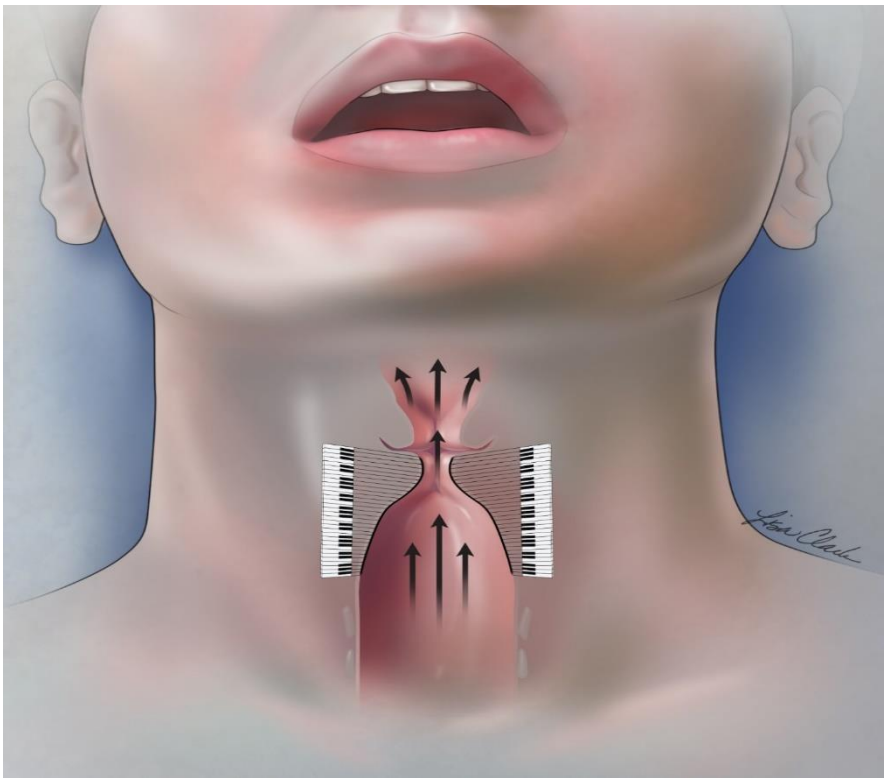


Communication and Swallowing Disorders

Physiology of swallowing, swallowing disorders, GERD

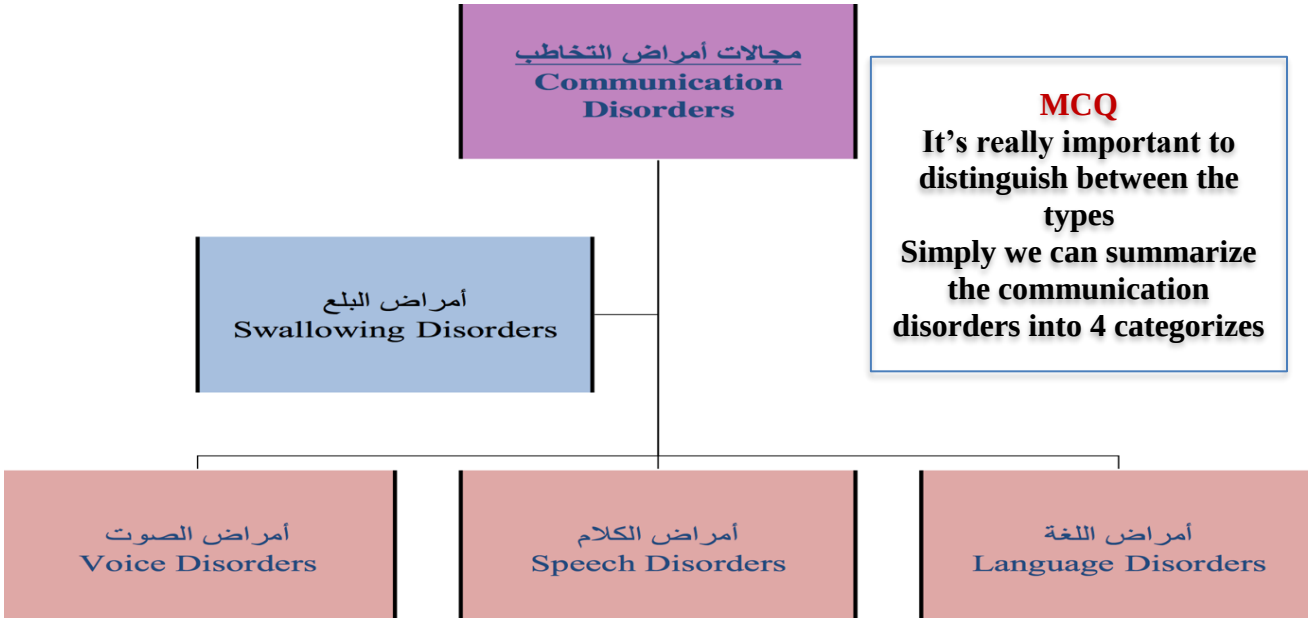
Voice disorder, language disorder, speech disorder

Done by : Reema AlRasheed

Reviewed by : Hadeel B. Alsulami

Correction File

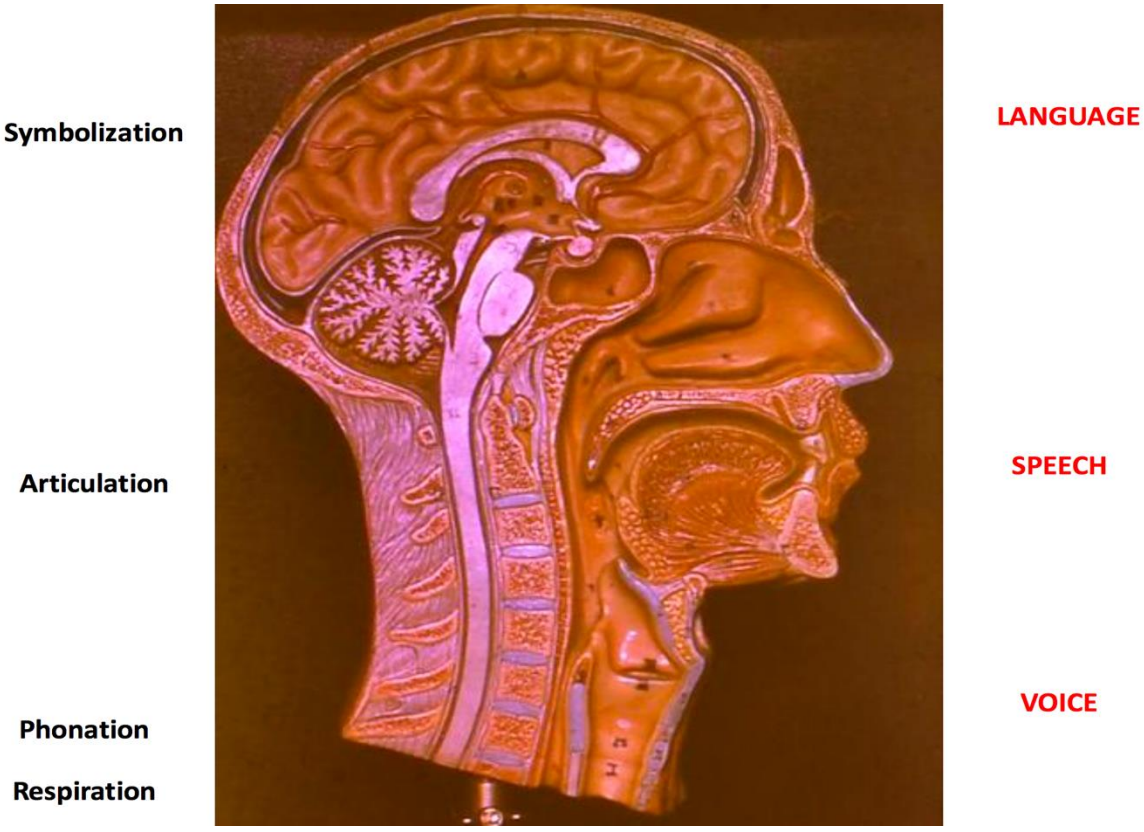
Color Index : Slides - Important Notes - Doctors' Notes - EXTRA



