NOTE: PART 2 OF THIS REPORT WILL BE PUBLISHED IN THE JANUARY, 1975 ISSUE OF THE CLEFT PALATE JOURNAL.

Status of Research in Cleft Palate Anatomy and Physiology July, 1973—Part 1

DAVID R. DICKSON, Ph.D.
J. C. B. GRANT, M.C., M.B., Ch.B., F.R.C.S.
HARRY SICHER, M.D., D.Sc.
E. LLOYD DUBRUL, D.D.S., Ph.D.
JOSE PALTAN, M.D.
Pittsburgh, Pennsylvania

I. INTRODUCTION

This report was prepared at the request of the National Institute of Dental Research. The work involved was supported by a contract awarded by NIDR to the American Speech and Hearing Association to review the status of information pertaining to cleft lip and palate, to identify gaps in our information and areas in need of research emphasis. The authors of this paper were specifically asked to cover areas of head and neck anatomy relevant to cleft lip and palate. K. Kenneth Hisaoka, Deputy Associate Director for Extramural Programs, NIDR, initiated the over all State-of-the-Art review. Richard F. Curlee, Associate Secretary for Research and Scientific Affairs, American Speech and Hearing Association, was the project director for ASHA. Zora J. Griffo, Chief, Developmental Biology and Orofacial Anomalies, Extramural Programs, was the Project Officer for NIDR. Richard L. Christiansen, Program Officer, was an NIDR observer.

Today there is a critical need to develop more efficient and effective methods of diagnosis and treatment of persons with cleft palate. Pre-surgically the surgeon must plan an operative procedure which takes into account the blood supply, nerve supply, and the position of the musculature relevant to velopharyngeal closure if a successful functional result is to be obtained. We are far beyond the misconception that simple structural obliteration of the cleft is sufficient. The necessity of a functional result has been recognized for many years. However, with this recognition, not all surgical results solve the physical and physiologic problems

This report has been prepared for the National Institute of Dental Research, Department of Health, Education and Welfare, supported, in part, by Contract NIH-71-643.

Presented at the Annual Meeting, American Cleft Palate Association, Boston, Massachusetts, 1974.

The Committee has requested that this article be dedicated in memory of Dr. Grant, who gave his invaluable assistance in its preparation.

Dr. Dickson is Professor of Speech, Professor of Anatomy, University of Pittsburgh.

Dr. Grant was Professor of Anatomy, Department of Anatomy, University of Toronto.

Dr. Sicher is Emeritus Professor of Anatomy and Histology, Loyola School of Dentistry and Chicago College of Dental Surgery.

Dr. Dubrul is affiliated with the Department of Oral Anatomy, College of Dentistry, University of Illinois.

Dr. Paltan is Associate Professor of Anatomy and Histology, Department of Anatomy and Histology, School of Dental Medicine, University of Pittsburgh.

of cleft palate or this report would be unnecessary. Diagnostically, we face the problem of the non-cleft palate which is not operating in a manner to provide normal function. Muscular forces relevant to closure, innervation patterns which may be disrupted, and the changing mechanical forces with growth become important.

An additional factor complicates our diagnostic and therapeutic planning in the case of cleft palate. It is well known that an intact, functioning velopharyngeal mechanism is necessary for normal speech. However, the palate is only a part of this mechanism. Muscles of the palate insert into, and presumably have a functional effect on the pharynx (which is intimately involved in velopharyngeal function) and on the tongue. From an acoustical standpoint, it is quite likely that the respiratory-phonatory-articulatory system is tightly coupled. This acoustic or aerodynamic coupling suggests that changing parameters in any part of the system may change the function of the entire system. The effect of cleft palate on the function of the larynx and on the function of the other articulators becomes relevant. There have been many statements in the literature which suggest that certain laryngeal pathologies may be related to cleft palate. Feeding problems in cleft palate have been alluded to over and over again in the literature. Recognizing that olfaction and taste are intimately related with each other and with the desirability of certain types of foods, information on the effects of changing nasal aerodynamics on olfaction and the status of the taste receptors of the oral mechanism of children with cleft palates would seem relevant. There are also suggestions in the literature that ear disease in cleft palate children may be related to mechanical factors associated with cleft musculature. There have been suggestions that changes in nasal and pharyngeal aerodynamics due to cleft palate may have a profound influence on air conditioning functions of these structures. Also, abnormal tongue function has been attributed to children with cleft palate. To return to the musculature physiology of the velopharyngeal mechanism, more than normal muscular position is necessary for normal function. The motor and sensory innervation of the muscles is also relevant. While some attention has been given to innervation, only recently has any concerted effort been made to analyze the proprioceptive status of the unrepaired or repaired cleft palate, or for that matter of the entire oral-pharyngeal system. Also of relevance, especially to the surgeon, is the blood supply to all of these structures.

Thus it seems imperative, in the review of anatomy and physiology relating to cleft palate, to consider not only the palate but all of the oral-facial-pharyngeal-laryngeal system; and, in addition, the respiratory apparatus if we are to understand the impact of cleft palate on this system and not simply relate to the velum. In addition, both normal and abnormal conditions must be reviewed.

In all areas of research on clinical problems, one of the prime requisites is an adequate body of normative data to act as a point of reference. It is our unfortunate posture at this point in time that the structure and function of many parts of the human respiratory-phonatory-articulatory system, including the velum and pharynx, are not well understood. In part this dilemma is due to a historical conception that normative information on sub-human animals could be rather directly applied to human beings. There is increasing evidence, and, in the case of the neck structures, overwhelming evidence, that this is not the case. Not only has the erect posture of man created distinct morphologic changes in his neck structures as compared to even the highest sub-human primates, there is increasing evidence of fundamental, functionally significant differences in musculature, skeletal components, blood supply, and innervation. That is not to say that the fields of comparative anatomy and animal cell biology have nothing to offer in our attempt to understand the human system. On the contrary, studies comparing and contrasting human and sub-human form and function are necessary in order to improve understanding and derive basic biomechanical concepts of function. By contrasting the

form and function of one adaptive arrangement with that of another, the workings of these different structural complexes may be, in part, deciphered. There are many instances in which light has been shed on problems of human function such as the overlapping innervation of the middle ear and mandibular musculature by comparative studies. In addition, work such as that done on E. Coli response to lactose in the 1950's which led to operon theory has shed much ight on the function of the biological time-clock in embryogenesis and has suggested the importance of the timing of cell differentiation in the formation of tissues and organs. The importance of these factors to an understanding of normal and abnormal human development is obvious. Still, if we are to use animal data, we must, from any scientific research standpoint, demonstrate equivalence and not take equivalence for granted.

Information on morphologic changes which occur from birth to adulthood is necessary when confronted with a clinical population which spans that range. In many areas of structure and function relevant to cleft palate, developmental information is lacking. While in some areas of human embryology information is available, it is generally sparse and inadequate with regard to head and neck structures relevant to this report. In many of these areas of concern information is based on such restricted samples that variability cannot be assessed adequately. Finally, in recognition of the complex interaction of muscular and aerodynamic forces in the operation of the palate in speech, various types of data, such as electromyographic, radiologic, acoustic, pressure and flow need to be related.

Time has dictated that major restrictions, some quite arbitrary, be placed on this review. The principal focus of the review is the velopharyngeal, lingual and laryngeal mechanisms and craniofacial maturation. Even within these areas, it has not been possible to review embryogenesis, or factors of aerodynamic flow and pressure. Research in some areas such as oral sensation and perception have been alluded to with no attempt to review in depth. These decisions were based on the original decision by the reviewers to cover areas in depth at the sacrifice of some breadth. Several factors led to this decision. The literature related to cleft palate and its management is replete with disagreements in anatomical and physiological descriptions. These disagreements seem to have three bases in addition to legitimate differences in research findings based on methodology or sampling. These three factors are: 1) writers' unfamiliarity with what is known from published research, 2) writers' assumption of information for which no data can be found, and 3) confusing entanglement of data and opinion in much of what has been published. For these reasons, the reviewers have attempted to cover all published human research beginning in about 1600 in those specific areas which have been emphasized. Obviously errors of omission will have occurred. Nonetheless it is felt that an accurate picture of what is and is not known is presented in these areas of emphasis. In general, only research reports are presented. Published opinions unsubstantiated by data will be included only in rare instances where data is altogether lacking in an area of research or where the opinion stated seems especially unique and pertinent.

The reviewers would like to acknowledge the immeasurable help of some of their associates in completing this review. Dr. Wilma Maue Dickson, Mercy Hospital, Pittsburgh; Dr. Raimund Rueger, Allegheny Hospital, Pittsburgh; and Dr. Lou Gallia and Mr. Ross Long, Jr., School of Dental Medicine, University of Pittsburgh, gave much of their time, thought and energy to this review.

II. THE VELOPHARYNGEAL MECHANISM

In considering the structure and function of the normal velopharyngeal mechanism and the effects of cleft palate on that mechanism it became clear that a broader view than "velopharyngeal" needed to be adopted. As the review proceeded it became apparent that the structure and function of the Eustachian tube and

possibly the middle ear may be intimately related and need to be viewed together if their functions and malfunctions are to be understood. Thus an overview of the status of research on the middle ear musculature and on Eustachian tube structure and function will be presented first. The impact of this information on understanding the patterns of movement, muscular physiology, and anatomy of the normal and cleft palate velopharyngeal mechanism will follow.

Middle Ear Musculature

Normal. The concept of the tensor tympani and stapedius muscles as providing the inner ear protection from loud sounds has been subject to serious dispute. Only two electromyographic studies of these muscles in humans have been found. Fisch and Schucthess (*41*) found stapedius to contract at SPL's above 100 db with increasing contraction as SPL was increased. They also found a lower threshold of contraction for frequencies around 2000 hz. and for noise than for higher frequencies. Salomon and Starr (*101*) found tensor contractions to occur following or coincident with voluntary contraction of muscles supplied by cranial nerve VII, with speech, yawning, swallowing and coughing; but only occasionally following presentation of loud sounds. Stapedius contraction was associated with voicing and coughing but not with contraction of facial muscles. Most importantly, the stapedius and tensor contractions preceded or were coincident with the correlated activities and so were not judged to be in response to the acoustic stimulus of speech.

