activity of the denervated esophagus. Recently, it has been reported that an increase in esophageal muscle thickness using a high-frequency intraluminal ultrasound (HFIUS) is an important feature of patients with primary esophageal motility disorder such as achalasia. We aimed to compare the clinical features, the radiological and manometric findings, and the esophageal mean muscle thickness and cross sectional area (CSA) between pre and post pneumatic dilation states in patients with achalasia. Methods: Fifteen patients (8 men and 7 women, mean age 39 ± 15 years) with achalasia treated by pneumatic dilation were enrolled in this study. The assessment of the clinical features, the maximal diameter of esophageal body on barium esophagogram, the LES resting pressure on manometry, and the esophageal mean muscle thickness and CSA at 3 cm above LES on HFIUS was performed before pneumatic dilation for baseline assessment. And these were repeated 6 months later after pneumatic dilation. Results: In post pneumatic dilation state, enrolled patients exhibited distinctly different features from pre pneumatic dilation state; an improvement of dysphagia symptom, a recovery of weight, a reduction of maximal diameter of the esophageal body, and a decrease of LES resting pressure. These differences between the two states were statistically significant. Esophageal muscle thickness and CSA were observed to be significantly lesser in post pneumatic dilation state than in pre pneumatic dilation state at 3 cm above LES (mean muscle thickness (mm); pre vs. post = 2.04 ± 1.02 vs. 1.67 ± 0.54, CSA (mm2); pre vs. post = 138 ± 44 vs. 90 ± 45, $P < 0.05$, respectively). Conclusions: Esophageal muscle thickness and CSA was recoveried after pneumatic dilation as was the clinical, radiological, and manometric findings in patients with achalasia in this study. This result may be aided to understand the functional motor disturbance of achalasia.

W1820

Acid Induced Symptom Generation (Heartburn) May Not Be Mediated By Esophageal Muscle Contraction

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(Background and Aim)Heartburn and regurgitation is considered as typical symptom of acid reflux, yet the symptom resolution is not always achieved by acid inhibition treatment, suggesting acid itself does not cause the symptoms directly. However the mechanisms of the symptom generation remain unknown. Accordingly we observed esophageal muscle contraction in esophageal acid perfusion test, by which we can monitor the physiological changes of the esophagus during heartburn symptom generation. (Subjects and Methods) Eight healthy subjects (6 males, mean age 26+3.5yrs, ranged 23 to 34) underwent acid perfusion test. Briefly, a double rumen gastric tube was inserted via nasal route and placed at the middle portion of the esophagus (15cm above LES) where the acid (0.1M) was applied in the rate of 8ml per min for 12 minutes (after two minutes application of saline). The ultrasonographic transducer was put and placed from the other rumen at 5cm above LES, by which the thickness of the esophageal muscle was monitored every 5 seconds and video recorded. The esophageal muscle thickness was measured on the image analyzing system. The intensity of the heartburn was evaluated real-timely using visual analogue scale. The mean baseline thickness (that of first two minutes during saline perfusion) and that of 10 to 12 minutes (last two minutes) was measured both in subjects with none or mild and moderate or severe heartburn. (Results) Of eight subjects, four had moderate to severe heartburn while the rest of four subjects had no or mild symptoms. The relative esophageal muscle thickness at last two minutes compared to the initial two minutes was1.14+0.25 and 0.98+0.0 (mean+SD) in the subjects with and without heartburn, respectively (p=0.231). Contraction amplitude as well as contraction duration was not significantly altered during acid perfusion both in the subjects with and without symptoms. (Conclusion) Our results indicate that acid induced symptom generation (heartburn) may not be mediated by esophageal muscle contraction.

W1821

The Virtual Esophagus: An Interactive Model of Esophageal Physiology, Pathophysiology, and Simulation of Esophageal Motility Disorders

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Background: A computer model simulating esophageal physiology and demonstrating the symptoms and test results of motility disorders would enhance trainee understanding of esophageal diseases and allow rehearsal of medical decision-making prior to patient care. **Aim:** To develop an interactive virtual esophagus that demonstrates automatic behavior reflective of underlying normal and diseased physiological functions and models motility and clinical management strategies. **Methods:** Knowledge engineers and subject matter experts collaborated to elicit knowledge rich in normal esophageal physiology, pathophysiology and clinical management. This included modeling normal esophageal concepts: anatomy, physiology and neuromuscular function, and abnormal concepts: acid reflux, inflammation, LES dysfunction, esophageal dysmotility and associated symptoms. The knowledge was encoded into machine tractable language using existing techniques developed by the collaborators. Computational methods were developed to reason with the encoded knowledge to achieve a virtual esophagus that functions in a realistic manner. **Results:** The knowledge and reasoning capacity successfully created an esophagus with automatic behavior capable of generating multiple qualities: (1) Intrinsic normal organ function such as basal lower esophageal sphincter pressure and lower esophageal sphincter relaxation and peristalsis in response to swallowing, (2) Symptoms of esophageal diseases such as heartburn and dysphagia, (3) Associated diagnostic test results on barium swallow, endoscopy, manometry, and 24 hr pH monitoring such as bird's beak, stricture, aperistalsis, and DeMeester score, (4) Natural disease progression such as progressive dysphagia as the esophageal lumen narrows, (5) Disease response to various treatments such as resolution of heartburn with a proton pump inhibitor or improvement of dysphagia with dilation of a stricture. The accumulation of these qualities provides a functioning virtual esophagus capable of demonstrating the normal esophagus, achalasia, gastroesophageal reflux disease (GERD), GERD with erosive esophagitis, Barrett's metaplasia, or adenocarcinoma. **Conclusions:** We have designed the backbone of an interactive virtual esophagus that models esophageal physiology, pathophysiology, and clinical management for common esophageal diseases. Use of the virtual esophagus may provide a novel and optimal teaching opportunity to allow a trainee to query the esophagus, diagnose esophageal diseases, perform treatments, and observe the results of treatments. This model will be further refined with addition of other esophageal pathology and expanded to include other organs.