MCQ

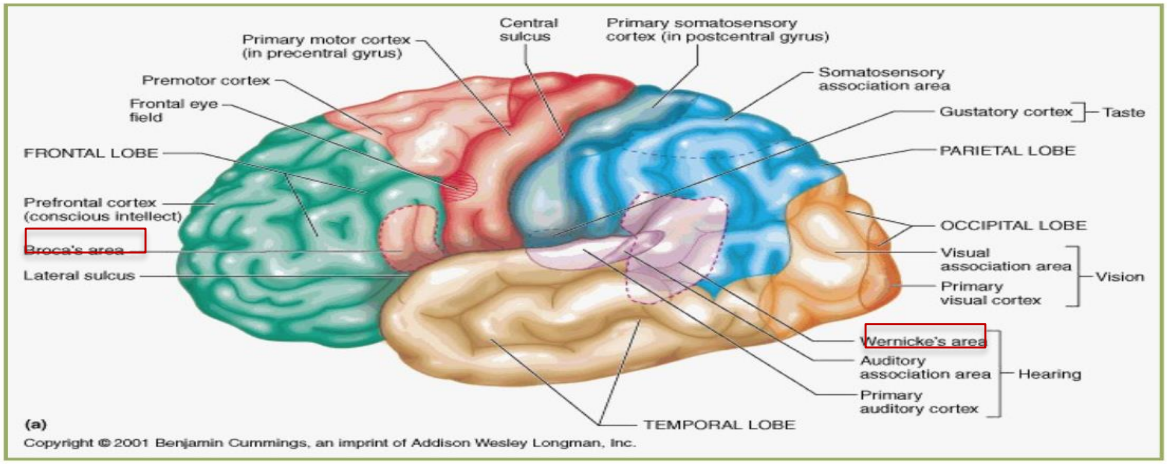
It's really important to distinguish between the types

Simply we can summarize the communication disorders into 4 categorizes

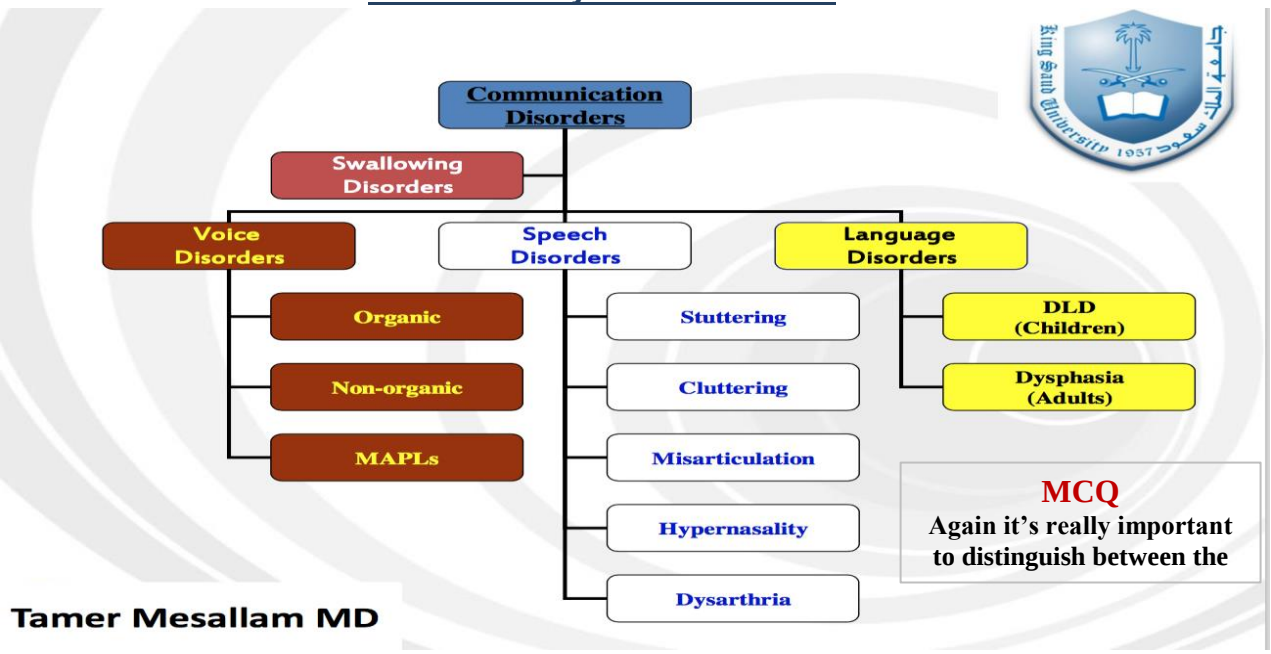


A.Physiology of communication

1.Language	A symbolic arbitrary system relating sounds to meaning. -The major area of language comprehension called Wernicke's area: 1. This area lies behind the primary auditory cortex in the posterior part of the superior gyrus of the temporal lobe. (problem will cause fluent aphasia but have no meaning) 2. The visual association area that feeds visual information conveyed by words read from a book into Wernicke's area is the language comprehension area "angular gyrus area", lying in the anterolateral region of the occipital lobe.
2.Speech	A neuro-muscular process whereby language is uttered تلفظ. It includes the coordination of respiration, phonation, articulation, resonation and prosody. -Prosody is the ability to change the tone while speeking=> lost in Parkinson disease= (1 tone speech) -Broca's area (problem will cause expressive aphasia understand but can't express), located partly in the cortex and partly in the premotor area, is responsible for expressing individual's words and short phrases. It works in association with Wernicke's area.
3.Voice	The result of vibration of the true vocal folds using the expired air. • 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.
4.Swallowing	The process of successful passage of food and drinks from the mouth through pharynx and esophagus into the stomach.



A.Disorders of communication



1.Language Disorders:

- Central language control

-The left hemisphere is the processor of language functions in almost all people regardless handedness. It is the dominant hemisphere.

