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THE EFFECT OF UNILATERAL PROGRESSIVE CONDYLAR COMPRESSION ON MANDIBULAR GROWTH IN THE GUINEA PIG

by

JOSEPH HUBBARD BELHOBEK

Submitted in partial fulfillment of the requirements

for the Degree of Master of Science

Department of Orthodontics

SCHOOL OF DENTISTRY

CASE WESTERN RESERVE UNIVERSITY

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CASE WESTERN RESERVE UNIVERSITY

GRADUATE STUDIES

We hereby approve the thesis of

JOSEPH HUBBARD BELHOBEK

candidate for the Master of Science

degree.

Signed: _____ (Chairman)

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THE EFFECT OF UNILATERAL PROGRESSIVE CONDYLAR COMPRESSION ON MANDIBULAR GROWTH IN THE GUINEA PIG

Abstract

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Mandibular prognathism is perhaps one of the most difficult skeletal imbalances which confronts the clinical orthodontist. Non-surgical correction of this condition has been attempted clinically in many ways including chin-caps, class III intermaxillary elastics, and distally directed inclined planes. A common factor in all these methods has been directing distal traction against the growing mandible. However, basic information concerning the effect of distal traction on mandibular growth is generally unavailable.

The present investigation was designed to obtain basic, quantitative information on the effect of transcondylar pressure on mandibular growth in twenty-two growing guinea pigs. On one side of the animals, a spring-loaded appliance was placed surgically between the zygomatic arch and the body of the mandible, while a free-sliding appliance was placed similarly on the other side of the animal. To assess mandibular growth by measurement of appliance elongation, cephalometric radiographs were obtained weekly for eight weeks. With this design, the free-sliding appliances served as a control to measure normal mandibular growth, while the spring-loaded appliances

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DEDICATED

To my Parents

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INTRODUCTION

Craniofacial growth and facial-skeletal balance are of great interest to the orthodontist. Malocclusions associated with skeletal imbalances between the maxilla and mandible prove to be very difficult types of orthodontic cases to treat. Perhaps the most difficult imbalances have been, and continue to be, those of true mandibular prognathism. Treatment of mandibular prognathism by surgical resection is an effective form of therapy, its chief disadvantage being that treatment cannot be performed until skeletal growth has ceased. Thus, the individual must live with a progressively worsening deformity for a considerable length of time. In addition, the procedure involves surgical trauma and the possibility of complications.

A more ideal approach to the problem lies in the possibility of controlling mandibular growth. All such attempts, to date, have essentially involved the application of posterior pressure against the mandibular condyle in order to retard or stop mandibular growth. While the clinical methods for producing this pressure have varied through the years, this concept of distal traction against the mandible remains basic, and to a considerable degree independent of the prevailing thought concerning the known mechanisms of craniofacial growth. Unfortunately, the efficacy of these clinical approaches has never been adequately demonstrated; indeed, perhaps it never can, since clinical evaluation of treatment is plagued by many uncontrolled variables,

chief among which is the impossibility of observing both treated and untreated effects in the same clinical patient. This lack of control in the clinical situation makes statistical evaluation of therapy unreliable, leaving treatment evaluation to the subjective judgement of the clinician.

Animal studies have the potential to overcome these problems. They offer better control of forces and the opportunity of having the animal serve, to a degree, as his own control. While one condyle is never completely independent of the other, the possibility of applying unilateral forces affords a considerable advantage over clinical trials. It will be the purpose of the present investigation to examine the effect of unilateral progressive condylar compression on mandibular growth in the guinea pig.

REVIEW OF THE LITERATURE

Control of Skeletal Growth

Long Bone Growth

Interest in bone, both as a tissue and an organ, is not limited to the orthodontic profession; the basic properties of osseous tissue have long been investigated. According to Gelbke (1951), Leuter and Volkmann, as early as 1862, advanced the theory that compression inhibits bone growth and produces atrophy, while tension stimulates osseous growth and formation. In 1892, Wolff postulated that bony tissue responds to altered function through both internal and external structural changes. He observed that increased function leads to bone formation, and that lack of use leads to atrophy. Various investigators have subsequently verified these theories. For example, Glücksmann (1938, 1942) used endosteal cultures derived from tibiae of embryonic and newly hatched chicks to study the effect of mechanical stress on skeletal tissue. He observed that orientation of spicules of bone cultivated in vitro responded directly to mechanical stress.

The way in which bones grow has been studied in great depth. According to Scott and Pease (1956), Hales observed in 1727 that holes drilled in the shaft of growing bones always remained the same distance apart. From this observation he concluded that growth of long bones occurred at their ends. Using fixed markers, duHamel in 1745, and Hunter in 1772, established the epiphyseal plate as the precise

center of growth.

Throughout this paper, the term "growth center" will denote, "a site of endochondral ossification with tissue separating force, contributing to the increase in skeletal mass," while the term "growth site" will denote, "regions of periosteal or sutural bone formation and modeling resorption adaptive to environmental influences." Both of these definitions were advanced by Baume (1961).

Lacroix (1951) removed the distal cartilage mass of the tibia in neo-natal rabbits and isologously transplanted them under the renal capsule. Cartilage elongation proceded along normal lines, and the implant was morphologically identical to a normal epiphyseal plate, indicating the ability of the epiphyseal cartilage of bones to pursue organogenesis out of normal organ context. In a similar study Koski and Rönning (1966) subcutaneously and intracerebrally transplanted the distal cartilaginous ends of rat radii between littermates. Their findings, like those of Lacroix showed that the transplants developed a well differentiated epiphyseal growth plate, an ossifying epiphysis, and a diaphysis of varying length.

Using transplanted embryonic rat femurs, Willis (1936) and Felts (1957a, b; 1959) established that whole immature long bones will continue normal growth and development when transplanted to a non-functional environment. Their investigations clearly revealed the inherent growth capacity of long bones. Furthermore, Felts (1957a, b; 1959) concluded that while the general size and shape of long bones exhibit a lack of dependency on the mechanical environment, the local

environment is responsible for small functional characteristics, such as tubercles and grooves.

Although such evidence strongly supports the concept that long bones grow in length at their ends and that the growth mechanism is the epiphyseal plate, Moss, Low, and Meehan (1968) do not agree. In an abstract they suggested that:

> the elongation of the epiphyseal growth plates is neither morphogenetically primary nor intrinsically determined. . . . The differential elongation of each long bone is related to the net compressive force exerted by related muscles at each articulator end; so that the greatest growth occurs at the end with the least net compression.

Experimental support was derived by isologously implanting rat proximal tibial growth plates into frontonasal, coronal, or metopic sutural areas. The observed growth-plate elongation was proportional to the amount of passive sutural expansion which normally occurs in these sites. They concluded that the growth of the epiphyseal plates in these sites was primarily determined by the expansion of the sutures, rather than an inherent growth potential of epiphyseal plates. To date, Moss et al. have not explained the theory more fully. However, the studies of Willis (1936), Felts (1957a, b; 1959), and Koski and Rönning (1966) clearly demonstrated that the epiphyseal plates of long bones grow well in passive transplantation sites.

Long Bone Inhibition

Based on these properties of epiphyseal plates in bony organogenesis, various means of controlling long bone growth have been developed. According to White (1935), Rizzoli in 1847, Taylor in 1916,

Brook in 1927, and numerous others equalized limb-length by surgical resection of appropriate segments of long bones. Phemister (1932) advocated correction of excessive long bone length by removing a portion of the epiphyseal plate and fusing the epiphysis to the diaphysis by bone grafts to inhibit further bone growth. Although these methods were effective in equalizing limb-length, problems in determining when to interfere surgically and the irreversible nature of these procedures led to further investigation for a better means of inhibiting bone growth. Haas (1945) bound the femoral epiphyseal plate in dogs by securing a wire loop through the epiphysis and diaphysis of the femur, and observed subsequent inhibition of femoral elongation. In similar animal studies, Haas (1948) and, later, Siffert (1956) found that wire staples placed across epiphyseal plates of long bones retarded their longitudinal growth. Siffert, in placing a staple below, but not across, the epiphyseal plate, found no bone growth inhibition. Blount and Clarke (1949) used this staple technique for the correction of limb-length inequality and angular deformities at the epiphyses in children. Haas (1950) placed pins diagonally across the epiphyseal plate of dogs and observed that this procedure also effectively inhibited long bone growth.

An interesting investigation by Monson and Felts (1961) examined the response of transplanted, immature long bones to longitudinal compressive force. The humeri of neo-natal mice were measured accurately and implanted subcutaneously either with or without a compression appliance in adults of the same strain. The results showed

that cessation of growth occurred earlier in the compressed implants than in the free implants. The force needed to stop the long bone growth, approximately 13 grams, was well beyond those encountered in the normal functional load of the limb.

The studies of Haas (1948, 1950), Blount and Clarke (1949), Gelbke (1951), Monson and Felts (1961), and Sijbrandij (1963) concluded that the ultimate effect of binding the epiphyseal plate was closure. Histologic examination of a closed femoral epiphyseal plate in the rabbit (Siffert, 1956) showed failure of the resting cells of the epiphyseal cartilage to form new cells. All the existing cells of the plate, however, continued to mature, hypertrophy, and be replaced by osseous tissue. This process progressively reduced the thickness of the epiphyseal plate until closure occurred. Upon removal of the mechanical block, Haas (1948, 1950), Blount and Clarke (1949), and Gelbke (1951), observed at least partial resumption of bone growth as long as skeletal maturation had not yet taken place. On the other hand, Siffert (1956) observed that bone growth inhibition was not reversible if the epiphyseal plates were bound long enough for complete closure of the epiphyseal plate to occur.

The work of Strobino, French, and Colonna (1952) analyzed the forces necessary to produce epiphyseal plate closure in long bones. Using calves, with springs wired across the tibial epiphyseal cartilage, they found that tensions up to 400 pounds produced no demonstrable effects on bone growth when compared to that of the contralateral side under normal physiologic tensions. The rate of long bone growth was

in no way altered when the epiphyseal plate was released from a prolonged force of 280 pounds. In a similar study using rabbits, Sijbrandij (1963) revealed that tibial growth inhibition did occur at load levels of three kilograms and that growth retardation occurred at somewhat lower compressive forces. However, these inhibition and retardation levels were substantially higher than the normal physiologic load required of this structure. Thus, the evidence provided by these studies indicates that growing epiphyseal plates produce great separating force and that relatively heavy compressive loads are required to inhibit cartilaginous growth centers.

Control of Mandibular Growth

Mandibular Growth

While there is a great deal known about long bone growth and inhibition, there is sufficient evidence to call into question the direct transfer of these data to mandibular growth. Embryologically, long bones and the mandible develop quite differently. Long bones develop from cartilaginous models by endochondral ossification, the epiphyseal plates persisting as cartilaginous growth centers until skeletal maturation occurs. In contrast, the mandible develops as a thin plate of intramembranous bone lateral to, and at some distance from, Meckel's cartilage, and the condyles develop secondarily in cartilage. These condylar cartilages grow rapidly to unite with the mandibular body and form large cone-shaped masses occupying the condylar process. This cartilage is then replaced by bone until only a

small portion remains just beneath the fibrous perichondrium of the condyle. During growth, three distinct cell zones are visible in the condylar cartilage of the mandible: 1) the fibrous articular zone, 2) proliferative cartilaginous zone, and 3) hypertrophic cartilaginous zone (Blackwood, 1966). Thus, while long bones develop endochondrally, the mandible develops both endochondrally and intramembraneously.

Classical Theory of Mandibular Growth

The mechanism by which the mandible grows is not as clearly established as that of long bone growth. The classical theory attributes mandibular translation to the growth that takes place at the condylar cartilage located at the top of the condyle. This theory was proposed by Charles (1925) and was based on his microscopic examination of human, fetal mandibles. This concept is supported by many investigators, including Brodie (1941), Rushton (1944), Sicher (1947), Symons (1951), and Scott (1967). This theory considers the condylar cartilage to be the primary growth center of the mandible and assumes as a major premise that it has an independent growth potential.

Many investigators have challenged the basic tenents of this classical theory of mandibular growth. Silbermann and Frommer (1972) examined histologically and autoradiographically the condylar cartilages of mice and observed that these cells lacked the specific arrangement and cellular homogeneity characteristic of more differentiated endochondral growth sites. They concluded that mandibular

condylar cartilage maintains the morphologic and metabolic characteristics of an embryonic type of tissue. Durkin (1972), studying rat and guinea pig condyles, came to similar conclusions.

The intrinsic growth potential of the condylar cartilage described in the classical theory of mandibular growth has been studied by many investigators using transplantation studies. As early as 1937, Baker examined the effects of isologous transplantation of rat mandibles to non-functional sites such as the eye, leg muscle, and brain. His investigation revealed that while intrinsic factors determine the shape of the mandible, function tends to control the overall size of the mandible. According to Felts (1961), a variety of people (Ostergren, 1958; Truax, 1958; Truax and Felts, 1961; Etem, 1959) subcutaneously implanted halves of fetal mandibles in laboratory animals and revealed that normal morphogenesis and size were never obtained. Normal chondrogenesis failed to occur, resulting in a loss of condylar cartilage. Koski and associates (1963, 1964, 1965, 1966), examining the growth of isologous condylar transplants to brain and subcutaneous tissue, reported that some transplants increased in size seemingly in a direction corresponding to the vertical dimension of the ramus. Histologic examination, however, revealed that, while the transplants partially resembled the ramus in situ the structure of the condylar cartilage was not similar to that of condylar cartilage in situ. In comparing transplanted condylar cartilage to transplanted epiphyseal cartilage, Koski concluded that the condylar cartilage deviated markedly from the typical growth cartilage

of the epiphyseal plate.

Duterloo's autologous implantation study of condylar cartilage utilizing tritiated thymidine cellular labelling (1967) produced somewhat different results. His investigation established that proliferation and differentiation of chondroblasts occurred in the transplants and, that while condylar chondrogenesis is not identical to epiphyseal chondrogenesis, the rat mandibular condylar growth is largely responsible for the increase in length of the ramus. However, he attributed the extent of longitudinal growth and the change in the shape of the condyle to be dependent on extrinsic factors. Meikle (1973) also found that condylar, cartilage cells retained their capacity to proliferate when transplanted into non-functional sites. Moreover, he reported that the condylar cells were multipotential forming either bone or cartilage depending upon environmental circumstances and suggested that the extrinsic mechanical stresses associated with functional activity of the mandibular joint provides the stimulus for the normal differentiation of the cells into chondroblasts. Thus, the present knowledge of the growth potential of mandibular condylar cartilage is incomplete.

