Velopharyngeal Incompetence in Cleft Palate Patients - Flexible Video Pharyngoscopy & Perceptual Speech Assessment

A DISSERTATION SUBMITTED IN PART FULFILLMENT OF

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Velopharyngeal Incompetence in Cleft Palate Patients

- Flexible Video Pharyngoscopy & Perceptual Speech Assessment

A Observational Co-relational pilot study

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'to Him be all the Glory....

CERTIFICATE

This is to certify that the dissertation Velopharyngeal Incompetence in Cleft Palate Patient Flexible Video Pharyngoscopy & Perceptual Speech Assessment-A Observational Co-relation pilot study is the bonafide work of Dr. Rajan Sundaresan V carried out in the DEPARTMENT OF ENT at CHRISTIAN MEDICAL COLLEGE VELLORE, TAMILNADU, under the guidance of Dr. Mary Kurien , Professor and Head, UNIT-II, DEPARTMENT OF ENT, Christian Medical College, Vellore for the M.S (Branch -IV) OTORHINOLARYNGOLOGY EXAMINATION OF THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY, to be held in March-2010.

DR. ACHAMMA BALRAJ, M.S, M.Sc in AUD.MED (LONDON)
PROFESSOR AND HEAD,
DEPARTMENT OF ENT,
CHRISTIAN MEDICAL COLLEGE
VELLORE, TAMILNADU.

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GUIDE

DR. MARY KURIEN MS., DLO.,
PROFESSOR & HEAD OF ENT II
DEPARTMENT OF ENT,
CHRISTIAN MEDICAL COLLEGE
VELLORE, TAMILNADU.

CONTENTS

INTRODUCTION

Velopharyngeal in-competence(VPI)[2] is a common abnormality seen in Cleft palate patients causing hyper nasal speech which is a major communicative disorder in such patients[3]. Assessment of this VPI is complex process due the Velopharyngeal apparatus being a combination of soft palate structures that regulate the airflow from the lungs and larynx through the mouth for oral sounds through the nose for nasal sounds [3].

In cleft palate patients there is a combination of structural, anatomical [5] and behavioural deficiencies which needs to assessed simultaneously and thoroughly [6]. Velopharyngeal insufficiency can affect both speech and swallowing, which may lead to situational difficulties. Disruptions of speech in children, regardless of the cause, are associated with reductions in scholastic performance and increased risk of retention in grade school. Speech impediments have been reported to disrupt social, emotional and educational development, effective communication, self-esteem, and participation in activities. Peers and caregivers often perceive these children negatively, and the resulting emotional impact may ultimately affect their activity, participation, and scholastic performance [7]This condition can affect the children and their families in many ways. In early life many patients undergo surgical repair and have speech, hearing and dental problems; during adolescence they may have cosmetic, orthodontic and emotional problems. Many parents are concerned about the genetic implications of the defect. This variety of problems requires management by several health care disciplines. Many health centres offer multidisciplinary team

management in a cleft palate-clinic. The child's primary care physician, with whom the team exchanges information, plays a significant role in helping the child and the family function optimally[8].

ENT problems appear in almost two-thirds of children with cleft palate (CP) / unilateral cleft lip and palate (UCLP). Hearing disability is common too. Nasal septum deviation or chronic hyperplastic rhinitis may cause impaired nasal breathing the great majority of the UCLP children. Voice disorders are frequently related to hearing loss and it is not more common than in the population of non-cleft children. Nasal speech is attributed to the presence of cleft palte. However post operatively following the correction of cleft palate if the patient has persistence of speech problem, the ENT surgeon and speech pathologist is approached.

Therefore, an Otolaryngologists has an active role early in the treatment of children with clefts palate in order to improve the children's hearing ability ,nasal breathing and to prevent functional voice disorders. Good cooperation with the maxillofacial surgeon and entire multidisciplinary team is also essential[9].

Otolaryngologists are increasingly being called upon to assist in the differential diagnosis of velopharyngeal valving disorders for speech, assisting in treatment planning and the assessment of treatment outcomes [10]. The most commonly used methods for direct visualization of velopharyngeal function remain nasendoscopy and videofluoroscopy. Literature supporting the use of either nasendoscopy followed by videofluoroscopy or the reverse can be found. Several studies also suggest that magnetic resonance imaging[11] can make important contributions to the evaluation of velopharyngeal anatomy and function. The routine use of magnetic resonance

imaging for evaluating the velopharynx is neither practical nor probable at the present time. Magnetic resonance imaging may be as effective as videofluoroscopy or nasendoscopy. The cost of magnetic resonance imaging and the radiation exposure of videofluoroscopy dictates nasendoscopy to be the most common technique for evaluating velopharyngeal function during speech. Several recent studies have documented the use of or assigning patients to a given surgical procedure, predicting surgical success or complications, and evaluating treatment outcomes [12]. There are still discussions and conflicting results regarding the best method for evaluating velopharyngeal function suggesting that no single method is best. The decision regarding the most appropriate evaluation protocol is to be guided by the information that the clinician is attempting to obtain and the relative benefits and risks of each method[4].Nasoendoscopy and speech assessment of the children early in the evaluation process not only gives good information for surgical correction but it also becomes a reference point for further management[3]. A good recording of both the endoscopy and the speech pathology can be used as bio feedback tools as the child grows[12].

AIMS AND OBJECTIVES

Primary Objectives

In patients who have cleft palate pathology:

- To establish a possible co-relation between the type of velopharyngeal Incompetence and the speech defect
- To assess the usefulness of Video endoscopy in evaluation of velopharyngeal incompetence.

Secondary Objectives

To assess the prevalence of type of

- Persistence of velopharyngeal incompetence (VPI)
- Perceptual speech defect in cleft palate

REVIEW OF LITERATURE

Velopharyngeal Insufficiency (VPI) is described by any of the following: velopharyngeal inadequacy, velopharyngeal insufficiency, velopharyngeal incompetence, or velopharyngeal dysfunction. These terms are used interchangeably to denote any type of velopharyngeal closure problem. Velopharyngeal Incompetence is a generic term used to describe the in ability to achieve complete closure of the Velopharyngeal apparatus during speech[13]. The velopharyngeal apparatus consists of the soft palate and the pharyngeal structures .In a normal structure the soft palate in coordination with pharyngeal structures regulate the air flow from the lungs and the larynx as it passes trough mouth for oral sounds and thro the nose for the nasal sounds. The presence of normal palate & pharyngeal structures is necessary for phonation , these can deformed in various forms of craniofacial deformities. [13]

Rise EN[14] as early as 1966 has described the function of the velum i.e. the soft palate and its surrounding structures as a the producers of pharyngeal closure for a good sound production. [15]

The "spoken word" (Voice "As we know it") results from three components of voice production: voiced sound, resonance, and articulation.

- Voiced sound: The basic sound produced by vocal fold vibration is called "voiced sound." This is frequently described as a "buzzy" sound. Voiced sound for singing differs significantly from voiced sound for speech.

- Resonance: Voice sound is amplified and modified by the vocal tract resonators (the throat, mouth cavity, and nasal passages). The resonators produce a person's recognizable voice.

- Articulation: The vocal tract articulators (the tongue, soft palate, and lips) modify the voiced sound. The articulators produce recognizable words

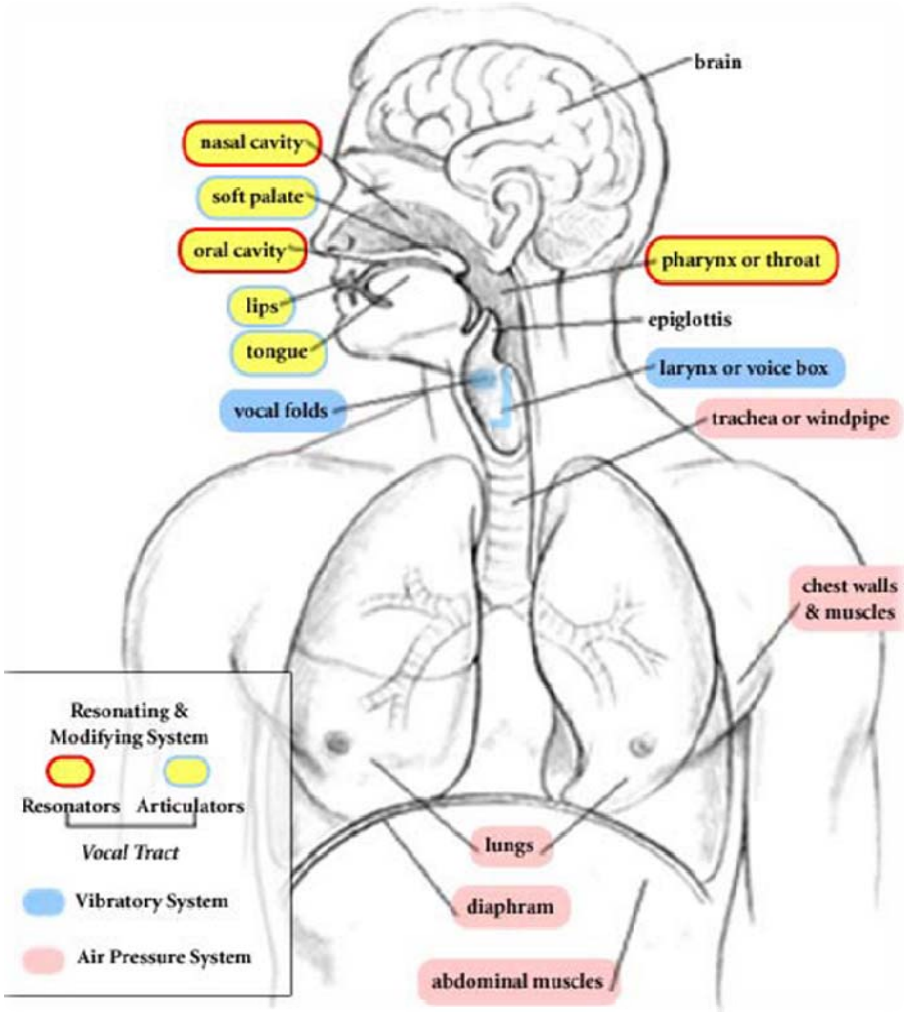


Figure 1

Voice Mechanism

Speaking and singing involve a voice mechanism that is composed of three subsystems. Each subsystem is composed of different parts of the body and has specific roles in voice production. (Figure 1)

Three Voice Subsystems

Subsystem	Voice Organs	Role in Sound Production
Air pressure system	Diaphragm, chest muscles, ribs, abdominal muscles Lungs	Provides and regulates air pressure to cause vocal folds to vibrate
Vibratory system	Voice box (larynx) Vocal folds	Vocal folds vibrate, changing air pressure to sound waves producing "voiced sound," frequently described as a "buzzy sound" Varies pitch of sound
Resonating system	Vocal tract: throat (pharynx), oral cavity, nasal passages	Changes the "buzzy sound" into a person's recognizable voice

The term velopharyngeal insufficiency or rhinolalia aperta is to describe the failure of apposition of the soft palate and or pharyngeal wall during speech. This is the main cause of hypernasality. In hypernasality, air escapes into the nose causing difficulty with speech, especially high pressure consonants such as plosives and fricatives. However, in the clinical management of these disorders, various aetiologies require different management approaches.

This study is on primarily on the valving abnormality and its association with speech pathology in patients with cleft palate and following surgeries for its correction.

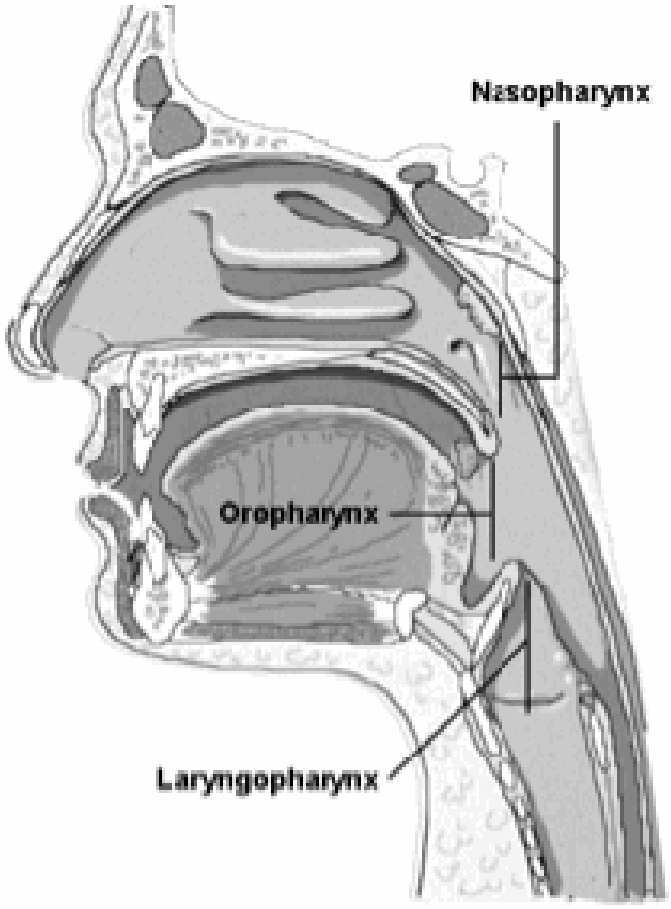


Figure 2

Anatomy:

The levator veli palatini muscle is the primary muscle in the function of velum as reported by an anatomical study [1]. This study of the levator veli palatini, palatopharyngeus, and superior constrictor muscles in 18 fresh cadaveric specimens of normal adults was done to analyze current controversies in velopharyngeal function and cleft palate surgery. The levator veli palatini was observed to form a muscular sling, suspending the velum from the cranial base. Its fibres occupied the middle 50 percent of the velum, lying in transverse orientation and without significant overlap across the midline. It is well placed to function as the prime mover in the velar component of velopharyngeal closure. The velar component of the palatopharyngeus consisted of two heads clasping the levator and inserting into the latter just short of the midline. Its pharyngeal component inserted into the superior constrictor in the lateral and posterior pharyngeal walls. Together, these two muscles formed a sphincter around the velopharyngeal port, suggesting that both muscles are involved in the pharyngeal component of velopharyngeal closure [16].

1. Nasopharynx(Figure 2)

The nasopharynx lies above the soft palate and behind the posterior nares (choanae) which allow free respiratory passage between the nasal cavities and the nasopharynx. The nasal septum separates the two posterior nares , each of which measures approximately 25 mm vertically and 12 mm transversely. Just within these openings lie the posterior ends of the inferior and middle turbinates. Except for the soft palate the walls of the nasopharynx are static and its cavity is never obliterated, in which respect it differs from the oral and laryngeal parts and resembles the nasal

cavities. Between the posterior border of the soft palate and the posterior pharyngeal wall the nasal and oral parts of the pharynx communicate through the pharyngeal isthmus, which is closed during swallowing by the elevation of the soft palate and constriction of the palatopharyngeal sphincter. The nasopharynx has a roof, a posterior wall, two lateral walls and a floor.

