



Communication & Swallowing Disorders I-II

Objectives:

- Understand physiology of communication.
- Recall different categories of communication and swallowing disorders.
- Differentiate different causes of communication and swallowing disorders.
- Assess and manage different communication and swallowing disorders.

437A: Most important VOICE, SWALLOWING AND HYPERNASALITY

Resources: Doctor Slides, Team 436F

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[Color index: **Important** | **Notes** | Extra]

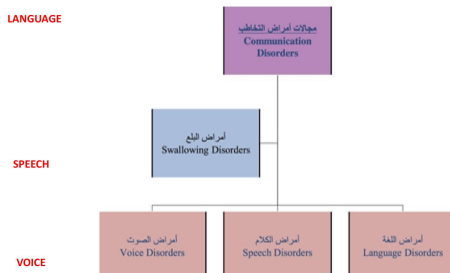
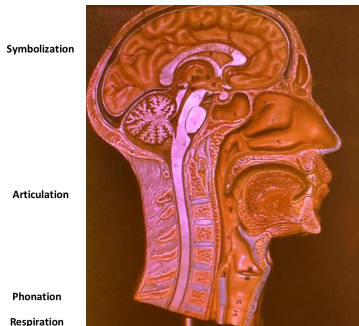
Definitions

Communication difficulties have an impact on the following aspects:
Academic, Social, Psychological, Employment, Professional, Financial, Family relations

Communication	<ul style="list-style-type: none"> - Exchange of thoughts, ideas, emotions between two parties - Types: Verbal, Non verbal - Parts of communication: 1.Voice, 2.Speech, 3.Language (try to mention them in this order)
Voice	- The result of vibration of the true vocal folds using the expired air
Speech (Articulators)	<ul style="list-style-type: none"> - A neuro-muscular process whereby language is uttered. تلفظ - It includes the coordination of respiration, phonation, articulation, prosody and resonance. الحروف والأصوات الواضحة، بطلاقة جيدة وبدون خنة
Language	- A symbolic arbitrary system relating sounds to meaning
Swallowing	- The process of successful passage of food and drinks from the mouth through pharynx and esophagus into the stomach.

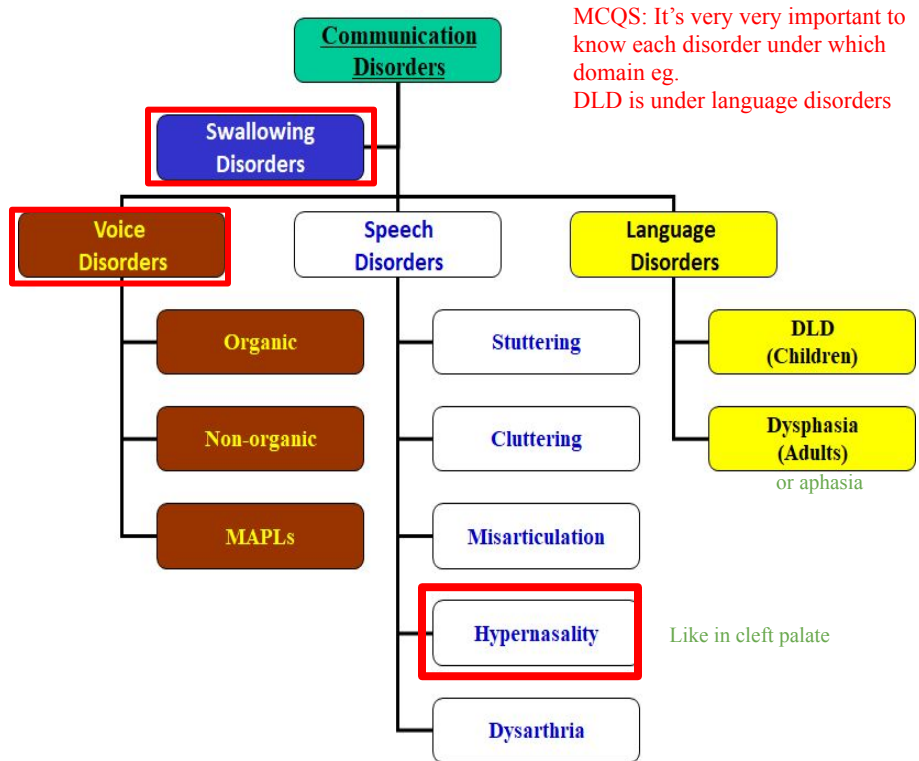
How communication happen? **Very Important**

- In order to communicate 4 physiological process should happen in certain arrangement :
 1. Inspiration → expiration → air passes through sound box larynx, expiratory phonatory airflow → 2. vibration of vocal folds “not cords!” → 1&2 gives voice, primary laryngeal sound → 3. Articulation by supralaryngeal compartments (pharynx, epiglottis, tongue, soft palate, teeth, sinuses) to articulators or resonators/speech, they produce a person's recognizable voice “Speech” → 4. symbolization by the brain (language)
- Function of supralaryngeal compartments: change of primary laryngeal sound (voice) into secondary sound (speech).
- Disorders of communication can occur in each of the following: Language, Speech, Voice.



Who is managing communication and swallowing disorders?

1. **Phoniatricians (MD's)**
 - a. A medical specialty that deals with communication and swallowing disorders.
 - b. It stems mainly from ORL (ENT), especially when dealing with voice disorders.
2. **Speech-Language pathologist**



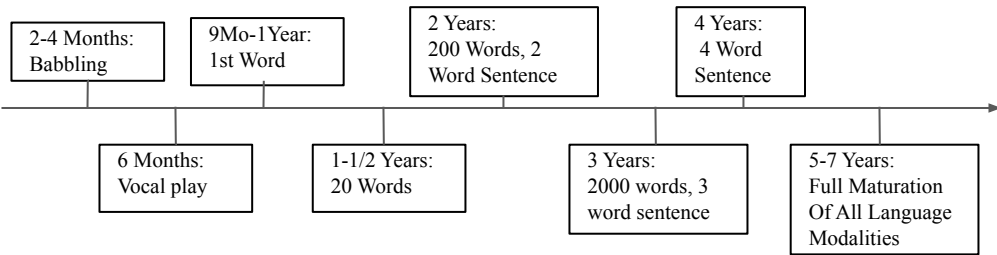
437A: The ones that circled with red are the most IMPORTANT

1. Delayed Language Development (DLD)

Definition:

Delay or failure to acquire language **matched** with age. Ex. 4 yrs. old child who knows 4 words only! i.e. an 8-month-old baby can't talk, you won't diagnose with DLD because at this age it's normal.

Stages of normal language development: any delay in stage could be considered as DLD



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. (ADHD , Autism)
4. Stimulating environment, very important **If everything is normal but the kid is left alone -> will not talk**

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.
- Anterior language area mainly in the temporal region concerned with expressive aspect.
- Posterior language area mainly in the parietal region concerned with receptive aspect.