Cancura (*28*) in placing loads on the two muscles concluded that while some protective function (less than 10 db) may occur for low frequencies, high frequency transmission could be facilitated. Kevanishvili and Gracharia (*62*) found that direct stimulation of tensor led to a change of 5–10 dB SPL with frequency differential. Differences in the richness of supply of proprioceptors between the two muscles has been observed (*118*). However, the possible role of these muscles in Eustachian tube clearance has not been investigated, even though there is evidence that the tensor tympani has fibers originating from tensor veli palatini (*3*, *71*, *96*), the innervation of the two muscles if from the same cranial nerve (*2*, *94*, *95*) and the tensor tympani may have dual innervation (*66*).

Cleft Palate. There is overwhelming evidence of a high incidence of middle-ear disease and hearing loss in children with cleft palate (*68*, *85*, *86*, *111*, *112*). In spite of this, no studies of the middle ear musculature in cleft palate persons have been found. Rather, investigators have turned their full attention to the Eustachian tube even though the possible role of the middle-ear musculature in the Eustachian tube function is unresolved.

The Eustachian Tube

Normal. The structure and function of the Eustachian tube would seem from the report of Proctor (*92*) to be clear and without question. However, much of the information presented by Proctor without verification is inconsistent with actual research or apparently without basis in the known research literature. In addition, many assumptions about Eustachian tube function come from apparently non-validated indirect measures such as the electro-acoustic bridge (see Donaldson, 1973).

Rood (*96*) provides an extensive review of literature pertaining to Eustachian tube anatomy and physiology and points out that much of the detailed anatomy of the tube, especially developmentally, is still lacking. In a classic series of two studies by Rich (*94*, *95*), one of which was reprinted in a 1970 issue of *Otolaryngologic Clinics of North America*, it was demonstrated that the Eustachian tube is opened by the function of the tensor palatini muscle. This is consistent with the anatomical findings of Cleland (*30*), Blakeway (*10*), McMyn (*78*), Korner (*64*), Simkins (*104*), Aschan and Nylen (*3*), Ross (*98*), Dickson and Dickson (*34*), Rood (*96*) and Dickson (*33*) that tensor attaches to the tube, though there is some disagreement about

its exact site of attachment. It is also known that while the action of tensor palatini is on the isthmus of the tube, the pharyngeal end of the tube opens during phonation and swallowing (*13*, *42*). The data of Thomsen (*118*) suggest that a pressure differential between the middle ear and pharynx may be necessary in addition to muscular force to open the Eustachian tube.

The weight of evidence is that Eustachian tube opening occurs at the isthmus and at the pharyngeal orifice and that the isthmus opening, caused by tensor veli palatini by its attachment to the lateral membranous wall of the tube (plus a possible pressure differential) is necessary for tube clearance. There is also evidence that ciliary action within the tube is essential to clearance (*100*, *103*).

While studies of the histology of the Eustachian tube and associated structures (*115*), and the embryology and fetal development of the tube (*43*, *96*, *119*) are fairly complete, developmental studies of tubal structure in children are lacking. In addition, there is disagreement about the structure and function of the tensor palatini muscle. This subject will be reviewed in a later discussion of velopharyngeal function.

Cleft Palate. Research on Eustachian tube structure and function associated with cleft palate has been motivated by the high incidence of ear disease which has been found in this population. Bluestone (*13*) in a x-ray study of 116 cleft-palate infants, all with chronic non-suppurative otitis media, found that the infants' Eustachian tubes were blocked at both the aural and pharyngeal ends. However, the aural obstruction disappeared in many of the infants after middle ear aeration while the pharyngeal obstruction did not. He further found a difference in the nature of the pharyngeal obstruction between cleft-palate and non-cleft-palate children with otitis media. Bluestone, Wittel and Paradise (*17*) indicated that the child with unoperated cleft palate has a specific problem of retrograde clearance, while non-cleft-palate children with otitis media have problems in both prograde and retrograde clearance. In a further study, Bluestone, *et al.* (*18*) concluded that tube dysfunction in children with cleft palate may relate to an increase in Eustachian tube compliance. This conclusion is of particular interest in light of Dickson's (*33*) suggestion that the Eustachian tube cartilage of cleft palate individuals may be abnormal. On the other hand, cleft-associated abnormalities of the tensor palatini muscle have also been noted and might relate to abnormal Eustachian tube function. Surina and Jagr (*116*) and Kriens (*65*) found tensor to be hypoplastic while Dickson (*33*) did not. However Kriens (*65*), Braithwaite and Maurice (*19*), Fara and Dvorak (*38*) and Dickson (*33*) all reported an abnormal inferior attachment of tensor at the hamulus in cleft-palate fetuses. Finally, Noone, *et al.* (*84*) demonstrated that hamular fracture in cleft-palate surgery has no demonstratable effect on Eustachian tube function. Whether this latter finding is related to the abnormal attachment of tensor reported above is a matter of speculation. However, it is evident that the possible mechanical or physiological factors responsible for Eustachian tube malfunction in cleft palate have still not been demonstrated.

Palate and Pharynx

Normal

Patterns of movement: Hilton (*56*), Falkson (*37*), Wardill and Whillis (*124*) Strong (*112*), Harrington (*53*), Simpson and Witchner (*106*), Perlman (*89*), Bloomer (*12*), Bosma (*15*), Calnan (*26*), Astley (*5*), Graber, Bzoch and Aoba (*48*), Bzoch, Graber and Aoba (*25*), Moll (*80*), Griffith, *et al.*, (*49*), Isshiki, Honjow and Morimoto (*59*), Pruzansky and Mason (*93*), Skolnick (*107*), Skolnick (*108*), Hynes (*58*), Ewanowski, Dickson, Dickson, and Crummy (*36*), Benson (*8*), Simpson and Austin (*105*), and Peterson (*90*), have studied patterns of movement of the velum and pharynx either by observing patients with facial defects which have revealed these structures or by cineradiography. All of the studies together present compelling

evidence of the following factors. There is a basic difference in function of the velopharyngeal mechanism between swallow and speech. In speech the velum is displaced posteriorly and superiorly against the posterior pharyngeal walls, while the lateral pharyngeal walls are displaced medially and posteriorly against the sides of the velum and obliterate the Rosenmüeller fossa. In swallowing, the pharynx is more totally involved in closure. While Bjork and Nylen (*9*) concluded from their study of transverse tomograms that there was "no evidence that muscular contraction of the lateral pharyngeal walls plays an important part in velopharyngeal closure" their tomograph tracings show decrease in both the anterior-posterior and lateral dimensions of the velopharyngeal port during closure. Also, while Pigott (*91*) reports almost no lateral pharyngeal wall movement in normal speech, his view of the upper lateral walls may have been obliterated since he was viewing the mechanism from below. Much of the evidence suggests that the principle site of lateral pharyngeal wall motion is at the level of the tori tubari, (*11*, *12*, *15*, *26*, *36*, *89*). Adams (*1*) in making direct measures of pharyngeal diameter of patients under light anesthetic following tonsillectomy and/or adenoidectomy found that reduction of the distance between the Eustachian tube orifices was frequently demonstrated and measured as much as two cm. He stated that "on one occasion, while the patient was retching, the examining finger was tightly gripped between the orifices of the Eustachian tubes moving medially." He went on to state that "such extensive contractions in the upper part of the nasopharynx and its anterior wall in relation to the cartilagenous eustachian tube, came as a surprise, as traditionally these areas are regarded as relatively rigid structures."

Recently, McKerns and Bzoch (*77*) presented evidence that males and females differ in the nature of velar elevation. In contrast to the female, they found that "velar length is greater in males, the height of elevation is greater, the amount of contact is less, and the inferior point of contact is most usually above the palatal plane." They found further that these differences could not be explained by differences in the ratio of velar length to pharyngeal depth. A number of investigators, including Bzoch, Graber and Aoba (*25*), Graber, Bzoch and Aoba (*48*), Pruzansky and Mason (*93*), and Simpson and Austin (*105*), have found that the velum is longer when in closure than at rest. Bzoch, Graber and Aoba (*35*), and Graber, Bzoch and Aoba (*48*) also demonstrated that the third quadrant of the velum contacts the posterior pharyngeal wall on closure at about the level of the palatal plane or slightly below, while the high point of the velum rises 4–5 mm above that plane. Simpson and Austin found the mean angle of velar movement in adults to be 54° with a range of 41° to 74°. Many studies (*24*) have demonstrated that specific height of the velum in speech is dependent on the sound being produced and on phonetic context. One investigator (*52*) provided evidence of possible racial differences in velar posturing.

Kelsey, Hixon and Minifie (*61*), and Minifie *et al.* (*79*) used ultrasound to assess lateral pharyngeal wall motion. However they measured motion below the level of the mandibular angle and so may not have been measuring motion involved in or related to velopharyngeal closure. This conclusion is supported by their finding that lateral wall motion was greater on the vowel [a] than on [i] with little movement on high vowels. This is contrary to what has been found for the velopharyngeal mechanism. The high correlation between movements of the velum and upper lateral pharyngeal walls has been noted by Skolnick (1969) and others.

In 1869 and again in 1969 Passavant (*87*, *88*) described a horizontal ridge on the posterior pharyngeal wall during velopharyngeal closure attempts in cleft palate patients. He assumed this to be a part of the normal mechanism for closure. Thus began approximately 100 years of debate over whether "Passavant's ridge" was part of normal velopharyngeal valving. It has been well documented (*11*, *27*, *51*, *127*), that this ridge is a compensatory mechanism seen not uncommonly in cleft palate but only rarely in persons with normal velopharyngeal mechanisms. In addition, it has been demonstrated that the ridge, when active, is not confined

to the posterior wall but is circular, extending via the lateral pharyngeal walls, to the sides of the velum (*15*, *123*).

Harrington (*53*), using cineradiography of the velopharyngeal mechanism, found that the degree of lateral wall movement in normal closure is directly related to the degree of velar elevation. This, and anatomical evidence to be presented later in this report, has led some investigators (*15*, *33*, *34*, *82*, *97*) to speculate that a single muscular mechanism may be responsible for both movements.

Muscular physiology: The muscles which have been thought to be responsible for velopharyngeal closure include the superior constrictor, levator, tensor, palatopharyngeus and salpingopharyngeus. Most authors have stated that at least two muscles (including various combinations of all of the above except salpingopharyngeus) are responsible for velar elevation. Lateral pharyngeal wall movement has been ascribed to salpingopharyngeus, superior constrictor, and/or levator.

