships with both the visual sense and the hearing and balance complex. Like olfaction, they are the special facial growth determinants for the other facial growth boundaries.

To locate the olfactory growth vector for the nasomaxillary complex, a line is drawn from the forward edge of the frontal lobe of the brain (or from the meningeal surface on the forward most endocranial point of the frontal bone) down perpendicular to and through the cribriform plane. This plane now represents the established Anterior Boundary of the developing nasomaxillary complex. When the growth of both AGB and SP mature and have merged at their final growth positions, Superior Prosthion lies on or within a millimeter or two (probably operator error) of the Anterior Border, which is the anterior and inferior most point of the maxilla. Prior to maturity, it will be moving on the pathway track of the olfactory growth vector moving toward the mature Anterior Border but yet still somewhere short of it. This plane of the Anterior Border is still undergoing a growth movement prior to about the fifth or sixth year of life because the frontal cerebral lobes are still enlarging. The Anterior Border and underlying soft tissue therefore develop progressively forward. The cribriform plane is also undergoing it's own growth movement adjusting to frontal lobe expansion. This continues until age five or six.

According to Enlow, the frontal lobes, anterior cranial fossae, ethmomaxillary complex, palate, maxillary arch, and other hard and soft tissue parts of the ethmomaxillary complex, are all mutual counterparts in relative positions, alignment, and growth limits. This is a key factor in the composite assembly of all the various major components of the head, as described in Chapter 9.

In summary, the intermediate extents of nasomaxillary growth movement toward the Anterior Growth Boundary can be predicted according to position along the olfactory pathway perpendicular to the cribriform plan. The biological limit of total growth along this growth vector, as seen above, is determined by the arrival of Superior Prosthion at the Anterior Boundary after five or six years of

age. In order to determine and measure growth-moving nasomaxillary positions during growth, it must be understood that a composite of important growth events are occurring in the movement process of SP. First, the entire maxilla is becoming secondarily displaced anteriorly by enlargement of the middle cranial fossae and temporal lobes of the cerebrum, and SP with it. At the same time, SP is becoming moved anteriorly through secondary displacement by the whole maxillary enlargement at the maxillary tuberosity. Third, SP is being moved inferiorly by the same secondary displacement at the circummaxillary sutures. SP itself is undergoing it's own remodeling and primary displacement. As seen above, the growth target to be reached by SP at its maturity is the Anterior Growth Boundary of the whole nasomaxillary complex. Early in childhood, SP lies well behind the mature boundary, and by means of remodeling, SP's size and configuration become progressively altered Simultaneously, all of these clinically significant and separate modes of movement by the regional categories of displacement, just mentioned, and the remodeling of this part of the maxilla itself, occur along the olfactory vector pathway. Superior prosthion is being moved progressively toward the simultaneously growing AGB until merging with it. Once the final, mature AGB attained, SP lies on or very near it and is expected to remain stable in that position thereafter.

Three major points are objectives of attempts to find a biologically accurate way for facial prediction. The first is to track movement pathways of the growth of the nasomaxillary complex that brings it to morphological maturity. Second, to account for the biological means of directional control of these growth movements. Third, and based on the biological information derived from the first two, is to determine if this information can be utilized in accurate biological forecasting of the directions and the magnitudes of nasomaxillary growth, the precise location it will attain upon reaching morphologic maturity, and at what point during whole facial development this will occur. In the present study, importantly, a distinct growth vector of the nasomaxillary complex toward a major facial anterior boundary is recognized. It relates directly to the olfactory growth plane and is seen clearly in children as well as adults. This study utilizes subjects ranging in age from new born to 22 years and employs standard three dimensional computed tomograms. The reliability and reproducibility of the measurements were evaluated by inter-examiner and intra-examiner calibrations. Intrusion criteria were determined according to age of the subject when the Computed tomogram (CT) was done, which ranged from newborn to 22 years of age, and if the nasomaxillary complex and the frontal lobe of the brain were included in the tomograms. Any Subject with pathology, or fracture affecting the nasomaxillary complex, the cribriform plate, or the anterior cranial fossa was excluded.

Subjects were divided into eight age groups (Table 21-1). The groups were defined according to the availability of the subjects representing each growth period. The first age group (Newborn to 4 years old) represents the period when most of the growth of the frontal cerebral lobe is achieved10. The computed tomogram (CT) scans were acquired by two different scanners following the exam protocol for the facial bones, (NEMC), Sensation 16 (SIEMENS), which uses a multi slice helical scan to acquire the image. The protocol included images generated at 120

kV and 120 mAs. Rotation time was 0.75 second, slice width 1mm. Collimation was 0.75 mm and the direction of rotation was 4.7 mm craniocaudal. The subject was in a supine position. Initial scout was lateral and the projection phase was axial. The anatomical range was from the top of the frontal sinus through the mandible. (Appendix I).