Functional Matrix Theory

According to Moss (1964), van der Klaauw in the late 1940's suggested a functional analysis of craniofacial growth. During the past decade, this synthesis has been examined and refined in numerous reports by Moss and associates (1960, 1964, 1968, 1969). Moss

attributes the origin, growth, and maintenance of the skeleton to the functional components which he defined as those tissues, organs, and functioning spaces related to any given skeletal element. His so-called "functional matrix" theory explains craniofacial growth on the basis of two types of matrices: periosteal and capsular. Periosteal matrices (muscles, teeth, glands, etc.) act to alter the size and shape of the skeleton by osseous deposition and resorption. Capsular matrices (brain, orbit, spaces, etc.), upon their expansion, are responsible for translational growth.

In describing mandibular growth, Moss (1960) credits the functioning oral and pharyngeal cavities as the capsular matrix responsible for mandibular translation. He states that expansion of this space is the primary event which causes the relocation of the mandible in space, and that condylar growth is merely an adjustive mechanism to maintain temporomandibular articulation. Thus, the functional matrix theory considers the condylar cartilage as a growth site rather than a growth center.

Mandibular periosteal matrices consist of: 1) mandibular muscle attachments, 2) neurovascular triads (arteries, veins, nerves), 3) associated salivary glands, 4) the teeth, 4) skin, fat and connective tissue, and 6) the tongue. These matrices are responsible for local, osseous remodelling of the mandible.

The main support for this functional theory of growth is derived from studies of growth of animals without condyles. The work of Jolly (1961), Gianelly and Moorrees (1965) and Pimenidis and

Gianelly (1972) examined the effects of condylectomies on mandibular growth in rats. Bilateral condylectomy of the rats resulted in relatively minor changes in post-natal length and spatial positioning of the rat mandible. In a human case report of a bilateral condylectomy performed on a seven year old girl for treatment of condylar ankylosis, Moss and Rankow (1968) reported continued mandibular growth after the condylectomies. This mandibular growth included: 1) mandibular translation, 2) increased body length, 3) horizontal movement, 4) increase in total projective width, and 5) continued normal dental maturation.

Sarnat and Engle (1951) performed unilateral condylectomies on Macaca rhesus monkeys and observed a marked asymmetry of the mandible and the middle third of the face. Performing condylectomies on dogs, Peskin and Laskin (1965) concluded from gross and histologic observations that removal of the entire condyle markedly retards the growth of the dog mandible and causes some changes in mandibular shape.

The central question in interpreting the evidence from these various studies is to determine if the mandibular growth seen in animals without condyles is the same as the mandibular growth in animals with condyles. Das, Meyer, and Sicher (1965) studied the effects of condylectomies in the rat and, like other investigators (Jolly, 1961; Gianelly and Moorrees, 1965; Pimenidis and Gianelly, 1972), observed that mandibular growth did occur. Nevertheless, they observed that the growth in the experimental animals was different from that seen

in the control animals. They concluded that normal "developmental" growth does not occur in the absence of the condylar growth center, but that adjustive growth does occur.

Inhibition of Mandibular Growth

With such an incomplete understanding of the mechanisms of mandibular growth, it is, perhaps, not surprising that no single method of inhibiting its growth has emerged. Surgical resection of the mandible is, of course, a solution, but this is of little help in treating the young growing patient.

Chin Cup Therapy

Early attempts at inhibiting mandibular growth involved the application of upward and backward pressure against the mandible by the use of the so-called "chin-cup" or "chin-cap." Basically, this appliance consists of a sling worn under the chin connected by either cloth or elastic straps to a cap worn over the back of the head. According to Weinberger (1926), Kneisel, as early as 1836, reported using the chin-cap to retard mandibular growth. While this appliance has enjoyed extensive clinical use, clinical assessments of its results are variable and in the past largely unpublished.

In a clinical study of 100 patients wearing chin-cups delivering 500 grams of force per side, Armstrong (1961) concluded that clinical success may result from a combination of limited mandibular inhibition and normal maxillary growth. There did not, however, appear to be actual retardation of the mandible.

In a study of 60 patients, Thilander (1963, 1965) evaluated the effects of chin-cups adjusted to 150-200 grams per side. Utilizing study casts and cephalometric radiographs, the study revealed that treatment produced lingual tipping of the mandibular incisors, labial tipping of the maxillary incisors, and some backward displacement of the mandible. However, not all cases responded well to chin-cup therapy. Success appeared to be dependent on: 1) the age treatment was started, 2) the presence of a deep overbite, and 3) the possibility of dental changes occurring.

Graber (1969) has reported a disturbance in the normal downward and forward development of the mandible as a result of chin-cups adjusted to deliver three to five kilograms of force per side. This disturbance, as judged cephalometrically, seemed to produce more vertical growth in a previously horizontally growing mandible.

In contrast, Janzen and Bluher (1965) applied 150 grams of distal traction per side in Macaca mullata and reported no alteration of the antero-posterior position of the mandible in relation to the cranial base. They did, however, observe a decrease in development of ramal height. Their histologic examination revealed resorption of the glenoid fossa and the posterior surfaces of the condyles, although no gross anatomic alteration was discerned. Interestingly enough, Ogston as early as 1878, postulated that the temporomandibular joint was capable of remodelling. Histologic analysis of the temporomandibular joint by Moffet, et al. (1964) and Blackwood (1966) have documented the remodelling ability of the joint.

Petrovic (1972) examined the effect of chin-cups applied six hours per day in twenty-eight rats. His study revealed that such therapy decreased the height of the prechondroblastic and chondroblastic zones of condylar cartilage, thereby decreasing the growth in the mandibular basal arch.

All these studies on the effects of chin-cups have suffered from a basic lack of methodological conformity including: 1) magnitude of forces used in distal traction, 2) time distal traction applied, 3) methods of quantifying results, and 4) type of animals used in the studies. Nonetheless, a survey by Armstrong (1961) showed wide clinical use of chin-cup therapy to treat mandibular prognathism.

Class III Intermaxillary Traction

At the turn of the 19th century, class III intermaxillary elastics superceeded the routine use of the chin-cup for treating mandibular prognathism. Essentially, class III intermaxillary force consists of traction, usually derived from elastics, directed from the posterior aspect of the maxillary dental arch to the anterior aspect of the mandibular dental arch. This approach was introduced by Case in 1893 and, soon, both Case (1904) and Angle (1907) abandoned attempts to reduce mandibular prognathism by extra-oral pressure on the chin via chin-cups. The use of class III intermaxillary force has, ever since, been a routine clinical procedure for treating mandibular prognathism

Since intermaxillary traction uses the dental arches for

anchorage, the question arises as to what degree of change is attributable to dental movement and what is attributable to skeletal alteration. In monkey studies utilizing class III intermaxillary elastics, Breitner (1940), Meikle (1970), and Adams, Meikle, Norwick, and Turpin (1972) observed a class II relationship of the dentures which they attributed largely to dental movement. This finding led Breitner (1940) to conclude that an external means of applying posterior traction, such as a chin-cup, may provide a more efficient means of producing skeletal changes than intermaxillary elastics. Breitner (1940), however, did report that in addition to the dental movement, there were skeletal changes occurring under class III intermaxillary traction. Histologically, the post-glenoid tubercle showed bony resorption on the side toward the condyle and deposition of new bone arranged in horizontal layers on the side away from the condyle. Bony deposition also occurred on the anterior aspect of the glenoid fossa and the anterior surface of the condyle. This remodelling was so extensive that Breitner concluded that the glenoid fossa was in a state of distal migration. In a similar study, Adams, et al. (1972) not only observed remodelling of the glenoid fossa but also observed regressive remodelling on the posterior surface of the condyle. However, Meikle (1970) reported that while class III intermaxillary traction produced histologic changes in the temporomandibular joint of Macaca mulatta, these changes were confined to the microscopic level and were of no clinical significance.

Cephalometric analysis has also shown that class III

intermaxillary elastics produce both dental as well as skeletal changes. Analyzing lateral headplates of monkeys under class III elastics, Adams, et al. (1972) reported counterclockwise forward and upward rotation of the maxilla. In addition, considerable alteration was observed in the alveolar process of the maxilla. In a similar manner, they observed a reciprocal downward and backward rotation of the mandible with extensive alveolar remodelling producing an upward and slightly backward rotation of the dentition. Thus, cephalometric evaluation of the effects of class III intermaxillary force tends to substantiate the clinical observation that class III intermaxillary forces produce dental as well as skeletal changes.

Inclined Planes

Still another method of applying distal traction against the growing mandible is through the use of inclined planes. According to Weinberger (1926), Fletcher in 1860 first reported the use of a type of inclined plane appliance to force the mandible in a posterior direction in order to correct mandibular prognathism. Essentially the modern appliance consists of an inclined plane oriented in such a manner as to force the mandible distally upon closure of the jaws (Neumann, 1969).

Since this appliance depends upon the teeth to generate posterior force against the mandible, once again the question arises as to the extent this force is transmitted to the mandible per se. Ramfjord and Hiniker (1966) examined the histologic and cephalometric effects of distally directed splints cemented to the dental arches of

four monkeys. They reported that, while the splints produced great trauma to the periodontium and adaptive repositioning of the teeth, the temporomandibular joint of the adult Macaca mullata was very resistant to dysfunctional changes produced by the bite planes. They observed no adaptive changes of the temporomandibular joint either on the inferior surface of the glenoid fossa or the superior surface of the condyle. It was their opinion that the temporomandibular joint in adult monkeys is well protected against distal traction by the cartilage present in the joint.

In an investigation utilizing rats with inclined planes cemented to the mandibular incisors, Folke and Stallard (1972) obtained different results. They found that distal displacement of the mandible via inclined planes produced both cellular and morphological changes in the condylar cartilage and glenoid fossa of the adult rat. Using histologic and radioautographic methods they observed that, while cellular activity (DNA synthesis) in the proliferative zone of the condyle decreased for the first week, the second through fourth weeks revealed substantial cellular increase over the controls. They attributed the initial decrease in cellular activity to pressure altering the blood supply of the joint and condyle, thereby reducing nutrition to the area and the subsequent decrease in cellular activity. According to Folke and Stallard, Trueta and Amata in 1960 and Trueta and Trias in 1961 observed this phenomenon in other joints. On the other hand, Folke and Stallard offered no explanation for the increase in cellular activity after the first week. Thus, if the ultimate

effect of distally directed inclined planes is only dental movement and possible stimulation of cellular activity of the condyle and glenoid fossa, their value in treating true mandibular prognathism may be questionable.

Conclusion

Clinical orthodontists, in their desire to help patients with mandibular prognathism, have tried many forms of therapy to inhibit mandibular growth. Often the basis for therapy rests not upon knowledge of craniofacial growth, but rather upon the presumed success of previous treatment. However, evaluation of treated cases is complicated by many factors. Clinical assessment of a treated case is largely subjective and, therefore, varies among different clinicians. Moreover, it is impossible to ever know the treatment and non-treatment results of the same case. Lack of standardization of such factors as: 1) appliance design, 2) magnitude and direction of force, and 3) patient cooperation, in clinical studies make treatment-evaluation even more difficult. In addition, statistical analysis of clinical treatment is of limited benefit due to the relatively small numbers of patients involved in clinical studies. For these reasons it is necessary to use animal investigations in order to obtain practical information concerning the effect of condylar compression on mandibular growth.

MATERIALS AND METHODS

The present investigation employed twenty-eight guinea pigs--eighteen males and ten females--of the CWR:(HA) strain obtained from the animal facility of Case Western Reserve University, School of Medicine, Cleveland, Ohio.

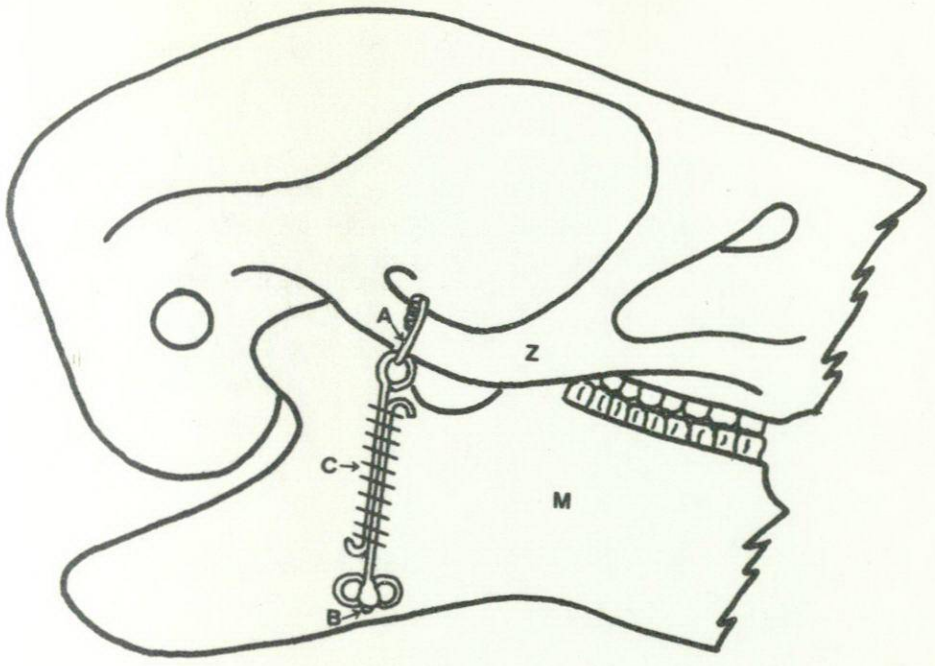
General Description

Progressive transcondylar traction in young growing guinea pigs provided a system for evaluating the effects of pressure on mandibular growth. On the experimental condyle, a spring-loaded appliance spanned the condyle between the zygomatic arch and the body of the mandible (Figure 1 and Plate 1). Initially, the spring was passive; however, as mandibular growth occurred, the appliance was compressed and produced progressively increasing pressure against the growing mandible.

The opposite side of the animal served as a control. A free sliding appliance was surgically positioned between the zygomatic arch and the body of the mandible (Figure 2 and Plate 2). The use of this free-sliding appliance provided a means of assessing uninhibited mandibular growth. To insure equal distribution of the unilateral forces employed in this study, the location of the control and experimental appliances was alternated from side to side in successive animals (Table I).

Figure 1. Placement diagram of spring-loaded appliance.
a. .010 stainless steel ligature wire.
b. insertion of mandibular clip in mandible.
c. spring-loaded appliance
m. mandible
z. zygomatic arch

Plate 1. Skull with spring-loaded appliance in place.


