The Craniofacial Complex in Cleft Lip and Palate: An Updated Review of Anatomy and Function

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This report has been prepared for the purpose of updating the previous State-of-the-Art reports on the status of research on the *anatomy* and *physiology* of *cleft lip* and *palate* (Dickson, et al., 1974, 1975; Maue-Dickson, 1977). It covers the literature from August, 1976 to August, 1978, on middle ear musculature, the auditory tube, the velopharyngeal mechanism, the lip, mandible, tongue, nose, and larynx. It also includes a review of the literature from August, 1972, through August, 1978, on the growth and development of the *craniofacial skeleton.* Significant advances have been made in our understanding of the velopharyngeal mechanism and auditory tube. Further investigation is needed on the innervation and blood supply to the velopharyngeal mechanism and on the effects of age, race, and sex on the development of the craniofacial skeleton in both normal and cleft-palate individuals. There is a continued need for interdisciplinary research.

Introduction

In 1971, the American Speech and Hearing Association was awarded a contract by the National Institute of Dental Research for the purpose of reviewing the status of a number of areas of research pertaining to cleft palate. The committee designated to accomplish this enormous task subsequently published its summary report (Spriestersbach et al., 1973) and a number of comprehensive reports, each covering specific areas of research. Among the comprehensive papers were those of Dickson et al. (1974, 1975). These reviewed the status of research in anatomy and physiology related to cleft palate from approximately the sixteenth century to 1972. In 1973, the American Cleft Palate Association established a committee to update the ASHA/NIDR report through August of 1976. This updated committee report was published in 1977 (Fletcher et al.) and included a section on the status of research in anatomy and physiology (Maue-Dickson, 1977).

The current material has been compiled for the purpose of updating the previous two states of the art on the status of research in the anatomy and physiology of cleft lip and palate. The section on growth and development of the craniofacial skeleton covers the period from August of 1972 to August of 1978. All other sections cover the period from August of 1976 to August of 1978. In addition, a small number of articles published prior to August of 1972 and August of 1976 respectively but not reviewed in the previous states of the art have been included. As in the previous reports, the current review includes normal, abnormal, and selected developmental studies of middle ear musculature, the auditory tube, the velopharyngeal mechanism, the tongue, larynx, facial musculature, and blood supply and innervation relevant to cleft lip and palate. In addition, the current review includes a section on craniofacial growth and development.

The author has attempted to remain faithful to the intent of the preceding two reports: (1) to review in depth the present status of research on cleft lip and palate anatomy and physiology; (2) to identify areas of missing or ambiguous information and unresolved controversies; (3) to identify areas which need no special research emphasis; and (4) to discuss approaches to provide additional information in those areas in need of further development.

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Middle Ear Musculature, the Auditory Tube, and the Velopharyngeal Mechanism

A. Middle Ear Musculature and the Auditory Tube. The status of research on the reflex activity of the tensor tympani and stapedius muscles remains very much as it was in August of 1976. At that time, the consensus among the authors whose studies were reviewed indicated a close association between stapedial contraction and acoustic stimuli and between tensor tympani contraction and nonacoustic stimuli. This association has been neither definitely confirmed nor challenged in the research literature during the past two years.

The role of the tensor tympani muscle in auditory tube function, however, has received increasing attention. As indicated by Dickson (1974, 1975), there is evidence that the tensor tympani muscle has fibers which originate from the tensor veli palatini muscle and that the two muscles share a common innervation. Dickson hypothesized that the two muscles might act together in auditory tube clearance by direct action on the tube (tensor veli palatini contraction) and by simultaneous action on the malleus (tensor tympani contraction) to adjust middle ear pressure. Since the two muscles share a common innervation and are separated from one another by an intermediate tendon, they might function as a two-bellied muscle unit. To investigate this hypothesis, Kamerer (1978) conducted an electromyographic study of the correlation of tensor tympani and tensor veli palatini muscle function in thirty human subjects undergoing surgery for conductive hearing loss. Among the findings was that the tensor tympani muscle consistently responded during swallowing and that the two tensor muscles had similar latency response times.

The probable functional association between the tensor tympani and tensor veli palatini muscles is supported by their anatomical relationship. Rood and Doyle (1978) investigated this relationship in three normal human fetuses and in six normal adult cadavers and demonstrated that the lateral fiber bundle of the tensor veli palatini muscle attaches superiorly to the tendon of the tensor tympani muscle as well as to the scaphoid fossa, the spine of the sphenoid bone, and the entire lateral osseous ridge of the sphenoid sulcus. These fibers were found to originate lateral and perpendicular to the auditory tube along its entire course from the osseous-cartilaginous junction to the nasopharyngeal orifice. The medial, or dilator tubae, fibers of the tensor veli palatini muscle attached to the membranous wall of the auditory tube, became tendinous inferiorly, rounded the hamulus, and terminated on the posterior margin of the palatine bone and in the anterior velum.

The possible association between the levator veli palatini muscle and auditory tube function, challenged in the preceding two states-of-the-art, has not received much attention in the recent literature. Only one report (Lavorato and Lindholm, 1977) mentioned the levator muscle with reference to the auditory tube. The authors of this study dissected the levator muscle in several normal adult cadaver heads and confirmed the absence of levator muscle fiber attachment to the tubal cartilage.

The recent literature shows a continued interest in the mechanisms underlying auditory tube dysfunction. Holborow (1975) noted that developmental changes in the specific morphology of the auditory tube may be associated with the decreasing liability of children to acute otitis media with increasing age. Unfortunately, the author presented no definitive data, and the validity of his conclusions cannot be evaluated. Jonas et al. (1978) conducted a cephalometric study of children with auditory tube dysfunction but without hearing impairment and suggested that children with "a vertical craniofacial growth pattern" may be predisposed to poor tubal function. Tos (1971) and Bak-Pedersen and Tos (1972) have suggested a possible association between an increased number of goblet cells and active infection within the auditory tube. Romanczuk et al. (1978) have hypothesized that secretory otitis media could result from a mucociliary-transport defect. The latter study is of particular interest since it demonstrated that mucin concentrations in children with cleft palate are significantly higher than necessary for normal tubal clearance, an observation consistent with the hypothesis that impaired clearance is a major factor in the retention of ear effusions. A study reported by Pulec et al. (1978) suggests that dysfunction

of the lymphatic system may also be involved in the genesis of secretory otitis media. The authors utilized histologic sections, light microscopy, and transmission electron microscopy to assess the status of auditory tube lymphatics in 107 specimens removed from fresh human cadavers during necropsy and have provided the first definitive description of the topography and distribution of the lymphatic capillaries of the human auditory tube. Most significantly, the authors have hypothesized that secretory otitis media with infected or inflamed tonsils could be the result of obstruction of the lymphatic capillaries rather than the mechanism often causally explained, i.e., that these tissues physically obstruct the tubal orifice.

In summary, the literature published between August of 1976 and August of 1978 has provided more definitive information on the specific morphology of the muscles of the auditory tube, the function of the tensor tympani and tensor veli palatini muscles, and the specific cellular morphology of the auditory tube. Data presented by Kamerer (1978) suggests that the equalization of pressure between the middle ear and nasopharynx may be accomplished via reflex contraction of the tensor tympani and tensor veli palatini muscles. Morphologic data indicate that, during this process, the medial fibers of the tensor veli palatini muscle are probably of particular importance in the displacement of the lateral membranous wall of the auditory tube (Rood and Doyle, 1978).

No new studies of the role of the middle ear musculature in response to acoustic vs. nonacoustic stimuli have been found. Several studies have suggested the need for further study of the cellular morphology of the auditory tube (Tos, 1971; Bak-Pedersen and Tos, 1972; Pulec et al., 1975; Romanczuk et al., 1978). One study (Jonas et al., 1978) has suggested that certain craniofacial growth patterns may be associated with auditory tube dysfunction. Further quantitative research is needed on the possible anatomical, developmental, biomechanical, and cellular factors responsible for auditory tube malfunction associated with cleft palate.

B. THE VELOPHARYNGEAL MECHANISM. The previous States-of-the-Art indicated a critical need for further anatomical and physiological research in normal and cleft areas to resolve questions regarding normal patterns of velopharyngeal motion, the level of motion relative to the torus tubarius, the role of specific muscles of the velum and pharynx in velopharyngeal closure, sex differences in velopharyngeal anatomy and physiology, developmental morphology of the velopharyngeal mechanism, and the specific innervation and blood supply of the muscles of the velopharyngeal mechanism. It was also pointed out in those reviews that research data in these areas is far more deficient in cleft palate than in the normal condition.

It seems obvious that definitive study of the velopharyngeal mechanism necessitates morphological analysis of its individual components as well as exploration of its function as a dynamic coordinated and integrated complex in both normal and abnormal conditions. The literature in the last two years has brought to our attention several excellent descriptions of the morphology and function of selected muscles of the velopharyngeal mechanism as well as some interesting methodological studies for the further investigation of the dynamic velopharyngeal mechanism.

In 1975, Dickson reviewed the anatomy of the velopharyngeal mechanism and the auditory tube and presented a series of speculations based strictly on anatomical considerations. He observed that, in histologic sections of a limited number of early human fetuses, the uvulus muscle was the most superior muscle of the velum and was a paired structure. Based on its location and configuration in the velum, Dickson speculated that contraction of the uvulus muscle could result in a humping of the third quadrant of the velum and produce what has been called the "levator eminence".

In 1976, Langdon and Dickson studied the morphology of the uvulus muscle in normal human fetuses of approximately 15 weeks gestational age. They concluded that the uvulus muscle in the human fetus in characteristically spindle-shaped and courses longitudinally through the posterior three-quarters of the velum superior to the bulk of the palatoglossus, palatopharyngeus, and levator muscles. Though the uvulus was observed to be bifid at one or both ends in nearly half of the specimens studied, the authors found no in-

dication that it was a true bilateral or paired structure. In all but one specimen, attachment of the uvulus to the hard palate occurred via separate tendons, the palatal aponeurosis, or less organized connective tissue pathways. In only one instance did the uvulus fibers extend as far anteriorly as the hard palate.