- Language areas are distributed along the rolandic fissure. (central fissure)

1.Anterior language area mainly in the temporal region concerned with expressive aspect. (Broca's)

2.Posterior language area mainly in the parietal region concerned with receptive aspect. (Wernicke's)

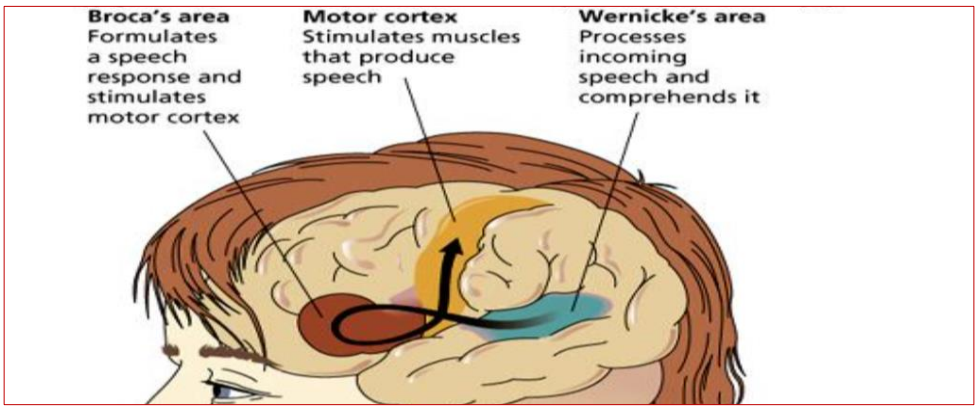
- Structural domains of language (skipped)

Semantics➔ meaning. Phonology➔articulation. Syntax➔grammar.

- Stages of normal language development (skipped)

2-4 mo	Babbling
6 mo	Vocal play
9 mo-1 yr	1st word
1-1/2 yrs	20 words

2 yrs	200 words, 2 word sentence
3 yrs	2000 words,3 word sentence
4 yrs	4 word sentence
5-7 yrs	Full maturation of all language modalities.



Delayed Language Development (DLD)	Dysphasia
○ Definition Delay or failure to acquire language matched with age. Ex. 4 yr old child who knows 4 words only!!	○ Definition: Language deterioration after its full development due to brain insult: infarction, hemorrhage, atrophy, etc.
○ Pre-requisites of normal language development: 1.Intact brain functions (conceptual, motoric and cognitive abilities). 2.Intact sensory channels; Auditory, Visual, Tactile, Kinesthetic. 3.Intact psyche. 4.Stimulating environment.	○ Types of dysphasia: 1.Expressive (e.g. broca's aphasia) Understand but cannot speak, very traumatic psychologically. (better prognosis) 2.Receptive Can speak but he doesn't understand 3.Mixed predominantly expressive 4.Mixedpredominantlyreceptive. 5.Global.
○ Etiology (opposite of pre- requisites) 1.Brain damage. • Diffuse subcortical lesion (M.R.). • Localized brain damage with motor handicap (BDMH). • Minimal brain damage (ADHD[1]),medication then speech therapy. 2.Sensory deprivation. • Hearing impairment: Conductive, Sensory-neural , Mixed , Central auditory processing disorder. • Visual impairment. 3.Psychiatric illness. Autism. , Autism Spectrum Disorder (ASD). 4.Environmental deprivation. Lonely child 5.Idiopathic.Specific Language Impairment (best prognosis)	○ Etiology; CVA / Neoplastic / Traumatic /Inflammatory / Degenerative / Metabolic / Poisoning Ex. 1.Expressive: Dr:Where's your son? Pt:Points to his son Dr:Ok, What's his name Pt:……. (unable to answer to a direct Q) 2.Receptive: Dr: How R U today Pt: oh, yes I slept well yesterday..
○ Assessment of DLD: 1. History taking. 2. Physical examination. 3. Investigations: -Psychometry (IQ). - Audiometry. -EEG. -Brian Imaging . -Ophthalmological consultation.	○ Assessment of Dysphasia: 1.History taking. 2.Physical examination: ..., neurological exam. 3.Investigations: -CT / MRI brain. - Dysphasia test. -Psychometry (IQ). - Audiometry.
Management: • Early detection. late=> worse prognosis • Providing the suitable aid: Hearing (HA or CI), Visual Aid, Physiotherapy. • Family counseling. encourage them to speak to him. • Direct language therapy (Individual- group). Medications (Autism and ADHD).	○ Management: • Treat the cause. • Physical rehabilitation (Physiotherapy). • Family counseling. • Language therapy. Alternative and augmentative communication: Cards, sign boards,

[1] Attention Deficit Hyperactivity Disorder

2. Speech disorders

1. Dyslalia (Misarticulation) لدغة	2.Stuttering تاتاة
○ Definition Faulty articulation of one or more of speech sounds not appropriate for age.	○ Definition: The intraphonemic disruptions resulting in sound and syllable repetitions, ككككورة sound prolongations, كووووورة and blocks. Worst prognosis
○ Types: Types: ○ Sigmatism (/s/ defect): سبورة 1. Interdental stigmatism. ثبورة 2. Lateral sigmatism. شبورة 3. Pharyngeal sigmatism. • Back-to-front dyslalia:كورة /k/ =/t/. /g/ =/d/ تورة • Rotacism (/r/ defect): ريما:غيما=ويما=ييما ☺ • Voiced-to-nonvoiced dyslalia: /g/ /k/- /d//t/ -/z//s/etc...	○ Types : •Normal dysfluency: 3-6 years. Only repetitions. No associated muscular activity. Not aware. • Incidence of stuttering: 1%. • Onset: Earliest = 18 months. Latest = 13 years. • Epidemiology: - More in families with history of stuttering. - Can occur in mentally retarded. - Very rare in the hearing impaired. • Gender ratio: 4 : 1 (male : female) , worst in females Theories of Stuttering: The exact cause is unknown. • Organic theory (doctor goes with organic more) • Neurosis theory. Learning theory
Assessment of DLD: 1. History taking. 2. Physical examination. 3. Investigations: -Audio recording. - Audiometry -Articulation test. - Psychometry (IQ)..	○ Assessment of Dysphasia: 1.History taking. 2.Physical examination 3.Investigations: - Audio & video recording. -Stuttering severity -Psychometry (IQ). -Articulation test -Auditory Perceptual Analysis (APA)(see nxt p)
Management: 1.Treatment of the cause: • Tongue tie (prevents elevation of the tongue (can't say La) • Dental anomalies (open bite) • Hearing aids 2.Speech therapy with assistance and counseling.	Management: Family and patient counseling. ○ Speech therapy: a. Indirect therapy: if not aware. on the family side(slowing their talk) b. Direct therapy: if aware.

- Auditory Perceptual Analysis (APA)

A. Core behaviors:

- Intraphonemic disruption.
- Repetitions.
- Prolongations.
- Blocks.

B. Secondary reactions: moving the shoulder/ twitch the muscle

- Muscular activity and struggle. – Interjection - Word substitutions and circumlocution.

C. Concomitant reactions: من الخوف ما يرد السلام

- Fear.
- Eye contact.
- Skin pallor/flushing
- Breathing (antagonism, interruption, prolongation, cessation, ...)

3. cluttering : is a fluency disorder characterized by a rapid and/or irregular speaking rate, excessive disfluencies.