Structural Domains Of Language:

- Semantics: meaning.
- Phonology: articulation.
- Syntax: grammar.

435 notes:

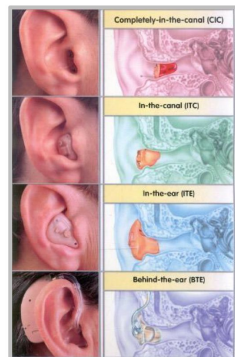
- Babies should say their first word at their 1st year (9 months – 1yr).
- At their 3 year he should be able to say more than one sentence if not they may have DLD.
- A parent with a baby who speaks no more than 2 word at age of 2 should seek advice.

Etiology Of Delayed Language Development: opposite of pre-requisites

1. Brain damage:
 - Diffuse subcortical lesion (Mental Retardation.)
 - Localized brain damage with motor handicapped child (BDMH)
 - Minimal brain damage (ADHD), medication then speech therapy.
 - Cerebral palsy (CP), hypoxia or trauma during delivery.
2. Sensory deprivation:
 - Hearing impairment: Conductive, Sensorineural, Mixed, Central Auditory Processing Disorder
 - Visual impairment
3. Psychiatric illness:
 - Autism, Autism Spectrum Disorder (ASD), childhood schizophrenia
4. Environmental deprivation:
 - Non-stimulating environment: lonely child, first and last child
5. Idiopathic (Specific Language Impairment), best prognosis. Everything intact

Assessment Of Language Development:

1. History taking
2. Physical Examination of articulators
3. Investigations:
 - *Psychometry (IQ) mental retarded or not
 - *Audiometry
 - *DLD sheet
 - *Brain Imaging for injury
 - *EEG
 - *Ophthalmological consultation

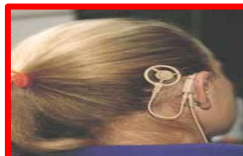
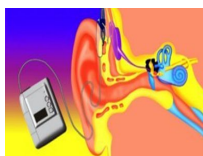


Management of DLD:

- Early detection, the earlier the better the prognosis due to brain plasticity being higher the younger a child is.
- Providing the suitable aid: Hearing aid (HA or CI) - Visual Aid - Physiotherapy
- Family counseling it is important for how to deal with child
- Direct language therapy (individual - group)
- Medications (autism and ADHD)

Saq Stop: Very Important

- What is this device called? Cochlear Implant. Cochlear Implant is contraindicated in children over 5 years old as they will benefit very little due to low brain plasticity.
- What are the indications for CI? Bilateral severe to profound sensorineural hearing loss.



2 is Implant part

2. Dysphasia

Definition:

Language deterioration **after** its full development due to brain insult: infarction, hemorrhage, atrophy, etc. Language center is in (Dominant)left hemisphere: anterior language zone is responsible for speech production and posterior language zone is responsible for comprehension. So when these areas are affected will lead to Broca's aphasia or wernicke's aphasia according to affected area, or expressive and receptive aphasia. When pt comes with infarction of left hemisphere could come with language problem; while in infarction of right hemisphere lead to dysarthria without language problem.

Etiology:

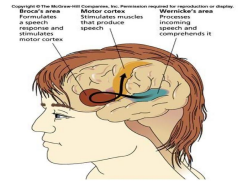
1. CVA
2. Neoplastic
3. Traumatic
4. Inflammatory
5. Degenerative
6. Metabolic
7. Poisoning
8. RTA

Full development of speech:

- Females: 7.5 years (more stutter)
- Males: 8 years.

Types Of Dysphasia:

1. Expressive: e.g. Broca's aphasia. Understands but cannot speak, very traumatic psychologically, better prognosis.
2. Receptive: Can speak but he doesn't understand, worse prognosis.
3. Mixed predominantly expressive
4. Mixed Predominantly Receptive
5. Global, most severe
 - Mixed types are more common
 - Examples:
 - a. 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)
 - b. Receptive: Dr: How R U today? Pt: oh, yes, I slept well yesterday...



Assessment Of Dysphasia:

1. History Taking
2. Physical Examination: Neurological Exam, sensory and motor.
3. Investigations:
 - *CT / MRI brain to see which domain of language is affected
 - *Dysphasia test to detect which area of language is affecting more
 - *Psychometry (IQ)
 - *Audiometry

Management of Dysphasia:

- Management of the cause. If tumor, if HTN causing Hemorrhage. etc.
- Physical rehabilitation 'Physiotherapy'
- Family counseling, they have big role! If isolated, the problem will deteriorate
- Language therapy

1. Dyslalia (Misarticulation) لدغة

Definition:

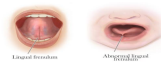
Faulty articulation of one or more of speech sounds not appropriate for age. And it is consistent.

Types: the first two are the most important

- Sigmatism (/s/ defect):** سبورة
* Interdental sigmatism ثبورة
* Lateral sigmatism شبورة
* Pharyngeal sigmatism خبورة
Sigmatism & back to front dyslalias more common in pediatric patients
- Rotacism (/r/ defect):** مگكب = مكب = موكب = ملكب : مركب more common in female
- Back To Front Dyslalia:** كورة
* k → t تورة * g → d
- Voiced To Nonvoiced Dyslalia:**
* g → k * d → t * z → s etc...
- Imitational Dyslalia:** parents have dyslalia → child never learned the correct sound

Assessment Of Dyslalia:

- History Taking
- Physical Examination “Tongue” check the articulators and tongue tie is “problem that’s frenulum is advanced anterior which attaches the tip of tongue and prevents elevation of the tongue, can’t say La or Ra ‘mostly letters affect’”.
- Investigations: Audio recording, Articulation test which letters affect, Psychometry (IQ), Audiometry for high frequency phonemes loss: pt who has problem in high frequency phonemes which can’t hear letters well like ص، س and pronounce them wrongly



Management Of Dyslalia:

- Treatment of the cause:
 - Tongue tie** treat by cutting the frenulum
 - Dental anomalies, open bite affects esp س
 - Hearing aids
 - Dyslalia sheet
- Speech Therapy** with assistance and counseling. Most cases treated this way and it very helpful. جلسات علاجية.



2. Stuttering أتاة أو تلثم

Time of onset is 1-13 years usually, most devastating psychosocially

Definition:

The **intrapliphonemic disruptions** resulting in sound and syllable repetitions أحمدأأأأ, sound prolongations أأأأأأ (first sound or whole word is prolonged), and blocks أأأأ (worst prognosis, hardest (MCQ!))

