CLEFT PALATE & MISARTICULATION

- INTRODUCTION
  - Between the 6th and 12th weeks of fetal gestation,the left and right sides of the face and facial skeleton fuse in the midddle. When they do fail to do so, the result is a craniofacial cleft. Various types of clefts may occur as isolated condition or as part of a syndrome. Since clefts involve the anatomy of the vocal tract, varying effects on speech are common.
  - Craniofacial Abnormalities are structural defects of the head and face. These defects may be part of a syndrome that includes other anomalies. Consequences include structuaal and developmentalanomalies.
  - Craniofacial - pertains to or involving bothe cranium (skull) and the face.
  - Craniofacial Anomalies - Malformations of the cranium and/or face
- INCIDENCE OF CLEFTS
  - Accepted estimate of incidence of vert cleft palate not associated with other malformations is 1 in 750 live births.
  - Cleft palate without cleft lip occurs more in females (57%) than males (43%), no significant racial differences
  - Cleft lip with or without cleft palate occurs twice as many males than females.
  - Native Americans (most), Asians, Caucasians, African Americans (least)
  - Gender differences may be related to slight differences in timing of embryological development.
- SPEECH PRODUCTION
  - 7 Point Man
    - Respiratory and laryngeal activities produce voice.
    - The vocal signal rises through pharynx or vocal tract.
    - The process by which the nasal branch is separated from or coupled with the rest of the vocal tract is referred as velopharyngeal closure.
    - Physical characteristics and activity of velopharyngeal region imposes feature of resonance.
    - Speech signal with characteristics of phonation and resonance modified by activity of tongue and lips imposes features of place and manner of articulation.
  - Voice or Resonance Disorder
- Velopharyngeal Inadequacy should be used as a generic term
- Velopharyngeal Insufficiency for cases for insufficiency of tissue
- Velopharyngeal Incompetence for cases in which movement patterns are inadequate
- ANATOMY OF EXTERNAL FACE & ORAL CAVITY
  - Nose
  - Lips
  - Oral Cavity
- EMBRYOLOGY
  - Neural crest theory of development holds that neural crest cells, one type of specialized embryonic cells, give rise to various connective and neural tissues of the skull and face
  - The cells migrate at different rates to destined locations
  - If the migration fails to occurs, or if there is an absence or inadequacy of related cells, clefts and other facial abnormalities may results.
  - The upper lip and premaxilla are formed by merging 3 structures:
    - frontonasal process
    - right and left nasomedial processes of the maxilla
  - Lip closure occurs during 5 or 6th week of embryonic development.
  - Palate is formed by union of palatal shelves that develop from the maxillary processes and close during 8th or 9th week of gestation

    - Embryonic tongue sits above the mandible between 2 sides of the palate and fusion of the 2 sections of the palate can occur after the mandible and tongue lower of the way. When this sequence of events is interrupted, it results in a condition called Robin sequence.
- Development of the External Face
    - Two mandibular processes result in the lower jaw (mandible), lower lip, and chin. By the end of the 4th or 5th week, these structures are formed and fused.
    - At 5 weeks, maxillary processes appear on the anterior edge of the mandibular arches. FIG 7-2 (Smith, 1983).
        - Two maxillary processes form most of the face, mouth, cheeks, and sides of the upper lip. These processes form most of the face, mouth, cheeks, and sides of the upper lip.
        - These processes evolve into most of the hard palate, alveolar ridge, soft palate.
    - Nasal Processes (lateral and medial) are prominent at 6 weeks. FIG 7-3 (Smith, 1983).
    - Frontonasal Process - develops into the nose, central part of upper face and primary palate.
        - Nasolateral Process will form the alae (lateral wings of the nostrils).
        - Nasomedial Process will contribute to the formation of several structures: midportion of the nose, upper lip, maxilla, primary palate (entire frontmost portion of the hard palate).
    - The nasomedial processes (medial nasal processes) fuses with maxillary processes by week 7.
    - By the end of the 7 week, the upper lip and primary palate are formed.
- Development of the Internal Face
    - Primary Palate
        - Prior to 6th week of development, the primary palate includes that portion of the upper airway that will develop into the lip, alveolar ridge, and the hard palate segment extending back to the incisive foramen (a hole behind the alveolar ridge through which passes nerve and blood supply to the palate). During 6th week, the separation between nasal and oral cavities occur. This early structure contains prolabium, premaxilla and four maxillary incisor teeth.
    - Secondary Palate (Hard palate 8-10 weeks)
        - During the sixth to 10th week of embryological development, the foundation for the hard palate and the floor of the nasal cavities, the palatine shelves, begins their migration toward the midline of the internal face. This movement toward midline coincides with the lowering of the tongue mass which had previously rested within the developing nasal chambers.
        - Between 5.5 - 8 weeks, the primary palate and the secondary palate (that portion formed by the joining of the palatine shelves behind the incisive foramen) coordinate their growth to form a single structure: the hard palate. Failure of the shelves to unite with the primary palate or with each other will result in a cleft of the hard palate.
    - Soft Palate
        - During the 10th to 12th week of orofacial development, the soft palate tissue fuses. Tissue that will form the soft palate's muscles begins to migrate toward the midline from both sides of the back of the mouth during the 5th to 6th week. By the 10 week this tissue has grown and moved into a horizontal orientation along with the palatine shelves.
- TYPES OF CLEFTS
    - Cleft is a separation or space between parts that are normally joined, thus interrupting the continuity of the structure. Although orofacial clefts can be acquired through various traumas, the vast majority of those of the lip and/or hard and soft palates are due to congenital failures of the structures to join. Of the many ways to classify types of congenital cleft (Berlin, 1971), a division into general types is convenient:
        - clefts of the lip and/or alveolar process (gum ridge), the embryological primary palate (Figures 9-4, 9-5, Hixon text), and
        - clefts of the hard and/or soft palates, the embryological secondary palate (Figure 9-6, Hixon text).