4. Hypernasality:

- Definition:

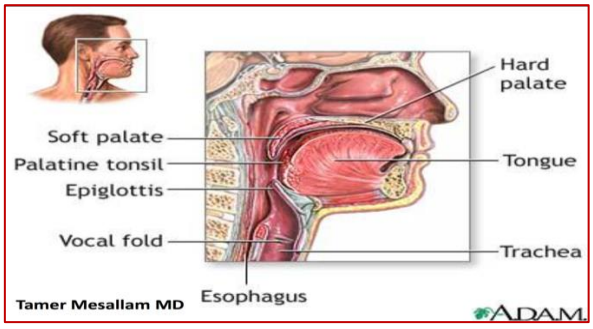
Faulty contamination of the speech signal by the addition of nasal noise. It results from velopharyngeal dysfunction (VPD) or insufficiency (VPI) . مناديل=بنديل

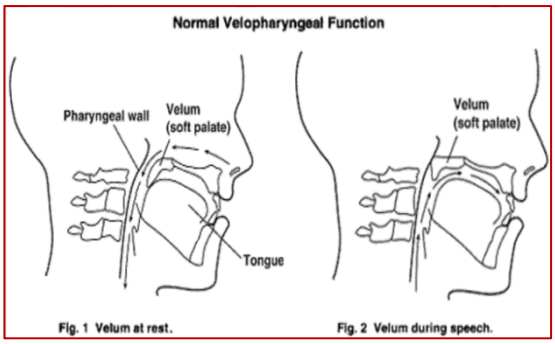
-All of us have nasal tone, we use it in M+N, however it doesn't exceed the limits.

-Hyponasality => close type=> Flu, Adenoid (more common)

-Hypernasality => open type=> velopharngeal dysfunction (VPD)

-We're concerned with soft palate(velum) which separates the nasal cavity from the mouth



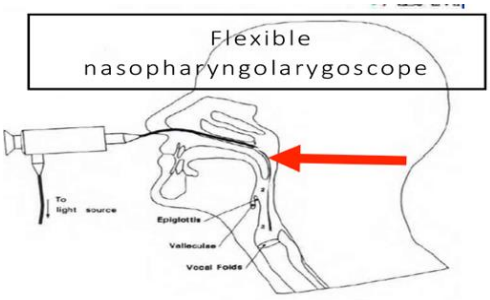


Etiology:

Organic	Non-organic (Functional) VP Mis- learning
1.Structural: a) Congenital: -Overt cleft palate. -Submucous cleft palate. -Non-cleft causes: 1.Congenital short palate 2.Congenital deep pharynx b)Acquired -Adenotonsillectomy. 2-4 weeks => temporary (pain=> decrease movement=> more inx are needed if it exceeds 4 weeks> surgical error -Palatal trauma -Tumors of the palate & pharynx. ***2. Neurogenic:*** *(VP Incompetence)* Palatal U motor neuron lesion/ Palatal L motor neuron lesion.	· Faulty speech habits. · Mental retardation · Hearing impairment. · Post-tonsillectomy pain.

Effects of VPD:

- Feeding problems: nasal regurgitation.
- Psychosocial problems.
- Communicative problems:
  - *Speech:* hypernasality.
  - Language: DLD.
  - *Voice:* hyper or hypofunction.



Assessment of hypernasality (VPD)

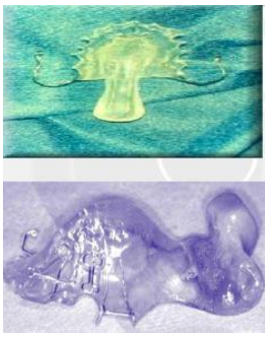
- Parent interview.
- Perceptual.
  - Simple tests:
    - Gutzman's (a/i) test.
    - Czermak's (cold mirror) test:
    - Resonance, Articulation, Nasal air emission, Voice. (no consndation while talking)
- Intra-oral evaluation
- Instrumental:
  - Nasopharyngoscopy
  - Nasometry = How much nasality u have?
  - Fiberoptic nasopharyngolarygoscope

  - Management:
    - Multidisciplinary team.
    - Management of feeding problem.
    - Palatal and lip surgeries.
    - Obturators.
    - Communication :(language, speech, voice)
    - Maxillofacial.
    - Hearing.
    - Feeding.

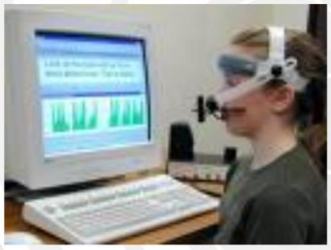
  - Treatment Decision
    - **Velopharyngeal insufficiency:** surgery (speech therapy post-op)
    - **velopharyngeal incompetence:** surgery (speech therapy post-op) / prosthetic devices / speech therapy
    - **velopharyngeal mislearning:** speech therapy

  - Surgery
    Pharyngeal flap / sphincter-platoplasty / post-pharyngeal wall augmentation.

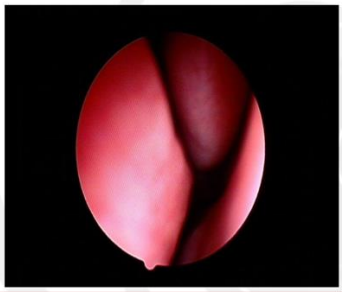
  - Prosthtic device
    - Palatal lift: to raise the velum when there is poor velar movement (i.e. dysartheria)
    - platal obturator: to occlude an open cleft or fistula
    - speech bulb: to occlude nasopharynx



Nasometry



Normal closure



Severe VPD

Pharyngeal flap

Submucus Cleft

Difficult to diagnose=> triad

1.bluish central line in soft palate

2.bifid uvula

3.post nasal notch (instead of spine)

- contraindicated to do adenoidectomy=> hypernasality

4. Dysarthria:

- Definition:

Any combination of disorders of respiration, phonation, articulation, resonance, and prosody, that may result from a neuromuscular disorder.

- Types of dysarthria:

Type	1. Flaccid	2. Spastic	3. Ataxic	4. Dyskinetic	5.Mixed
Lesion:	lower motor neuron level.	upper motor neuron level.	cerebellum level.	basal ganglia level.	may the most common
Communication	breathy phonation.	strained strangled phonation.	increased equal stresses.	A. Hypokinetic type (Parkinsonism): * breathy phonation. * rapid rate. * short rushes of speech with final decay.	- Examples: 1.Motor neuron disease: Flaccid +Spastic. 2.Multiple sclerosis: Ataxic + Spastic. 3. Wilson's disease: Ataxic + Spastic + Hypokinetic.
	hypernasality .	labored breathing.	irregular articulatory breakdown.	B. Hyperkinetic type: i. Quick hyperkinetic (Chorea): variable rate and loudness. ii. Slow hyperkinetic (Athetosis): slow rate.	

- Assessment of dysarthria:

I. History taking.
II. Physical examination: mouth, palate ,neurological exam,
III. . Investigations:

- Audio recording.
- CT/MRI brain
- Articulation test.
- Audiometry.
- Aerodynamics (Aerophone II).
- Fiberoptic nasopharyngolaryngoscopy.
- MDVP.
- Dysphasia test.
- Psychometry (IQ).
- Nasometry.