Types:

- Prolongation,** 2. **Repetitions,** 3. **Blocking** الكلمة توقف خالص

Most important problem facing children with stuttering is the avoidance:

يبدأ الطفل بتجنب المشاركة والتحدث ويصير كل أحد يتجنبه وهذا من أكثر الأشياء التي يزيد المشكلة سوء.

هذا اسمها عدم طلاقة طبيعية يعني ممكن تعدي بدون مشاكل ولكن الطفل عرضة للتلعثم + يقول كلام أكبر من عمره

3 to 6 years - Only repetitions - No associated muscular activity - Not aware - **NO blocks, any blocks is pathologic**

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) worse in females, because they are more shy and they get anxious about the problem too much that make them deteriorates.

Theories of Stuttering:

The exact cause is unknown. Although the cause it's not psychological, but this problem has a huge psychological impact.

- Organic/Genetic theory
- Neurosis theory
- Learning theory

Assessment Of Stuttering:

1. History Taking.
2. Physical Examination (APA, VPA, ...)
3. Investigations: Audio & video recording if there are reactions do during stuttering and try to avoid them. - Stuttering severity index (SSI) - Psychometry (IQ) - Articulation test.

Auditory Perceptual Analysis (APA)

1. Core Behaviors:	2. Secondary Reactions	3. Concomitant Reactions
- Intraphonemic disruption. - Repetitions. - Prolongations. - Blocks.	- Muscular activity and struggle (tremors) - Interjection will say مثلا a lot - Word substitutions and circumlocution to avoid saying the word they cannot pronounce	- Fear. - Eye contact (poor) - Skin pallor/flushing - Breathing (antagonism, interruption, prolongation, cessation, ...)

Management Of Stuttering: The only treatment till now is speech therapy جلسات علاجية

- **Family and patient counseling.** if they have a bad attitude
- **Speech therapy:** يكون فيها تعويد الطفل على إبطاء سرعة الكلام + التحكم في النفس
 - A- Indirect therapy: if not aware. on the family side (slowing their talk).
 - B- Direct therapy: if aware

3. Dysarthria

Here Language Center is intact, not like dysphasia

Definition:

*like parkinson's patients, masking of tone and emotion in speech

Any combination of disorders of respiration, phonation, articulation, resonance, and prosody* (intonations), that may result from a **neuromuscular disorder**. Brain and language are normal, the problem is in brainstem or nerves that supply muscles that produce speech sound. It affect the supralaryngial compartments

Types Of Dysarthria:

Type	Flaccid	Spastic	Ataxia	Dyskinetic	Mixed
Lesion	Lower motor neuron level	Upper motor neuron level	Cerebellum level	Basal ganglia level	May the most common
Communication	- Breathy phonation -Hypernasality	- Strained strangled. phonation. - Labored breathing	- Increased equal stresses. - Irregular articulatory breakdown.	a. Hypokinetic type (Parkinsonism): - breathy phonation - rapid rate - short rushes of speed with final decay b. Hyperkinetic type: i. Quick hyperkinetic (Chorea): variable rate and loudness. ii. Slow hyperkinetic (Athetosis): slow rate	- Examples: a. Motor neuron disease (Flaccid+Spastic) b. Multiple sclerosis: (Ataxic+ Spastic) c. Wilson's disease: (Ataxic + Spastic + Hypokinetic)

Assessment Of Dysarthria:

1. History Taking
2. Physical Examination: Mouth, Palate, Neurological Exam...
3. Investigations:
 - *Audio recording
 - *Fiberoptic nasopharyngolaryngoscopy
 - *CT/MRI brain
 - *Dysphasia test

dysarthria can come with dysphasia (Mixed)

 - *Psychometry (IQ)
 - *Articulation test
 - *Audiometry
 - *Nasometry
 - *MDVP
 - *Aerodynamics (Aerophone II)

Management Of Dysarthria:

- Individualized: no language therapy b/c it's intact unless it's mixed
 - Management of the cause
 - Patient counseling and speech therapy
 - Communicative therapy: depending on the area affected
 - Articulation - Phonation - Resonance - Respiration - Prosody
 - Alternative and augmentative communication. If not response

Pt tries to speak fast which is not appropriate with speed of articulators (muscles) so need counseling for how to slow speed of speech and exaggerate the production of sound.

4. Hypernasality: Imp

جميع الحروف تخرج من الفم عدا (م, ن) تخرج من الأنف
In Hypernasality some letters come also from the nose

Definition:

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

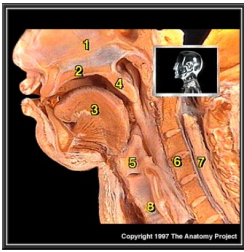
- Is a disorder that causes abnormal resonance in a human's voice due to increased airflow through the nose during speech.
- Nasal tone (soft palate is open) is used in the letter M (M nasal = B oral) and N
- Hyponasality → soft palate closed (closed nasality) e.g. common cold, polyp inflammation, edema, deviated septum adenoid, turbinate hypertrophy, chronic sinusitis. (M becomes B) .
- Hypernasality → soft palate open (open nasality) → velopharyngeal dysfunction (VPD).

Causes of hypernasality:

Organic	Non-organic (functional) VP mis-learning
<p>1- Structural (VP insufficiency):</p> <p>a. Congenital:</p> <ul style="list-style-type: none"> - Overt cleft palate - Submucous cleft palate - Non-cleft causes: <ul style="list-style-type: none"> • Congenital short palate • Congenital deep pharynx <p>b. Acquired cause velopharyngeal insufficiency:</p> <ul style="list-style-type: none"> - Palatal trauma - Tumors of the palate and pharynx - Adenotonsillectomy injury to tonsil <p>2-4 weeks => temporary (pain=> decrease movement=> more inx are needed if it exceeds 4 weeks> surgical error</p> <p>2- Neurogenic (VP Incompetence):</p> <p>a. Palatal upper motor neuron lesion.</p> <p>b. Palatal lower motor neuron lesion.</p>	<ul style="list-style-type: none"> - Faulty speech habits. - Mental retardation. - Hearing impairment. - Post-tonsillectomy pain so pt doesn't move palate. (transient) more than 3 weeks= surgical or iatrogenic trauma. - Neurosis or hysteria. <div data-bbox="621 816 994 1047"> <p>Normal Velopharyngeal Function</p> <p>Fig 1 Velum at rest. Fig 2 Velum during speech.</p> </div> <p>Picture: velum: at rest and during speech.</p> <div data-bbox="652 1078 1004 1509" style="background-color: #e0f0e0; padding: 10px;"> <p>velopharyngeal: consists of *soft palate + anterior, posterior and lateral pharyngeal walls. function: be closed during swallowing or speech. During speech completely close velopharyngeal with posterior and lateral tight which separates oral and nasal so the speech production comes from the mouth except in two letters "m" and "n" be opened and resonance happen in nasal cavity. In submucous cleft, the velum doesn't close the passage and air is allowed to pass through the nasal passage. This is what gives hyper resonance.</p> </div>

Effects of VPD:

- Feeding problems: nasal regurgitation = cleft palate
- Psychosocial problems
- Communicative problems:
 - Speech: hypernasality
 - Language: DLD like in cleft palate undergo to many surgeries and be hospitalized which prevent patients from exposure to environment also compensate leakage in velopharyngeal on vocal cord = hyper abduction during phonation.
 - Voice: hyper or hypofunction
- Ear Infections (tensor palati : CN V).