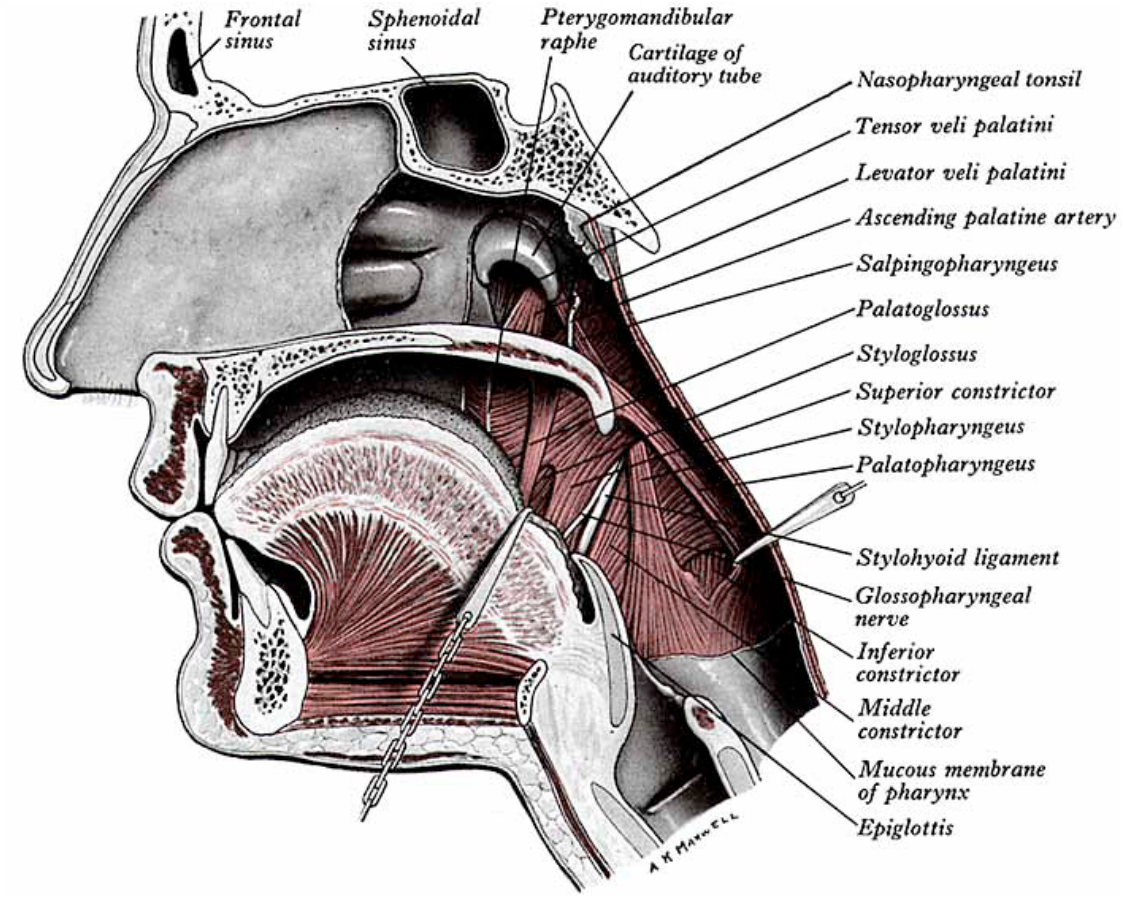


Figure 3[1]

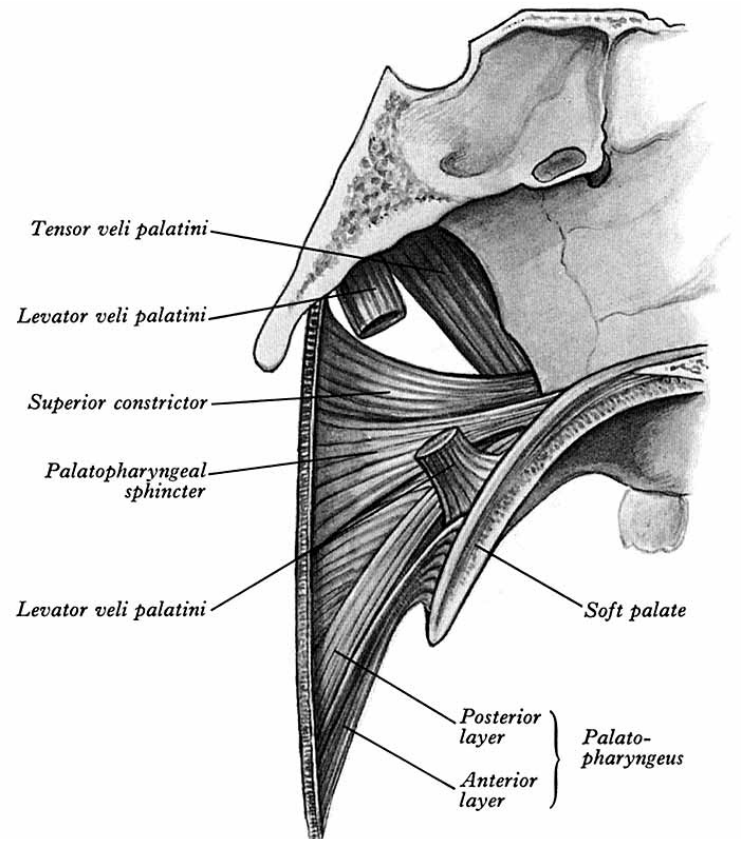

Tensor veli palatini
Levator veli palatini
Superior constrictor
Palatopharyngeal sphincter
Levator veli palatini
Soft palate
Posterior layer
Anterior layer
Palato-pharyngeus

Figure 4[1]

The roof and posterior wall together form a continuous, concave slope leading down from the nasal septum to the oropharynx, bounded above by mucosa overlying the posterior part of the body of the sphenoid and further back by the basilar part of the occipital bone as far as the pharyngeal tubercle. Following the posterior wall further downwards, the mucosa overlies the pharyngobasilar fascia and the upper fibres of the superior constrictor, and behind these, the anterior arch of the atlas. A lymphoid mass, the nasopharyngeal tonsil, lies in the mucosa of the upper part of the roof and posterior wall in the midline.

The lateral walls of the nasopharynx have a number of important surface features. On either side each receives the opening of the Pharyngotympanic tube (also termed the auditory or Eustachian tube), situated 10–12 mm behind and a little below the level of the inferior nasal turbinate's posterior end .The tubal aperture is approximately triangular in shape, bounded above and behind by the tubal elevation consisting of mucosa overlying the protruding pharyngeal end of the tubal cartilage; the prominent posterior margin of this elevation facilitates the introduction of catheters passed along the floor of the nasal cavity for the intubation of the Pharyngotympanic tube.

Behind the tubal opening, a vertical salpingopharyngeal fold of mucosa descends from the tubal elevation, covering the salpingopharyngeus muscle in the wall of the pharynx. In front of the aperture, a smaller salpingopalatine fold extends from the anterosuperior angle of the tubal elevation to the soft palate. The levator veli palatini, entering the soft palate, produces an elevation of the mucosa

immediately below the tubal opening . In the mucosa immediately posterior to the opening of the pharyngotympanic tube is a small mass of lymphoid tissue, the (bilateral) tubal tonsils. Further behind the tubal elevation the lateral wall has a variable depression, the pharyngeal recess or fossa of Rosenmüller. The floor of the nasopharynx is formed by the upper surface of the soft palate.

Superior Constrictor[1] **:**(Figure 3) It is a quadrilateral sheet of muscle, thinner and paler than the other two constrictors, attached anteriorly to the pterygoid hamulus (and sometimes to the adjoining posterior margin of the medial pterygoid plate), the pterygomandibular raphe, below to the posterior end of the mylohyoid line of the mandible and by a few fibres to the side of the tongue .

Its fibres curve back into the median pharyngeal raphe; some are also prolonged by an aponeurosis to the pharyngeal tubercle on the basilar part of the occipital bone, the superior fibres curving under levator veli palatini and the pharyngotympanic tube and leaving an interval below the cranial base for passage of the pharyngotympanic tube. This interval is limited anteriorly by the medial pterygoid plate and closed by the pharyngobasilar fascia .A constant band of muscle sweeps backwards from the anterolateral part of the upper surface of the palatine aponeurosis, lateral to levator veli palatini, to blend internally with the superior constrictor near its superior border). This band is the palatopharyngeal sphincter; it ridges the pharyngeal wall (ridge of Passavant) visibly when the soft palate is elevated. It is hypertrophied in cases of complete cleft palate. The change from columnar, ciliated, 'respiratory' epithelium to stratified, squamous epithelium on the superior palatal aspect occurs at the attachment of the palatopharyngeal sphincter to the palate.

Soft Palate[1]

The soft palate is a mobile flap suspended from the posterior border of the hard palate, sloping down and back between the oral and nasal parts of the pharynx. It is a thick fold of mucosa enclosing an aponeurosis, muscular tissue, vessels, nerves, lymphoid tissue and mucous glands. In its usual position, relaxed and pendant, its anterior (oral) surface is concave, with a median raphe; its posterior aspect is convex and continuous with the nasal floor. Its anterosuperior border is attached to the hard palate's posterior margin, its sides blend with the pharyngeal wall and its inferior border is free, hanging between the mouth and pharynx. A median conical process, the uvula, projects downwards from its posterior border; the palatal arches, two curved folds of mucosa containing muscle, descend laterally from each side of the soft palate . The anterior of these, the palatoglossal arch, contains the palatoglossus muscle and descends to the side of the tongue at the junction of its oral and pharyngeal parts, forming the lateral limits of the oro-pharyngeal isthmus. The posterior palatopharyngeal arch contains the palatopharyngeus muscle, and descends on the lateral wall of the oropharynx. The isthmus of the fauces is the aperture between the oral cavity and oropharynx guarded on either side by the palatoglossal folds. Just behind and medial to each upper alveolar process, in the lateral region of the anterior part of the soft palate, a small bony prominence can be felt. This is produced by the pterygoid hamulus, an extension of the medial pterygoid plate The pterygomandibular raphe, a tendinous band interposed between buccinators and the superior constrictor muscle, passes downwards and outwards from the hamulus to the posterior end of the mylohyoid line. When the mouth is opened wide, this raphe elevates a fold of mucosa which marks internally the posterior boundary of the cheek.

Palatine Aponeurosis[1]

A thin, fibrous palatine aponeurosis supports the muscles and strengthens the soft palate; it is attached to the posterior border and inferior surface of the hard palate behind the palatine crest . It is thick in the anterior two-thirds of the soft palate but very thin further back. It is composed of the expanded tendons of the tensores veli palatini; near the midline it encloses the musculus uvulae. All the other palatine muscles are attached to it. The anterior third of the soft palate contains little muscle, consisting mainly of the palatine aponeurosis, inferior to which are many mucous glands; this region is less mobile and more horizontal than the rest of the soft palate and is the chief area acted upon by the tensores veli palatini.

Muscles of the Palate (Figure 4)[1]:

The muscles of the palate are classified into

1) Intrinsic

2) Extrinsic

The Extrinsic include the levator veli palatini, tensor veli palatini, palato pharyngeous, The only intrinsic muscle is the musculus uvulae

Levator Veli Palatini[1]

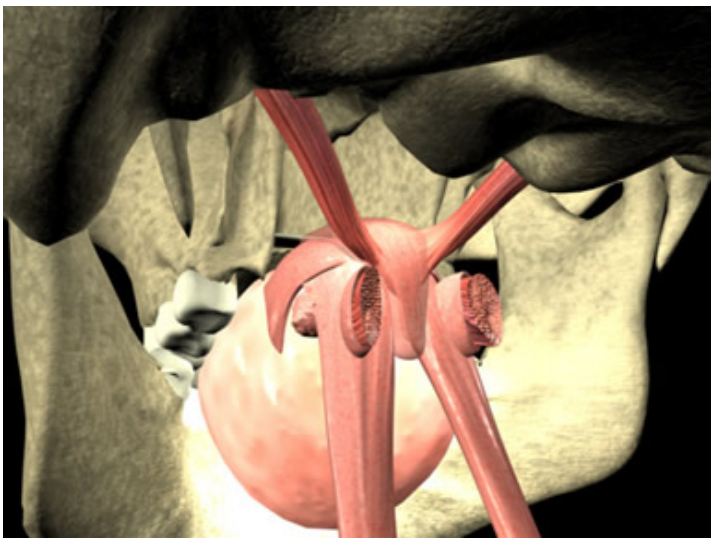
This muscle is cylindrical and lies lateral to the posterior nasal aperture. It's attachements are

- by a small tendon on the inferior surface of the petrous temporal bone(in front of the lower opening of the carotid canal)

- by muscle fibres to a sheet of fascia descending from the vaginal process of the tympanic bone to form the upper part of the carotid sheath

- by a few fibres to the inferior aspect of the cartilaginous part of the pharyngotympanic tube.

At its origin the muscle is inferior rather than medial to the pharyngotympanic tube and only crosses medial to it at the level of the medial pterygoid plate. Passing medial to the upper margin of the superior constrictor and in front of salpingopharyngeus, its fibres spread in the medial third of the soft palate between the two strands of the palatopharyngeus, its fibres being attached to the upper surface of the palatine aponeurosis as far as the midline where they interlace with those of the contralateral muscle. Thus the two levator muscles form a sling above and just behind the palatine aponeurosis.



The primary role of the levator veli palatini is to elevate the almost vertical posterior part of the soft palate and pull it slightly backwards. During swallowing, the soft palate is at the same time made rigid by the contraction of the tensores veli palatini and touches the posterior wall of the pharynx, thus separating the nasopharynx from the oropharynx. By additionally pulling on the lateral walls of the nasopharynx posteriorly and medially, the muscles also narrow that space. The levator veli palatini has little or no effect on the pharyngotympanic tube. [1]

Tensor Veli Palatini[1]

This is a thin, triangular muscle, lateral to the medial pterygoid plate, pharyngotympanic tube and levator veli palatini. Its lateral surface contacts the upper and anterior part of the medial pterygoid muscle, the mandibular, auriculotemporal and chorda tympani nerves, the otic ganglion and the middle meningeal artery. It is attached to the scaphoid fossa of the pterygoid process and posteriorly to the medial aspect of the spine of the sphenoid; between these two sites it is attached to the anterolateral membranous wall of the pharyngotympanic tube, including its narrow isthmus where the cartilaginous medial two-thirds meets the bony lateral one-third. Some fibres may be continuous with the tensor tympani muscle. Inferiorly, the fibres converge on a delicate tendon which turns medially around the pterygoid hamulus to pass through the attachment of buccinator to the palatine aponeurosis and the osseous surface behind the palatine crest on the horizontal plate of the palatine bone. Between the tendon and the pterygoid hamulus is a small bursa.

Acting together the tensors it tautens the soft palate, principally its anterior part, depressing it by flattening its arch. Alone, the muscle pulls the soft palate to one

side. Although contraction of the tensors will slightly depress the anterior part of the soft palate, it is often assumed that the increased rigidity aids palatopharyngeal closure. However, it is now believed that the primary role of the tensor is to open the pharyngotympanic tube, for example during deglutition and yawning, thereby equalizing air pressure with the middle ear and nasopharynx. [1]

Musculus Uvulae[1]

A bilateral structure, this arises from the posterior nasal spine of the palatine bone and the dorsal surface of the palatine aponeurosis, between the two laminae of which the uvular muscles lie. It runs posteriorly above the levator sling to insert beneath the mucosa of the uvula. A paired structure at its anterior and posterior attachments, for most of its length the two sides are united. It Elevates and retracts the uvula by thickening the middle third of the soft palate and it aids the levators in palatopharyngeal closure. Running at right angles to each other, contraction of the levatores and musculi uvuli raises a 'levator eminence' which seals off the nasopharynx 'like a cork in a bottle'..

Palatoglossus

This is a small fasciculus narrower at its middle than at its ends and forming, with the mucosa overlying it, the palatoglossal arch or . It arises from the oral surface of the palatine aponeurosis about half-way along the soft palate where it is continuous with its fellow and extends forwards, downwards and laterally in front of the palatine tonsil to the side of the tongue; some of its fibres spread over the dorsum of the tongue, others passing deeply into its substance to intermingle with the transversus linguae. Palatoglossus elevates the root of the tongue and

approximates the palatoglossal arch to its fellow, thus shutting off the oral cavity from the oropharynx.

Palatopharyngeus [1]

This forms, with its overlying mucosa, the palatopharyngeal arch . Within the soft palate it is composed of two fasciculi which are attached to the upper surface of the palatine aponeurosis in the same plane but separated from each other by levator veli palatini. The thicker anterior fasciculus is attached to the posterior border of the hard palate as well as to the aponeurosis where some fibres interdigitate across the midline. The posterior fasciculus is in contact with the mucosa of the pharyngeal aspect of the palate; it joins the posterior band of the opposite muscle in the midline. At the soft palate's posterolateral border the two layers unite and are joined by fibres of salpingopharyngeus. Passing laterally and downwards behind the tonsil, palatopharyngeus descends posteromedial to and in close contact with stylopharyngeus, to be attached with it to the posterior border of the thyroid cartilage. Some fibres end on the side of the pharynx, attached to pharyngeal fibrous tissue and others cross the midline posteriorly, decussating with those of the opposite muscle. The palatopharyngeus thus forms an incomplete internal longitudinal muscular layer in the wall of the pharynx. Together, the palatopharyngei pull the pharynx up, forwards and medially, thus shortening it during swallowing. They also approximate the palatopharyngeal arches and draw them forwards.

Vessels[1]

The arteries of the palate are the greater palatine branch of the maxillary artery, the ascending palatine branch of the facial artery and the palatine branch of

the ascending pharyngeal artery. The veins drain largely to the pterygoid and tonsillar plexuses. The lymph vessels pass to the deep cervical lymph nodes.

Nerves[1]

The sensory nerves issue from the greater and lesser palatine, and nasopalatine branches of the maxillary nerve, and also the glossopharyngeal nerve (posteriorly). The lesser palatine nerve also contains taste fibres of facial nerve (greater petrosal) origin supplying taste buds in the oral surface of the soft palate. Parasympathetic postganglionic secretomotor fibres arising from the facial nerve via the pterygopalatine ganglion run with these nerves to the palatine mucous glands; it is also possible that some parasympathetic fibres pass to the posterior parts of the soft palate from the glossopharyngeal nerve, perhaps synapsing in the otic ganglion. Sympathetic fibres run from the carotid plexus along arterial branches supplying this region.

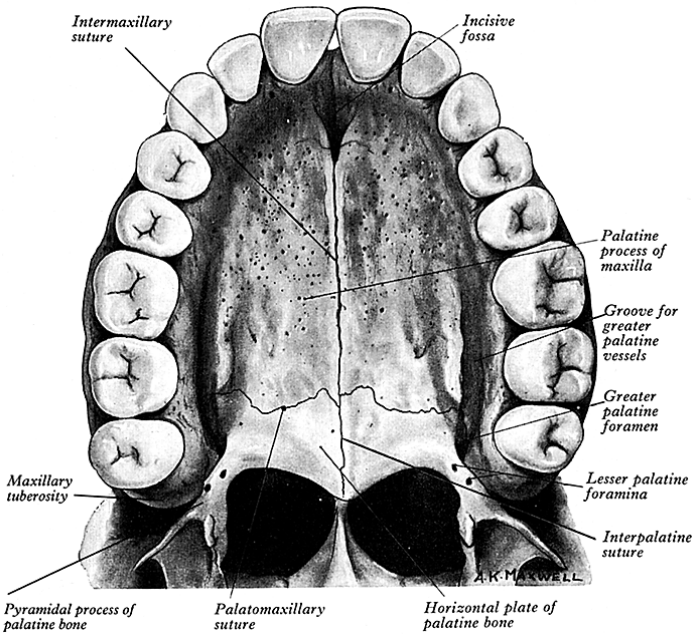
Nerve Supply of Palatine Muscles

Except for tensor veli palatini, which is innervated by the mandibular nerve , all the palatine muscles are supplied by nerve fibres which leave the medulla in the cranial part of the accessory nerve and reach the pharyngeal plexus via the vagus nerve and possibly the glossopharyngeal. More controversially, several investigators have suggested that levator veli palatini is also supplied by the facial nerve. Ibuki et al in 1978[17]reported electromyographic evidence that in monkeys this motor route involves the greater petrosal nerve, pterygopalatine ganglion and lesser palatine nerves. In contrast, Keller at al (1984), using [18]retrograde axonal transport in cats, found levator veli palatini motor neurons in the nucleus ambiguus but not in the facial

nucleus. These authors also confirmed that tensor veli palatini motor neurons are situated in the trigeminal motor nucleus.