Management of dysarthria:

- Individualized:
- Management of the cause.
- Patient counseling.
- Communicative therapy:
  - * Articulation. * Phonation. * Resonance. * Respiration. * Prosody.
  - Alternative and augmentative communication.

1. Voice disorders:

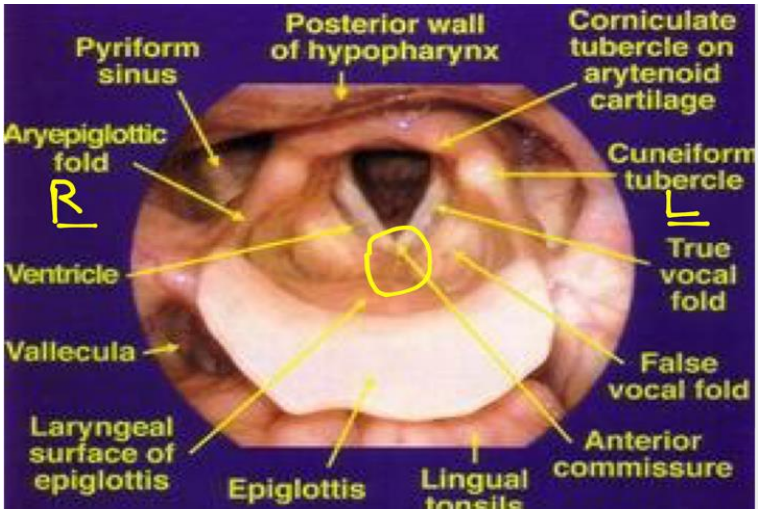
- Prerequisites of "normal" voice production:

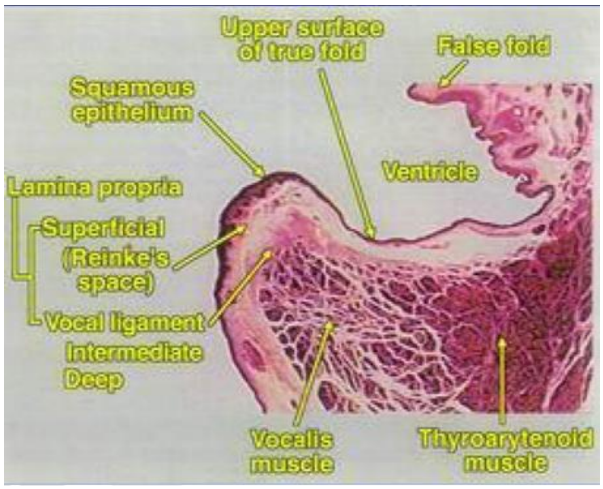
1. Normal range of movement of vocal folds.
2. Normal mobility of mucosa on deep layers.
3. Optimal coaptation of vocal folds' edges.
4. Optimal motor force.
5. Optimal pulmonary support.
6. Optimal timing between vocal fold closure and
2. pulmonary exhalation.
7. Optimal tuning of vocal fold musculature (int. & ext.).

- Usually the presenting symptoms in voice disorders are:

Dysphonia:	Any change of the patient's voice from his habitual one. -"Difficulty in phonation". - "Change of voice from his /her habitual". - "Hoarseness" = roughness & harshness of voice. = Objective, we don't like+ Dysphonia is more broad (includes high pitched)
Aphonia:	Loss of the patient's voice (functional or organic);
Phonasthenia: Voice fatigue	a subjective complaint of dryness, tightness, globus feeling and voice fatigue, while the patient's voice and larynx is normal.
Dysodia:	Change of the singing voice while the speaking voice is normal.

VERY IMPORTANT: Know the anatomy and the sides (right or left, which depends on Ant. Commissure)



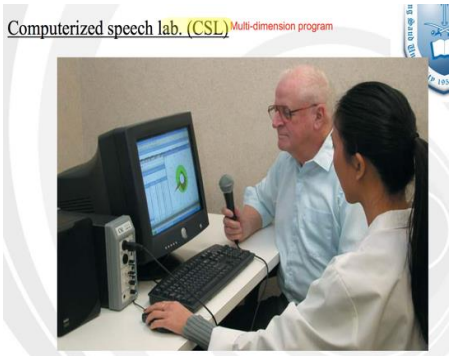


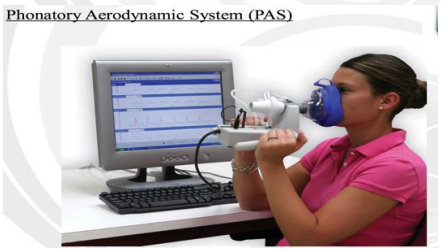
- Etiological classification of dysphonia:

Organic	Non-organic Normal phx		**Minimal Associated Pathological Lesions (MAPLs)**	Accompaniment of Neuro-psychiatric Ailments
1.Congenital. 2.Inflammatory. 3.Traumatic. 4.Neurological. 5.Neoplastic. 6.Hormonal. 7.Status post-laryngectomy.	Habitual	Psychogenic	1. Vocal fold nodules. 2. Vocal fold polyps. 3. Vocal fold cysts. 4. Reinke's edema. 5. Contact granuloma.	
	1.Hyperfunctional childhood dysphonia. 2. Incomplete mutation. 3. Phonasthenia (Voice fatigue). 4.Hyperfunctional dysphonia. 5. Hypofunctional dysphonia. 6. Ventricular dysphonia.	1.Psychogenic dysphonia. 2.Psychogenic aphonia. عاوزين يقوزوها ابن عمها وهي رافضة ☺!		

- Assessment of dysphonia:

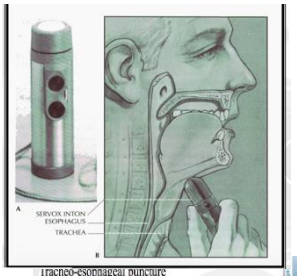
I. History taking.
II. Physical examination: APA , neck ,
III. Investigations:
- Audio recording. - Digital laryngostroboscopy.
- Digital laryngokymography. - Acoustic analysis (MDVP).
- GERD (LPR) work-up. - CT neck.
- Aerodynamic analysis (Aerophone II).
-Rigid scope is better, but use flexible with in-cooperative pts.
-CSL=Acoustic analysis of voice, intensity and frequency(Quantities).

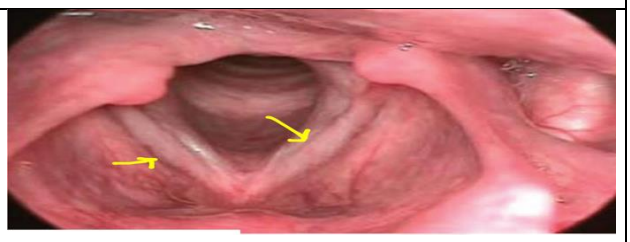
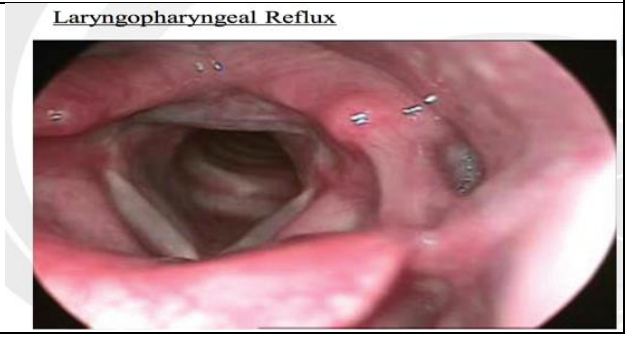




- Management of voice disorders:

Pharmacological agents. Ex.GERD
Surgical procedures (Phonosurgery).
Technical aid devices.
Voice therapy.