Important for SAQ

1- Nasal Cavity 2- Hard Palate 3- Tongue

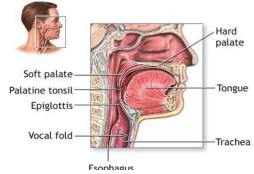
4- Soft Palate : هو المتحكم بخروج الأصوات مع الأنف

“Cleft Palate” فمتلا في حالات ال

هنا ما راح يقفل ويتحكم بخروج الأصوات لأن فيه مشكلة بالتالي بعض الأصوات راح تطلع مع الأنف على غير العادة

5- Larynx 6- Spinal column “Cervical part”

7- Spinal cord “Cervical part” 8- Trachea

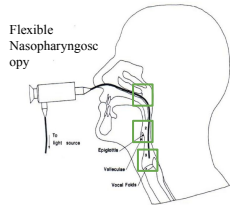


Assessment Of Hypernasality (VPD):

- Parent interview: **trauma, cleft abnormality..**
- Perceptual:
 - * Simple test: Gutzman’s (a/i) test - Czermak’s (cold mirror) test
 - * Resonance
 - * Articulation
 - * Nasal air emission **audible in severe**
 - * Voice
- Intra-oral evaluation
- Instrumental: **Nasopharyngoscopy - Nasometry**



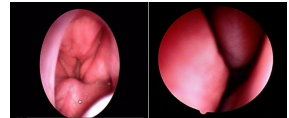
Early Condensation on surface of cold mirror



Normal closure

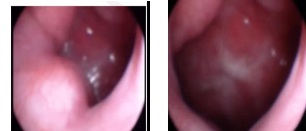
Examination:

- General
- ENT examination: palate inspection, palpation
 - 1- Examine soft palate (Speech and Hypernasality)
 - 2- Examine swallowing
 - 3- Examine vocal folds (Voice)



Investigation:

- Audio recording
- Psychometry (IQ)
- Audiometry
- **Fiberoptic Nasopharyngolaryngoscopy is gold standard**
- Articulation test
- Hypernasality sheet
- Nasometry: Hypo or Hypernasality



VPD

Management of VPD:

- Multidisciplinary team
- **Family counseling**
- Management of feeding problem **regurgitation**
- Management of otological and audiological problems
- Surgical intervention **close the gap**
- Orthodontic intervention
- Phoniatric intervention (language, speech, voice)



Nasometry



Fiberoptic Nasopharyngolaryngoscopy

- Teamwork: Feeding, Hearing, Maxillofacial, Palatal and lip surgeries, Obturators.
- Communication (Phoniatic intervention):
 - *Language: Language therapy
 - *Speech: Speech therapy
 - *Voice: Voice therapy
- Family counseling.

Treatment Decision:

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

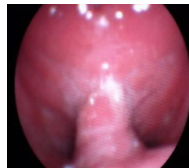
Surgery:

- Pharyngeal flap **most common**
- Sphincter - palatoplasty
- Post-pharyngeal wall augmentation

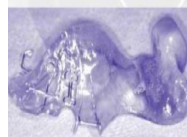
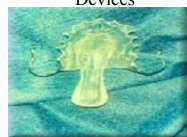
Prosthetic Device: (For Elderly) almost non favorable

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

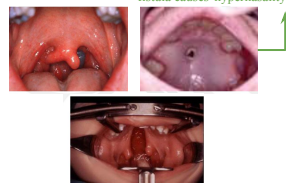
Pharyngeal Flap



Prosthetic Devices



Palatal Fistula after surgical repair from cleft palate, fistula causes hypernasality



Cleft palate

Submucous Cleft:

Difficult to diagnose > triad:

- Bifid Uvula (1st sign)
- Bluish central line in soft palate
- Post nasal notch (instead of spine)
- Contraindicated to adenoidectomy > Hypernasality

Submucous Cleft



5. Cluttering

Definition: Is a fluency disorder characterized by a **rapid** and/or irregular speaking rate, excessive disfluencies. Rx: first by making the person aware of this problem.

Voice Disorders

- 10% of communications disorders
- Prerequisites of “normal” voice production:
 - Normal range of movement of vocal folds.
 - Normal mobility of mucosa on deep layers.
 - Optimal coaptation of vocal folds’ edges.
 - Optimal motor force.
 - Optimal pulmonary support.
 - Optimal timing between vocal fold closure and pulmonary exhalation.
 - Optimal tuning of vocal fold musculature (int. & ext.)
- **Functions of the larynx:** Airway, Protection, Phonation, Increasing intrathoracic pressure.

Usually The Presenting Symptoms In Voice Disorders Are:

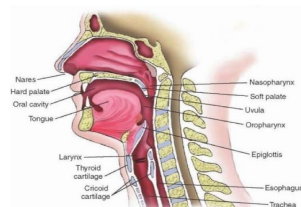
- **Dysphonia:** Any change of the patient's voice from his habitual one
 - * Difficulty in phonation
 - * Change patient’s voice from his/her habitual
 - * Hoarseness: roughness & harshness of voice
 - * Objective term. Dysphonia is broader (includes high pitched)
- **Aphonia:** Loss of the patient’s 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. **singers/ quran reciters**
- **MCQ :** Difference between Dysphonia (Change of habitual sound) and Dysodia (Change of Singing Sound)



True vocal cord movement:

→ During breathing (**Abduction**)

→ During Phonation (**Adduction**)



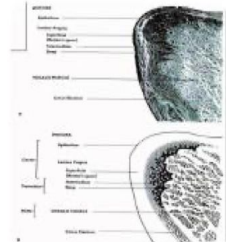
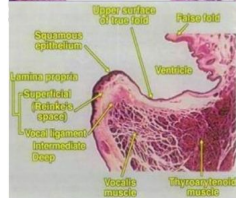
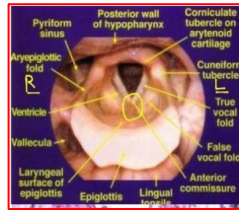
Etiology of dysphonia:

1. **Organic:** there is clear seen pathology.
2. **Non-organic:** (normal Phx): no clear seen pathology, but there is complaint.
 - Habitual
 - Psychogenic
3. **Benign vocal Fold lesion** = Minimal Associated Pathological Lesions (MAPL) Both, Start as non-organic then become organic due to damage. Accompaniment of neuro-psychiatric ailments.
4. Accompaniment of Neuro-psychiatric Ailments

Anatomical Landmarks Of The Larynx:

- IMP understand the picture so you can locate the lesion
- Anteriorly: Anterior commissure and epiglottis
- Posteriorly: Arytenoid cartilage and hypopharynx
- Pictures: As seen in the second picture the false vocal cords are on top of the true vocal folds and not next to them as it might appear in the first picture.
- FF=False folds, V=Ventricle, TF=True folds.
- How to know **Right from Left?** By the Anterior commissure (no posterior commissure), so in the first picture the patient is facing us.
- Cross Section Of The Vocal Cords Which Contains:
 - Mucosa:
 1. Squamous epithelium
 2. Lamina propria which contains:
 - *Superficial layer.
 - *The intermediate and deep layers (vocal ligament)

MCQ/SAQ



1. Organic Voice Disorders

- Congenital (Laryngeal web, Subglottic stenosis, Laryngomalacia)
- Inflammatory
- Traumatic
- Neurological
- Neoplastic
- Hormonal
- Status post-laryngectomy

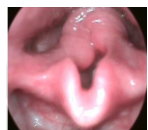
*f2: all pic from doctor slides are covered, there are extra pics "gary frame"

1- Normal

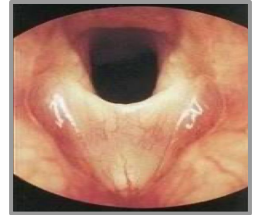
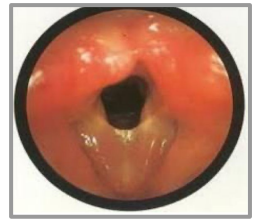


2- laryngomalacia (congenital)

- Epiglottis is folded, almost touching arytenoids and doesn't change shape during breathing. Collapse during inspiration.
- Present complaint: Stridor
- Management: resolves with time in 90% of cases, if severe subglottoplasty



3- Congenital vocal folds web (congenital) Web



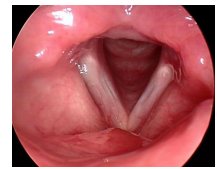
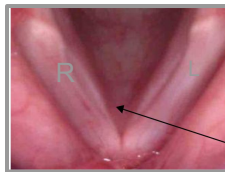
- Can be complete / incomplete (complete is life-threatening)
- Presenting complaint: SOB or dysphonia
- Management: excision of web and add laryngeal tube to avoid formation of new adhesions
- Pt w/ incomplete web may be fine until they get the flu then from the inflammation and edema the airway narrows and they develop SOB

4- Laryngeal cleft. (congenital) Arytenoid cleft



- Has 4 types, most imp is that it could lead to defect between trachea and esophagus
- Presenting complaint: aspiration and swallowing problem, food will go down trachea and breathing
- Management: surgical close.

5- Sulcus vocalis (congenital) bilateral groove



- common in ksa mostly genetic problem
- Congenital lesion
- On the free edge of the true vocal fold
- Presenting complaint: Dysphonia
- Treated by: in mild case: voice therapy, in severe case: vocal folds injection(filler) (close the gap)
- Typical bilateral sulcus vocalis
- Grove on the vocal fold edge
- Can be bilateral or unilateral, including the whole length or small portion of the vocal folds

6- Laryngopharyngeal Reflux



7- Fungal infection (inflammatory)



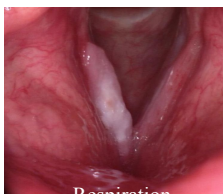
- Candidiasis
- Pt is DM/ Immunocompromised/ steroid treatment
- Management: Medical therapy

8-laryngoscleroma (Inflammatory)



- Chronic inflammation of the larynx
if left untreated it will cause granulation tissue to form and narrow the airway with
time subglottic stenosis
- Etiology: infection with Klebsiella Rhinoscleromatis that targets respiratory
epithelium

9- LARYNGEAL CARCINOMA: (neoplastic)



-Severe dysphonia
 -Whitish lesion occupying the full length of the right true vocal fold. Irregular surface with hyperkeratosis (suspicious). Needs biopsy. Right vocal cord carcinoma.

Squamous cell carcinoma is the most common. Larynge

-Risk factors: Tobacco use, Excessive ethanol use, Infection with human papillomavirus, Increasing age.

10- Cancer (neoplastic)

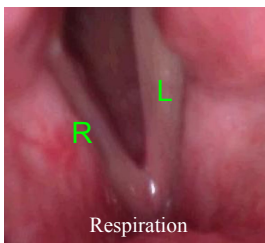


Extended lesions (transglottic carcinoma)



Left Mass

11- Left vocal cord paralysis (neurological)



Left vs right? left true vocal cord immobility (don't describe it as paralysis, because the diagnosis could be something else).
 1-Determine anatomical landmarks (anterior and posterior) to know which side is left and which is right
 2-During respiration vocal cords should be abducted if one of them is not abducted → paralysis. During phonation vocal cords should be adducted if one is not → paralysis.
 You cannot determine which cord is paralyzed if you don't know if the picture is during inspiration or phonation. How it presents? Dysphonia- aspiration (if unilateral) Airway obstruction - swallowing abnormality (if bilateral)
 Management: vocal cord injection, augmentation, medialization thyroplasty.

12- Foreign body



Chicken bone in posterior pharyngeal wall. In foreign bodies mostly are in sites of infection like in sinus and follicular.

2. Non-Organic Voice Disorders

normal anatomy, medical treatment

A. Habitual:

1. **Hyperfunctional childhood dysphonia.** Bad habit. Child is always screaming
2. **Incomplete mutation,** occurs in males, 13-17 years during puberty, change of voice from high frequency to low frequency voice.
3. **Phonasthenia (voice fatigue)** مشجع أو شخص يتكلم كثير They have dryness, tenderness, frequent throat cleaning. It's the only voice disorder that we can't see any abnormality or hear any abnormality, voice is normal but they feel pain with repetitive use (they used to speak for long periods put now they can't).
4. **Hyperfunctional dysphonia, with excessive use.** Also overuse could lead to 5&6
5. **Hypofunctional dysphonia, laryngitis** > pain with speech > they start speaking with low and soft voice b/c it's painful > patient remain in this state of hypofunction even after the disease resolved b/c brain got used to it. E.g. **acute pharyngitis.** Patient won't talk because of pain. If they stopped talking for more than 3 weeks, it will be difficult to get back normal voice.
6. **Ventricular dysphonia, with very high voice and straining,** they even start using the false vocal folds "which is not normal".
 - i. Misuse of voice will cause the ventricles to hypertrophy until they touch each other and dysphonia will occur (patient will sound like WWE fighter).
 - ii. Normal function of ventricle: Helps shape the cords, holds glands which decrease friction between false and true vocal cords, fine tuning.