Opinions regarding the specific muscles responsible for these patterns of movement are almost as numerous as authors who discuss them. Several researchers have reported results of electromyographic investigations of the palate and pharyngeal musculature in non-cleft human subjects. Li and Lundervold (*67*), Basmajian and Dutta (*7*), and Basmajian (*6*), used needle electrodes but were unable to specify specific muscles under study. Neither Harris and Schvey (*55*), Lubker and Curtis (*70*) nor Lubker (*69*) specify activity of individual muscles since they used surface electrodes on the velum and pharyngeal wall. Only two investigators (*31*, *45*, *46*) have studied a number of the muscles of the palate and pharynx simultaneously by electromyography. With surface suction electrodes Cooper studied various sites on the soft palate, and also sites on the posterior pharyngeal wall and anterior and posterior pillars. He found, "Most of the placements showed activity during speech, but only those on the posterior palate near midline gave all-or-nothing potentials that correlated with closure." Fritzell used needle electrodes to study levator, tensor, palatopharyngeus, palatoglossus, and superior constrictor during speech. While in his preliminary report (*45*) he reported tensor and levator active for speech, in 1969 he reported that only the potentials from superior constrictor and levator consistently correlated with closure. During speech, palatoglossus activity correlated with velopharyngeal opening.

In two studies by Rich (*94*, *95*) the concept of tensor palatini as a tensor (or levator) of the velum was denied. In this careful study involving nerve and muscle sectioning in animals and studies of human subjects with known cranial nerve lesions, Rich concluded that tensor's sole and unique function is to open the Eustachian tube. The conclusion that tensor is not related to velar motion is consistent with the experimental study of Fritzell (*46*). Bohme, Sram and Kalvodova (*14*) also studied EMG patterns of tensor and levator. Tensor activity was found during some phonatory attempts but only inconsistently.

Muscular anatomy: While studies of the muscles of the velum and pharynx date back to 1869, there is still disagreement about the anatomy of this system in modern texts. Major findings relevant to function will be reviewed. The most superior fibers of superior constrictor have been found to insert into the velum in the majority of dissections by Wardill (*123*), Whillis (*125*), Harrington (*53*, *54*) and Dickson and Dickson (*34*). They have speculated that these fibers may be responsible for Passavant's ridge. Dickson and Dickson (*34*) speculated that contraction of superior constrictor should draw its circumference (through the lateral and posterior pharyngeal walls) toward its most fixed attachment (the pterygoid plates). This action, they reason, is inconsistent with medial and posterior movement of the lateral pharyngeal walls seen during velopharyngeal closure. In addition, Harrington (*54*) reported that from his dissection of ten cadavers, all evidenced anterior attachment of superior constrictor at the hamulus rather than higher in the pterygoid fossa as presented in many text books. These findings taken with those of Perlman (*89*), Bloomer (*11*, *12*), Bosma (*15*), Calnan (*26*), Adams (*1*) and Ewanowski, *et al.* (*36*), all of which suggests that the site of lateral pharyngeal

wall motion is high in the nasopharynx at the torus tubarius, make it extremely unlikely that superior constrictor causes this motion in normal closure for speech. Swallowing, however, may involve a different mechanism, since pharyngeal activity occurs at a lower level (*106*).

Many authors of text books describe the salpingopharyngeus muscle as responsible for lateral pharyngeal wall movement and/or opening of the Eustachian tube. However, without exception, anatomical investigations of this muscle (*15, 30, 34, 78, 97, 113*), have revealed the muscle to be extremely inconsistent, often absent, and rarely of substantial size. The salpingopharyngeal fold has been found to be primarily glandular.

The anatomy of the tensor palatini muscle has been fairly well accepted for many years except for the site of its attachment to the Eustachian tube and its relation to the hamulus. On the basis of dissections and histologic examination of the tensor, Cleland (*30*), Körner (*64*), Simkins (*104*), and Aschan and Nylen (*3*), describe tensor as attaching only to the cartilaginous portion of the tube. Blakeway (*10*), McMyn (*78*), and Ross (*98*), describe attachments of tensor to both the cartilaginous and membranous walls. McMyn (*78*), Ross (*98*), Dickson and Dickson (*34*) and Rood (*96*) found the primary attachment to the tube to be to the lateral membranous portion. Ross (*98*), in a study of 52 Eustachian tubes by dissection and histologic examination, reported that in approximately ten percent of his cases tensor fibers attached to the maxillary tuberosity and in over one third of the cases there was attachment to the hamulus. There is also debate as to whether there exist common fibers of tensor palatini and tensor tympani (*3, 71*). Rood (*96*) demonstrated the continuity of fibers of tensor palatini and tensor tympani and also, on the basis of histologic and gross anatomic studies found tensor palatini to be composed of two distinct bundles separated by a fascial plane. The lateral bundle was described as attaching to the cranial base, pterygoid fossa and hard palate (as the velar aponeurosis) while the medial bundle (continuous with tensor tympani) arose from the lateral membranous wall of the Eustachian tube and joined the tendon of the lateral bundle inferiorly.

While the anatomy of the levator muscle is well agreed upon by those authors who have studied its structure (*10, 17, 30, 34, 64, 78, 99, 104, 121*) this agreement is not consistently expressed in current texts. It is apparent that levator passes inferior to, but without attachment to, the Eustachian tube; that it passes lateral to the torus tubarius prior to insertion into the velum; and that its velar insertion is broad, extending from the anterior third of the velum to the uvula. Anatomically it would be in position to displace the torus medially, posteriorly, and superiorly on contraction. There seems to be no anatomical evidence for the opinion suggested by many texts that levator lacks the proper vector to singularly accomplish normal velar movement (*105*).

There exists little disagreement among investigators regarding the anatomy of the palatopharyngeus (*10, 18, 34, 78, 83, 102, 125, 128*). Most of these investigators have emphasized the broad cephalocaudal insertion of the palatopharyngeus in the pharynx and intertwining of fibers of this muscle with fibers of superior constrictor. Townshend (*120*) and Negus (*83*) suggested that the sphincter fibers of superior constrictor entering the velum described by Whillis and others represent an inaccurate description. They stated that palatopharyngeus and superior constrictor are hopelessly intertwined in the adult pharynx, and that, by definition, fibers of the pharynx which enter the velum should be termed palatopharyngeus. The work of Dickson and Dickson (*34*), however, suggests that in the fetal pharynx these two muscles can be more clearly differentiated.

There has been little anatomical study of the palatoglossus (*4, 125*) or the uvulus muscle (*124*). Assis found no racial or sex differences in the anatomy of the palatoglossus among Brazilian Mulatos, Negroes and Whites.

Nerve supply: The nerve supply to the velopharyngeal musculature is described in modern texts as coming from the pharyngeal plexus. This plexus is not well understood but is generally considered to be supplied by cranial nerve IX and X, and also, according to some authors, from XII and from the sympathetic system. The exception to this arrangement is the innervation of tensor palatini which is thought to be from cranial nerve V. However, there is a great deal of disagreement. As Rich (*95*) points out in his historical review, up to 1920 the innervation of levator had been ascribed to cranial nerves, V, VII, IX, X, XI, and XII; while that of tensor had been ascribed to V, VII, X, and XI. Rich (1920b) in nerve degeneration studies on dogs, demonstrated that levator was supplied by the bulbar portion of XI. He also found, in dogs, that only stimulation of the mandibular branch of V caused contraction of tensor. He further reported a patient with unilateral paralysis of III, V, and VII, and bilateral paralysis of VI who demonstrated no velar disability but had unilateral Eustachian tube paralysis. Broomhead (*21*, *22*) conducted histological examinations of human material to determine the innervation pattern of the velum and pharynx. He, too, found tensor to be supplied by the mandibular branch of cranial V. He also found the uvular muscle to be innervated by the lesser palatine nerves (cranial IX), and the palatoglossus to be supplied by branches of cranial IX. Other muscles of the mechanism he found to be supplied by the pharyngeal plexus, made up of anastomosing branches of cranial IX and the pharyngeal branch of the vagus. Vidic (*122*) also found anastomoses of IX, X, and XI which supplied this area.

One point of confusion becomes apparent in any discussion of the Vagus nerve. Studies by Rich (*95*) and others have demonstrated that the Vagus contains cranial X and the bulbar portion of XI. Thus "Cranial X" and the "Vagus" are not synonymous. These studies also present evidence that the pharyngeal branch of the Vagus originates in bulbar XI. This same confusion has been evident in laryngeal research as will be pointed out later in this report.

Kanagasuntheran, Wong and Chen (*60*), presented human histological evidence that cranial IX is sensory to the velum. Winckler (*126*) found a generous supply of neuromuscular spindles in the human tensor muscle.

Blood supply: Except for the study of Broomhead (*21*) extensive research on the blood supply to the velopharyngeal mechanism prior to 1960 was not found in the current literature search. However, seven investigations since that time have been extensive and seem to have answered a number of questions, specifically about the arterial supply. In 1962, 1963, and 1964, Maher and Swindler (*72*, *73*, *74*) reported a series of studies of 78 normal human fetuses. They describe the distribution of the arterial supply from the greater palatine foramen in detail. They also illustrate the left-right anastomosis of these vessels. Girgis (*47*), in a study of "many" human palates described an arterial vessel which enters the uvular region of the velum via the posterior pillar. He points out that while all prior descriptions indicate that arterial supply to the velum comes from superior laterally, this vessel is an exception. He found this vessel to be, usually, from a separate branch of the external carotid, though sometimes it arose from the ascending pharyngeal artery and once from the lingual artery. In 1963, Zolotko (*129*) reported on a dissection of the arterial supply of 100 cadavers. He found that in 79 percent of his dissections the velar arterial supply was from the ascending palatine artery. In the other 21 percent, the supply was from the ascending pharyngeal artery. In either case, the artery divided into a muscular branch and a glandular branch. The latter, he found, anastomosed with the hard palate arteries. The course of each of the branches into the velum is presented in detail. Frederiks (*44*) reported on vascular patterns of the lip and palate in human embryos, and Fiamminghi and Gennari (*40*) found a rich blood supply to the hard palate mucosa and velum with

little crossing of midline. More extensive midline crossing was found in the velum than in the hard palate.

Cleft Palate

Patterns of movement: In terms of patterns of movement of the velum and pharynx, the presence of Passavant's ridge in many persons with cleft palate has already been documented. In his radiographic study of normal and cleft palate persons, Buck (*23*) found the cleft subjects to have a greater degree of posterior pharyngeal wall movement in speech than the non-cleft subjects. Knobeloch and Buck (*63*), however, in a similar study of Puerto Ricans found no difference in this measure. Hagerty and Hill (*50*) agreed with Buck. Carpenter and Morris (*29*) found Passavant's ridge to be consistent in those subjects who exhibited it and reported that at least in some cases the ridge seemed effective in aiding velopharyngeal closure.