Pictures of cases by laryngoscopy with explanation (you don't have to memorized it)	
Piriform fossa : is a site of constriction **which means it's a** common place for food to become trapped injury in this area gives a feeling of food stuck in throat. It's also a commonest site for hypophyseal carcinoma	
Congenital vocal cords web: incomplete recanalization of the laryngotracheal tube most common site in level of vocal cords Present: mild dysphonia to significant airway obstruction **Management : surgical lysis of the webs with laser or cold knife**	 
Congenital laryngeal cleft Rare abnormality in the posterior laryngeo-tracheal wall resulting in incomplete closure of larynx during swallowing Present with: abnormal feeding , and failing to gain weight. Treatment : filler injections but depends on degree	 
sulcus vocalis (sulcus) is a groove or infolding of mucosa along the surface of the vocal folds it has a genetic predisposition present with dysphonia treated by: vocal folds injection (filler)	
Laryngophayngeal reflux Present with :throat clearing, cough, and hoarseness Usually examined with flexible laryngoscope Findings :edema and erythema Management : PPIs and diet changes , lifestyle modifications , stress management	

Laryngomalacia : means soft larynx

The epiglottis becomes soft and floppy and cannot maintain it arch shape so it forms a omega shape.

It present with stridor at babies usually 6 months it's self corrected around 12-18 months if not resolved surgery

Diagnosed by : laryngoscopy

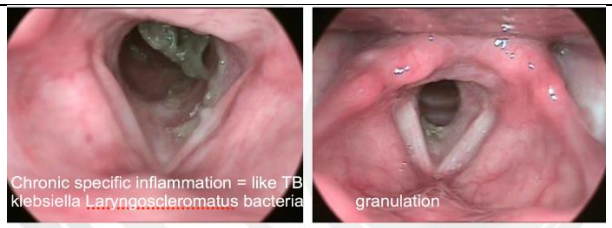
Fungal infection

Seen in immunocompromised patients , Diabetics , or those taking inhaled steroids mostly candidiasis

superficial colonization of the mucosa; the oral cavity is often involved

Laryngoscleroma

is rare chronic specific granulomatous infection of larynx cause by a bacteria called *K rhinoscleromatis*



Vocal cords paralysis

Here left vocal cord is paralyzed

How it present? (unilateral)

Dysphonia – aspirations

If it's bilateral how it present ?

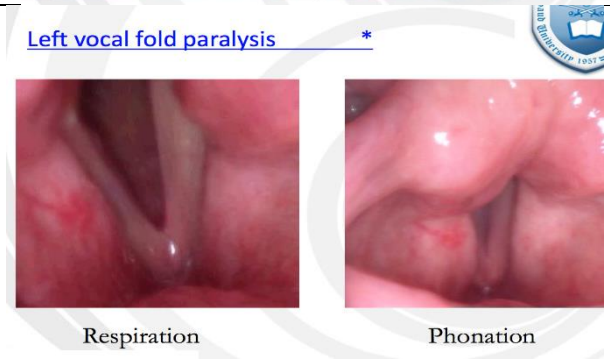
Airway obstruction – swallowing abnormality

How is it diagnosed ?

History (course , trauma , trauma in delivery if infant , CNS abnormality , intubation , surgery)

Management ?

Tracheostomy(ER) or Posterior cordotomy or Suture lateralization



Laryngeal carcinoma

squamous cell carcinoma is the most common

Risk factors :

Tobacco use - Excessive ethanol use

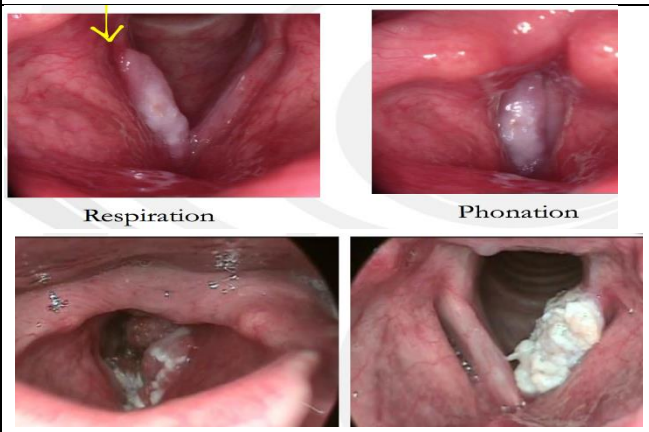
Infection with human papillomavirus

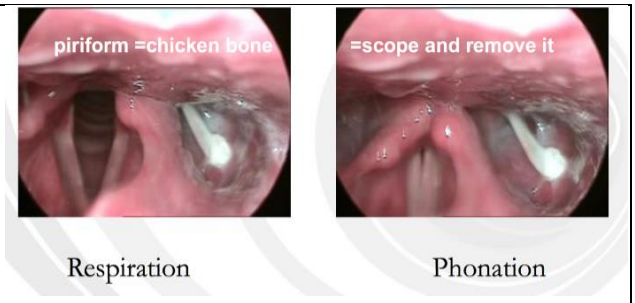
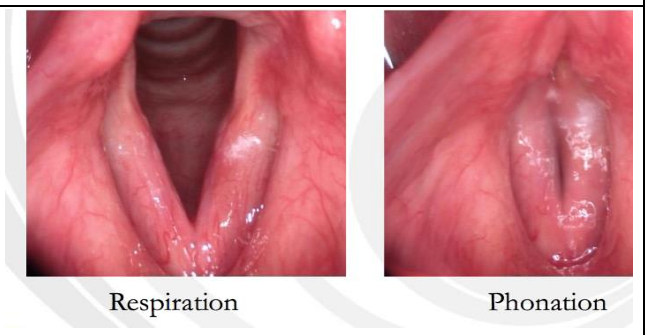
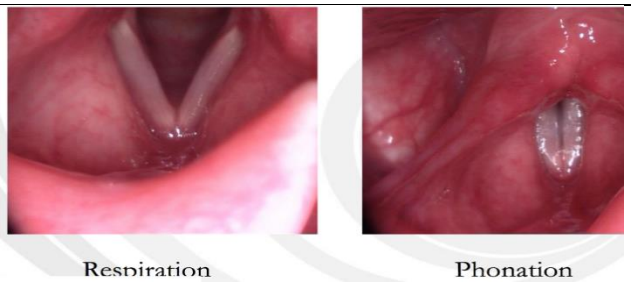
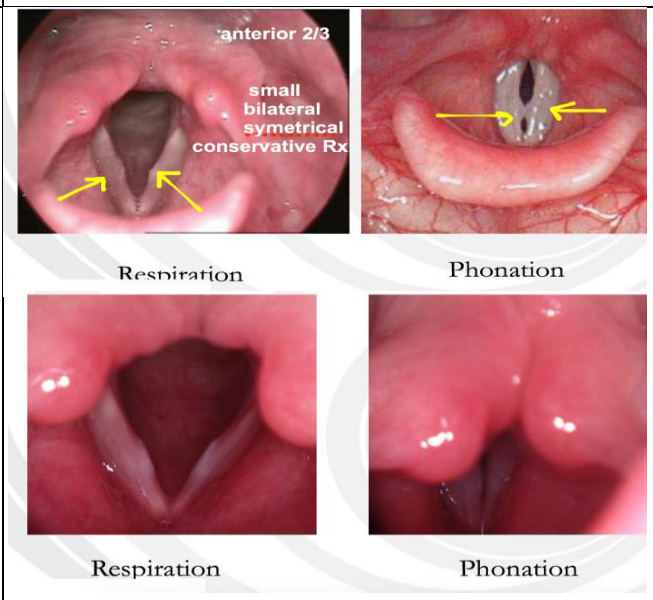
Increasing age