Bones of the Plate[1]:

The pre maxilla is situated anterior to the incisive foramen and includes the anterior nasal spine and four incisor teeth. The paired maxillae form the anterior portion of the palate, and the paired palatal bones form the posterior palate including the posterior nasal spine. The palatine bone articulates with the medial plate of the pterygoids, from which projects the pterygoids hamulus, which acts as a pulley for the tensor velli palatine



Summary of Soft Palate Muscle Attachments

In the soft palate the muscles are arranged as follows: the palatine aponeurosis (tendon of the tensores veli palatini) is an intermediate sheet, enclosing the uvular muscles near the midline; the levatores veli palatini and the palatopharyngi are attached to its upper surface, the two fasciculi of the latter lying in the same plane, one in front of and the other behind levator veli palatini. The palatoglossi are inserted into the inferior surface of the aponeurosis.

Embryology:

The head is composed of the skull surrounding the brain, and an outer covering of muscles, glands and skin. The skull has two distinct portions: that surrounding the brain and special sense organs—the neurocranium—and the lower face and jaws (also the palate, hyoid, epiglottis and larynx)—the viscerocranium. Each part derives from different mesenchymal populations and by different methods. The neurocranium develops from the paraxial mesenchyme in the head, i.e. the first five somites and the unsegmented somitomeres rostral to the first somite [19], and from ectoderm via the neural crest. The basal portion of the skull is similar in structure and development to the vertebral column and is preformed in cartilage. The viscerocranium derives from ectoderm via invaginated head neural crest which streams into the developing arches forming all the connective tissue elements of the face. Bones of the viscerocranium form in the main from membranous ossification but there are cartilage models in each arch. The contribution of neural crest to the neurocranium in mammals is not yet clear, although it has been established in the chick that neural crest mesenchyme gives rise to the large bones lateral and dorsal

to the brain by membranous ossification. Lateral plate mesenchyme does not extend into the head.

The Pharyngeal Arch

Generally each pharyngeal arch consists of an epithelial covering exteriorly and a mesenchymal core interiorly. The epithelium may be ectodermal entirely (as in the first arch), or ectoderm covering the external aspect of the arch and endoderm covering the internal aspect of the arch (as in the remaining arches). The mesenchyme within each arch derives from neural crest, paraxial and angiogenic mesenchymal populations. The motor and sensory roots of a cranial nerve are associated with the epithelium and mesenchyme of each arch.

Development of the Pharyngeal Arches[1]

The human circumoral first pharyngeal arch consists, on each side, of two main regions: a ventral part or mandibular prominence and a dorsal part or maxillary prominence. Each mandibular prominence, first seen at stage 10 (22 postovulatory days), grows ventromedially in the floor of the pharynx to meet its fellow in the midline, being situated between the primitive mouth and the cardiac (pericardial) prominence. The maxillary prominences are not seen until stage 13; their enlargement coincides with proliferation of neural crest mesenchyme between the ectoderm and prosencephalon forming the frontonasal prominence. The enlargement of the first arch is particularly rostral to the site of the buccopharyngeal membrane; thus inner and outer aspects of this arch are covered with ectoderm. The second or hyoid arches, seen from stage 11, are caudal to the maxillomandibular; they similarly grow ventrally to meet and fuse in the midline. The third arches are

seen at stage 12 (26 days) and the fourth arches by stage 13 (28 days); the latter especially are not prominent, being largely sunk in a depression produced by the caudal overlapping of the hyoid arch. The fifth and sixth arches cannot be recognized externally and can only be identified by the arrangement of the mesenchyme and by slight projections into the pharynx.

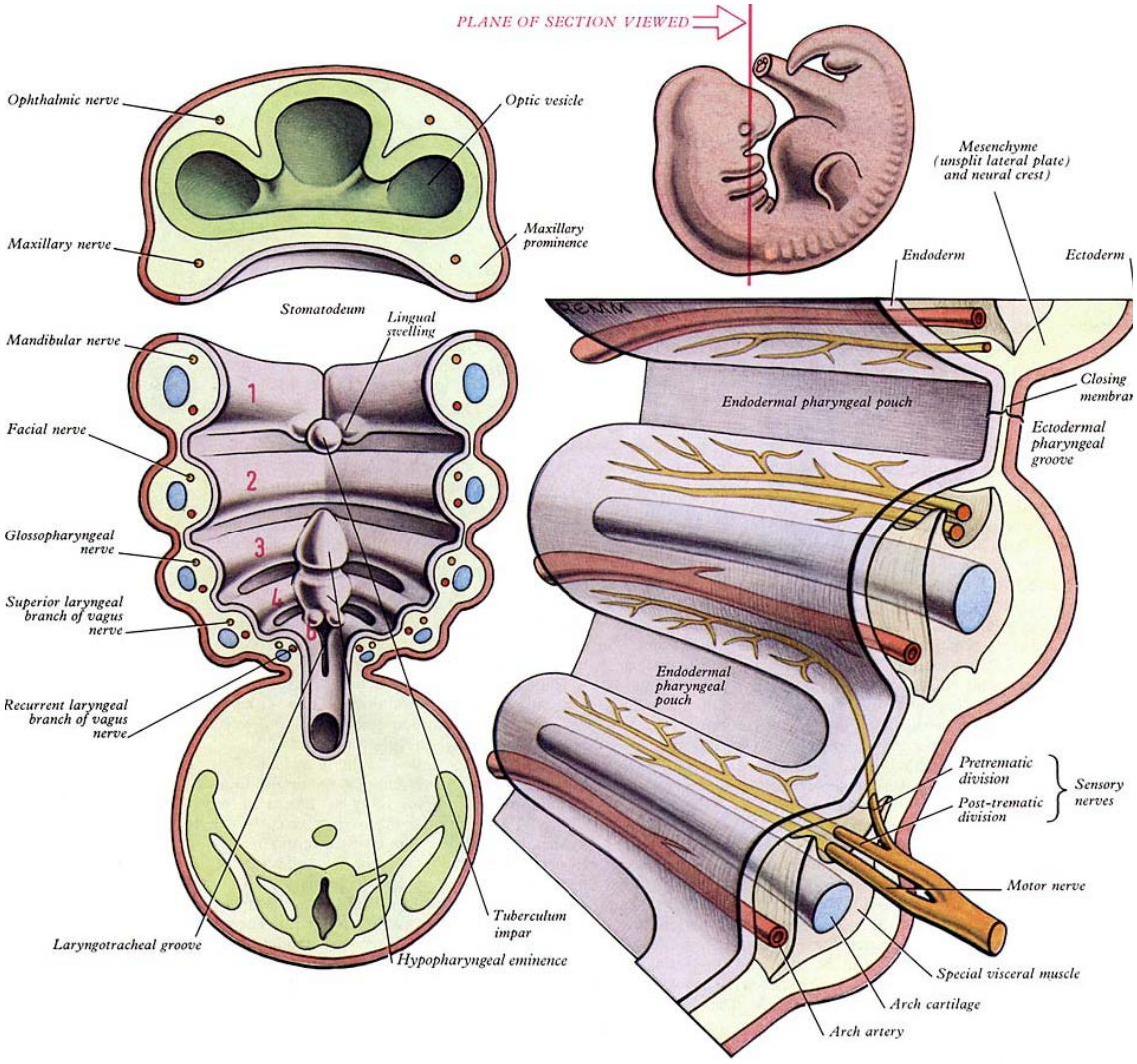


Figure 6: Pharyngeal arches[1]

The First Pharyngeal Arch

The first pharyngeal arch is sufficiently different, both in its structure and development, from the subsequent caudal arches for its separate examination. Unlike the other arches it possesses dorsal and ventral prominences, appearing C-shaped in lateral view. The dorsal (maxillary) prominences interact with ectodermal epithelia and neural crest mesenchyme of the frontonasal prominence, and generally form more extensive skeletal structures than the other arches; particularly, these skeletal elements fuse with the chondrocranium. The first arch is completely clothed with ectoderm unlike the caudal arches which are dependent on the proximity of pharyngeal endoderm for their development. The ectoderm originates (in the 3-somite chick) from a territory lateral to the mesencephalic neural folds (see figure 7)The mesencephalic folds themselves give rise to both the ectodermal placodal cells and neural crest cells which contribute to the trigeminal ganglion, and the mesenchymal population which streams into the mandibular and maxillary prominences.

The first arch contains on each side a dorsal and ventral cartilage. The former represents the palatopterygoquadrate bar, a prominent element in earlier vertebrates forming part of the upper jaw but much reduced in mammals. The ventral cartilage (of Meckel) extends from the developing otic capsule into the mandibular prominence, meeting its fellow at its ventral end. The dorsal end of Meckel's cartilage becomes separated, and was often held to form the rudiments of both malleus and incus.

The cells which give rise to the muscle of the first arch arise from the paraxial mesenchyme localized to somites 2 and 3. The muscle mass of the mandibular part of the first arch forms the tensor tympani, tensor veli palatini and the masticatory muscles, including mylohyoid and the anterior belly of digastric .The tensor tympani retains its connection with the skeletal element of the arch through its attachments to the malleus, and the tensor veli palatini to the base of the medial pterygoid process, which may be derived from the dorsal cartilage of the first arch, but the masticatory muscles transfer to the mandible, a dermal bone. All these muscles are supplied by the mandibular nerve, the mixed 'post-trematic' nerve of the first arch.

Face (Figure 7)

While the mandibular prominences are invading the floor of the pharynx, mesencephalic neural crest cells migrate rostrally and laterally between the prosencephalic neuroepithelium and the surface ectoderm to form the extensive frontonasal prominence. During the fifth week the sites of the olfactory or nasal placodes are established ventrolateral to the frontonasal prominence, dividing the latter, on each side, into medial and lateral nasal prominences or folds; the olfactory placodes originate from the neural folds . The placodes are at first widely separated and coplanar with the surface ectoderm but, as the nasal prominences develop, they soon become depressed to form the olfactory pits (nasal sacs). The lateral nasal prominences are the more evident, but the medial nasal prominences, still separated by the median remainder of the frontonasal field, project caudally beyond the former. Extensions of mesenchyme from the medial prominence into the roof of the stomodeum proliferate to form the premaxillary fields. Each nasal sac has a ventral fold from which develops an epithelial nasal fin passing caudally to fuse with the

While these changes are progressing a somewhat triangular elevation swells ventrally from the cranial aspect of the dorsal region of each mandibular prominence. This is the maxillary prominence, and like the frontonasal prominence it consists of proliferating neural crest mesenchyme covered by ectoderm. Each maxillary prominence grows in a ventral direction and fuses with the lateral nasal prominence, the two being at first separated by a nasomaxillary groove (naso-optic furrow). The opposed margins of the lateral nasal and maxillary prominences growing together thus establish continuity between the side of the future nose and the cheek. The ectoderm along the boundary between them does not entirely disappear; it gives rise to a solid cellular rod, which at first develops as a linear surface elevation, the nasolacrimal ridge, and then sinks into the mesenchyme . Its caudal end proliferates to connect with the caudal part of the lateral nasal wall, while its cranial extremity later connects with the developing conjunctival sac. The solid rod becomes canalized to form the nasolacrimal duct . The relatively wide primitive mouth or stomodeal fissure is progressively reduced, and the epithelial and connective tissues of the cheek enlarged, by fusion between the adjacent surfaces of the mandibular and maxillary prominences. This proceeds from the para-otic region to the angle of the definitive oral fissure.

Nasal Cavity[1]

The rounded apex of the triangular maxillary prominence extends beyond the lateral nasal prominence, crossing the caudal end of the olfactory pit to meet and fuse with the premaxillary elevation developing at the extremity of the frontonasal field. This closes off the lower or caudal edge of the olfactory pit, the upper part of the opening of which is thus defined as the primitive external naris. The growth of the

surrounding mesenchyme leads to a deepening of the pit to become a primitive nasal cavity, or nasal sac, the epithelial wall of which, in the dorsocaudal part of its extent, the nasal fin, retains contiguity with the epithelium of the stomodeal roof. This contact area becomes progressively greater as growth continues, and the nasal fin is eroded, ultimately forming a thin layer, the oronasal membrane, which also disappears later. By these changes a new cranial boundary is set for the oral opening, consisting of the fused premaxillary and maxillary regions. This is the future upper lip, but it has not yet become separated from the deeper tissues which will form the maxillary alveolus. At the same time the nasal cavity acquires a floor through the fusion of the nasal prominences and the maxillary prominences. At this stage the two external nares are still widely separated by an area derived from the frontonasal field, but this separation becomes reduced by the fusion of the premaxillary mesenchyme from the two sides. The maxillary mesenchyme is thus considered by some to contribute substantially to the formation of the philtrum of the upper lip, thus accounting for its maxillary innervation.

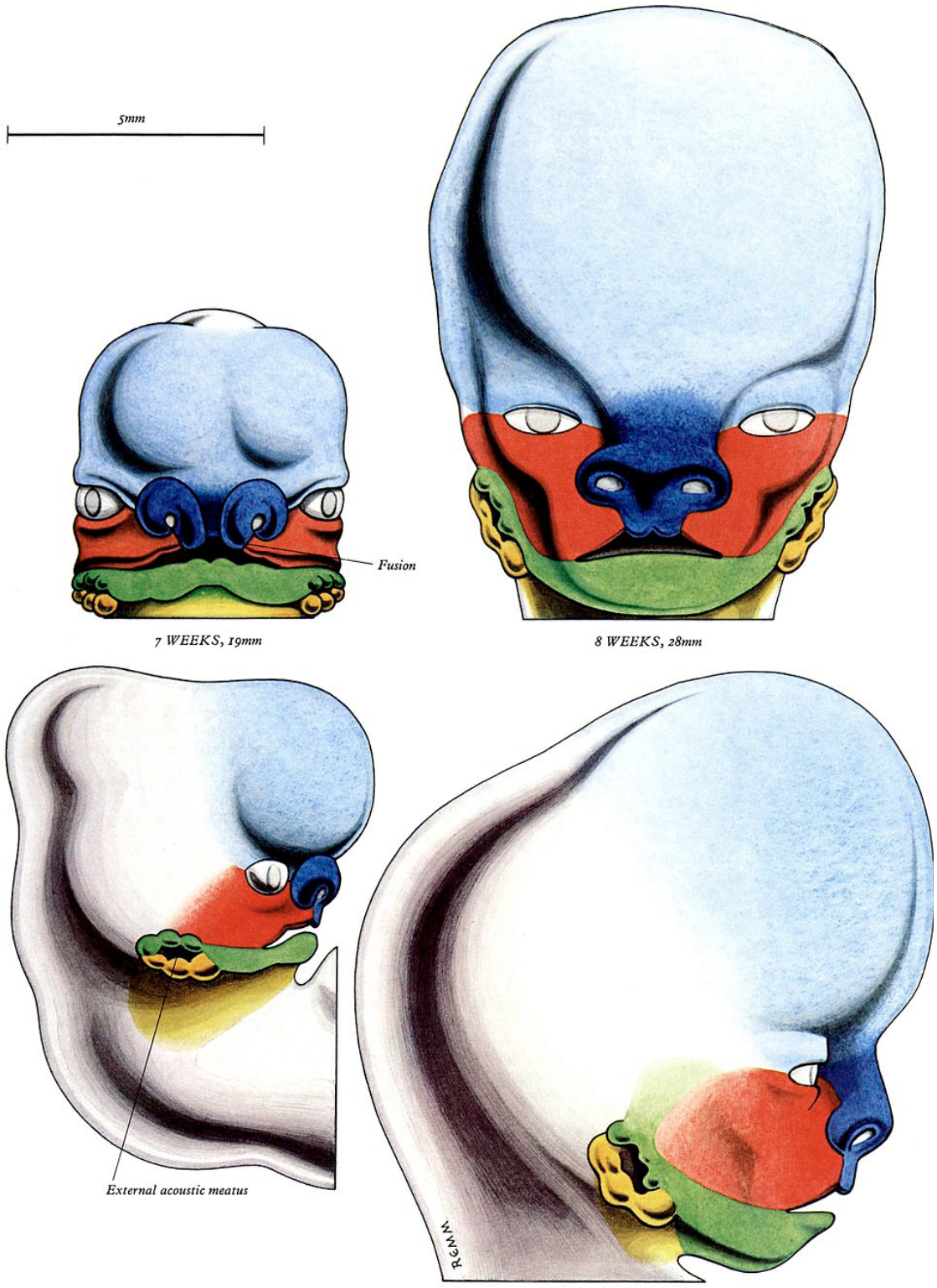

5mm
Fusion
7 WEEKS, 19mm
8 WEEKS, 28mm
External acoustic meatus
REMM

Figure 7[1]

Palate[1]

Once the primitive nasal cavities are defined the ventral part of the roof of the oral cavity can be regarded as the primitive palate (median palatine prominence). It is formed by the premaxillary regions and maxillary prominences, which become confluent and establish continuity with the thick median nasal septal prominence (primitive nasal septum). As the head grows in size, the region of mesenchyme between the forebrain and oral cavity increases greatly by proliferation and the nasal cavities deepen, extending towards the forebrain. Simultaneously they also extend dorsally from the primitive choanae as two narrow and deep grooves in the oral roof which are separated by a partition. The grooves and the partition deepen together, and the latter becomes the nasal septum, continuous rostrally with the primitive nasal septum. The broad dorsocaudal border of the nasal septum is at first in contact with the dorsum of the developing tongue , the right and left nasal cavities still communicating freely with the mouth except where the nasal floor is already established ventrally by the primitive palate.