NOTES:

Misuse vs abuse of voice: Misuse > incorrect use of voice e.g. shouting / Abuse > overuse of voice

Voice frequencies: Children: 200-250 / Males: 100-125 / Females: 200

Male vs Female vocal cords: Male: 20-24 mm / Female: 18-19 mm

1. Hyperfunctional dysphonia

-In professional voice users. **Normal anatomy** > **non-organic.**
-This is a male



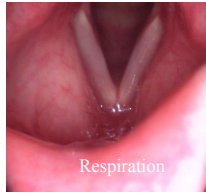
Respiration



Phonation

2. Phonasthenia: most common

- Example: teacher can't talk after 5th period (fatigue) إجهاد صوتي - خطيب المسجد يشتكي من هذا النوع.
- Won't hear or see anything on examination IMP
- If left untreated it can develop: 1.hyperfunctional dysphonia, 2.it can lead to vocal folds nodules or polyps (benign vocal fold lesions).



B. Psychogenic: it has happened before, mostly in female+ middle age+low socioeconomic and education+history of recurrent

1. Psychogenic dysphonia
2. Psychogenic aphonia

3. Benign Vocal Cord Lesion

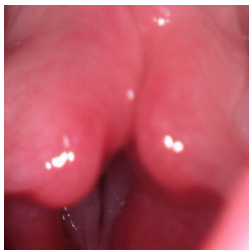
Minimal associated pathological lesions (MAPLs)

- 1.Vocal folds nodules, 2.Vocal folds polyps, 3.Vocal folds cysts, 4.Reinke's edema, 5.Contact granuloma. **All are very Important with their management**

1. Vocal Fold Nodule (bilateral true vocal fold nodule, In females) + voice therapy

Juvenile type

Kissing nodules - More broad base not clear ;small

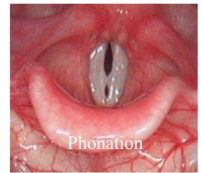


Adult type

Describe: bilateral nearly symmetrical vocal cords lesions at junction of anterior 1/3 and posterior 2/3.

Presenting complaint: dysphonia or hoarseness
Causes: phono-trauma, voice misuse and abuse, shouting.

Treatment: **voice therapy** and vocal hygiene advices. More common in female adult and male children, very rare in adult male



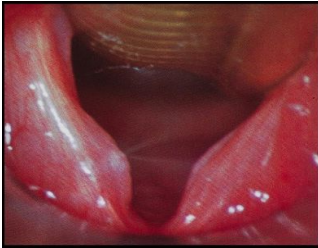
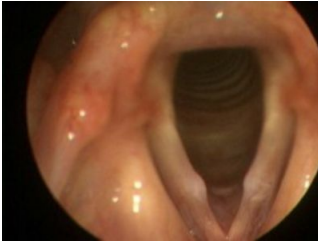
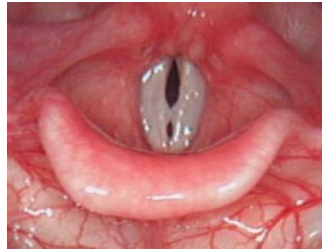
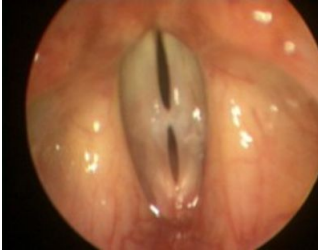
Glottal gap, due to facing of both nodules.



1. Vocal Fold Nodule cont. : EXTRA

SAQ: What is the diagnosis?

Answer: Bilateral true vocal folds nodules



2. Vocal Fold Polyp

- Different shapes, More common in males
- From epithelial layer (mucosa) → well defined

Left vocal fold polyp with a **reaction**

- reaction occurs on the opposite side due to friction during phonation



Left true vocal fold polyp

- Describe: unilateral vocal fold mass or lesion at the left side in the middle third protruding medially.
- Presenting complaint: dysphonia or hoarseness.
- Causes: phono-trauma, voice abuse and sudden shouting.
- Treatment: mainly surgical excision, and voice therapy



Right Vocal Fold Polyp

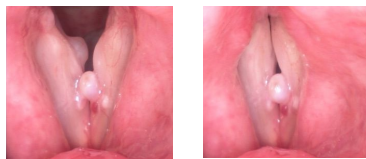
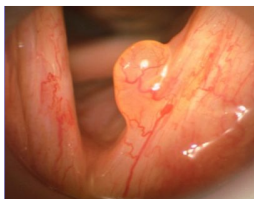
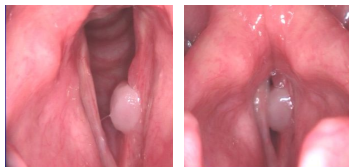
pedunculated
Movable
with
breathing



2. Vocal Fold Polyp cont.: EXTRA

SAQ : What is the diagnosis?

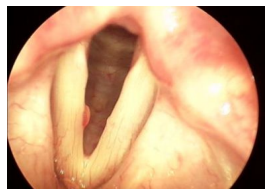
Answer : Left true vocal fold polyp



Multiple polyps, bilateral and Asymmetrical



Right true vocal fold polyp



Right true vocal fold polyp,
we know it's right from the
anterior commissure

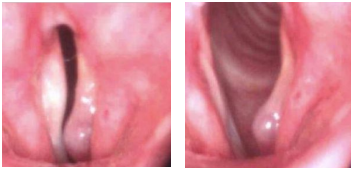
3. Vocal fold cyst

Arises from **deeper** layers causing elevation of the covering mucosa → **ill defined**

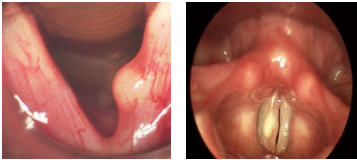
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Left Vocal folds cyst

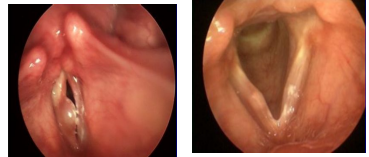
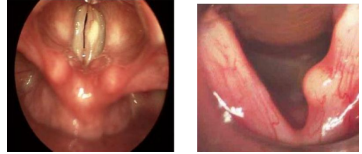


Left True Vocal folds cyst



Left True Vocal folds cyst Intrafolder cyst

- Causes: phono-trauma, congenital, duct closure voice abuse.
- Treatment: primary management Surgery. (Excision)
- We can differentiate between the polyp and the cyst by the outer mucosa, in the polyp is changing, reddish and hemorrhagic. cyst is well defines unlike polyp.