Mazaheri, Millard and Erickson (*75*) studied normal and non-cleft subjects with velopharyngeal inadequacy cineradiographically. They reported the velum in the latter group to be short and thin in contrast to the normal. Hoopes, *et al.* (*57*), in another cineradiographic study, reported that in persons with submucosal clefts the levator eminence was more anterior than in the normal. In addition, they found that the eminence was even more anteriorly placed in persons with open clefts.

Isshiki, Honjow and Morimoto, (*59*) reported that there was a poor correlation between velar and lateral pharyngeal wall movement in their pre-pharyngeal flap patients. However, their data was difficult to interpret because of the angle they used in taking their radiograms.

Muscular physiology: Only three electromyographic studies of persons with cleft palate have been found in the literature (except for those related to the pharyngeal flap operative procedure which will be referred to later in this report). In 1969 Surina and Jagr (*116*) reported their study of tensor palatini and levator palatini. They concluded that the action potentials they observed, as well as histologic evidence they collected, demonstrated reduced contractile tissue and increased connective tissue in these muscles in persons with cleft palate. Broadbent and Swinyard (*20*) and Li and Lundervold (*67*) used needle electrodes to study cleft palate musculature, but neither could adequately specify which muscles were being recorded. In addition, Broadbent and Swinyard studied swallowing movements only.

Muscular anatomy: Only four studies of the anatomy of the cleft velum have been found. In 1960, Deuschle, *et al.*, (*32*) reported on dissections of an unreported number of full-term fetuses with cleft palate. They reported that tensor palantini could be described in three parts: an anterior part which attached to the scaphoid fossa and spread into the velum; a middle part which attached to the scaphoid fossa and lateral lamina of the Eustachian tube and descended to attach to the hard palate; and a posterior part from the angular spine and Eustachian tube cartilage to the palatine aponeurosis.

In 1967, Kriens (*65*) reported on his dissection of one newborn with cleft palate. He found, in general, that the musculature arose from normal attachments but was atypical in its insertions into the velum. He found fibers of both palato-pharyngeus and levator to attach to the hard palate at or in the margin of the cleft. He also found the most superior fibers of superior constrictor to enter the velum with some attaching to the hard palate. In addition, he reported that muscular fibers either from superior constrictor or palatopharyngeus attached to the hamulus. He found the tensor muscle to be hypoplastic.

Braithwaite and Maurice (*19*) also reported that their dissection of cleft palate revealed levator to insert into the edge of the hard palate. Most recently, Fara and

Dvorak (*38*) reported the results of autopsies of 18 unoperated clefts and 4 normal palates of mature stillborn children. While they mentioned the presence of isolated anatomical anomalies they also reported a series of consistent differences between cleft and non-cleft. Like Kriens they reported that the cleft muscles had normal origins, atypical insertions and were hypoplastic. They found palatoglossus, palatopharyngeus and the anterior part of levator to attach to the hard palate in the cleft margin. They also reported the presence of the circular fibers reported earlier by Whillis, presence of uvular muscle fibers on both sides of the cleft, well developed palatoglossal and palatopharyngeal muscles, and enlarged hamuli. They stated that the degree of hypoplasia of levator seemed correlated with the extent of the cleft.

It is interesting to note that all of these anatomical findings with the exception of the well developed palatoglossus by Fara and Dvorak (*38*) are consistent with the detailed descriptions of the cleft musculature presented by Veau (*121*) in 1931. Finally, there has been general agreement since the early work of Lushka (*121*) that the anterior aponeurosis is lacking in cleft palate. Further evidence of these findings was provided by Dickson (*33*).

Nerve supply: No specific investigations of the nerve supply to the cleft palate have been found. However, McCoy and Zahorsky (*76*) described the innervation of superior constrictor in five cadavers. They found small branches from cranial nerve IX and the ganglion nodosum joined one branch from the vagus which entered superior constrictor laterally.

Blood supply: No specific investigations of the blood supply to the cleft palate have been found except for Frederiks (*44*) who reported slight differences between cleft and non-cleft.

Pharyngeal flap: While surgical procedures and their results are outside the scope of this report, a number of studies of the mechanism of the pharyngeal flap are pertinent to this review since they shed light on the function of the velopharyngeal mechanism, particularly the role of the lateral pharyngeal walls. The discussion which has arisen in the literature has to do with whether motion of the flap as seen in lateral cineradiography is important to the success of the procedure. Unfortunately, the fact of correlation between flap motion and speech result has been interpreted as defining a causal relationship. Mechanically, the flap can serve only as an obturator since vertical movement of the flap cannot close the lateral ports around the flap. All of the research in this area has demonstrated that medial movement of the lateral pharyngeal walls is the active mechanism for closure. Morris and Spriestersbach (*81*) in 1967 found that mesial movement of the lateral walls was a better predictor of a good speech result than movement of the velum after pharyngeal flap surgery. Skolnick and McCall (*109*) found lateral wall motion to be essential for closure. Fara and Vele (*39*) found that while a superiorly based flap resulted in more atrophy and fibrosis than inferiorly based flaps, the superiorly based flap was more active and gave a better clinical result. They concluded that "the functional state of the flap did not have a decisive influence on palatopharyngeal closure." Still, concern about the functional status of the flap resulted from the work of Subtelny, *et al.* (*114*) who found reduced velar activity after pharyngeal flap surgery. McCoy and Zahorsky (*76*), on the premise that a flap must be functional to work, studied the innervation to the donor site. Their finding that the innervation of the flap would be sacrificed in the usual procedure led them to devise a new procedure even though they state that "the clinical improvement commonly observed from conventional flaps in patients must be explained on another basis (than the functional status of the flap)."

If the musculature which moves the velum (levator palatini) is also responsible for lateral wall motion (by levator's action on the torus tubarius) the correlation between flap motion and speech result is explainable since there would also be a

high correlation between velar and pharyngeal wall motion. In any case, it is apparent that lateral pharyngeal wall motion is the dynamic mechanism for closure for persons with pharyngeal flap.

Discussion

The need for further anatomical and physiological research in the areas of normal and cleft palate seems apparent from this review. With regard to normal patterns of movement, the specific nature of lateral pharyngeal wall involvement needs to be further delineated. The site of maximum motion relative to the torus tubarius needs particular attention. Detailed electromyographic investigation of all the muscles of this area is still necessary to resolve the conflicts apparent in the available literature. Work such as Fritzell's must be continued if we are to come to an understanding of this mechanism. However, this work must be preceded by or coincident with more detailed anatomical study of the normal mechanism. For example, there have been no studies of sex differences in the anatomy even though the work of McKerns and Bzoch (*77*) suggests that such differences may occur. There is little specific information on the developmental morphology of this musculature. Detailed studies of the palatopharyngeus, palatoglossus, and uvular muscles are almost non-existent. With regard to nerve supply there is still a need to sort out the pharyngeal plexus and the origins of the vagus nerve. This may be possible through embryologic and fetal studies.

Studies of the anatomy and physiology of the palate and pharynx of the person with cleft palate is not nearly as advanced as studies of the normal structure and function. Radiographic and motion picture studies of these movement patterns and their degree of variability needs to be pursued in a manner similar to studies of the normal. The field of electromyography of the cleft palate is practically virgin territory. While some very good information is available on the musculature of the cleft palate, more detailed information is necessary to indicate the degree of variability which should be expected with various types of cleft palate. In addition, no studies of the detailed anatomy of the Eustachian tubes of persons with cleft palate have been found. Nerve supply and blood supply in cleft palate have received very little attention.

In all of these areas of study more attention needs to be given to the investigation of relevant variables as well as control of known variables such as age, sex, race and type of cleft.

References

1. Adams, W. S., The transverse dimensions of the nasopharynx in child and adult with observations on its contractile function. *J. Laryngol. Otol.*, *72*, 465–471, 1958.
2. Almeida, H. R. de, Contribuição para o estudo da anatomia do "musculus stapedius" no homen. *Rev. Med. Cirur de São Paulo*, *1*, 275–298, 1941.
3. Aschan, P. E. and B. Nylen, The reconstruction of large defects in the palate or the cavity of the mouth using a tubed flap. *Trans. Internat. Soc. Plast. Surg.*, 219–220, 1955.
4. Assis, J. E. de Paula, Observações sobre os músculos glósso-palatine e amigdaloglósso, *Rev. Bras. Oto-Rino-Laring.*, *22*, 39–46, 1954.
5. Astley, R., The movements of the lateral walls of the nasopharynx: A cineradiographic study. *J. Laryngol. Otol.* (*London*), *72*, 325–328, 1958.
6. Basmajian, J. V., *Muscles Alive—Their function revealed by electromyography*. Williams and Wilkins, Baltimore, 1962.
7. Basmajian, J. V. and C. R. Dutta, Electromyography of the pharyngeal constrictors and levator palati in man. *Anat. Rec.*, *139*, 561–563, 1961
8. Benson, D., Roentgenographic cephalometric study of palatopharyngeal closure of normal adults during vowel phonation. *Cleft Palate J.*, *9*, 43–50, 1972.
9. Bjork, L. and B. Nylen, The function of the soft palate during connected speech. *Acta Chir. Scand.*, *126*, 434–444, 1963.
10. Blakeway, H., Investigations in the anatomy of the palate. *J. Anat. Physiol.*, *48*, 409–416, 1913.
11. Bloomer, H., Observations of the palatopharyngeal action with special reference to speech, swallowing and blowing. *Cleft Palate Bull.*, *2*, 4, 1952.

