Conclusion: PGE2 was used to detect significantly abnormal tissue architecture in BE patients with dysplasia and neoplasia. Significant increases in the depolarization ratio were detected both in the squamous mucosa of the distal esophagus and within the BE segment. These findings support further studies for remote detection of BE dysplasia and neoplasia based on the field carcinogenesis concept.

Figure 1. Significant differences were found in the depolarization ratio (d) obtained from within BE segments in patients with associated dysplasia and neoplasia.

Distal Baseline Impedance (DBI): A Specific Predictor of Major Motility Abnormality
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Purpose: The purpose was to test the hypothesis that patients with major motility abnormalities exhibit significantly lower DBI values on esophageal impedance manometry when compared to other patients with dysphagia and to establish a DBI value that yields the highest sensitivity/speciﬁcity for a diagnosis of MMA. Other comparisons included MMA versus diffuse esophageal spasm, and 81 with mixed features of two or more MMs. An ROC curve comparing MMA patients with all other diagnoses showed that at a DBI value of ≤634Ω there was a sensitivity of 45.56% and a speciﬁcity of 95.24% for the diagnosis of MMA.

Conclusion: The results of this study further reinforced the current belief that distal baseline impedance is often significantly lower in patients with major esophageal motility abnormalities. Moreover, we found that a DBI value of ≤634Ω yielded 95.24% speciﬁcity for the diagnosis of MMA. Therefore, DBI appears to be a valuable diagnostic aid in the diagnosis of a major motility abnormality when esophageal manometry ﬁndings are indeterminate.

The Potential Pathogenetic Role of Esophageal Prostaglandins in Patients with Barrett’s Esophagus: Its Clinical Implication
Presidential Poster
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Purpose: The majority of patients with chronic symptoms of gastroesophageal reﬂux disease (GERD) are likely to never develop endoscopic esophagitis. This patient’s population is known under acronym NERD, i.e. negative endoscopically reﬂux disease. On the other side of the wide GERD spectrum, there is a small subpopulation evolving into Barrett’s esophagus (BE) dysplasia and adenocarcinoma. Prostaglandin E2 generated through the cyclooxygenase 1 (COX-1) pathway is protective towards the columnar epithelium, however, its very high levels elaborated by COX-2 seem to promote the malignant potential within the intestinal metaplasia (Lewis et al. Surg Endosc, 2011). Little is known, however, regarding the rate of luminal release of PGE2 in patients with BE as compared to subjects with NERD. The following specific aims were designated to our study protocol: 1.) To measure the rate of PGE2 generation in esophageal secretion in patients with BE, and 2.) to compare obtained results with corresponding values recorded in NERD patients.

Methods: The study, approved by IIRB, was conducted in 10 patients with the long segment (>3cm) of BE (2 female [F] and 8 male [M], mean age of 49 years, 30-69 range), and in 10 patients with a long history of NERD (4F and 6M, mean age of 40, 27-64 range). The esophageal secretions from the mucosa and submucosal mucous glands were collected during mucosal exposure to initial NaCl followed by HCl/Peptic (H2/C) and final saline, mimicking the natural gastroesophageal reﬂux scenario, using a specially designed esophageal perfusion catheter (Wilson Cook Med. NC). In collected samples PGE2 was measured using RIA (Amersham, MA). Statistical analysis was performed using Σ-Stat (SPSS, IL).

Results: The basal rate of esophageal PGE2 secretion in patients with BE during exposure to saline was (Mean, ±SEM) 7.2-fold higher than in patients with NERD (9624 ±1416 vs. 1333 ±141 pg/min, P<0.01). Furthermore, the rate of esophageal PGE2 secretion in BE patients remained also 5.8-fold higher than in NERD patients during exposure to HCl/P (4030 ±1748 vs. 695 ±203 pg/min, P<0.05). Esophageal PGE2 secretion in BE patients remained also 10.4-fold higher than in NERD patients during the ending mucosal perfusion with saline (5716 ±2686 vs. 351±618 pg/min, P<0.05).

Conclusion: 1.) Profoundly lower rate of esophageal PGE2 generation in patients with NERD confirms that equilibrium between aggressive factors and protective mechanisms within the esophageal squamous mucosa is well preserved. 2.) Several fold higher rate of esophageal PGE2 secretion in BE may imply that the development of the columnar epithelium of BE could potentially be driven by excessive generation of prostaglandins in mucosal inflammatory changes, potentially setting the stage for further complications.

Correlation between Preprandial Reflux and Total Reflux Episodes in Prolonged Impedance Studies
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Purpose: The purpose of this study was to determine if preprandial reflux is correlated to total reflux during a prolonged multichannel intraluminal impedance study.