Right True Vocal folds cyst

4. Reinke's edema: Usually bilateral, In superficial lamina propria

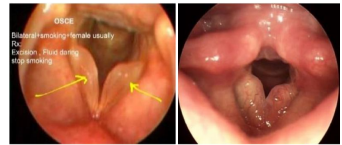


Right-sided Reinke's edema



Bilateral Reinke's edema

- **Bilateral Reinke's edema**

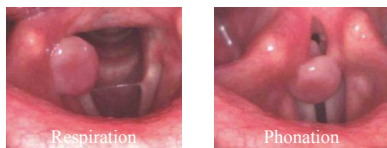


- Causes: smoking, laryngopharyngeal reflux, voice abuse.
- Treatment: stop smoking and surgical removal.
- Common amongst middle aged female smokers but also in male.

5. Contact Granuloma

- No change in voice
- 437A: from the cartilaginous part of the vocal fold “in vocal process not vocal folds”
- *Read this please: **Contact granulomas** are benign lesions usually located on the **posterior** third of the vocal fold, which corresponds to the vocal process of the arytenoid cartilage in larynx. Contact granulomas may occur unilaterally or bilaterally.

Right-sided Intubation Granuloma

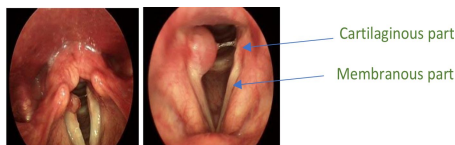


- Intubation granuloma is a different type and it's removed by surgery

no dysphonia, global sensation, problem appears after cardiac surgery for 9h, what is the diagnosis?

Intubation granuloma

Right-sided Contact Granuloma



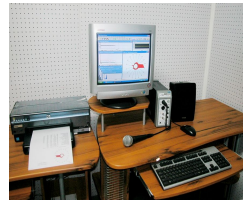
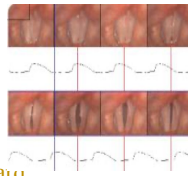
- Unilateral swelling in the cartilaginous part. IMP
- Complain of dryness, pain, phlegm
- Goes by voice therapy
- Two types of granuloma:
 - 1- **intubation** granuloma
 - 2- **contact** granuloma (due to reflex).

- Causes: Laryngopharyngeal reflux, After intubation.
- Presenting complaint: pain or discomfort but rarely dysphonia unless very large.
- **Treatment:** Treat the cause (the reflex), voice therapy, surgery only if failed medical treatment, or increasing in size.
- Why not polyp? It's in the posterior part (Usual place for a granuloma).
- It is very similar to polyps but differ in location, here it involves the posterior cartilaginous part which does not vibrate, therefore no dysphonia.
- **Intubation granuloma is a different type and it's removed by surgery**

Assessment Of Dysphonia:

1. History taking
2. Physical examination: APA, neck...
3. Investigations:

- Audio recording
- **Digital laryngostroboscopy is gold standard**
- Digital laryngokymography
- Digital high speed imaging
- Acoustic analysis (MDVP)
- Aerodynamic analysis (Aerophone II)
- GERD (LPR) work-up
- CT neck
- **Stroboscopy**
- Pharyngeal pH Monitoring
- Phonatory aerodynamic system (PAS)
- **Computerized speech lab (CSL): Acoustic analysis of voice, intensity and frequency (Quantities)**
- Voice sheet
- Special kind of real imaging: strobe and high speed



CSL (MDVP)

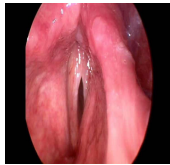
CSL : Computerized Speech Lab
MDVP : Multidimensional voice program



laryngostroboscopy

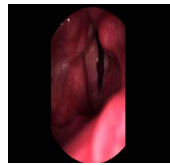


Stroboscopy

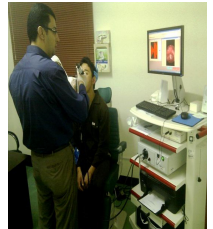


Strobe

Takes sample from each side



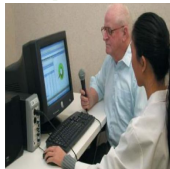
High Speed



High speed laryngeal imaging



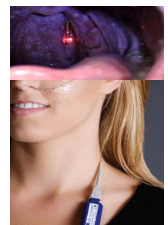
Video laryngostroboscopy



CSL



Phonatory aerodynamic system (PAS)



Pharyngeal PH monitoring

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 up to 1000**
- **Males reach 120-130 cycle/ sec up to 200**

Management Of Voice Disorders:

- Pharmacological agents Ex.GERD
- Surgical procedures (Phonosurgery) polyp,cyst
- Technical aid devices in total laryngectomy, like artificial larynx
- Voice therapy

Treatment of Benign vocal folds lesions in summary: IMP

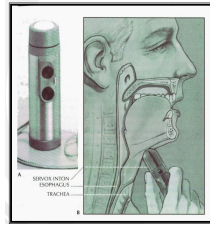
1-Polyps 2- Cysts 3-Reinke's edema > Surgical removal followed by voice therapy

4-Nodules > Voice therapy

5- Contact granuloma > Voice therapy and anti-reflux management

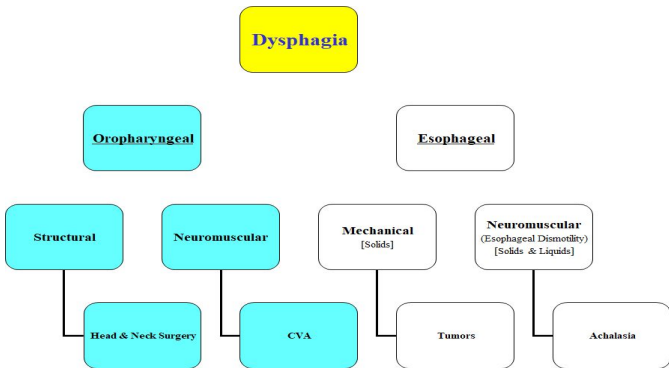


Tracheoesophageal puncture



Swallowing Disorders

CVA: Cerebrovascular Accident, in ENT they are concerned about the oropharyngeal causes.

