TRANSVERSE MAXILLARY RESTRAINT IN GROWING RABBITS AND COMPENSATORY GROWTH CHANGES

by

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Abstract

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Compensatory responses to experimentally induced alterations of craniofacial structures by the application of active or passive force systems has received little attention in the literature. Results of previous studies indicate that post-restraint compensatory responses are varied.

The present study was performed to examine the nature of compensatory responses during and after passive, transverse restraint of the maxillary complex of rabbits. The sample consisted of 12 rapidly growing New Zealand White rabbits (30 days of age), with 5 rabbits serving as controls. The experimental group was subjected to 15 days of passive restraint of the maxillary complex utilizing custom-fabricated acrylic appliances. The second phase of the experiment involved removal of the restraining appliances which allowed for a 30 day period of unrestricted growth.

Data were collected from both groups and analyzed in three categories:

1. Dental cast measurements which assessed structural compensatory responses of the maxillary complex;

2. Cephalometric measurements which assessed the compensatory responses of surrounding structures in the anteroposterior and transverse dimensions; and,

3. Histological observation to evaluate the relative amount of post-restraint bone deposition between groups.

Dental cast analysis showed compensatory responses expressed as an increase in the depth of the palatal vault during restraint with a corresponding decrease in the experimentally induced palatal vault depth, and regression toward normality during the post-restraint period.

The cephalometric analysis showed that surrounding structures such as the zygomatic and maxillary bones, as well as the cranial base, all responded similarly during and after restraint. Anteroposterior snout development, however, differed in its compensatory response to transverse restraint of the maxillary complex. The posterior portion of the calvarium appeared to be unaffected by restraint of the maxillary complex.

Histological examination showed a relatively greater amount of bone deposition in the experimental group subsequent to removal of the restraining appliance as compared to the same period of unrestricted growth in the control group.

The results of the study may be summarized:

1. Passive transverse restraint of the maxillary complex can alter its structural and transpalatal dimensions and inhibit its growth potential.

2. Compensatory changes seen during restraint include an increase in palatal depth and anteroposterior snout development and a decrease in the rate of transverse growth of surrounding craniofacial structures.

3. Compensatory changes seen in the post-restraint period include a decrease in the experimentally induced palatal vault depth and a reassertion of inhibited growth potential in surrounding craniofacial structures with a regression toward normality; i.e., rebound.

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DEDICATED

TO

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who have enriched my life and for
whom my love knows no bounds.

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INTRODUCTION

The idea of using extrinsic restraint to alter the growth patterns of hard and soft tissue is not new. Indeed, the application of such forces to alter various parts of the human body has a long historical and cultural background.

Darwin points out that throughout history, man has deformed himself for any number of reasons (Perez-Martinez, 1960). Whether simply by painting his body, altering body proportions to terrify enemies in battle, or by using various markings to denote the achievement of manhood, strange and exotic alterations of human form have been made in the observance of religious, cultural, and tribal rites.

Alterations of the human body were not done to actually lessen one's appeal; indeed, the feeling of a need to enhance a given concept of beauty is a consistent theme for these alterations. Chinese women of high society bound their feet to restrict growth to a length of only about four inches. Deformation of Guanes Indian skulls was produced by binding pieces of wooden slabs to the head, or simply by binding the head with strips of cloth until the desired

configuration was achieved. These alterations of form by growth restraint suggest that the size, shape and spatial relationships of bones may be altered to various extremes.

The theme of restraint has received a considerable amount of attention in the orthodontic literature. In the early part of the nineteenth century clinicians employed extraoral force to reduce mandibular and maxillary prognathism (Cellier, 1802; Fox, 1803; Gunall, 1822). The occiput was used as a base for anchorage with various attachments to the areas to be restricted. Since elastics were not yet in use for force generation in these appliances, the main objective was passive restraint of ongoing growth. Case (1908) categorized extraoral force as "orthopedic" and stated:

> A movement of other parts quite as important in the reduction of certain facial deformities as the movement of teeth, has long been recognized as within the possibilities of dental force application.

At the turn of the twentieth century, elastic force was used in restraining appliances, and the force system was further categorized as either orthodontic or orthopedic. Lighter forces, whose

effect appeared limited to tooth movement, were categorized as "orthodontic"; whereas heavy forces, which produced apparent skeletal changes, were categorized as "orthopedic". This categorization continues today, and much investigation on the effects of such forces is in the literature. Several investigators (Klein, 1957; Newcomb, 1958; and Ricketts, 1960) questioned whether clinically imperceptible facial and cranial basal bone changes accompanied the obvious dentoalveolar changes seen after extraoral force application. Moore (1959), Ricketts (1960), and Henry (1976) suggested that a posterior force on the teeth could potentially inhibit or redirect maxillary growth. These experimental studies have shown that both direction and magnitude of force are important considerations in the application of extraoral restraint to the craniofacial complex.

Unfortunately, there has been little experimentation relevant to the phenomenon of compensatory growth change after restraint. The present investigation attempts to explore such compensatory phenomena. The maxillary complex of young, growing rabbits was restrained in the transverse dimension by use of a passive, intraoral appliance. These

restraining appliances were removed after a period of time and unrestricted growth and compensatory responses were then allowed to proceed for the remainder of the experimental period. Growth and compensatory changes were analyzed by means of gross observation and direct measurements from serial dental casts of the maxillary dentition serial roetgenographic cephalograms, and histological preparations.

LITERATURE REVIEW

Growth and Development

The growth and development of the craniofacial complex has received considerable investigation. Growth changes have been studied in attempts to determine the developmental mechanisms responsible for the changes in the size, shape, and spatial relationship of the bones of the craniofacial complex.

Brash (1934) analyzed the growth changes of the skulls of madder-fed pigs. Brash concluded that skull growth may be explained solely on the basis of ectocranial surface "accretion" of bone and endocranial resorption. Brash (1934) proposed that this theory of surface accretion and resorption could explain the growth changes of all bones and stated:

> ...that bone growth and the moulding of bone form are solely surface phenomena is in reality the true key to all the problems raised in the consideration of how individual bones grow.

The lateral growth changes seen in the maxillary complex may, therefore, be explained by primary surface accretions on the buccal surfaces of the bones, followed by secondary bone resorption

to maintain form and spatial relationships.

Sicher (1946) largely attributes the growth of the maxillary complex to sutural areas. The sutures of the craniofacial complex most important in the growth of the maxilla are fronto-maxillary suture, the zygomatico-maxillary suture, and the suture between the pyramidal process of the palatine bones and the pterygoid process of the sphenoid bone. Sicher (1946) further hypothesized that these sutures are all parallel to one another. Connective tissue proliferation in this suture system would be responsible for downward and forward growth changes of the maxillary complex. Sicher attributes growth changes in the lateral dimension of the maxillary complex as being due to bone proliferation by the sagittal suture system. This suture system is made up of the median palatine, intermaxillary and metopic sutures, as well as the mandibular symphysis. Sutural growth in these areas is explained as appositional growth on opposing surfaces by which a "spreading" of the sutural area is accomplished (Weinmann and Sicher, 1955). The spreading of a suture is presumably caused by the proliferation of the connective tissue cells of the sutural membrane, with the subsequent apposition of new bone at the two

opposing edges representing a secondary process. In this manner, the lateral dimension of the maxillary complex is increased.

Scott (1953, 1954, 1956, 1957, 1958) theorized that the primary force responsible for craniofacial growth changes is in the cartilage of the chondrocranial and chondrofacial skeleton. Sutural growth changes are considered as passive, secondary, and affected by local and environmental factors. Therefore, the growth of the maxillary complex was presumed to be genetically regulated by endochondral proliferation in the associated masal septum together with the growth of adjacent organs such as the brain, tongue and orbital contents (Scott, 1953, 1956).

Theories of growth and development of the bones of the craniofacial complex could, to this point, be summarized by some combination of the following:

1. That growth changes are explained by the processes of osseous deposition and resorption (Brash, 1934);
2. That the primary expansive forces are within sutural tissues which "pushed" bones apart (Sicher, 1955), or;
3. that expansive forces are within the cephalic cartilages (Scott, 1953).

Implicit in the above presumptions is that the genetic regulatory mechanisms for such growth reside in the sutural and cartilage cells themselves.

Moss (1960 b, 1962, 1968 b, 1969 b, 1971 a, 1972 a) denied any intrinsic regulatory control within cartilage or bone tissues. Moss based his interpretation of the growth process on a concept of interrelated functional cranial components. Simply stated, the head is a composite structure having very different and independent regional functions. Every function is completely carried out by a specific functional cranial component composed of two important parts:

(1) a functional matrix which carries out the function, and

(2) a skeletal unit whose biomechanical role is protective and supportive to its associated functional matrix (Moss and Salentijn, 1969 a).

The term functional matrix is all inclusive of muscles, glands, nerve, fat, blood vessels, teeth, as well as the functioning spaces relative to the bones which they approximate. Fundamental differences between two types of functional matrices exist. The designation reflects the site of activity. Periosteal functional matrices are responsible for transformative growth changes through osseous

deposition and resorption allowing changes in size and shape. Capsular matrices are responsible for translative growth changes, or changes in spatial relationships without directly involving osseous deposition and resorption (Moss and Salentijn, 1969 b).

Skeletal units are variably composed of bone, cartilage, and tendinous tissues - not the equivalent of the "bone" of classical osteology. When such "bone" consists of a number of skeletal units, they are referred to as microskeletal units; i.e., the maxilla itself is composed of a number of microskeletal units. When adjacent processes of neighboring bones are united to form a single cranial component, this is a macroskeletal unit; the maxillary complex is an example.

The basic postulate, then, of the functional matrix theory of growth is that, with the exception of embryonic tissue differentiation, all aspects of craniofacial skeletal tissue growth (osseous, cartilaginous and fibrous) are secondary and compensatory to the primary volumetric expansion of the cranial functional matrices (Moss and Salentijn, 1969 b; Moss and Greenberg, 1967; Moss and Young, 1960).

Enlow (1963, 1965, 1966, 1968, 1975) attests to bones being translated by the expansive force of all the growing soft tissue matrices associated with it -- a process he terms primary or secondary displacement. Primary displacement is the physical movement of a bone that occurs in conjunction with the bone's own growth and remodeling by resorption and deposition. Secondary displacement is the movement of a whole bone caused by the separate enlargement of other bones (Enlow, 1975).