In a follow-up study, Azzam and Kuehn (1977) analyzed histologic sections of the uvulus muscles from seven normal adult cadaver heads and found that, in all specimens studied, the musculus uvulus was a true paired structure composed of two discrete muscle bundles separated by a median septum. The separate bundles coursed posteriorly and converged medially, approximating each other as cylindrically-shaped bundles in an area overlying the levator sling. In agreement with Dickson (1972) and Langdon and Dickson (1976), Azzam and Kuehn observed that the most anterior fibers had no direct attachment to the hard palate, that within the velum the uvulus muscle organized itself into smaller bundles or fascicles, that the anterior portion of the velum was void of muscle tissue, and that the uvulus muscle somewhat disorganized in the area of the uvula proper. However, in disagreement with Dickson, Azzam and Kuehn found that the first observable muscle fibers posterior to the hard palate were identifiable as the most anterior fibers of the uvulus muscle. Both studies provide substantial anatomical support for the earlier hypothesis that the so-called "levator eminence" is produced by the uvulus muscle.

Several functional studies of the velopharyngeal mechanism in cleft palate subjects reported during the past two years have also provided substantive empirical support for the latter hypothesis. In 1976, Shprintzen *et al.* published a combined multi-view videofluoroscopic, panendoscopic, and nasendoscopic study of 100 subjects with hypernasal speech. They found that, in 11% of the subjects studied, velopharyngeal insufficiency was due to the presence of small midline gaps between the velum and the posterior pharyngeal wall. The gap was typically triangular in shape and two to five millimeters in width. The lateral walls were observed to move medially to the lateral edges of the velum. While the authors did not comment on the underlying dysmorphology of this central velar gap, it would not be inconsistent with hypoplasia or absence of the uvulus muscle.

In 1978, Croft et al. reported a combined nasendoscopic and videofluoroscopic study which further elucidated the nature of these central velar gaps. The authors studied 20 patients with central velar gaps, hypernasal speech, and palatal morphology which appeared normal on oral examination. Gap size ranged from 1.0 $mm.^2$ to 12.0 $mm.^2$ with a mean of 5.0 $mm.^2$. All subjects in this group exhibited an absence of the muscular uvulus bulge on the nasal surface of the velum. On videofluoroscopic examination, 14 of the 20 subjects exhibited normal patterns of velopharyngeal closure in lateral view, though air bubbles in the barium coating of the nasopharynx indicated velopharyngeal insufficiency. Central velar gaps were subsequently identified in these subjects with frontal and base view films. The authors compared these subjects with 20 normal subjects, all of whom exhibited complete velopharyngeal closure during connected speech and a clearly-defined uvulus bulge during velopharyngeal closure for speech. It is significant that no subjects in either group exhibited abnormalities of the lips, dental arches, hard palate, velum, or uvula and that all subjects in both groups exhibited good velar and lateral pharyngeal wall mobility. This well-designed definitive study lends support to the functional importance of the uvulus muscle in normal velopharyngeal closure for speech and to the hypothesis that the V-shaped defect described in some patients with hypernasality probably represents either absence or hypoplasia of the uvulus muscle. It is also of interest to note that the authors found that all of the subjects with central velar gaps had been misdiagnosed prior to nasendoscopic and videofluoroscopic evaluation; the misdiagnoses ranged from palatal paresis to congenital pharyngeal insufficiency to disproportion to idiopathic hypernasality and even to hysterical conversion reaction.

Only one study on the anatomy of the velopharyngeal musculature in cleft palate has been found in the current review. Long et al. (1976) studied the velopharyngeal musculature in a five-month-old human cadaver with an unoperated cleft of the palate. Their three-dimensional reconstructions of serial

transverse and coronal histologic sections demonstrated convergence of the fibers of the levator, palatopharyngeus, and uvulus muscles to form a compact bundle which inserted into the posterior nasal hemispine and the medial margin of the bony cleft palate. In addition to these aberrant insertions, the authors found that other fibers of these muscles terminated in a reduced dome-shaped palatal aponeurosis formed by the tendon of the tensor veli palatini muscle after it had rounded the hamulus. The superior constrictor muscle was also found to contribute fibers to the velar musculature. Its anterior fibers inserted into the lateral part of the palatal aponeurosis. Finally, the authors found a small but distinct salpingopharyngeus muscle arising from the auditory tube cartilage bilaterally. Their findings serve to confirm the overall deficiency of tissue in the velum and to define further the specific abnormal course of the various muscles of the velum.

The question of which muscles are primarily responsible for normal velopharyngeal closure has continued to be a subject of interest during the past few years. The dissimilar biomechanical potential of the levator veli palatini and the superior constrictor muscles, plus the observation that the lateral walls move posteriorly and medially during phonation, led Dickson (1972) to hypothesize that the levator muscle alone is responsible for normal velopharyngeal closure in speech and that the lateral wall motion which contributes to velopharyngeal closure occurs at the level of the torus tubarius. Subsequent studies conducted by Bell-Berti (1973, 1975) provided physiologic evidence in support of this hypothesis. However, several other studies cited in the previous review (Skolnick et al., 1973; Shprintzen et al., 1974; Shprintzen et al., 1975; Zagzebski, 1975) presented evidence in opposition to this hypothesis. The latter studies described maximum medial excursion of the lateral pharyngeal walls during speech as occurring at the level of the full length of the velum and palate, well below the "levator eminence" and below the level of the torus tubarius. The issue remained unresolved.

In 1976, Honjo, Harada, and Kumazawa studied the role of the levator muscle in lateral pharyngeal wall motion in three adult patients whose nasopharynges were directly visible after surgical removal of the nose or the maxilla and in four normal adult cadaver heads. The living subjects each participated in two test sequences, one radiographic and one cinematographic. For the radiographic portion of the study, the authors pasted lead markers on each torus tubarius just below the pharyngeal orifice of the auditory tube at the point where lateral pharyngeal wall motion was most marked. By the use of these markers and contrast media, they were able to observe the entire velopharyngeal area in frontal radiographs of the pharynx taken during rest and phonation. They then filmed velopharyngeal motion during the production of plosive, fricative, and nasal syllables and subjected the films to frame-by-frame analyses. The most significant of their findings were that the motion of the lateral pharyngeal walls during speech occurs most prominently at the torus tubarius, not in the salpingopharyngeal fold descending from it, and that the amount of lateral wall motion is directly related to the extent of velar elevation. They were also able to demonstrate close synchrony between lateral-wall and velar motion. The authors' analysis of the relationship between the levator muscle and the torus tubarius in cadavers indicated that, from a mechanical point of view, contraction of the levator sling would produce a medial displacement of the torus tubarius, which lies just medial to the levator sling. The authors concluded that medial movement of the lateral pharyngeal wall is caused by contraction of the levator muscle and that the primary site of the movement is at the torus tubarius. Their findings are in agreement with the hypotheses published by Dickson in 1972.

In 1976, Bell-Berti conducted an electromyographic study of the velopharyngeal musculature in three normal adult subjects. Speech samples consisted of nonsense words composed of vowels, stop consonants, and nasal consonants to form various stop-nasal and nasal-stop contrasts. Palatopharyngeus showed consistent activity for each of the subjects, although the activity was greater for open than for closed vowels. The superior constrictor and middle constrictor contributed relatively little to closure, and the middle constrictor was strongly active only for vowel production, particularly for open vowels. The

palatoglossus was found to be active for tongue body movements and for pharyngeal narrowing but not for palatal lowering. The strength of levator EMG potential was highly correlated with velar height and was greater for closed than for open vowels. Velopharyngeal port opening was accomplished by suppression of the activity of the levator muscles and not by increased activity in any other muscle. Thus, the data collected in this study provide support for the theory that velopharyngeal closure for speech is achieved primarily by contraction of the levator muscle, that velopharyngeal closure is not a binary on/off mechanism, and that the pharyngeal constrictors are primarily active during pharyngeal cavity adjustments involving variations in vowel quality. This does not contradict the possible primary role of the superior constrictor in velopharyngeal closure for non-speech activities. As indicated in the previous review, Minifie et al. (1974) demonstrated electromyographically that the constrictor muscles are more active during swallowing than during speech production. However, since there are still two strong schools of thought on this issue, additional data are necessary to further elucidate the questions and resolve the existent controversies.

While the evidence which has accumulated during the past few years appears to be remarkably consistent regarding the role of the pharyngeal constrictors in normal velopharyngeal closure for speech, there has been considerable debate over the role of these muscles in individuals with cleft palate. The superior fibers of the superior constrictor muscle are of particular interest since it is these fibers which appear to be responsible for the formation of Passavant's ridge. Data presented by Honjo et al. (1975) suggest that this ridge is of little significance in compensation for velopharyngeal inadequacy. The authors examined the ridge, marked with radioopaque contrast material, radiographically and cineradiographically in ten adult cleft palate patients. Their data indicate that the height of the ridge varies with specific vowels and that the formation of Passavant's ridge is not associated with the degree of velopharyngeal closure necessary for the production of specific speech sounds but is closely associated with tongue position for vowel production. These data suggest that, like in normal subjects (Bell-Berti, 1976), the pharyngeal constrictors in individuals with cleft palate may be involved in pharyngeal cavity adjustments during the production of vowels. The hypothesis that Passavant's ridge lies too low to be effective as a compensatory mechanism in cleft palate has not been addressed in the recent literature.

In 1977 Shprintzen et al. studied velopharyngeal imcompetence resulting from absent lateral pharyngeal wall motion in the presence of velar motion in two patients and absent velar motion in the presence of lateral pharyngeal wall motion in three patients. The authors suggested that, if velar motion is controlled by levator activity while pharyngeal wall motion is controlled by the superior constrictor, then incongruous motions of the velum and lateral pharyngeal walls are readily explicable. However, the incongruous motions could not be explained by acceptance of the hypothesis that the levator muscle is responsible for both velar motion and lateral pharyngeal wall motion. This reasoning would appear to support the contention by Shprintzen and others that velopharyngeal closure for speech is a function of both the levator and superior constrictor muscles. However, it should be noted that abnormalities of the levator muscle, known to exist in cleft palate, may preclude normal patterns of velopharyngeal function. It is not indicated how many of their subjects had cleft lip and/or palate, how many had surgically repaired palates or pharynges or how many, if any, had palatopharyngeal scarring or other anomalies.

