Barrett’s Esophagus: State of the Art Management

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Hx

- First described in 1950 by two surgeons, Norman Barrett in the United Kingdom and Jean-Louis Lortat-Jacob in France
Norman Barrett (1903)
What is Barrett’s Esophagus?

• Esophagus lined by “Columnar mucosa” (Columnar lined epithelium, CLE)
• Specialized intestinal metaplasia (SIM) present on histological examination
Barrett’s Esophagus

The condition in which a metaplastic columnar epithelium that predisposes to cancer development replaces the stratified squamous epithelium that normally lines the distal esophagus.

Affects 5.6% of adult Americans

What is the significance of BE?

• By itself: nothing!
• The chances of development of dysplasia in this metaplastic tissue is what makes it apparently important
• What are these chances
  – Initially estimated at 1%/year or less
  – Later 0.45%/year
  – Now: 0.12-0.27%/year
Estimates of Cancer Risk for Individual Patients with Non-Dysplastic Barrett’s Have Been Falling

- **1990s** Estimate: 1% per year
  1 in 100 patients per year

- **2000s** Estimate: 0.5% per year
  1 in 200 patients per year

- **2014** Estimate: 0.25% per year
  1 in 400 patients per year
• The absolute risk of esophageal adenocarcinoma developing in a person 50 years of age or older is approximately 0.025% per year.

• However, up to 40% (in some reports over 50%) of all patients with esophageal cancer do not report GERD symptoms.

• 80-90% of esophageal adenocarcinomas are seen in pts without known Barrett esophagus
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<tr>
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Hypothetical scenario

- In the US 77 million over 50 Y/O
- If 14% weekly GERD & BE prevalence 14% about 10 million BEs
- If all adenoCas arise in BE 6500 will develop adenoCa
- Annual Ca incidence rate: 0.00065
- If 50% of adenoCas rise in GERD pts: 0.00033
- If 20% of adenoCa rise in GERD pts: 0.00017
- The figure in Iran is much lower because BE is much less infrequent (probably 1/10th or less: 0.000033)
- Annual incidence rate of serious injury/death due to car accidents in Iran: 0.00412
- So driving in Iran or simply crossing the streets is at least 125x more dangerous than developing esophageal adenoCa if you have BE!
Guidelines for Endoscopy in GERD

• “Upper endoscopy is indicated in men and women with heartburn and alarm symptoms (dysphagia, bleeding, anemia, weight loss, and recurrent vomiting).”  


• “Upper endoscopy is indicated in men and women with typical GERD symptoms that persist despite a therapeutic trial of 4 to 8 weeks of twice-daily proton pump inhibitor therapy.”

• “Upper endoscopy is not required in the presence of typical GERD symptoms.”

• “Endoscopy is recommended in the presence of alarm symptoms and for screening of patients at high risk for complications [Barrett’s esophagus].”  

AGA Medical Position Statement on Endoscopic Screening for Barrett’s Esophagus

- We recommend against screening the general population with GERD for Barrett’s esophagus.

- In patients with multiple risk factors associated with esophageal adenocarcinoma, we suggest screening for Barrett’s esophagus.

  Chronic GERD, hiatal hernia, age ≥50, male gender, white race, elevated BMI, intra-abdominal body fat distribution

How is BE diagnosed?

- In a relaxed patient undergoing UGIE
- During scope withdrawal while the esophagus is partially deflated
- Finding columnar lined mucosa in the distal esophagus (above the proximal end of gastric folds)
- Confirm presence of SIM by histological examination
- NOTE: Please DO NOT report “Barrett’s Esophagus” in your endoscopy notes. Just report “columnar lined mucosa”.

Proposed Endoscopic Landmark for GEJ
Proximal extent of gastric folds

McClave. Gastrointest Endosc 1987; 33: 413.
The major endoscopic landmark for the gastro-oesophageal junction
The top of the gastric mucosal folds
How is it reported or categorized?