12. Bloomer, H., Observations on palatopharyngeal movements in speech and deglutiation. *J. Speech Hear. Dis., 18,* 230–246, 1953.
13. Bluestone, C. D., Eustachian tube obstruction in the infant with cleft palate. *Annals of Otol. Rhinol., Laryngol., 80* (Suppl. 2): 1971.
14. Bluestone, C. D., R. A. Wittel, and J. L. Paradise, Roentgenographic evaluation of Eustachian tube function in infants with cleft and normal palates (with special reference to the occurrence of otitis media). *Cleft Palate J., 9,* 93–100, 1972.
15. Bluestone, C. D., *et al.*, Certain effects of cleft palate repair on Eustachian tube function. *Cleft Palate J., 9,* 183–193, 1972.
16. Bohme, G. Von, F. Sram, E. Kalvodova, Elektromyographische untersuchungen uber das verhalten der mm. und tensor veli palatini bei der atmung und bei phonation von vokalen. *Folia phoniat., 18,* 9–10, 1966.
17. Bosma, J. F., A correlated study of the anatomy and motor activity of the upper pharynx by cadaver dissection and cinematic study of patients after maxillo-facial surgery. *Annals Otol., Rhinol., Laryngol., 62,* 51–72, 1953.
18. Bosma, J. F. and S. G. Fletcher, The upper pharynx. Part II. Physiology. *Annals Otol., Rhinol., Laryngol., 71,* 134–157, 1962.
19. Braithwaite, F., D. G. Maurice, The importance of the levator palati muscle in cleft palate closure. *Brit. J. Plast. Surg., 21,* 60–62, 1968.
20. Broadbent, T. R., C. A. Swinyard, The dynamic pharyngeal flap. Its selective use and electromyographic evaluations. *J. Plast. reconstr. Surg., 23,* 301–312, 1959.
21. Broomhead, I. W., The nerve supply of the muscles of the soft palate. *Brit. J. Plast. Surg., 4,* 1–15, 1951.
22. Broomhead, I. W., The nerve supply of the soft palate. *Brit. J. Plast. Surg., 10,* 81–88, 1957.
23. Buck, M., Velopharyngeal movements and tongue carriage during speech in adults with unrepaired incomplete cleft palate. *Cleft Palate Bull., 10,* 8–10, 1960.
24. Bzoch, K., Variations in velopharyngeal valving: The factor of vowel changes. *Cleft Palate J., 5,* 211–218, 1968.
25. Bzoch, K. R., T. M. Graber, T. Aoba, A study of normal velo-pharyngeal valving for speech. *Cleft Palate Bull., 9,* 3–4, 1959.
26. Calnan, J. S., Movements of the soft palate. *Speech* (London), *19,* 14–20, 1955.
27. Calnan, J. S., Modern views on Passavant's ridge. *Brit. J. Plast. Surg., 10,* 89–113, 1957.
28. Cancura, W., Der einfluss der binnenohrmuskulatur auf die schallubertragung im mittelhohr. *Monatssch. Ohrenheilk, 1,* 3–46, 1970.
29. Carpenter, M., H. Morris, A preliminary study of Passavant's pad. *Cleft Palate J., 5,* 61–72, 1968.
30. Cleland, On the question whether the Eustachian tube is opened or closed in swallowing. *J. Anat. Physiol. 3,* 97–103, 1869.
31. Cooper, F. S., Research techniques and instrumentation: E.M.G., proceedings of the conference: Communication problems in cleft palate. *Asha Reports, 1,* 153–168, 1965.
32. Deuschle, F. M., *et al.*, Soft palate muscular (*sic*), an anatomical study of full-term cleft palate fetuses using stereoscopic dissection microscope. *Cleft Palate Bull., 10,* 60–61, 1960.
33. Dickson, D. R., Normal and cleft palate anatomy. *Cleft Palate J., 9,* 280–293, 1972.
34. Dickson, D. R., W. M. Dickson, Velopharyngeal anatomy. *J. Speech Hear. Res., 15,* 372–381, 1972.
35. Donaldson, J. A., Physiology of the Eustachian tube. *Arch. Otolaryng., 97,* 9–12, 1973.
36. Ewanowski, S., W. M. Dickson, D. R. Dickson, A. B. Crummy, Lateral pharyngeal wall motion. Scientific exhibit. Amer. Cleft Palate Assoc. Convention, Chicago, 1971.
37. Falkson, The Functions of the soft palate and the pharynx. *Virchow's Arch., 79,* 477, 1880. (Summary: *Med. Rec.* 71, 1881.)
38. Fára, M., J. Dvorak, Abnormal anatomy of the muscles of palatopharyngeal closure in cleft palates. *Plast. reconstr. Surg., 46,* 488–497, 1970.
39. Fára, M., F. Véle, The histology and electromyography of primary pharyngeal flaps. *Cleft Palate J., 9,* 64–72, 1972.
40. Fiamminghi, L., U. Gennari, Sulla microvascolarizzazione sauguifera fetale umana della mucosa del palato. *Acta Bio-Medica, 42,* 3–20, 1971.
41. Fisch, U., G. V. Schulthess, Electromyographic studies on the human stapedial muscle. *Acta Otolaryng., 56,* 287–297, 1963.
42. Flisberg, K., *et al.*, The 'valve' and 'locking' mechanisms of the Eustachian tube. *Acta Otolaryng., supp. 182,* 42–68, 1963.
43. Frazer, J. E., The early development of the Eustachian tube and nasopharynx. *Brit. Med. J., 2,* 1148–1151, 1910.
44. Frederiks, E., Vascular patterns in normal and cleft primary and secondary palate in human embryos. *Brit. J. Plast. Surg., 25,* 207–223, 1972.
45. Fritzell, B., An electromyographic study of the movements of the soft palate in speech. *Folia Phoniat., 15,* 307–311, 1963.
46. Fritzell, B., The velopharyngeal muscles in speech. *Acta Oto-Laryngol., Suppl. 250,* 1969.
47. Girgis, I. H., Blood supply of the uvula and its surgical importance. *J. Laryngol. Otol., 80,* 397–402, 1966.
48. Graber, T. M., K. R. Bzoch, T. Aoba, A functional study of palatal and pharyngeal structures. *Angle Orthod., 29,* 30–40, 1959.

49. Griffith, B. H., *et al.*, Motion of the lateral pharyngeal walls during velopharyngeal closure. *Plast. reconst. Surg., 41,* 338-342, 1968.
50. Hagerty, R. F., M. J. Hill, Pharyngeal wall and palatal movement in postoperative cleft palates and normal palates. *J. Speech Hear. Res., 3,* 59-66, 1960.
51. Hagerty, R. F., *et al.*, Posterior pharyngeal wall movement in normals. *J. Speech Hear. Res., 1,* 203-210, 1958 (a).
52. Hagerty, R. F., *et al.*, Soft palate movement in normals. *J. Speech Hear. Res., 1,* 325-330, 1958 (b).
53. Harrington, R., A study of the mechanism of velopharyngeal closure. *J. Speech Hear. Dis., 9,* 325-345, 1944.
54. Harrington, R., M. pterygopharyngeus and its relation to m. palatopharyngeus. *Laryngoscope, 55,* 499-508, 1945.
55. Harris, K. S., M. M. Schvey, Component gestures in the production of oral and nasal labial stops. *J Acoust. Soc. Amer., 34,* 743, 1962.
56. Hilton, A large bony tumor in the face completely removed by spontaneous separation. *Guy's Hospital Report, 1,* 493-506, 1836.
57. Hoopes, J. E., *et al.*, The locus of levator veli palatini function as a measure of velopharyngeal incompetence. *Plast. reconst. Surg., 44,* 155-160, 1969.
58. Hynes, D. M., A radiological assessment of the oropharynx with special reference to tomography. *Clinical Radiol., 21,* 407-414, 1970.
59. Isshiki, N., I. Honjow, M. Morimoto, Cine-radiographic analysis of movement of the lateral pharyngeal wall. *Plast. reconst. Surg., 44,* 357-363, 1969.
60. Kanagasuntheram, R., W. C. Wong, H. L. Chan, Some observations on the innervation of the human nasopharynx. *J. Anat., 104,* 361-376, 1969.
61. Kelsey, C. A., T. J. Hixon, F. D. Minifie, Ultrasonic measurement of lateral pharyngeal wall displacement. *IEEE Trans Bio-Med. Engin., Apr.* 143-147, 1969.
62. Kevanishvili, Z. Sh., and Z. V. Guacharia, On the role of the tensor tympani muscle in sound conduction through the middle ear. *Acta Otolaryng., 24,* 231-239, 1972.
63. Knobeloch, C., M. Buck, Velopharyngeal movements and tongue carriage during speech in adults with unrepaired complete cleft palates. *Cleft Palate Bull., 10,* 10, 1960.
64. Korner, F., Die mm. tensor und levator veli palatini. *Z. Anat., 111,* 508, 1942.
65. Kriens, O., Anatomische untersuchungen am gespaltenen waich en gauemen. *Chir. Plast. Reconstr., 4,* 14-32, 1967.
66. Lawrence, M., The double innervation of the tensor tympani. *Ann. Otolaryn., 71,* 705-817, 1962.
67. Li, C., and A. Lundervold, Electromyographic study of cleft palate. *J. Plast. Reconstr. Surg., 21,* 427-432, 1958.
68. Linthicum, F. H., H. Body, Incidence of middle ear disease in children with cleft palate. *Cleft Palate Bull., 9,* 23-25, 1959.
69. Lubker, J. F., An electromyographic-cinefluorographic investigation of velar function during normal speech production. *Cleft Palate J., 5,* 1-18, 1968.
70. Lubker, J. F., and J. F. Curtis, Electromyographic-cinefluorographic investigation of velar function during speech production. *J. Acoust. Soc. Amer., 40,* 1272, 1966.
71. Lupin, A., The relationship of the tensor tympani and tensor palati muscles. *Ann. Otol. Rhinol. Laryngol., 78,* 792-796, 1969.
72. Maher, W. P., P. F. Swindle, Submucosal blood vessels of the palate. *Dent. Progress, 2,* 167, 1962.
73. Maher, W. P., P. F. Swindle, Mucosal blood vessels of the palate. *Dent. Progress, 3,* 106, 1963.
74. Maher, W. P., P. F. Swindle, Variations in the network of submucosal arteries in the human fetal palate. *J. Amer. Dent. Assoc., 69,* 106-111, 1964.
75. Mazaheri, M., R. Millard, D. M. Erickson, Cineradiographic comparison of normal to noncleft subjects with velopharyngeal inadequacy. *Cleft Palate J., 1,* 199-209, 1964.
76. McCoy, F. J., C. L. Zahorsky, A new approach to the elusive dynamic pharyngeal flap. *Plast. reconst. Surg., 49,* 160-164, 1972.
77. McKerns, D., K. R. Bzoch, Variations in velopharyngeal valving: The factor of sex. *Cleft Palate J., 7,* 652-662, 1970.
78. McMyn, J. K., The anatomy of the salpingopharyngeus muscle. *J. Laryngol. Otol., 55,* 1-22, 1940.
79. Minifie, F. D., *et al.*, Lateral pharyngeal wall movement during speech production. *J. Speech Hear. Res., 13,* 584-594, 1970.
80. Moll, K. L., A cinefluorographic study of the velopharyngeal function in normals during various activities. *Cleft Palate J., 2,* 112-122, 1965.
81. Morris, H. L., D. C. Spriestersbach, The pharyngeal flap as a speech mechanism. *Plast. reconstr. Surg., 39,* 66-70, 1967.
82. Moss, M. L., Normal and abnormal velopharyngeal anatomy. *Plast. reconst. Surg., 17,* 226-234, 1958.
83. Negus, V. E., The mechanism of swallowing. *Proc. Royal Soc. Med., 36,* 84-97, 1943.
84. Noone, R. B., *et al.*, The effect on middle ear disease of fracture of the pterygoid hamulus during palatoplasty. *Cleft Palate J., 10,* 23-33, 1973.
85. Paradise, J. L., C. D. Bluestone, Diagnosis and management of ear disease in cleft palate infants. *Trans. Amer. Acad. Ophthalmol. Otolaryngol., July-Aug.* 709-714, 1969.
86. Paradise, J. L., C. D. Bluestone, H. Felder, The universality of otitis media in 50 infants with cleft palate. *Pediat., 44,* 35-42, 1969.