The second was the Volume Zoom (SIEMENS): which uses a multi slice helical scan to acquire the image. The protocol included images generated at 140 kV and 100 mAs. Rotation time was 0.75 second, slice width 1.25mm. Collimation was 1mm and the direction of rotation was 3.5 mm craniocaudal. The subject was in a supine position. Initial scout was lateral and the projection phase was axial. The anatomical range was from the top of the frontal sinus through the mandible. (Appendix II). The resultant image slice data were stored on Picture archive communication (PACS) system and then loaded onto the LEONARDO work station (SIEMENS). Images were reformatted in the sagittal plane to be used for the evaluation of the growth of the nasomaxillary complex. The protocol included window values of: 2500/200 which were adjusted as needed using the LEONARDO soft ware to help in clarifying the anatomical land marks. (Appendix III). Selection of the sagittal section that passes through the cribriform plate and the superior prosthion (SP) point was completed after adjustment of the midline on both the coronal and axial sections. Jones *et al.* (2002) had concluded that there was no statistically significant difference between left and right sides demonstrable for any parameter, irrespective of pathological status. Distance of the selected sagittal section from the adjusted midline was measured using the LEONARDO distance measurement software. Locating the olfactory plane on 2-D cephalograms is not possible because of the superimposition of anatomical land marks. The use of three dimensional CT with formatted sagittal sections makes determination of the olfactory plane (cribriform plate) easier and clearer. Anatomical landmarks of the nasomaxillary complex were identified which are the Cribriform plate and a line connecting the anterior most and posterior most aspects of the cribriform plate at the ethmoid/nasal junction. The cribriform plate is a thin wafer of bone with distinct anterior and posterior ends. The plane for the Anterior Boundary of the nasomaxillary complex: is a line originating from the anterior most point of the frontal lobe drawn inferiorly perpendicular through cribriform plates. Determination of this 90 degree angle was done using the LEONARDO soft ware for image processing. The target is the anterior and inferior most point of the maxilla and is represented by Superior Prosthion. It's anticipated position lies on the Anterior Border, but while expected (programmed) to lie on this precise point, our findings show that it may normally be a millimeter short or long. The nasomaxillary complex thus becomes vertically aligned during development along the vertical axis of olfactory nerve distribution. The distance between the Anterior Boundary and SP was measured using The Leonardo Software. (See Appendix IV and V for image samples). If SP is short of AGB, it is recorded as a positive (+) value, and if SP is beyond AB, it is a negative value (-) Intra-examiner calibration of the measurement was done by randomly selecting thirty five subjects and then reexamined by the same examiners without looking at the first measurement. Time between the two readings was at least 2 weeks (Appendix VII). Inter-examiner calibration was done by randomly selecting

ten subjects then reexamined by another examiner (E. A.), (Appendix VIII). Standard deviation for each group is shown in (Table 21-2).

The mean of Age Group 2 is more than Age Group 1, which means that the distance between SP and AB is increasing in this age group. In all the older age groups; the mean is decreasing continuously. Age group 7 and 8 have an increased mean because there is an outlier value of 1.02 in age group 7, while all the other values are (0.07,0.03,0.01,0.04,0.09), and a value of 0.44 in age group 8. The other values are all zero (Appendix VI).

Regression analysis was done between age (independent variable) and the distance between SP and the anterior boundary (dependant variable). When the scatter plot was done, there was no clear interpretation for the data for subjects below 10 years old, but then there is a clear change where the measurement starts to decrease then it plateaus. (Figure 21-1)

When age was used as a continuous variable, P value: 0.0001 which indicates that there is a significant relation. See (Figure 21-2), which shows statistical significance for the whole population.

When age was grouped as shown in (Table 21-2), there is a decrease in the distance between SP and the anterior boundary with the increase in age group. See (Figure 21-3) which shows a P value of 0.0001 which indicates that there is a significant relation.

The population was divided into 3 samples:

Sample 1: New born to 14 years old

(A) When age was considered a continuous variable results were not significant, p value: 0.767, (Figure 21-4) shows the curve fit and the model summary.

(B) When age was considered as a constant variable, results were not significant with a p value 0.774. (Figure 21-5) shows the curve fit and the model summary.

Sample 2: 14 years old to 22 years old

(A) When age was considered as a continuous variable, P value: 0.066 . (Figure 21-6) shows the curve fit and the model summary.

(B) When age was considered as a constant variable, (Figure 21-7) shows the curve fit.