- Cleft Lip - A cleft of the lip involves only soft tissue and extend through the red part of the lip or vermillion border, into the upper portion of the upper lip toward the nostril. If the cleft is unilateral, it commonly occurs on the left side. Patient may have a short columella, which is the strip of tissue between the base and the tip of the nose.
- Cleft Palate - The palate serves as the partition between the nasal and oral cavities. The anterior two-third of the palate make up the hard palate, commonly referred to as the roof of the mouth. The soft palate or velum consists of the posterior one-third of the palate. Isolated cleft palate, without cleft lip, may include all of the hard palate posterior to the incisive foramen and the soft palate; it may involve only a small portion of the posterior part of the soft palate; or it may between these two extremes.
- Alveolar Ridge - A part of the prepalate, partially encircles the hard palate and consists primarily of supporting structures for the teeth.
- Cleft of Prepalate - Median Cleft - clefts of the upper lip is median defect extends to the nose. The missing tissue is the problabium. Bilateral cleft lip with absence of the prolabium or complete median cleft lip.
  - Premaxilla Protrusion & Detachment
  - Premaxilla - intermaxillary bone
  - Prolabium - central prominence of lip
- Submucous Cleft Palates - The palate appears to be structurally intact, but there are both muscular and bony deficits. The defect include a bony notch in the hard palate, a bluish line at the midline of the soft palate, and a bifid uvula.
  - Hard Palate - The bony notch can be seen or felt where normally the posterior nasal spine is found along the posterior border of the hard palate. The submucous cleft of the hard palate is not functionally significant.
  - Soft Palate - The muscular deficit found in the soft palate often is functionally significant. This cleft is seen as a bluish line through the length of the soft palate. The levator muscles in these cases are often found to be inserted into the hard palate instead of interdigitating (interlock as or like fingers of both hands) to form the normal levator sling. This condition foreshortens the functional soft palate and creates a further complexity which may contribute to velopharyngeal incompetence requiring surgery. The levator muscle elevates the posterior part of the soft palate, the velum, and pulls it slightly backward which effectively closes the nasal from the oral pharynx.
  - Unilateral Clefts
  - Bilateral Clefts

- COMMUNICATION DISORDERS
  - SPEECH DISORDERS
    - Good place of articulation and intelligible speech in association with nasal emission and consonant weakness resulting from lack of intraoral breath pressure.
    - Compensatory Learned Behaviors - Second pattern involved only nasal escape, but also nasal snort, glottal stops, pharyngeal fricatives, nasal grimace, and other articulatory sibilants.
  - MISARTICULATION OF SPEECH SOUNDS
    - Reduced intra-oral pressure (McWilliams Audio 1,2)
      - Some speakers with VPI seem to accept their loss of intra-oral pressure and continue to articulate as accurately as possible,. These speakers may have weak consonants and audible escape, but placement is accurate. Even intelligibility may be only slightly impaired.
    - Sounds Most Frequently Misarticulated
      - Most frequently in error with VPI: /s/. In short, velopharyngeal competence is a requirement for adequate consonant production. The first consonant to deteriorate will be /s/. This is the phoneme most frequently in error in individuals with VPI of

a marginal or borderline nature. As the incompetence increases, other sibilants and fricatives (f,v,th,sh,z,s,zh) become involved. The symptom is decreased intraoral pressure because of loss of air at the velopharyngeal portal. Still greater incompetence in this critical valve will lead to reduced intra-oral pressure in association with plosives.