A
Z
C
M
B

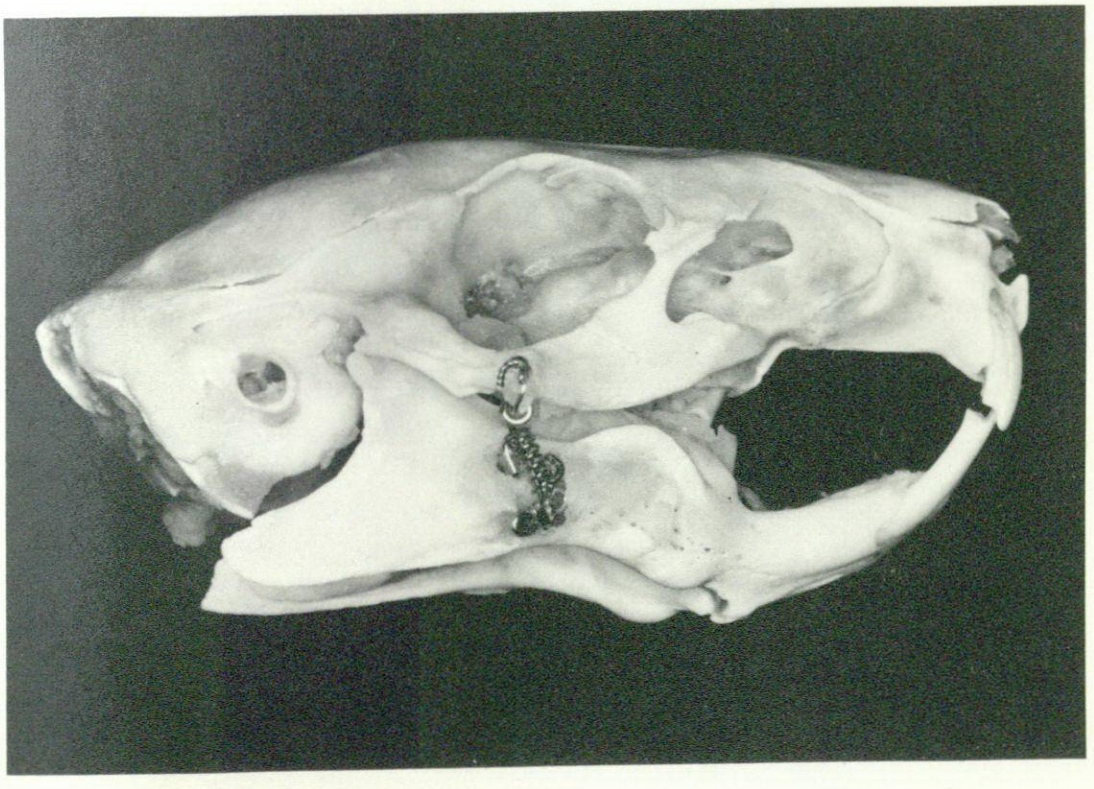
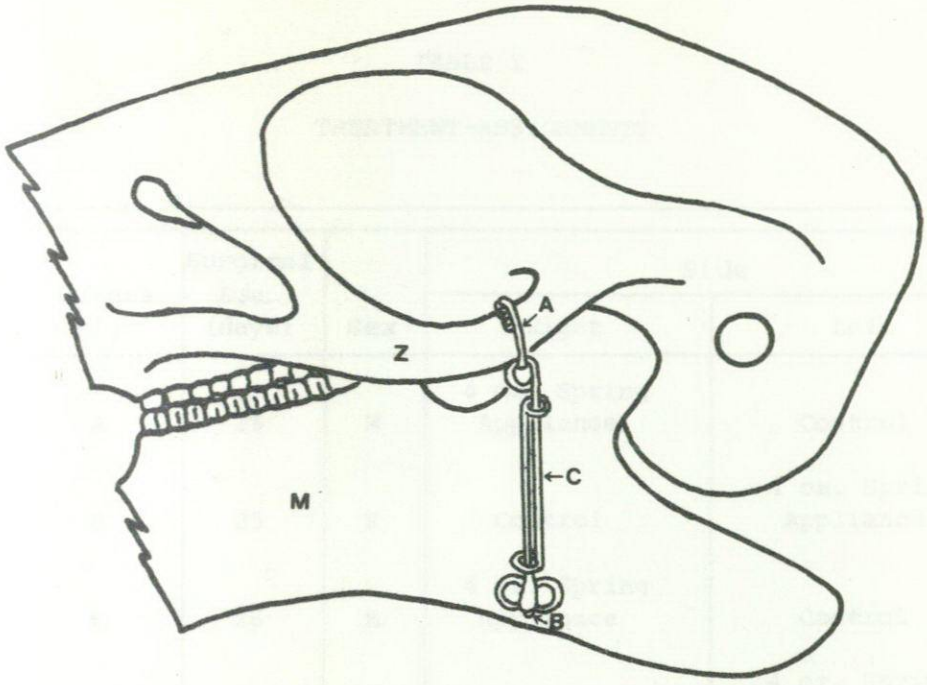


Figure 2. Placement diagram of free-sliding appliance.
a. .010 stainless steel ligature wire.
b. insertion of mandibular clip in mandible.
c. free-sliding appliance.
m. mandible.
z. zygomatic arch.

Plate 2. Skull with free-sliding appliance in place.


A
Z
C
M
B

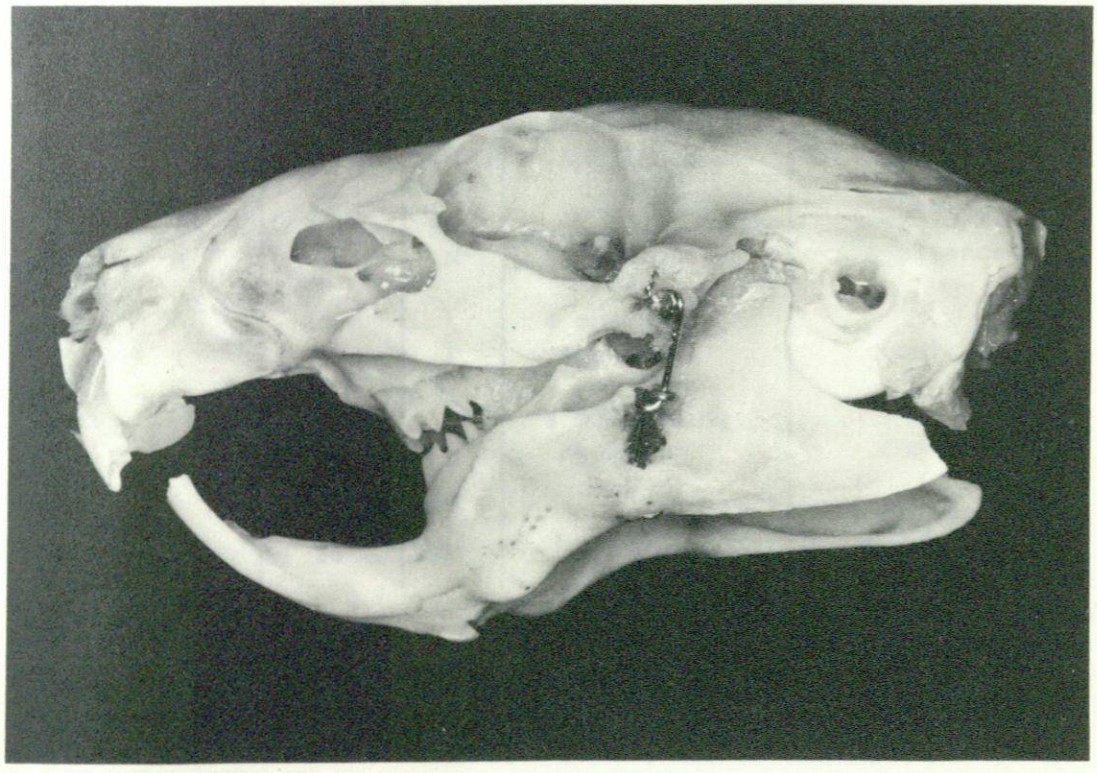


TABLE I

TREATMENT-ASSIGNMENTS

Guinea Pig	Surgical Age (days)	Sex	Side	
			Right	Left
A	25	M	4 oz. Spring Appliance	Control
B	25	F	Control	4 oz. Spring Appliance
C	26	M	4 oz. Spring Appliance	Control
D	26	F	Control	4 oz. Spring Appliance
E	28	M	Control	4 oz. Spring Appliance
F	27	M	Control	4 oz. Spring Appliance
G	27	M	4 oz. Spring Appliance	Control
H	25	M	Control	4 oz. Spring Appliance
I	27	F	4 oz. Spring Appliance	Control
J	28	M	4 oz. Spring Appliance	Control
K	25	F	Control	8 oz. Spring Appliance

TABLE I continued

Guinea Pig	Surgical Age (days)	Sex	Side	
			Right	Left
L	25	M	8 oz. Spring Appliance	Control
M	25	M	Control	8 oz. Spring Appliance
N	25	F	8 oz. Spring Appliance	Control
O	25	F	Control	8 oz. Spring Appliance
P	25	F	8 oz. Spring Appliance	Control
Q	25	M	8 oz. Spring Appliance	Control
R	25	M	Control	8 oz. Spring Appliance
S	27	F	8 oz. Spring Appliance	Control
T	26	M	8 oz. Spring Appliance	Control
U	26	M	Control	8 oz. Spring Appliance
V	26	M	8 oz. Spring Appliance	Control

In order to evaluate the effects of the appliances, cephalometric radiographs were taken at weekly intervals. Measurement of the distance between the ends of the appliances provided a means of quantifying growth of both the experimental and control sides. This comparision between the control and experimental appliances in the same animal provided a means of evaluating the effects of unilateral forces on mandibular growth.

After death, the animals were examined to determine the gross effects of the experimental forces. Such factors as incisal wear and mandibular deviation were noted. In addition, the tissue surrounding the implants was subjected to histologic analysis.

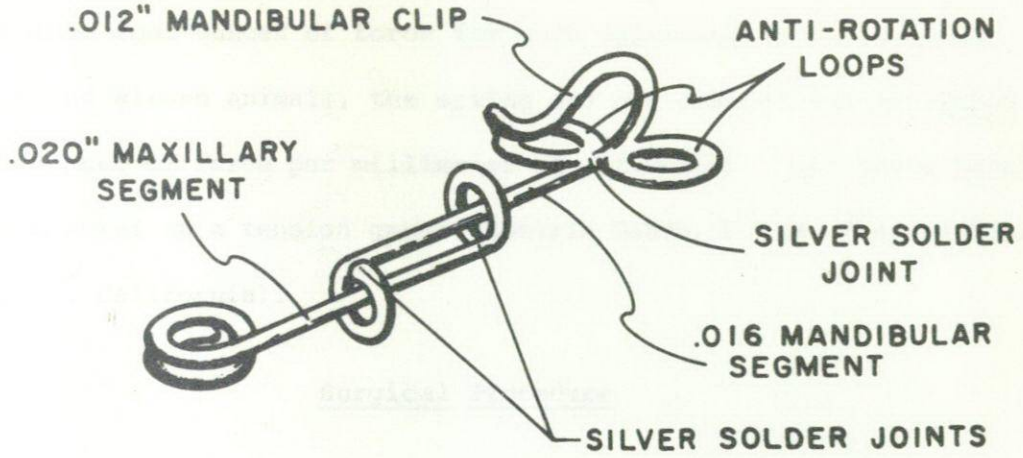
Appliance Design

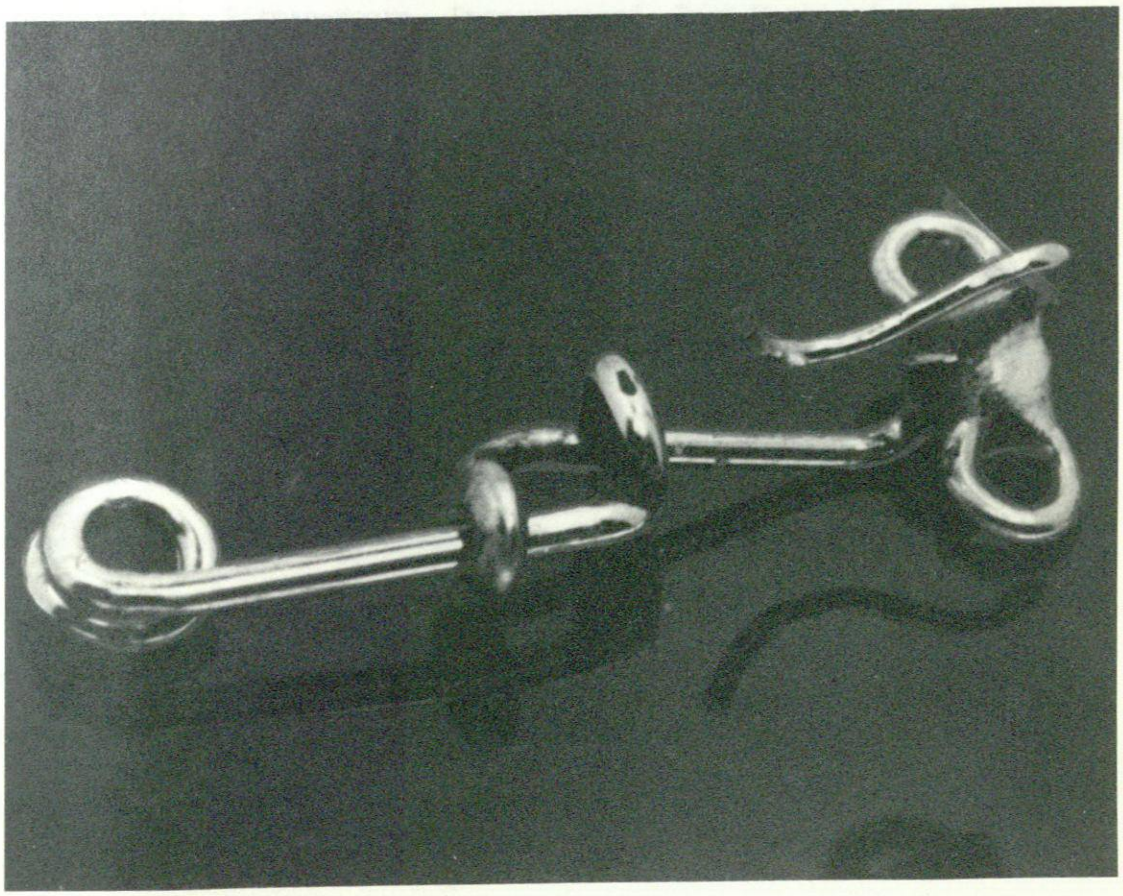
All appliances were constructed from stainless steel wire (Rocky Mountain/Orthodontics, Denver, Colorado). The maxillary portion of the appliances was constructed from .020 inch wire, while the mandibular portion was fashioned from .016 inch wire. The wire clip on the mandibular portion of the appliance was reduced to approximately .012 inches in diameter in an electrolytic polisher (Model #710, Rocky Mountain/Orthodontics, Denver, Colorado), in order to produce a more flexible clip for easier insertion of the appliance through the mandible. The free-sliding appliance was fabricated as shown in Figure 3 and Plate 3.