Sample 3: 11 to 18 years old:

(A) When age was considered a continuous variable it showed a statistical significant P value, 0.003 , (Figure 21-8) shows the curve fit and the model summary.

A prediction formula for this group can be derived from the graph in figure 21-8 where the X axis is represented by the age

Y axis is represented by the distance between SP and The anterior boundary of the nasomaxillary complex.

According to the curve the slope of the curve is m, and the intercept is b.

Y = m times X + b
Y is the distance between SP and the anterior boundary, X is age
m is the slope of age = -0.085
b is the intercept = 1.722
Distance = -0.085 times Age + 1.722

If a clinician would like to know where is the adult position of the anterior boundary of the nasomaxillary complex, he or she would apply the formula:

Y=-0.085 times (patients age) + 1.722

Y would be the magnitude which the current anterior boundary of the nasomaxillary complex had move forward. Additional research on this point for validation is recommended for clinical use.

(B) Age was considered as a constant variable: highly significant P=0.001. (Figure 21-9) shows the curve fit and the model summary.
Regression analysis was done by comparing each group to the last, and the results show that there is an increase in the (standard estimate) between the first group and the second group, then there is a continuous decrease in the 3rd, 4th, 5th 6th and 7th standard estimate. (Table 21-4).

The p value was highly significant for the first 3 age groups. There is an increase (standard estimate) between the first group and the second group; which is logical since the frontal lobe is still growing forward until age 5 years, so the anterior boundary for this age group will be in a posterior position because the frontal lobe is not fully mature yet. Then there is a continuous decrease in the 3rd, 4th, 5^{th}, 6th and 7th standard estimate.

Analysis was done for Age Group 7 to see if the value approaches zero, Age Group: 7 (>18- 19 years old), there was an outlier with a value of 1.02. This measurement was repeated twice. (Table 21-5)

For age Group 8 descriptive analysis was done to see if the value stays stable. Age there was an outlier with a value of 0.44, and the measurement was repeated twice. (Table 21-6)

Correlation analysis for the intra-examiner calibration shows that there is significant $P < 0.0001$ correlation between the two readings, Pearson correlation 0.962. (Table 21-7)

Inter examiner correlation analysis shows that there is significant ($P < 0.0001$) correlation between the two readings. Pearson correlation is 0.900. (Table 21-8)
(Table 21-9) shows the descriptive analysis for the variable (the distance of each selected sagittal section from mid line). The standard deviation is 0.04495.

Discussion

This is the first study attempting to apply an intrinsic biological approach to prediction of human facial growth using three-dimensional imaging data to determine the growth vector of the nasomaxillary complex. Importantly, the intrinsic biological information to be directly used for prediction of an individual

child's face is derived from that same individual child and is not a "normative value" of averages from some group of individuals. In this study sagittal sections were formatted from axial and coronal sections, thus the formatted sagittal section is a true representative of the anatomical landmark points. A clinician can recognize the advantages that the third dimension gives to clinical diagnosis, treatment planning and patient education. In the first two age groups, Age Group 1 included subjects ranging in age from newborn to four years. Age Group 2 included subjects ranging in age from more than four years to eight years and showed an increase in the distance between SP and the anterior boundary. This is supported by the findings of Farkas (2003), stating that early rapid growth of the forehead width takes place between one and six years, and that head circumference shows continuous increase to six years, but with mild growth rates after that. The anterior fossa shows a rapid growth rate before the first 5 years of age (Farkas et al. 1992, Broadbent et al. 1975) . Two main growth periods were observed: before and after the first five years of life. During the first five years, the anterior fossa showed a rapid growth rate with respect to its anterior projection in males, whereas in the females there was a more concentric growth pattern. The body of the sphenoid bone and the middle cranial fossa showed a rapid growth rate in both sexes but was greater in the females. These findings provide new insight into the pattern of growth of the various parts of the basicranium[2]. In the present study, a continuous decrease in the standard estimate in Age Groups 3, 4, 5, 6 and 7 when each group was compared to the last (Table 21-4). The findings indicate that there is constant growth of the nasomaxillary complex along the olfactory growth vector. As described above, this is a perpendicular plane established by the olfactory bodies. It enables the cribriform plate to serve as a template for orienting an olfactory-directed maxillary growth direction. This plane, perpendicular to the cribriform plane as described elsewhere, represents the olfactory vector for the growth direction and magnitude of the nasomaxillary complex. McMahon *et al.*[4] in his PhD work on humans and anthropoids, clearly verified this biological relationship.