In 1977, Lavorato and Lindholm reported a fiberoptic nasendoscopic study of velopharyngeal activity in one normal adult male and in one normal adult female. Both speech and non-speech activities were observed. The torus tubarius was seen to move in a postero-superomedial direction and, during maximum displacement, nearly approached midline. It was most mobile during swallowing, blowing, and the production of speech sounds which demanded high intraoral pressure; it was also mobile during oral and nasal breathing, and during the production of nasal sounds and /a/ but to a lesser degree. The authors noted that their observations demonstrated the mo-

bility of the torus tubarius and that such movement was intimately related to activity of the levator veli palatini muscle. The authors also observed that, during swallowing, velopharyngeal activity began at the level of the oropharynx rather than in the nasopharynx and that, in general, activity for blowing and swallowing occurred much lower in the pharynx than did such activity for speech. In the male subject, lateral pharyngeal wall motion was described as occurring at the level of the torus tubarius and, in the female subject, just below the level of the tip of the torus. The authors concluded that activity of the levator at both levels generated movement of the lateral pharyngeal wall during velopharyngeal activity. Subsequent dissections done by the authors on male and female cadaver heads demonstrated that the levator muscle lay in an appropriate position to act upon either the torus tubarius or the torus-tubarius, salpingopharyngeal-fold complex.

An interesting study on the effect of changes in velopharyngeal orifice area on vowel intensity has been presented in the recent literature by Bernthall and Beukelman (1977). The authors utilized prosthetic appliances positioned in the velopharynx to study the influence of changes in velopharyngeal orifice size on vowel intensity in normal speakers when subglottal air pressure was maintained at specific levels. They discovered that the subjects were able to produce a relatively consistent subglottic pressure in the presence of velopharyngeal orifices of different sizes while visually monitoring a subglottic pressure trace on an oscilloscope. A reduction in vocal intensity (dB SPL) was observed as velopharyngeal orifice area was increased from the control condition to the large experimental orifice condition when subglottic pressure was monitored at a specified level. The magnitude of the SPL decrements recorded in this experiment ranged from 1.9 dB to 4.1 dB. The authors indicated that SPL reductions of this magnitude reflect very significant losses in sound energy, and they hypothesized that a speaker may compensate for this loss of sound energy by increasing the output from the source (the larynx). Previous studies have indicated that a 5 dB increase in vocal intensity may require more than a 50 per cent increase in subglottic pressure. As indicated by the authors, the increased effort required to drive the vocal system in order to effect an adequate level of sound energy provides the potential for vocal abuse. This study provides strong support for the cautions put forward by McWilliams, et al. (1969), who advised close scrutiny for any sign of hoarseness in children with velopharyngeal valving difficulties. Bernthal and Beukelman indicate that a clinician should be aware of the sizeable increase in vocal effort and the potential for laryngeal abuse in children with inadequate velopharyngeal mechanisms when increased vocal intensity is an instructional goal.

Several studies specifically aimed at the improvement of methodology for the definition of velopharyngeal function or pathology have been published in the recent literature. Cotton and Quattromani (1977) have suggested the addition of supine videofluoroscopy in the Towne projection to diagnostic evaluations for the purpose of improving preoperative diagnostic procedures aimed at defining the degree of lateral pharyngeal wall motion in velopharyngeal closure. It is difficult, as of this writing, to see what this would contribute that is not available by other more usual means. Hawkins and Swisher (1978) have described the quality of information obtainable from an ultrasound system with specific relevance to clinical judgements of lateral pharyngeal wall motion. While they concede that videoradiographic techniques still provided more total information, the study which they conducted on three adults with ultrasound indicated that the quality of information from an ultrasound system could be of sufficient value to make clinical judgments regarding lateral pharyngeal wall motion. As indicated by the authors, ultrasonography has the potential for providing the same information as videoradiographic techniques without the disadvantage of exposing the patient to irradiation. Beery (1976) compared the speech profiles of 33 subjects with palatal clefts or suspected velopharyngeal insufficiency or both with viedotape fluoroscopy, pressure-flow analysis, and cephalometrics. His findings suggested that, while each technique provides valuable information under specified circumstances, at least two of the techniques should be employed for the best evaluation. He indicated that cephalometrics

yielded the most questionable results, particularly in borderline cases. Finally, Michel et al. (1978) described the use of lateral plane and cineradiographic x-rays, palatal tomography, audiometry, and tympanometry in the diagnosis of children with speech and hearing abnormalities. Those children in whom the question of a palatal defect was raised also underwent trispiral palatal tomography. The authors found that nine of the 12 patients selected for tomography demonstrated a palatal defect that was not detected by other means; the defect was located in the posterior hard palate in the region of the posterior nasal spine. The authors used the CGR Stratomatic tomographic unit and stated that "12 cuts were usually sufficient". As correctly noted by the authors, the primary problems often associated with submucosal clefts of the palate are hypernasality and serous otitis media, both of which can be diagnosed without the aid of tomography. It is difficult to see how the methodology suggested by the authors can improve patient care, add significantly to presurgical diagnostic information, or change the outcome of treatment and planning. The disadvantages of excess radiation would seem to far outweigh the advantages of the procedure.

Only two studies on the innervation of the palatopharyngeal complex were found in the current review of the literature. Domenech-Ratto (1977) studied the development and peripheral innervation of the palatal muscles in ten human embryos and fetuses. The authors found that the tensor veli palatini muscle is innervated by a branch of the mandibular nerve, that the nerve fibers to the levator and palatopharyngeus muscles come from the glossopharyngeal and vagus nerves, and that the only nerve branches observed to penetrate the uvulus muscle come from a plexus formed at the expense of the posterior palatine nerve. The author was careful not to deny the possibility that the fibers which arrive at the levator and palatopharyngeus muscles may originate with the accessory nerve, but, at least peripherally, the nerves are separate. In the case of the palatopharyngeus muscle, the nerve fibers proceeding from the vagus nerve were observed to enter the muscle body without passing through any nerve plexus. This was the only study of human innervation of the velopharyngeal complex found in the current review.

The only other study of palatal innervation found in the recent literature was that of Ibuki et al. (1978), who described the course of the facial nerve innervation for the levator veli palatini muscle in ten rhesus monkeys. The authors utilized recordings of evoked EMG responses of the levator muscle and of the orbicularis oris muscles by electrical stimulation to both the facial nerve and its branches within the cranium. The authors concluded that the course of the facial nerve to the levator veli palatini muscle is through the greater petrosal nerve. Since this is a study of subhuman primates, its equivalence in the human levator muscle would need to be demonstrated.

Only one study of the human palatal blood supply was found in the recent literature. Maher (1977) conducted a landmark study of the distribution of the palatal and other arteries in cleft and non-cleft human palates in 12 near-term human fetuses, three with cleft palate and nine without. The specific arterial branches described by the author include the pathways and ramifications of the greater palatine, posterosuperior alveolar, infraorbital, and facial arteries in near-term human fetus. These descriptions deserve to be read in their entirety. Maher noted in particular that the major branches of the arteries are commonly present on both sides but that there are numerous variations in each facial half in both the cleft and non-cleft-palate groups. He concluded that the theoretical considerations proposed originally by Spriestersbach and his co-workers (1973) and by Dickson et al. (1975) that there may be variability in arterial arrangement associated with cleft lip and palate has now been demonstrated.

In summary, the literature in the past two years has added significant new data on the morphology and function of specific musculature of the velopharyngeal mechanism, on patterns of velopharyngeal closure in the normal and cleft condition, and on the innervation and blood supply to specific parts of the velopharyngeal mechanism.

The current consensus on the morphology of the uvulus muscle is that it typically has no direct attachment to the hard palate, that it

is subdivided into smaller bundles or fascicles, that its major bulk occurs in the third quadrant of the velum and that it is probably the uvulus muscle and not the levator veli palatini which is positioned appropriately for the production of the so-called "levator eminence" (Dickson, 1975; Langdon and Dickson, 1976; Azzam and Kuehn, 1977). There is still disagreement about whether the uvulus muscle is paired (Azzam and Kuehn, 1977) or unpaired (Langdon and Dickson, 1976). The disparity may reflect developmental differences, since the former study was conducted on adult subjects and the latter on fetuses. The central velar gaps described by Shprintzen et al. (1976) and by Croft et al. (1978) strongly suggest hypoplasia or absence of the uvulus muscle. This is completely consistent with the morphology of the uvulus muscle as described by Dickson (1975), Langdon and Dickson (1976), and Azzam and Kuehn (1977). It is apparent that the broadened applications of nasopharyngoscopy and the recent advent of the small-diameter flexible nasopharyngoscope have had an enormous and positive impact on both research and clinical studies of the velopharyngeal mechanism. It is of particular significance that nasopharyngoscopy has been demonstrated to provide direct full-color visualization of the velopharyngeal port and that, in many instances, it can provide adequate diagnostic information without the need for additional procedures involving radiation.

The studies of Honjo et al. (1976), Bell-Berti (1976), and Lavorato and Lindholm (1977) taken together provide strong support for the thesis that the levator veli palatini muscle is the primary activator in normal velopharyngeal closure for speech and that the levator produces synchronous lateral wall and velar displacement during this function. These data also support the thesis that maximum lateral wall motion does occur at the level of the torus tubarius. However, additional studies of this mechanism are in order.

Incongruous motion of the velum and lateral pharyngeal walls in velopharyngeal closure for speech has been described in conditions of abnormal velopharyngeal closure (Shprintzen et al., 1977) and may indicate significant functional differences between normal and abnormal patterns of velopharyngeal closure. The effectiveness of Passavant's pad as a compensatory mechanism for velopharyngeal inadequacy in cleft palate has been challenged by Honjo et al. (1975).

Data presented by Bernthall and Beukelman (1977) suggest a functional and pathological relationship between velopharyngeal incompetence and subglottic pressure. Their data indicate that small increments in vocal intensity may require enormous increments in subglottic pressure and may be associated with vocal abuse.

Several studies reported in the recent literature suggested altered approaches in methodology for the diagnosis of velopharyngeal dysfunction. Cotton and Quattromani (1977) suggested the inclusion of the Towne projection for definition of lateral wall motion videofluoroscopically; Michel et al. (1978) suggested the addition of trispiral tomography for the dectection of submucosal clefts. Both approaches clearly involve the use of additional irradiation of the subject for access to diagnostic information which can be gained in alternative ways without the necessity for increased radiation. Beery (1976) suggested the use of two of three standard techniques (videofluoroscopy and air pressure-flow measurements) for maximal efficiency in the diagnosis of speech profiles of individuals with cleft palate or suspected velopharyngeal insufficiency.

Only two studies of the innervation of muscles of the velopharyngeal mechanism have appeared in the recent literature. Both described the innervation of the levator veli palatini muscle, one in man (Domenech-Ratto, 1977) and one in the rhesus monkey (Ibuki et al., 1978).

Maher (1977) has contributed a landmark study of the blood supply to the palate in normal human fetuses and in fetuses with cleft palate and has demonstrated variability in arterial patterns in both cleft and non-cleft-palate human fetuses.