- Sounds Most Frequently Misarticulated - McWilliams (1953)
  - /s/ - 63%
  - /z/ (61%)
  - /d/ (48%)
  - /ch/ - (44%)
  - /p/ - (11%)
  - /b/ - (9%)
  - /s/ is th espeech sound most frequently and most consistenly misarticulated by cleft palate individuals.
- Substitutions (McWilliams Audio 10)
  - Generally sound substitutions are seen in speech problems that are characteristic of young children who are still in the process of learning speech. Some writers have referred to the fact that speakers with cleft palate substitute a nasal consonant for a consonant that requires intraoral pressure (for example, substituting /m/ for /b/). In some instances, this response is regarded as a nasally distorted /b/ rather than the substitution of /m/. Viewed in this context, the speech sound error clearly points to velopharyngeal incompetence rather inappropriate learning.
- Omissions (McWilliams Audio 11)
  - Could delete final consonants as a means of avoiding nasal emission, because sounds lacking normal oral air pressure could be interpreetedd as omissions even though some energy marked the final consonants.

- Compensatory Strategies for VPI (McWilliams Audio 12,14)
  - Some speakers with VPI seem to accept their loss of intra-oral pressure and continue to articulate as accurately as possible,. These speakers may have weak consonants and audible escape, but placement is accurate. Even intelligibility may be only slightly impaired.
  - Others attempt to compensate for their valving deficits by seeking new approaches to consonant articulation. These individuals discover that it is easier to produce a sibilant-like consonant lingua-palatal or pharyngeally than it is to produce it in the front of the mouth and that plosives created glottaly are unaffected by the defective valve.They adopt gross articulation errors, which are referred to as pharyngeal fricative and glottal stops. These compensations are rarely accompanied by nasal escape because that critical valve is bypassed.
- Stop plosives, fricatives, affricates misarticulated cannot manage airstream for development and maintenance of high oral air pressure. Therefore, leading to deterioration of sound (Hixon, 372).
  - Hypernasal resonance, nasal air emission, and weak pressure consonants cause phonetic distortions, compensatory gestures cause phonetic substitutions.
  - Individuals with clefts tend to substitute non English sounds for English phonemes. Substitute glottal stop (whispered cough) for stop-plosives (p, t, k, b, d, g). Glottal stops are made by closing the glottis, building up pressure within the trachea, and then suddenly reopening the glottis to release a transient puff of air. Substitute pharyngeal fricatives for fricative consonants. Pharyngeal fricatives are produced by positioning the tongue close to the back wall of the throat or by decreasing the side to side diameter of the throat by moving the walls of the pharynx inward. Air is then forced through the constricted pharyngeal airway to produce a turbulent sound.

        - Substitution of Glottal Stops - GO BACK TO WITZEL VIDEO
            - Speakers with VPI problems demonstrate glottal articulation for plosives, typically back plosives. The interruption of the air stream at the level of the glottis results in a cough-like speech sound that has been labeled the glottal stop /?/.
        - Substitution of Pharyngeal Fricative - GO BACK TO WITZEL VIDEO
            - Typically, fricatives and affricates are the result of a constriction of the air stream in the anterior portion of the oral cavity (lips, tongue, teeth).
            - Speakers with velopharyngeal incompetence are not able to produce the friction noise because the air pressure escapes from the oral cavity through the velopharyngeal opening. Such speakers sometimes distort fricatives and affricates in order to create the friction noise by constriction between the tongue and pharyngeal wall or perhaps by constriction of the pharyngeal tube.
    - Distortion by Nasal Emission (McWilliams Audio 13,3,4)
        - Description -The type of error sometimes referred to as the distortion-nasal is typical of what most listeners regard as "cleft palate speech" and is demonstrated by many cleft palate speakers. The air pressure is emitted through the nostrils during the production of the pressure consonants. If the escape is sufficient in quantity, the nasal emission of air pressure is audible and distorts the acoustic signal of the speech sound.
        - Nasal air escape associated with production of consonants requiring high oral pressure. It occurs when air is forced through an incompletely closed velopharyngeal port. Nasal emission may be audible or not.
- RESONANCE
    - Hypernasality that occur supraglottally is referred to as "resonance disorders."
        - Voice disorders refers to a problem that occurs at the level of the larynx, a disorder of phonation.
            - Hypernasality is not a voice problem that implies phonation.
        - Resonance is the vibratory response of a body or air-filled cavity to a freqeuency imposed upon it. (Wood, 1971).
        - Individuals with velopharyngeal incompetence have traditionally been described in the literature as having an hypernasal voice quality. In actuality, hypernasality is not a problem associated with phonation. It is the result of alterations that occur when the oral and nasal cavities are coupled when they should be separated by action of the velopharyngeal mechanism.
        - Definition - The perception of excessive nasal resonance during the production of vowels. This results from inappropriate coupling of the oral and nasal caviities. Low vowels and vowels in nasal consonant contexts are normally somewhat nasalized.
        - Factors Responsible for Hypernasality
            - Results form inadequate closure of the VP valve during speech, but it may caused by entrance of air into nasal cavity through an open cleft palate or fistula in hard or soft palate.
            - Oral Cavity constriction during speech may increase hypernasal resonance by forcing more sound waves into the nasal cavity. This constriction may be the result of :
                - restricting mouth opening during speech,
                - posterior or superior positioning of the dorsum (upper surface) of the tongue during articulation of some sounds,
                - or abnormal positioning or tension of the pharynx.
    - Hyponasality is the speech characteristic associated with a reduction in nasal resonance with /m,n,ng/ approaching but not matching /b,d/g/.