There was no significance or reliable pattern in the regression analysis done for the first three age groups. Different explanations may be proposed in which the differences in the head forms may be attributed to the large series of variables or to the differences in ethnic back ground. The distance between SP and the anterior boundary approaches zero around 18 years of age, there was an outlier in the data and the measurement was repeated twice. The distance between SP and the anterior boundary does not exceed the zero value in the Age Group 21 years of age, which indicates that the forward growth of the nasomaxillary complex has ceased in that age group. There was an outlier in the data, and the measurement was repeated twice. McMahon *et al.* (1999) demonstrated that the cribriform plate orientation in anthropoids mainly affects changes in the nasomaxillary skeleton by influencing the degree of facial rotation during development . There is a highly significant correlation between age and the decrease of the distance between the nasomaxillary complex and it's biological potential limit (Table 21-4), for subjects with an age range between 1 and 18 years of age (Table 21-4). This further demonstrates that the distance between SP and the anterior boundary of

the nasomaxillary complex decreases with the increase of age. Forward growth of the nasomaxillary complex can be predicted from age 11 to 18 years. A prediction formula for this group would be:

Distance = -0.085 times Age + 1.722.

The sagittal sections were selected by viewing both coronal and axial sections, and then choosing the sagittal section that passes through the anatomical land marks, the distance from the midline was recorded, and the results showed that the standard deviation was minimal when there was consistency in the location of the sagittal section from the midline. Intra-examiner calibration of the measurements revealed a high correlation between the two readings that were done by the same examiner with a time interval of more than 2 weeks between the two readings. Measurements done by another examiner (E.A.) for ten randomly selected subjects showed a highly significant correlation, which indicates that the readings are reliable and reproducible.

Summary of Results

There is a highly significant correlation between age and the decrease of the distance between the nasomaxillary complex and its biological potential limit, shown in (Figure 21-3) and (Table 21-4), which supports the hypothesis that the distance between SP and the anterior boundary of the nasomaxillary complex decreases with the increase of age.

In conclusion, this study demonstrated a method for growth prediction based on intrinsic biological potential of each individual child. It shows that there is a continuous decrease in the distance between the Superior Prosthion and the anterior boundary of the nasomaxillary complex with increasing age. Statistical significance (P value of 0.0001)

The forward growth of the nasomaxillary complex can be predicted from age 11 to 18 years according to a prediction formula. Forward growth of the nasomaxillary complex stops at around 18 years of age and stays stable until at least 22 years of age, which was the oldest age examined.

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22

Maturation of the Orofacial Neuromusculature

Robert E. Moyers, D.D.S., Ph.D.*
David S. Carlson, Ph.D.

PRENATAL MATURATION

During prenatal life the human neuromuscular system matures unevenly. It is not accidental that the orofacial region matures (in the neurophysiologic sense) ahead of limb regions, because the mouth is the primary site of respiration, nursing, and protection of the oropharyngeal airway. In the human fetus, by about the eighth week, generalized uniform reflex movements of the entire body can be elicited by tactile stimulation. A few spontaneous movements, in response to as yet unidentified stimuli, have been observed as early as 9½ weeks. Localized specific and more peripheral responses can be produced before 11 weeks. At this time, stimulation of the nose-mouth region causes lateral body flexion. By 14 weeks, the movements have become much more individualized, and very delicate activities can be executed. When the mouth area is stimulated, general bodily movements no longer are seen; instead, facial and orbicular muscle responses are produced. Stimulating the lower lip, for example, causes the tongue to move. Stimulation of the upper lip causes the mouth to close and, often, deglutition to occur.

Respiratory movements of the chest and abdomen are first seen at about 16 weeks. The gag reflex has been demonstrated in the human fetus at about 18½ weeks (menstrual age). By 25 weeks, respiration is shallow but may support life for a few hours if established.

Stimulation of the mouth at 29 weeks has elicited suckling, although complete suckling and swallowing are not thought to be developed until at least 32 weeks.

Davenport Hooker and Tryphena Humphrey have shown us that there is an orderly, sequential staging of events in prenatal orofacial neuromuscular maturation—a staging seen throughout the body, but which is much more advanced in the oropharyngeal region. All this has to be established by the time of birth in order for the child to survive (Humphrey, 1970).

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* Deceased

NEONATAL ORAL FUNCTIONS

At birth, tactile acuity is much more highly developed in the lips and mouth than it is in the fingers. The infant carries objects to his mouth to aid in the perception of size and texture; later they go into the mouth as a part of teething. The neonate slobbers, drools, chews toes, sucks thumbs, and discovers that gurgling sounds can be made with the mouth.

Freudians consider all of this oral eroticism, as they do adult smoking; but in the infant surely it is also explanatory and exercises the most sensitive perceptual system in the body at that time. Oral functions in the neonate are guided primarily by local tactile stimuli, particularly those in the lips and front part of the tongue.