Thus, while there have been significant advances in our information on the structure and function of the velopharyngeal mechanism in both the normal condition and in cleft palate, further study is still needed in the following areas: the anatomy and physiology

of the normal velopharyngeal valving mechanism, the nature and degree of difference in patterns of velopharyngeal closure in normal and cleft-palate individuals; developmental changes in the morphology of the velopharyngeal mechanism and associated structures and possible functional and clinical implications of these changes; and the innervation of the muscles of the velopharyngeal mechanism.

The excellent endoscopic and morphologic studies of the uvulus muscle evident in the recent literature have resulted in a major breakthrough in our recognition and understanding of central velar gaps. They have also led certain groups to suggest defect-specific correction of these gaps, such as midline injections of teflon paste (Shprintzen et al., 1979) and the surgical construction of narrow superiorly-based pharyngeal flaps (Dickson and Maue-Dickson, 1977). While both procedures have produced good functional results to date and have simplified correction of the defect, recent personal communication with Shprintzen (1978) has documented the abandonment of teflon injections into the velum because of potentially serious or lethal complications, such as the inadvertent injection of teflon into the blood stream.

Our ability to quantify the functional potential of the velopharyngeal mechanism in the cleft palate population is still in need of further study. A major problem in this regard is the apparent extreme variability in the degree of functional insult resulting from clefts of the palate.

It is encouraging to note that the recent literature suggests that a great deal of cross-fertilization of ideas is occurring in the study of velopharyngeal structure and function; the dialogue and debate evident in the literature indicates a very healthy and active concern for the solution of many of the questions still surrounding our understanding of the velopharyngeal mechanism. It is also encouraging to note that many of the research questions pursued recently reflect needs stated in previous states-of-the-art.

The Lip and Prolabium

A number of interesting studies of the lip and prolabium have appeared in the recent literature. Lehman and Artz (1976) have published a report on minimal clefts of the lip. They have noted that the deformity is usually more extensive than it first appears to be, that the skin overlying the area is often devoid of hair follicles and sweat glands, that the medial side of the lip is displaced relative to the lateral side, and that, beneath the skin groove, there is a break in the continuity of the lip musculature which can be confirmed histologically.

Rees et al. (1975) examined the contents of the prolabium via tissue biopsies from 18 patients with cleft lip. Sections prepared from these biopsies indicated that muscle tissue was absent or markedly diminished in all cases and that normal muscle bundle architecture was not present in any of the samples studied. The authors also collected electromyographic recordings from the prolabia of four subjects with secondary bilateral clefts. None showed normal action potential, indicating that the motor units in these prolabial segments were either sparse or absent. The authors observed that incomplete clefts of the lip are characterized by relatively more muscle tissue than are complete clefts. The absence of anatomically and functionally significant muscle in the prolabium in bilateral clefts of the lip was also observed earlier by Duffy (1971), who found that muscle tissue in the philtrum probably comes exclusively from the lateral segments in normal development.

In 1976, Ehmann et al. examined qualitative and quantitative differences in the tissues surrounding unoperated clefts of the lip and palate in an attempt to identify the anatomical and topographical characteristics of these differences. Photographs, panoramic radiograms of the maxilla, and study models collected prior to surgery indicated a close relationship between the degree and condition of the cleft and the extent of the vermilion, the number of tooth buds adjacent to the cleft, the form and size of the alveolar borders, and the number and type of incisors. The authors found that cleft edges were typically symmetrical in isolated clefts of the palate.

In 1972, Franz and Sokol studied philtral width in 40 normal subjects ranging in age from birth to four months. They derived an equation for determination of the proper philtral width for bilateral cleft lip repair based on head circumference, intercanthal distance, commissural distance, and philtral width.

They calculated the mean of philtral width to be 7.0 mm. and, interestingly, found no significant differences between philtral widths in the newborn group and the one-to-four month-old group.

In summary, while there is not an extensive new body of information in this area, there have been several studies which have documented the type and extent of tissue deficiencies associated with cleft lip.

Anatomy and Development of the Mandible, Tongue, Nose, and Larynx

Only two studies relevant to the function of the masticatory muscles have been found in the recent literature. Kubota and Masegi (1977) studied the distribution of muscle spindles in the masticatory muscles in one eight-month human fetus and in one newborn cadaver and found a dramatic concentration of spindles in the elevator muscles of the mandible (temporalis, masseter, medial pterygoid, and lateral pterygoid). As stated by the authors, this would suggest a high degree of proprioceptive control of the extensive mobility of the human temporomandibular joint. The authors also found that the temporalis muscle contained 342 spindles, 65.7 per cent of the sum total on one side of the face, and suggested that this muscle may play an important role in the maintenance of condylar posture during mandibular movements.

One additional finding by Kubota and Masegi was that the muscle spindles of the human masticatory muscles studied consisted of four to six intrafusal muscle fibers, one or two nuclear bag fibers, and two to eight nuclear chain fibers. A definite connective tissue capsule surrounded the intrafusal muscle fibers. The vast majority of the spindles (98.8 per cent) were located in the elevator muscles while only a few (1.2 per cent) were located in the depressors. The authors also observed that the human masticatory muscles appear to be richer in muscle spindles than do those of other primates. While the principal objective of the study was to determine muscle spindle distribution and density, the authors also presented detailed descriptions of the topographic arrangements of each of the jaw muscles.

Yemm (1977) utilized needle electromyography, tension recordings with a forced transducer, and surface EMG to assess the recruitment of motor units of the masseter and temporalis muscles during voluntary isometric contractions. The data, collected from three normal adults, strongly suggested a mechanism of orderly recruitment, with no regional differences in the distribution of units within the relatively large muscles studied. The relationship between force threshold or unit size and contraction time was found to be weak, at least in those human muscles studied by the authors thus far.

Only two studies on the tongue have been found in the recent literature. Cohen and Vig (1976) studied the growth of the tongue and intermaxillary space in 25 male and 25 female subjects ranging in age from four to 19 years. The authors' hypothesis was that if soft tissues influence the position of the teeth, then growth changes in the tongue are also likely to be of significance. Cephalometric data collected by the authors indicated that lingual growth curves in the female subject resembled somatic growth curves, while the male subject showed a much later slowing of lingual growth relative to somatic growth. The authors also observed that the tongue becomes relatively larger during growth in relation to the intermaxillary space. The second study of the human tongue (Barnwell et al, 1978) described the morphology of the superior longitudinal muscle in histologic sections from 28 normal human fetuses of approximately 15 weeks gestational age. The authors described the muscle as a bilateral fiber stratum lying just beneath the dorsal lamina propria in the anterior two-thirds of the tongue. They were able to identify its origin at the lamina propria along the sulcus terminalis and its termination on the superior aspect of the median septum. They also provided a detailed description of its complex interrelationships with other lingual muscles and presented evidence that the dorsal muscular stratum of the human fetal tongue consists of both intrinsic and extrinsic fiber groups.

The only study of the larynx found during the current review was on the postnatal descent of the epiglottis in man (Sasaki et al., 1977). The authors described the descent of the epiglottis in 15 subjects between birth and 18 months of age. Between the ages of one and six months, they found that the epiglottis

and the soft palate lay in contact in five of the subjects, indicating a functional relationship characteristic of the newborn. Other subjects demonstrated wide separation of the larynx and nasopharynx at the age of six months. The larynx was observed to rise to contact the nasopharynx only during deglutition. The authors also observed that the larynx and nasal cavities remained interlocked during respiration surrounding the feeding period but separated during the act of crying and during subsequent respiratory efforts. The three radiograms of infants 12 to 18 months of age all suggested the establishment of laryngeal-velar separation. The authors also observed that, in this age group, palatal-epiglottic contact was variably accomplished during deglutition, while, during tidal respiration, the larynx and nasopharynx appeared separated and remained disengaged during both vocalization and crying. The authors concluded that the age group from four to six months seems to represent a transitional period from obligate nasal breathing to potential oral tidal respiration, a transition of particular interest because it is peculiar to man and also because it seems to reflect a period of potential respiratory instability which coincides with the peak incidence of sudden infant death syndrome. Data analyzed by the authors were obtained from cineradiographs obtained for clinical purposes unrelated to the project reported.

One study on the morphology of the nasal cartilages and one on the normal patterns of growth sites of the nasal septum have been found in the current review. De Lara Galindo et al. (1977) studied the morphology and interrelations of the nasal cartilages in 11 full-term human fetuses and seven adults. The authors found that the septodorsal cartilage was made up of one piece. Their data confirm previous assertions that the lateral cartilages lie under the nasal bone at their junction and not in the end-to-end relationship often described. The histologic survey conducted by the authors demonstrated that the lateral cartilages join the major alar cartilages by fusion, overlapping, or rolling to form one functional unit. The attachment of the septodorsal cartilage to the nasal bone takes place from two to five millimeters in the inner aspect of the latter cartilage.

In 1977, Melsen described the normal pattern of the growth sites of the nasal septum according to age and sex in nasoseptal tissue blocks from 66 male and 57 female cadavers ranging in age from zero to twenty years. The increase in size of the vomer appeared to result from apposition on the superior surface and on the posterior margin. After the ethmoid bone was ossified, the connection between the vomer and the perpendicular plate of the ethoid was a slight sinuous suture in which growth occurred until puberty. This growth pattern implied that a forward, downward sliding of the vomer must have taken place in relation to the ethmoid bone and the cartilaginous septum. Finally, the author found that a cartilage island lay embedded in the vomer after the establishment of the suture between the vomer and the perpendicular plate; this cartilage still persisted in specimens from adults. The author concluded that there is no reason to believe that the septal cartilage plays a major role in the forward, downward growth of the maxillary complex in man.

In summary, very few studies on the anatomy and developmental morphology of the mandible, tongue, larynx, and nose have been reported in the recent literature, and none of these studies has related directly to the cleft-palate individual. Kubota and Masegi (1977) have provided important data on the topographical details of the muscle spindle supply to the muscles of mastication. Data on the growth of the tongue and intermaxillary space has been provided by Cohen and Vig (1976). Barnwell et al. (1978) have provided a detailed description of the morphology of the superior longitudinal muscle in the fifteen-week human fetus. The study reported by Sasaki et al. (1977) on the descent of the epiglottis in man is one of the few studies which has provided a rational explanation of the change from obligatory nasal breathing to oral breathing. Finally, Melsen (1977) and De Lara Galindo et al. (1977) have provided important new data on the normal morphology of the human nose from birth to early adulthood.

Further study of the anatomy and developmental morphology of the mandible, tongue, larynx, and nose is clearly needed. The previous state-of-the-art reviews have indicated an urgent need for further definitive study of these areas in association with cleft palate. This need remains urgent.