        - a reduction in normal nasal resonance usually resulting form blockage or partial blockage of the nasal airway by any number of causess, including upper respiratory tract infection and a wide obstructiing pharyngeal flap, and moderately large adenoids were present.
        - If the nasal airways were completely occluded, speech would be denasal, meaning that nasal air flow associated with /m, n, ng/ would be eliminated and the sound wave altered; the nasal consonants would approach but not match /b, d, and g/.
- VOICE DISORDERS
    - Hoarseness
        - Hamlet (1973) speculated that glottal tightness might contribute to vocal abuse which would lead to hoarseness, harshness, and vocal nodules secondary to hypernasality. This conclusion appears reasonable in view of the clinical evidence of patients who attempt to control hypernasality by tightening both the respiratory muscles and laryngeal muscles to control air flow before it reaches the crucial velopharyngeal valve.
    - Soft Voice Syndrome
        - Because of loss of air through the velopharyngeal port, some cleft patient shave difficulty creating a voice that is of sufficient loudness to serve them well in communication or they use reduced loudness as a compensatory strategy. Patients who adopt this voice pattern may have little nasal escape, although their velopharyngeal valving mechanisms are usually deficient. In order to increase loudness, their subglottic pressures must be higher than normal, and there is air loss leading to an increase in nasal emission, together with alterations in the perception of hypernasality. This problem is identified on the basis of clinical experience.
    - Reduced loudness
        - General characteristic of client with repaired cleft palate is the reduced loudness. Aerodynamic and acoustical factors contributes to the reduced loudness rather reduced self confidence or self consciousness.
        - For vowels, diphthongs, and many voiced consonants, the problem is related to incomplete velopharyngeal mechanism which allows sounds produced at the larynx to pass through the nasal segment of the upper airway.
        - For many consonant sounds of speech, an incompetent velopharyngeal mechanism precludes the adequate buildup of oral air pressure that is required behind airway occlusions and obstructions to generate normal speech sounds (Subtelny, Worth, and Sakuda, 1966). This means that sounds like voiceless stop-plosives, fricatives, and affricates will fail to their normal crispness when produced. Consequently, they are perceived as being too soft. Trying to generate sounds orally in the face of velopharyngeal incompetence is like trying to blow up a tire that has a leak in it.
    - Monotone
        - Often accompanying the soft-voice syndrome is the monotonous voice with little pitch variation. Patients with problem are often unable to demonstrate pitch variations of more than 3 or 4 tones. This problem is identified on the basis of clinical experience.
    - Strangled Voice
        - This problem is not one of the more common phonation disorders found inpatients with valving problems, but is encountered often enough to warrant mention here. Strangled voice quality appears to be associated with an attempt to be non-nasal in the presence of VPI. Phonation is associated with extreme tension in the abdominal, diaphragmatic, thoracic, laryngeal, and supraglottal muscles. It is almost as if the person were trying to hold his breath and talk at the same time. The introduction of relaxation results in an increase in hypernasality.

- DELAYED LANGUAGE
    - Research suggests that there may a lag in language acquisition during early years of development for many children with orofacial clefts, but as the individual gets older the gap closes. By adulthood, no significant language differences remain.
    - Disruption in early experience disruption of oral touch-pressure sensation, oral cavity exploration, perceptual motor deprivation, prolonged deprivation during formative language period and reduced hearing sensitivity.
    - Insufficient language stimulation due to low expectations for verbal responsiveness given child's physical problem.
    - Negative reactions of listeners cause child to limit the frequency and elaboration of speech language attempts.
- HEARING DISORDERS
    - Hearing disorders are prevalent among individuals with orofacial clefts. These disorders are a result of middle ear dysfunction, involving both ears. Exist in all infants with unrepaired clefts of palate under the age of 2, 70 to 80% with repaired clefts of older children
    - Prevalence is due to a disease called otitis media involves an inflammation and/or infection of middle ear system and presence of liquid in middle ear root of otitis media problem is eustachian tube dysfunction muscle to open ET are cleft and do not function normally consequently ET remains closed, pressure and liquid builds up in ME inflammation and infection occurs middle ear sound transmission system is impaired conductive type of loss occurs