Enlow (1965, 1966, 1968) takes into account the differential growth processes of other tissues when considering the dictates of the growth of the maxillary complex. For example, neurological growth precedes general body growth; therefore, the template for the lateral width of the maxillary complex is influenced by the lateral extent to which the brain and basicranium develop. The rate of growth and development of the anterior cranial fossa is proportional to the development of the frontal lobes of the brain and sets the template for maxillary width from an anatomical standpoint due to the sutures common between the maxilla and its counterpart - the anterior cranial fossa (Enlow, 1973).

The "V" principle of Enlow (1963) is also operative in providing increases in the width of the maxillary complex. This principle accounts for the growth sequence of a V-shaped structure in which increases in size and direction are brought about by a combined process of apposition on the inner surface of the V-shaped structure and resorption from the outer surface. When viewed from the frontal aspect, the maxillary complex may be thought of as an inverted V. Methods of mapping the localized distribution of the various kinds of endosteal and periosteal fields of bone deposition, as documented by Enlow and Bang (1965), have made possible the analysis of the growth of the maxillary complex in just such a view. The inner surface of the V is represented by the oral surface of the maxillary complex, while the outer surface is represented by the nasal surface of the complex and the labial cortex. Accordingly, new fields of osseous deposition are found on the oral surface of the maxillary complex with corresponding, proportionate bone removal from the nasal surface of the maxillary complex. Increase in its transverse dimension is, therefore, brought about by apposition and resorption of bone in specified areas.

Growth Restraint

Restraint of growth, whether delivered through an active or passive force system, experimentally or naturally induced, has been practiced for centuries for varied reasons. Perhaps the most familiar to those in the orthodontic profession is the restraint of prognathic jaws. Cellier (1802) and Fox (1803) utilized extraoral occipital anchorage and a modified hairnet with leather bands attached to a chin cap. Since elastics were not yet in use, the force delivered by nineteenth century restraining appliances was necessarily passive and restrictive. The turn of the twentieth century brought with it the utilization of elastic force in the design of restraining appliances, and such methods were employed by Angle, Knapp and Giulford (1905). Such restraining appliances can be considered to represent active force systems.

Headgear therapy has received much attention in the orthodontic literature in the twentieth century. Studies utilizing experimental animals have examined growth alterations after application of various types of force systems. Contemporaries, such as Droschl (1973), Elder and Tuenge (1974), Meldrum (1975), Henry (1976), and others, have dramatically shown that the

maxillary growth of monkeys can be restrained antero-posteriorly using orthopedic forces (100-800 grams per side) delivered by extraoral high-pull headgear.

Cephalometric and histologic examination of anatomic areas affected by orthopedic force application suggests that extraoral force exerted on the maxillary teeth in a posterosuperior direction is capable of slowing, stopping, or reversing the normal downward and forward displacement of the maxillary complex (Meldrum, 1975; Henry, 1976). These experimental findings tend to agree with similar cephalometric and clinical findings in actual orthodontic cases involving human subjects, as documented by Graber (1967), and Merrifield and Cross (1970).

Orthopedic surgeons, perplexed by the problem of equalizing limb length after an attack of poliomyelitis, began devising methods that would bilaterally equalize the linear growth of long bones. In 1862, Hueter found greater longitudinal growth of a bone when exposed to diminished compressive forces. Volkmann (1869), in founding the basis for epiphyseal stapling, observed that leg growth could be retarded on the side where pressure is abnormally increased. Haas (1945), and Blount and Clarke (1949) used

epiphyseal stapling by inserting staples on each end of the excessively long bone so as to bridge the epiphyseal plate on its medial and lateral aspects. In this manner, growth may be temporarily arrested until the lengths of the right and left limbs were equalized. Various reactive changes in epiphyseal growth rates were noted after removal of the staples. Growth rate can be normal, slower, or faster (Blount and Clarke, 1949).

Post-Restraint Compensatory Changes

Haas (1948) found the restraining force of a tight wire loop encircling the epiphyseal plate of the growing femur in dogs, did not permanently destroy the proliferative properties of the cartilage cells of the plate, and that bone growth resumed after release of the restraining force. Wire loop restraint of the epiphyseal plate of the dog femur increased the rate of growth of the plate, relative to normal growth increases, after loop removal (Haas, 1948). Similar findings in humans to correct linear and angular deformities of long bones do not show such clear-cut results as do the animal experiments. An epiphyseal plate which has become temporarily "thinned" as a

result of compressive epiphyseal stapling, rapidly regains its former thickness after removal of the staples. Blount and Clarke (1949) noted that whether the staples were removed, or left *in situ*, epiphyses which had been restrained with staples usually fused two to six months earlier than normal. This shortening of normal developmental timing was believed to be due to the trauma of the operation and not specifically to the stapling. Normal rate of growth after removal of epiphyseal staples was observed in the correction of idiopathic knock-knee (Blount and Clarke, 1949).

Studies examining the effects on the craniofacial complex during treatment of scoliosis with the Milwaukee brace provide further information on the nature of post-restraint compensatory changes. Orthopedists have been successful in treating spine curvature with the use of this type of brace, although the effects on the facial skeleton can be severe. The brace is constructed so that a pelvic girdle fits tightly around the waist and atop the iliac crests. Two vertical bars extend to the neck region where supports for the mandible and occiput are engaged. The vertical bars may be lengthened to exert pressure upon the occiput and mandible until the curvature of the

spine decreases. Because most cases of scoliosis exhibit themselves, and treatment is initiated while the patient is young and growing, detrimental secondary effects on normal development of the face, cranium and occlusion can result. The first documented case was reported by Howard in 1926 (Blount and Clarke, 1949) involving patients confined for about six months in a body cast. In one year post-treatment studies, Howard found that the patients showed a slight tendency of the facial form to rebound to its former normal state. In one other study demonstrating a rebound effect, Logan (1962) reported a case of a child who wore a Milwaukee brace for nine years. The patient's facial profile exhibited a reduced vertical dimension, yet nine months after the brace was removed, a spontaneous bite-opening and repositioning of teeth took place. Logan attributes this response to assumption of "genetic shape".

Henry (1976) restrained and redirected the growth of the maxillary complex of growing monkeys with heavy, posteriorly-directed forces (800 grams per side) delivered by a high-pull headgear. The positions of the facial and cranial bones were altered relative to each other. During a 26 day post-headgear observation

period, a partial "recovery" of experimentally induced skeletal and dental changes occurred. The craniofacial bones translated toward their original positions (Henry, 1976). Although no long-term cephalometric documentation was available, Henry postulated that the recovery observed was from bone deformation and/or soft tissue compression at the sutures, and that additional rebound was unlikely. This reasoning parallels the rationale presented by Zimring and Isaacson (1968) in a clinical study utilizing humans. Zimring and Isaacson found that, following rapid palatal expansion, an initial rapid phase of rebound occurred when the accumulated stresses of expansion were not yet fully dissipated.

Anatomy

For a detailed account of the anatomy of the rabbit, the interested reader is referred to Bensley's PRACTICAL ANATOMY of the RABBIT (Craigie, 1948).

The maxillary complex is composed of the following bony elements: the paired maxillae, the paired palatine bones, and the paired premaxillary bones. Each maxilla is composed of a body, and five processes -- alveolar, palatine, orbital, zygomatic, and sphenorbital (Sarnat and Wexler, 1965).

The body of the maxilla is extensively fenestrated on its external and internal surfaces and the perforated area extends posteriorly to the anterior rim of the orbit. The alveolar process is that portion of the maxilla that contains the sockets of the cheek-teeth. The cheek-teeth, in the adult rabbit, include three premolars and three molars in each maxilla. The palatine process of the maxilla extends medially and together with the opposite process forms two-thirds of the bony palatine bridge. The orbital process is continuous with the fenestrated portion of the body of the maxilla, and its appearance as a process is mainly due to the solid character as compared to the fenestrated surface anterior to it. The zygomatic process of the maxilla forms the anterior root of the zygomatic arch and fuses with the zygoma prior to the tenth week of postnatal life, thereby closing the zygomatico-maxillary suture (Ritsila, et al., 1972). The ventral surface of the zygomatic process of the maxilla bears a prominent masseteric spine for the attachment of the ligament of the masseter muscle. The sphenorbital process of the maxilla lies on the medial wall of the orbit in a position opposite to the middle portion of the ridge lodging the posterior cheek-teeth (Bensley, 1948).

The palatine bone consists of two portions -- horizontal and perpendicular. The horizontal portion is that portion lying in the plane of the palatal surface. It articulates anteriorly with the palatine process of the maxilla. The perpendicular portion is a vertical plate that extends posteriorly from the palatine bridge (Craigie, 1948).

The premaxilla forms the anterior portion of the maxillary complex. The body of the premaxilla contains an alveolar portion containing the large and small incisor teeth, a frontal process, and a palatine process. The body forms a portion of the palatal surface of the skull (Craigie, 1948).

STATEMENT OF THESIS

Restraint studies (Droschl, 1973; Elder and Tuenge, 1974; Meldrum, 1975; Henry, 1976) have shown that the growth of the maxillary complex may be slowed, stopped or reversed in its normal downward and forward displacement.

It is unclear how much of the growth inhibited during restraint is compensated for when the restraint is removed (Haas, 1948; Blount and Clarke, 1949; Logan, 1962; Henry, 1976). Therefore, it is the objective of this investigation to evaluate the nature of compensatory changes that occur in the maxillary complex of growing rabbits subsequent to removal of a maxillary growth-restraining appliance. Growth and compensatory changes, in comparison to control groups, will be quantitated and evaluated cephalometrically, histologically, and by the use of dental casts to determine to what extent the potential for full maxillary growth will become expressed when the constraining effect is released.

METHODS AND MATERIALS

Animals

Twelve growing New Zealand White rabbits were utilized in this study. All rabbits were 30 days old at the commencement of the experimental period and fed on Purina Rabbit Chow (Ralston Purina Co., St. Louis, Missouri) and tap water *ad libitum*. Sex of the experimental animals was not considered to be a significant factor, as based on a study by Lowrance (1955) in which no significant differences between increases in actual lengths and diameters of the femora between male and female rabbits were found. Initial weights ranged from 357 grams to 814 grams (Table I).

The rabbit was chosen as the experimental model primarily because of its rapid growth rate. By 15 days after birth, 32-44 per cent of adult growth has occurred (Lowrance, 1955). In the period between 15 and 43 days postpartem, growth is rapid and the rate is relatively constant (Lowrance, 1955). Therefore, the experimental phase of restraint was selected to occur between the 30th and 45th days of life.