At 41 days the internal aspects of the maxillary prominences produce palatine processes (shelves), which grow towards the midline but are for some time separated from each other by the tongue. At this stage the roof of the oral cavity projects ventrally beyond its floor and the tip of the developing tongue actually lies in contact with the cranial (superior) surface of the primitive palate. With further growth, the mandibular region and the tongue are carried forwards (ventrally), and the lingual tip passes round to the caudal surface of the primitive palate. At 56–57 days the palatine processes rapidly elevate, assuming a horizontal position which allows them to grow towards each other and thus to fuse .

The change of position occurs very rapidly caused by the progressive region specific synthesis and accumulation of hyaluronic acid within the palatal process mesenchyme. The hyaluronic acid will bind up to 10 times its own weight of water, thus causing swelling and expansion of the palatial shelves. This process is further aided by the alignment of collagen fibrils and palatal mesenchymal cells (the latter contract in response to acetylcholine and serotonin which they secrete thus regulating the elevation of the shelves), and by the epithelium which restrains the swelling. Once these forces are in concert and exceed the resistance factors, the palatal shelves will mechanically elevate. Such elevation occurs at a time of craniofacial growth when there is constant growth in head height but almost no growth in head width. This latter factor is important: if palatal shelf elevation is delayed so that they elevate in a period of growth in facial width, the unfused processes are unable to touch physically and cleft palate may result. Other factors affecting palatal closure are the growth in length of the first arch cartilage (Meckel's) which allows the tongue to lower into the developing mandible. Further, the change in position of the maxilla relative to the anterior cranial base, which is maintained at about 84° during weeks 9 and 10, has the effect of lifting the head and upper jaw upwards from the mandible so permitting withdrawal of the tongue from between the palatal shelves and creating space for them to elevate. Mouth opening, tongue protrusion and hiccup movements have also been noted at this time; these movements and their associated pressure changes may assist palatal shelf elevation. Generally in female embryos palatal shelf elevation occurs 7 days later than in males, making congenital cleft palate more likely in female embryos. After elevation the palatine processes grow medially along the inferior borders of the primitive choanae, uniting with them and with the margins of the median palatine

prominence, except over a small area in the midline where a naso-palatine canal maintains connection between the nasal and oral cavities for some time and marks the future position of the incisive fossa. (The plates which form the early (primitive) palate are sometimes known as median palatine processes, the maxillary contributions being then named the lateral palatine processes.)

As the medial borders of the maxillary palatine processes fuse together, fusing also with the free border of the nasal septum, the nasal and oral cavities are progressively separated and the tongue is excluded from the former. The nasal cavities are thus extended dorsally and the choanae reach their final position, leaving the caudal edge of the nasal septum free in about its dorsal quarter as the partition between them. Slightly later the dorsomedial extremities of the palatine processes, which extend dorsally beyond the choanae, fuse together rostrocaudally to form the future epithelia and connective tissues of the soft palate. There is later an upgrowth of myogenic mesenchyme from the third and, probably, other pharyngeal arches into the palate and around the caudal margins of the auditory tube, along a line corresponding in the final state to the palatopharyngeal arches.

Embryology of Naso-pharynx:[1]

In the neonate this is one-third of the relative length in the adult. The nasopharynx is a narrow tube which curves gradually to join the oropharynx without any sharp junctional demarcation. An oblique angle is formed at this junction by 5 years of age and in the adult the nasopharynx and oropharynx join at almost a right angle.

Anatomical defects of the Palate

The condition of congenital cleft palate has been noted already as a developmental defect. Rarely, palatopharyngeal incompetence may be due to muscle hypoplasia, particularly of the musculus uvulae; submucous clefts resulting from this may be revealed clinically as a V-shaped notch in the midline of the soft palate during function. Paralysis of the soft palate may follow diphtheria due to the action of the toxin on the nerve cells of the medulla oblongata; in this state, the voice becomes nasal and fluids regurgitate into the nose during swallowing; the palate is visibly flaccid and motionless and also anaesthetic. Other pathological processes involving the glossopharyngeal, vagus and accessory nerves or their nuclei in the medulla oblongata also cause palatal paralysis.[1]

Classification of Cleft Palate:

Davies & Ritchie [20, 21] reported one of the earliest system of classification based on morphological features of alveolar defects in to 3 groups, Veau in 1931 proposed a 4 group classification system based on morphologic features: Group 1: lefts of the soft palate only, Group 2: Clefts of soft and hard palate, Group 3: complete unilateral cleft of lip, alveolus and palate, Group 4: bilateral complete cleft.

Pruzansky[22] proposed a classification based on individual structures as lip only(incomplete/complete) and palate alone, but alveolus was left alone. The fourth category in this classification, congenital insufficiency of the palate, comprised velopharyngeal in sufficiency of any aetiology (e.g. submucucous clefts of the palate, congenitally short palate).

Kernahan and Stark[23] developed a classification based on embryological development, in a effort to unify the various efforts the American Association for cleft palate rehabilitation [24]formed a nomenclature committee which classified cleft palate:

Pre-palate

Lip

Alveolar process (to incisive foramen)

Palate

Soft Palate

Hard Palate (to incisive foramen)

As with other classification systems, provisions were made for Velopharyngeal insufficiency. Over time many other classification and documentations have been proposed, each has their merits and deficiency

ENT pathology in Cleft Palate patients

Children born with cleft palate face many ENT problems, majority of them have Recurrent Otitis media (ROM) or Secretory Otitis media (SOM). The incidence ranges from 40-100% & this causes a mild-to-moderate conductive hearing loss which can result in impaired speech, language and even cognitive development. Nasal problems include reduction in the airway and mouth breathing. Voice and

articulation are very prevalent which includes laryngeal voice symptoms' such as hoarseness, breathiness, low volume and abnormal pitch. McWilliams et.al[25] reported 59% velopharyngeal insufficiency in CP patients.

Godbersen[26] presented a classification system of the speech disorders in cleft palate children;

1. Primary speech disorder-: Caused by incomplete velopharyngeal closure resulting in hypernasality, weak plosives, fricatives and affricatives

2. Secondary speech disorders: Substitute mechanisms for plosives, fricatives and affricatives

3. Tertiary speech disorders: Functional dysphonias which derive from primary and secondary speech disorders.

Kawano et.al[27]studied the site of origin of articulatory defects in cleft palate children using flexible endoscopy and videofluroscopy. They reported that velopharynx is the primary site of defect for hypernasality and that laryngeal fricatives, affricatives, pharyngeal stop and glottal stop are produced in various other non laryngeal sites.

Articulation of Speech: [28, 29]

It is the precision of speech sound production, is achieved through rapid co-ordination of oral, Velopharyngeal, and laryngeal structures.

Definitions in Speech assessment:

Phonetics is the general study of the characteristics of speech sounds.

Articulatory Phonetics is the study of how speech sounds are made or articulated- this is where the defect in the palate/velopharynx plays a role.

Auditory (or perceptual) phonetics is the perception of the sound/ phonemes (words).

Place of articulation is the placement of the palate and alteration of the various parts of the oral cavity and the laryngo-pharynx during sound production.

Bilabials are sounds produced by using both lips, ex: pat, bat, and mat.

Labiodentals are the sounds produced by using the upper teeth and lower lip,

ex: laugh, cough, initial part of photo.

Dentals are sounds formed with the tongue tip behind the upper front teeth,

ex: the, there, then and thus.

Alveolars are sounds formed with the front part of the tongue on the alveolar ridge,

ex: initial part of top, dip, sit, zoo and nut.

Alveo-Palatals sounds which are produced with the tongue at the very front of the palate, near the alveolar ridge, ex: shoe-brush, church.

Velar are the sounds produced with back of tongue against the velum. The

voiceless sound represented by “k” ex: kill, car, kid, kick, coke, and kaka.

Glottals are sounds that are produced without the active use of the tongue and other parts of the mouth. It is the sound [h] which occurs at the beginning of “have and house”.

Manner of articulation describes the way the sounds are pronounced, for example we can say [t] and [s] are both alveolar sounds. But they differ in the manner of articulation, the sound [t] is one set of sounds called ‘stops’ and the [s] sound is a set called ‘fricatives’.

Stops, the set of sounds [p],[b], [t], [d], [k] and [g] are all produced by some form of complete ‘stopping’ of the air stream(very briefly) and then letting it go abruptly. This type of consonant sound resulting from a blocking or stopping effect on the air stream is called a ‘stop’ or a Plosive.

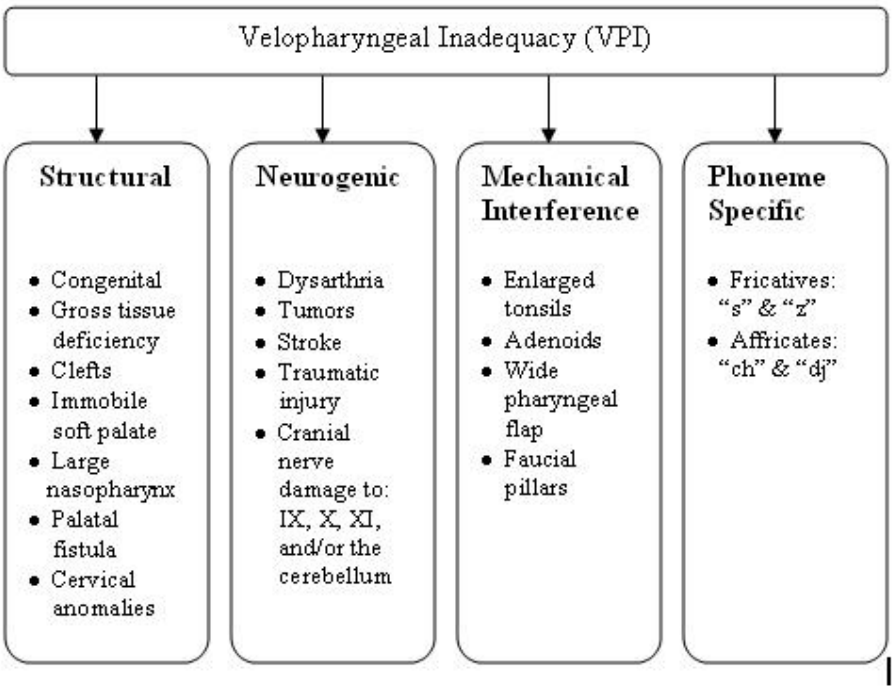
Fricatives are manner of articulation used in producing the set of sounds [f], [v], [z] and [θ], which involves almost blocking the air stream, and having the air push through a narrow opening causing a type of friction.

Affricatives are sounds produced by the combination of brief stopping of the airstream with obstructed release which causes some friction. Ex: at the beginning of words jeep & cheap.

Nasals. Most of the sounds are produced orally, with velum raised, preventing air flow from entering the nasal cavity. However, when the velum is lowered and the airstream is allowed to flow out through the nose to produce [m], [n] and [rj].

Pathology of Velopharyngeal Incompetence (VPI):

There are various types of VPI [15][30]



1. Classified according to its aetiology [3]:

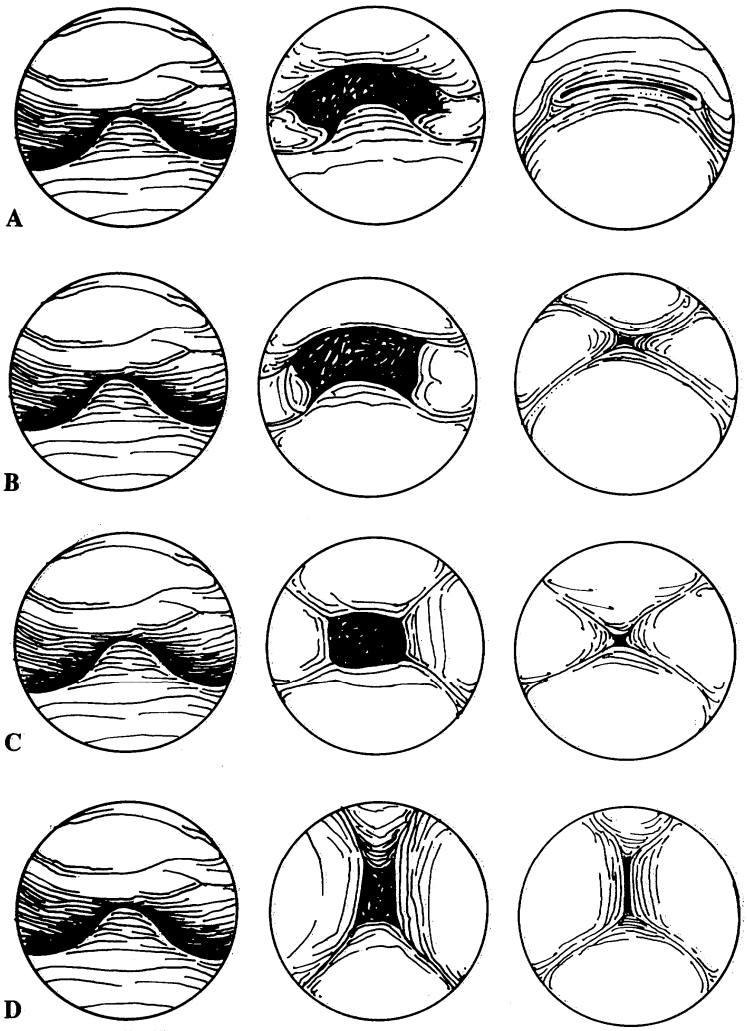
A. Neuro-muscular dysfunction where in there is anatomical deficiency of neuromuscular co-ordination

B. Tissue deficit- as in the absence of the formation of the palate.

2. Based on diagnostic criteria's [4]:

- VP Insufficiency due to structural defect
- VP Incompetence due to neurologic impairment
- VP Mislearning which is caused by neither of the above factors.

The basic pathology in Velopharyngeal incompetence is the defect in the valving mechanism of the velopharynx, either due to structural defect or neuromuscular. Literature describes 4 patterns of primary closure of velum. These patterns are best understood by thinking of the residual gap orientation if the velopharynx does not have complete closure. The defect which are commonly seen:



The velopharyngeal valve as seen on nasopharyngoscopy at rest and during partial and complete closure. The posterior pharyngeal wall and adenoid tissue are at the top of the circle and the soft palate is at the bottom. *A*, Coronal pattern; *B*, Circular pattern; *C*, Circular pattern with a Passavant's ridge; *D*, Sagittal pattern.

Figure 8

1. Coronal -55%of the population, the palate moves posteriorly without much contribution lateral walls or posterior wall

2. Sagital -10 to 15% of the population leaves a gap oriented towards the sagital because the major contribution to closure is from the lateral walls.

3. Circular closure pattern (10% to 20% of the population) has significant motion of the velum and lateral pharyngeal walls, leaving a circular central gap.

4. Circular closure with a passavant's ridge is seen in 15% to 20% of the population where there is motion of the velum and lateral walls with additional presence of a Passavants ridge which may not contribute the closure. Skolick et al,1973; 1975 [31], Zwitman et al [32], 1976; Sprintzen et al,1977[33]; Croft et al, 1981[34].

The closure pattern may assist in prescribing surgical or prosthetic intervention. knowing where the "gap" is, helps one to decide how best to obturate the gap.

Speech and Language Defect in VPI:

Children with cleft palate & Lip are mainly affected in 3 major areas[35]:

1. Speech production
2. Language comprehension and
3. Language production

Children with oral clefts are the most affected of all the cleft palate & lip defects and are at higher risk for delays and disorders in language and reading skills compared with their peers [36].

Even after surgical repair, children with oral clefts are at high risk for impaired speech production. Speech production can be broken into three main components: articulation, resonance, and voice quality.

Resonance: Resonance is a quality of speech that is created by shaping the vocal signal with movements of the articulators: the pharynx, velum, tongue, lips and jaw. Surgical repair of the cleft palate restores the normal barrier between the nose and the mouth, which is required for the normal resonance. In English only the words n, m and ng (ex: mama, nana, swing) require nasal resonance created by air flowing through the nose. For all other consonants and vowels, the velum closes against the posterior pharyngeal wall(velopharyngeal valve) to prevent sound energy from leaking into the nose and to maintain oral air flow and pressure. Velopharyngeal valve closure is necessary for high oral pressure sounds that stop the air(ex: b, p, t, d, k, g) and those that maintain constant air flow (ex: f, v, s, z, sh, th).

When there is lack of nasal resonance, as when a person has a cold, the Quality of speech is affected by a decrease in the expected nasality on nasal sounds(hyponasality), so that “maybe” sounds as “baby”. Leaks can be caused by residual fistulae in the hard palate or soft palate after surgery or can occur when the soft palate fails to close against the posterior pharyngeal wall, known as velopharyngeal inadequacy.