Growth and Development of the Craniofacial Skeleton

It is encouraging to note that the literature from August of 1972 to August of 1978 reflects a growing body of increasingly more definitive information on the growth and development of the craniofacial skeleton in both normal individuals and in those with cleft lip and/or palate. These studies span age groups from birth through late adult life and include both cross-sectional and longitudinal data on specific morphologic features of the craniofacial complex, on age differences, race differences, and sex differences. They also bring to our attention new perspectives on the pre- and post-surgical status of the craniofacial complex.

In addition, there is a separate but equally interesting body of new literature on human embryologic and fetal growth and development and on animal studies which are relevant to our understanding of the structure and function of the craniofacial complex in cleft lip and palate. The latter two categories have not been included in the current review for lack of space but certainly deserve in-depth review. An attempt has been made in the current report to summarize recent human studies which further elucidate both the character and the extent of what should now, perhaps routinely, be called the cleft palate syndrome.

The majority of the studies in this area have usually presented data on more than one aspect of the craniofacial complex. Data relevant to a specific part or area of the craniofacial complex, e.g., the maxilla, has, therefore, been extracted from each study in which data on that part or area have been reported.

A. THE MAXILLA AND PREMAXILLA. There is continued interest in the effects of primary palatal surgery on the subsequent growth and development of the craniofacial complex, and, therefore, continued interest in the anatomy and growth patterns of the maxilla. In addition, there seems to be a growing awareness that the palate, both morphologically and functionally, does not exist in isolation from even relatively distant craniofacial structures. The normal growth and development of each component of the craniofacial complex probably very literally depends on the concomitant normal growth and developmental integrity of all associated structures of the craniofacial complex. Developmental interlocking would also be a logical expectation in dysmorphologies of the craniofacial complex, and, at least in cleft lip and palate, there is growing evidence that many regions of the face and cranial base are subject to variances from their normal shape, size, and/or relative position.

The recent literature contains numerous studies on the structure, relative position, and growth of the maxilla in normal individuals and in individuals with cleft lip and palate both preceding and following surgical repair of the cleft. Nakamura et al. (1972) compared longitudinal samples of 45 male and 40 female children grouped according to the extent of their deformities with norms for normal children. The authors found that children with clefts of the lip showed significantly greater bizygomatico-maxillary suture width and wider maxillae than did normal children. Fish (1973) studied 30 children with cleft palate and 30 normal children between birth and three years of age and found that, while the posterior width of the maxillary arch was greater at birth in children with clefts than in normal children, by three years of age the mean posterior and intercanine widths of the arch in the cleft group were significantly less than normal. No other maxillary arch dimensions were found to be significant. In 1974, Mapes et al. studied alveolar length in 40 patients with complete unilateral clefts of the lip and palate. Their photocopies of maxillary casts indicated that tissue deficiency in alveolar length on the affected side had been compensated for by additional growth on the unaffected side by the age of six years so that, by this time, the combined alveolar arch lengths on the two sides were not significantly different from those in normal subjects. Wada and Miyazaki (1975) studied growth and changes in maxillary arch form in 62 normal children and 87 children with complete unilateral clefts of the lip and palate. At six months, prior to lip closure, the cleft lip and palate group exhibited protrusion of the larger alveolar segment. Data reported by Krogman et al. (1975) on craniofacial growth patterns in normal and cleft-palate children between birth and six years indicates an association between palatal clefting and de-

creased maxillary length. In 1976, Bishara et al. studied 12 individuals with unoperated clefts of the lip and/or palate and 12 normal individuals matched in age, sex, and ethnic background. They found distinct differences in the dento-alveolar and skeletal relationships between both cleft groups and normals and between the two cleft groups. Their clinical findings indicated that the alveolar process in the cleft area rolled superiorly with infra-occlusion and tipping of the adjacent teeth, leading to an open-bite tendency in the cleft area. Also, medial collapse of the cleft segment was present with slight rotation and fanning of the maxillary incisors on the non-cleft side. The authors were careful to point out that the population studied were of Dravidian origin and that they are generally smaller in stature than North American Caucasians and exhibit a greater tendency to have bimaxillary dental protrusion.

Hayashi et al. (1976) studied 255 Japanese children with cleft palate and found that the maxillae were more posteriorsuperiorly positioned than in unaffected populations. Crabb and Foster (1977) studied the effects of unrepaired unilateral clefts of the lip and palate on the growth of the maxillary dento-alveolar structures in ten Burmese subjects. Their data suggested that forward development of the maxillary arch as a whole and lateral development of the arch in the molar region were satisfactory. However, at least a third of their subjects exhibited deficient development in the cuspid region on the cleft side and either a malpositioned or hypoplastic central incisor nearest to the cleft. In 1977 Foster et al. published a longitudinal study of dental arch growth in 40 subjects, half male and half female, ranging in age from 2.5 to 10 years. Their data indicate that peak maxillary arch growth occurs between six and seven years in boys but between seven and eight years in girls. Maximum changes in arch shape were found to occur between five and six years in both sexes.

In 1973, Bishara published a study of the cephalometric evaluation of facial growth in 32 normal children and in 12 operated and eight unoperated individuals with isolated clefts of the palate. Comparison of the total cleft group with the normal group indicated that the maxilla and mandible were both positioned more posteriorly in the cleft group but that the two structures still had an acceptable relationship with each other. Maxillary depth was also smaller in the cleft group. No significant differences were found between the two cleft groups, and the author suggested that there seems to be a "natural" tendency for the maxilla and mandible to rest in a more posterior relationship to the cranial base in both the operated and unoperated condition than in the normal condition. Two Southern Indian subjects and two normal individuals matched on the basis of age, sex, and race were studied by Bishara et al. (1978). Both had unoperated bilateral clefts of the lip and palate. The authors found that both subjects had rotated maxillary central incisors and absent lateral maxillary incisors. Both also showed protrusive maxillae in comparison with the normal control subjects.

Two studies of the premaxillary-vomerine complex and one on the intermaxillary and transverse palatine suture areas have been published in the recent literature. Pruzansky (1971) studied the premaxillary-vomerine suture in human skeletal material and histologic sections as well as in serial casts and radiographs from 54 infants with unoperated clefts of the lip and palate. He found that, in the unoperated infants, the premaxilla extended beyond the palatal shelves 12.5 to 22.5 mm. In one subject, the protrusion of the premaxilla increased from 18.5 to 22 mm. from the fifth to the fourteenth month. In comparing incomplete forms of cleft lip with the complete form, the author stated that it is clear that even a small Simonart's band across the cleft is sufficient restraint to alter the configuration of the osseous segments and to mold their development. Friede (1973) studied the premaxillary-vomerine suture in histologic sections of the unoperated side of a nine-month-old human cadaver with a bilateral cleft of the lip and palate. The author stated that the premaxillary-vomerine suture resembled an ordinary facial suture with five different zones discernible. The main sutural direction was oblique from the inferior side of the septum upward and forward. Sparse conglomerates of cartilage cells were found on the bony edges of the suture, but there was no evidence of an epiphyseal line. Like Pruzansky (1971), Friede interpreted his findings to mean that this

cartilage was secondary to the overgrowth of the premaxillary-vomerine complex and was not causative of the protrusion characteristic of an infant born with complete bilateral cleft of the lip and palate. Persson and Thilander (1977) studied the intermaxillary and transverse palatine sutures in 14 male and 10 female subjects ranging in age from 15 to 35 years. Data collected in this study of normal individuals indicated clear evidence that some of the facial sutures start to close in the juvenile period at markedly younger ages than has been supposed in the orthodontic literature. Although beginning obliteration of the posterior part of the intermaxillary suture was observed in the 15-year-old subject included in the study, marked closure of the sutures was not noted typically until the third decade of life. The authors did not observe any marked sex differences.

In summary, the current review of the literature has revealed a number of contrasts in the patterns of growth and development of the maxilla in children with cleft lip and/or palate as compared with those in normal children. There is some evidence to suggest that the maxilla is wider than normal at birth but significantly narrower by the age of three years in the cleft population (Fish, 1973). Wider maxillae may also be associated with clefts of the lip (Nakamura et al., 1972). Findings on the association between palatal clefting and maxillary length are not all in agreement. Some data indicate decreased maxillary length (Krogman, et al., 1975); other data suggest no difference (Fish, 1973; Crabb and Foster, 1977). One study (Mapes et al., 1974) indicated that, in unilateral complete clefting of the lip and palate, combined arch length is not significantly different from normal. Palatal clefting may also be associated with more posterior positioning of the maxilla (Bishara, 1973) or with more posterosuperior positioning (Hayashi et al., 1976). The rate of maxillary growth and concomitant changes in configuration have also been studied (Foster et al., 1977); peak maxillary arch growth appears to occur between six and seven years in males and between seven and eight years in females, while peak changes in configuration appear to occur in both sexes between five and six years. A number of these studies have also provided data on the position and configuration of the alveolar segments in unilateral clefts of the lip and palate (Mapes et al., 1974; Wada and Miyazaki, 1975; Bishara et al., 1976; and Crabb and Foster, 1977). The combined impact of these studies tends to confirm positional deviations of the larger segment in the unoperated condition and deficiencies in vertical and lateral development of the cuspid region on the affected side. It remains unclear whether the forward growth of the maxilla is normal or whether both arches are more posteriorly positioned though normal in relationship to each other. The morphology of the premaxillary-vomerine complex in clefts of the lip and palate has also been described in greater detail (Pruzansky, 1971; Friede, 1973). The suture appears to resemble an ordinary facial suture, and cartilage cells present at its edges appear to be secondary to overgrowth of the premaxillary-vomerine complex and not causative of the premaxillary protrusion characteristic of an infant born with complete bilateral cleft of the lip and palate.

The studies reviewed have presented data on subjects of widely varied racial origins. While most subjects have been North American Caucasians, some of the data are from subjects from Japan (Hayashi et al., 1976), Burmo (Crabb and Foster, 1977), and India (Bishara et al., 1973; Bishara, et al., 1976). Many of the non-American populations cited have provided researchers with an invaluable opportunity to study individuals with unrepaired clefts of the lip and/or palate and to contrast growth and development in these groups with that in matched normal individuals across a very broad age range. However, it has not yet been possible to determine what effects, if any, race has on these data.