- Prague classification
- In a relaxed patient while withdrawing the scope locate the proximal end of gastric folds as distance (cm) from incisors
- Pull back and locate the most proximal end of “circumferential” columnar lined esophagus (distance from incisors, cm)
- Pull back and locate the proximal end of “tongues” of columnar lined esophagus (distance from incisors, cm)
- Report as:
  - Columnar lined esophagus: CxMy
Endoscopic recognition of Barrett’s Oesophagus

Columnar lined oesophagus
CLE detected on EGD

- Four quadrant bx every 1-2cm
- SIM+, No Dysplasia - □ repeat q3-5y
- SIM+, LGD+ □ repeat q6-12m or ablation
- SIM+, HGD+ □ confirmed by SECOND Pathologist □ Intervention
What is dysplasia and how is it diagnosed?
Histological Features of Dysplasia in Barrett’s Esophagus

Nuclei:
- enlargement
- pleomorphism
- hyperchromatism
- stratification
- atypical mitoses

Villiform surfaces and tubules show crowding

No Dysplasia  Dysplasia
How is dysplasia classified?

• High grade dysplasia (HGD) vs Low Grade Dysplasia (LGD)
• What is the difference?
• What is their significance?
Illustrations of Barrett’s dysplasias in a new GI pathology textbook

Low-grade  High-grade

These don’t look alike. Any fool can tell the differences between them. Where does LGD end and HGD begin?
The standard diagnoses used for biopsies of Barrett’s mucosa

0
Neg

Indef

LGD

HGD

Follow

Treat

0
3

Highest LGD
Lowest HGD
How concordant are pathologists in diagnosing dysplasia?

• Concordance for LGD: 0.33%
• Concordance for HGD: 0.67%
Endoscopy with systematic biopsy sampling for dysplasia remains the clinical standard for managing patients with Barrett’s esophagus.
Endoscopic Surveillance Might Not Decrease Mortality from Esophageal Adenocarcinoma

8,272 pts. with Barrett’s esophagus (BE)

Surveillance endoscopy within 3 years was NOT associated with decreased risk of death from esophageal cancer (adjusted odds ratio 0.99; 95% CI 0.36-2.75)

38 pts. with confirmed death from esophageal cancer

55% surveillance endoscopy performed within 3 years

101 living Barrett’s pts. matched for age, sex, follow-up duration

60% surveillance endoscopy performed within 3 years

Randomized Trial of RFA vs. Surveillance for Low-Grade Dysplasia (LGD) in Barrett’s Esophagus

Data & Safety Monitoring Board early termination: RFA superior to surveillance for preventing neoplastic progression

Potential for patient safety issues if trial continued

- 1.5% RFA group
- 26.5% surveillance group
- 25% ↓ risk of progression (95% CI 14.1-35.9%, P<.001)

Progression to cancer at 3 years

- 1.5% RFA group
- 8.8% surveillance group
- 7.4% ↓ risk of cancer (95% CI 0-14.7%, P=.03)

Phoa KN. JAMA 2014;311:1209.
Endoscopic Eradication Therapy for Mucosal Neoplasia in Barrett’s Esophagus 2015

- EMR of mucosal irregularities for staging and therapy
- Ablate the remaining Barrett’s metaplasia to minimize metachronous neoplasia
Subsquamous Intestinal Metaplasia

Protected from destruction by RFA?

Squamous Epithelium

Hidden from endoscopist

Intestinal Metaplasia
Chronic GERD symptoms (e.g., heartburn and regurgitation) and ≥1 risk factor for esophageal adenocarcinoma:
- age ≥50 yr
- male sex
- white race
- hiatal hernia
- elevated BMI
- intraabdominal body-fat distribution
- or tobacco use

No further screening

No Barrett's esophagus

Consider screening endoscopy for Barrett's esophagus

Columnar-lined esophagus seen endoscopically, and esophageal-biopsy specimens show intestinal metaplasia

