LONGITUDINAL MEASUREMENTS OF TOOTH WEAR IN PATIENTS WITH GASTRO-OESOPHAGEAL REFUX DISEASE (GORD). DW Bartlett, DF Evans, L Blunt, BGN Smith. Dept of Conservative Dentistry, UMDS, Guy's Hospital, London, "GI Science Research Unit, St Bartholomew's and the Royal London Schools of Medicine, Department of Manufacturing and Mechanical Engineering, University of Birmingham, UK.

GORD is known to cause palatal dental erosion in susceptible patients, particularly those experiencing regurgitation. The destruction of enamel and dentine caused by continuous oral or regurgitated gastric juice can be catastrophic leading in some cases to the complete loss of coronal enamel and dentine. It is important to identify the aetiology of the erosion and initiate preventive measures to prevent further tooth wear. Accurate measurement of tooth wear is important to establish the damage caused by regurgitation and therefore the effect of GORD on teeth. The aim of this study was to measure tooth wear in patients with palatal erosion and GORD and compare it to a group of controls with no symptoms or erosion.

Tooth wear was measured over a 6 month period in 23 patients with GORD and erosion and 12 controls. Metal disks 0.1 mm thick and 2 mm in diameter were cemented to the palatal surface of upper central incisors and clear of the opposing dentition. Impressions of the disk and surrounding tooth surface were taken and repeated after 6 months. Wear was measured with a contacting laser profilmeter using the metal disks as fixed reference points. Wear was recorded as a change in depth from the centre of the metal disk to a point on the surrounding tooth surface which was reproducible on both impressions. Reproducibility was within acceptable limits (r = 0.85). Wear was successfully measured in 13 erosion patients and 7 controls. The most common reason for failure was decementation of the metal disks over the 6 month assessment period.

A significant increase in enamel loss was observed between the patients (median 33.5 μm, range 11.4-108.2) when compared to the controls (2.6 μm, range 0.5-15.8, p = 0.004).

In conclusion, palatal tooth wear was significantly increased in patients with GORD compared to controls. We recommend that dentists should consider anti-reflux medication or a specialist referral in patients with worsening palatal tooth wear and symptoms of GORD.

T91

PRIOR DIAGNOSIS OF BARRETT'S OESOPHAGUS IS RARE IN PATIENTS WITH OESOPHAGEAL ADENOCARCINOMA. C.M. Brown, R.Jones, T.Shirazi, B.Codling, R.M. Valori. Gloucester Gastroenterology Group, Gloucestershire Royal Hospital, Great Western Road, Gloucester, GL1 3NN, England.

Background: Endoscopic screening is usually offered to patients with Barrett's oesophagus to detect treatable oesophageal adenocarcinoma. This policy will only be effective if the group at risk is identified by endoscopy after consulting with dyspepsia.

Aims: To determine the frequency of prior consultation for dyspepsia, endoscopy and diagnosis of Barrett's oesophagus in a consecutive series of patients with oesophageal adenocarcinoma.

Methods: All new diagnoses of oesophageal adenocarcinoma from a well defined postcoded district (pop: 280,500) during a 5 year period (1990-4) were identified. Open access endoscopy has been available in this district for 17 years. The overall rate of endoscopy is 1% of the population per year. Details of previous consultation, investigation and diagnosis of Barrett's oesophagus were obtained from general practitioner and hospital records.

Results: Full details were available for 58 of 67 patients presenting with oesophageal adenocarcinoma. 30 (58%) of these patients had consulted their general practitioner at least once for dyspepsia, 11 (19%) had been endoscoped, 14 (24%) had had a barium meal and only 4 (7%) had a diagnosis of Barrett's oesophagus prior to the diagnosis of oesophageal adenocarcinoma.

Conclusions: In the presence of an established open-access endoscopy service a diagnosis of Barrett's oesophagus prior to diagnosis of oesophageal adenocarcinoma is rare. If current referral patterns for endoscopy remain unchanged 95% of patients destined to develop oesophageal adenocarcinoma will never present with a pre-cancerous lesion. If all patients consulting with dyspepsia are endoscoped <50% of prospective cancer patients will be available for screening. Other risk factors are required to select patients for endoscopic screening.

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BARRETT'S OESOPHAGUS, HIGH GRADE DYSPLASIA AND EARLY ADENOCARCINOMA. COMPARISON OF ENDOSCOPIC BIOPSY AND SURGICAL PATHOLOGY FINDINGS. A.J. Cameron, H.A. Carpenter. Mayo Clinic, Rochester, Minnesota 55905, USA.

Because of the increased cancer risk, we follow patients with Barrett's oesophagus with endoscopy every 1-2 years. Using standard Olympus GIF-100 instruments, 4-quadrant biopsies are taken every 2 cm and from any focal abnormality. Oesophageal resection is usually advised for invasive adenocarcinoma (AC) or high grade dysplasia (HGD). To determine the extent and distribution of early AC and HGD in Barrett's oesophagus, and the value of endoscopic biopsy, we examined 30 consecutive oesophageal resection specimens in detail. None of these patients had a visible tumour and the endoscopy, but all had a biopsy interpreted as HGD or AC. Following surgery, the resected oesophagus was mapped in 2 cm blocks, and about 100 microscopic slides per case examined.

19 patients (14 male, mean age 60) had oesophagectomy for a preoperative diagnosis of HGD. Histological mapping showed submucosal AC in 2 (10.5%), 17 having HGD only. 12 patients (11 male, mean age 62) had a preoperative biopsy suggesting AC, without obvious cancer on endoscopy. One patient had node metastases, and resection was not done. Histological mapping in the other 11 confirmed AC in 5 cases (4 intramucosal, 1 submucosal); in 6, only HGD was found.