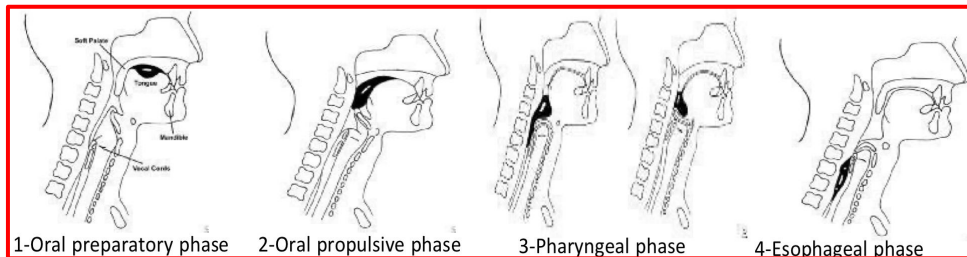


Definitions:

- **Swallowing:** is the successful (timely and efficiently) passage of food and drinks from the mouth to the stomach. Happens 2,000-3,000 times/day.
- **Dysphagia:** difficulty in moving food from the mouth to the stomach (pain, discomfort and/or difficulty in initiation or completing the act of swallowing)
- **Odynophagia:** painful swallowing due to a disorder of the esophagus.

Phases Of normal Swallowing:

1. Oral (**voluntary**)
2. Pharyngeal
3. Esophageal

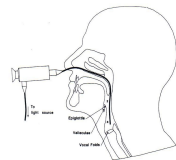


Consequences Of Dysphagia:

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

Assessment of dysphagia:

1. History taking
2. Physical examination:
 - General examination.
 - Language and speech assessment.
 - Vocal tract examination.
 - Neck examination.
 - Trail feeding (bedside assessment) **give them food and try swallow**
 - Dysphagia sheet.

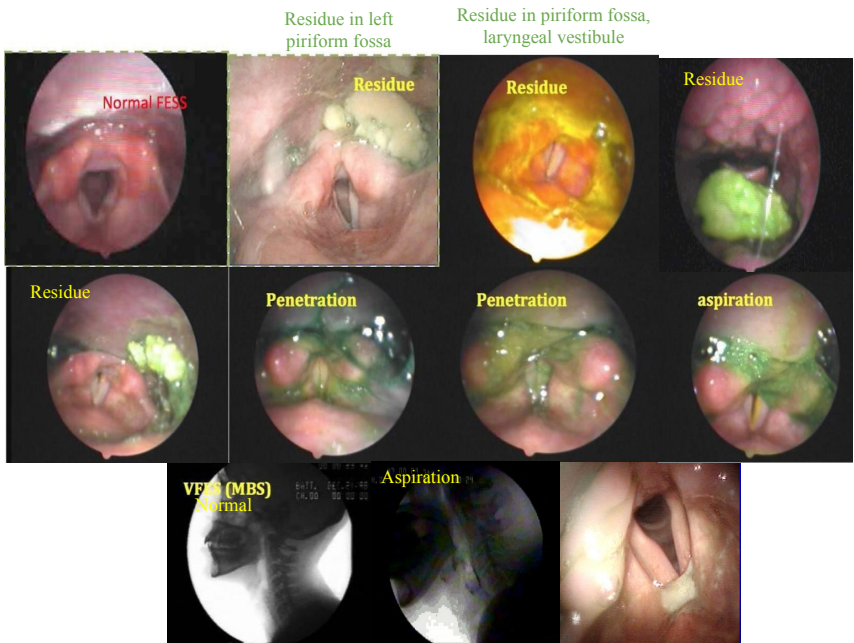


3. Investigations:

- **FEES “Fiberoptic endoscopic evaluation of swallowing”**
 - **VFES (MBS) “Video fluoroscopic evaluation of swallowing”(Modified barium swallow)** It shows the path way from lips to upper esophageal sphincter (oral and pharyngeal steps)
 - GERD (LPR) workup
- FEES protocol of evaluation (Langmore, 2003):
- a. Anatomic and physiologic assessment.
 - b. Assessment of food and liquid swallowing.
 - c. Assessment of therapeutic interventions.



FEES



- Residue = in the pharynx.
- Penetration= at the true vocal folds.
- Aspiration= below vocal folds.
- Normally when drinks or food enters the airway there is cough reflex, but if for e.g. an old age patient with CVA or stroke and the fluids enter the airway and no cough reflex, this is called **Silent Aspiration** which is very dangerous.
- If aspiration occurs, **penetration** must have occurred first (except in tracheoesophageal fistula where aspiration occurs without penetration).
- Aspiration of liquid is **more common** but less severe than aspiration of solids.

Penetration on anterior commissure

Management of dysphagia:

- Oral vs non oral feeding
 - Non oral feeding when
 - Aspiration >10%
 - Oral + pharyngeal transit time >10 s
- Direct vs Indirect therapy
 - Direct: food or liquid given to the patient
 - Indirect: no food or liquid given (only saliva)
- Compensatory vs Therapy technique
 - Compensatory: elimination of symptoms but no change in swallowing physiology, such as postural techniques
 - Therapy techniques: change of swallowing physiology such as swallowing maneuvers.

Management of dysphagia cont. :

1. Swallowing therapy:
 - a. Diet modification. (if the problem is with solids only or fluids only)
 - b. Postural techniques.
 - c. Swallowing maneuvers.
 - d. Sensory enhancement techniques.
 - e. Motor exercises.
 - f. Bolus control cup
2. Surgical treatment, e.g. medialization laryngoplasty. Like in vocal fold paralysis, cricopharyngeal muscle spasm
3. Medical (Drug) treatment, e.g. anti-parkinsonism drugs.
4. Intraoral prosthesis. hypernasality VDD, VDI insufficiency / nasal regurgite
5. Alternative routes of feeding, e.g. NG tube feeding. Temporary (not more than 6 weeks) > 6 weeks → gastrostomy.

*From doctor
slides

KAUH-Strobe Examination Report

Name: Al-Bulaihi, Haila, M Patient ID: 00465849 RKH
Exam Original Date: 3/13/2004 9:46:18 AM

Selected Stills (Image Compression - 15:1)

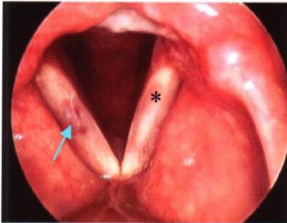


Figure (1) - Fully abducted position



Figure (2) - Fully adducted position

Thank you for referring this patient.

Telescopic videolaryngostroboscopy done, and showed:

I. Continuous light examination:

- ❖ Left vocal fold paralysis (asterisk).
- ❖ Paralytic phonatory glottal gap of about 2-3 mm at maximum width posteriorly (Figure 2).
- ❖ A patch of submucosal hematoma at the middle third of membranous part of the right vocal fold (arrow).
- ❖ Mild ventricular hypertrophy.

II. Stroboscopic light examination:

- ◆ Decreased amplitude and mucosal waves on the left vocal fold.
- ◆ Asymmetry in amplitude and mucosal waves between both vocal folds.
- ◆ Aperiodicity in amplitude and glottal cycle time at the left vocal fold.
- ◆ Phase is predominantly open.

Diagnosis:

Left vocal fold paralysis with glottal gap of about 2-3 mm at maximum width posteriorly.