TABLE I

INITIAL WEIGHTS

OF ANIMALS (grams)

EXPERIMENTALS		CONTROLS	
#1	475	#1	751
#2	490	#2	706
#3	430	#3	619
#4	477	#4	814
#5	357	#5	599
#6	417	-	
#7	467	-	

The rabbits were weaned at 3 weeks and were allowed to become acclimated to their environment and feeding habits. At the beginning of the experimental period the rabbits were therefore weaned, acclimated, feeding on rabbit chow and water, physically able to withstand the surgery necessary to place the appliance, and still well within a period of rapid growth. After the designated period of rapid growth rate (15-43 days), the rate of growth continues with a very slight decrease (Lowrance, 1955). Therefore, Phase II (unrestricted growth) is still within a period of relatively rapid growth. If the potential for compensatory growth exists upon removal of the restraining influence, these compensations should more intensely express themselves while the growth rate is rapid (Stutzmann and Petrovic, 1977).

Experimental Design

Two groups of rabbits were used. Group I consisted of seven littermates that received a maxillary restraining appliance. Group II was composed of five littermates that served as unoperated controls.

Serial maxillary impressions were made from all the animals in both groups, and dorsoventral

cephalometric radiographs were taken at three intervals. Initially, maxillary impressions of each rabbit were made, utilizing silicone impression material (ORMCO, Glendora, CA.) in custom acrylic trays one day prior to the surgical placement of the restraining appliance. The dental casts of the rabbits at this initial phase served a two-fold purpose; (1) as the first serial study cast to compare with later study casts by a quantitative analysis, and (2) as working casts upon which the restraining appliances were fabricated. All rabbits in Group I underwent 15 days of passive restraint before appliances were removed. The rabbits of Group II received no restraining appliances. Serial maxillary impressions and dorsoventral cephalograms of all rabbits were taken after the initial 15 days of passive restraint (Group I), or no appliance therapy (Group II). The second phase of the experiment involved a period of unrestricted growth which began when the appliances were removed. All animals remained in an unrestricted growth phase for 30 days until their sacrifice. Maxillary impressions and dorsoventral cephalograms were taken after the sacrifice of the animals.

Two important phases were thereby created:

1. Phase I consisted of passive restraint of the maxillary complex and spanned experimental days 1 through 15.

2. Phase II consisted of unrestricted growth of the maxillary complex and spanned experimental days 16 through 45.

Restraining Appliance

The appliance utilized in this experiment was designed to passively restrain the growth of the maxillary complex in its transverse dimension. The transpalatal dimension of growth would, therefore, necessarily be altered.

Maxillary silicone impressions were made of all rabbits in Groups I and II, and the impressions were poured in dental die-stone (Kerr Velva-Mix, Emeryville, California). The appliance was fabricated directly on the working casts of all rabbits in Group I. Self-curing acrylic (Caulk Orthodontic Resin, L. D. Caulk Co., Milford, Delaware) was applied to the working casts to form an appliance having the following specifications:

1. The appliance is to be made entirely of acrylic, and
2. extend from the depth of one buccal vestibule of the maxillary complex, over and across the teeth of the maxilla proper to the buccal vestibule of the opposite side, while it
3. traverses and contacts the soft tissue of the palatal vault.

4. The appliance is to be thick enough (1 mm ± 0.25 mm) to act as a biteplate and resist the forces of mastication.

5. The appliance is to extend posteriorly no further than the maxillary tuberosity area and the soft palate, and

6. extend anteriorly only as far as the maxillary alveolus that supports the first premolar.

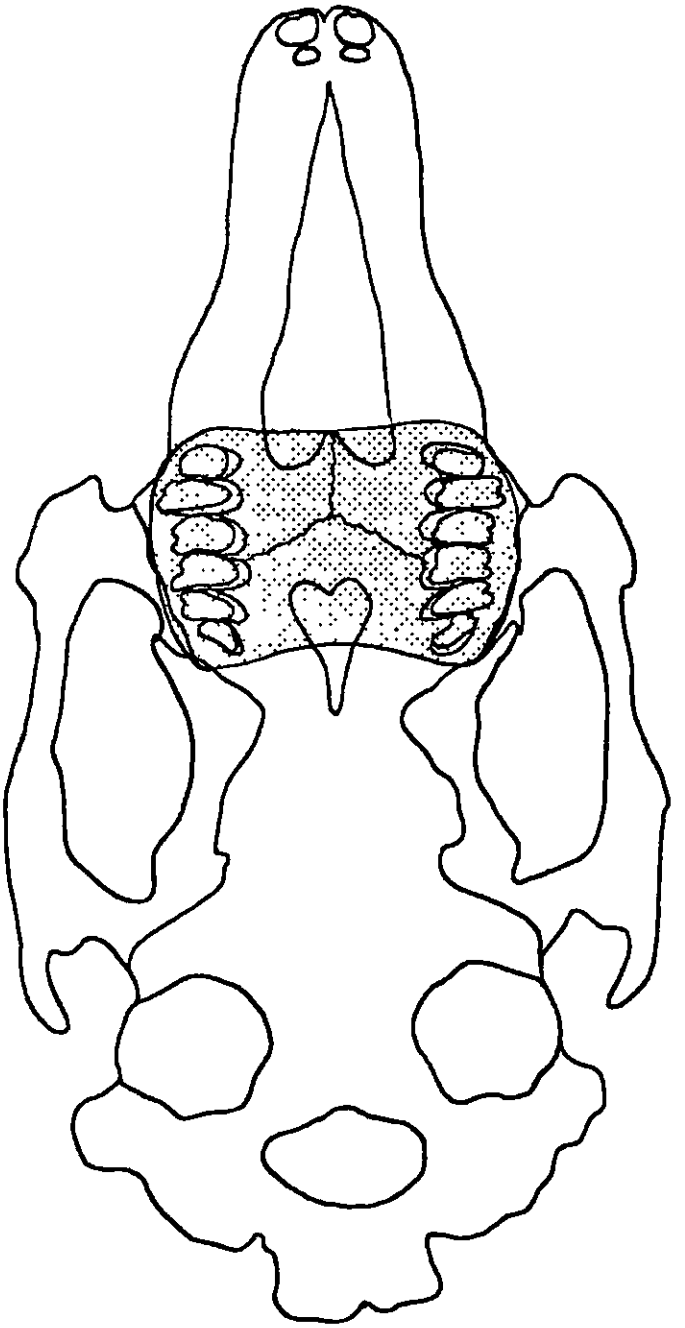
The diastema between the first premolar and the incisors of the premaxilla, and the incisors proper, are not to be encompassed by the appliance (Figure I).

The appliance, by design, has its own retentive properties from the undercuts and embrasures of the teeth and of the maxillary alveolus. However, to insure that the appliance could not be dislodged by the active, feeding rabbit, two small holes were drilled into the appliance opposite the first premolar and the first molar to act as eyelets for .010" stainless steel ligature wires for circumzygomatic ligation.

Anesthesia

Intramuscular injection of Xylazine (Rompum, 5 mgs/Kg. of body weight; Cutter Labs, Inc., Shawnee, Kansas), and ketamine hydrochloride (Ketaset, 30-50 mgs./Kg. of body weight; Bristol Labs, Syracuse, New York), were utilized to induce anesthesia.

Figure 1. - Schematic
Representation of anatomical area
covered by the restraining appliance.



Surgical Procedure

The surgical procedure was performed when the rabbits were 30 days old. Once anesthetized, the experimental animals of Group I were secured on an operating board in a semiprone position. Access to the oral cavity was aided by two .020" brass wire loops hooked around the upper and lower incisors. To gain access to the zygomatic process of the maxilla, a 5 mm incision was made anteroposteriorly parallel to the long axis of the orbit approximately 5 to 7 mm inferior to the lower eyelid along the bony prominence of the zygomatic arch. A sterile, swedged, straight needle was placed through the incision in an anteroinferior direction medial to the zygomatic arch until it appeared intraorally in the buccal vestibule opposite the maxillary first molar. At that time, forceps were introduced into the oral cavity to secure the needle and pull it through until the .010" stainless steel ligature secured to the end of the 3-0 black silk suture thread passed through the infraorbital incision appeared intraorally. The needle was then passed through the posterior eyelet of the appliance superoinferiorly and then passed through the anterior eyelet of the appliance inferosuperiorly,

thus threading the ligature wire through the appliance. The needle was then guided with forceps to the buccal vestibule opposite the first premolar to puncture the oral mucosa. The needle was then passed in close approximation to the anterior border of the zygomatic arch until it appeared through the infraorbital incision. In doing so, the appliance was secured tightly to the maxillary complex by twisting the two ends of the ligature wires present at the infraorbital incision. Once twisted securely, the long "pigtail" was shortened to approximately 3 mms. The infra-orbital incision was swabbed off with Povidone-iodine (Betadine surgical scrub, Purdee-Frederick Co., Norwalk, Connecticut) and the incised edges were approximated with one black silk suture. The steel ligature was left protruding slightly through the incision to aid in the removal of the appliance. Infection proved to be a minimal complication and sub-sided a few days post-operatively.

Vital Dyes

Immediately post-operatively, Oxytetracycline (Terramycin Oxytetracycline, 50 mgs/Kg. of body weight; Pfizer Labs, New York, New York), was injected

intramuscularly. Its purpose was two-fold; to provide an *in vivo* histologically detectable indicator of newly proliferated bone tissue, and to serve as a prophylactic antibiotic to minimize the potential for infection in the period immediately following the surgical placement of the appliance. The beginning of the restraint period could, in this manner, be identified microscopically by the presence of a fluorescent band of bone tissue. A second vital dye indicator, Alizurin Complexon (30 mgs/Kg. of body weight; Eastman Kodak Co., Rochester, New York) in a 2 per cent solution of $NaHCO_3$, was injected intraperitoneally at the beginning of the unrestricted growth phase when the appliance was removed, thereby marking this important phase histologically.

Serial Cephalometric Roentgenography

Dorsoventral cephalograms were taken of each rabbit in Group I immediately after the appliance was placed, upon removal of the appliance, and at death. The control group received a similar serial regimen of dorsoventral cephalograms. The cephalostat used was a modification of that designed by Selman and Sarnat, 1953 (see Plate I).

Plate I - Rabbit cephalometer

The radiographs were taken on a standard dental occlusal film (Kodak Occlusal Film, DF-46), which was used at a duration of 3 seconds at 15 mA and 70 KVP. The central ray was perpendicular to the film and directed at the midpoint of the orbital axis. All films were developed in an automatic film processor to insure constancy.