No dysplasia

Surveillance endoscopy every 3–5 yr; if surveillance biopsies show low- or high-grade dysplasia, follow the guidelines for dysplasia

Suspected low-grade dysplasia

Have diagnosis confirmed by expert gastrointestinal pathologist

Low-grade dysplasia confirmed

Surveillance endoscopy every 6–12 mo or endoscopic eradication therapy

Suspected high-grade dysplasia or intramucosal carcinoma

High-grade dysplasia or intramucosal carcinoma confirmed

Endoscopic eradication therapy
Is Barrett Esophagus a Complication of GERD?
CHRONIC PEPTIC ULCER OF OESOPHAGUS

CHRONIC PEPTIC ULCER OF THE OESOPHAGUS AND 'OESOPHAGITIS'

BY N. R. BARRETT, LONDON

THE BRITISH JOURNAL OF SURGERY


The terms 'oesophagitis' and 'peptic ulcer of the oesophagus' connote one thing to some people and something quite different to others. Confusion has

In 1884 Mackenzie defined 'oesophagitis' as "acute idiopathic inflammation of the mucous membrane of the oesophagus giving rise to extreme odynophagia and often to aphagia"; this condition was the entity which surgeons meant when they used the term 'oesophagitis', and in this sense the disease
According to Mackenzie the cause was unknown, but the chief symptom was excruciating burning or tearing pain—odymphagia—induced by any attempt to swallow or any movement of the laryngeal muscles. Being unable to get relief by drinking, he could not endure the torment. In adults there was constant expectoration of frothy saliva; the patients were not pyrexial, but might become delirious, and Mackenzie, having already discovered syphilitic eroded patches, proceeded to abscess formation or suppuration. He judged the lesion to be diffuse catarrhal inflammation of the mucosa at the upper end of the gullet mycosis, syphilis, or growths. To-day we do not recognize the disease which Mackenzie described, but we use ‘œsophagitis’ to describe another entity which chiefly affects the lower part of the gullet.
Painting to show the characteristic appearances of 'reflux usoohanitis' and 'oeotic ulcer of the esophagus' as seen at endoscopy.
I submit that most of these cases are in truth examples of congenital short esophagus, in which there is neither general inflammation nor stricture formation, but in which a part of the stomach extends upwards into the mediastinum -or even to the neck-and that in this stomach a typical chronic GU can form. I urge that, as accurate surgery must rest upon accurate pathology, we must distinguish between gastric and esophageal ulcers.
THE OESOPHAGUS LINED WITH GASTRIC MUCOUS MEMBRANE

BY

P. R. ALLISON AND A. S. JOHNSTONE

Leeds

- Peptic oesophagitis and peptic ulceration of the squamous epithelium of the oesophagus are secondary to regurgitation of digestive juices, are most commonly found in those patients where the competence of the cardia has been lost through herniation of the stomach into the mediastinum, and have been aptly named by Barrett (1950) "reflux oesophagitis."
Allison and Johnstone 1953

- It describes accurately in two words the pathology and aetiology of a condition which is a common cause of digestive disorder.

- In the same paper Barrett for the first time drew a sharp distinction between the above and lesions which had previously been described by pathologists as peptic ulcers of the oesophagus, but which in fact were gastric ulcers occurring in gastric mucous membrane lining a viscus which, from the outside, looked like oesophagus.
Reflux esophagitis ulcer (peptic ulcer of the esophagus):
• Dense submucosal fibrosis
• Superficial ulcer (missed easily)
• May ooze, rarely bleeds
• Does not perforate
• Trivial findings on necropsy
• Nothing interesting to keep in museum

Ulcer in the esophagus with a gastric lining:
• Behaves like an ulcer in the abdominal stomach
• Causes stricture
• May bleed profusely
• May perforate
• Easily found at necropsy
• Kept in museum
The result has been that pathologists have been describing one thing and clinicians another, and they have had the same name.