B. The Mandible and Temporomandibular Joint. The current literature includes a number of studies on differences between the growth and development of the mandible in normal subjects and in individuals with clefts of the lip and/or palate. Malinowski and Strzalko (1971) collected data on mandibular development in 58 subjects ranging in age from birth to nine months and found significant increments in vertical growth of the ramus during that period. Bergersen (1972) studied the male adolescent facial growth spurt in relation to skeletal maturation and found a significant correlation between the onset of this spurt, facial dimensions, and standing height.

A longitudinal study of dental arch growth conducted by Foster et al. (1977) indicated that peak growth of the mandible occurs between nine and ten years in both males and females. Peak change in mandibular arch shape were observed to occur in both boys and girls at five to six and seven to eight years. Bishara et al. (1978) compared two Southern Indian individuals with unoperated bilateral clefts of the lip and palate and two normal individuals matched on the basis of age and sex. They noted that mandibular length was relatively shorter and that the mandibular plane was relatively steeper in the cleft individuals. The lower incisors of the cleft subjects were lingually inclined in spite of the lack of occlusal contact between the maxillary and mandibular incisors.

Nakamura et al. (1972) found that children with clefts of the palate showed significantly shorter mandibles than did normal children and that children with clefts of the lip and palate exhibited smaller mandibles than did normal children. Cephalometric and radiographic data collected by Fish (1973) indicated no significant differences in any of the mandibular arch dimensions studied in normal and cleft palate individuals between birth and three years. The study of Krogman et al. (1975) on craniofacial growth in children with cleft palate and in normal children from birth to six years indicated that, with regard to mandibular growth, the cleft group exhibited retrognathism but no micrognathism, and no significant differences in mandibular symphyseal height. However, gonial angle was more obtuse, the SNA angle was greater, and the data on the temporomandibular joints suggested the possibility of retropositioning. Bishara et al. (1976), in their study of 12 normals and of 12 individuals with unoperated clefts of the lip and palate, found that there was a tendency for the mandibular plane to be steeper and for the lower incisors to be significantly more lingually positioned in the cleft group. Horowitz et al. (1976) found all of the mandibular angles to be increased in individuals with cleft lip and palate. The study of Hayashi et al. (1976) on Japanese children with cleft palate indicated that the mandibular rami were shorter, the gonial angle was more obtuse, and the chin was typically retropositioned. In 1976, Graft-Pinthus studied 39 children with repaired unilateral clefts of the lip and palate and a carefully matched control sample. His observations and evaluation of growth patterns in the most-cleft and least-cleft groups within this sample indicated wide variability. In the two subjects judged to be most cleft, one showed midfacial hypoplasia, pseudoprognathism, and a total cross-bite by eight years of age, while the other did not develop either hypoplasia or cross-bite and had a well-balanced facial appearance at the age of seven years. Of the least cleft subjects evaluated, one exhibited a tendency toward mandibular prognathism at 14 years of age, while the other showed normal clinical growth. The author concluded that it is not possible to identify cleft lip and palate infants as difficult or simple clinical cases based only on cephalometric evidence.

Vora and Joshi (1977) studied mandibular growth in 25 surgically-repaired Indian children with cleft palate and in control subjects without cleft palate. Their findings indicated poor vertical growth of the mandibular ramus in the cleft palate group, a high gonial angle, a tendency toward retrusion of the chin relative to the cranial base, and retroinclination and lingual placement of the mandibular incisors in relation to the mandibular plane.

Only one study of the postnatal development of the human temporomandibular joint was found in the current review. Wright and Moffett (1974) studied 44 subjects under 10 years of age and six subjects between 10 and 21 years of age. X-ray and histologic sections of the joint area obtained by the authors indicated that throughout the postnatal developmental period, the articular tissue and discs consist of fibrous tissue; no cartilage cells were observed in any of the specimens studied. The disc at birth was found to be flat, and the S-shaped profile was found to develop as the articular tubercle developed. The tubercle at birth consisted of only a small elevation formed by a combination of endochondral, immature, and ordinary intramembranous bony tissue. The authors also found that the characteristic profile of the temporal portion of the temporomandibular joint is achieved during the first two or three years of life, after which time gradual enlargement and compaction of the bony structures occurs. Growth of the mandibular condyle is in a constant

posterosuperior lateral direction, and a mature contour is not achieved until the age of mixed dentition. The condylar cartilage decreased from 1.5 cm. to .05 mm. shortly after birth and did not show the columnar arrangement of chondrocytes seen in epiphyses. The closing plate of bone was observed to coalesce below the condylar cartilage at about 16 or 17 years of age.

In summary, the literature on normal mandibular growth and development suggests that significant increases in the height of the mandibular ramus occur within the first nine months of life (Malinowski and Strzalko, 1971), that mandibular length is highly correlated with other general body dimensions during the adolescent growth spurt (Bergersen, 1972), and that peak growth of the mandible occurs between nine and ten years in both males and females while peak configurational changes occur between five and six and between seven and eight years (Foster et al., 1977). Data on the normal growth and development of the temporomandibular joint has been presented in detail by Wright and Moffett (1974) and indicates that curvature of the disc develops as the articular tubercle develops while the characteristic configuration of the temporal part of the joint is achieved within the first two to three years of life.

The studies of the growth and development of the mandible in individuals with clefts of the lip and/or palate indicate that clefting is associated with retrognathism but not micrognathism (Krogman et al., 1975), a more obtuse gonial angle (Krogman et al., 1975; Horowitz et al., 1976; Hayashi et al., 1976; Vora and Joshi, 1977; Bishara et al., 1976, 1978), and lingual inclination of the lower incisors (Bishara et al., 1976, 1978; Vora and Joshi, 1977). However, Fish (1973) found no significant differences in any of the mandibular dimensions between subjects with and without clefts in the birth-to-three-year age range. Several studies suggested retropositioning of the chin (Hayashi et al., 1976; Vora and Joshi, 1977) or temporomandibular joint (Krogman et al., 1975). Bishara et al. (1978) suggested relative reduction of mandibular length in clefts of the palate. Hayashi et al. (1976) and Vora and Joshi (1977) observed poor vertical growth of the ramus. It is clear from the disagreements noted on mandibular form and development in both normal and cleft-palate individuals that further study is needed in this area.

C. Facial Proportions. Changes in facial proportions have been a topic of interest during the past few years. Bergersen (1972) presented an excellent review of the literature on the relationship between facial growth and general body growth. He reported that skeletal maturation is significantly correlated with the facial growth spurt in male adolescents from at least five years prior to the onset of the adolescent growth spurt to one year following its onset. Nakamura et al. (1972) found that facial growth rates appeared to be the same in children with cleft deformities as in normal children. Krogman et al. (1975) found that individuals with cleft palate exhibit increased upper and lower facial height, while Hayashi et al. (1976) found that upper facial height is reduced while lower facial height is increased. Horowitz et al. (1976) indicated that both upper and lower facial height are affected in cleft lip and palate; upper anterior facial height is reduced while lower anterior facial height is increased. The authors found posterior facial height to be similarly affected; upper posterior facial height was found to be reduced while lower posterior facial height was found to be increased. In a study of mandibular growth in 25 Indian children with surgically-repaired cleft palate, Vora and Joshi (1977) found upper anterior facial height to be unaltered, while lower anterior facial height was increased.

In summary, only one study of normal facial growth has been found in the recent literature Bergersen (1972) found a significant correlation between skeletal maturation and facial growth during the growth spurt in male adolescents. Nakamura et al. (1972) found that facial growth rates in normal and cleft-palate individuals did not differ.

Several researchers have found significant cleft/normal differences in the facial skeleton. Hayashi et al. (1976) presented data which indicate that upper facial height is decreased and lower facial height is increased in the cleft condition, while Krogman et al. (1975) found both upper and lower facial height to be increased. Horowitz et al. (1976) and Vora and Joshi (1977) both found clefting to be

associated with increased lower anterior facial height but did not agree on upper anterior facial height. Horowitz found the latter dimension to be decreased in cleft-palate individuals, while Vora and Joshi found it to be the same as in normal individuals. Horowitz et al. (1976) found upper posterior facial height to be decreased and lower posterior facial height to be increased in individuals with cleft palate.

The obvious lack of unanimity of facial proportions in cleft-palate individuals clearly indicates the need for further research on this question. Part of the disagreement may result from differences in methodology, particularly in the selection of measurement points, or from racial differences. Very little data seems to be available on the patterns and rates of facial growth in normal and cleft-palate individuals. Finally, if growth rates are not significantly different in normal and cleft-palate individuals (Nakamura et al. 1972), but facial proportions do differ significantly (Hayashi et al., 1976; Horowitz et al., 1976; Vora and Joshi, 1977), then the facial characteristics of the two groups must be different to begin with. This particular issue has not been addressed in the recent literature.

D. The Cranial Base. Possible dysmorphology of the cranial base in cleft lip and/or palate has also been a topic of concern in the recent literature. Bishara and Iversen (1974) noted in their study of 30 normal and 27 cleft-palate subjects that the cranial base was "smaller" on the average in the cleft group. The data of Krogman et al. (1975) indicate that anterior cranial base length and clival length in children with cleft palate are greater than normal. The cleft group which they studied also showed greater flexion of the sellar angle than did the normal group, and both linear and angular measurements were noted to be more severe in the cleft-lip-and-palate group than in the cleft-palate-only group. One of the statements made by these authors is worthy of direct quotation: "We feel that all of this points to basion and foramen magnum as the major areas of adjustment to CL(P) and CP clefting." The authors further suggested that there is a close relationship between the various structural components of the craniofacial midline and, specifically, that palatal clefting may have repercussions in adjacent bony structures in both the cranial base (occipital, sphenoid, and ethmoid bones) and facial areas (involving the midfacial complex).

In 1976 Bishara et al. observed that, in general, the cranial base and skeletal face are not extensively malrelated in individuals with either unoperated clefts of the lip and palate or of the lip and alveolus as compared with matched normals but that distinct differences in the dento-alveolar and skeletal relationships between the combined cleft groups and normals and between the two cleft groups do exist.

Several other studies relevant to the dimensions of the cranial base have also published recently. Maue-Dickson et al. (1976) studied human abortuses with and without cleft palate and found increased distances between the left and right pterygoid plates on each side. They suggested that cleft palate may be associated with an abnormally wide cranial base. Hayashi et al. (1976) noted that the cranial base angle was more obtuse in their cleft subjects than in the normal controls. Bishara, Olin, and Krause (1978), in their study of two Indian subjects with unoperated bilateral clefts of the lip and palate and two normal control subjects of the same population, found that the size and relationships of various structures of the cranial base were comparable in the cleft and normal subjects.