87. PASSAVANT, G., *Ueber die Verschliessung des Schlundes Beim Sprechen.* J. D. Sauerlander, Frankfurt, 1863.
88. PASSAVANT, G., Ueber die verschliessung des schlundes beim sprechen. *Arch. Path., Anat., Physiol., 46,* 1, 1869.
89. PERLMAN, H. B., Observations on the Eustachian tube. *Arch. Otolaryngol., 53,* 370–385, 1951.
90. PETERSON, S. J., Velopharyngeal function: Some important differences. *J. Speech Hear. Dis., 38,* 89–97, 1973.
91. PIGOTT, R. W., The nusendoscopic appearance of the normal palato-pharyngeal valve. *Plast. reconst. Surg., 43,* 19–24, 1969.
92. PROCTOR, B., Anatomy of the Eustachian tube. *Arch. Otolaryng., 97,* 2–8, 1973.
93. PRUZANSKY, S., R. MASON, The "stretch factor" in soft palate function. *J. Dent. Res., 49,* 972, 1969.
94. RICH, A. R., The innervation of the tensor veli palatini and levator veli palatini muscles. *Johns Hopkins Hosp. Bull., 355,* 305–310, 1920 (a).
95. RICH, A. R., A physiological study of the Eustachian tube and its related muscles. *Johns Hopkins Hosp. Bull., 352,* 206–214, 1920 (b).
96. ROOD, S. R., A functional anatomic study of the muscles related to the Eustachian tube. Unpubl. Ph.D. thesis, University of Pittsburgh, 1972.
97. ROSEN, L., The morphology of the salpingopharyngeus muscle. Unpubl. M.S. thesis, University of Pittsburgh, 1970.
98. ROSS, M.E., Functional anatomy of the tensor palati—its relevance in cleft palate surgery, *Arch. Otolaryng., 93,* 1–3, 1971.
99. RUDING, R., Cleft Palate. Anatomic and surgical considerations. *Plast. reconst. Surg., 33,* 132–147, 1964.
100. SADÉ, J., Mucociliary flow in the middle ear. *Ann. Otol. Rhinol. Laryngol., 80,* 336–341, 1971.
101. SALOMON, G., A. STARR, Electromyography of middle ear muscles in man during motor activities. *Acta Neurol., 39,* 161–168, 1963.
102. SANVICHIEN, S., Palatopharyngeus muscle and elevation of the epiglottis after swallowing. *Anat. Rec., 136,* 271, 1960.
103. SHIMADA, T., D. J. LIM, Distribution of ciliated cells in the human middle ear. *Ann. Otol. Rhinol. Laryngol., 81,* 203–211, 1972.
104. SIMKINS, C. S., Functional anatomy of the Eustachian tube. *Arch. Oto-Laryngol., 38,* 476–484, 1943.
105. SIMPSON, R. K., A. A. AUSTIN, A cephalometric investigation of velar stretch. *Cleft Palate J., 9,* 341–351, 1972.
106. SIMPSON, W. L., J. E. WITCHER, A. study of the Eustachian tubes and their orifices and lumens in patients with large operative defects giving direct visualization. *Ann. Otol. Rhinol. Laryngol., 56,* 357–367, 1947.
107. SKOLNICK, M. L., Video velopharyngography in patients with nasal speech with emphasis on lateral pharyngeal motion in velopharyngeal closure. *Radiol., 93,* 747–755, 1969.
108. SKOLNICK, M. L. Videofluoroscopic examination of the velopharyngeal portal during phonation in lateral and base projections—a new technique for studying the mechanics of closure. *Cleft Palate J., 7,* 803–816, 1970.
109. SKOLNICK, M. L., G. N. MCCALL, Velopharyngeal competence and incompetence following pharyngeal flap surgery: A video-fluoroscopic study in multiple projection. *Cleft Palate J., 9,* 1–12, 1972.
110. SPRIESTERSBACH, D. C., *et al.,* Hearing loss in children with cleft palates. *Plast. reconst. Surg., 30,* 336–347, 1962.
111. STOOL, S. E., P. RANDALL, Unexpected ear disease in infants with cleft palate. *Cleft Palate J., 4,* 99–108 1967.
112. STRONG, L. H., Factors in nasopharyngeal occlusion. *Anat. Rec., 100,* 459–460, 1943.
113. STRONG, L. H., Muscle force components in the occlusive mechanism of the nasopharynx. *Anat. Rec., 106,* 510, 1949.
114. SUBTELNY, J. D., *et al.,* Pre- and post-operative analysis of velar and pharyngeal flap mobility. *Cleft Palate J., 7,* 748–762, 1970.
115. SUCHESTON, M. E., M. S. CANNON, Eustachian tubes of several mammalian species. *Arch. Otolaryng., 93,* 58–64, 1971.
116. SURINA, I., AND JAGR, Action potentials of levator and tensor muscles in patients with cleft palate. *Acta Chir. Plast., 11,* 21–30, 1969.
117. TERRACOL, J., *et al.,* Proprioceptive function of ossicular muscles. *Rev. Laryngol. Otol. Rhinol., 87,* 25–26, 1966.
118. THOMSEN, K. A., Investigations of Toynbee's experiment in normal individuals. *Acta Otolaryng., suppl. 140,* 263–268, 1958.
119. TOS, M., Growth of the foetal Eustachian tube and its dimensions. *Arch. Klin. Edp. Ohr. Nas. Kehlk, Heilk., 198,* 177–186, 1971.
120. TOWNSHEND, R. H., The formation of Passavant's bar. *J. Laryngol. Otol., 55,* 154–165, 1940.
121. VEAU, V., *Division Palatine.* Masson et Cie, Paris, 1931.
122. VIDIC, B., L'innervation du muscle releveur du voile du palais chez l'homme et chez certains mammiferes. *Arch. Anat. Histol. Embryol., 47,* 336–346, 1964.
123. WARDILL, W. E. M., Cleft palate—results of operation for cleft palate. *Brit. J. Surg., 16,* 127–148, 1928.
124. WARDILL, W. E. M., AND J. WHILLIS, Movements of the soft palate. *Gynecol. Obstet., 621,* 836–839, 1936.
125. WHILLIS, J., A note on the muscles of the palate and the superior constrictor. *J. Anat., 65,* 92–95, 1930.

126. WINCKLER, —., L'equipment nerveaux du muscle tenseur du voile du palais. *Arch. Anat.*, *47*, 313-316, 1964.
127. WOLFE, W. G., X-ray study of certain structures and movements involved in nasopharyngeal closure. Unpubl. M.A. thesis, University of Iowa, 1942.
128. WOOD-JONES, F., The nature of the soft palate. *J. Anat.*, *74*, 147-170, 1939.
129. ZOLOTKO, V. S., On the blood supply of the soft palate. *Stomatologia* (Moskva), *42*, 24-27, 1963.

Introduction

III. MAXILLARY GROWTH DEVELOPMENT

One of the most perplexing, yet fundamental problems encountered in the habilitation of patients with congenital cleft palate seems to be the effect of primary palatal surgery on subsequent growth and development of the craniofacial complex. The maxilla, for obvious reasons, has received the "lion's share" of attention in this regard in the literature, and therefore is the focus of the present literature review.

In 1949 Graber's (*9*) indictment of early and repeated traumatic surgery touched off a flurry of intense reaction and critical re-evaluation of surgical procedures being used. However, some of the research done since that time suffered from a shortcoming encountered frequently in this complex area of investigation, namely, lack of adequate controls. Any critical review of the literature with a comparison of the many diversified and oftentimes diametrically opposed findings apparent in this field necessitates stringent attention to all relevant variables. For this reason a review of methodological considerations will precede a review of research on normal and abnormal growth.

Methodological Considerations

Several aspects must be considered in any evaluation of maxillary growth. First of all the subject groupings must be judiciously constructed and tightly controlled with respect to several variables including race, sex, and type and extent of original defect. As suggested by Krogman and Sassouni (*12*), race must be considered in growth studies ". . . for there are structural (proportional) differences, size differences, and possibly growth-time differences based on cultural variables such as health, hygiene, diet, and so on." Sex differences in the growth and development of the maxilla have been documented by the work of Savara and Singh (*20*) using dental cast measurements. These studies demonstrate sex differences not only in maxillary growth timing and sequence but also in amount. Age, whether chronological, dental, or skeletal, also requires strict grouping in growth studies since growth itself is a function of time. As shown by Savara and Singh (*20*, *25*, *26*), increments in growth are constantly changing with chronological age, being most marked in the first few years postnatally, and circumpubertally. Growth changes associated with dentition and dental age are pointed out by Lebret (*14*), Sillman (*25*), and Richardson (*19*), and also serve to emphasize the need for underscoring the consideration of the type of age grouping used (chronological, dental, etc.). Lastly, cleft subjects must be classified as to type and extent of the original defect. To date, it seems to be fairly well established that cleft lip (with or without associated cleft palate) and isolated cleft palate are embryologically and genetically two different and independent disorders (*7*, *27*). These researchers fairly well agree that cleft lip (with or without cleft palate) occurs most often in males and is largely inherited, while isolated cleft palate occurs more frequently in females and is in-

frequently inherited, being influenced more by environment. In addition, Coupe and Subtelny (*4*) have shown that unilateral complete clefts, bilateral complete clefts, and isolated cleft palate, all differ significantly in the unoperated state in the amount of deficiency, and in the degree of displacement of palatal tissue. In their study using cephalometric laminography, all palatal cleft types showed a deficiency of palatal tissues, with bilaterals being most affected, then isolated clefts, and least of all unilaterals. The bilaterals also showed the greatest degree of displacement followed by the unilaterals and last by the isolated clefts. Also, within each of these categories of the cleft types, severity of the original defect should be considered. However, such subgroupings are obviously difficult because of the great individual variation in extent of defect from patient to patient.