The clarification of this point has been so important, and the description of a gastric ulcer in the oesophagus so confusing, that it would seem to be justifiable to refer to the latter as Barrett's ulcer.

The use of the eponym does not imply agreement with Barrett's description of an oesophagus lined with gastric mucous membrane as "stomach."

Such a usage merely replaces one confusion by another.
Allison and Johnstone 1953

• A variable amount of the oesophagus below the aortic arch may be lined by gastric mucosa of cardiac type.
• This is presumably, but not necessarily in every case, a congenital abnormality, and
• in the series presented has always been associated with herniation of the true stomach through the diaphragmatic hiatus, with the cardia lying in the mediastinum.

• Barrett accepted this view
Heterotopic mucosa, a common finding in the "normal" esophagus, may take one or more forms

1. During early fetal life, the lining of the esophagus is derived from the simple columnar epithelium of the primitive foregut.

2. In the 5th or 6th month epidermoid mucosa begins to replace the cylindrical cells.

3. This process originates in the midesophagus and extends caudally and orally so that by the end of gestation the fetal esophagus is covered by squamous epithelium.

4. At birth, persisting islands of columnar mucosa may be found in the cervical esophagus, which is the last portion to become stratified.

2. By the 7th week of fetal life, caudal descent of the stomach has been completed.

3. When clusters of potentially gastric cells remain within the esophagus, characteristic nests of ectopic gastric fundic epithelium may occur after birth at any level of the esophagus.

2. The superficial cardiac glands of the lamina propria may also represent true gastric rests. Although general agreement on this point is lacking.
THE COLUMNAR-LINED ESOPHAGUS (BARRETT SYNDROME)---AN ACQUIRED CONDITION?

Sanford M. Mossberg, M.D.

Department of Gastroenterology, Medical Division, Montefiore Hospital and Medical Center, New York, New York, and the Department of Medicine, New Rochelle Hospital, New Rochelle, New York
• M 18 Y/O in 1946
• Daily nausea/self-induced vomiting
• 1960: Heartburn/black stool \( \Box \) antacids/sedatives
• 1961: admission for heartburn, pp abd pain, hematemesis \( \Box \) XR: duodenal deformity, small SHH, questionable esophagitis
• Esophagoscopy: slight redness of the distal esophagus \( \Box \) Bx
Squamous epithelium (1961)
• Anticholinergics, antacids, head of bed elevation □ persistent SS
• 1962: Admission for heartburn, mid-dorsal pain, regurgitation, and melena
• Cinefluorography: ulcer in posterior wall of the esophagus about 5 cm above the cardioesophageal junction.
  – At this point the esophagus was dilated and the ulcer appeared to lie within an intraluminal filling defect.
  – The radiological diagnosis was "probable ulceration in a leiomyoma but carcinoma cannot be excluded."
• Esophagoscopy: reddened esophageal mucosa distal to 37 cm and a normally located esophagogastric junction at 40 cm from the incisor teeth. At 38 cm a deep, posterior wall crater was seen.
  – Bx of the superior margin of the ulcer
acute and chronic inflammation in gastric mucosa
• 2 m later admitted again:
  – Hematemesis
  – WN man, mild epigastric tenderness and tarry stool (guaiac, 4+) in the rectum
  – BP: 120/80 mm Hg; PR: 136/min; RR: 20/min; and T: 99 F.
  – Hb: 8.6 g/100 ml; WBC: 21,800/mm3, BUN: 37mg/l00 ml; BS: 102mg/l00 ml; and U/A: negative.
• NG tube: black, guaiac-positive material
• After 2 units of whole blood, Hb increased to 10.3 g/l00 ml
large ulcer approximately 2 cm above a sliding esophageal hiatal hernia
• Esophagoscopy: red, friable, bleeding esophageal mucosa starting at 20 cm from the incisor teeth and extending to the gastroesophageal junction (40 cm).
  – A mass of heaped-up mucosa at 37 cm
Columnar epithelium lining the esophagus (at 23, 30, & 37 cm)
Squamous epithelium at 18cm
• 1 week later vagotomy/pyloropoplasty
• Since surgery: no recurrence of SS
• His daily episodes of vomiting ceased
• Esophagogram, 6 months later: negative except for a small, residual hiatal hernia
• There are animal models where induced severe esophagitis heals with columnar metaplasia
• Most esophagitis episodes heal with squamous epithelium
• The origin of the columnar cells is not clear:
  – Esophagus,
  – Cardia,
  – Bone marrow
• Good epidemiologic evidence to link GERD and increased EAC prevalence
• Little evidence to link GERD and BE causally
Take home message