Resonance Disorders Frequently Associated with cleft Palate[35]

1. Hypernasal Resonance

- Inordinate nasal resonance during speech
- Nasal quality noticed across vowels
- Occurs due to in appropriate coupling of the oral and nasal cavities produced in the oral cavity in normal speech

2. Hyponasal Resonance

- Reduction in normal nasal resonance
- Occurs due to partial or complete blockage of the nasal airway
- Mixed Resonance (Hyper-Hyponasality)
- Simultaneous Hypernasality and hyponasality
- Occurs when a speaker has both incomplete velopharyngeal closure and high nasal airway resistance

3. Nasal Emission of Air

- Audible nasal air escape during production of consonants that require high oral pressure
- Occurs when air is forced through an incompletely closed velopharynx

Articulation: An articulation disorder involves problems making sounds. Sounds can be substituted, left off, added or changed. These errors may make it hard for people to understand you. Young children often make speech errors. For instance, many young children sound like they are making a "w" sound for an "r" sound (e.g., "wabbit" for "rabbit") or may leave sounds out of words, such as "nana" for "banana." The child may have an articulation disorder if these errors continue past the expected age. Not all sound substitutions and omissions are speech errors. Instead, they may be related to a feature of a dialect or accent. For example, speakers of African American Vernacular English (AAVE) may use a "d" sound for a "th" sound (e.g., "dis" for "this"). This is not a speech sound disorder, but rather one of the phonological features of AAVE[37]. Children with cleft lip and palate are at considerable risk for speech sound errors such as sound distortions related to malocclusion, developmental speech sound disorders, or compensations made because the palate is not working appropriately. Sounds such as s, am or sh that require relatively high oral pressure consonants such as h and w or nasal consonant such as m, n or ng.

Voice Quality: This is produced by the vocal folds, abnormal voice qualities of hoarsness, unusual pitch, breathiness and reduced loudness are more common in the people with cleft palate are thought to be caused by the speaker using the vocal folds to compensate for the nasal air leak in the velopharynx[37].

Clinical Evaluation of VPI

Children with cleft palate visit the paediatrician initially for the birth defect who in turn refers the child to the Plastic surgeon. The Plastic surgeon evaluates the child for the type of cleft and amount of defect at 9 months when the child has adequate weight and if fit enough to be operated corrects the cleft palate. Most of the children do well and develop a good amount of intelligible speech, the rest due to incomplete closure of the velum. They return to the plastic Surgeon with complaints of nasal speech or unclear speech, that is when the role of the Otolaryngologist and his team come in to the picture which includes a As the presence of good hearing mechanism is very essential for a good outcome of speech, the child is evaluated for middle ear pathology, hearing loss, any developmental delay especially if associated with syndromes. In all the above mentioned type of classifications the common denominator is the presence of VPI which needs to be addressed and they often present to the Otolaryngology clinic for evaluation and advice regarding management. The correct approach to treatment of VPI depends upon practical diagnostic method which will give accurate and complete information, failing which inappropriate or inadequate interventions which are potentially dangerous can be done (1).

Protocol for Evaluation (Annexure 1)

(I) Preliminary Diagnostic Measures:

1. Patient's and parent's interview.

2. Auditory perceptual assessment (APA) of the patient's speech.

3. Visual perceptual evaluation (examination) of the vocal and oral tract.

4. Simple clinical tests for the detection of the presence and degree of hypernasality.

(II) Clinical Diagnostic Aids:

1. Visual aids: a. Endoscopies, b. Roentgenological methods.
2. Formal tests for quasi-objective evaluation of the patient's language, articulation, mental, social and cognitive abilities.

Literature describes various techniques for the assessment of VPI, furthermore, the diversity of approaches to surgical management renders evidence regarding speech outcome inconclusive. A recent survey revealed that 201 different European teams used 194 protocols for one cleft subtype, making comparison of outcomes impossible[37], which can be categorized in to 2 group's i.e.

A. In-direct

B. Direct

A. The Indirect methods of Assessments involve quantitative and qualitative measurement of VPI sequelae using various modalities

- Hyper nasal Resonance- measured with Nasometer
- Nasal Air escape/air flow-measured using a heated pneumatochograph
- Perceptual Parameters of speech –assessed by a trained Speech Pathologist

The advantages of the In-direct type of assessment is, it gives the quantity of deficiency but it seldom gives a data regarding the structural inadequacy that is present which can be corrected and the same data finding can be used for a latter comparison.

The Nasometer (Kay-Pentax, Inc. Fig.9) is a device which measures acoustic energy during speech. A ratio of acoustic data is acquired by two microphones, situated on a metal plate on a headset that separates the nose from the mouth. This ratio is termed nasalance and is defined as the acoustic correlate of perceived nasality. Nasalance is displayed as a percentage on the software program, with higher percentages representing increased nasalance.

Although the Nasometer does not give the patient a view of the VP mechanism, it does give auditory feedback coupled with the instrument's real-time visual feedback during therapy or assessment tasks. Other advantages include; it is non-invasive, easy to clean and has an established normative database. The data collected is often useful in documenting patient performance before, during and after therapy, surgery, or prosthetic fitting. The Nasometer can be used in therapy to provide visual, real-time nasalance percentages as the patient reads, talks or repeats prescribed material. One useful protocol is to set a threshold 10% higher than the patient's average nasalance score, while the patient attempts to stay below the set threshold. The threshold is lowered in 5-10% increments as the individual successfully produces the treatment stimuli at or below the target. The use of the Nasometer to reduce nasality was researched by Fletcher [38] and was generally found to be successful with most VPI patients. Results indicated that patients with various VPI conditions were able to decrease nasalance by a significant

margin and perceptual judgment of nasality also decreased . Those with mild, inconsistent hypernasality are the best candidates for nasality reduction using the Nasometer.

The results needs to be verified with various clinical settings, false positive results could occur if there is an opening elsewhere along the vocal tract anterior to the Velopharyngeal valve such as large palatal fistula [(1)]. False negative results can occur in case of patients with significant deviated nasal septum and hypertrophied turbinate (due to allergic rhinitis) which is a common finding in cleft palate patients [(3)].

Figure 9[39]

Nasal Air flow/pressure measurement using a heated Phacometer which involves fixing small catheters in the nares and the oral cavity and record the pressure in a transducer. Thought this measurement gives a quantitative data indicating the structural integrity of the palate, this is cumbersome and the patient should be intellectually capable of obeying the commands and if so so-operative [(3).]

Palatal Efficiency Rating Computed Instantaneously (PERCI):

Nasal airflow can also be detected and monitored through various instruments that measure aerodynamic data such as subglottal pressure, translaryngeal and nasal airflow. The PERCI system was developed by Warren and Dubois in 1964 , and has been modified and packaged to be commercially available [40]The PERCI system is typically used for assessment purposes but can be used during treatment by providing visual feedback of nasal and oral flow during speech production. PERCI is a non-invasive method for assessing and monitoring VP function.

Perceptual Speech Evaluation Perceptual Assessment:

Although review of the current literature shows no universally consistent method of judging speech and defining speech outcomes in VPI, several different methods of success measurement have been used by individual craniofacial anomalies teams. Rating systems for perceptual measures of speech characteristics are almost always used during pre and post surgery assessments. Rating systems that have been reported in the literature include the Cleft Audit Protocol for Speech, or "CAPS" [41], the Base-10 Index [42], and numerous similar rating systems developed by individual centres published a study supporting the use of the paediatric voice outcome speech(PVOS) [43-45]as a functional outcome assessment to measure success following surgery for VPI. These authors proposed the idea that success of surgery can only be determined by the quality of the patient's communication performance in everyday situations as compared to their pre-surgery status.

The pathology to assess:

A. Resonance

1. Hyper nasal sound- the perception of inordinate resonance during the production of vowels.

2. Nasal Emission- escape of air during production of consonants like S/P/K

B. Articulator assessment includes,

1. Nasal Substitution-substitution of different letters instead like B with M and D with N

2. Compensatory articulation- in appropriate use of articulation to form plosive, Fricative words

C. Phonation assessment

Sibilant distortion-incorrect tongue placement often due to malocclusion for the sounds of S& Z.

Perceptual speech evaluation is a useful tool in assessment of an intelligent English speaking patient. In the Indian scenario the ISHA monograph has been recommended where in articulatory assessment is done in various Indian languages.

The protocol for evaluation of Perceptual:

Annexure (i)

DIRECT ASSESSMENT:

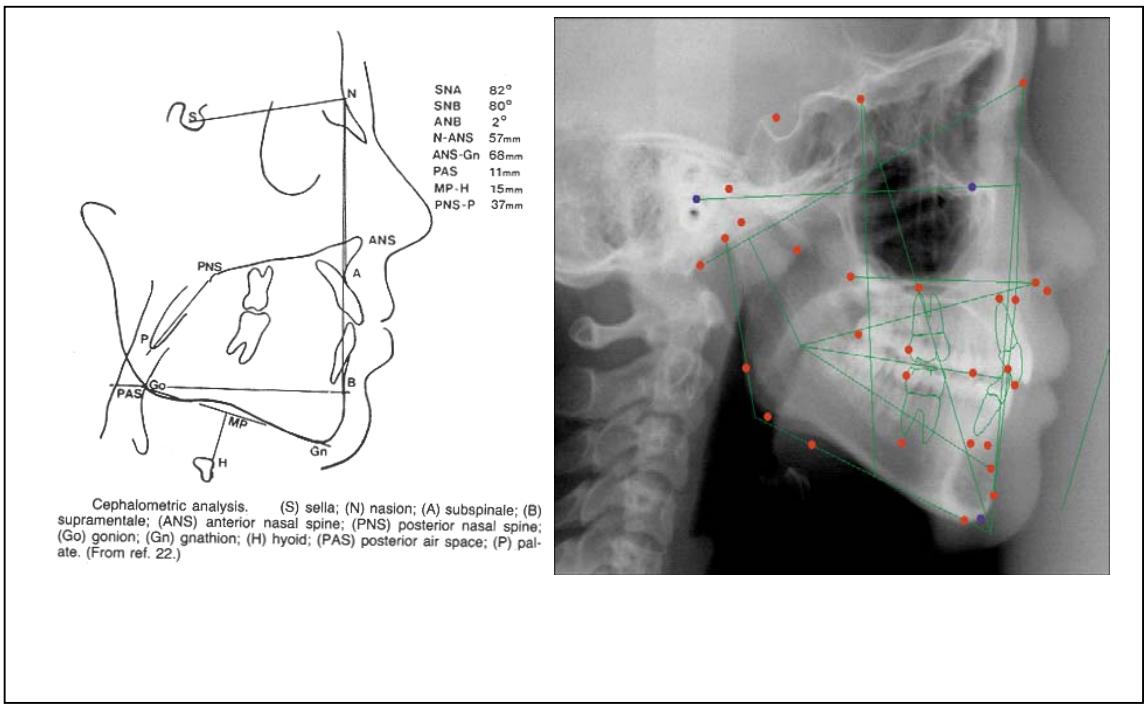
Many objective techniques have been devised and used to overcome the problem of assessing the velopharyngeal (VP) function in speech, both in normal and pathological conditions. The following domains have witnessed significant development:

1. Technological advances in radiology have moved from cephalometrics and tomography to cineradiography, Videofluoroscopy also replaced cephalometrics and tomography. Radiographic techniques moved from lateral, extended and frontal views of the mechanism to the base view. CT scans were also tried, and recently, MRI studies have been attempted.
2. Endoscopy has evolved from the pan endoscope to sophisticated endoscopes often coupled to videotape equipment.
3. Various aeromechanical devices have also been developed to measure nasal and oral pressures, to estimate the size of VP openings and to measure nasal airway resistance.
4. Acoustic measures that correlate with perceived hypernasality have also been developed, tested and used clinically.
5. Electromyography has provided basic information about muscle activity during speech. The behaviour of individual muscles and their dynamic interactions have helped to explain the movements observed by videofluoroscopy and endoscopy and to illuminate the sequence of events within motor patterns

Cephalometric assessment: This is by tracing a lateral view radiograph of the skull taken during phoneme specific nasal emission in older children. The tongue palate contact is studied and a rough estimate is obtained. This method does not give a 3 dimensional picture and more so the lateral pharyngeal wall movement's very important factor cannot be assessed [(5)]. The cephalograph is the accepted standard of craniofacial assessment for both comparison to established norms[46]and long term comparison of repeated cephalographs for the same patients. Using both linear and angular measurements, orthodondist, surgeons, and others can quantitatively appreciate the change in tha mandibular position and the related soft tissue structures.

Multi view Cine video fluoroscopy: is a dynamic study of Velopharyngeal apparatus in a co-operative child. Its superiority in comparison to Cephalometry is established (1), but it is less preferable by the surgeons in comparison to Nasopharyngoscopy has been established in various centres (1).It a very effective tool in assessment of VPI but the long term effects of the radiation exposure is not studied. Cost factor also plays an important role in the installation of the equipment and the affordability of the patient. Hence the routine use of this methodology is debatable (1).

The assessment is done in both en face view for the velar, lateral pharyngeal wall and posterior pharyngeal wall movement. Analysis should be made of the displacement and of the size, shape and position of the gap. The same criteria and procedures should be applied as were used for nasopharyngoscopy.



Cephalometric analysis. (S) sella; (N) nasion; (A) subspinale; (B) supramentale; (ANS) anterior nasal spine; (PNS) posterior nasal spine; (Go) gonion; (Gn) gnathion; (H) hyoid; (PAS) posterior air space; (P) palate. (From ref. 22.)

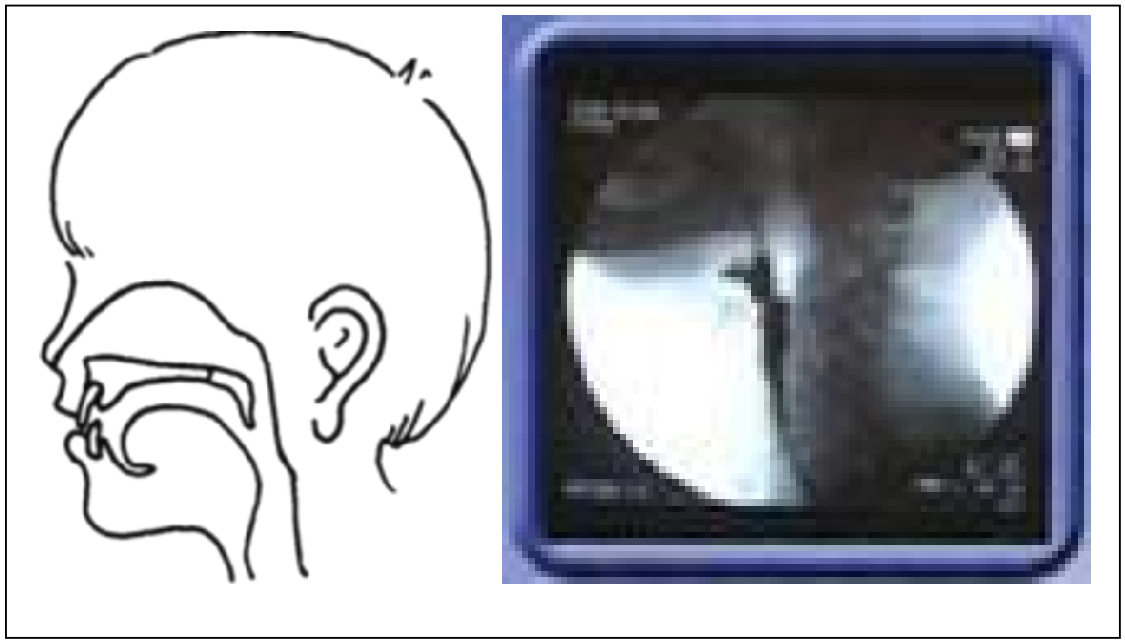


Figure 10 and 11[47]

Flexible Nasopharyngoscopy:

In 1969, Piggott and his colleagues in Europe presented this unique method of using Storz rigid side viewing endoscope for the visualization of the Velopharyngeal apparatus [48] . Shprintzen from USA studied the use of End viewing Machida Flexible cope and presented the need for the tailor made flap surgery in treating Cleft Palate in 1979 [49]. He also mentioned that Video-fluoroscopy and video-pharyngoscopic assessment is the best method of presurgical investigation for planning intervention [49].

A 1993 survey of speech-language pathologists on cleft palate/craniofacial teams was conducted by Pannbacker et al[50]. Results revealed the majority opinion that naso-pharyngoscopy should be included as an important piece of assessment of VPI. Sixty-four percent of these respondents also believed that nasopharyngoscopy should be performed by a speech-language pathologist, and 78.5% of respondents believed this should be done under medical supervision. However, only 35.5% of the speech-language pathologists surveyed at the time actually performed naso-pharyngoscopic examinations [51]. A similar survey was conducted by D'Antonio et al [52]. A questionnaire was sent to all ACPA craniofacial/cleft palate teams, for which 90% of the respondents indicated that nasendoscopy was available to their teams. Of the responding teams, 90% reported that they believed nasendoscopy to be necessary for "difficult diagnostic problems," but only 50% of those who responded believed that nasendoscopy was appropriate for all patients with velopharyngeal insufficiency, and 41% believed that nasendoscopy was an appropriate instrumental measure for patients for whom secondary palatal management is planned.

Golding-Kushner[53] and her international working group stressed the importance of utilizing videofluoroscopy in addition to nasopharyngoscopy when conducting an evaluation of velopharyngeal structure and function. At the end of their 1993 report, they concluded that “the evaluation of velopharyngeal movement must be comprehensive and include, at the very least, frontal (P-A) and lateral view videofluoroscopy, and en face view or nasopharyngoscopy”. All endoscopic observation should be based on views where all, or most of the VP orifice is seen in a single field of view.