Cephalometric Landmarks

The radiographs were mounted in a photographic enlarger and enlarged to provide 3 times magnification to aid in the identification of the cephalometric landmarks. The following points were punched directly onto 8½" x 11" white paper (Figure 2):

1. the average of right and left superior prosthion, (P);
2. the anterior-most portion of the maxillary alveolus as depicted by the anterior-most point on the concavity formed by the periodontal membrane of the maxillary first premolar, (Ma);
3. the anterior-most point on the concavity formed by the zygomatic arch (Mx) representing maxillary basal bone;
4. the construct of a point (Zg) on the most lateral aspect of the zygomatic arch resulting from a line drawn perpendicular to the midsagittal line (M) and bisecting points (Zg);

5. the most anterior point on the convexity formed by the tympanic bullae (Tb);

6. the most lateral point on the convexity formed by the tympanic bullae (Cb);

7. the anterior-most portion of the foramen magnum (B);

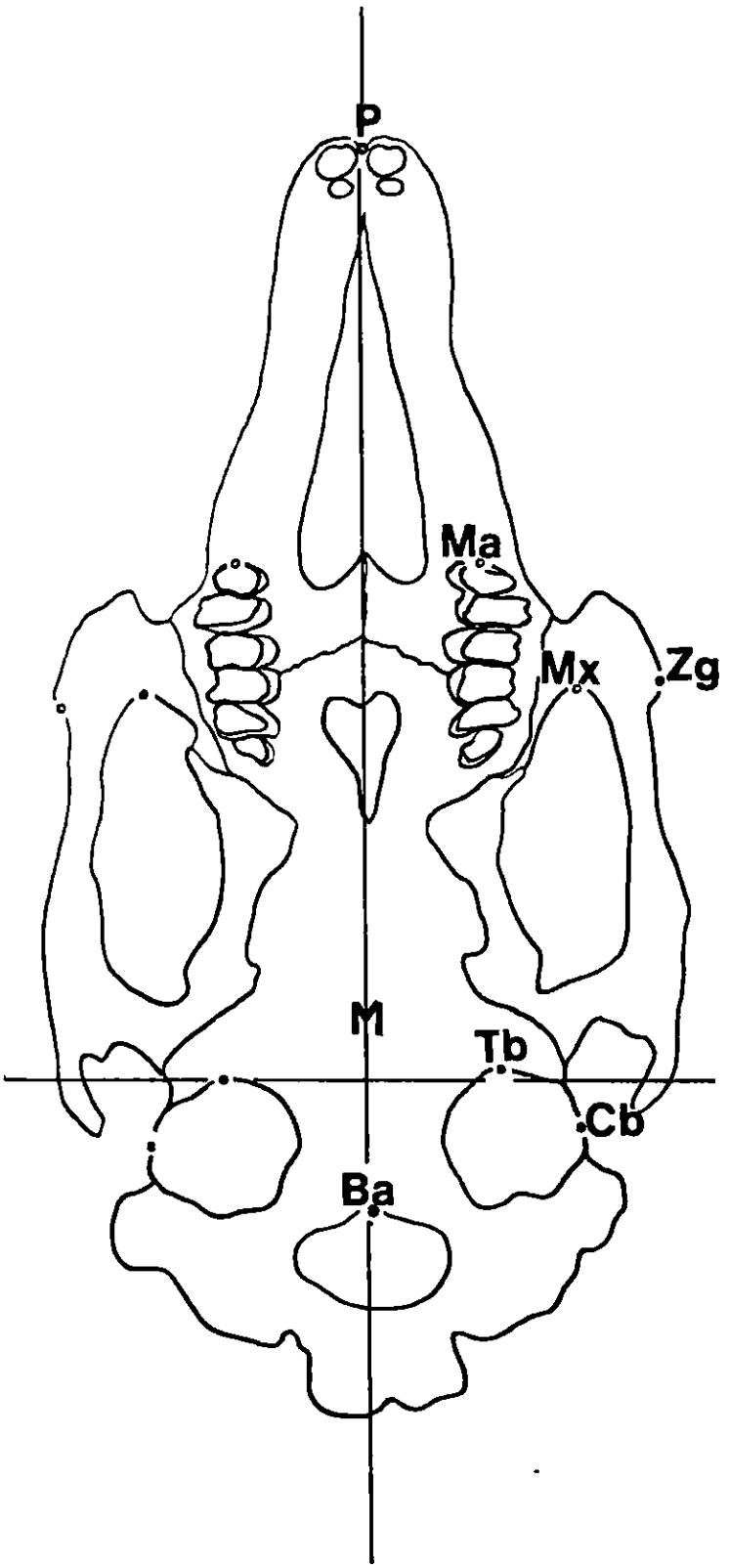
8. a midsagittal line constructed by connecting points (P), (B), (M).

Cephalometric Analysis

Direct measurements for the transverse dimension of the maxillary complex were taken between the right and left cephalometric points. Each cephalogram was oriented using acetate tracing paper (.003" matte) with fine perpendiculars (abscissa and ordinate) placed on it with a cephalometric tracing pencil, (J-87 Rocky Mountain Orthodontics). The cephalogram was oriented so that the abscissa passed through the right and left tympanic bulla (Tb) points, and the ordinate passed through the midsagittal plane (M) defined by a line passing through basion (B) and the average of superior prosthion (P).

A total of six measurements were used to quantitate growth changes of the maxillary complex. Measurements taken to the midsagittal ordinate were perpendicular to it. Two of the six measures were to quantitate growth anterposteriorly and were diagonal

Figure 2. -- Cephalometric landmarks


P
Ma
Mx
Zg
M
Tb
Cb
Ba

to the midsagittal plane. The various measures are listed in Table II and illustrated by Figure 3.

Dental Cast Analysis

Direct measurements quantifying the transverse dentition changes were taken on each serial study model. Transverse measurements were made on the most prominent point of each group of cheek-teeth, and these values were averaged to determine the mean change in transpalatal width between experimental phases.

Palatal depth measurements were taken of the palatal vault on the midline raphe in the area of the maxillary first molars with the use of a depth guide. Measurements were taken to the nearest 0.05mm with dial vernier calipers (Helios Calipers, DCA Corporation, Washington, D.C.)

Histological Evaluation

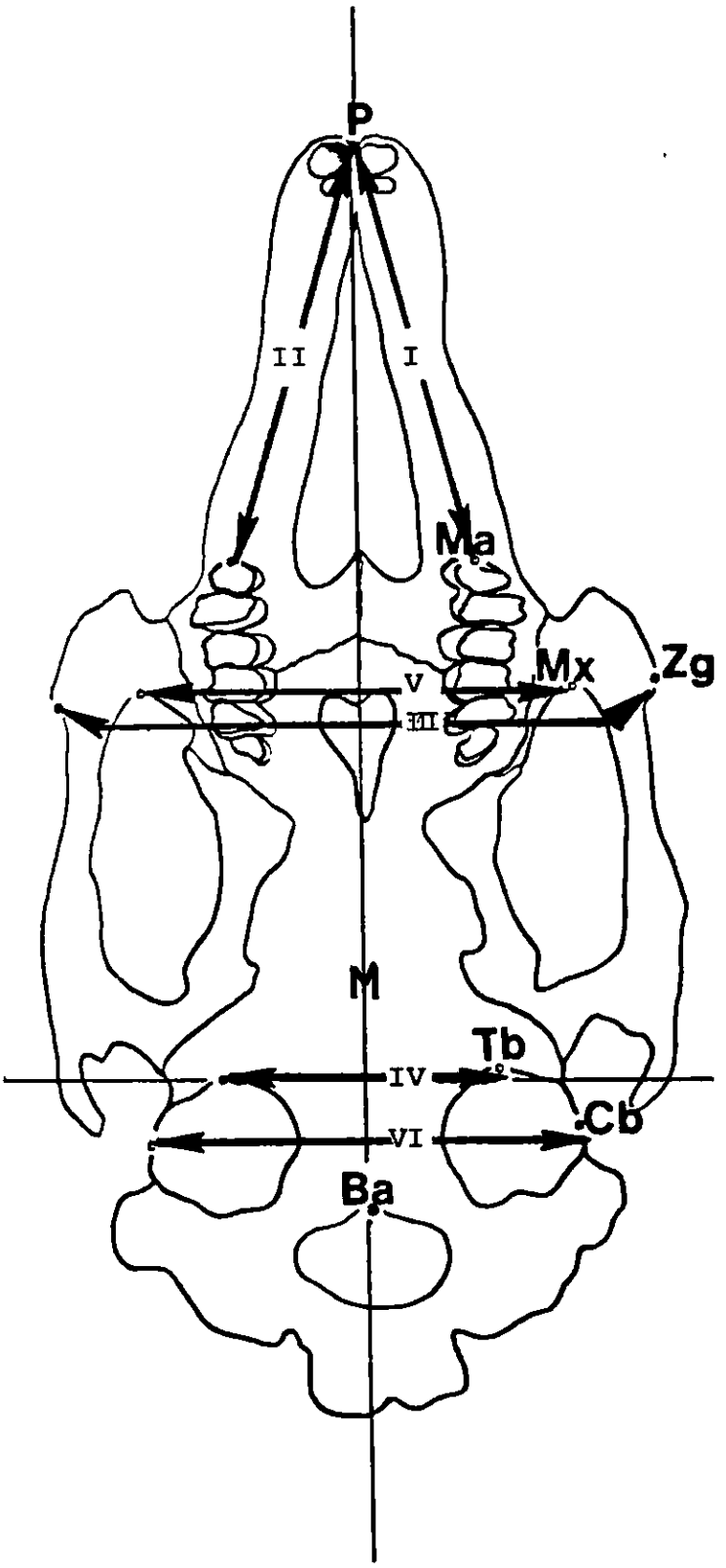
The animals were killed by a lethal dose of pentobarbitol sodium and perfused with approximately 300cc of 10 per cent buffered formalin. Immediately after perfusion the heads were removed and placed in formalin. Undecalcified thin sections and routine hemotoxylin and eosin stained and decalcified sections

TABLE II

Cephalometric Analysis

	Measure	Landmarks Employed	
I	Anteroposterior snout development (right)	Ma right	P
II	Anteroposterior snout development (left)	Ma left	P
III	Zygomatic width	Zg right	Zg left
IV	Cranial base width	Tb right	Tb left
V	Maxillary width	Mx right	Mx left
VI	Posterior skull width	Cb right	Cb left

Figure 3. -- Cephalometric analysis
Measures I and II represent anteroposterior development.
Measures III through VI represent transverse development.