In summary, the recent literature presents little definitive information on the cranial base in the cleft-palate population. While there is some evidence to suggest that changes in linear and angular dimensions of the various cranial base structures are significantly different in normal and cleft-palate individuals (Krogman et al., 1975; Maue-Dickson et al., 1976; Hayashi et al., 1976), one study has indicated that the size and relationships of cranial base structures appear to be comparable in the two groups (Bishara et al., 1978), and another study has indicated that, while the cranial base and skeletal face are not extensively malrelated in the cleft group, there are deviations from normal in cranial base and dento-alveolar relations (Bishara et al., 1976). Further longitudinal and cross-sectional study of the cranial base and related structures is needed, both in operated and unoperated cleft-palate individuals and in matched normal subjects. The possible influences of sex, race, and age should also be

studied further. There do not appear to have been any studies which have focused on possible cranial base malformations associated with palatal clefts of varying degrees of severity.

E. The Nasal Septum and Nasal Airway. Another area of the craniofacial skeleton which has received some attention is the nasal septum and nasal airway. Drettner (1960) studied nasal airway dimensions in 53 subjects with repaired clefts of the lip and palate or palate only. In this group, deviation of the nasal septum, usually to the side of the cleft, was common in unilateral cleft lip and palate, and the incidence of nasal airway constriction was significantly higher in cleft lip and palate combined than in cleft palate alone. Drettner hypothesized that nasal obstruction may be the end product of a series of events beginning with irritation caused by food and saliva and followed by upper respiratory infections, inflammation of the nasal mucosa, and, finally, hyperplasia of the nasal mucosa and conchae. The author emphasized the importance of not neglecting the physiological respiratory function of the nose along with the cosmetic problems involved in the treatment of cleft lip and palate.

One other study found in the current review documented the prevalence of deviated nasal septa and nasal obstruction in cleft palate. Chaudhuri and Bowen-Jones (1978) studied 245 children with cleft palate and noted a 50% incidence of deviated septa producing nasal obstruction. Both of these studies reveal a gap in our understanding of the mechanisms underlying nasal obstruction in cleft-palate individuals and the functional importance of such obstruction.

F. Multivariate Analyses of the Craniofacial Skeleton. A number of studies reported in the recent literature have indicated a growing interest in patterns of covariation among various spatial, linear, and angular measurements of the craniofacial complex in order to determine what combination of craniofacial skeletal features might best describe the cleft lip and palate individual and in order to determine what portions of the face might be particularly vulnerable to growth disturbances in cleft lip and palate. In an analysis of lateral cephalometric x-rays of 39 children with repaired clefts of the lip and palate and 39 normal children matched for age and sex, Horowitz et al. (1976) were able to identify six factors which together accounted for 92% of the observed variance in skeletal morphology. These included the nasopharyngeal-maxillary complex, cranial base, body and ramus of the mandible, the palate, and the lower face. The authors also identified certain portions of the face which appeared to be particularly vulnerable to growth disturbances in cleft lip and palate individuals. For example, the angles formed between the upper posterior facial region and the palatal plane were significantly larger in the cleft group and reflected rotation of the palatal plane in a clockwise direction. As described earlier, they also found significant differences in the dimensions of upper and lower facial height in the cleft group. Finally, the authors presented data which indicated that the nasomaxillary complex and nasopharynx in cleft lip and palate are significantly smaller.

Foster et al. (1977) was able to identify marked variations in dental arch growth between male and female subjects, between different age groups, and between maxillary and mandibular dental arches by the multivariate statistical analysis of longitudinal data from normal subjects. Liebgott (1977) utilized factor analysis on radiographic measurements of the craniofacial complex and was able to identify a large number of uncorrelated factors which accounted for 91% of the total variance. These factors included retrognathic facial type, anterior dento-alveolar height, maxillary body length, cranial base and facial width, ramus height, anterior maxillary body height, mandibular length, cranial vault height, vertical position of the condyles, cranial vault and clivus length, bigonial width, and cranial vault width. Lavelle (1977) demonstrated significant contrasts between angle Class I, II and III malocclusions by the use of multivariate analyses and contributed new data on the varying relationships of the craniofacial skeleton in each occlusal category.

The use of multivariate analyses is a necessary and appropriate approach to the discovery of growth patterns and structural deviations which affect several or many components of the craniofacial complex.

G. The Effects of Surgery on Craniofacial Growth and Development. Numerous investigators have noted that cleft individuals

who have undergone surgery in childhood exhibit differences in craniofacial relationship as compared with non-cleft individuals. As pointed out by Bishara et al. (1976), however, the causes of the differences are still controversial. A number of investigators whose publications were reviewed for the current report have attempted to determine whether these differences result from surgery or exist independently of the effects of surgery on subsequent growth and development of the craniofacial complex.

Bishara (1973) studied facial growth cephalometrically in 32 normal children, in 12 subjects with surgically repaired palates, and in eight subjects with unrepaired isolated clefts of the palate. As noted previously in this report, he found significant differences in the relative size and position of the maxilla and mandible and reduced maxillary depth in the cleft group. His comparisons of the repaired cleft group with the unrepaired, obturated cleft group revealed no significant differences. These data suggest that, although differences between the cephalometric morphologic characteristics of the cleft face and the normal face do exist, these differences are not necessarily a result of palatal surgery. Bishara has stated that his cephalometric data can be considered the "normal" morphological pattern for the cleft-palate-only face and has concluded that, when these individuals are compared to normal individuals, the latter should be used primarily as a reference or baseline and not to detect differences since the cleft and normal samples are representative of essentially two different populations with different craniofacial characteristics.

In 1974 Mapes et al. studied photocopies of maxillary casts from 40 patients with complete unilateral clefts of the lip and palate and noted that the growth rate of the maxillae in the cleft group lagged temporarily during the interval immediately following surgery but then accelerated temporarily beyond the normal rate to a point where normal length was achieved. Wada and Miyazaki (1975) also studied growth and changes in maxillary arch form following surgery. Their data, based on 62 normal children and 87 children with complete unilateral clefts of the lip and palate, suggest less temporary changes as a result of surgery. In the cleft subjects who had had lip repair at six months and palatal closure at two years, the depths of all alveolar dimensions were found to be decreased at the age of two years. At four years, the latter group exhibited alveolar height and depth which were significantly smaller than in the normal group. These differences could, at least in part, be the result of surgical intervention or, as pointed out by Bishara in 1973, could simply demonstrate further the existence of two different populations with two different arrays of craniofacial characteristics.

Bishara et al. (1976) suggested that the resolution of this question might lie in the study of unrepaired cleft individuals at later stages of development. Such a study might offer some insights into which types of growth inhibition are inherited, which are biomechanically compensatory, and which are induced. They studied 12 individuals with unoperated clefts of the lip and palate ranging in age from seven to 29 years and compared these subjects with 12 normal individuals matched in age, sex, and ethnic background. Their cephalometric comparisons indicated no significant differences in the maxillary and cranial base parameters between the cleft and normal groups. However, there was a tendency for the mandibular plane to be steeper than normal and for the total face height to be greater in the cleft group.

Crabb and Foster (1977), whose data have been summarized previously, have pointed out that certain of the growth deficiencies associated with repaired clefts of the lip and palate may be present in the absence of surgery. Most particularly, this includes deficiencies in vertical and lateral growth in the maxillary cuspid region immediately behind the alveolar cleft. Their data clearly suggest the presence of localized growth defects which cannot be accounted for by operative trauma.

In 1977, Mazaheri et al. conducted a longitudinal analysis of growth of the soft palate and nasopharynx from six months to six years in 20 children with unilateral complete clefts of the lip and palate, 10 children with bilateral clefts of the lip and palate, 13 children with clefts of the palate only, and 15 normal controls. As pointed out by the authors, it has been postulated that shortness of the soft palate may result from prenatal velar underdevelopment, palatal bony deficiency, or nonfunctional velar atrophy. Data collected in the current study reveal a continuous relative

growth rate of the soft palate from six months to six years and do not suggest nonfunctional velar atrophy. Velar length appeared to be affected more in the unilateral-cleft-lip-and-palate and bilateral-cleft-lip-and-palate groups than in the cleft-palate-only group. This suggested to the authors that intrauterine underdevelopment and palatal bony deficiency may contribute to velar deficiency at birth. Data collected by the authors do not support the concept of more superior positioning of the posterior border of the hard palate or of deficiency in vertical maxillary growth. Nasality in the cleft groups was noted to decrease from the age of three to six years without intervention or secondary surgery.

The function status of the velopharyngeal mechanism following maxillary advancement has been reported by two groups during the period of the current review. Unfortunately, both studies were single-case reports. Witzel and Monroe (1977) stated that velopharyngeal insufficiency did result from the procedure, while Bralley and Schoeny (1977) stated that the procedure did not adversely affect either articulatory ability or voice.

Kaplan (1978) studied 112 patients with unilateral complete clefts of the lip both pre- and post-surgically. His data indicated that a cleft lip has ten to 20% greater growth in the transverse direction parallel to the orbicularis oris muscle than in the vertical direction perpendicular to the muscle. He concluded that surgical procedures which transpose tissue from transverse to vertical will lead to excessive vertical growth. Lip growth was found to be slightly decreased along the cleft margins. Lip shortness was observed to occur soon after repair because of scar contracture but tended to resolve with the passage of time. On the side of the cleft, transverse growth was found to be slightly less than normal (82% of normal), whereas vertical growth was significantly reduced (67% of normal at the philtrum and 50% at the ala). As stated by the author, this study did not determine the reason for differential growth of lip skin. He hypothesized that abnormal skin tension and muscle force are the most important factors controlling lip growth aside from genetic influences.

Enemark and Jorgensen (1978) observed that unilateral-cleft-lip-and-palate patients operated on by the Tennison-Veau procedure exhibit a rather high percentage of maxillary asymmetry. Their tomographic study of 20 children who had had Tennison repairs at two months of age and Wardill palatoplasties at 24 months of age demonstrated bony development in the hard palate as well as fusion between the nasal septum and the maxilla on the cleft side in all subjects studied. In most patients, this bone formation was combined with an irregular thickening of the base of the bony septum with marked asymmetry of the hard palate.