The study of Velopharyngeal function includes [54],

- Movement of the Velum in the Postero-superior direction towards the Posterior pharyngeal wall(PPW) in adults and towards the adenoids in children
- Movement of the Lateral pharyngeal wall medially
- Movement of the posterior pharyngeal(Passavant’s ridge) wall anteriorly in 20%

Velar Movement:

Quantitative: The degree of velar movement is measured from a reference point on the musculus uvulae if it is present. A reference point is chosen as anatomic midpoint on the velum and during inspiration/phonation of the syllable ‘K’ as the velum moves posterior to close, the distance between the velum and the posterior pharyngeal wall or the adenoid is defined in terms of ratio.

Qualitative: Movements should be observed for symmetry, asymmetry if present is noted for the side both intersyllabic and intrasyllabic consistency should be noted. The presence of sound specific VPI or closure and consistency at repeated attempts of the same task should be made.

Lateral Pharyngeal Wall Movement

Quantitative: Each lateral wall is measured separately for movement from its normal resting position in inspiration along a reference line to a most medial position of the lateral pharyngeal wall. The point of maximum movement of each lateral wall is measured in ratio.

Qualitative: The qualitative assessment of lateral pharyngeal wall is similar to the described for velar movement and should include notation of symmetry. If movement is asymmetric, the side with better motion is noted and any discrepancy in vertical height of motion is described.

Posterior Pharyngeal Wall and Passavant's ridge

Quantitative: A line is constructed between the posterior pharyngeal wall (PPW) or location of Passavant's ridge(PR), if present, and the midpoint of velum(during inspiration).

Qualitative: Presence of adenoids, their relation to the midline, shape, and surface is noted.

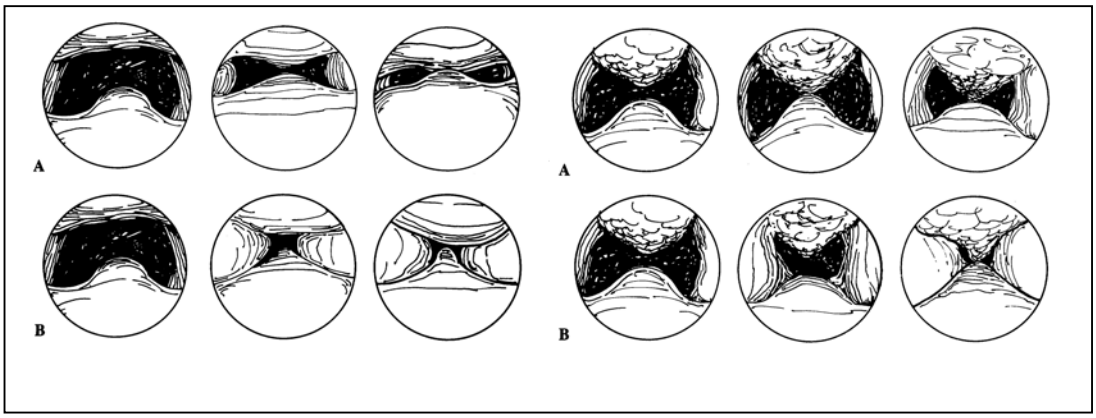
Velo Pharyngeal Gap description

Size of the Gap: a rough size of the VPI gap is done either manually/computerized. If no gap is seen / Bubbling is seen through a pin hole. This is to be recorded.

Shape of the Gap: the coronal, sagital, circular system described by Skolnick et al[55] for classification of closure patterns is used to describe the shape of the gap. Both circular and circular with Passavant's ridge closure pattern's yield circular gaps. Small central triangular gaps is listed as circular, or the shape most closely resembled. Irregularly shaped gaps is listed as "other" and described or drawn.

Other Information

The nasopharyngoscopy report also indicates the nostril(s) through which examination was done, if The endoscope is passed between the middle turbinate and the inferior turbinate. Factors that may affect measurement and/or interpretation of observation is noted including the presence and location of palatal fistula or pharyngeal scarring. If tonsils are present in the pharynx, their size, position, and any observed intrusiveness on the velopharyngeal closure [34]



[56] Valving Patterns of the Velopharynx in VPI

A. Decreased velar motion with & without adenoids

B. Decreased lateral wall movement with & without adenoids

Patients & methods

This prospective observational study was done in the ENT department of Christian Medical College Vellore. Since there was no sufficient data from India or internationally comparing the type of VPI and the speech defect a pilot study was suggested for estimation of the sample size. Hence, consecutive patients attending Plastic Surgery or ENT OPD diagnosed to have Cleft palate (pre or post operative) with complaints of unclear or nasalized speech were enrolled.

Inclusion Criteria

1. Patients with cleft palate and unclear/nasalized speech
2. More than 9 months of age

Exclusion Criteria

1. Children with multiple syndromic deformities

Informed Consent:

Informed consent was taken in patients' language from all being enrolled in this study, Consent forms are attached as Appendix III

Methods: All subjects fulfilling criteria were subjected to detailed ENT evaluation. Their ears were examined to look at the status of the tympanic membrane and hearing was evaluated by Pure tone and Impedance audiometry. Radiograph of the soft tissues of the neck (lateral view) was done to ascertain adenoid status.

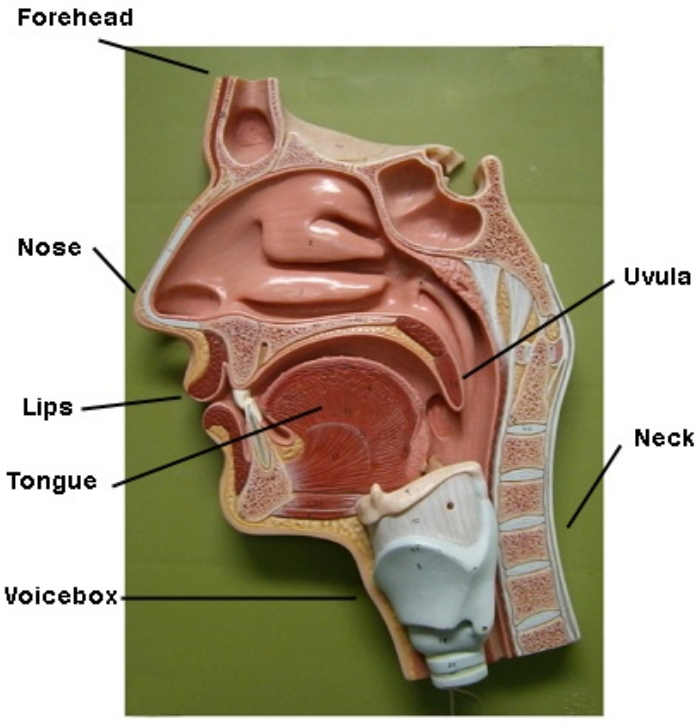
The speech pathologist who is routinely involved in assessment and training children with nasalized speech assessed the speech defect according Cleft Audit Protocol Speech (CAPS).The findings were then noted and subsequently analyzed.

The Principal investigator then performed the Flexible video endoscopy to ascertain the type of VPI. Nasal spray consisting of a solution of oxymetazoline and 2% xylocaine mixed in a 1:1 solution, which helps in opening the nasal passages and numbing each side of the nasal cavity for easy and comfortable insertion was used. Following this either a Pentax (FNL-10S; Englewood, CO) or Olympus (ENF-P; Melville, NY), 3mm flexible end viewing nasopharyngoscope was used to view the velopharyngeal port. The scope was passed over the middle meatus of the nose in order to provide a higher angle view above the palate and not moved with soft palate movement[54]. Once the palate & pharyngeal walls were visualized, the scope was kept stationary. Following this the velopharyngeal apparatus was examined and the mobility of the respective walls was observed during cry (in infants/young children)/speech in older children. In the latter and young adults the subjects' name, age, counting from 1 to 20, sustained vowel productions, consonant vowel repetitions, sustained /s/, connected speech (sentence repetition), and repeating a standard sentence that has the consonants 'K' was repeated. The scope was moved from side to side in the pharynx so that both the lateral pharyngeal walls could be observed and then passed deep into the pharynx until the larynx was seen in order

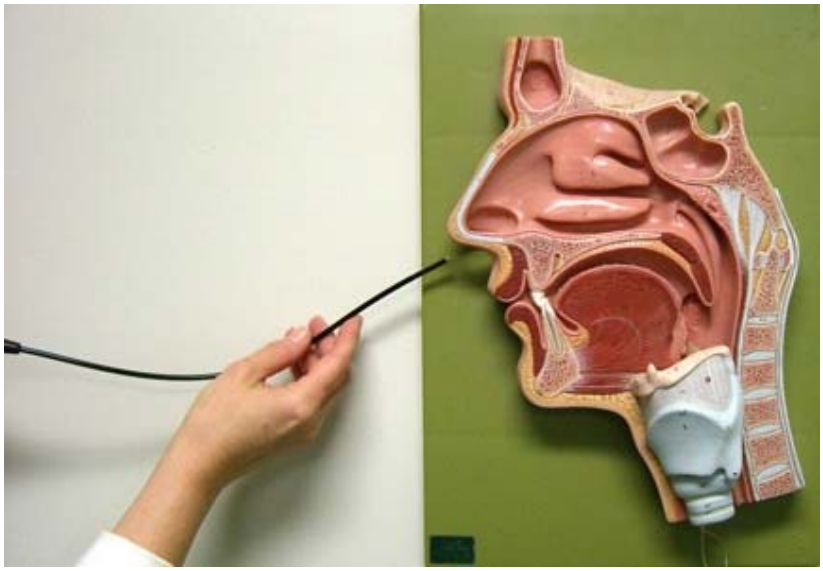
to observe all possible vertical levels of movement[54]. The videotaped studies were then evaluated by the primary investigator/guide.

Pentax Flexible Scope being used and video being recorded on imimo software

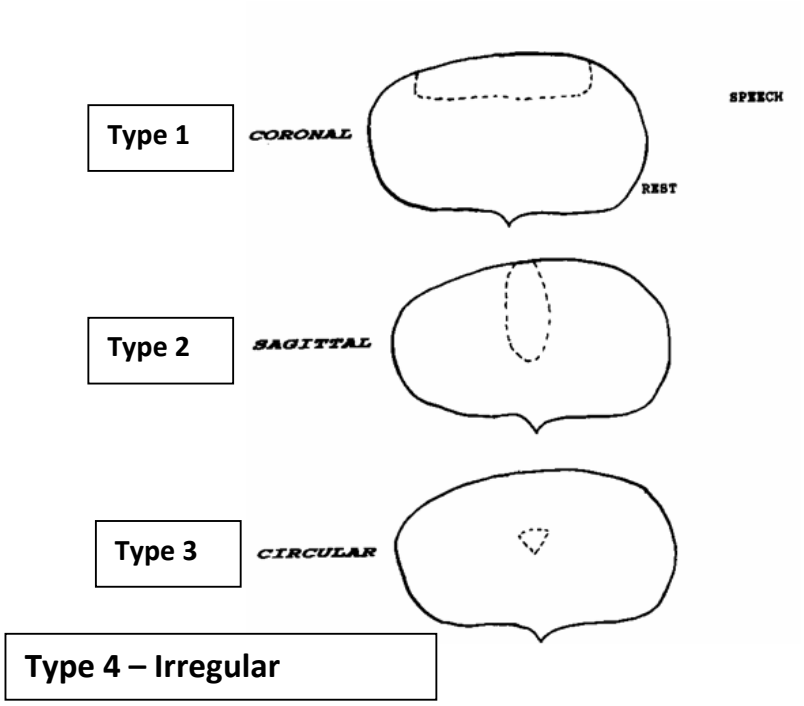
Imimo soft ware on which the videos are recorded



Structure of the nose and pharynx

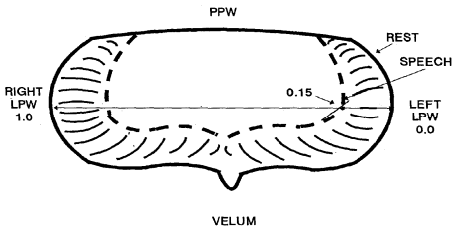


Endoscope as it is passed

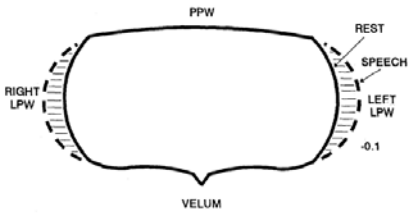


[54]

Various Patterns of velopharyngeal closure seen in VPI



[57, 58]



[2]

Pictorial representation of the movements of the Velum and lateral pharyngeal wall

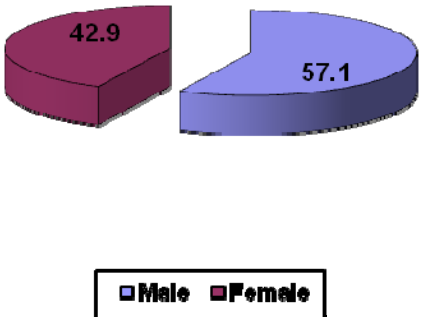
Results & Analysis

1. Statistical Analysis:

All stastical analysis were performed using Statistical Package for Social Science version 16.0 (SPSS Inc, Chicago IL)

2. Patient Profile:

There were 28 patients in this study. Among them 16 (57.1%) were males and 12 (42.9%) were females.

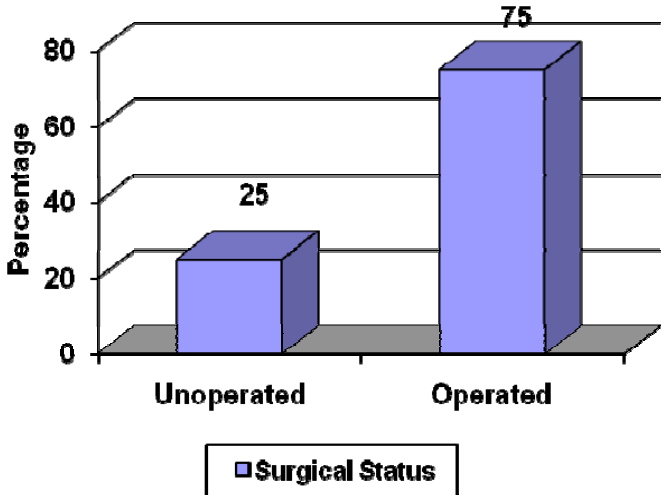


3. Age distribution:

Ranged from 1 year to 23 years, with a mean age of 7.71 and Standard deviation of 5.28.

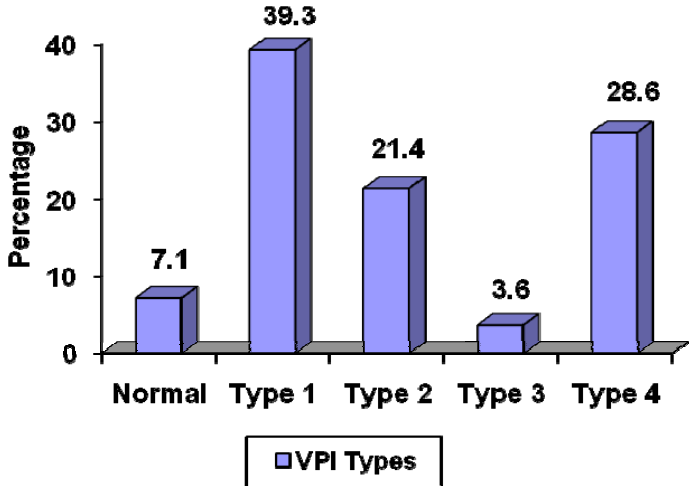
3. Surgical Status:

Among these patients 7 (25%) were pre-operative and 21 (75%) were post operative.



4. Types of VPI:

It was noted that Type 1 VPI was the commonest (39.3%) followed by Type 4 (28%) and Type 2 (21.4%). Type 3 was least common (3.6%)



5. Comparison of surgical status and types of VPI

Table 1.

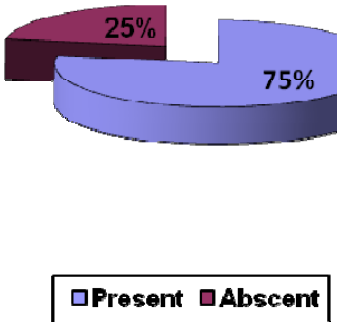
Variables	VPI Types										p - Value
	Normal		Type1		Type2		Type3		Type 4		
	n	%	n	%	n	%	n	%	n	%	
Surgical status											
Operated	2	100.0	7	63.6	4	66.7	1	100.0	7	87.5	0.431
Unoperated	-	-	4	36.4	2	33.3	-	-	1	12.5	

Pre-operative patients appear to have more commonly type 1 VPI (63.6%),

Post operative patients appears to have type 4 VPI more commonly (87.5%)

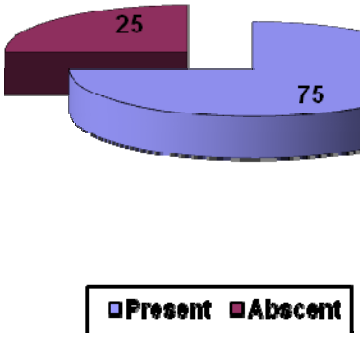
6. Hyper Nasal Sound:

Among these patients 21 (75%) were found to have hyper nasal speech.