• Barrett esophagus is an endoscopic AND histopathologic Dx
• The risk of Ca is extremely low in nondysplastic BE tissue
• Prevalence of BE is much lower in Iran than that reported in the west
• Although surveillance for dysplasia in BE is recommended by most international societies, its efficacy is in serious question
• The problems with sampling and histopathologic Dx
• The patient with Barrett Esophagus will get relieved when his/her endoscopist dies!
CHRONIC PEPTIC ULCER OF THE OESOPHAGUS AND
‘OESOPHAGITIS’

By N. R. BARRETT, LONDON

The terms ‘oesophagitis’ and ‘peptic ulcer of the oesophagus’ connote one thing to some people and something quite different to others. Confusion has overtaken us partly because the rich legacy of clinical observations recorded by Morell Mackenzie and his contemporaries have not been sufficiently carefully aligned with the recent advances in the pathology of the living oesophagus.

In 1884 Mackenzie defined ‘oesophagitis’ as “acute idiopathic inflammation of the mucous membrane of the oesophagus giving rise to extreme odynophagia and often to aphagia” ; this condition was the entity which surgeons meant when they used the term ‘oesophagitis’, and in this sense the disease was first named by John Peter Frank in the eighteenth century. In 1732 Boehm described the acute pain which “reached down even to the stomach and which was accompanied by hiccup and a constant flow of serum from the mouth”. In 1785 a physician called Bleuland was struck down with the disease himself and he carefully recorded the course of the malady. In 1828 Billard published a statement concerning the ailments of the newborn, and it was he who first reported ‘oesophagitis’ in children. In the following year Mondiere (1829) wrote a thesis describing an attack of oesophagitis he had experienced personally, and the subject was further illuminated by communications from Hamburger, Padova, and many others.

Diseases, decubitus, aneurysms, catarhal inflammations; those associated with diverticulum, tuberculosis, syphilis, varicosities, and ulcers due to thrush. Apart from these he drew special attention to peptic ulcer of the oesophagus, which he said exactly simulated chronic gastric ulcer. It was a rare but definite entity which had first been described by Albers in 1839 and from then onwards had been occasionally reported by pathologists. Rokitansky had given the weight of his authority to support the view that peptic ulcer of the lower oesophagus existed; it was considered to be due to the presence of gastric juice in the gullet. Tileston, who reviewed the literature, stated that up till 1906 there had been 44 undoubted examples published. The ulcers concerned were generally single, but were often associated with chronic peptic ulcer in the stomach or the duodenum. They were large, penetrating lesions, sometimes 6 to 8 cm. in length, which were disposed longitudinally but might encircle the gullet, and they were considered to occur in that part of the oesophagus which lay “above the cardiac sphincter”. The patients were usually elderly, and many had no symptoms referable to the oesophagus until shortly before admission to hospital. Death generally resulted from perforation either into a large vessel, the pericardium, the mediastinum, or the pleural cavity; some died of pneumonia, but few had symp-
According to Mackenzie the cause was unknown, but the chief symptom was excruciating burning or tearing pain—odynophagia—induced by any attempt to swallow or any movement of the laryngeal muscles. The patient generally experienced great thirst, but being unable to get relief by drinking, he could but endure the torment. In adults there was constant expectoration of frothy saliva; the patients were not pyrexial, but might become delirious, and Mackenzie, who gave details of 5 personal cases, said that none proceeded to abscess formation or suppuration. He judged the lesion to be diffuse catarrhal inflammation of the mucosa at the upper end of the gullet and diagnosis was based upon the extreme pain and the absence of pharyngeal inflammation.