The spring-loaded appliance employed .010 x .036 inch stainless steel open coil spring (Unitek Corporation, Monrovia, California)

Figure 3. Construction of free-sliding appliance.

Plate 3. Completed free-sliding appliance.


.012" MANDIBULAR CLIP
ANTI-ROTATION LOOPS
.020" MAXILLARY SEGMENT
SILVER SOLDER JOINT
.016 MANDIBULAR SEGMENT
SILVER SOLDER JOINTS



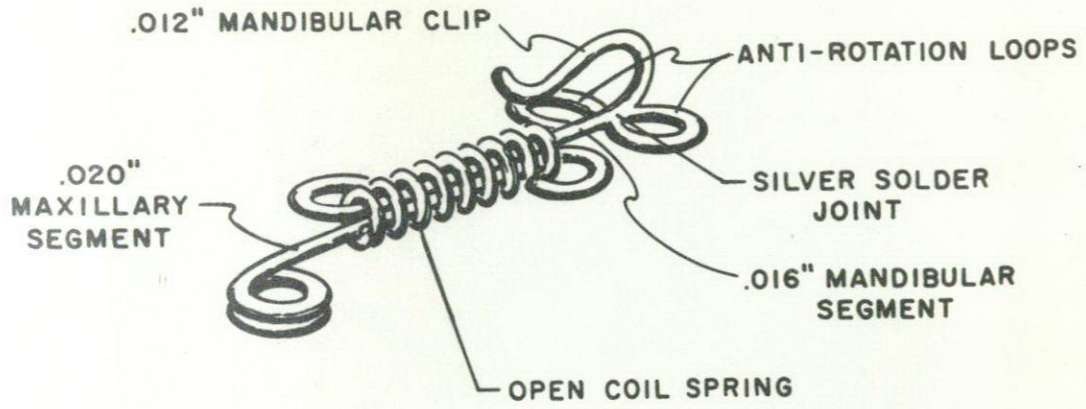
(Figure 4 and Plate 4). In the first eleven animals, the spring was reduced for two and one-half minutes in an electrolytic polisher so as to produce four ounces of force for each millimeter of activation. In the second eleven animals, the spring was not reduced and delivered eight ounces of force per millimeter of activation. All force levels were measured on a tension gauge (Dontrix Gauge, E.T.M. Corporation, Monrovia, California).

Surgical Procedure

All experimental animals underwent surgery at between twenty-five and thirty days of age. Surgical anesthesia was obtained by intramuscular injection of droperidol (13.2 mg/kg) and fentanyl (.26 mg/kg) (Innovar-Vet, Pitman-Moore, Incorporated, Fort Washington, Pennsylvania), followed ten minutes later by 20 mg/kg sodium pentobarbital (Veterinary Nembutal, Abbott Laboratories, North Chicago, Illinois) injected intraperitoneally as described by Fuller (1974). The surgical site was shaved and then swabbed with nitromersol (Metaphen Tincture, Abbott Laboratories, North Chicago, Illinois). A vertical incision was made from the zygomatic arch, just under the canthus of the eye, to the inferior border of the mandible. The skin was reflected and the masseter muscle exposed. Horizontal incisions were made in the masseter muscle at the inferior border of the zygomatic arch and midway between the zygomatic arch and the inferior border of the mandible. The muscle was then freed from the body of the mandible by blunt dissection (Plate 5). At a distance from the zygomatic arch

Figure 4. Construction of spring-loaded appliance.

Plate 4. Completed spring-loaded appliance.


.012" MANDIBULAR CLIP
ANTI-ROTATION LOOPS
.020" MAXILLARY SEGMENT
SILVER SOLDER JOINT
.016" MANDIBULAR SEGMENT
OPEN COIL SPRING

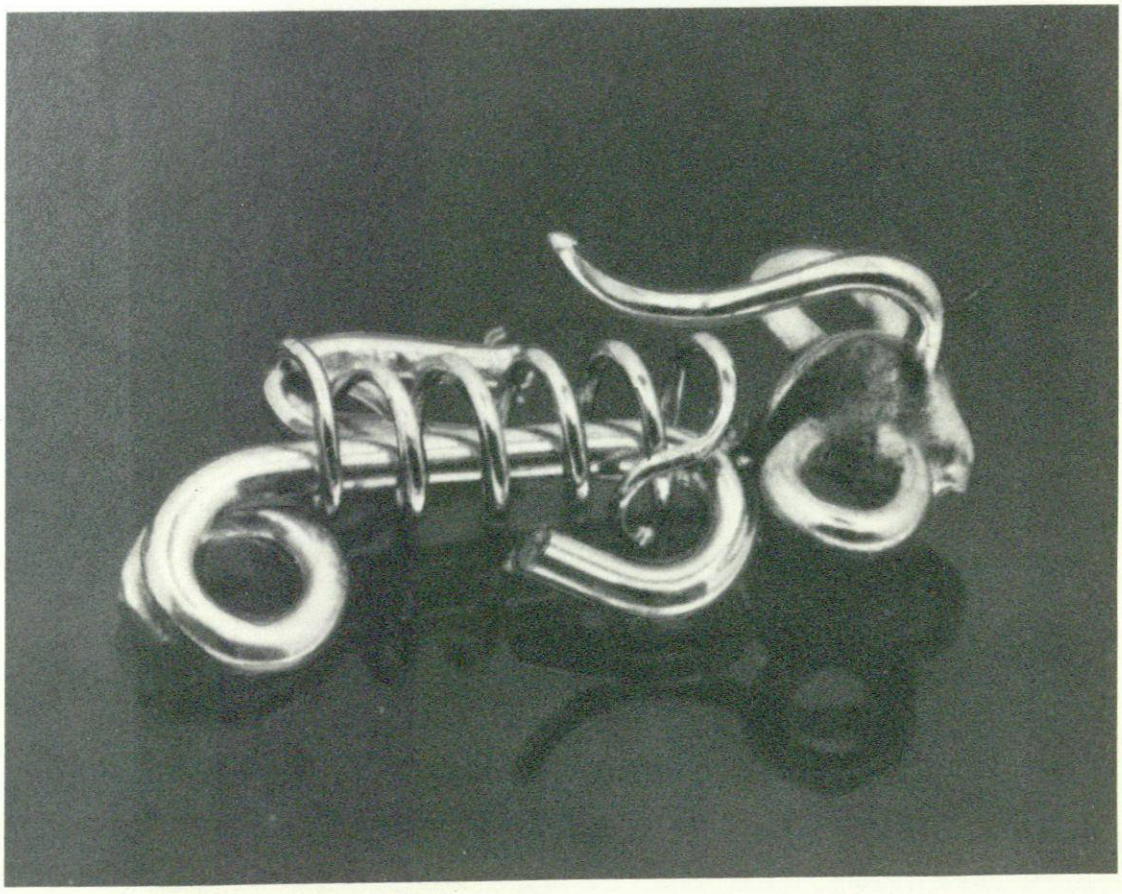
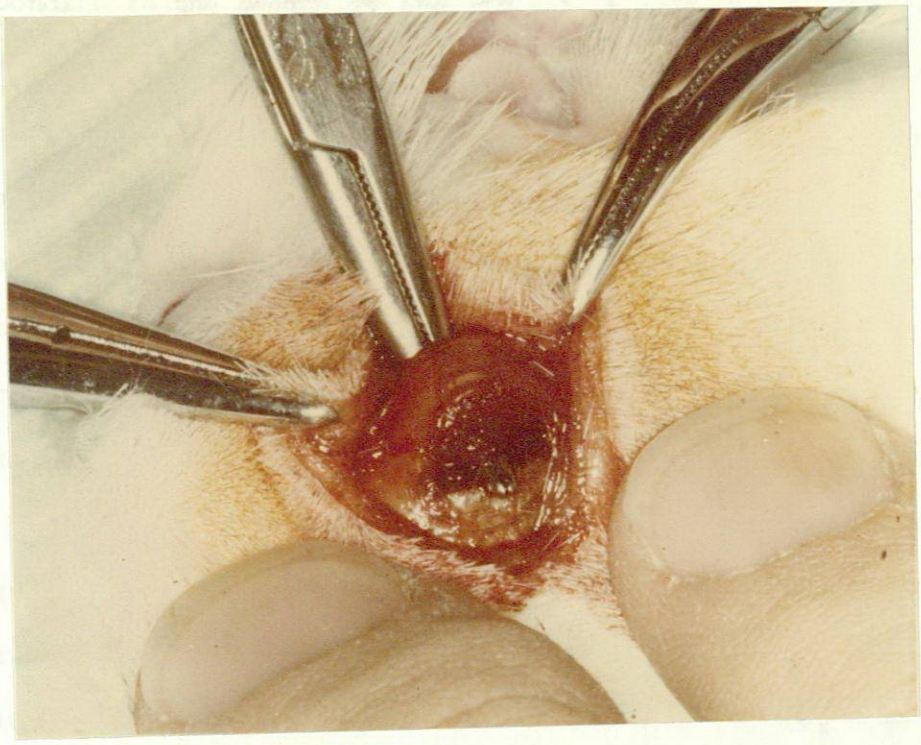
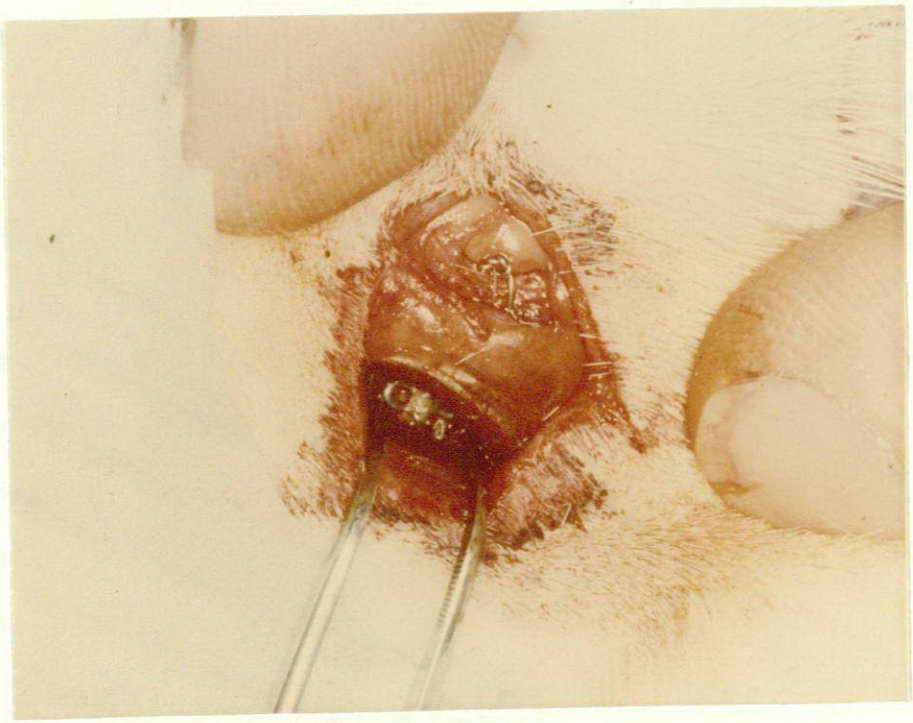


Plate 5. Reflection of masseter muscle from mandible.

Plate 6. Surgical placement of appliance





corresponding to the length of the implant, a hole was drilled through the body of the mandible using a #1/2 round dental bur (Ransom and Randolph Company, Toledo, Ohio). The implant was insinuated beneath the masseter muscle and the clip portion of the implant inserted through the hole in the mandible. The maxillary end of the implant was secured to the zygomatic arch by .010 inch soft stainless steel ligature wire (Malin Company, Cleveland, Ohio) (Plate 6). The jig was then checked for freedom of movement. Nitrofurazone (Furacin Soluble Powder Veterinary, Eaton Veterinary Laboratories, Norwich, New York) was sprinkled into the wound to control infection and the muscle and skin sutured with 4-0 surgical gut (Sherwood Medical Industries, Incorporated, St. Louis, Missouri). The closed incision was swabbed with Nitromersol. The same surgical procedure was used on both the experimental and control sides of the animals.

Post-surgically, the animals were bottle-fed a mixture of powdered guinea pig feed (Guinea Pig Chow, Ralston Purina Company, St. Louis, Missouri), dextrose (J.T. Baker Company, Chicago, Illinois), oatmeal (The Quaker Oat Company, Chicago, Illinois), and water until they regained their pre-surgical weight. To control any infection sodium sulfamethazine (Solumet, American Cyanamid Company, Princeton, New Jersey) was added to their drinking water as needed. After returning to their pre-surgical weight, the animal's diet consisted of Purina Guinea Pig Chow, cabbage, and water, ad libitum.

Data Collection

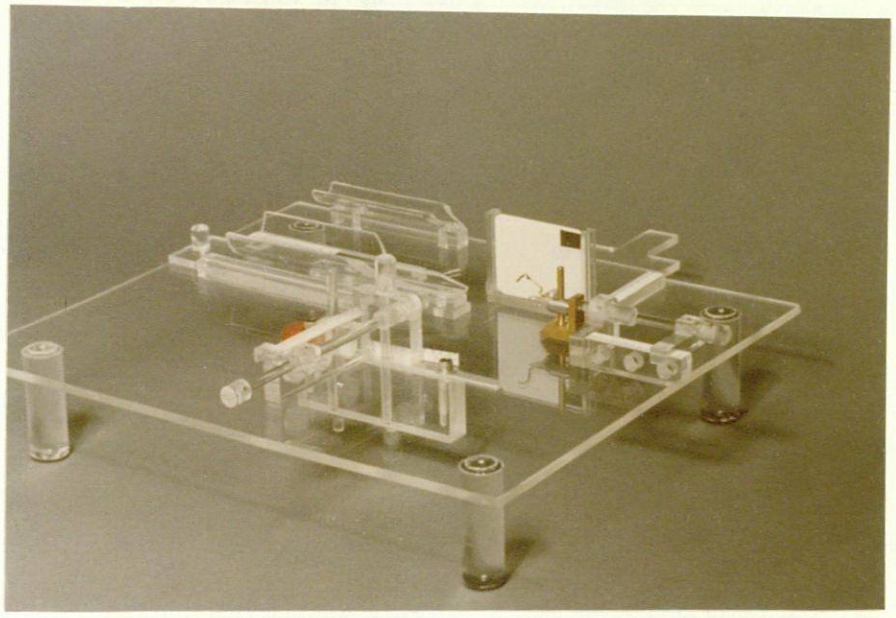
Cephalograms

Cephalometric radiographs were used to evaluate the growth of each side of the mandible. The animals were radiographed immediately post-surgically while still anesthetized and on a weekly basis thereafter for eight weeks. The procedure for radiographing the animals consisted of sedating the animals by intramuscular injection of droperidol (2 mg/kg) and fentanyl (.04 mg/kg) (Innovar-Vet). A rubber band was placed over the animal's snout in order to hold the jaws together and the animal positioned in a specially designed cephalostat, similar to the one used by Gange (1973)--See Plate 7. The cephalostat insured a relatively constant source-film distance (215 mm.) and an object-film distance (30 mm. from the sagittal plane) for each animal. Standard occlusal radiographic films (Kodak, DF-46; Eastman Kodak Company, Rochester, New York) were used (Plate 8). The radiographs were taken on a conventional dental radiographic unit (Ultron 90, Weber Dental Manufacturing Company, Canton, Ohio) with an exposure time of one-half second at 90 kV and 15 mA. All films were developed in an automatic film processer (Auveloper, S.S. White Company, Great Neck, New York) to insure consistency of technique.