P
II
I
Ma
Mx
Zg
V
III
M
Tb
IV
Cb
VI
Ba

of the maxillary complex were prepared for microscopic examination to determine the nature of the histological compensatory changes which occurred.

Data Reduction:

A determination of a statistically significant difference between the initial weights of the control group and the experimental group was based on the t - test. The initial weights of the littermates of the control group were significantly greater than the initial weights of the littermates of the experimental group (Table I). No satisfactory adjustment factor could be derived to allow for comparisons of the data collected from each group for the purpose of statistical inference. Therefore, strong statistical inference could not be derived due to a small sample size coupled with a large difference in initial weights. Therefore, the results of this study will be presented from a descriptive, rather than inferential statistical evaluation. Mean ($\bar{X}$) and standard deviation (S.D.) values for the control and experimental groups (Table III) were utilized in the construction of all graphs.

RESULTS

The results of the present study will be presented under the following headings: (1) Gross Observations; (2) Dental Cast Analysis; (3) Cephalometric Analysis; and (4) Histologic Evaluation.

Gross Observations

Visual examination of the animals after the appliance had been surgically placed revealed an experimentally induced open-bite due to the biteplane effect of the restraining appliance. This effect prevented the usual attrition of the incisors and allowed for overeruption of the incisor teeth with the result that an anterior cross-bite was created (Plate II). No attempt was made to correct this experimentally induced malocclusion once the restraining appliance was removed because no deleterious effects in eating habits were noted.

Eye infections proved to be a troublesome post-operative complication. Three of the 7 animals in Group I developed a purulent exudate about the eyes which later crusted over and resolved itself without any interceptive treatment.

Plate II - Treatment - induced
incisor over-eruption

Dry skull examination of one of the experimental rabbits showed epiphytic bone proliferation in the area of the right masseteric spine of the zygoma. This same rabbit had an obvious post-operative subcutaneous infection in this area.

Dental Cast Analysis

The results of the dental cast analysis will be presented in two parts: (1) the average maxillary dental arch width; and (2) the average palatal depth. Treatment data are summarized in Table III.

The data collected at three intervals are graphically depicted in Figures 4 and 5. The rate of increase in maxillary dental arch width (Figure 4) for the control group was gradual in Phase I and greater in Phase II. In the experimental group, the rate of maxillary dental arch width increase was very slight during the restraining period (Phase I), but only very gradually increased during the unrestricted growth period (Phase II).

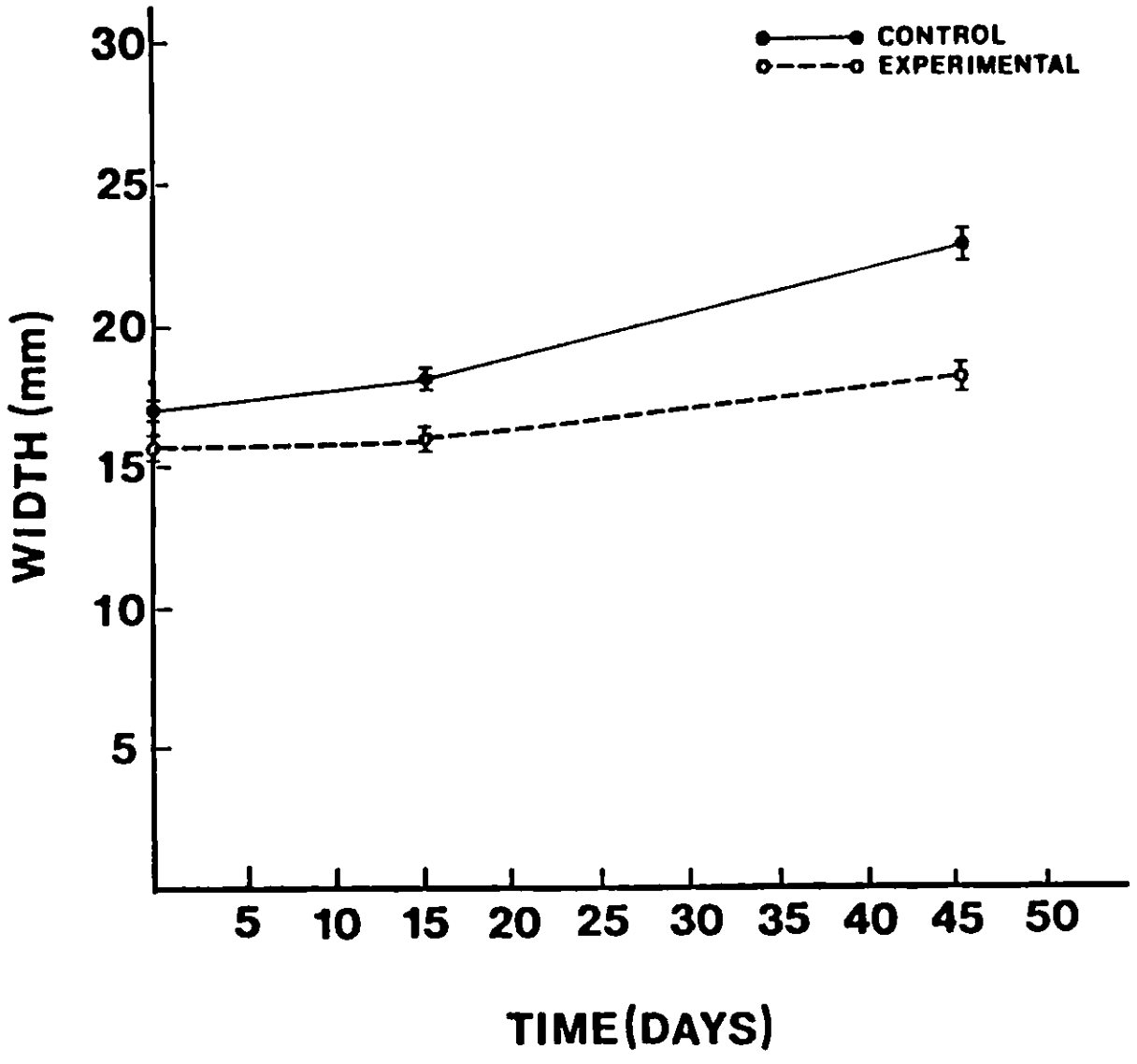
The average palatal depth of the control group gradually increased throughout the course of the experiment. The average palatal depth of the experimental group, however, increased sharply in

TABLE III

SUMMARY TABLE FOR RESULTS
of the
CEPHALOMETRIC AND DENTAL CAST ANALYSES

Measure	ANIMAL GROUP											
	CONTROL						EXPERIMENTAL					
	Day 1		Day 15		Day 45		Day 1		Day 15		Day 45	
	$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.
Maxillary Dental arch width	16.87	0.31	18.09	0.29	20.82	0.89	15.56	0.37	15.85	0.53	18.04	0.67
palatal depth	4.47	0.05	4.58	0.05	5.27	0.08	4.00	0.19	5.33	0.19	4.92	0.11
cephalom. I	54.54	1.78	57.05	1.28	70.74	2.72	44.82	3.19	48.78	2.46	60.68	3.85
cephalom. II	53.97	1.71	56.35	2.13	70.25	3.12	44.58	2.98	48.81	2.56	60.31	3.62
cephalom. III	80.90	2.39	93.06	2.73	102.63	4.49	76.73	1.48	83.70	2.17	97.69	3.17
cephalom. IV	49.05	1.59	53.39	1.82	58.68	2.69	46.71	1.70	49.33	1.49	55.51	3.16
cephalom. V	63.54	1.97	72.26	1.99	81.04	1.98	60.15	1.41	66.04	1.59	76.57	2.06
cephalom. VI	71.06	2.70	78.29	2.24	84.84	2.77	68.28	1.12	72.65	0.58	77.02	3.80

Figure 4. -- Dental cast analysis
Average maxillary dental arch width


CONTROL
EXPERIMENTAL
30
25
20
15
10
5
WIDTH (mm)
5
10
15
20
25
30
35
40
45
50
TIME(DAYS)

the restraint phase and then gradually decreased throughout the remainder of the period (Figure 5).

Cephalometric Analysis

Cephalometric analyses comprised two measures of anteroposterior development and four measures of transverse development. Cephalometric measures I and II reflect the average extent of anteroposterior development of the snout. The control group shows a gradual increase in the rate of snout development during Phase I of the experiment and an increased rate of anteroposterior development during Phase II (Figure 6). In comparison, the experimental group shows similar trends, although the rate of increase in anteroposterior snout development is greater in the experimental animals during the restraint phase. It is comparable to the control group in terms of the rate of increase in anteroposterior snout development during the unrestricted growth phase.

The cephalaometric assessment of transverse development is depicted by Figures 7 - 10. All cephalometric measures relative to transverse development show similar trends. For the control group, the