We can be satisfied that Mackenzie was not referring to inflammation the result of swallowed corrosives, nor to the ulceration of specific fevers such as diphtheria; nor to thrush, tuberculosis, actinomycosis, syphilis, or growths. To-day we do not recognize the disease which Mackenzie described, but we use 'esophagitis' to describe another entity which chiefly affects the lower part of the gullet.

In 1900 all inflammatory conditions affecting the gullet were considered as varieties of 'oesophagitis', but it was about this time that one of these began to arouse especial interest. Writing in 1906, Wilder Tileston pointed out that at least twelve different types of ulceration could be clearly separated from each other—namely, those due to carcinoma, corrosives, foreign body; those complicating acute infectious tmons of oesophageal obstruction. The histology of these ulcers was identical with chronic gastric ulcer, and the adjacent mucosa was gastric in type; it was assumed to be 'ectopic' because it lined the lower part of the gut in the mediastinum.

Twenty years later 'peptic ulcer of the oesophagus' became a matter of special interest. The pathologists continued to report isolated examples of chronic peptic ulcer occurring at the lower end of the gullet. Meanwhile clinicians, who were dealing with patients, were also reporting a variety of oesophageal lesions which had been discovered as a result of improvements in radiological diagnosis and in oesphagoscopy.

The confusion in which we find ourselves to-day dates from this time, because the pathologists, the clinicians, and the endoscopists assumed that they understood each other and that they were talking about the same entity when they referred to 'peptic ulcer of the oesophagus' or to 'oesophagitis'. By 1929 it was clear that all was not well, for Chevalier Jackson claimed to have seen 88 cases in 4000 consecutive endoscopies, whereas Stewart and Hartfall (1929) had found but 1 example in 10,000 consecutive autopsies. Since 1929 there have been many important contributions, such as those by Lyall (1937), Chamberlin (1939), Dick and Hurst (1942), and by Allison and Johnstone, of Leeds (1943, 1946, and 1948). None of these authors find themselves in difficulty as to what they mean by 'peptic ulcer of the oesophagus' or 'oesophagitis'. 
Natural Hx of BE

• Longitudinal studies:
  – most cases of BE do not progress beyond nondysplastic intestinal metaplasia or transient low-grade dysplasia

• In cases of progression to HGD:
  – A meta-analysis, 4 studies, 236 pts, 1234 pt/yr FU, 69 adenoCa
  – 6 per 100 patient-years during the first few years of follow-up
How is natural hx affected by ablative Rx?


• Patients:
  – nondysplastic BE (NDBE), low-grade dysplasia (LGD), or high-grade dysplasia (HGD) and
  – follow-up of at least 6 m
  – The rate of cancer in patients undergoing ablation and from the natural history data was calculated using weighted-average incidence rates (WIR)

• RESULTS:
  – 53 articles met the inclusion criteria for the natural history data. Cancer incidence of:
    • 5.98/1,000 patient-years (95% CI 5.05-6.91) in NDBE;
    • 16.98/1,000 patient-years (95% CI 13.1-20.85) in LGD;
    • 65.8/1,000 patient-years (95% CI 49.7-81.8) in HGD patients
  – 65 articles met the inclusion criteria for BE pts undergoing ablation (1,457 patients, NDBE; 239 patients, LGD; and 611 patients, HGD). The WIR for cancer was:
    • NDBE: 1.63/1,000 pt-yr (95% CI 0.07-3.34)
    • LGD: 1.58/1,000 pt-yr (95% CI 0.66-3.84)
    • HGD: 16.76/1,000 pt-yr (95% CI 10.6-22.9)

