long-acting β-agonist. None were on oral steroids. All 48 patients completed the 3 month study and remained compliant with Aciphex® therapy. AQLQ scores improved at month 1 by 40.7 points ($P < 0.001$) and continued to improve at month 3 (43.1 points from baseline, $P < 0.001$). Likewise peak flow values improved significantly from baseline (285 ml) to the assessment at month 3 (323 ml; $P = 0.008$). GERD symptoms were reduced to nearly half during the study period as well (QOLRAD baseline = 89.9 to month 3 = 47.3; $P < 0.001$). Improvements in AQLQ, QOLRAD, and peak flow during Aciphex® treatment remained significant even after adjustment for age, BMI, gender, and asthma treatment regimen. There was good correlation between improvement in the AQLQ and QOLRAD ($r = 0.71$; $P < 0.001$). No patient required hospitalization, oral steroids, or additional medical therapy for CPA during the study period.

**Conclusion:** Our data supports the published literature that potent acid suppression in patients with CPA can improve respiratory symptoms. Objective improvement in FEV1 as measured by peak flow correlated with subjective assessment of improvement. Improvement was independent of patient characteristics such as age, gender, and BMI. Improvement was similar in patients on or off inhaled steroids, β-agonists, or leukotriene receptor antagonists. Our study should be replicated in a larger group using a double-blinded therapeutic trial design.

**10 A Case of Severe Heartburn**

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**Purpose:** A 64 yo white male with a history of recurrent paroxysmal atrial fibrillation was admitted to the hospital for elective surgery. He had failed multiple conventional therapies including anti-arrhythmic agents and two previous ablations, but continued to be symptomatic. He was deemed a candidate for minimally invasive pulmonary vein ablation with left atrial appendage ligation, or the mini-MAZE procedure. He underwent the mini-MAZE procedure and tolerated the procedure well. His post-operative course was complicated by weakness, inability to ambulate and recurrent atrial arrhythmias requiring continued anticoagulation. By POD day #5, he began to complain of anorexia, nausea and abdominal discomfort. A preliminary work-up included normal liver and pancreatic enzymes, abdominal ultrasound and CT scan. Despite this, his WBC count revealed a well-appearing male with a normal abdominal exam. He was noted to be tachycardic with a heart rate of 104. The patient was placed on an esomeprazole infusion and the decision was made to proceed with an EGD to evaluate upper GI bleeding.

EGD revealed what appeared to be a full thickness burn and necrosis at 25–30 cm from the incisors with formation of a pseudodiverticulum. The findings were suspicious for a contained perforation, likely related to the radiofrequency ablation 11 days prior. An emergent CT scan was obtained which revealed a small mediastinal space tract that appeared to connect to an esophageal defect. There was no evidence of free air in the mediastinum to suggest an ongoing leak. The patient was taken urgently to surgery and findings included the above mentioned anterior full-thickness perforation of the esophagus that extended for approximately 2 cm. The patient underwent flexible esophagogastroscope, placement of esophageal drainage mesogastric tube, exploratory laparotomy, feeding jejunostomy and Stamm gastostomy placement. The esophageal perforation was followed with periodic EGDs and gastrografin swallow studies. He began tolerating a soft diet and was eventually discharged to a rehabilitation facility 50 days after the initial mini-MAZE procedure.

In conclusion, there needs to be a high index of suspicion for esophageal perforation after the mini-MAZE procedure.

**11 Proton Pump Inhibitor and Nonsteroidal Anti-Inflammatory Use and the Development of Neoplasia in Barrett’s Esophagus**

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**Purpose:** The role of chemoprevention in Barrett’s esophagus (BE) is unclear. Evidence suggests a protective role for proton pump inhibitors (PPIs), non-steroidal anti-inflammatories (NSAIDs), and possibly statins. However, human data are limited.

**Methods:** This is a retrospective study of a well characterized large cohort of patients with documented BE diagnosed between 1985 and 2005. Prescription information was collected from pharmacy records before and after BE diagnosis. Patients were followed until the development of dysplasia, adenocarcinoma, death, or 12/2005. Cox regression analyses were performed to examine the association between NSAID, PPI, or statin prescription and the risk of developing dysplasia or cancer.

**Results:** We examined 408 patients with BE with a mean age of 61 at the time of BE diagnosis; Caucasian 91.2%, men 94.4%. The mean duration of follow-up was 6.6 years (SD 4.9). During 2690 patient-year follow-up, 125 developed dysplasia (20 high grade) yielding an incidence of 4.65 per 100 PY and 29 patients developed adenocarcinoma (1.08 per 100 PY). Approximately 38.4% were prescribed NSAIDs for a mean duration of 12.9 months, 66.4% were prescribed a PPI for a mean duration of 31.5 months, and 26.2% were prescribed a statin for a mean duration of 10.5 months.

In unadjusted analyses, only patients with BE segment > 3 cm, and more recent time of BE diagnosis were associated with increased risk of dysplasia or cancer, whereas PPI prescription was associated with reduction in that risk. This persisted in multivariable analysis (Table), and were exaggerated in analysis limited to those developing dysplasia or cancer after the first year of diagnosis; for example, PPI use (0.23, 95% CI: 0.10–0.61). No consistent associations were observed for NSAID or statin use where neither any prescription nor prescriptions > 12 months was associated with the risk of dysplasia or cancer.

**Conclusion:** PPI use seems to reduce the risk of neoplastic changes in patients with BE. NSAID or statin use is not associated with the risk of neoplasia.

<table>
<thead>
<tr>
<th>Multivariable COX Ph model predicting the risk of dysplasia</th>
<th>Hazard Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-Caucasian</td>
<td>0.51</td>
<td>0.24–1.10</td>
</tr>
<tr>
<td>BE length &gt; 3 cm</td>
<td>1.53</td>
<td>1.01–2.29</td>
</tr>
<tr>
<td>PPI prescription</td>
<td>0.64</td>
<td>0.43–0.94</td>
</tr>
</tbody>
</table>

**12 Measurement of Lower Esophageal Sphincter (LES) Characteristics during Esophageal Manometry Does Not Differ with Severity of Ineffective Esophageal Motility**

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**Purpose:** Manometric assessment of Lower Esophageal Sphincter (LES) includes Resting Pressure and LES relaxation characteristics (Residual