Figure 5. -- Dental cast analysis
Average palatal depth

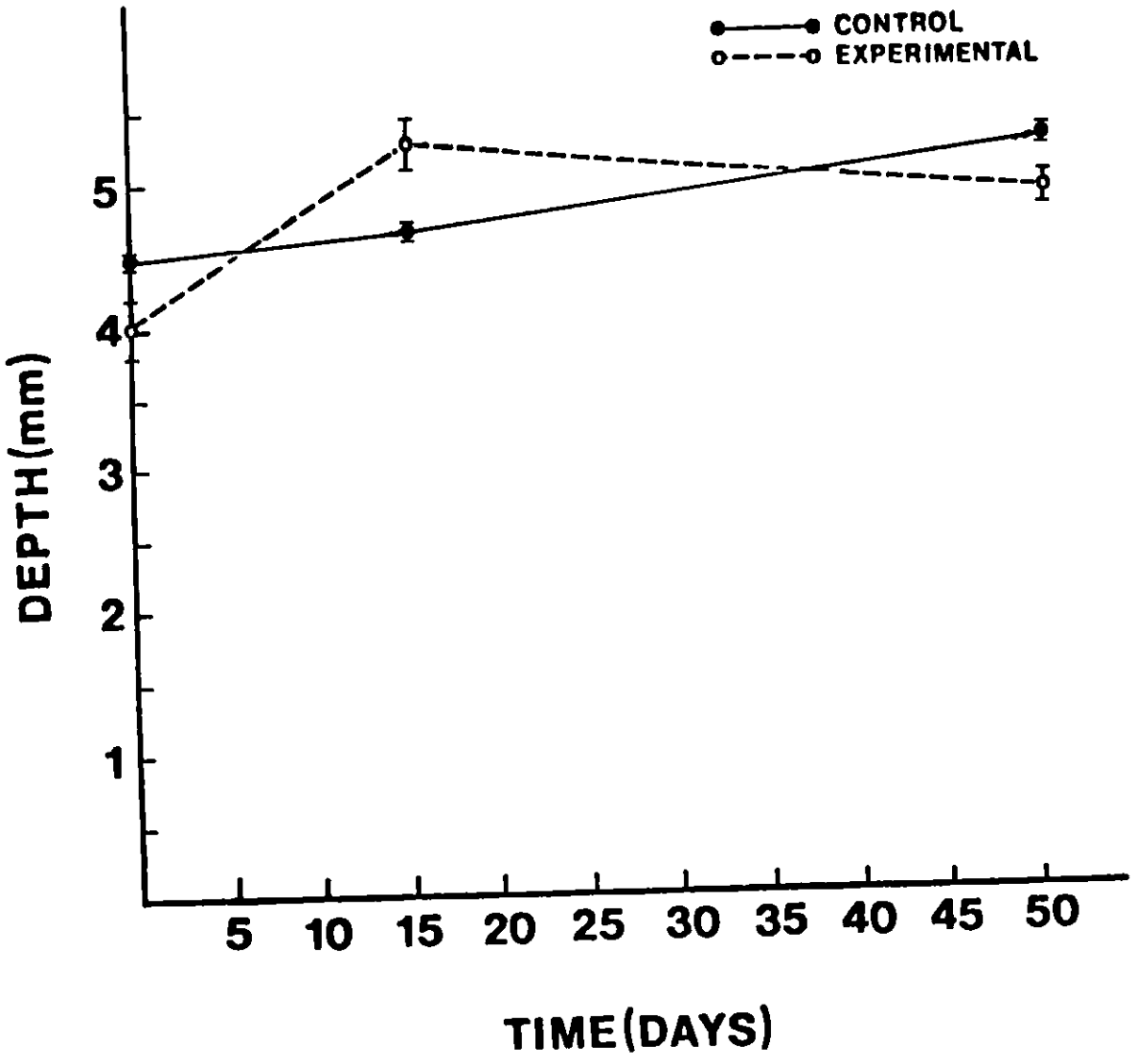
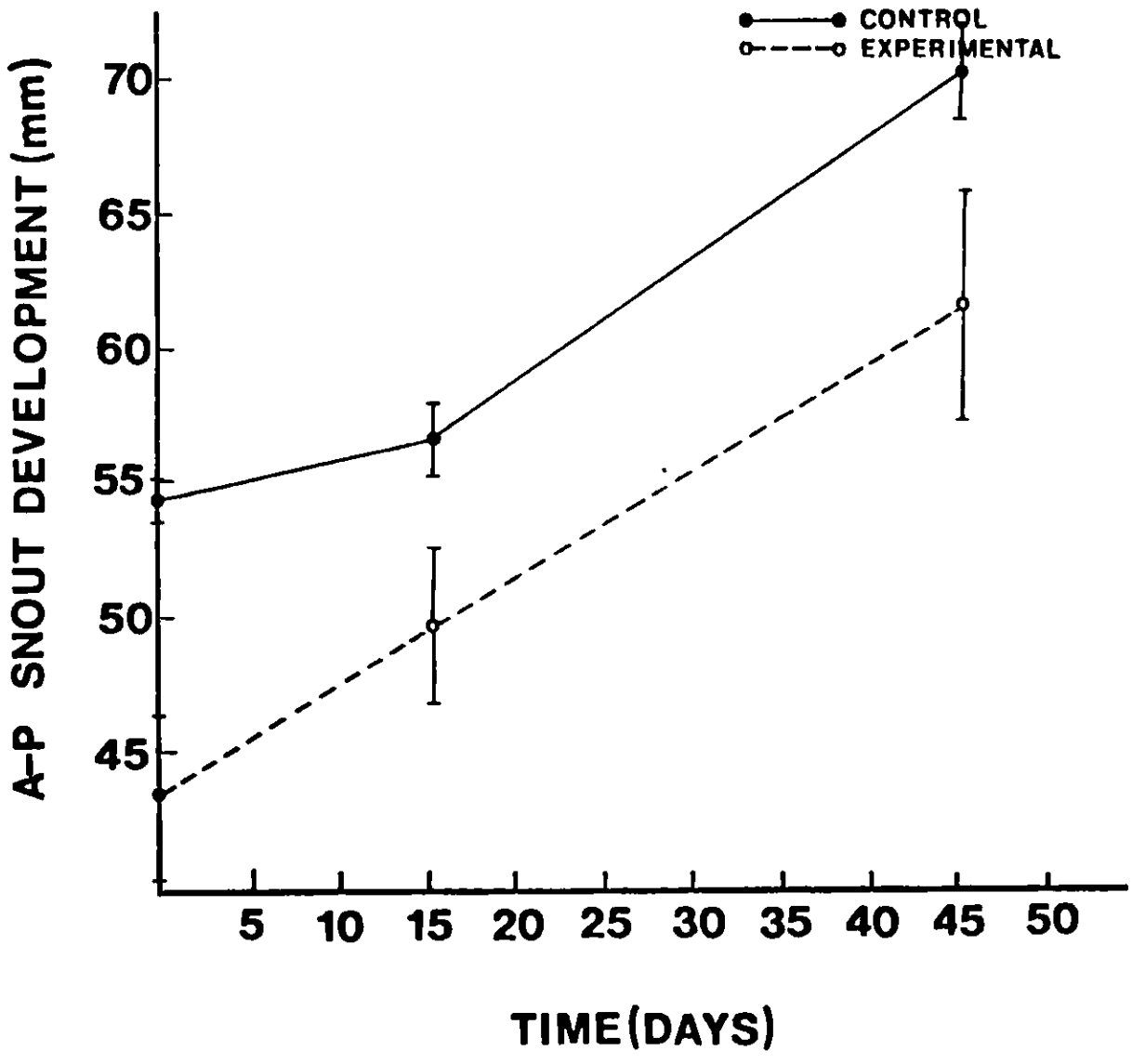

CONTROL
EXPERIMENTAL
DEPTH(mm)
1
2
3
4
5
5
10
15
20
25
30
35
40
45
50
TIME(DAYS)

Figure 6. -- Cephalometric analysis
Average anteroposterior snout development.
All measurements are X3 due to
photographic enlargement.


CONTROL
EXPERIMENTAL
A-P SNOUT DEVELOPMENT (mm)
70
65
60
55
50
45
5
10
15
20
25
30
35
40
45
50
TIME (DAYS)

average zygomatic width, cranial base width, maxillary bone width, and posterior skull measurements all increased rapidly during Phase I of the experiment, but exhibit a slower rate of increase thereafter (Figures 7-10). Variability in growth rate of these areas between Phases I and II can be recognized by the differences shown in these graphs. The increase in average transverse development of these same areas of the experimental group, however, is more linear, and a distinction in growth rate between Phases I and II is not as readily discernible.

Histological Evaluation

Histologically detectable differences between the control and experimental groups were not apparent upon examination of decalcified sections. Midpalatal and maxillopalatine suture activity, as well as bony remodeling activity on the oral and nasal surfaces of the palatal vault were essentially identical in both groups. Microscopic examinations of thin undecalcified sections were performed with a fluorescent microscope at 40X magnification. This provided a determination of the amount of bone tissue deposition that occurred between the vital dye lines and, therefore,

Figure 7. -- Cephalometric analysis
Average zygomatic width.
All measurements are X3 due to
photographic enlargement.

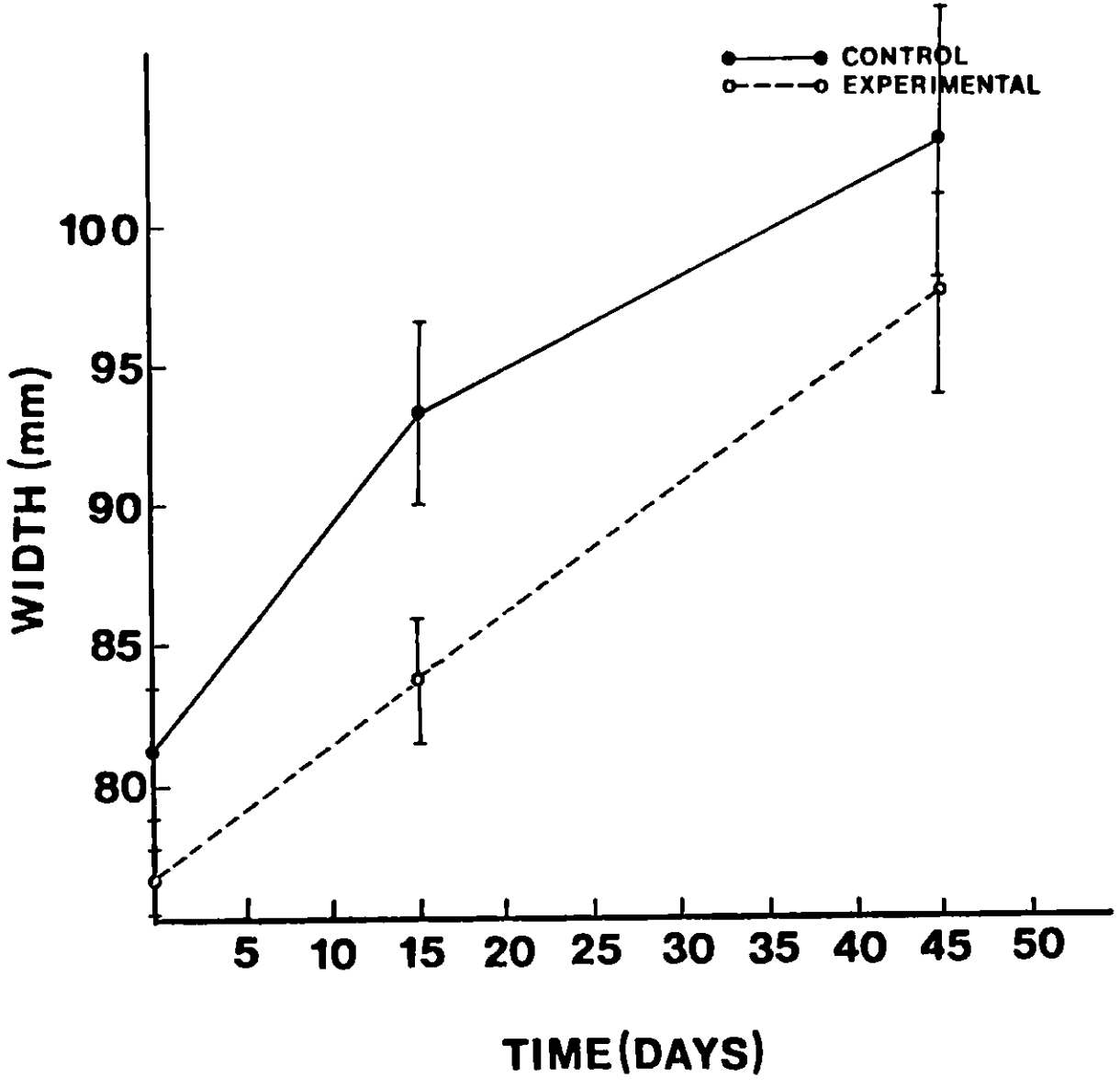

CONTROL
EXPERIMENTAL
100
95
90
85
80
WIDTH (mm)
5
10
15
20
25
30
35
40
45
50
TIME(DAYS)

Figure 8. -- Cephalometric analysis
Average cranial base width.
All measurements are X3 due to
photographic enlargement.