A second consideration required in a critical evaluation of this topic is the method of analysis used by the researcher. One of the most common methods of analysis is radiography. In evaluation of research using cephalometric methods, however, one must be acutely aware of the myriad of possible sources of errors in interpretation, ranging from human misinterpretation in the tracing of oftentimes hazy and deformed structures (as in the case of ANS & PNS in clefts), to the choice of planes for orientation and superimposition. Krogman and Sassouni (*11*) report that one of the reasons for discrepancies in profile measurements from one study to another seems to be the selection of a reference place. Those methods using Frankfort Horizontal for reference tend to interpret the maxilla as more protrusive and the mandible as less retrusive, than methods using sella-nasion.

Another frequently used method of growth analysis is by measurements taken on dental casts. Toward this end various auxillary procedures have been incorporated, including sectioning the cast at desired levels and landmarks (*14, 18*), and photographing the cast to give a two-dimensional representation (*15, 28*). In comparing data using such methods, however, one must be aware of the need for identical standardized impression techniques, landmark choice, and the reliability of human interpretation of landmarks. Also comparison of such data with research done using roentgenographic cephalometrics should be done with caution because of the independence of skeletal, alveolar, and dental measurements in growth, as shown by Richardson and Brodie (*19*).

A final methodological point to be expressed here is the need for adequate and well-chosen non-cleft control subjects for comparison. Although the "ideal" normal control group would involve unaffected twins of the cleft patients, this is obviously not entirely feasible. Therefore, using the study by Swanson, McCollum, and Richardson (*30*) as a guideline, the comparison should not be made with an "ideal" normal, but rather with a randomly selected matched normal group which would include, in their proper proportions, the spontaneously and randomly occurring cases of skeletal Class I, Class II, and Class III jaw relations and other developmental differences naturally evident in normal population. This, of course, has its basis in the supposition that such differences will similarly occur in the cleft population regardless of the palatal defect (*3*).

Normal Growth Patterns

Before one can attempt to summarize aberrations in growth and development, a precise understanding of the normal processes is mandatory. This topic, specifically regarding the maxilla and craniofacial complex, has been investigated and discussed by many researchers. While a complete review of all investigations is beyond the scope of this paper, several salient points should be mentioned.

First of all, in spite of the importance of the topic of normal craniofacial growth and its apparent interest to so many fields, one striking finding is the lack of agreement and oftentimes even the espousal of diametrically opposed theories concerning

this basic topic. For instance, Craig (*5*) mentions that the greatest changes in facial growth occur in the vertical dimension, with anterior-posterior dimensions next, and breadth least. Krogman (*11*) Krogman and Sassouni (*12*), however, suggest that the greatest dimensional increases occur in the anterior-posterior direction, with height being second, and breadth last. As a second example, Brodie (*3*) suggests that the maxillary suture system is the primary impetus behind the downward component of growth and repositioning of the maxilla, with appositional growth at the tuberosity region supplying the forward component. Scott (*21*, *22*, *23*, *24*), in a convincing series of reports, sets forth his notion of the cartilage of the nasal septum being the prime supplier of both downward and forward aspects of maxillary movement while relegating the maxillary suture systems to a position of mere "fill-in" type of growth as a response to the septally induced separation of facial bones.

From the examples cited above it becomes increasingly evident that data concerning the identification, description, and study of maxillary growth aberrations will automatically be limited to an extent directly related to the degree of agreement and/or disagreement about the normal processes.

On the other hand, there does evolve from examination of growth research, a number of important details about which there seems to be sufficient conclusive evidence and general agreement to warrant mention: (1) in accordance with the ideas forwarded by Scott (*21*), the cartilagenous nasal septum, without question, seems to be intimately involved in maxillary growth and development in the first six years of life; (2) as described by Enlow and Bauq (*6*), appositional growth in the tuberosity region seems to be an important aspect of a-p development of the maxilla; (3) at least portions of Moss' "functional matrix" theory (*8*) appear to have relevance in relation to the necessity for the integrity of intact functioning intraoral, circumoral, and circumpharyngeal soft tissues in order to have concomitant normal maxillary development; (4) as evidenced by the research of Singh and Savara (*26*), Savara and Singh (*20*), and Lebret (*14*), the development of the maxillary alveolar process, and, correspondingly, increases in maxillary vertical dimensions are directly related to the presence and normal eruption of the teeth; (5) data by Sillman (*25*), Singh and Savara (*26*), Savara and Singh (*20*), Scott (*25*), Krogman (*11*), and Krogman and Sassouni (*12*) indicate that maxillary growth is extremely prolific from birth to age two or three and again circumpuberally, being about 90% complete by age seven, being largely remodelling, apposition, and deposition thereafter.

The above mentioned aspects of normal maxillary growth are but a few of the many findings on the subject, but seem to elicit a more general agreement among investigators and represent important features integral to the understanding of the literature concerning the effects of palatal surgery on maxillary growth.

Unoperated Cleft Palate Growth Patterns

As mentioned previously, accurate description of maxillary growth deficiencies in cleft palate patients presupposes a certain amount of foreknowledge of not only expected normal growth patterns, but also of cleft palate growth patterns uninfluenced by surgical intervention. There is obvious difficulty in obtaining such a population for study in this day and age of early and total treatment of cleft patients. Nevertheless, several studies have been reported.

Isolated Cleft Palate. In an early study of nasopharyngeal width, Subtelny (1955), using frontal cephalometric laminography at the level of the pterygomaxillary fissure, compared a random normal sample with a group of 91 unoperated cleft palate subjects, 29 of whom, ranging in age from birth to three years, had isolated cleft palate. Among other things, Subtelny found that the isolated cleft group (as well as unilaterals and bilaterals) all presented significantly larger width measurements than normals when structures close to the cleft (bihamular, and

between the tooth germs of maxillary first molars) were examined. While he found no difference from normals at the foramen rotundum or the zygoma, the birotundum measure may not be a valid expression of cranial growth. In addition, the pterygoid plates were found to be inclined laterally to a significantly greater degree than normal for isolated as well as all other cleft types. Of interest is the fact that these measurably wider dimensions compared to the normals were maintained over the age ranges studied.

There are two additional points of interest in Subtelny's work. First, the three cleft groups involving clefts of the hard and soft palate were not significantly different from each other. Secondly, the normals showed significant growth increases over the first two years but tapered off to statistical insignificance between years two and three. The cleft groups showed a similar growth pattern with the only differences reported being those of width.

In 1962, Innes (*10*) found an adult population of unoperated cleft palate persons in North Borneo which included four cases of posterior palatal clefting which he was able to compare to other adult members of the tribe by coronal and sagittal measurements taken on dental casts. These studies indicated to Innes that in clefts not involving the alveolus and hard palate, development was normal. However, his isolated cleft palate group had soft palate clefts only, and it is therefore difficult to generalize his thinking to include isolated cleft palate cases with soft and part of the hard palate affected. He found no differences in intermolar measurements or sagittal dimensions in the isolated cleft palate group. Thus, the maxillary growth of patients with unoperated isolated cleft of the soft palate seems to proceed normally, although no conclusions were reached as to what effect, if any, an isolated cleft including the hard palate would have.

Another study using frontal cephalometric laminography, this one through the most inferior point of the zygomatico-maxillary suture, was done by Coupe and Subtelny (*4*). The results of this work, mentioned earlier, indicate that the posterior cleft group of 40 subjects aged 1–3 years had greater width at this laminographic level than the bilateral cases but less than the unilaterals, and significantly less than a group of matched normals at all age levels. The authors state that their linear measurements from medial to lateral extent of the palatal shelves on frontal section demonstrate a deficiency of palatal tissue. However, it may be that this measure is demonstrative of a significant difference in transverse palatal configuration at this level as a result of lack of continuity of the oral structures. A second finding was that the posterior cleft subjects showed significantly laterally displaced maxillae as calculated by the width of the nasal cavity when compared to normals, although this width was again less than that shown by bilateral cleft subjects. Finally, no deviations were found in structures distant from the cleft, as reported in an earlier study (*29*).

Mestre, DeJesus, and Subtelny (*16*) examined the facial skeleton of a group of adult unoperated cleft patients, 22 of whom had isolated cleft palate. They used proportional and angular measurements, rather than linear dimensions, taken from lateral cephalometric tracings. They found no significant differences from normal in unoperated posterior cleft palate subjects in anterior-posterior and vertical chin position, proportional size of mandibular componants, anterior maxillary height, anterior position of the maxilla, posterior maxillary height, or maxillary body length expressed as a proportion of mandibular length. The same findings applied for unilateral cleft subjects. Thus the authors concluded that any growth deficiencies in operated posterior cleft groups must be a consequence of certain types of surgery.

Unilateral Complete Cleft Lip and Palate. Much greater mention of unoperated unilateral cleft lip and palate appears in the literature. Subtelny's 1955 report (*28*) demonstrated that the unilateral cleft subjects started life with width measurements in the area examined which were significantly greater than normal.

These were associated with laterally-inclined pterygoid plates. In addition, these subjects were found to have vomer inclination varied from perpendicular.

The study by Innes (*10*) describing the dental arches of North Borneo natives with unoperated clefts included a group of five with complete lip and palate clefts which was not specified as either bilateral or unilateral. Nevertheless, Innes found some ". . . degree of medial collapse of the lateral alveolar segment associated with diminished downward vertical growth of the alveolus." The deviations in width dimension seemed mainly limited to the canine region with the inter-molar measurements being apparently within normal limits. This group showed marked asymmetry in incisor to molar dimensions between the unaffected and affected side, the latter being less.

The subjects with unilateral complete clefts examined by Couple and Subtelny (4) showed significant deficiency of tissue as compared to normals, but nonetheless exhibited larger palatal shelf measurements than the other two cleft groups studied. Also, the unilateral group demonstrated significant lateral maxillary displacement but to a lesser degree than the bilateral group. Further, this displacement was marked only in the age group 0–1 years.

The data of Mestre, DeJesus, and Subtelny (*16*) contained a sample of 27 adult unoperated unilateral cleft palate subjects. However, most of these subjects had had surgical lip repair.

Ortiz-Monasterio, *et al.* (*17*) examined 18 Mexican subjects with unilateral clefts, four of whom had been operated. The Frankfort-facial angle was found to be comparable to normal standards. On the other hand, Frankfort-mandibular and convexity angles were said to be larger than normal. No tests of statistical significance were reported. Also, this Mexican sample was compared to normative data collected on North American subjects. In a later study, Ortiz-Monasterior, *et al.* (*18*) attempted to tighten the controls and eliminate some of the variability of the aforementioned research. They examined a group of unoperated cleft subjects, 42 of whom had complete unilateral clefts. They compared their results to data on normal Mexican adults. In essence, their new results coincided with those they obtained seven years previously.