The tongue at this age does not guide itself; rather, it follows superficial sensation. The posture of the neonate's tongue is between the gum pads, and it is often far enough forward to rest between the lips, where it can perform its role of sensory guidance more easily. The young infant, to a great extent, interprets the world with the mouth, and the integration of oral activities is therefore by sensory mechanisms.

If you touch a young child's lips or tongue and have him follow your finger, both the head and body turn. A little bit later the head turns separately from the body, and still later the mandible is moved without moving the head. It is only last of all that the neonate can follow with the tongue, while not moving the mandible. These stages appear in a natural sequence, just as teeth erupt on a kind of schedule.

The infant uses the mouth for many purposes. The perceptual functions of the mouth and face are combined with the sensory functions of taste, smell, and jaw position. The neonate's primary relationship to its environment is by means of the mouth, pharynx, and larynx. Here a high concentration of readily available receptors becomes stimulated and modulates the already matured brain stem coordinations that regulate respiration and nursing and determine head and neck positions during breathing and feeding.

The sensitivity of the tongue and lips is perhaps greater than that of any other body area. The sensory guidance for oral functioning, including jaw movements, is from a remarkably large area. These sensory inputs are compounded by many dual contacting surfaces, such as the tongue and lips, the soft palate and posterior pharyngeal wall, and the compartments of the temporomandibular articulation. A great array of sensory signals is required for the integration, coordination, and interpretation of this complex system.

INFANTILE SUCKLING AND SWALLOWING

The effectiveness of oral motor activities is a good indication of the neurologic maturation of premature infants. It has been found that a child will follow the same patterns in certain oral reflex movements years after initial learning. For example, a study was made of children whose records had been kept from infancy. As long as 9 years after weaning, if given a bottle from which to suckle, they produce the

same suckling, swallowing, and respiratory rhythms they had when infants. If they swallowed in a suckle-suckle-swallow type of pattern (i.e., two suckles for one swallow, two-for-one), this same rhythm appeared years later. It may be a three-for-one or even a four-for-one ratio, but the pattern is maintained. Such primitive reflexes are difficult for us to change. How foolish it is for us, with our present ignorance about conditioning such basic mechanisms, to try and alter some of these reflexes. We must spend more time with those problems that we have at least a theoretical chance to condition.

Rhythmic elevation and lowering of the jaw provide sequential changes in positions of the tongue in coordination with its suckling contractions. The activities of suckling are closely related temporally to the motor functions of positional maintenance of the airway.

Electromyographic studies in our own laboratory have confirmed visual observations reported in England by a number of people, revealing that while the mandibular movements are carried out by the muscles of mastication, the mandible is primarily stabilized during the actual act of infantile swallowing by concomitant contractions of the tongue and the facial (rather than masticatory) muscles (Moyers, 1964). At the actual time of the infantile swallow, the tongue lies between the gum pads and in close approximation with the lingual surface of the lips. Thus, the infantile swallow is neuromuscularly a different mechanism from the mature swallow.

Characteristic features of the infantile swallow are that (1) the jaws are apart, with the tongue between the gum pads; (2) the mandible is stabilized primarily by contractions of the muscles of the seventh cranial nerve and the interposed tongue; and (3) swallowing is guided, and to a great extent controlled, by sensory interchange between the lips and the tongue.

Maintenance of the Airway

The oral-jaw musculature is responsible for the vital positional relationships that maintain the oral pharyngeal airway. While the infant is resting, a rather uniform diameter for the airway is provided by (1) maintaining the mandible anteroposteriorly and (2) stabilizing the tongue and posterior pharyngeal wall relationships.

The axial musculature around the vertebrae is also involved. These primitive neonatal protective mechanisms provide the motor background upon which, with growth, all the postural mechanisms of the head and neck region are developed. Physiologic maintenance of the airway is of vital, continuing importance from the first day throughout life.

This little neonate who cannot focus his eyes, who cannot make a purposeful movement of his limbs, who cannot hold his head upright, who has absolutely no control of the lower end of his gastrointestinal tract has absolutely exquisite control of some functions in the orofacial regions. Why? Such control is necessary for survival.

Infant Cry

When the aroused baby is crying, the oral region is unresponsive to local stimulation. The mouth is held wide open, while the tongue is separated from the lower lip and from the palate. The steady stabilization of the size of the pharyngeal airway is given up during crying, and there are irregular, varying constrictions during expiration of the cry and large, reciprocal expansions during the alternating inspirations.