Methods: We analyzed 158 24-hour impedance and pH studies from a prospectively maintained database of patients with symptomatic reflux and compared the preprandial reflux, defined as any reflux episodes before the first meal, to the total number of reflux episodes in the study. All reflux episodes were calculated as a frequency per minute to account for differences in time before first meal and total study length. Symptom correlation as well as medical therapy were also noted. Patients with previous esophageal surgery as well as patients with low distal baseline impedance were excluded.

Results: Of the 158 patients, there was a correlation coefficient of 0.57 between preprandial reflux frequency and total reflux frequency. The median number of preprandial reflux episodes per minute was 0.39, with a mean of 0.74 and a standard deviation of 0.98. Six data values outside of two standard deviations of the mean were excluded. Median number of total reflux episodes per minute was 0.031 with a mean of 0.053 and a standard deviation of 0.025.

Conclusion: There is a moderate correlation between preprandial reflux and total reflux frequency. Implications for this data could include decreasing prolonged multichannel intraluminal impedance study lengths, which would decrease patient discomfort and may increase testing availability to a larger patient population.
Prevalence and Association of Helicobacter pylori in Downtown Brooklyn’s Minority Population
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Purpose: Helicobacter pylori (H. pylori) infection is a pandemic disease; about 70% of the developing world population and 40% of the U.S. population harbor this bacteria as per the CDC. H. pylori has been implicated in the development of peptic ulcer disease and low frequency gastric malignancy, namely adenocarcinoma and lymphoma of the gastric mucosa-associated lymphoid tissue. Due to the serious consequences of this common infection and the lack of studies addressing H. pylori prevalence among minority patients, we aimed to investigate the prevalence of this infection in a downtown Brooklyn population. One of the most diverse urban populations in the country.

Methods: Charts of all adult patients who underwent upper endoscopy with biopsy at our medical center in two years period were reviewed. Data about demographics, endoscopic, and histological findings were collected and analyzed. The presence of H. pylori infection was based on the immunohistochemical analysis of the biopsy samples. SAS software was used for statistical analysis.

Results: Our cohort included 970 patients (37% males and 63% females). African Americans and Hispanics represented 52.5% and 28.3% of the study population, respectively. The prevalence of H. pylori was 24.6%. There was no association found between H. pylori prevalence and age, race, or sex (P = 0.16, P = 0.92, and P = 0.87, respectively). Peptic ulcer disease was found in 11.5% of our cohort which was significantly associated with H. pylori (16.3% vs 9.9% in non-infected patients, P = 0.007). Gastric intestinal metaplasia (in 11.6% of the patients) was also associated with H. pylori infection (18.4% vs 9.4% in non-infected patients, P = 0.0002).

Conclusion: H. pylori prevalence among the downtown Brooklyn population is lower than the national average reported by CDC and even lower than any North American population reported in the literature. The environmental, bacteriological and host factors behind these findings need to be evaluated.

Pancratic Heterotopia in the Setting of Eosinophilic Esophagitis: A Case Report and Review of the Literature
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Purpose: A 37-year-old Caucasian male with a 5-year history of dysphagia to solid foods and intermit-tent postprandial vomiting was investigated with an upper GI endoscopy. Visualization revealed circular rings, linear furrows and Los Angeles grade A esophagitis along the length of the esophagus. Additionally, inflamed gastric cardia along with flat subtle polyloid lesion noted at gastric cardia. Histologic specimens harvested from the proximal and distal esophagus demonstrated up to 43 eosinophils per high-powered field consistent with eosinophilic esophagitis (EoE). Additional biopsies from the cardia revealed chronic carditis along with pancreatic heterotopia. A subsequent skin allergen testing revealed multiple food allergies that were determined to be contributing to his esophagitis and consequent dysphagia. Patient was started on budesonide and also undergoing sublingual immunotherapy (SLIT) to decrease his immune response to known allergens. Eosinophilic esophagitis is a chronic immune-antigen mediated disease process believed to be caused by a variety of both environmental and genetic factors. Patients often present with dysphagia in the third-fourth decade of life and are commonly found to have multiple food allergies. Management often includes immunotherapy to decrease responsiveness to known allergens, dietary modifications as well as medi-cal therapy with swallowed topical steroids. Interestingly in our patient pancreatic heterotopia, when aberrant pancreatic tissue is found throughout the body and is usually asymptomatic, was also noted at gastric cardia and the combination of the two is exceedingly rare and the existence of the two in tandem is infrequently reported in literature, making our case a unique presentation of two relatively uncommon findings together.