Cephalometric Evaluation

The amount of growth of both the control and experimental condyles was evaluated by measuring the amount of elongation of the appliances from their radiographic images. This was accomplished by punching holes in the radiographs at both ends of the appliance images and measuring these distances with a dial vernier caliper (Helios

Plate 7. Cephalostat.



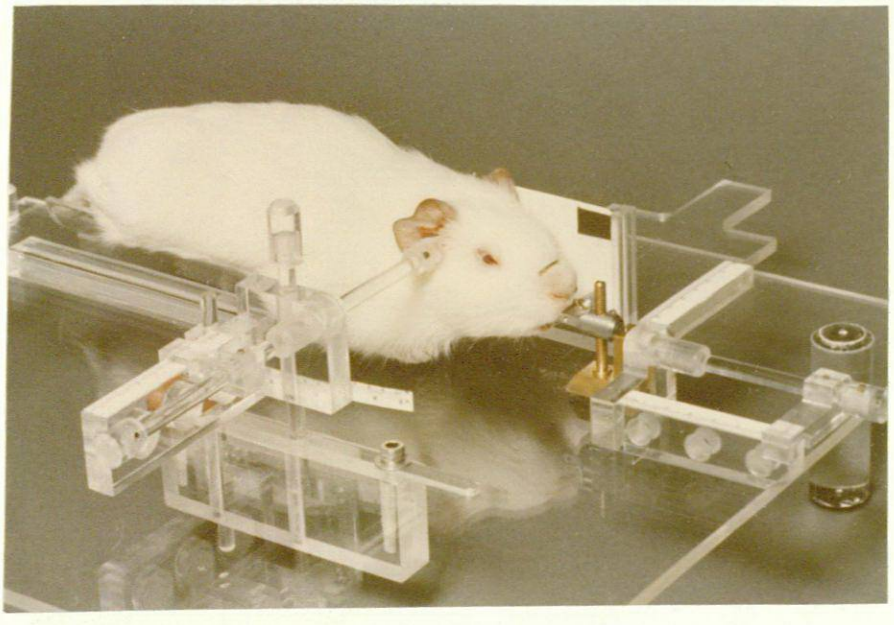
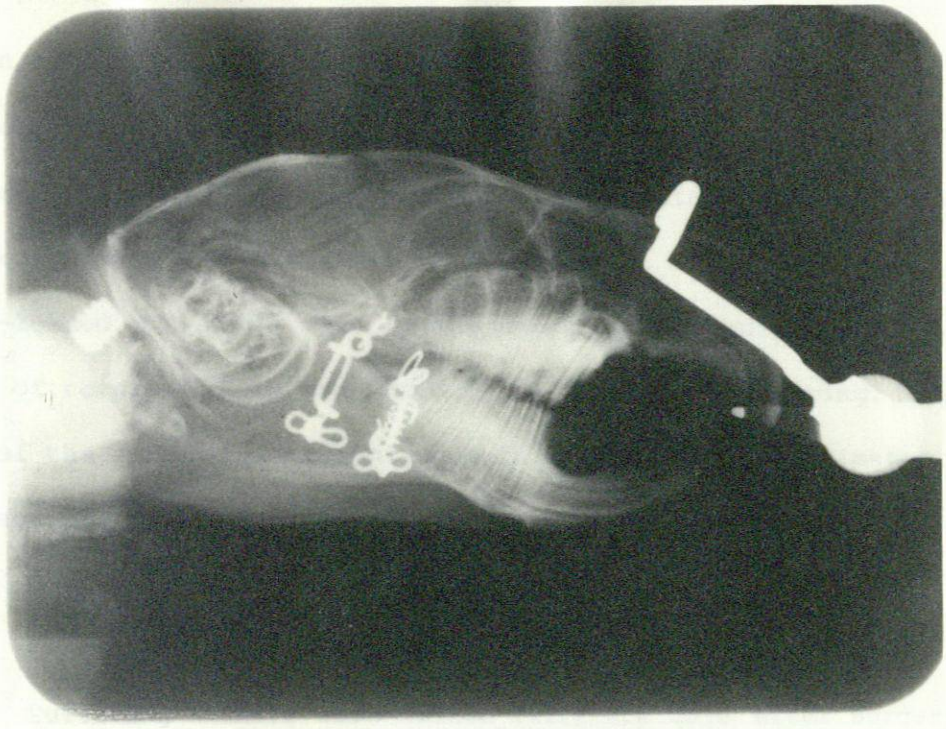
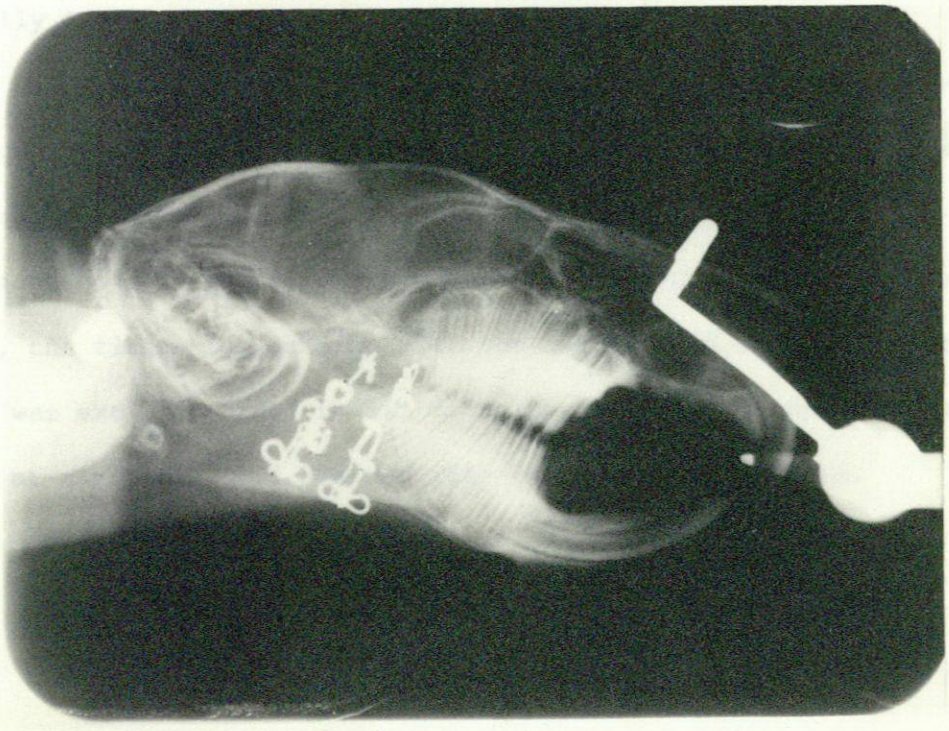


Plate 8. Radiographs
a. Initial radiograph
b. Eight-week radiograph





Gauge, D.C.A., Washington, D.C.). The image-length of the maxillary portion of the appliance was measured in a similar manner. This length, when compared to the actual length of the maxillary portion of the appliance provided a means of correcting for radiographic magnification. After correction of radiographic image-lengths, the force values of the spring-loaded appliances were easily determined by multiplying millimeters of compression by the known force level of the spring. The force level of the spring-loaded appliance in each animal approximated the force against which the mandible grew.

Anatomic and Histologic Examination

Surviving animals were killed fifty-six days after surgery by an over-dose of sodium pentobarbital (Diabutol, Diamond Laboratories, Des Moines, Iowa). Incisal wear and mandibular deviation were checked carefully in all animals. In addition, the surgical sites were opened and the tissue resistance of the appliances measured by cutting the zygomatic arches on either side of the appliances, freeing the ligature ties from the bone, and subjecting the tissue embedded appliances to a load analysis by means of a tension gauge. In one animal, the tissue surrounding both the control and experimental implants was excised, fixed in 10% formalin, embedded in paraffin, sectioned at six micra, and stained with hematoxylin and eosin.

Data Reduction

The present sample was divided into two sub-samples based

upon the two sizes of springs (four and eight ounces of force per millimeter) employed. The two groups numbered ten and twelve respectively.

The measurements taken from the cephalometric radiographs were analyzed in several ways: 1) cumulative condylar growth, 2) weekly growth increments, and 3) mean growth rate. The means and standard deviations for the corrected weekly incremental and cumulative growth were calculated for the control and experimental condyles in each sample. Animals with broken appliances were included in the sampling to determine mean and standard deviations until the time of breakage.

Due to several instances of appliance breakage and the resulting missing data, a randomized block analysis of variance was used on each sample to resolve differences between the mean growth rates for the control and experimental condyles. The mean growth rate of all animals was determined by dividing total growth by the number of weeks the appliances remained functional. This procedure allowed the use of animals in which appliance-breakage occurred in the final weeks of the experiment.

The linear model for this statistical design may be expressed as:

$\chi_{ij} = \mu + \tau_i + \beta_j + \varepsilon_{ij}$, where

χ_{ij} = the measurement of the ith treatment in the jth individual

μ = the population mean

τ_i = the effect of the ith treatment

β_j = the effect of the jth block

ε_{ij} = the experimental error associated with the ith treatment

in the jth individual.

In this design, each animal is considered to be a block containing two plots, the right and left condyles. Treatment-assignment to the condyles was random.

All statistical calculations were performed on an electronic calculator (Model 9100A; Hewlett-Packard Company, Loveland, Colorado).

RESULTS

The results of the present investigation are presented in two categories: 1) cephalometric findings and 2) general observations.

Cephalometric Findings

Table II shows the weekly means and standard deviations of the cumulative condylar growth for the four and eight ounce samples. These weekly means are graphically depicted in Figures 5 and 6. In general, the mean cumulative growth of the experimental (loaded) condyles was smaller than the growth of the control (unloaded) condyles. However, the large standard deviations indicate that there was wide scatter about the means.

The overall cumulative growth also provided the data for determining the degree of condylar compression produced by the spring-loaded appliances. The mean condylar loadings of the samples were calculated to be between 1 1/2 and 2 ounces (42.5 to 56.5 grams). The articular area of the condyle was estimated by graphic reconstruction to be approximately 6.8 $mm.^2$. Thus, the pressure exerted by the spring-loaded appliances ranged from .6 $oz./mm.^2$ to .8 $oz./mm.^2$ (6 $gms./mm.^2$ to 8 $gms./mm.^2$).

The means and standard deviations of the weekly growth increments are summarized in Table III. The means are graphed in Figures 7 and 8. Generally, the loaded condyles showed smaller weekly growth

TABLE II

MEAN CONDYLAR GROWTH
CUMULATIVE
(in mm)

Weeks	Control Side		Experimental Side	
	Mean	Standard Deviation	Mean	Standard Deviation
Four Ounce Sample				
1	.11	.29	-.03	.18
2	.40	.42	.05	.20
3	.56	.58	-.02	.17
4	.62	.62	.07	.22
5	.66	.73	.16	.25
6	.71	.75	.26	.28
7	.81	.74	.33	.36
8	.95	.59	.41	.37
Eight Ounce Sample				
1	.15	.22	.18	.36
2	.25	.36	.17	.38
3	.38	.46	.13	.33
4	.44	.47	.18	.35
5	.58	.57	.24	.35
6	.74	.85	.28	.40
7	.73	.88	.28	.43
8	.82	.93	.24	.50

Figure 5. Cumulative condylar growth: four ounce sample.

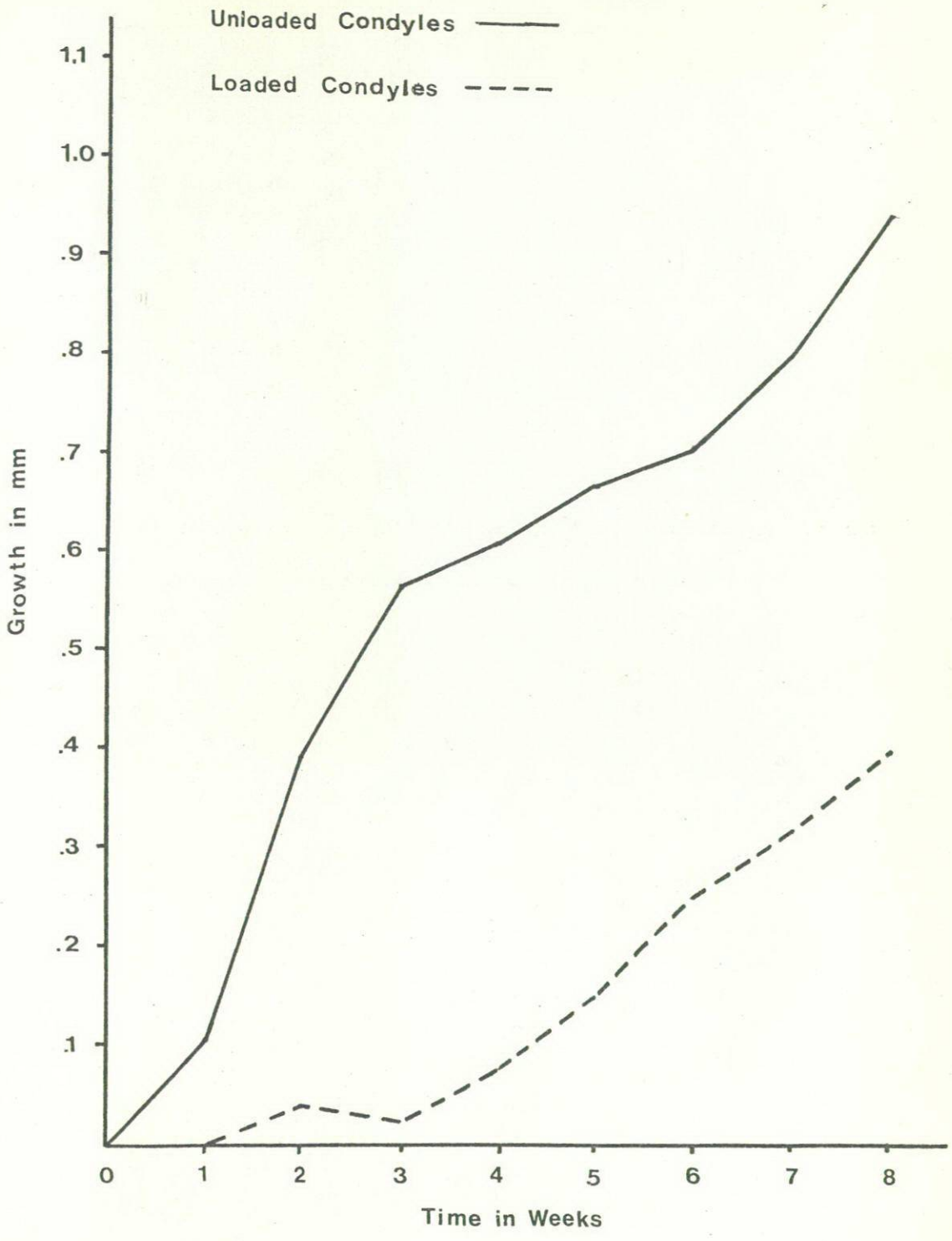

Unloaded Condyles
Loaded Condyles
Growth in mm
1.1
1.0
.9
.8
.7
.6
.5
.4
.3
.2
.1
0
1
2
3
4
5
6
7
8
Time in Weeks

Figure 6. Cumulative condylar growth: eight ounce sample.