Gagging

Gagging, the reflex refusal to swallow or accept foreign objects in the throat, is an exaggeration of the protective reflexes guarding the airway and alimentary tract. The gag reflex is present at birth, but it changes as the child grows older in order to accommodate visual, acoustic, olfactory, and psychic stimuli that are remembered and thus condition it.

EARLY POSTNATAL DEVELOPMENT OF ORAL NEUROMUSCULAR FUNCTIONS

Mastication

The interaction between the rapidly and differentially growing craniofacial skeleton and the maturing neuromuscular system brings about sequentially progressive modifications of the elementary oral functions seen in the neonate (Moyers, 1964, 1969, 1988). Mandibular growth, downward and forward, is greater during this time than midfacial growth and is associated with a greater separation of the thyroid bone and thyroid cartilage from the cranial base and mandible.

Maturation of the musculature and delineation of the temporomandibular joint help provide a more stable mandible. Although mandibular growth carries the tongue away from the palate and helps provide differential enlargement of the pharynx, patency of the airway is maintained—a most important point.

The soft palate and the tongue are commonly held in apposition, but as the tongue is no longer lowered by mandibular growth, its functional relationship with the lips is altered, an alteration aided by the vertical development of the alveolar process. So the morphologic relationship of the tongue and lips is strained. At rest now, the tongue is no longer in generalized apposition with the lips, buccal wall, and soft palate. The lips elongate and become more selectively mobile; the tongue develops discrete movements that are separate from lip and mandibular movements. The labial valve mechanism is constantly maintained during rest and feeding so that food is not lost.

The development of speech and mastication as well as of facial expression requires a furthering of the independent mobility of the separate parts. In the neonate, however, the lips tightly surround a plunger-like tongue, moving in

synchrony with gross mandibular movements. Speech, facial expression, and mastication require the development of new motor patterns as well as greater autonomy of the motor elements. Not all the developmental aspects of these functions are known. But mastication certainly does not gradually develop from the infantile nursing. Rather, it seems that the maturation of the central nervous system permits completely new functions to develop. These functions are triggered to an important extent by the eruption of the teeth.

One of the most important factors in the maturation of mastication is the sensory aspect of newly arriving teeth. The muscles controlling mandibular position are cued by the first occlusal contacts of the antagonistic incisors. Serial electromyographic studies at frequent intervals during the arrival of the incisors have demonstrated conclusively that the very instant the maxillary and mandibular incisors accidentally touch one another, the jaw musculature begins to learn to function in accommodation to the arrival of the teeth (Moyers, 1964).

Thus, since the incisors arrive first, the closure pattern becomes more precise anteroposteriorly before it does mediolaterally. All occlusal functions are learned in stages. The central nervous system and the orofacial and jaw musculature mature concomitantly, and usually synchronously, with the development of the jaws and dentition.

The earliest chewing movements are irregular and poorly coordinated, like those during the early stages of the learning of any motor skills. As the primary dentition is completed, the chewing cycle becomes more stabilized, using more efficiently the individual's pattern of occlusal intercuspation. In the very young child, sensory guidance for masticatory movement is provided by the receptors in the temporomandibular articulation, the periodontal membrane, the tongue, and the oral mucosa and muscles; of these it seems by far that the most important are those of the temporomandibular articulations, and next those of the periodontal membrane. Cuspal height, cuspal angle, and incisal guidance (which is usually minimal in the primary dentition) play a role in the establishment of chewing patterns in the infant. However, condylar guidance is not important at this age, since the eminentia articularis is ill-defined and the temporal fossae are shallow. Rather, it may be supposed that the bone of the eminentia articularis forms where temporomandibular function permits (or causes) it to develop. In a similar fashion, the plane of occlusion is established by the growth of the alveolar process, during eruption of the teeth, to heights permitted by the configuration and functioning of the neuromusculature.

The individual's movements during the chewing cycle are a developed, integrated pattern of many functional elements. In the young child, at the time of completion of the primary dentition, masticatory relationships are nearly ideal, since all three systems (bone, teeth, and muscle) still show the adaptability characteristic of development. Cusp height and overbite in the primary dentition are more shallow, bone growth more rapid and adaptive, and neuromuscular learning more easily cued because pathways and patterns of activity are not yet well established. Adaptations to masticatory change are much more difficult in later years, as every dentist knows.

Facial Expression

In a not dissimilar way, most subtle facial expressions are learned, largely by imitation, so we think, and begin about the time the primitive uses of the seventh nerve musculature for infantile swallowing are abandoned. Those of us who are parents imagine all sorts of facial expressions in the young neonate. Actually, observing the infant objectively, we must admit that the expression is often rather blank. The reason is that the facial muscles are busy being used for the massive efforts of mandibular stabilization necessary during infantile swallowing. Eventually the mandible becomes controlled and stabilized more by the muscles of mastication, particularly during unconscious reflex swallowing, and the delicate muscles of the seventh cranial nerve become truly "muscles of facial expression."