Management :

Early stages(I-II : radiation or surgical techniques

Late stages (III-IV) : total laryngectomy, reconstruction, and adjuvant postoperative chemoradiation therapy



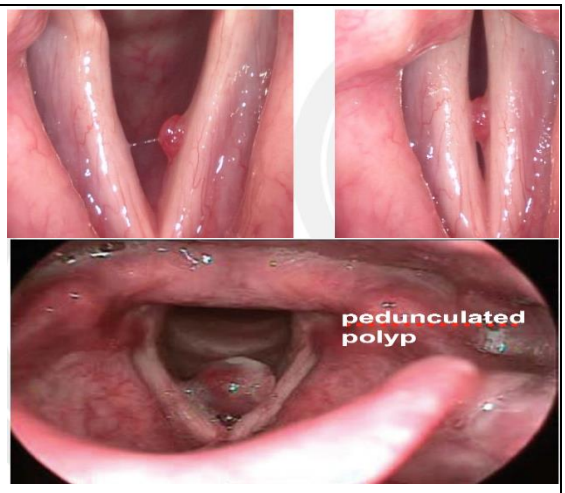
Like mentioned in 1st case piriform fossa is a common site for foreign body to get trapped in the good thing is you can scope and remove it in clinic	
Hyperfunctional dysphonia : • Usually due to miss use of voice (kids, singers , teachers) • Might be found in males that didn't acquire the voice change during their adolescence • No organic changes found, normal larynx maybe increase in vascularization , and decreases in phonatory gap	
Phonasthenia : Phonasthenia is the most common one, could be due to voice problem or reflux. Treated by voice therapy. With voice fatigue . also phonatry gap . T: voice thereby (team 432)	
Vocal fold nodules (adult) Bilateral nearly small symmetrical lesions at the junction of anterior and middle two-thirds of membranous vocal folds. Present with dysphonia Causes: vocal misuse and abuse, and chronic repetitive phonotrauma. Treatment: voice therapy. , voice arrest .with vocal hygiene advice) Surgery (rearly !)is indicated if case of asymmetrical lesions, fibrotic lesions, or failed therapy. (team 432) **Vocal fold nodules (juvenile)** Same but in pediatrics	

Left vocal fold polyp

Etiology :by acute phonotrauma.

Describe : usually Unilateral Can be bilateral and nearly in the same area. Red polyp = hemorrhagic wait and re evaluate

Treated by surgical excision and post-op voice thereby (team 432)

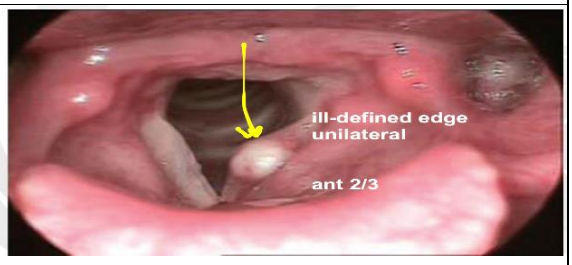


Vocal cord cyst

Localized intra-foldal lesion with il-defined edge unilateral usually in anterior 2/3

Etiolgy : phonotrauma and could be congenital. The mucosal waves on digital laryngostroboscopy are missed because they can't cross the dense mass.

Treatment: surgical excision followed by post-op voice therapy with voice hygiene advice. (team 432)



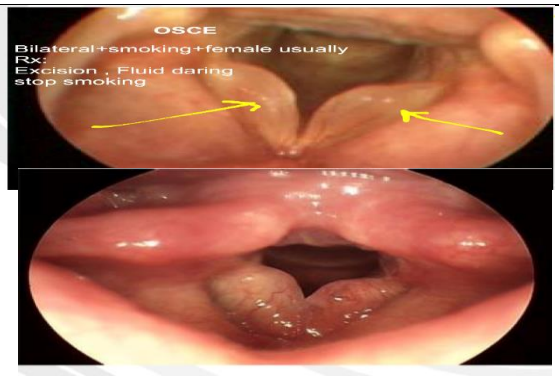
Bilateral reinke's edema :

bilateral Reinke's edema is usually bilateral and could be unilateral.

Etiolgy : phonotrauma, smoking and reflux (GERD).

It is common among middle-aged smoker active women. Treated :

smoking cessation, reflux medications and surgery (team 432)



Right contact granuloma

Contact granuloma grows on the posterior glottis.

Etiolgy : intubation – phonotrauma - severe laryngopharyngeal reflux.

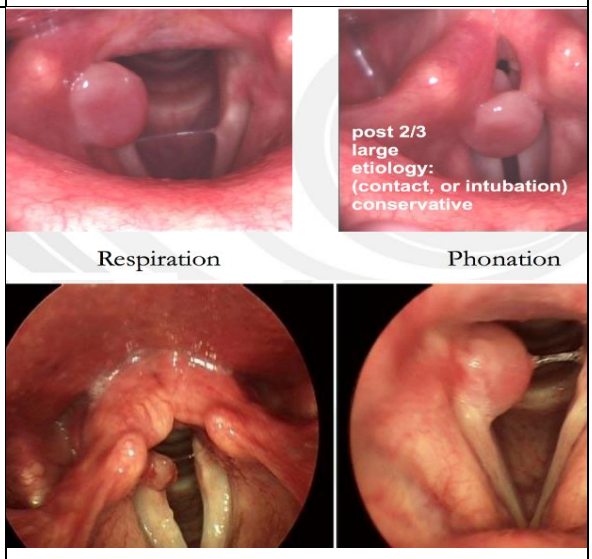
If there is a history of intubation it's intubation granuloma

Treat : the cause, and avoid surgery because of risk of recurrence, unless the lesion was big enough to obstruct the airway. (team 432)