Latham and Burston (*13*) examined histologic sections of the circum-maxillary suture system of a newborn infant with a complete unilateral cleft and compared it to similar sections from a normal infant. Of importance in this study was Latham's finding that the zygomaticomaxillary sutures of the cleft infant seemed to be the site of a hinge or rotary-type mechanism which was swinging the lower half of the separate maxillae downward and laterally, compressing the facial half of this suture, while the upper or orbital half moved correspondingly medially separating the orbital part of the suture and stimulating bone deposition there. This effect was noticeably greater on the non-cleft side and was exactly opposite to that found in the normal infant. Latham suggested that the nasal septum attached only to the non-cleft side exerts an imbalanced growth force driving the non-cleft maxilla laterally and inferiorly and initiating compensatory growth at the sygomaticomaxillary suture. Further, the cleft side, separated from the influence of the septum, would show a corresponding decrease in its growth. Thus, the infero-laterally inclined maxillae seen in unilateral subjects, and the asymmetry mentioned by Subtelny (*29*) and Coupe and Subtelny (*4*) seem to be given support. This study also implied a degree of support for the theories of Scott (*21*) regarding the importance of the nasal septum in maxillary growth and development. Latham and Burston (*13*) found no growth imbalance in the palato-pterygo-maxillary suture system.

Finally, Atherton (*1*) used descriptive morphology to analyze the maxillary development of 17 skulls with complete unilateral unoperated clefts. The specimens ranged in age from birth to old age. Although based on subjective evaluation, his findings lend support to the previously mentioned data. First, infant specimens exhibited marked laterally inclined maxillary bones and pterygoid-plates, as shown

by Subtelny (*29*) and Couple and Subtelny (*4*), which seemed to become more normal in older specimens as implied by Innes (*10*), Mestre, DeJesus, and Subtelny (*16*), and Ortiz-Monasterio, *et al.* (*17, 18*). Atherton (*1*) also found, in agreement with the observation of Latham and Burston (*13*), a slightly decreased size of the cleft-side maxilla in all three dimensions. Third, Atherton observed normal or greater than normal vertical growth on the cleft side (in areas other than the canine region) in spite of its lack of continuity with the nasal septum. This was contrasted to less vertical development of the non-cleft side, an occurrence perhaps related to the deviant direction of the vomero-septal structures. In general, these data appear to support earlier findings of deviations at adulthood mainly limited to the cleft site with normal development of the rest of the maxilla.

Bilateral Complete Cleft Lip and Palate. The unoperated complete bilateral cleft has received little thorough investigation. However, many of the investigations already reviewed contained bilateral subjects.

Subtelny's (*29*) 1955 laminographic study contained 13 unoperated complete bilateral subjects. His findings for these were similar to those of the other groups, namely, significantly wider nasopharynges and more laterally inclined pterygoid plates than normal. The bilaterals were not significantly different from either unilaterals or isolated cleft palate groups in this respect.

Nineteen bilateral cases were included in the 1960 work by Couple and Subtelny (*4*). Within the limits of the author's interpretations of the findings these subjects exhibited a deficiency and displacement of hard palate tissue which not only was significantly greater than normal at all three age intervals, but also was greater than either of the other two cleft palate groups.

The second paper of Ortiz-Monasterio, *et al.* (*18*) presented data on a sample of 14 adult complete bilateral subjects. The cephalometric measures used apparently revealed normal growth and development in these cases, although the objective data from the bilateral subjects was not grouped separately from other cleft types. Subjective clinical evaluation, however, prompted the authors to point out a severely underdeveloped columella and prolabium in all cases with a prominently positioned and unstable premaxilla. The lateral segments in these cases were described as presenting lateral external displacement yielding increased transverse dimensions. These data seem to suggest that with no repair of the lip the premaxilla continues to grow forward maintaining its anterior position into adulthood, while the lateral segments demonstrate no spontaneous medial collapse or under-development in spite of their lack of continuity with the nasal septum. On the contrary, their increased transverse dimension, continued into adulthood, implies that the bilaterals do not show the same degree of normalizing tendencies with age as did the unilateral and isolated cleft palate subjects.

In one final piece of research examining bilateral clefts only, Boo-Chai (*2*) clinically and subjectively evaluated the maxillary development of 27 adult subjects with bilateral clefts, 12 of which were completely unrepaired, 10 of which had lip repair only, and the remaining 5 of which had lip repair with surgical extirpation of the premaxilla. According to the author, the first group, in general, showed prominent forward growth of the premaxilla and good anterior growth of the lateral maxillary segments to give a "fairly good profile." Normal transverse molar relationships were found, but some degree of medial collapse of the lateral segments behind the premaxilla in half the cases, and anterior open bite in the vicinity of the cleft, were reported.

Discussion

It is apparent that further work is needed in order to resolve existing differences of opinion regarding the mechanisms of normal maxillary growth. In addition, as in other areas of research, much more work is needed in order to understand the growth disturbances associated with various types and extents of cleft palate. For example,

there is need for longitudinal growth studies, data on subjects between three years and adulthood, and a great need for better systems of classification of clefts on some other basis than superficial appearance, so that meaningful comparative studies can be completed.

reprints: *Craniofacial Anomalies Program*
National Institute of Dental Research
National Institute of Health
Bethesda, Maryland 20014

References

1. Atherton, J. D., Morphology of facial bones in skulls with unoperated unilateral cleft palate. *Cleft Palate J.*, *4*, 18-30, 1967.
2. Boo-Chai, K., The unoperated adult bilateral cleft of the lip and palate. *Brit. J. Plast. Surg.*, *24*, 250-257, 1971.
3. Brodie, A. G., Behavior of normal and abnormal facial growth patterns. *Amer. J. Orthod.*, *27*, 633-647, 1941.
4. Coupe, T. B., J. D. Subtelny, Cleft Palate—deficiency or displacement of tissue. *Plast. reconst. Surg.*, *26*, 600-612, 1960.
5. Craig, R. D. P., The management of complete clefts of the lip and palate. *Brit. J. Surg.*, *54*, 923-931, 1967.
6. Enlow, D. H., S. Bang, Growth and remodeling of the human maxilla. *Amer. J. Orthod.*, *51*, 446-464, 1965.
7. Fögh-Anderson, P., Cleft lip and palate: Remarks on etiology of harelip and cleft palate with special reference to heredity. *Trans. Intern. Soc. Plast. Surg.*, 156-160, 1955.
8. Grabb, W., S. W. Rosenstein, K. R. Bzoch (Eds.), *Cleft Lip and Palate*. Little, Brown, and Co., Boston, 1971.
9. Graber, T. M., Craniofacial morphology in cleft palate and cleft lip deformities. *Surg. Gynec. Ob.*, *88*, 359-369, 1949.
10. Innis, C. O., Some preliminary observations on unoperated hare-lips and cleft palates in adult members of Dusan tribes of North Borneo. *Brit. J. Plast. Surg.*, *15*, 173-181, 1962.
11. Krogman, W. M., The problem of the cleft palate face. *Plast. reconst. Surg.*, *14*, 370-375.
12. Krogman, W. M., V. Sassouni, *Syllabus in Roentgenographic Cephalometry*. College Offset, Philadelphia, 1957.
13. Latham, R. A., W. R. Burston, The effect of unilateral cleft of the lip and palate on maxillary growth pattern. *Brit. J. Plast. Surg.*, *17*, 10-17, 1964.
14. Lebret, L., Growth changes of the palate. *J. Dent. Res.*, *4*, 1391-1404, 1962.
15. Mazaheri, M., *et al.*, Changes in arch form and dimensions of cleft patients. *Amer. J. Orthod.*, *60*, 19-32, 1971.
16. Mestre, J. C., J. DeJesus, J. D. Subtelny, Unoperated oral clefts at maturation. *Angle Orthod.*, *30*, 78-85, 1960.
17. Ortiz-Monasterio, F., *et al.*, Cephalometric measurements on adult patients with non-operated cleft palates. *Plast. reconst. Surg.*, *24*, 53-61, 1959.
18. Ortiz-Monasterio, F., *et al.*, A study of untreated adult cleft palate patients. *Plast. reconst. Surg.*, *38*, 36-41, 1966.
19. Richardson, E. R., A. G. Brodie, Longitudinal study of growth of maxillary width. *Angle Orthod.*, *34*, 1-15, 1964.
20. Savara, B. S., I. J. Singh, Norms of size and annual increments of seven anatomical measures of maxillae in boys from three to sixteen years of age. *Angle Orthod.*, *38*, 104-119, 1968.
21. Scott, J. H., The cartilage of the nasal septum. *Brit. Dent. J.*, *95*, 37-43, 1953.
22. Scott, J. H., The growth of the human face. *Proc. Royal Soc. Med.*, *47*, 91-100, 1954.
23. Scott, J. H., Growth at facial sutures. *Amer. J. Orthod.*, *42*, 381-387, 1956.
24. Scott, J. H., Further studies on the growth of the human face. *Proc. Royal Soc. Med.*, *52*, 263-268, 1959.
25. Sillman, J. H., Dimensional changes of the dental arches: longitudinal study from birth to 25 years. *Amer. J. Orthod.*, *50*, 824-842, 1964.
26. Singh, I. J., B. S. Savara, Norms of size and annual increments of seven anatomical measures of maxillae in girls from three to sixteen years of age. *Angle Orthod.*, *36*, 312-324, 1966.
27. Spriestersbach, *et al.*, Clinical research in cleft lip and cleft palate. *Cleft Palate J.*, *10*, 113-165, 1973.
28. Stockli, P. W., Application of a quantitative method for arch form evaluation in complete unilateral cleft lip and palate. *Cleft Palate J.*, *8*, 322-341, 1971.
29. Subtelny, J. D., Width of the nasopharynx and related anatomic structures in normal and unoperated cleft palate children. *Amer. J. Orthod.*, *41*, 889-909, 1955.
30. Swanson, L. T., D. W. McCollum, S. O. Richardson, Evaluation of the dental problems in cleft palate patient. *Amer. J. Orthod.*, *42*, 749-765, 1956.

NOTE: PART 2 OF THIS REPORT WILL BE PUBLISHED IN THE JANUARY, 1975 ISSUE OF THE CLEFT PALATE JOURNAL.