Although many facial expressions are learned through imitation, some facial responses are not learned and can be traced back to reflexes of earlier primates. Similar facial displays have evolved in the four lines of modern primates in which monkey-like forms have developed. Comparative studies have been made revealing similar reflex expressions of protective anger, for example, in various primates—the same primitive expressions you have seen on your best friend.

Speech

Purposeful speech is different from the reflex infant cry. Infant crying is associated with irregular tongue and mandibular positions related to sporadic inspirations and expirations. Speech, on the other hand, is performed on a background of stabilized and learned positions of the mandible, pharynx, and tongue. The infant cry is usually a simple displacement of parts, accompanied by a single explosive emission, whereas speech can only be carried out by polyphasic and sequential motor activities synchronized closely with breathing. Speech is regular; the infant cry is sporadic. Speech requires complicated, sophisticated, varying sensory conditioning elements during learning; the infant cry is primitive and not learned.

Speech consists of four parts: (1) language—the knowledge of words used in communicating ideas; (2) voice—sound produced by air passing between the vibrating vocal cords of the larynx; (3) articulation—the movement of the speech organs used in producing a sound, (i.e., the lips, tongue, teeth, mandible, palate, and so forth); and (4) rhythm—variations of quality, length, timing, and stress of a sound, word, phrase, or sentence. If there is no impairment of hearing, sight, or oral sensation, the child will learn to speak from the speech that is heard. Speech defects are a loss or disturbance of language, voice, articulation, and rhythm or combinations of such losses and disturbances.

Mature Swallow

During the latter half of the first year of life, several maturational events usually occur that alter markedly the orofacial musculature's functioning. The arrival of the incisors cues the more precise opening and closing movements of

the mandible, compels a more retracted tongue posture, and initiates the learning of the mastication. As soon as bilateral posterior occlusion is established (usually with the eruption of the first primary molars), true chewing motions are seen to start, and the learning of the mature swallow begins. Gradually, the fifth cranial nerve muscles assume the role of muscular stabilization during swallowing, and the muscles of facial expression abandon the crude infantile function of suckling and the infantile swallow and then begin to learn the more delicate and complicated functions of speech and facial expressions. The transition from infantile to mature swallowing takes place over several months, aided by maturation of neuromuscular elements, the appearance of upright head posture, and hence a change in the direction of gravitational forces on the mandible, the instinctive desire to chew, the necessary ability to handle textured food, dentitional development, and so forth. Many children achieve features of the mature swallow at 12 to 15 months, but there is a great variability. Characteristic features of the mature swallow are as follows: (1) the teeth are together (although they may be apart with a liquid bolus); (2) the mandible is stabilized by contractions of the fifth cranial nerve muscles; (3) the tongue tip is held against the palate above and behind the incisors; and (4) minimal contractions of the lips are seen during swallowing.

Neural Regulation of Jaw Positions

Jaw position, like a number of other automatic-somatic activities, normally is largely reflexively controlled, even though it can be altered voluntarily. A surprising number of jaw functions are carried out at the subconscious level, even though conscious control is possible and sometimes necessary. Receptors in the temporomandibular capsule area are far more important than previously thought.

Since more research on the neurophysiologic regulation of jaw position and function has been done on the adult, there has been a tendency to transfer prosthodontically oriented concepts, based on sound adult clinical practice, to children. Our knowledge about the developmental aspects of orofacial and jaw neurophysiology is incomplete at this time, although much research is under way. We must remember that many of our attitudes are victims of our experience with degenerating occlusions in adults, and the critical clinical factors that apply under those circumstances may not be present in the child or may have different relative significance during development.

Unconditioned jaw positions and functions include mandibular posture for the maintenance of the airway and unconscious or reflex swallowing. The neural mechanisms that determine mandibular posture are important to the dentist, because mandibular posture (sometimes in dentistry called the rest position) is a determinant of the vertical dimension of the face. In the opinion of many, the position of the mandible during unconscious swallowing is an important factor in occlusal homeostasis, because every time a person swallows unconsciously, the occlusal relationship is stabilized or, because of tooth interferences, shifts interfering teeth by lower jaw movement until a stable occlusal relationship finally is obtained.

Conditioned jaw positions and functions include all those of mastication, the mature swallow, and speech and most of facial expression.