7. Distortion of speech:

Among these patients 21 (75%) were found to have distortion of speech



8. Comparison of Speech defects (Resonance) in various types of VPI

Table 2

Variables	VPI Types										p - Value
	Normal		Type1		Type2		Type3		Type 4		
	n	%	n	%	N	%	n	%	n	%	
Hyper Nasal Sound											
Present	1	50.0	7	63.6	5	83.3	1	100.0	1	100.0	0.041
Absent	1	50.0	4	36.4	1	16.7	-	-	-	-	
Nasal emission											
Present	1	50.0	8	72.7	4	66.7	-	-	7	87.5	0.438
Absent	1	50.0	3	27.3	2	33.3	1	100.0	1	12.5	
Hypo Nasal Sound											
Present	-	-	1	9.1	-	-	-	-	-	-	0.431
Absent	2	100.0	10	90.9	6	100.0	1	100.0	8	100.0	
Mixed Hypo / Hyper Nasal Sound											
Present	-	-	1	9.1	-	-	-	-	-	-	0.431
Absent	2	100.0	10	90.9	6	100.0	1	100.0	8	100.0	

Hypernasal speech is pathognomic of VPI. and is most common in type 1 (P value=0.041)

Nasal emission is also seen more in type1 VPI (72%). However it was not statistically significant.

9. Comparison of Speech defects (Substitution/addition) with various types of VPI

Table 3

Variables	VPI Types										p - Value
	Normal		Type1		Type2		Type3		Type 4		
	n	%	n	%	n	%	n	%	n	%	
Substitution											
Present	-	-	5	55.6	4	80.0	-	-	6	75.0	0.190
Absent	2	100.0	4	44.4	1	20.0	1	100.0	2	25.0	
Addition											
Present	-	-	4	36.4	2	33.3	-	-	6	75.0	0.049
Absent	2	100.0	7	63.6	4	66.7	1	100.0	2	25.0	

1. Compensatory/addition articulation- plosive, Fricative words is pathognomic of type 2 VPI- significant(0.049)

2. Nasal Substitution (B with M and D with N) –nasal substitution is seen more in type 1(55%) and type 2 (80%). These were also not statistically significant.

10. Comparison of Speech defects (distortion) with various types of VPI

Table 4

Variables	VPI Types										p - Value
	Normal		Type1		Type2		Type3		Type 4		
	n	%	n	%	n	%	n	%	n	%	
Distortion											
Present	-	-	6	54.5	1	16.7	-	-	5	62.5	0.386
Absent	2	100.0	5	455	5	83.3	1	100.0	3	37.5	

Distortion of words is absent in type 2 VPI (83%) and present in type 1 and type 4 VPI. These are, however, not stastically significant.

11. Comparison of presence of adenoids and type of VPI

Table 5

Variables	VPI Types										p - Value
	Normal		Type1		Type2		Type3		Type 4		
	n	%	n	%	n	%	n	%	n	%	
Adenoids											
Normal	2	100.0	7	63.6	6	100.0	-	-	5	62.5	0.393
Minimal	-	-	3	27.3	-	-	-	-	2	25.0	
Enlarged	-	-	1	9.1	-	-	1	100.0	1	12.5	

Adenoid tissue was absent in 71.4% patients and among them 63.6% had type 1 VPI

Hearing was normal with no evidence of middle ear effusion in 92.8% patients while 2 post operative patients had unilateral attico-antral disease.

Discussion

Velopharyngeal in-competence(VPI)[2] is a common abnormality seen in Cleft palate patients causing hyper nasal speech. Otolaryngologists assist in the differential diagnosis of velopharyngeal valving disorders for speech, assisting in treatment planning and the assessment of treatment outcomes [10]. The most commonly used methods for direct visualization of velopharyngeal function remain nasendoscopy and videofluoroscopy. Nasoendoscopy and speech assessment of the children early in the evaluation process not only gives good information for surgical correction but it also becomes a reference point for further management[3]. A good recording of both the endoscopy and the speech pathology can be used as bio feedback tools as the child grows[12].

The present study was a prospective investigation of 28 subjects between the ages of 1 and 23 years who were diagnosed with velopharyngeal dysfunction secondary to cleft palate. All the subjects were endoscoped sucessfulluy irrespective of their age, unlike in the study by Antonio et al where 3 children could not be endoscoped even after 2 seperate attempts[59] Results of the present study indicate that a significant and clinically relevant relationship exists between the perceived characteristics of hypernasality and velopharyngeal type of insufficiency . 85 % of patients came with complaints of unclear speech. Among them 75% were post operative. 50 % of these patients were then diagnosed to have VPI clinically and referred for management. Rest of the patients were sent for detailed evaluation and confirmation of VPI.

Coronal / type 1 VPI defect was the most common followed by type 4, type 2 and type 3. This has been earlier reported by Harlan et al[60]. On further analysis it was noted that type 1 Velopharyngeal dysfunction or the coronal type deficiency had strong co-relation with hypernasality (P value -0.041). This has not been previously reported. Perceptual ratings of hypernasality and distortion of speech contributed significantly to the prediction of type of VPI. Perceptual characteristics accurately predicted type 1 and type 2 VPI with 63.6% and 75% accuracy, respectively. Compensatory / addition articulation- plosive, fricative words was pathognomic of type 2 VPI (P - 0.049). Nasal emission was also seen more in type1 VPI (72%). However it was not statistically significant. Nasal Substitution (B with M and D with N) is seen more in type 1 and type 2 (Table – 2). These were also not statistically significant. These observations have not been previously reported. Distortion of words was absent in type 2 VPI (83%) and present in type 1 and type 4 VPI. These are, however, not stastically significant. These has also not been previously reported.

In comparing the surgical status of the patients and type of VPI pre-operative patients appear to have more commonly type 1 VPI (63.6%) and post operative patients appears to have type 4 VPI more commonly (87.5%). The latter appears to be probably due the inadequate surgical correction for speech.

It was also observed that hearing was normal with no evidence of middle ear effusion in 92.8% patients while 2 post operative patients had unilateral attico-antral disease.

In the present study adenoid tissue was absent in 71.4% patient. In patients with absence of adenoid tissue type 1 VPI was more common (63.6%). This has also not been previously reported.

Clinical Implications:

Although the specific diagnostic protocols for the direct assessment of velopharyngeal function vary among different centres, perceptual assessment is always performed. The results of this pilot study suggest that perceptual assessment of speech can determine not only the speech pathology but in many cases can also be used to predict the relative type of the velopharyngeal gap. This can be important because the size and type of the velopharyngeal gap and not the severity of the speech distortion, may be the primary determinant for appropriate treatment. Treatment of velopharyngeal dysfunction through speech therapy alone has been reserved for patients with such characteristics as phoneme-specific nasal emission (as can occur with the use of a posterior nasal fricative), inconsistent hypernasality or nasal emission, stimulable closure, or a small velopharyngeal gap (3 mm) as observed through objective measures[3]. However, when the speech distortion seems severe, as is usually the case with hypernasality, clinicians may not consider speech therapy as an option until after physical / surgical management.

The results of the present investigation indicate that when a hyper nasal speech is noted in, the cause could probably be type 1 VPI and when speech substitution is noted the type 2 VPI could be predicted. Predicting velopharyngeal insufficiency type based on an individual's speech is not an exact science. However, the results from the present investigation revealed that some predictions of VPI type

can be made on the basis of the speech assessment alone. Confidence in the prediction is strongest if the patient has hypernasal sound, which predicts a Type 1 opening. Confirmation of the structural and physiological causes of the opening can be identified only through velopharyngeal endoscopy / imaging.

CONCLUSION

The present study of perceptual speech and flexible video endoscopy in patients with cleft palate pathology indicates a co-relation between speech defect and type of VPI. In management of patients with cleft palate, it is important that surgical correction of the defect is done at the same time achieving velopharyngeal competency for speech without creating nasal airway obstruction. Velopharyngeal endoscopy with speech assessment will define the anatomic and functional bases for the velopharyngeal correction and also to plan /tailor pharyngeal flaps. This approach also appears to be a useful and necessary tool for 'surgical feedback'. Hence a multidisciplinary approach involving Otolaryngologists / Plastic surgeons / Speech pathologists for preoperative evaluation of the defect with perceptual speech analysis and velopharyngeal endoscopy is mandatory.

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Anexure-1

Cleft Palate Articulation & Resonance Evaluation in Developing Nations:

Assessing in a non-native language

Kelly Nett Cordero, M.A., CCC-SLP

& Anna Thurmes, M.A., CCC-SLP

Outline

I. Overview of Velopharyngeal Closure and Velopharyngeal Dysfunction

II. Assessment Protocol for English Speakers at a U.S. Cleft Palate Clinic

III. Adapting Assessment Protocols for Other Languages

IV. Team Collaboration in Assessment Process

Overview of Velopharyngeal Closure

Velopharyngeal Dysfunction

Impacts many aspects of speech:

• Resonance

• Articulation

• Voice

• Intelligibility

SLP Role in Assessing Velopharyngeal Dysfunction

Goal: Determine if speech errors are physically and/or behaviorally based and what treatment is needed

Speech Samples

• Physical Management

• Physical and Behavioral Management

• Behavioral Management

Assessment Protocol for English Speakers in CPC

Goal: A wide variety of speech tasks to determine where breakdown occurs and under what contexts

Conversation, Sentence, Syllable…

Phonetic Context: Orals, Nasals, Mixed

Protocol in Cleft Palate Clinic

Recorded Speech Sample:

• Connected Speech (Reading passage or picture description task)

• Sentence Repetition (phonetically balanced)

• Counting

• Sustained Vowels

'Low-Tech'

• Nasal Flutter (nasal occlusion, cul-de-sac test)

• Mirror Test (stethoscope or straw)

'Some-Tech'

• Standardized Articulation Tests

'High-Tech'

• Nasometry

• Pressure Flow

• Nasendoscopy

• Videofluoroscopy

Adaptation vs. Translation of Assessment Tools

_ Determine if a tool exists in the target language

_ Consider general rules for test adaptation

_ Look for guidelines to adapt/develop your own

• Brøndsted et al, 1994;

• Hutters & Henningsson, 2004

• "Universal Parameters for Reporting Speech Outcomes in Individuals with Cleft Palate" (Henningsson, Kuehn, Sell, Sweeney, Trost-Cardamone & Whitehill, 2008)

• 7 areas to evaluate (Henningsson et al, 2008)

• Suggestions for speech sample: single words, short sentences

• Operation Smile Ratings

General Linguistic Considerations in Test Adaptation

• What language(s) is(are) spoken, and when relevant, what dialect(s)?

• Who will be your interpreter(s)?

• What are key socio-pragmatic rules to consider?

Key Linguistic Traits in Assessment of Velopharyngeal Closure

Presence and frequency of occurrence of the following in the linguistic system:

• High pressure consonants (Hutters & Henningsson, 2004)

• Vowels (Guirao & Jurado, 1990)

• Phonemically nasalized vowels (Leeper, Rochet, & MacKay, 1992)

• Nasal phonemes (Heimbach, 1980) (Kan & Kohnert, 2004).

• Phonemic glottal stop and other pharyngeal or uvular sounds (Shahin, 2002).

• Presence of lexical tones (Gibbon, Whitehill, Hardcastle, & Stokes, 1998; Stokes &

Whitehill, 1996).

Nature of Cleft Palate Speech Errors

Cross-Linguistic

• 'Universal' patterns exhibited

• Eurocleft (Brøndsted et al, 1994; Hutters & Brøndsted, 1987)

Language-Specific

• Arabic (Shahin, 2002)

• Cantonese (Stokes & Whitehill, 1996)

• Japanese (Yamashita & Tsukada, 1985)

• Mandarin (Wu, Chen & Noordhoff, 1988)

• Spanish (Guillen & Barlow, 2006)

BOTH

Non-English Speaker CPC Evaluation

Profile: Somali Speaker with repaired cleft palate

Tasks:

• Study Phonology of Language

• Develop stimuli to assess VPC and articulation

• Adjust the plan as needed during interaction

The Protocol Utilized

Recording

• Count to 50

• Word repetition task

• Sustained Vowels

Nasal flutter and mirror test

• Use list of words with oral only and mixed consonants

• Syllable repetition

• Sustained Fricatives

- Nasendoscopy
- Treatment Recommendations
- Decision

• Physical Management?

• Speech Treatment?

A more familiar language…Spanish

Profile: Bilingual Spanish-English speaker with repaired cleft lip and palate

Impressions of Articulation and Resonance in Spanish vs. English

Judging Cleft Palate Speech in an Unfamiliar Language

Vietnamese (Landis, 1973; Landis & Cuc, 1975)

Slovak (Morris, 1978)

Sinhala (Sell, 1992; Sell & Grunwell, 1990; Sell & Grunwell, 1993)

Collaborating with Team

_ Surgical Management Decisions

_ Collaborating with local professionals

_ Continuity of care when team leaves

Role of SLP During Evaluation Abroad

Depends on:

_ Model utilized by team

_ Type of cases being seen

_ Types of surgical and dental procedures available from visiting and local teams

_ Evaluation tools available

_ Experience of SLP

_ Relationship of team memb

Annexure II

INFORMATION SHEET

Dear Patient/Guardian,

This is to bring to your attention that the procedure you/your ward is going to undergo is a routine ENT evaluation which involves the examination of his/her cleft palate using a flexible metallic endoscope measuring 3mm in diameter. This instrument will be passed through one of the nasal cavities after application of local anaesthetic liquid soaked cotton. The duration of this examination will be 15 mins, during which time we will study the nature of the defect in the palate and its subsequent outcome in the speech of the patient.

The whole procedure will be recorded on a computer and latter analysed by a team of specialist including the Plastic Surgeon, ENT Surgeon & Speech pathologist. This will help us in planning a suitable treatment schedule for your Child/self. There are no major foreseen risks during or after this procedure, occasionally there might be prolongation of the anaesthetic and very rarely bleeding in case of mucosal injury.

The other alternative procedure to study this defect involves the child undergoing a radiographic procedure called Video-fluoroscopy, which involves being exposed to X-rays for more than 15 mins. Under no circumstances will your identity be revealed to third Parties in any information or published article. No one other than the treating doctors and the investigators of this study shall have access to your medical records. Your participation in the study is voluntary and hence you have the option of withdrawing from this investigation as and when so intended.

The cost of this evaluation would be Rs.480/-(Four Hundred and eighty rupees only) apart from your other investigation expenses, Kindly sign in the following consent form after reading through this information and if you have any query kindly feel free to ask

Dr. Rajan or any other doctor performing the procedure.

Thank you for your co-operation and we pray for a speedy recovery.

ANNEXURE III

A. Information Sheet and Consent form in English

Informed Consent form to participate in a clinical trial

Study Title: **Velopharyngeal Incompetence in Cleft Palate Patients-**

Flexible Video Pharyngoscopy & Perceptual Speech Assessment.

Study Number:

Patient Bradma: _____

Date of Birth / Age: _____

(i) I confirm that I have read and understood the information sheet dated _____ for the above study and have had the opportunity to ask questions. [ ]
(ii) I understand that my / my child's participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. [ ]
(iii) I understand that the Sponsor of the clinical trial, others working on the Sponsor's behalf, the Ethics Committee and the regulatory authorities will not need my permission to look at my / my child's health records both in respect of the current study and any further research that may be conducted in relation to it, even if I withdraw from the trial. I agree to this access. However, I understand that my / my child's identity will not be revealed in any information released to third parties or published. [ ]
(iv) I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s) [ ]
(v) I agree to my / my child's taking part in the above study. [ ]

Signature (or Thumb impression) of the Subject/Legally Acceptable

Representative: _____Date: _____/_____/_____

Signatory's Name: _____

Signature of the Investigator: _____
Date: _____/_____/_____
Study Investigator's Name: _____

Signature of the Witness: _____
Date:_____/_____/_____
Name of the Witness: _____

B. Information Sheet and Consent form in Hindi

C. Information Sheet and Consent form in Tamil

D. Information Sheet and Consent form in Bengali

ANNEXURE IV

PROFORMA

Study Title: **Velopharyngeal Incompetence in Cleft Palate Patients-**

Flexible Video Pharyngoscopy & Perceptual Speech Assessment.