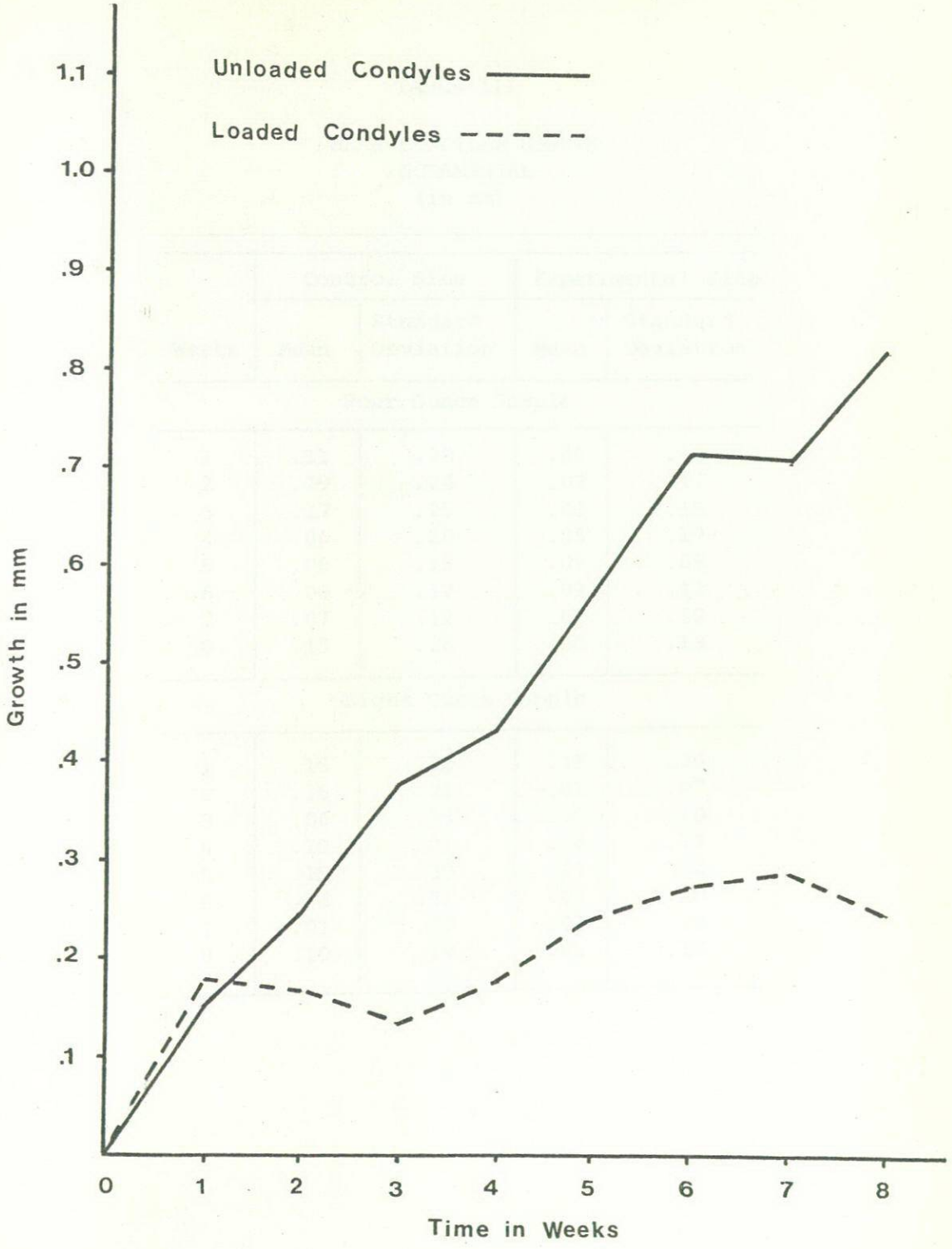

Unloaded Condyles
Loaded Condyles
1.1
1.0
.9
.8
.7
.6
.5
.4
.3
.2
.1
Growth in mm
0
1
2
3
4
5
6
7
8
Time in Weeks

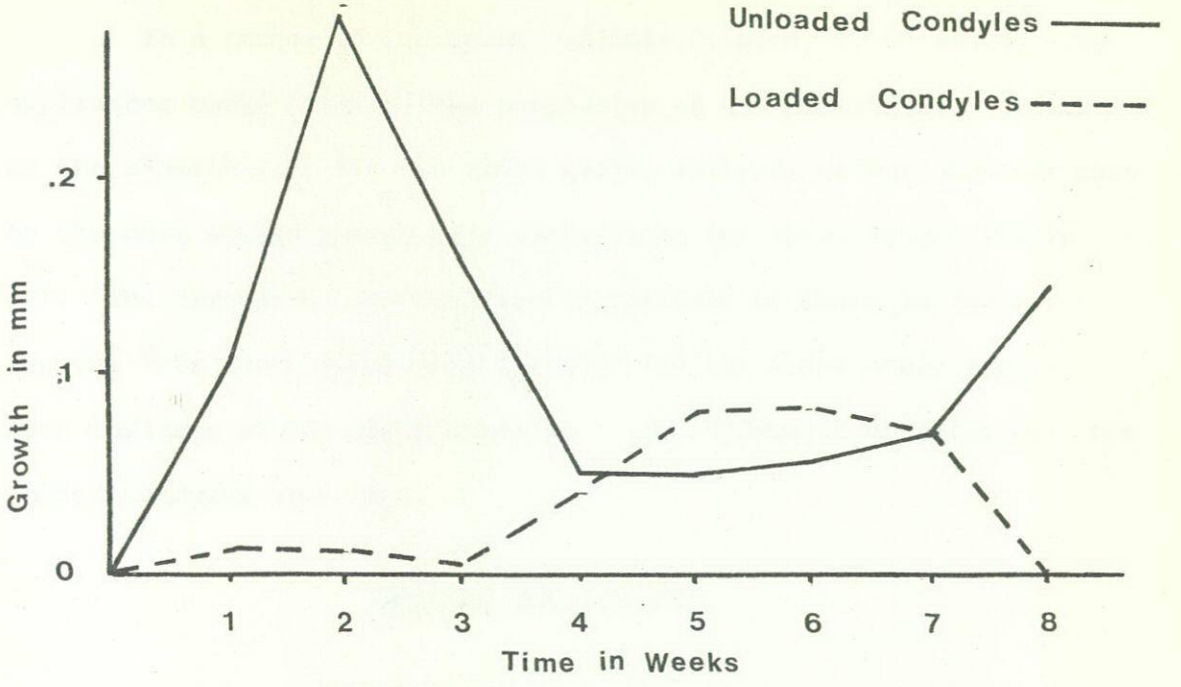
TABLE III

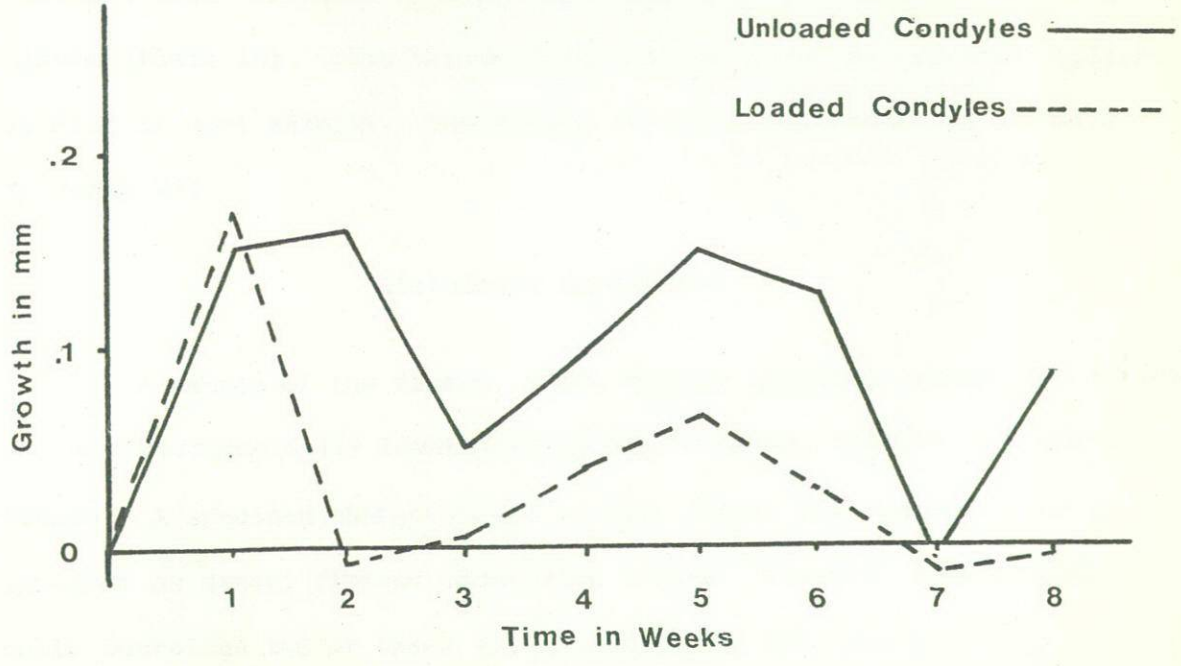
MEAN CONDYLAR GROWTH
INCREMENTAL
(in mm)

Weeks	Control Side		Experimental Side	
	Mean	Standard Deviation	Mean	Standard Deviation
Four Ounce Sample				
1	.11	.29	.02	.13
2	.29	.24	.02	.17
3	.17	.25	.00	.15
4	.06	.28	.05	.17
5	.06	.19	.09	.09
6	.06	.17	.09	.11
7	.07	.12	.07	.12
8	.15	.26	.00	.13
Eight Ounce Sample				
1	.15	.22	.18	.36
2	.16	.21	-.01	.07
3	.06	.25	.00	.18
4	.10	.21	.04	.17
5	.15	.30	.07	.14
6	.14	.51	.03	.20
7	-.01	.23	-.02	.19
8	.10	.19	-.03	.10

Figure 7. Weekly growth increments: four ounce sample.

Figure 8. Weekly growth increments: eight ounce sample.


Unloaded Condyles
Loaded Condyles
Growth in mm
.2
.1
0
1
2
3
4
5
6
7
8
Time in Weeks


Unloaded Condyles
Loaded Condyles
Growth in mm
.2
.1
0
1
2
3
4
5
6
7
8
Time in Weeks

increments than the unloaded condyles.

In a number of instances (animals C, G, H, O, U, and V), the appliances broke prior to the completion of the experiment. Because of the missing data for the final weeks, analysis of variance was done on the mean weekly growth rate rather than the total growth (Table IV). The results of the analysis of variance is shown in Table V for the four ounce sample and Table VI for the eight ounce smaple. Both analyses showed significant ($p < .05$) treatment differences: the loaded condyles grew less.

General Observations

Tissue Reaction to Appliances

Two types of tissue were commonly found surrounding the appliances. One type grossly appeared as a thin, soft, transparent tissue (Plate 9) while a second type appeared as a firmer, more opaque tissue (Plate 10). This tissue formation was found to increase appliance loading in most animals. The degree of tissue resistance is summarized in Table VII.

Histologic Appearance

A sample of the tissue, which grossly appeared transparent (Plate 9) was histologically identified as normal dense, fibrous connective tissue. A specimen characterized as firm (Plate 10), microscopically appeared as dense, fibrous connective tissue. However, some of these cells contained two or three nuclei and looked dysplastic.

TABLE IV

MEAN GROWTH RATE
(in mm/week)

Guinea Pig	Control Side	Experimental Side
Four Ounce Sample		
A	.02	.14
B	.04	.07
C	.20	.00
D	.11	.06
E	.07	.02
F	.05	.04
G	.14	.00
H	.21	.04
I	.21	.01
J	.15	.02
Eight Ounce Sample		
K	.14	.03
L	.05	.02
M	.04	.07
N	.12	.10
O	.09	.03
P	.06	-.03
Q	.08	.03
R	.39	.01
S	.00	-.08
T	.21	.13
U	-.08	.01
V	.14	.04

TABLE V

ANALYSIS OF VARIANCE:
MEAN GROWTH RATE
FOUR OUNCE SAMPLE

Source	df	SSQ	MSQ
Treatments	1	.032	.032
Blocks	9	.014	. .
Error	9	.052	.005
Total	19	.097	. .

$F(1,9) = 5.56$
$F_{.95}(1,9) = 5.12$

TABLE VI

ANALYSIS OF VARIANCE:
MEAN GROWTH RATE
EIGHT OUNCE SAMPLE

Source	df	SSQ	MSQ
Treatments	1	.032	.032
Blocks	11	.111	. .
Error	11	.069	.006
Total	23	.212	. .

$F(1,11) = 5.13$
$F_{.95}(1,11) = 4.84$

Plate 9. Tissue Proliferation around Spring-loaded appliances.

Plate 10. Tissue proliferation around free-sliding appliances.





TABLE VII

TISSUE RESISTANCE OF APPLIANCES

Guinea Pig	Control Side	Experimental Side
Four Ounce Sample		
A	++	O
B	++	+
C	++	+
D	+	+
E	++	++
F	++	+
G	++	+
H	+	O
I	+	+
J	++	+
Eight Ounce Sample		
K	++	+
L	O	+
M	++	+
N	++	O
O	O	++
P	++	+
Q	O	+
R	O	O
S	++	+
T	+	O
U	+	+
V	++	+

O Appliance movement unaffected by tissue proliferation.

