

#### VitalStim Therapy

Practicing Clinical Reasoning: Everything you need to know about the UES

#### Slide 2

#### **UES**: Upper Esophogeal **Sphinctor**



- A 2.5 to 4.5 cm manometric highpressure zone located between the pharynx and esophagus
- Also known as the Pharngo-esophogeal Segment (PES)
- The Cricopharyngeous Muscle (CPM) is only one component of the UES

#### Slide 3

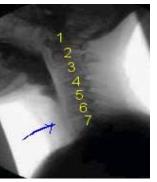
# Components of the UES erior Pharyngeal



#### Location of the UES



Located at C6-C7



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#### **Sphinctor**



- A ring of muscle fibers located around an opening in the body that <u>regulates</u> the passage of substances
- A circular muscle <u>constricting an opening</u>. Sphincter muscles control bowel and bladder evacuation
- A ring-shaped muscle that <u>relaxes or</u> <u>tightens</u> to <u>open or close</u> a passage or opening in the body

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#### CPM



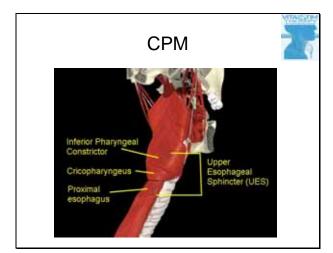
- C-shaped muscle that attaches to the lateral lamina of the cricoid cartilage
- The only component of the UES that contracts and relaxes during reflexive tasks
- Comprised of both Type I and Type II muscle fibers (90% Type I-tonic)
- Maintains a constant tone to allow it to contract and expand when necessary

#### CPM

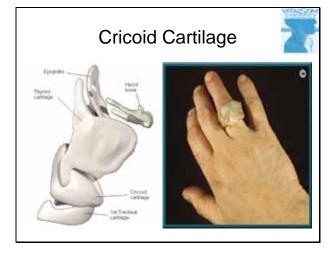
- The state of the s
- \*maintain a tonic contraction to prevent aspiration of refluxed material \*tonically contracted at rest \* Passive Compon \*open to deglutiti burping, \*occurs traction
  - Passive
    Component

    \*open to allow for deglutition, burping, vomiting
    \*occurs by way of a traction force

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#### Innervation of the CPM



- CPM receives DUAL innervation from the pharyngeal plexus and the recurrent laryngeal nerve
  - CN IX and CN X
- This innervation allows for the tonic contraction
- The inhibition of this innervation allows for its relaxation

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# CN Innervation for the UES Whenever start on a Y Wh

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#### The function of the UES



- Again....
  - #1- prevent reflux of GI material
  - #2- open to allowpassage of food, liquids: 3 phases burping vomiting

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#### UES seal - 1



- Relaxation UES
  - Cessation of neural activity to cricopharyngeus
- <u>Prepares</u> for esophageal cha opening





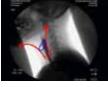
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#### UES seal - 2

- Traction force to open
- Laryngeal extrinsics
- Pharyngeal shortening muscles
- Opens esophageal chamber





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#### UES seal - 3



- Closure UES
  - Resumption neural activity to cricopharyngeus
- <u>Closes</u> esophageal chamber

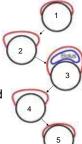


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#### Phases of UES opening



- 1. Inhibition of tonic contraction
- 2. Hyolaryngeal excursion causes passive opening
- 3. Distention of the UES through bolus size and weight
- 4. Passive collapse of UES as food passes through
- 5. Closure UES through active contraction



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#### **UES** opening



- · Muscles involved:
  - Suprahyoids and Infrahyoid cocontract
  - Pharyngeal shortening muscles contract
- Norm: Barium column passes through UES in one single swallow, no indentations of column during moment of maximum opening of the UES



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#### Opening....



- Openers
  - -Supra & Infra hyoids co- contracting to pull the hyoid up and forward
  - -"the degree of preservation of UES opening was significantly associated with the degree of preservation of geniohyoid shortening." (2)
  - -Pharyngeal constrictors shortening an distending the pharyngeal space
  - -"Swallowing in individuals who can relax their CP but cannot elevate their larynx has not been observed." (1)
- Influencers
  -size & weight of the bolus
- -tongue base retraction:
- "in the absence of a pharyngeal swallow, "UES opening was possible if sufficient intrapharyngeal pressure was generated in response to compensatory tongue base motion." (2)

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#### Dyfunction with the UES



• Due to:

Lack of function in the traction force to pull open the UES

OF

Lack of compliance at the level of the UES

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## Disorders/Disease – UES dysfucntion



- Oropharyngeal carcinoma, Dermatomyositis, Diabetes
- Esophageal carcinoma, Inclusion body myositis, Diphtheria
- Benign esophageal tumor, Hyperthyroidism, Rabies
- Zenker's diverticulum, Idiopathic, Lead poisoning
- Laryngopharyngeal Reflux, Radiation therapy, Polymyositis,
- · Pharyngitis, Brainstem tumor, Scleroderma
- · Post-surgical change, ALS, Muscular dystrophies
- Foreign body, Huntington's chorea, Myxedema
- CVA, Poliomyelitis, Botulism
- Parkinson's, Spinocerebellar degeneration, Trauma (iatrogenic)
- Inflammatory myopathies, Syringobulbia (1)

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#### Manifestations of Dysfunction-1



- . CP Bar= hypertrophy, prominence in the CPM
- -can result from any lesion that interferes with esophageal neuromuscular activity.
- -can be a normal variant in 10-15% of the population
- -can be a compensatory response induced by chronic gastroesophageal reflux.
- Neuromuscular causes include central and peripheral nervous system abnormalities (multiple sclerosis, ALS, syringomyelia, cerebral vascular disease) inflammatory myopathies, and myoneural junction disorders (myasthenia gravis, diphtheria, tetanus).
- Can exacerbate symptoms of dysphagia and increase propensity of development of a Zenker's diverticulum (3).

#### **CP** Bar





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#### **CP** Bar



 "The presence of a bar does NOT imply the existence of dysphagia.

An individual with a CP bar and normal PES opening should be evaluated for an alternative cause for his/her swallowing complaints." (1)

 So- if there are dysphagia complaints, identified dysfunction, and the presence of a bar, the SLP should note the presence and consult the ENT or GI

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#### Achalasia vs. CP Bar



- Achalasia
   -LES issue
- <u>CP Bar</u> -UES issue

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#### Achalasia

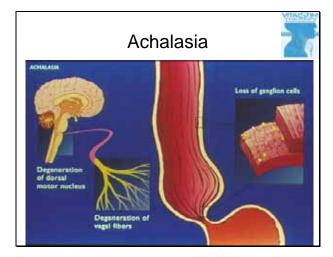


- Achalasia = caused by failure of the lower esophageal sphincter to relax.
- Symptoms are caused by food becoming trapped in the esophagus and unable to pass into the stomach. With time, the esophagus can become enlarged with marked twists and bends to accommodate the retained food.
- Patients can present with dysphagia, regurgitation of undigested food, and even chronic aspiration.

  Radiographically the column of barium will remain in the esophagus and come to a point resembling a 'bird's beak" at the lower esophageal sphincter.

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#### Identifying the Dysfunction



- True CP or PES dysfunction is at the level of the CP
- · Remember: the UES will not open on its own.... Traction force
- Dysfunction leads to symptoms such as pyriform sinus residue, and pharyngeal residue

But- we must ID the cause of these symptoms

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### Protocol for Diagnosing dysfunction



- When the symptoms of pryriform sinus residuals and poor clearance of the bolus through the UES are noted:
- If there is noted weakness/inadequate pharyngeal constriction and weakness/inadequate hyolaryngeal excursion-TREATEMENT (of those issues-the openers)
- If there is noted adequate strength/function in pharyngeal constriction and hyolaryngeal excursion- CONSULT ENT or GI (claim it; don't name it)

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#### SO....



- The UES is a true sphincter; it will behave as one
- The UES will NOT open on its own- muscles pull it open
- If there are symptoms of UES dysfunction, look at the openers
- Know your role on the medical team of experts -consult with Radiology, GI, ENT
  - -SLPs do not diagnose esophogeal dysfunction; we can observe it and report it
- Know and Use the protocol for diagnosing dysfunction in the UES

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#### References



- 1. Belafsky, P. The Pharyngo-Esophageal Segment (PES). <u>www.vitalstimtherapy.com</u>. 2006
- 2. Wiliams, R, Wallace, K. L., Ali, G. N., Cook, I, J. Biomechanics of failed deglutitive upper esophageal sphincter relaxation in neurogenic dysphagia. AJP-Gastrointest Liver Physiol. 2002 July; vol 283.
- 3. http://gi.vghtc.gov.tw/Teaching/Eso/EsoAGA/20. htm



#### **Questions & Answers**

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#### Thank you!

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