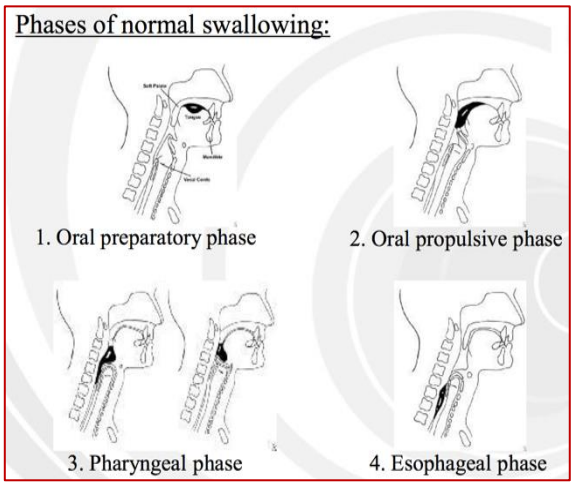
Stroboscope: is a special method used to visualize vocal fold vibration. It uses a synchronized, flashing light passed through a flexible or rigid telescope. The flashes of light from the stroboscope are synchronized to the vocal fold vibration at a slightly slower speed, allowing the examiner to observe vocal fold vibration during sound production in what appears to be slow motion

females reach 300 cycle/ sec

Males reach 120-130 cycle/ sec



3. Swallowing disorders:



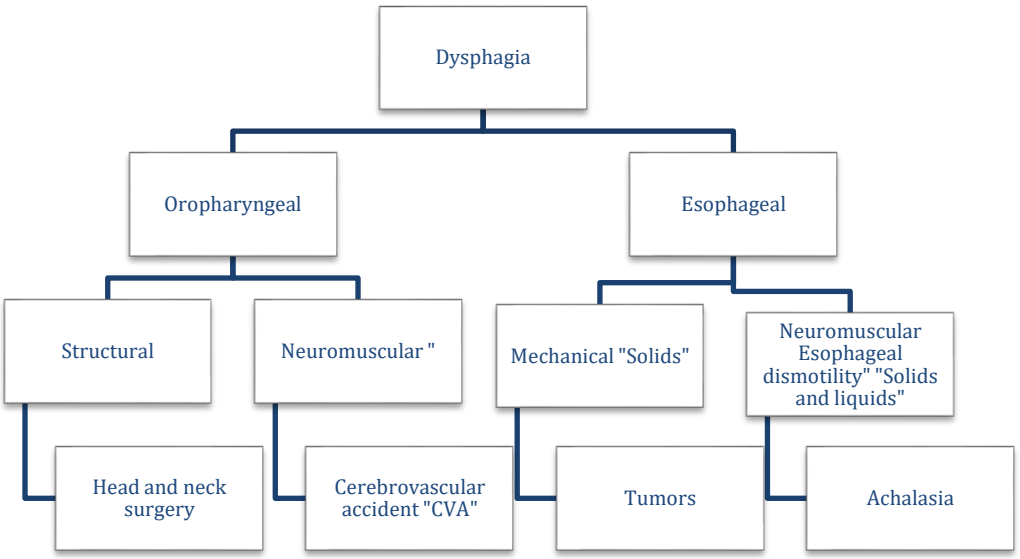
- Definition:

Difficulty in moving food from the mouth to the stomach".
"Odynophagia" = painful swallowing due to a disorder of the esophagus.

- Consequences of dysphagia:

-Dehydration.
-Weight loss.
-Aspiration pneumonia. (MOST dangerous)
-Airway obstruction.
0Loss of joy of eating.

- Causes of dysphagia:



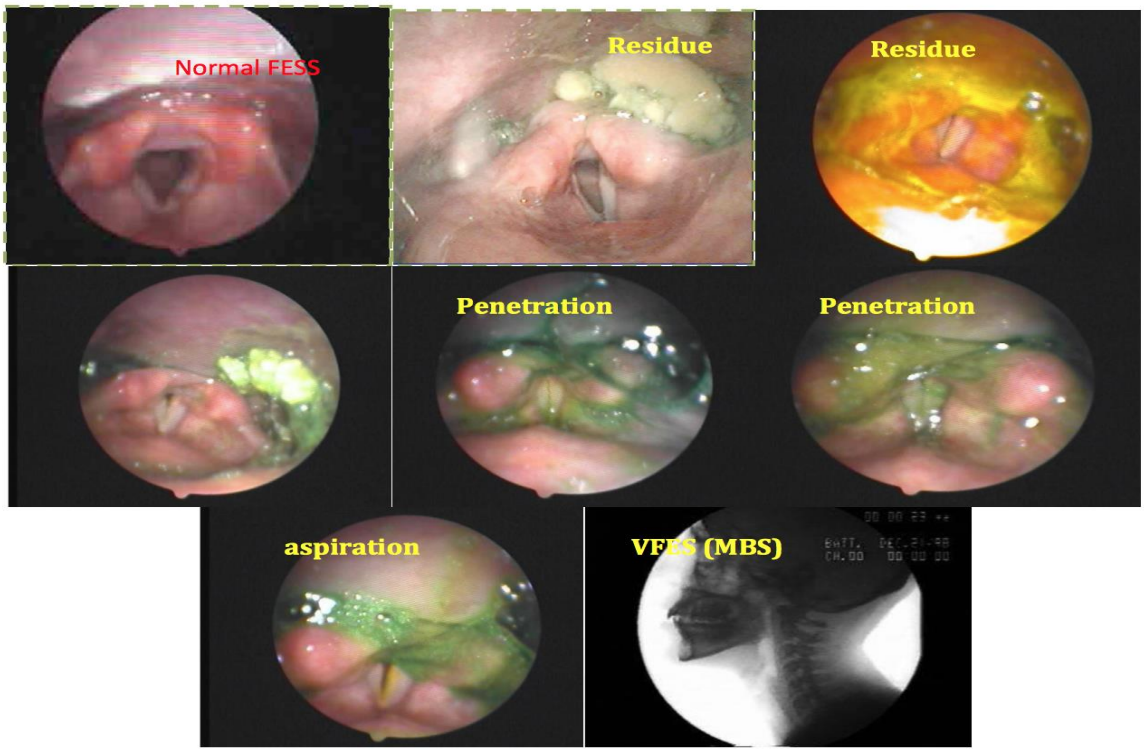
Assessment of dysphagia:

I. History taking
II. Physical examination:
--- General examination
--- Language and speech assessment --- Vocal tract examination
--- Neck examination
--- Trail feeding
III. Investigations:
--- FEES "Fiberoptic endoscopic evaluation of swallowing"
--- VFES (MBS) "Video fluoroscopic swallowing exam" (Modified barium swallow)
--- GERD (LPR) work---up

FEES protocol of evaluation (Langmore, 2003):
I. Anatomic and physiologic assessment.
II. Assessment of food and liquid swallowing.

III. Assessment of therapeutic interventions.

Note:
Penetration= at the vocal cords
down to trachea= aspiration



Management of dysphagia:

1. Swallowing therapy:
- Diet modification.

Dysphagia with water?? Try make it more thick=> juice

- Postural techniques.
- Swallowing maneuvers.
- Sensory enhancement techniques.
- Motor exercises.

2. Surgical treatment, eg medialization laryngoplasty.
3. Medical (Drug) treatment, eg anti-parkinsonism drugs.
4. Intraoral prosthesis.
5. Alternative routes of feeding, eg NG tube feeding.