• CONCLUSIONS:
  – Compared to historical reports of the natural history of BE, ablation may be associated with a reduction in cancer incidence,
  – although such a comparison is limited by likely heterogeneity between treatment and natural history studies.
  – The greatest benefit of ablation was observed in BE patients with HGD.
Shaheen et al
NEJM 2009: 2277

298 ablation session in 84 pts (3.5/pt)

1 UGIB □ Endoscopic Rx
5 strictures □ Endoscopic dilatation (2.6/pt)

Over 12 m, 13,573 bx (9517 ablations, 4056 controls)
1260 bx from LGD pts+ablation:
97.5% free of SIM at 12m vs. 56.9% in controls
1464 bx HGD pts+ablation:
98.5% free of SIM at 12m vs 58.6% in the controls
Our findings should be viewed with caution:
Cancers occurred in only 5 patients (1/84 with ablation & 4/43 in controls
So the shift of a single incident cancer would have resulted in a loss of statistical significance
What other options?

• Surgery:
  – Curative
  – mean mortality: 2.7% (up to 14%)
  – 28% morbidity (anastomotic leaks, strictures, wound dehiscence, recurrent aspiration pneumonias) may require a second operation

• One of the longest F/U (BMJ 2000):
  – 143 BE patients
  – Followed for a mean of 4.4 years (up to 10 y)
  – Regular surveillance
  – Only 1 adenoCa found
  – He died of surgery not of the Ca
EMR, ESD

- Laborious
- Complications are real
- Recurrence happens even beneath a now normal looking mucosa
Cost–utility model
Markov model
Base case scenario: endoscopic surveillance of
   - BE q3y
   - LGD q1y
   - HGD q3m
Does more harm than good when compared with no surveillance
Surveillance produces:
   - fewer quality-adjusted life-years (QALYs)
   - for higher cost than no surveillance,
The cost per cancer identified:
   - ~ £45,000 in the surveillance arm
   - with no apparent survival advantage
   - owing to high recurrence rates
   - and increased mortality due to more surgical interventions (i.e. oesophagectomies) in this arm.
The input parameters to which the model is most sensitive:
- the rate of recurrence of adenocarcinoma after oesophagectomy in the surveillance compared with the no surveillance arm
- the rate at which adenocarcinoma becomes symptomatic once it has developed
- the utility value (quality of life) attached to the health states for Barrett’s oesophagus.

One-way sensitivity analyses (for 3-yearly surveillance to become costeffective at usual levels of willingness to pay £30,000 per QALY):
- rate of recurrence of AC post-op reduces to 4.5% in the surveillance arm (from the base case of 9.3%)
- rate of recurrence of AC post-op reduces to 7% in the non-surveillance arm (from the base case of 26%)
- progression from undetected to symptomatic AC increases to at least 23% per year (from the base case of 14.3%)
- utility values for Barrett’s oesophagus health states fall to ≤ 0.63 (from the base case of 0.81)
New techniques for detection

Compared with gastroscopy:

Sensitivity: 73.3% (95%CI 44.9-92.2%)
Specificity: 93.8% (95%CI 91.3-95.8%)
for ≥1 cm circumferential length

Sensitivity: 90.0% (55.5-99.7%)
Specificity: 93.5% (90.9-95.5%)
For segments ≥2 cm

Kadri SR et al, BMJ 2010;341:c4372
doi:10.1136/bmj.c4372
Take Home Message

• Barrett’s Esophagus is defined endoscopically AND histologically
• “Columnar lined esophagus” can be reported but NOT “Barrett’s esophagus”
• Prague classification for CLE reporting
• Most patients with BE never die from it
• Current guidelines are far from perfect
• Dysplasia Dx is not consistent between pathologists
• Current Rx modalities are far from perfect (serious mortality/morbidity)
• Cost-utility analyses do not suggest any benefit (even may be harmful
• Only high risk patients (male, white, older, fat) may benefit from current surveillance/interventions
The BE patient is relieved only when his/her Endoscopist is dead!