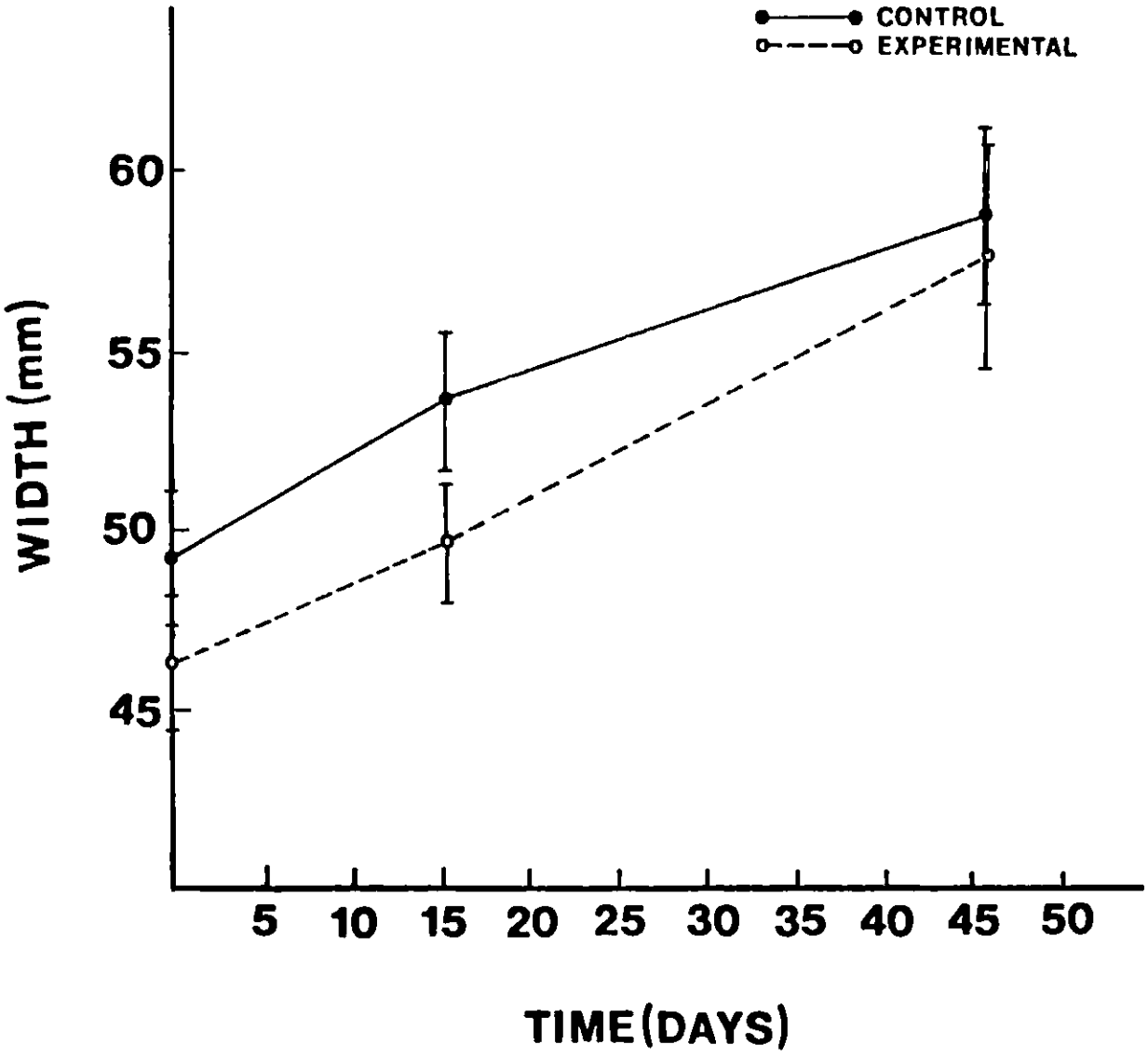

CONTROL
EXPERIMENTAL
WIDTH (mm)
60
55
50
45
5
10
15
20
25
30
35
40
45
50
TIME (DAYS)

Figure 9. -- Cephalometric analysis
Average maxillary width.
All measurements are X3 due to
photographic enlargement.

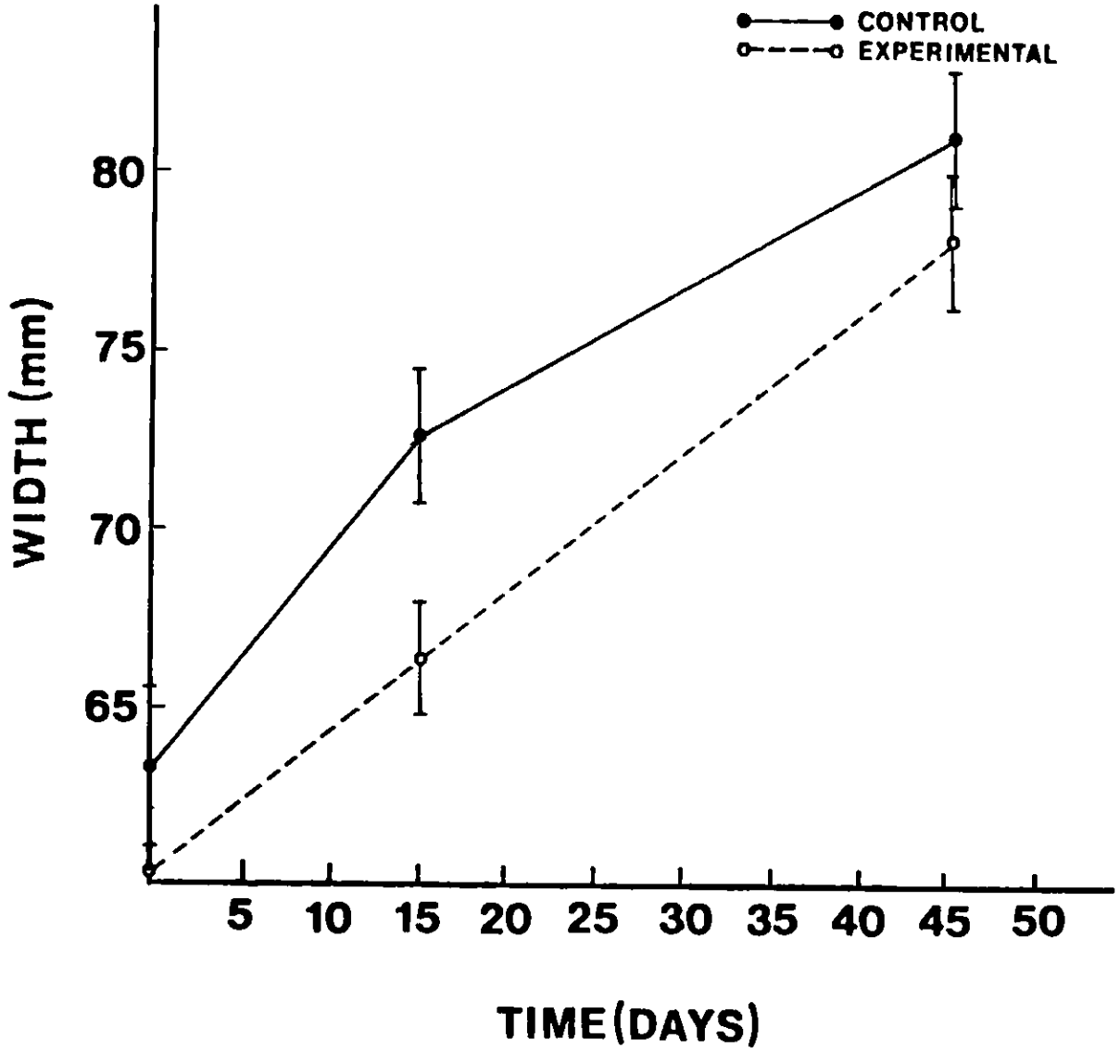
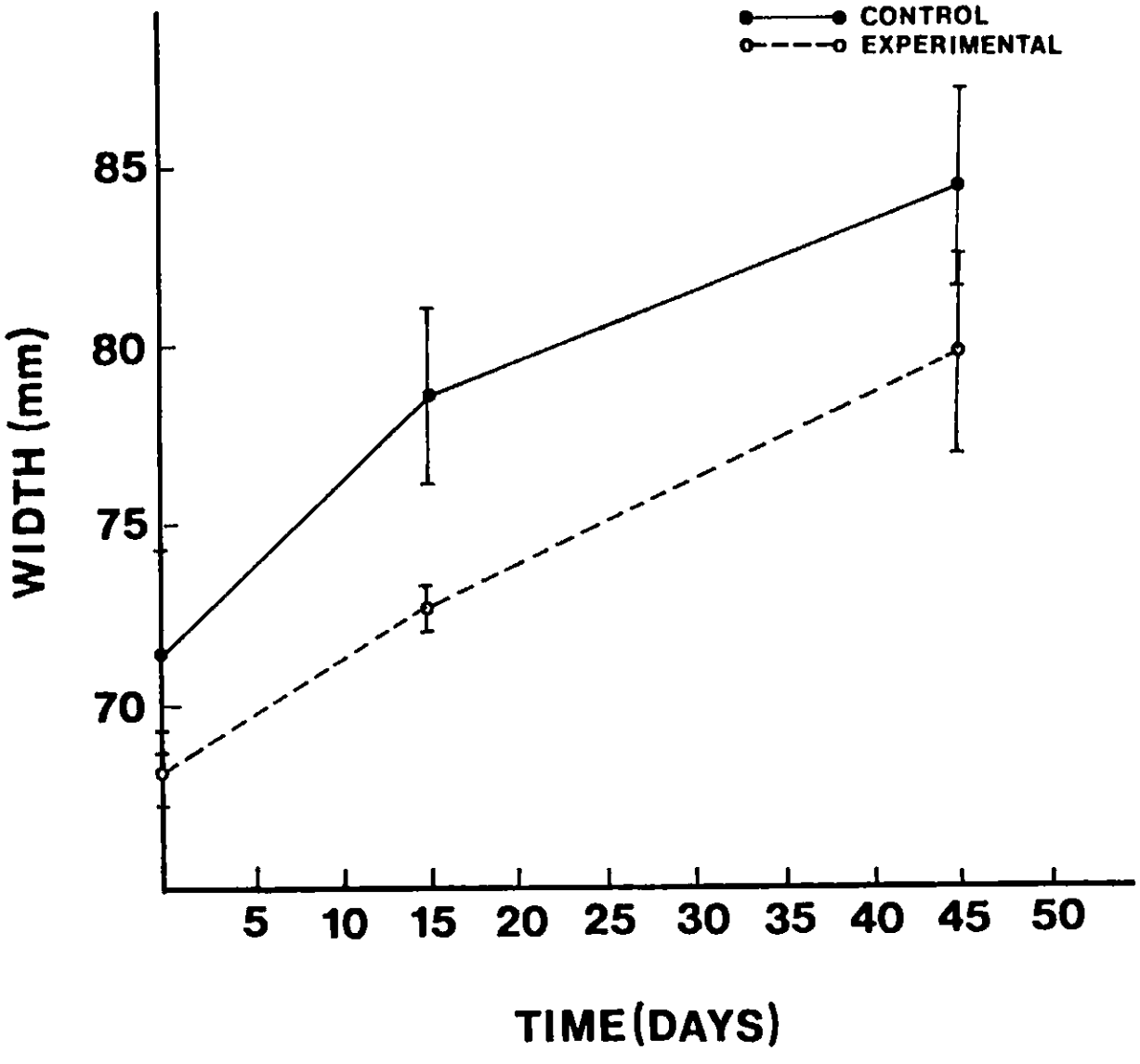