+ Appliance movement hindered by tissue proliferation.

++ No appliance movement with sixteen ounce loading.

Gross Observations

Unilateral forces applied to the condyle by the appliances were associated, in certain instances, with uneven wear of the incisor teeth (Plate 11). Incisal wear was observed in both the four and eight ounce samples, and was examined by means of contingency tables. There was no observable relationship between incisal wear and tissue resistance. However, there was an observable relationship between incisal wear and condylar loading (Table VIII). According to a 2 x 2 test, this relationship between incisal wear and condylar loading was significant ($p < .05$).

Mandibular deviation was observed in various animals from time to time, but no consistent pattern could be discerned.

A summary of each animal's weight throughout the experiment is shown in Table IX. After a post-surgical weight drop of forty to sixty grams, the animals generally showed a steady weight increase. By the end of the experiment, the mean weight had more than doubled.

TABLE VIII

CONTINGENCY TABLE
INCISAL WEAR
AND CONDYLAR
LOADING

		Spring R	Spring L
Incisal Wear	R	1	3
	L	8	1

Plate 11. Incisal Wear: The spring-loaded appliance was on the right side; incisors are worn toward the control side (left).

BRARY

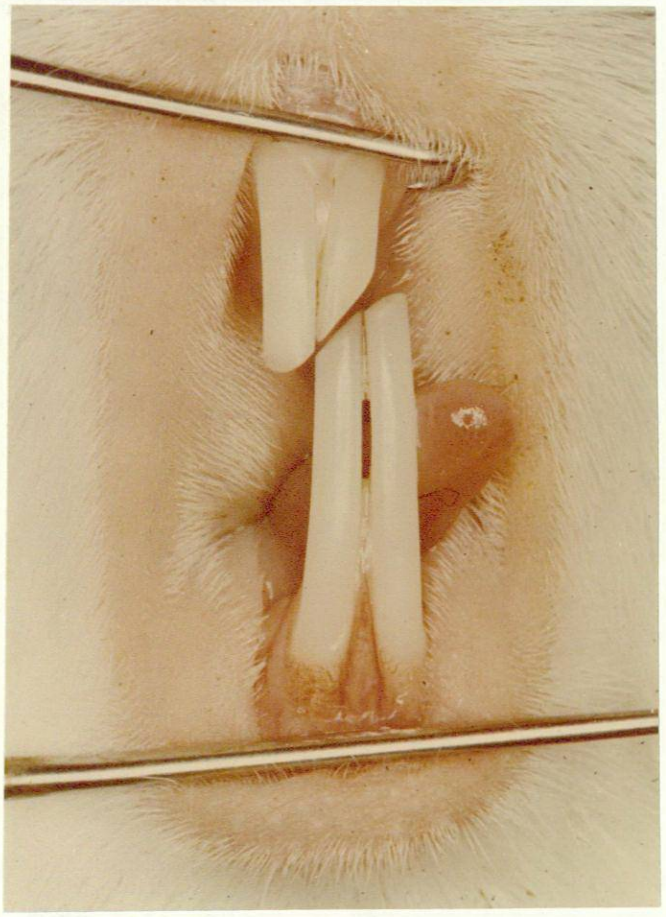


TABLE IX

WEIGHT GAIN
(in grams)

Guinea Pig	Initial	Four Weeks	Eight Weeks
A	274	313	592
B	246	301	487
C	272	324	549
D	203	321	523
E	250	267	429
F	260	352	609
G	234	382	607
H	341	431	600
I	242	307	465
J	218	269	398
K	220	350	582
L	249	314	549
M	219	383	651
N	257	391	565
O	239	444	635
P	356	395	499
Q	238	385	635
R	265	445	699
S	312	423	623
T	238	359	613
U	365	439	642
V	334	474	672
Mean	269.2	366.8	573.8

BRARY

DISCUSSION

The results of the present investigation indicate that loaded mandibular condyles grew significantly less than did unrestrained condyles.

Certain technical details warrant discussion. Guinea pigs of sufficient size to permit the placement of appliances had only a limited amount of mandibular growth remaining. For this reason, technical error might be expected to have a considerable impact on the power of statistical analysis. It is assumed that the use of a precise radiographic technique (well-designed cephalostat, careful positioning of the animal, correction of magnification, etc.) served to minimize these problems. Other technical variables, however, were not so easily controlled.

Although care was exercised to ensure that the spring-loaded appliances were passive at the start of the experiment, it is probable that some were slightly active and others slack at the start of the experiment.

Another possible source of error was tissue proliferation around the appliances (Plates 9 and 10) which might serve to add to the force exerted by the appliances, both experimental and sham. This tissue proliferation did, in fact, increase appliance loading in a number of instances (Table VII). Indeed, there was a significant tendency ($p < .05$, Steel and Torrie) for greater proliferation

on the control side. As a result, condylar loading was, in all probability, frequently higher than calculated and the differential loading between control and experimental sides somewhat reduced. A solution to this problem of tissue proliferation and subsequent interference with the action of the appliances would be to sheath the appliances in a protective casing.

The incisal wear seen in some animals (Plate 11) is largely angled toward the control side. Such wear indicates that the animal may have been able to alter--by functional shifting--the load on the condyles. In future studies, an incisal guidance appliance could be used to avoid this problem.

The choice of a simple randomized block analysis of variance was based on: 1) the presumed need to account for variability among experimental animals, and 2) the necessity of estimating missing data points late in the experiment. By using the average growth per week instead of total growth, the problem of missing data was circumvented, although differences among weeks could not be estimated.

As may be seen in Tables V and VI there was no great gain in precision by accounting for the sum of squares for blocks in either sample, inasmuch as animal variability accounted for only a small proportion of the total variability in average rates. However, there seems to be little theoretical justification for changing, after the fact, to a completely randomized design. This design would raise the levels of significance, but would not alter the conclusions.

The use of a two-tailed test was a very conservative method

of evaluating the data. Based on a variety of long bone inhibition studies (Haas, 1945, 1948; Blount and Clarke, 1949; Siffert, 1956; Strobino, et al., 1961; Sijbrandij, 1963) the only effect of compression on growth cartilages is that of inhibition. Since only changes of a given sign are expected, a one-tailed test has greater power to resolve significant departures from hypothesis. If it were not for an article by Folke and Stallard (1972) showing increased cellular activity in loaded condyles, a one-tailed test would have been employed. It would, however, be questionable to change the hypothesis after data are gathered, even though the levels of significance would be somewhat elevated.

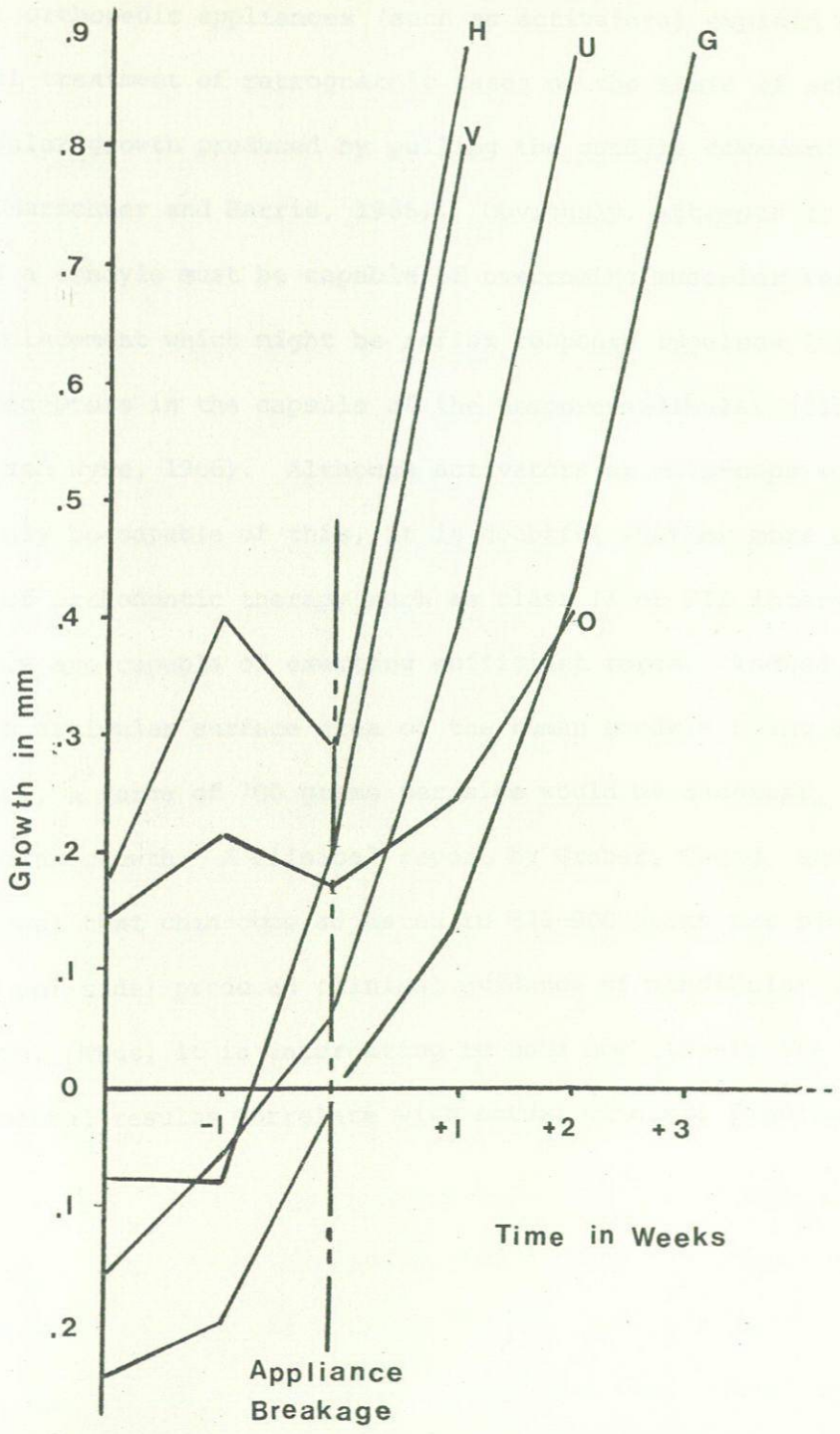
As has been reported elsewhere (Leash, et al., 1973), the use of droperidol and fentanyl (Innovar-Vet) as an anesthetic agent produced some adverse effects. Intra-muscular injection in the thigh led to self-mutilation of the hind feet on the injected side in six out of twenty-four animals. It is assumed, however, that this had little effect on the outcome of the experiment.

Baume (1961) and Sicher (1965), among others, have suggested that the condylar cartilage resembles an epiphyseal growth plate, functionally if not structurally. Experimentally, Sijbrandij (1963) showed that the growth of an epiphyseal plate is inversely proportional to the magnitude of compressive forces directed against it. Using rabbits, Sijbrandij found that 11.5-19.5 grams/mm.^{2} blocked tibial epiphyseal growth. In the present study, the cumulative longitudinal growth of the four ounce samples (.40 mm.) was almost twice that of

the eight ounce sample (.24 mm.). Using an approximation of the condylar area (6.8 $mm.^2$), this amounts to a pressure of 7 $grams/mm.^2$, approximately one-half of Sijbrandij's findings. This force reduction may be related to the appositional-type growth of the condylar cartilage (Meikle, 1973) being more responsive to pressure than the purely interstitial growth seen in the epiphyseal plate. Moreover, it should be noted that while these forces slowed mandibular growth, they did not stop it. Perhaps, condylar growth would occur in the face of even greater pressure if such an experiment were conducted over a longer period of time. In this context, it is suggested that the use of somewhat lighter forces might be advantageous in that it would allow more growth and, thereby permit more reliable measurements.

The effect of reduced or negative loading on growth cartilage has also been studied. Melanson and Van Dyken (1972) unloaded condyles in rats by surgical removal of the glenoid fossa. They reported that, when scarring did not occur, the condyle on the experimental side grew faster than did the control. Fuller (1974), isolated the condyle from the rest of the mandible in growing guinea pigs, and found that the freed, unloaded condyles grew faster than did the controls. Furthermore, in the present study, when the spring-loaded appliances broke, the condyle responded with an apparent surge of growth (Figure 9). It might be assumed therefore, that if 1) condyles normally grow against some resistance, and 2) condylar growth is inversely proportional to the degree of loading, then condylar growth can either be accelerated or retarded by adjusting the resistance.

Figure 9. Growth rate change after breakage of spring-loaded appliances.


.9
.8
.7
.6
.5
.4
.3
.2
.1
0
.1
.2
Growth in mm
H
V
U
G
O
-1
+1
+2
+3
Time in Weeks
Appliance
Breakage

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In effect, clinical efforts to alter mandibular growth have long been based on this assumption. For example, advocates of functional orthopedic appliances (such as activators) explain their successful treatment of retrognathic cases on the basis of accelerated mandibular growth produced by pulling the condyle downward and forward (Marschner and Harris, 1966). Obviously, attempts to load or unload a condyle must be capable of overcoming muscular resistance to displacement which might be reflex response impulses initiated from receptors in the capsule of the temporomandibular joint (Greenfield and Wyke, 1966). Although activators or chin-caps would almost certainly be capable of this, it is doubtful whether more conventional forms of orthodontic therapy such as class II or III intermaxillary elastics are capable of exerting sufficient force. Indeed, with the average articular surface area of the human condyle being approximately 100 $mm.^2$, a force of 700 grams per side would be necessary to retard mandibular growth. A clinical report by Graber, Chung, and Aoba (1968) did reveal that chin-cups adjusted to 675-900 grams per side (1.5-2 pounds per side) produced clinical evidence of mandibular growth inhibition. Thus, it is interesting to note how closely the present experimental results correlate with actual clinical findings.

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SUMMARY AND CONCLUSIONS

Mandibular prognathism is perhaps one of the most difficult skeletal imbalances which confronts the clinical orthodontist. Non-surgical correction of this condition has been attempted clinically in many ways including chin-caps, class III intermaxillary elastics, and distally directed inclined planes. A common factor in all these methods has been directing distal traction against the growing mandible. However, basic information concerning the effect of distal traction on mandibular growth is generally unavailable.

The present investigation was designed to obtain basic, quantitative information on the effect of transcondylar pressure on mandibular growth in twenty-two growing guinea pigs. On one side of the animals, a spring-loaded appliance was placed surgically between the zygomatic arch and the body of the mandible, while a free-sliding appliance was placed similarly on the other side of the animal. To assess mandibular growth by measurement of appliance elongation, cephalometric radiographs were obtained weekly for eight weeks. With this design, the free-sliding appliances served as a control to measure normal mandibular growth, while the spring-loaded appliances generated progressively increasing transcondylar loading. The effects of treatment were analyzed by a randomized block analysis of variance.