OCCLUSAL HOMEOSTASIS

Occlusal stability at any moment is the result of the sum of all forces acting against the teeth. Some of these forces have been measured in research, but it is not yet possible to describe precisely in summation all the forces and counterforces that produce occlusal homeostasis. Occlusal homeostasis is dependent upon elaborate and sophisticated sensory feedback mechanisms from the periodontal membrane, temporomandibular joint, and other parts of the masticatory system. Such sensory feedback serves as a regulating mechanism helping to determine the strength and nature of muscle contractions. Each individual tooth is positioned between contracting sets of muscles. It is also in contact with adjacent teeth and in occlusion with the teeth of the opposite arch. A number of physiologic forces determine the tooth's position occlusally, including eruption, the occlusal force during swallowing, the forces of mastication, occlusal wear of the crown of the tooth, and so forth. Occlusal interferences in or near the unconscious swallowing position of the mandible tend to diminish reflexively the force of muscle contractions during swallowing. Because reflex swallowing occurs so frequently, it plays an important role in occlusal homeostasis. Other factors involved in occlusal homeostasis include the natural mesial drifting tendencies of the teeth, the anterior component of force, the growth of bones of the craniofacial complex, and alveolar bone growth and remodeling. It is now believed that the neuromuscular mechanisms and bone growth factors are far more important in the nature of occlusal relations than are the oft-mentioned factors of cuspal inclination, cusp height, condylar guidance, and so on. The occlusal relationships are now generally held to be nowhere near as stable as depicted in some dental textbooks, if for no other reason than that occlusal adaptations must occur constantly to accommodate, in their way, changes in the neuromusculature and the craniofacial skeleton. Occlusal homeostasis is achieved and maintained in a complex system of responses and adaptations in several tissue systems.

EFFECT OF NEUROMUSCULAR FUNCTION ON FACIAL GROWTH

From the earliest periods of embryonic growth, an intimate functional relationship exists between muscles and the bones to which they are attached. Obviously, as the bones grow, the muscles must also change their size. Therefore, a relationship exists between the overall growth of any bone and the muscles attached to that bone; and adjustments between muscle and bone are a normal part of growth and development. During growth, muscles also must migrate to occupy relatively different positions with time. As the skeleton grows, there is a constant adjustment of the attachment relationships between muscle and skeleton.

Functional use and disuse determine to some extent the thickness of the cortical plate of limb bones. However, the relationship of muscle function and bone form and growth in the craniofacial skeleton is much more difficult to assess. Certain parts of some of the facial bones are very dependent on function—for example, the alveolar process around the roots of the teeth and the coronoid process

to which the temporal muscle is attached. In a more general way, the conformation of the bone and the craniofacial relationships are determined by such factors as mouth breathing, excessive masticatory function, and so forth. In the case of the calvaria, cranial base, and nasomaxillary complex, functional features other than those of muscle apparently play an important role in development and growth—namely, the growth of the brain, the eyeballs, cartilage growth, and so on.

The mandible, with its important condylar cartilage, holds a special interest for dentists, particularly orthodontists. Although there is general agreement that variations in muscle function affect markedly the areas of muscle attachment and that the development and use of the dentition affect the alveolar process, there is some dispute over whether or not muscle function can have a more general effect on the size and form of the mandible. The point is a very important one for orthodontists treating Class II malocclusions in children who are still growing.

Although the evidence is still not complete, most workers now believe that function plays a more dominant role in the determination of mandibular size and conformation than was previously thought. For example, extensive experimental research has shown that the masseter and lateral pterygoid muscles may play a major role in the growth of the mandibular condylar cartilage. It remains unclear, however, whether this effect is a direct one, or whether muscle function influences condylar growth simply by alteration of the biomechanical environment.

EFFECTS OF ORTHODONTIC TREATMENT ON THE MUSCULATURE

It is known that severe malocclusion is often associated with pathologic changes in the temporomandibular articulations, which in turn impair the sensory receptors within the joints, causing such orthodontic patients to have a less precise ability to determine mandibular position than persons who have normal occlusion. After malocclusions have been treated orthodontically, there is a significant change in the range of mandibular movements and an improvement in the precision of the determination of mandibular positions. Occlusal equilibration on treated orthodontic patients has been shown to change significantly teeth-apart swallowing to teeth-together swallowing (Moyers, 1988). Thus, orthodontic treatment including occlusal equilibration conditions swallowing reflexes, which in turn help stabilize the orthodontic occlusal result. Occlusal disharmonies, at the end of orthodontic treatment, have been shown to be disruptive to the stability of treated orthodontic occlusions and thus an important cause of relapse in treated malocclusions. Other adaptive muscular changes following orthodontic therapy may include an altered lip posture, tongue posture, mandibular posture, chewing stroke, and method of breathing.

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