One other study designed to assess the results of a specific surgical procedure was conducted by Subtelny and Nieto in 1978. The authors conducted a longitudinal study of maxillary growth following pharyngeal flap surgery carried out prior to the approximate time of the pubertal growth spurt. Data from the 24 subjects in this group were compared with data from a group of 18 cleft palate individuals who had had palatal surgery but not a pharyngeal flaps. Both cleft groups showed some post-surgical reduction in maxillary growth. However, the pharyngeal-flap group showed significantly greater reduction in maxillary forward growth. Interestingly, the pharyngeal-flap group showed normal vertical maxillary dimensions with growth, whereas the non-flap group showed some reduction in vertical growth of the maxillary complex. This study may reflect surgically-induced abnormalities in maxillary growth, or it may simply reflect pre-existent differences in the morphology and growth potential of the two cleft groups.

In summary, the recent literature indicates that some of the craniofacial growth deficiencies observable in cleft palate, e.g., abnormal maxillary and mandibular growth, are apparently unrelated to surgical trauma (Bishara, 1973; Bishara et al., 1976; Crabb and Foster, 1977; Mazaheri et al., 1977). Other studies suggest that surgery may retard maxillary growth temporarily (Mapes et al., 1974) or perhaps permanently (Wada and Miyazaki, 1975). The data of Enemark and Jorgensen (1978) suggest that maxillary asymmetry may result from surgery. One study (Subtelny and Nieto, 1978) suggested a significant association between retarded forward growth of the maxilla and pharyngeal flap surgery. However, the cleft-palate group in whom this was observed exhibited normal vertical growth of

the maxilla, while the non-flap cleft group exhibited some reduction in vertical growth of the maxilla. This could suggest a number of different influences: non-interference with vertical growth by pharyngeal flap surgery, interference with vertical growth by non-flap surgery, or differences in the velopharyngeal insult which led to the selection of flap or non-flap surgery. Further study of each of these possibilities is clearly warranted.

We are still a great distance away from unanimity on the possible effects of surgery on subsequent growth and development of the craniofacial complex in individuals with cleft palate. Nonetheless, it has become apparent that certainly not all deviations from the "normal" can be assigned to the negative effects of surgery and that, at least in some cases, surgery may have only a temporary negative effect or, possibly, even a positive effect on the subsequent normalcy of craniofacial growth and development.

H. Methodologic Problems. Since the vast majority of studies of the craniofacial skeleton involve the use of cephalometrics, it seems appropriate to bring up the matter of x-ray validation and methodology utilized commonly in the collection and analysis of data. Cross (1977) has very succinctly pointed out the fallacies which may result from the selection of any particular facial landmark in making judgements about the direction of facial growth. He has stated very simply that superimposition on the bottom of the feet of a child will project the child's growth in an upward direction, while superimposition on the top of the head will project the child's growth in a downward direction. Isaacson et al. (1977) also addressed this problem. They have pointed out that; in a study of mandibular growth, the placement of metallic markers in the growing jaws has permitted superimposition on the jaws themselves rather than having to rely on superimposition on some presumably stable sutures. By employing metallic markers, the authors have found that jaw growth is not linear as supposed previously; rather, facial growth is a primarily rotary phenomenon which would result in major changes in dental occlusion if it were not compensated for by dissimilar alveolar growth. While the authors' principal objective was to show how vertical growth of the ramus is capable of producing anteroposterior dental and profile changes, the implications of their methodology extend to a wide range of studies of the craniofacial complex.

Slagsvold and Pedersen (1977) measured gonial angle distortion in lateral head films and demonstrated a difference between the roentgenocephalometric and craniometric registrations of 8.48 degrees for the side closer to the film and 5.5 degrees for the side further from the film. For the intermediate gonial angle, the difference was 6.65 degrees. All of the differences were statistically significant and the data presented demonstrate conclusively that the differences were primarily attributable to a systematic error in the roentgenocephalometric method involving a magnification of the gonial angles. The authors concluded that lateral head films do not, in general, permit reliable registrations of the gonial angle and that, at present, no method is available for an exact assessment of the distortion in head films in the single living person. This study, like that of Isaacson et al. demonstrates the need for very careful assessment of methodology and serves as a reminder of the need for the demonstration of both reliability and validity.

I. Craniofacial Morphometrics. The recent literature evidences an increasing interest in the study of morphometric techniques. Walker and Kowalski (1972) have described the use of computer morphometrics in craniofacial biology and have reported on the application of their method in the analysis of some 30,000 cases, each with records and plots of 177 coordinate points which comprised the authors' model of craniofacial morphology. As stated by the authors, the importance of this type of computer modeling is in the prediction of results of the treatment, or lack thereof, and in predictions of the future size of craniofacial structures, the timing of surgical changes, and the velocity and direction of growth. Berkowitz et al. (1974) have described a new method for the quantitative analysis of cleft palate casts. The method which they have described, stereophotogrammetry, produces digital representations of a structure by means of measurements taken from photographs. The application of the method for the analysis of patients with craniofacial abnormalities has been reviewed by Berkowitz and Cuzzi (1977) on five patients. The latter study demonstrates that a useful

coordinate transformation system can be created by connecting points supernasala and subnasala for the establishment of the YZ plane and the construction of the XZ plane at subnasala.

In 1977, Rabey described a method by which craniofacial disharmony can be evaluated. Individual analytic morphograms are combined to form analytic histograms, which, in turn, are connected to form analytic histomorphograms, which are used to provide three-dimensional statements about the variation in craniofacial structures within a given population. The author mentioned that this methodology is being converted to a computer-graphic technology for large-scale studies.

It seems clear that the recent advances in computerized tomography will also become extremely important and useful in the quantitative analysis of craniofacial morphometrics and in the pre- and post-surgical evaluations of craniofacial anomalies of many types, including cleft palate (Maue-Dickson and Trefler, 1977). One of the main advantages of computerized tomography in this regard is that it permits the analysis of soft-tissue as well as hard-tissue structures.

All of these studies relate directly to the development of techniques which will supplement and extend our understanding of both external and internal morphology in the assessment of the craniofacial complex. However, if these systems are to be used effectively in the prediction of growth and development of craniofacial structures for the purpose of treatment planning, far more definitive information than is currently available will be needed on "normal" growth curves for specific subgroups within the category of craniofacial anomalies. Further research in this area and on the functional correlates of static morphology is needed.

Summary and Discussion

The purpose of this report has been (1) to review the recent literature on the anatomy and function of the craniofacial complex in cleft lip and palate; (2) to identify areas of missing or ambiguous information and unresolved controversies; (3) to identify areas which need no special research emphasis; and (4) to discuss approaches to provide additional information in those areas in need of further development. The active debate and dialogue evident in the recent literature has made this a worthwhile challenge.

The recent literature on the middle ear musculature, the auditory tube, and the velopharyngeal mechanism has provided growing support for the following viewpoints: The tensor veli palatini and tensor tympani muscles function as a unit in normal auditory tube clearance. Their contraction, which provides active pressure equalization between the tympanic cavity and the nasopharynx, is probably not linked obligatorily to swallowing, a hypothesis which needs to be tested. The anatomical, functional, and developmental parameters involved in disruptions of this function in cleft palate are still poorly understood.

There is increasing evidence to suggest that the levator veli palatini muscle may be the prime muscle in normal retraction and elevation of the velum for speech and in anteromedial movement of the lateral pharyngeal walls for speech. The superior and middle constrictor muscles, perhaps aided by the palatopharyngeus muscle, subserve pharyngeal adjustments in the articulation of vowels. The superior fibers of the superior constrictor muscle, thought to produce Passavant's pad, appear to produce a circum-pharyngeal buldge too low to compensate for velopharyngeal inadequacy. The dysfunction of the levator and constrictor muscles is still poorly understood in cleft palate. Patterns of velopharyngeal closure in cleft palate are characterized by extreme variability which has not as yet been studied adequately.

The uvulus muscle contributes a central dorsal velar mass which has been identified as the "levator eminence"; this mass should now be termed the "uvulus eminence." Its presumed absence, paresis, or hypoplasia produces central velar gaps which obviate normal closure of the velopharyngeal port for speech. The morphology of this muscle is now well understood in normal subjects but should be studied in the cleft-palate population. The innervation of the velopharyngeal musculature is still poorly understood in both the normal and the cleft-palate condition.

The recent literature on the lip, mandible, tongue, nose, and larynx suggest that our information is still incomplete in many regards. Further studies of both normal and abnormal growth and development are

needed. The anatomy of the lip, tongue, nose, and larynx requires further study in both normal and cleft-palate individuals. Further, there have still not been any studies of the tongue in cleft palate individuals even though the previous reviews have suggested abnormal lingual configuration associated with cleft palate. Labial and lingual innervation and blood supply in cleft palate have not been studied.

The growth and development of the craniofacial skeleton has been extensively investigated during the past few years. The studies reported span age groups from birth through late adult life and include both cross-sectional and longitudinal data on specific morphologic features within the craniofacial complex and on age, race, and sex differences. They also present new perspectives on the pre-and post-surgical status of the craniofacial complex. Nonetheless, there are still several obvious gaps in our information in this area. The findings on the association between palatal clefting and various maxillary, mandibular, and cranial base dimensions are not in agreement, nor is there unanimity on the nature and timing of developmental changes associated with craniofacial growth in individuals with cleft palate. The possible influences of race, sex, and age are frequently unclear and have proven to be difficult to document. The literature indicates a concerted effort to discern the possible influences of surgery on morphologic status, and the available data indicate that not all abnormalities seen post-operatively result from surgical trauma. On the contrary, some appear to be intrinsic to the cleft-palate population.

It is clear from the disagreements on craniofacial dimensions and proportions associated with cleft palate that further study is badly needed in this area. The literature also suggests the need for more extensive study of the patterns of co-variation among various spatial, linear, and angular measurements of the craniofacial complex in both normal and cleft-palate subjects.

It is apparent that at least some of the disparity in data found in the literature may reflect methodologic differences between and among subjects. This would suggest that the standardization of techniques and the more precise definition of data points could be beneficial in the collection of similar data by different researchers and in the subsequent pooling of these data.

Further, in many instances our understanding of function would be greatly enhanced by the simultaneous use of several different methodologies. For example, velopharyngeal function can be analyzed in part by a number of different methods, including radiographic, cinematographic, nasopharyngoscopy, electromyographic, pressure/air flow measurements, and acoustic studies, but frequently only one of these methods is utilized at a time. The simultaneous recording of velopharyngeal function with cine, EMG, and acoustic recordings would provide correlative data not obtainable by combining data from these three sources if the data has been obtained during different time periods in the same subjects or if the three types of data have been obtained from different subjects.

Finally, it is imperative in all areas in which our information on cleft-palate anatomy and function is deficient to encourage continued collaboration and utilize more fully interdisciplinary and interinstitutional resources.

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