The results of this investigation revealed that:

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1) loaded mandibular condyles grew significantly less than unloaded condyles

2) condylar growth produced condylar loading of about 6-8 grams/mm.^{2}.

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BIBLIOGRAPHY

Adams, C. D., Meikle, M. C., Norwich, K. W., and Turpin, D. L. 1972. Dentofacial remodelling produced by intermaxillary elastics in Macaca mulatta. Arch. Oral Biol., 17: 1519-1535.

Angle, E. H. 1907. Malocclusion of the Teeth, 7th ed., S. S. White Dental Mfg. Co., Philadelphia.

Armstrong, C. J. 1961. Clinical evaluation of the chin-cup: preliminary report. Aust. Dent. J., 6: 338-346.

Baker, L. W. 1937. Prefunctional and functional influences on bone growth. Harvard Dent. Rec., 11: 2-10.

Baume, L. J. 1961. Principles of cephalofacial development revealed by experimental biology. Am. J. Orthod., 47: 881-901.

Blackwood, H. J. J. 1966. Growth of the mandibular condyle of the rat studied with tritiated thymidine. Arch. Oral Biol., 11: 493-500.

Blount, W. P. and Clarke, G. R. 1949. Control of bone growth by epiphyseal stapling. J. Bone Joint Surg., 31A: 464-478.

Breitner, C. 1940. Bone changes resulting from experimental orthodontic treatment. Am. J. Orthod., 26: 521-546.

Brodie, A. G. 1941. On the growth pattern of the human head from the third month to eighth year of life. Am. J. Anat., 68: 209-262.

Case, C. S. 1893. Some principles governing the development of facial contours in the practice of orthodontia. Dental Cos., 35: 959-969.

Charles, S. W. 1925. The temporo-mandibular joint and it's influence on the growth of the mandible. Br. Dent. J., 46: 845-855.

Das, A., Meyer, J., Sicher, H. 1965. X-ray and alizarin studies of the effect of bilateral condylectomy in the rat. Angle Orthod., 35: 138-148.

du Hamel, H. L. 1745. Sur le developpement et la crue des os des amimaux. Histoire de l'academie royale des sciences: memoires de methematique et de physique: 354-370. Cited from Scott, B. L. and Pease, D. C. 1956. Electron microscopy of the epiphyseal apparatus. Anat. Rec., 126: 465-495.

Durkin, J. F. 1972. Secondary cartilage: a misnomer. Am. J. Orthod., 62: 15-41.

Duterloo, H. S. 1967. In vivo implantation of the mandibular condyle of the rat. Ph.D. Thesis, University of Nijmegen, The Netherlands.

Felts, W. J. L. 1957a. Tissue survival in mouse whole bone transplants. Am. J. Phys. Anthropol., 71: 201-216.

Felts, W. J. L. 1957b. A comparison of subcutaneous implants of isologous and homologous immature whole mouse bones. Transplant. Bull., 4: 131-134.

Felts, W. J. L. 1959. Transplantation studies of factors in skeletal organogenesis. I. The subcutaneously implanted immature long-bones of the rat and mouse. Am. J. Phys. Anthropol., 71: 201-216.

Felts, W. J. L. 1961. In vivo implantation as a technique in skeletal biology. Int. Rev. Cytol., 12: 243-302.

Folke, L. E. A. and Stallard, R. E. 1966. Condylar adaptation to a change in intermaxillary relationship. J. Periodont. Res., 1: 79-89.

Fuller, D. S. 1974. Personal communication.

Gange, R. S. 1973. Personal communication.

Gelbke, H. 1951. The influence of pressure and tension on growing bones in experiments with animals. J. Bone and Joint Surg., 33A: 947-954.

Gianelly, A. A. and Moorrees, C. F. 1965. Condylectomy in the rat. Arch. Oral Biol., 10: 101-106.

Glücksmann, A. 1938. Studies on bone mechanics in vitro. I. Influence of pressure on orientation of structure. Anat. Rec., 72: 97-115.

Glucksmann, A. 1942. The role of mechanical stresses in bone formation in vitro. J. Anat., 76: 231-239.

Graber, T. M., Chung, D. D. B., and Aoba, J. T. 1968. Dentofacial orthopedics versus orthodontics. Aust. Dent. J., 1: 84-105.

Graber, T. M. 19691 Dentofacial orthopedics. Current Orthodontic Concepts and Techniques. Vol. II, ed. T. M. Graber, W. B. Saunders Co., Philadelphia, 1969, pp. 919-988.

Greenfield, B. E. and Wyke, B. 1966. Reflex innervation of the temporomandibular joint. Nature, 211: 940-941.

Haas, S. L. 1945. Retardation of bone growth by a wire loop. J. Bone and Joint Surg., 27: 25-36.

Haas, S. L. 1948. Mechanical retardation of bone growth. J. Bone and Joint Surg., 30A: 506-512.

Haas, S. L. 1950. Restriction of bone growth by pins through the epiphyseal cartilaginous plate. J. Bone and Joint Surg., 32A: 338-343.

Hunter, J. 1772. The works of John Hunter, F.R.S., with notes. Vol. 4, p. 219, ed. James F. Palmer, Longman, Rees, Ornie, Brown, Green, and Longman, 1835-1837, London.

Janzen, E. K. and Bluher, J. A. 1965. The cephalometric, anatomic and histologic changes in the Macaca mulatta after application of a continuous acting retraction force on the mandible. Am. J. Orthod., 51: 823-855.

Jolly, M. 1961. Condylectomy in the rat. Aust. Dent. J., 6: 243-256.

Koski, K. and Makinen, L. 1963. Growth potential of transplanted components of the mandibular ramus of the rat. Suom. Hammaslaak Toim., 59: 296-308. Cited from Cranio-facial Growth in Man, ed., R. E. Moyers and W. M. Krogman, Pergamon Press, New York, 1971, pp. 125-138.

Koski, K. and Mason, K. E. 1964. Growth potential of transplanted components of the mandibular ramus of the rat II. Suom. Hammaslaak Toim., 60: 209-217. Cited from Cranio-facial Growth in Man, ed. R. E. Moyers and W. M. Krogman, Pergamon Press, New York, 1971, pp. 125-138.

Koski, K. and Rönning, O. 1965. Growth potential of transplanted components of the mandibular ramus of the rat III. Suom. Hammaslaak Toim., 61: 292-297. Cited from Cranio-facial Growth in Man, ed. R. E. Moyers and W. M. Krogman, Pergamon Press, New York, 1971, pp. 125-138.

Koski, K. 1966. Some characteristics of craniofacial growth cartilages. Cranio-facial Growth in Man, ed. R. E. Moyers and W. M. Krogman, Pergamon Press, New York, 1971, pp. 125-138.

Lacroix, P. 1951. The Organization of Bones. Blakiston Div., McGraw-Hill Book Co., New York.

Leash, A. M., Beyer, R. D. and Wilbur, R. G. 1973. Self-mutilation following Innovar-Vet injection in the guinea pig. Lab. Anim. Sci., 23: 720-721.

Marschner, J. F. and Harris, J. E. 1966. Mandibular growth and class III treatment. Angle Ortho., 36: 89-93.

Melanson, E. and Van Dyken, C. 1972. Studies in condylar growth. M. S. Thesis, University of Michigan, Ann Arbor, Michigan.

Meikle, M. C. 1970. The effect of a class II intermaxillary force on the dentofacial complex of the adult Macaca mulatta monkey. Am. J. Orthod., 58: 323-340.

Meikle, M. C. 1973. In vivo transplantation of the mandibular joint of the rat--an autoradiographic investigation into the cellular changes at the condyle. Arch. Oral Biol., 18: 1011-1020.

Moffett, B. C., Johnson, L. C., McCabe, J. B., and Asker, H. C. 1964. Articular remodelling in the adult human temporomandibular joint. Am. J. Anat., 115: 119-142.

Monson, J. W. and Felts, W. J. L. 1961. Transplantation studies of factors in organogenesis. II. The response of the immature, mouse humerus to longitudinal compressive force. Am. J. Phys. Anthropol., 19: 63-77.

Moss, M. L. 1960. Functional analysis of human mandibular growth. J. Prosth. Dent., 10: 1149-1159.

Moss, M. L. 1964. Vertical growth of the human face. Am. J. Orthod., 50: 359.

Moss, M. L. 1968. The primacy of functional matrices in orofacial growth. Dent. Pract., 19: 65-73.

Moss, M. L., Low, W. D., and Meehan, M. 1968. Functional analysis of epiphyseal growth plate elongation. Anat. Rec., 160: 397 (Abstr.).

Moss, M. L. and Rankow, R. 1968. The role of the functional matrix in mandibular growth. Angle Orthod., 38: 95-103.

Moss, M. L. and Salentijn, L. 1968. The primary role of functional matrices in facial growth. Am. J. Orthod., 55: 566-577.

Moss, M. L. and Salentijn, L. 1969. The capsular matrix. Am. J. Orthod., 56: 474-490.

Neumann, B. 1969. Removable appliances, Current Orthodontic Concepts and Techniques. Vol. II., ed. T. M. Graber, W. B. Saunders Co., Philadelphia, 1969, pp. 817-874.

Ogston, A. 1878. On the growth and maintenance of the articular ends of the bones. J. Anat. and Physiol., 12: 503-517.

Peskin, S. and Laskin, D. M. 1965. Contributions of autologous condylar grafts to mandibular growth. Oral Surg., Oral Med., Oral Path., 20: 517-533.

Petrovic, A. G. 1972. Mechanisms and regulation of mandibular condylar growth. Acta Morphol. Neerl.-Scand., 10: 25-34.

Phemister, D. B. 1933. Operative arrestment of longitudinal growth of bones in the treatment of deformities. J. Bone and Joint Surg., 15: 1-15.

Pimenidis, M. Z. and Gianelly, A. A. 1951. The effects of early postnatal condylectomy on the growth of the mandible. Am. J. Orthod., 62: 42-47.

Ramfjord, S. P. and Hiniker, J. J. 1966. Distal displacement of the mandible in adult rhesus monkeys. J. Prosth. Dent., 16: 491-502.

Rushton, M. A. 1944. Growth of the mandibular condyle in relation to some deformities. Br. Dent. J., 76: 57-68.

Sarnat, B. C. and Engle, M. B. 1951. A serial study of the mandibular growth after removal of the condyle in the Macaca rhesus monkey. Reconstruct. Surg., 7: 364-379.

Scott, B. L. and Pease, D. C. 1956. Electron microscopy of the epiphyseal apparatus. Anat. Rec., 126: 465-495.

Scott, J. H. 1967. Dento-facial Development and Growth. Pergamon Press, Oxford.

Sicher, H. 1947. Growth of the mandible. Am. J. Orthod., 33: 30-35.

Sicher, H. 1965. Oral Anatomy. C. V. Mosby Co., St. Louis.

Siffert, R. S. 1956. The effects of staples and longitudinal wires on epiphyseal growth. J. Bone and Joint Surg., 38A: 1077-1087.

Sijbrandij, S. 1963. Inhibition of tibial growth by means of compression of its proximal epiphyseal disc in the rabbit. Acta Anat., 55: 278-285.

IBRARY

Silbermann, M. and Frommer, J. 1972. Vitality of chondrocytes in the mandibular condyle as revealed by collagen formation. An autoradiographic study with ^{3}H-Proline. Am. J. Anat., 135: 359-370.

Strobino, L. J., French, G. O. and Colonna, P. C. 1952. The effect of increasing tension on the growth of epiphyseal bone. Surg. Gynec. and Obstet., 95: 694-700.

Symons, N. B. B. 1951. Studies on the growth and form of the mandible. Dent. Rec., 71: 41-43.

Thilander, B. 1963. Treatment of angle class III malocclusion with chin-cap. Trans. Europ. Orthod. Soc., pp. 384-398.

Thilander, B. 1965. Chin-cap treatment for angle class III malocclusion. Trans. Europ. Orthod. Soc., pp. 311-327.

Weinberger, B. W. 1926. Orthodontics, A Historical Review of its Origin and Evolution. St. Louis, the C. V. Mosby Co.

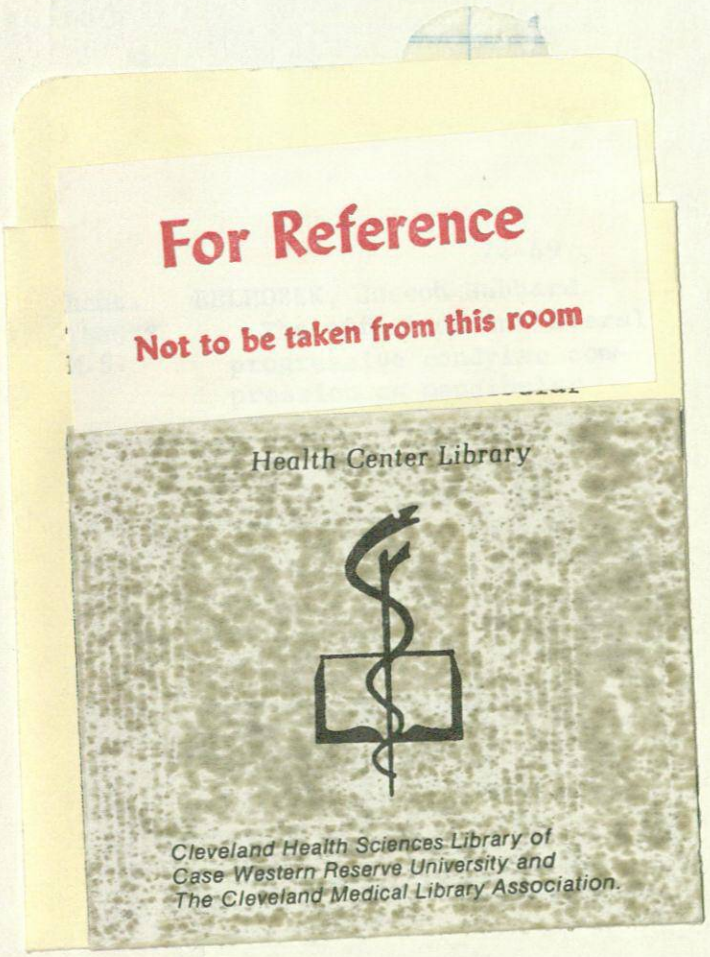
White, J. W. 1935. Femorial shortening for equalization of leg length. J. Bone and Joint Surg., 17: 596-604.

Willis, R. A. 1936. The growth of embryo bones transplanted whole in the rat's brain. Proc. Roy. Soc. Lond.(B), 120: 493-496.

Wolff, J. 1892. Das Gesetz der Transformation der Knochen, August Hirschwald. Cited from Gelbke, H. 1951. The influence of pressure and tension on growing bones in experiments with animals. J. Bone and Joint Surg., 33A: 947-954.

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