CONTROL
EXPERIMENTAL
80
75
70
65
WIDTH (mm)
5
10
15
20
25
30
35
40
45
50
TIME (DAYS)

Figure 10. -- Cephalometric analysis
Average posterior skull width.
All measurements are X3 due to
photographic enlargement.


CONTROL
EXPERIMENTAL
WIDTH (mm)
85
80
75
70
5
10
15
20
25
30
35
40
45
50
TIME (DAYS)

the extent of bone deposition during each phase of the experiment. No attempt was made, however, to precisely quantitate the amounts of new bone tissue deposited during the two growth phases because slight differences in planes of sections among the different preparations can introduce misleading errors in measurement. Rather, only approximations involving significant bony pattern differences were noted.

The amount of bone deposition between the initial tetracycline line and the alizurin line, was proportionate to the total amount of bone deposited in the control group. In the experimental group the tetracycline and alizurin lines were much closer, with less new bone between them, thus indicating that less new bone tissue was deposited during the restricted phase. A relatively greater amount of bone deposition took place subsequent to the alizurin line in the experimental group, as compared to the same period in the control group, indicating a compensation for inhibited growth by reassertion of bone deposition in the unrestricted growth phase took place during the unrestricted growth phase.

Fluoroscopic examination of the undecalcified sections also allowed for "mapping" of the

osseous activity of the palatal vault. The undecalcified frontal sections of the palate and alveolus were examined and some generalizations may be made. The palate is depository on both the oral and nasal sides in the anterior two-thirds of the bony palatal bridge. The posterior one-third is made up mainly of the horizontal portion of the palatine bones. This area was found to be depository laterally but resorptive in all other areas on the nasal surface of the palate and depository on the whole oral surface. Similar findings were found in both the control and experimental groups.

DISCUSSION

The purpose of this investigation is to examine the nature of compensatory responses during and after passive restraint of the maxillary complex. The findings show that passive transverse restraint placed on the maxillary complex can retard its transverse growth rate. During the post-restraint period, however, a reassertion of the growth potential, i.e., rebound, appears to occur in which changes toward "normal" dimensions take place.

Results of the dental cast analysis assessing compensations with respect to (1) maxillary dental arch changes in its transverse dimension and, (2) palatal depth changes suggest that both were structurally affected by the restraining appliance. Growth increases in the maxillary dental arch were slight during the restraint phase in the experimental group (Figure 4). This effect may have been caused by : (1) a decrease in general body development due to impaired eating habits resulting from the appliance; (2) retardation of midpalatal sutural growth activity; or (3) a "warping" of the dental arch form and maxillary alveolus.

A study performed by Stutzmann and Petrovic (1976), in which local mechanical restraint from traction between intraosseous hooks placed adjacent to the premaxillo-maxillary suture, showed a significant slowing of sutural growth in this region. This finding supports an explanation for decreased transverse growth of the maxillary dental arch width as caused by slowing of midpalatal suture activity. The histological findings of this study do not support a decrease in midpalatal suture activity during restraint. Since the histological preparations were done on animals that were long released from the restraint period, it may be argued that midpalatal sutural activity, slowed during and immediately after restraint, was reasserted and a histologic return to normal sutural activity occurred by the time the histological preparations were made.

Palatal depth alterations were the most dramatic compensatory responses noted. During the restraint phase, palatal depth in the experimental group, which was initially less than the palatal depth of the control group, increased dramatically to the extent that it was deeper than the palatal depth of the control group. However, after the restraining

appliances were removed, there was a correspondingly dramatic decrease in the depth of the palate, with regression (rebound) toward normality (Figure 5). The observed result may be due to an unaltered development of the vertical dimension of nasomaxillary growth, as the appliance was not intended to inhibit downward palatal movement and remodeling.

If growth of the vertical dimension proceeds normally while the transverse dimension of growth is slowed, a deepening of the palatal vault could result.

The cephalometric analysis was designed to assess compensatory changes that may have occurred in the anteroposterior and transverse dimensions of the maxillary complex. Analysis of the anteroposterior compensatory changes during restraint in the experimental group shows that a greater amount of anteroposterior snout development occurred in comparison to the control group. This suggests that growth may have compensated for the inhibition in the transverse dimension by increasing growth and development in the anteroposterior dimension.

The results of the cephalometric assessment of compensatory changes in the transverse dimension, with one exception, all show a similar trend.

The zygomatic arch, cranial base, and bony maxillary widths of the experimental group were all affected similarly in that the rates of increase in these areas were reduced to a more linear relationship (Figures 7-9). The control group exhibited a marked rate of increase in these same areas during Phase I of the experiment, and a decrease in the rate of increase during Phase II. These findings demonstrate that normal increases in the transverse dimension of the maxillary complex were slowed by the restraining appliance. The one exception was in the width of the posterior skull area (Figure 10).

Measures assessing the transverse width of the posterior portion of the calvaria suggest that this area was not significantly affected by the restraining appliance. Any slight differences noted between the groups can probably be explained by normal variance in growth between different litters, as implied by the similar patterns in growth changes seen between the groups (Figure 10).

Histological evaluation of undecalcified sections was done in an effort to assess the amount of bone deposition between vital dye indicators. Due to the limited number of sections taken from each

group, only broad conclusions can be made concerning the histological response of the maxillary complex during and after its restraint. No histologic evidence was found to suggest compensatory changes in the form of increased or decreased midpalatal sutural activity in the experimental group as compared to the control group. However, the trend for less bone deposition between vital dye indicators in the experimental group was consistently noted. This finding correlates with the observations derived from dental cast and cephalometric analyses.

It is therefore suggested that differences seen between the control and experimental groups with respect to the rate of development of the maxillary complex in its transverse dimension during the phase of unrestricted growth is due to a reassertion of inhibited growth potential and a regression toward normal structural and spatial relationships (rebound).

SUMMARY AND CONCLUSIONS

Compensatory responses to experimentally induced alterations of craniofacial structures by the application of active or passive force systems has received little attention in the literature. Results of previous studies indicate that post-restraint compensatory responses are varied.

The present study was performed to examine the nature of compensatory responses during and after passive, transverse restraint of the maxillary complex of rabbits. The sample consisted of 12 rapidly growing New Zealand White rabbits (30 days of age), with 5 rabbits serving as controls. The experimental group was subjected to 15 days of passive restraint of the maxillary complex utilizing custom-fabricated acrylic appliances. The second phase of the experiment involved removal of the restraining appliance which allowed for a 30 day period of unrestricted growth.

Data were collected from both groups and analyzed in three categories:

1. Dental cast measurements which assessed structural compensatory responses of the maxillary complex;

2. Cephalometric measurements which assessed the compensatory responses of surrounding structures in the anteroposterior and transverse dimensions; and,

3. Histological observation to evaluate the relative amount of post-restraint bone deposition between groups.

Dental cast analysis showed compensatory responses expressed as an increase in the depth of the palatal vault during restraint with a corresponding decrease in the experimentally induced palatal vault depth, and regression toward normality during the post-restraint period.

The cephalometric analysis showed that surrounding structures such as the zygomatic and maxillary bones, as well as the cranial base, all responded similarly during and after restraint. Anteroposterior snout development, however, differed in its compensatory response to transverse restraint of the maxillary complex. The posterior portion of the calvarium appeared to be unaffected by restraint of the maxillary complex.

Histological examination showed a relatively greater amount of bone deposition in the experimental group subsequent to removal of the restraining appliance as compared to the same period of unrestricted growth in the control group.

The results of the study may be summarized:

1. Passive transverse restraint of the maxillary complex can alter its structural and transpalatal dimensions and inhibit its growth potential.

2. Compensatory changes seen during restraint include an increase in palatal depth and anteroposterior snout development and a decrease in the rate of transverse growth of surrounding craniofacial structures.

3. Compensatory changes seen in the post-restraint period include a decrease in the experimentally induced palatal vault depth and a reassertion of inhibited growth potential in surrounding craniofacial structures with a regression toward normality; i.e., rebound.

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