W1822

Vigorous Achalasia and Classic Achalasia: Two Similar But Separate Entities?

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BACKGROUND: Achalasia is primarily defined manometrically by an aperistaltic esophagus. Traditionally, hypertensive LES, and incomplete lower esophageal sphincter (LES) relaxation are considered part of the manometric definition. Vigorous achalasia is identified in a subset of patients having distal esophageal amplitude (DEA) greater than 37 mmHg, while still having total absence of peristalsis. Combined multichannel intraluminal impedance and manometry (MII-EM) allows both a functional and a manometric evaluation of esophageal motility and also identifies chronic fluid retention. AIM: To compare manometric and MII characteristics in patients with achalasia and vigorous achalasia. METHODS: Review of 73 MII-EM tracings (38 females; mean age 53.5; range 14 - 95 years) from patients with achalasia and 73 patients with normal motility (49 females; mean age 54; 22-82 years). Manometric and MII characteristics were compared during 10 liquid and 10 viscous swallows. Patients were divided into 3 groups: classic achalasia, vigorous achalasia, and normal. RESULTS: There were 59 (81%) patients with classic achalasia and 14 (19%) patients with vigorous achalasia. EM and MII details are shown in Tables 1 and 2 respectively. In 73 patients with normal motility, the mean baseline MII was 1955 + 229 ohms, 1985 + 232 ohms after 10 liquid swallows and 2102 + 246 ohms after 10 viscous swallows. There was also a significant difference ($p<0.05$) between the baseline MII in patients with both classic and vigorous achalasia and normals. CONCLUSION: Patients with vigorous achalasia are likely to have a hypertensive LES, higher number of swallows with complete bolus transit for liquid and viscous swallows, and higher impedance suggesting less fluid retention than those with classic achalasia. Classic and vigorous achalasia are similar but have distinct features. Combined MII-EM helps confirm the diagnosis.

Table1: Manometric data

	Distal esophageal amplitude	LES pressure	LES residual pressure
Classic	22	36±4.3	10.4±1.4
Vigorous	79	47±5.6	11.4±3.1
P	< 0.05	< 0.05	NS

Table 2: Impedance data

	CBT (median) (liquid)	CBT (median) (viscous)	Baseline MII (ohms)	Liquid MII (ohms)	Viscous MII (ohms)
Classic	0 (0-4)	0 (0-3)	689	389	343
Vigorous	1.5 (0-6)	0 (0-6)	1265	812	650
p	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05

CBT = complete bolus transit; MII measured at baseline, post 10 liquid and viscous swallows.

W1823

Novel MII-pH Methods in the Evaluation of Physico-Chemical and Spatiotemporal Characteristics and Peristaltic Clearance Mechanisms with Reflux Events (RE) in Infants with Chronic Lung Disease of Infancy

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BACKGROUND The role of physico-chemical properties of GER in prematurely born infants with CLDI (oxygen need at >36 wk corrected gestation age, GA) is not well understood. OBJECTIVES To characterize the association between the physical (gas, mixed, liquid), chemical (acid, minor-acid, non-acid) and spatiotemporal (height of refluxate and duration) characteristics of RE with aerodigestive symptoms and clearance mechanisms (primary peristalsis, PP and secondary peristalsis, SP). METHODS Six neonates (27±1 wk GA) with CLDI associated with dysphagia were studied. First, esophageal manometry was performed to determine the segmental lengths of the aerodigestive tract. Next, a 24-hr concurrent MII-pH recording was performed following manometry. Clinical symptoms were documented in real time. The physical and chemical nature and the extent of the refluxate were determined for each RE. Symptoms were considered associated if they occurred during, before and after 1-min of RE. Clearance mechanism (PP, SP, and none) with each RE was recorded based on the proximal extent of the refluxate and aboral propagation. Proportions were compared between the RE characteristics, and symptom sensitivity index (SSI = # of RE with symptoms/ total RE * 100). RESULTS A total of 318 RE (gas 36%, mixed 19%, liquid 45%) were analyzed. Of these, 23 % were acid pH < 4.0, 10% minor acid as pH-decrease by 1 unit, and 67% non-acid events. Proximal extent of all RE was up to pharynx (PX, 76%), proximal esophagus (PE, 17%), mid esophagus (ME, 7%), and distal esophagus (DE, 1%). The bolus clearance time for mixed events was 17± 18 sec, liquids 19 ± 29 sec (P=NS). Response time for peristaltic reflex for gas RE was 3.6 ± 5.6 sec, mixed RE 32± 18 sec, and liquid 16 ± 13 sec (ANOVA P<0.001). 134 symptoms were noted with 318 RE; of which 20% with gas, 28% with mixed, 52% with liquid. CONCLUSIONS The symptoms associated with RE and the clearance mechanisms are dependent on the physical properties and proximal extent of the refluxate. The gas and mixed RE constitute the majority of the RE with symptoms. Response times for clearance reflex responses were dependent on the physical property of refluxate. Occurrence of PP (esophago-deglutition response) with refluxate stimulating DE, ME, and PE are noted.

Peristaltic clearance reflexes and SSI based on physical and spatiotemporal parameters