Are There any Histologic Changes Suggestive of Acid Exposure in the Esophageal Mucosa Surrounding Inlet Patches?
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Purpose: The frequency and clinical significance of heterotopic gastric mucosa in the upper esophagus, also called inlet patch, is not known. It is thought to result in acid production causing GERD. Many histologic changes have been described in the esophageal squamous mucosa in patients with GERD such as ballon cells, basal cell hyperplasia, and intraepithelial eosinophils. The aim of this study was to assess if there were any histologic changes in esophageal squamous mucosa surrounding inlet patches as well as in the upper gastrointestinal tract.

Methods: Patients from 2/2012 to 5/2013 were identified who by endoscopic appearance had an inlet patch. Biopsies were obtained from the inlet patch as well as from esophageal mucosa surrounding the inlet patch. The mucosa surrounding the inlet patch was evaluated for the presence or absence of eight previously reported reflux-associated histologic changes including dilated intercellular spaces, balloon cells, intrapapillary vessel dilation, elongated papillary, basal cell hyperplasia, squamous metaplasia, intraepithelial eosinophils, and Langerhans cells. Use of proton pump inhibitors, presence of hiatral hernia, reflux esophagitis, Barrett’s esophagus, Helicobacter pylori were also noted.

Results: Thirteen patients were identified. Baseline characteristics: mean age 54 years (32-69), gender 12 males: 1 female, nearly all were Caucasian, majority reported heartburn (79%). All heterotopic gastric mucosas were eosinoytic type on histologic analysis. All esophageal biopsies of mucosa surrounding the inlet patches revealed normal esophageal squamous mucosa. All histologic markers for GERD were absent. 5/13 had evidence of Barrett’s esophagus, 2/13 had H. pylori, 7/13 had hiatal hernia on endoscopic evaluation, 1/13 had reflux esophagitis, and 8/13 were on proton pump inhibitor prior to endoscopy.

Conclusion: Although heterotopic gastric mucosas in the esophagus are thought to produce reflux-like symptoms, there were no histopathological changes consistent with GERD identified in mucosa surrounding the inlet patch in our small pilot study. This may be attributed to the large portion of patients on acid suppression medication prior to endoscopy.

An Unusual Presentation of Achalasia
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Purpose: Achalasia is a rare motor disorder with an incidence of 1 per 100,000 per year. Achalasia is a primary esophageal motor disorder of unknown etiology characterized by loss of esophageal peristalsis and manometrically by insufficient lower esophageal sphincter (LES) relaxation. We present an unusual case of a 50 year old gentleman with no past medical history admitted to our hospital for evaluation of cough, low grade fever and sweats for 2 months. He had been sent by his PMD for evaluation of an abnormal chest X-ray as outpatient. Physical exam was not significant, and abdominal exam showed no masses or organomegaly or lymphadenopathy. In the ED, he was found to have dry cough and fever, and an urgent CT scan of the chest was done which showed severe diffuse distension of the esophagus from the level of the thoracic inlet to the gastro-esophageal junction and nodular pleural/parenchymal opacities throughout both lungs suspected to be aspiration pneumonia. Gastroenterology was consulted and emergent Esophago-gastroduodenoscopy (EGD) was performed. EGD showed dilated esophagus with retained liquid and semisolid food particles, GE junction was puckered consistent with achalasia. The contents in the esophagus were suctioned and removed. He later underwent manometry confirming the diagnosis. Lab tests were within in normal limits. The patient was then scheduled for laparoscopic Heller myotomy. These motor abnormalities resulted in stasis of ingested food in the esophagus, leading to clinical symptoms, including dysphagia, regurgitation of ingested food, retrosternal pain, weight loss and aspiration pneumonia. Although it is well demonstrated that loss of myenteric esophageal neurons is the underlying problem, it still remains unclear why these neurons are preferentially attacked and destroyed by the immune system. The most successful therapies are clearly pneumatic dilatation and Heller myotomy with short-term success rates of 75% declining to 50-65% after more than 15 years. The above case demonstrates that respiratory symptoms such as cough and pneumonia should be worked up further to rule out esophageal diseases such as Achalasia.

Comparison of Radio-Frequency Ablation and Cryotherapy for the Treatment of Barrett’s Esophagus with Dysplasia

Purpose: The incidence of esophageal adenocarcinoma (EA) continues to rise in the United States. Barrett’s Esophagus (BE) is a major precursor of esophageal adenocarcinoma. Dysplasia is harbored in the Barrett’s mucosa, and is the premalignant lesion for EA. Multiple modalities are available for the