Endoscopic findings did not distinguish early AC from HGD: Endoscopic appearance Adenocarcinoma High grade dysplasia

<table>
<thead>
<tr>
<th>Area of Barrett's</th>
<th>1 (5%)</th>
<th>10 (43%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nodular area</td>
<td>3 (15%)</td>
<td>2 (9%)</td>
</tr>
<tr>
<td>Single nodule</td>
<td>1 (5%)</td>
<td>1 (4%)</td>
</tr>
<tr>
<td>Ulcer</td>
<td>2 (10%)</td>
<td>8 (35%)</td>
</tr>
<tr>
<td>Stricture</td>
<td>0</td>
<td>1 (4%)</td>
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The mean length of Barrett's oesophagus was 7.4 cm, mean area 37.3 sq. cm. The mean surface area of 7 adenocarcinomas was 1.1 sq. cm. HGD was found in all 30 cases, occupying a mean area of 6.5 sq. cm. The HGD involved large areas of the Barrett's in 11 cases, scattered multiple foci in 11, and a single area in 8. In 15 cases the total HGD area was 1 sq. cm or less. The mean area of low grade dysplasia was 15.7 sq. cm. Conclusions: #1 The area of Barrett's oesophagus involved with HGD or early AC is often very small. #2. Multiple biopsies can fail to detect these lesions due to sampling error. #3 The distinction between HGD and AC is not always possible on biopsy.
OESOPHAGEAL CANCER IS AN UNCOMMON CAUSE OF DEATH IN PATIENTS WITH BARRETT'S OESOPHAGUS. A. van der Burgh, W.C.J. Hop, J. Dees, M. van Blankenstein, Division of Gastroenterology, University Hospital Rotterdam and Institute of Epidemiology and Biostatistics, Erasmus University, Rotterdam, The Netherlands

The incidence and outcome of Barrett's Carcinoma (BC) in patients with Barrett's Oesophagus (BO) was ascertained in a cohort of 166 patients with BO >3cm and without carcinoma who had been identified between 1973 and 1986 at upper GI endoscopy. Their vital status or cause of death was ascertained in 1986 and 1994. In both studies 155 patients (93%) were traced. In 1986 4 cases of BC had developed, in 1994 another 4. The incidence of oesophageal carcinoma was 1:170 and 1:180 patient-years respectively, the final follow-up comprised 1440 patient-years (average 9.3 years).

In 7 out of 8 patients the tumour was symptomatic: 6 complained of dysphagia and 1 of recurrent reflux symptoms. One tumour was diagnosed at endoscopic follow-up. Three patients had carcinoma in situ, five invasive cancer. Six patients underwent surgical resection. Three survived, one died from unrelated causes, one from postoperative complications and one from metastases. One refused treatment and another was considered unfit for surgery. Both the latter died from unrelated causes.

Of the total group 79 patients have died at a mean age of 75 years, but only two from BC (2.5%).

Conclusions:
1. The incidence of oesophageal cancer in patients with BO is one in 180 patient-years.
2. Oesophageal cancer is an uncommon cause of death in patients with BO (2.5%).
3. The patients in this cohort would not have benefited from an endoscopic surveillance programme.

EARLY EXPERIENCE WITH BOTULINUM TOXIN IN THE TREATMENT OF ACHALASIA. RBSH Greaves, HE Mulcady, SE Patchett, PD Fairclough, EM Alistead, MJG Farthing. Digestive Diseases Research Centre, St Bartholomew's and the Royal London School of Medicine and Dentistry, London.

The ideal treatment of achalasia should be effective, safe and long-lasting. Recent reports have suggested that intra-sphincteric injection of botulinum toxin fulfills at least some of these criteria. Its efficacy is reported to be about 70% compared to placebo, no cases of oesophageal perforation have been reported, and benefits are maintained for up to one year. We report our experience of botulinum toxin injection in a prospective series of unselected patients with achalasia.

Methods: Eleven consecutive patients with achalasia (8 male, mean age 55, range 20-87) were treated with 60 units of Botulinum toxin (Dysport® Porton Products Ltd.) into each of 4 quadrants at the lower oesophageal sphincter using a 5mm sclerotherapy needle under direct vision. Diagnosis was based on clinical, radiological and manometric criteria. Patients were assessed pre-treatment and 1 month after treatment using a symptom score (dysphagia, regurgitation and chest pain, each scored on a 0-3 scale with 3=every meal, 2=daily, 1=occasional) and oesophageal manometry. Median follow-up was 12 months (range 6-28).

Results: The injection procedure was simple to perform and free of adverse effects. Although treatment had a beneficial effect on dysphagia (median pre-treatment score 3 [inter-quartile range 3-3]; post-treatment score 2 [0-3]; p=0.03) one month following therapy, there was no significant improvement in chest pain or regurgitation scores. Similarly, no significant reduction in median lower oesophageal sphincter pressure was observed (29.5mmHg [21-42] pre-treatment, 28.5 [17.5-55.5] post treatment p=0.67). Furthermore 4 patients (36%) required further therapy within 3 months and the overall relapse rate was 64% (7/11) after 2 years. 1 patient refused further therapy despite apparent lack of improvement.

Conclusions: Although botulinum toxin injection has potential advantages over established treatment modalities in terms of safety and tolerance, this study questions its efficacy as a promising treatment for achalasia.