1. Study Number:

2. Hospital Number:

3. Patient Bradma/Name: _____

4. Date of Birth / Age: _____

5. Address:
6. Diagnosis:

7. Operated/Un operated:

8. Complaints;

9. Pure tone audiogram & Impedance Audiogram

10. X-ray neck soft tissue lateral view:

11. Speech Assessment:

A. Resonance

1. **Hyper nasal sound-** **Present/Absent**
2. **Nasal Emission during S/P/K-** **Present/Absent**
3. **Hypo nasal sound- (nasal obstruction, turbinate hypertrophy)Present/Absent**
4. **Mixed Hyper- nasality-** **Present/Absent**

B. Articulator assessment includes, (Articulatory assessment in their respective language of Communication Tamil/Bengali/Hindi/English)

1. Nasal Substitution (B with M and D with N) - **Present/Absent**

2. Compensatory/addition articulation- plosive, Fricative words **Present/Absent**

C. Phonation (Sibilant distortion- for the sounds of S& Z) - **Present/Absent**

Flexible Nasopharyngoscopy

R **L**

1. Nasal Cavity:

2. Floor

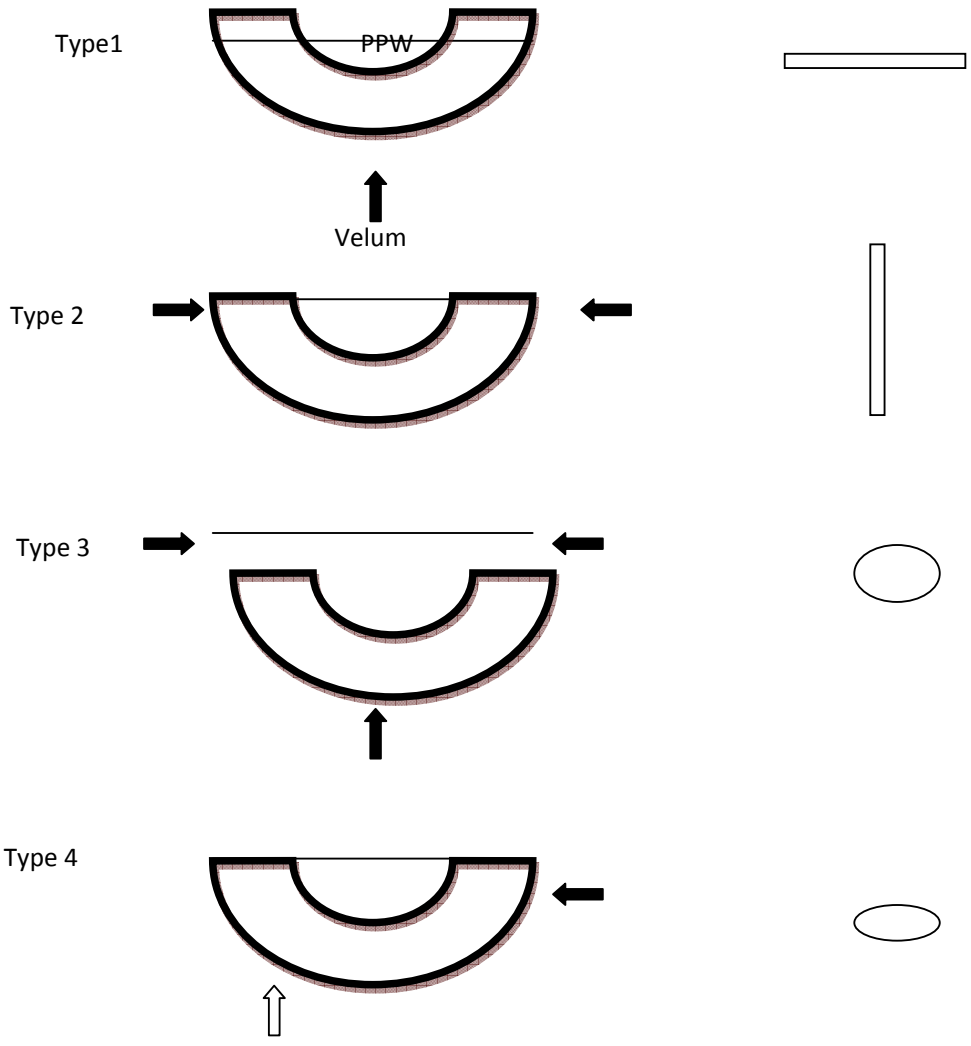
3. Turbinate

4. Soft palate

5. Posterior Pharyngeal wall/Passavant's ridge

6. Lateral Pharyngeal wall

7. Type of defect:



Comments: Signature

তথ্য প্রদান (1)

শ্রদ্ধেয় রোগী/ রোগীনি/ গার্জিয়ান,

আপনাকে বিশেষভাবে জানানো যাচ্ছে যে, আপনাকে নিয়মমাফিক ENT চেক করার প্রক্রিয়ায় নেয়া হচ্ছে যা আপনার ক্লেপট প্যালেট পরীক্ষার একটি অংশ এবং এটি মাত্র ৩ মি.মি ডায়ামিটার বিশিষ্ট একটি ধাতব এবং নড়াচড়া করে এমন একটি এন্ডোস্কোপ। এই যন্ত্রটি আপনার নাকে এ্যানেসথেসিয়া মেশানো তুলো দেওয়ার পর নাকের একটি ছিদ্র দিয়ে ধীরে ঢুকানো হয়। পরীক্ষাটি করার সময় মাত্র ১৫ মিনিট। [redacted] নাকে কী ধরনের সমস্যা আছে তা ~~এন্ডো~~ দেখে এবং [redacted]-এর কথা শুনে এই সময়ে [redacted] পরীক্ষা-নিরীক্ষা করা হয়।

সমস্ত প্রক্রিয়াটি কম্পিউটারে রেকর্ড করা হয় এবং পরবর্তীতে একজন প্লাস্টিক সার্জন ~~সহ একটি বিশেষজ্ঞ টিম~~ ইএনটি সার্জন এবং একজন সিনিয়র প্যাথলজিস্ট সহ একটি বিশেষজ্ঞ টিম এই রেকর্ড ~~দেখে~~ পর্যালোচনা করেন। এই প্রক্রিয়া/ পদ্ধতি আপনাকে/ আপনার শিশুর যথার্থ চিকিৎসা প্রদানের ক্ষেত্রে আমাদেরকে সহায়তা দেয়। এই পদ্ধতিতে ~~প্র~~ নেওয়ার সময় কিংবা পরে তেমন কোন বিপদ নেই। ব্যতিক্রম হিসেবে নিউকাসে ক্ষত হয়ে সামান্য রক্ত বের হতে পারে।

এই প্রক্রিয়ার বিকল্প প্রক্রিয়াটি হচ্ছে- ভিডিও ফ্লুরোসকপির মাধ্যমে সমস্যাটি পর্যবেক্ষণ করা। এটি [redacted] ১৫ মিনিটের এক্স-রে-এর একটি বাড়তি ক্ষতিকর পদ্ধতি। কোন অবস্থায়ই [redacted] আপনার পরিচয় অন্য কাউকে জানানো হবে না বা প্রচার করা যাবে না। কেবলমাত্র ~~এই~~ চিকিৎসা প্রদানকারী ডাক্তার এবং অনুসন্ধানকারী [redacted] আপনার মেডিকেল রেকর্ডপত্র দেখতে পারবেন।

এই অনুসন্ধানকারী প্রক্রিয়ায় অংশগ্রহণে আপনি বাধ্য নন এবং/ অথবা আপনি চাইলে যে কোন সময় এই প্রক্রিয়া থেকে সরে যেতে/ বন্ধ করতে পারেন।

এই পরীক্ষণ প্রক্রিয়ার ফি ৩০০/- টাকা। এই তথ্যাদি পড়ার পর আপনি সম্মত হলে উল্লিখিত 'কনসেন্ট ফরম'-এ স্বাক্ষর করুন। আপনার যদি আরও কিছু জানার ইচ্ছে থাকে তাহলে নির্দ্বিধায় ডঃ রাজন অথবা এই প্রক্রিয়ার সাথে জড়িত যে-কোন ব্যক্তিকে তা জিজ্ঞেস করুন।

আপনার সহযোগিতার জন্য ধন্যবাদ। [redacted] আমরা দ্রুত রোগমুক্তির জন্য প্রার্থনা করি।

চিকিৎসার্থে পরীক্ষণে অংশগ্রহণকারীর তথ্যাবলী ফরম

পর্যবেক্ষণ : ভিডিও ফারিংগোস্কপি এবং ক্লেফট প্যালেট পেশেন্ট-এর প্রদত্ত মৌখিক তথ্যের ভেলোফেরিনজেলসহ বিশ্লেষণ।

পর্যবেক্ষণ
~~পরীক্ষণ~~ নং:—

রোগীর পরিচিতি :

জন্মতারিখ/বয়স :

i) আমি নিশ্চিতভাবে প্রদত্ত তথ্যাবলী পড়েছি এবং বুঝেছি যা তারিখে প্রদত্ত এবং নিম্নোক্ত বিষয়ে জানার সুযোগ পেয়েছি. [ ]

ii) আমি বুঝেছি যে আমার/আমার সন্তানের জন্য উপরোক্ত শিক্ষা-পরীক্ষণে অংশগ্রহণ স্বেচ্ছা প্রণোদিত এবং কোন প্রকার কারণ দর্শানো ছাড়া অথবা কোন চিকিৎসার স্বার্থ ছাড়া অথবা আমার অধিকার ক্ষুণ্ন হচ্ছে মনে করে যে কোন সময় এই প্রক্রিয়া থেকে নিজকে প্রত্যাহার করার অধিকার রাখি।

iii) আমি বুঝেছি যে, এই চিকিৎসা-প্রোফাইল-এ অংশগ্রহণ, অংশগ্রহণকারীর প্রাসঙ্গিক বৈশিষ্ট যথাযথ কর্তৃপক্ষ এবং ঐ নিয়ন্ত্রিত পর্যবেক্ষণ কর্তৃপক্ষ আমার/আমার সন্তানের স্বাস্থ্য-সংক্রান্তে বর্তমান পর্যবেক্ষণ এবং ভবিষ্যতে অনুসন্ধানের ক্ষেত্রে আমার অনুমতি নেওয়ার কোন প্রয়োজন নেই; অধিকন্তু - আমি উক্ত পরীক্ষণ পদ্ধতি থেকে নিজেকে প্রত্যাহার করলেও। আমি এই অনুপ্রবেশে সম্মত আছি। আমি বুঝেছি যে, আমার/আমার সন্তানের পরিচিতি অন্য কাউকে জানানো হবেনা বা প্রচার করা হবেনা।

iv) এই পর্যবেক্ষণ থেকে প্রাপ্ত ডাটা বা ফলাফল বৈজ্ঞানিক প্রয়োজনে ব্যবহারের ক্ষেত্রে আমার কোন অসম্মতি নেই।

v) আমি/আমার সন্তান উপরোক্ত পর্যবেক্ষণ কর্মসূচীতে অংশগ্রহণে সম্মত আছি।

স্বাক্ষর/বৃদ্ধাঙ্গুলির ছাপ (সম্মতি জ্ঞাপন)

जानकारी पत्र

प्रिय संरक्षक,

यह आपकी जानकारी के लिए है कि आपके बच्चे के इलाज मे यह एक साधारण जाँच है। इस जाँच में एक मुड़ने वाला एण्डोस्कोप के जरिए नाक के द्वारा तलुए के हेड की जांच कि जाएगी।

इस प्रक्रिया से पहले नाक के अन्दर रुई पर दवा स्रव कर उसे सुन्न किया जाएगा ताकि कोई दर्द महसुस न हो। इस प्रक्रिया का समय लगभग 15 मिनिट है। इसके द्वारा बच्चे के तलुए के हेड व इसके आवाज पर प्रभाव का विश्लेषण किया जाएगा।

यह सारी प्रक्रिया कम्पयुटर पर रिकार्ड कि जाएगी व विशेषज्ञो द्वारा विश्लेषण के बाद सही इलाज का निर्धारण किया जाएगा। इस प्रक्रिया से कोई विशेष असुविधा व खतरा नही है लेकिन यदाकदा थोड़ा खुन नाक से आ सकता है।

इस प्रक्रिया के अलावा जो प्रक्रिया काम में ली जा सकती है जैसे कि फ्लोरोस्कोपी में बच्चे को रेडिएशन का हानिकारक प्रभाव हो सकता है।

आपकी पहचान पुर्णत: गुप्त रही जाएगी। आपके इलाज करने वाले चिकित्सक व जाचकर्ता के अलावा कोई भी आपके रिकार्ड नही प्राप्त कर सकता।

स्टडी मे भाग लेना आपकी इच्छा पर निर्भर करता है आप इससे कभी भी हट सकते है।

इस जाच का खर्च 500₹ होगा. इस पत्र को पुरा पढने के बाद आप अनुमति पत्र पर हस्ताक्षर करे। अधिक जानकारी के लिए आप डॉ. राजन से सम्पर्क करे।

आपके सहयोग के लिए धन्यवाद!

क्लिनिकल स्टडी मे भाग लेने अनुमति पत्र

स्टडी का नाम : विड्यो फनडेस्कोपीक विसनेस्पा- क्लेफट पेलेट रोग - विलोफारिन्जीयल इनकामपिटेन्स।

स्टडी नं०:

मरीज का नाम : SOHINI MITRA 438852D 9m17d F

अस्पताल No ; C PLS Q1W 3995531 HIN

(i) मैंने उपरोक्त स्टडी के सूचनापत्र को पढ़ व समझ लिया है व उससे सम्बन्धित प्रश्न पूछने का मौका दिया गया।

(ii) मैं जानता हु कि स्टडी में भाग लेना मेरी इच्छा पर है तथा किसी भी समय मैं इससे हट सकता हु बिना किसी कारण के व बिना मेरी चिकित्सा व कानुनी अधिकार प्रभावित हुए।

(iii) मैं जानता हु कि क्लीनिकल स्टडी के स्पोन्सर व इससे सम्बन्धित लोग व इथीक्स कमेटी, रेगुलेटरी अथोरिटी को मेरी स्वास्थ्य रिकार्ड, इस स्टडी व अनुसंधान मे शामिल करने के लिए मेरी अनुमति की जरूरत नही है। मैं जानता हु कि मेरी पहचान गुप्त रखी जाएगी।

(iv) मैं इस स्टडी के परिणाम व किसी अन्य तथ्य के वैज्ञानिक उपयोग के लिए सहमत हु।

(v) मैं उपरोक्त स्टडी में भाग लेने के लिए सहमत हु।

हस्ताक्षर (अंगुठे का निशान) K Sughendr Mitra.

दिनांक

हस्ताक्षरकर्त्ता का नाम

जांचकर्ता के हस्ताक्षर mr Rajiv

दिनांक

जांचकर्ता का नाम

गवाह के हस्ताक्षर

दिनांक

அன்புள்ள பெற்றோர்களே,

உங்களுக்கு அளிக்கப்படும் சிகிச்சையால் உள்நோக்கு பரிசோதனை, உள்நோக்கு கருவி மூலம் அறியலாம். இதன் மூலம் உங்கள் பேசும் திறனையும் ஆய்வு செய்யலாம். இந்த சிகிச்சை முறையின் கால அளவு 15 நிமிடங்கள் மட்டுமே. மயக்க மருந்து தேய்த்துக் பட்ட பரிசோதனை முக்கில் வைத்து பின் இந்த ஆய்வு செய்யப்படும்.

இந்த சிகிச்சை முறை கணினியில் பதிவுசெய்யப்பட்டு அறுவைசிகிச்சை நிபுணர்கள் ஆய்வு செய்வார்கள், இவற்றைச் செய்யும் போழதோ அல்லது அதற்குப் பிறகோ எந்த ஒரு பெரிய பக்கவிளைவுகள் ஏற்பட வாய்ப்பில்லை. சிலசமயம் மயக்க மருந்தின் செயல்பாடு நீடித்தோ அல்லது மிகக் குறைய அளவு இரத்தக்கசிவு ஏற்பட வாய்ப்புண்டு.

மற்றும் ஒரு மாற்று சிகிச்சை (Video fluoroscopy) செய்யும் போது, 15 நிமிடங்கள் கதிர் வீச்சு (Radiation) உட்படுத்தப்படும் உங்களுக்கு அளிக்க படும் அனை சிகிச்சை விவரங்கள் முழுதும் மொழக்கல் நிகழ்படல் பதிவு செய்யம் படும், உங்கள் சிகிச்சை அளிக்கும் மருத்துவர் இதனைக் தவிர மற்றவர்களுக்கு அளிக்க முடியாது.

இதற்கு ஆகும் செலவு Rs 500/- மட்டுமே, இவற்றை முழுமையாகப் பழத்துபிறகு உங்கள் விருப்பத்தை கை யொப்பமிட்டு தெரியப் படுத்தவும். உங்கள் சந்தேகங்களுக்கு டாக்டர் ராஜன் அவர்களோடு, அல்லது இந்த சிகிச்சை அளிக்கும் மற்ற டாக்டரிடமும் கேட்டு தெரிந்து கொள்ளலாம்.

உங்கள் ஒத்துழைப்புக்கு மகிழ்ந்து நன்றி.

நீங்கள் விரைவில் நலம் பெற நாங்கள் பிரார்த்தனை செய்கிறோம்.

மருத்துவ மாதிரியில் பங்கேற்பதற்கான ஒப்புதல் படிவம்

தலைப்பு → உள் நாக்கு மினுதகலை அறியும் எண்டோஸ்கோபி முறைகள்.

பாடத்தின் எண்:—

நோயாளியின் பெயர்:— 158862D @ F
RAMYA P.

பிறந்த தேதி / வயது:— 1½ yrs old.

கருத்து: _____ தேதியில் இந்த படிவத்தையும் மேற்கண்ட வகுப்பின் நோக்கத்தையும் நன்கு அறிந்து கொண்டேன்.

இந்த வகுப்பில் நான்/என் குழந்தை தேர்வது என்னுடைய விருப்பத்தின் நான், எனக்கு விருப்பம் இல்லையென்றால் உடனடியாக விலகிக்கொள்ள எனக்கு சந்த தடைகள் இல்லை என்று எனக்கு முழுமையாக தெரியும்.

இந்த மருத்துவ மாதிரியினை நடத்தும் திரு; மற்றும் திட்டபூர்வமான குழுவை சார்ந்தவர்களுக்கு என் குழந்தையின் மேலதிக்கான விவரங்களை அறிந்து கொள்ள என்னுடைய அனுமதி தேவையில்லை. (நான் இதில் இருந்து விலகி கொண்டாலும்) என்பதை நான் நன்கு அறிவேன்.

எனது சம்மந்தப்பட்ட விவரக்களும், இந்த படிவின் முடிவுகளும் அறிவியல் சம்மந்தமாக பயன்படுத்த நான் மறுக்க மாட்டேன்.

(ஆ) மேற்கண்ட படிப்பில் பங்குபெற சம்மதிக்கிறேன்.

நோயாளின் / அல்லது அவர் சம்மந்தப்பட்டவர் கையொப்பம்.

தேதி -

பெயர் -

மருத்துவரின் பெயர்:— R. Pachani
MR. R. PACHANI

தேதி:—

கையொப்பம்:—

சாட்சியாளரின் பெயர்:—

தேதி:— 4/4/19

கையொப்பம்:—