17% of men and nearly 50% of women in the study population.[1] For men weighing 59-118 kg and women of 60-90 kg it may be safer to use a 1 inch (25mm) needle. A woman over 90 kg may need a 1.5 inch (38mm) needle.

Healthcare professionals may hesitate to use longer needles on the grounds that they are likely to cause the patient more discomfort. However, skeletal muscle has a poor supply of pain fibres compared with skin and subcutaneous tissue.[10]

Consideration should be given to needle gauge.[11] A wider bore needle ensures that the vaccine is dissipated over a wider area, thus reducing the risk of localised redness and swelling.[12]

A standard size of needle will not guarantee successful intramuscular injection in all people. When intramuscular vaccine administration is needed to ensure optimal immunogenicity and minimise local reactions, a selection of non-fixed needles (pre-filled syringes that may be provided with a needle fixed on the barrel) should be available to allow healthcare professionals to select a length and gauge of needle appropriate to each patient.

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Barrett's oesophagus: the continuing conundrum

Surveillance should be confined to the surgically fit

In 1950 Barrett wrote a treatise to clarify confusion over oesophagitis which "connote[s] one thing to some people and something quite different to others."[1] He described gastric mucosa extending into the tubular oesophagus as the result of a congenitally shortened oesophagus. The presence of columnar lined epithelium in the oesophagus is now referred to as Barrett's oesophagus. It is associated with chronic gastro-oesophageal reflux disease and an increased risk of oesophageal adenocarcinoma.[2] Quantifying this risk, and the best methods for early diagnosis, are still the subjects of considerable debate.

Endoscopically the distal end of the pearly white oesophagus is readily distinguished from the salmon red of the proximal stomach: the so called "Z line" or squamocolumnar junction. However, the location of the Z line may be difficult to identify in cases of intense inflammation, hiatal hernia, and stricture patients with oesophagitis. Extension of the Z line proximally—representing columnar replacement of the squamous epithelium of the distal oesophagus (Barrett's oesophagus)—is seen in 5-15% of patients with peptic oesophagitis.[2] Historically one point of confusion has been whether a minimal length of columnar metaplasia is needed to qualify for the diagnosis of Barrett's oesophagus: is it >2 cm, >3 cm, or >5 cm? In part, these arbitrary criteria were established to avoid "false positive" biopsies of intestinal metaplasia which often occur in the gastric cardia. The requirement of a minimum length to establish Barrett's oesophagus has been abandoned. Histologically, the columnar based epithelium can be one of three types: gastric fundic gland, junctional type epithelium with cardiac mucous glands, or a distinct type of columnar metaplasia called specialised columnar (intestinal) epithelium.[3] Only patients who have the specialised columnar epithelium are at an increased risk of cancer and should be considered for endoscopic surveillance.

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About 10% of patients who have Barrett's oesophagus at the time of the initial endoscopic examination have coexistent oesophageal adenocarcinoma.[4 5] The incidence of oesophageal adenocarcinoma has rapidly increased over the past two decades in Western Europe and the United States.[6] Unfortunately, the 5 year survival rate is 11%. The risk factors for this cancer are longstanding gastro-oesophageal reflux disease, the presence of Barrett's specialised columnar epithelium, male sex, and white race.[6 7] In a case-control study Lagergren et al showed that a greater risk of oesophageal adenocarcinoma was associated with more frequent, more severe, and longer lasting symptoms of acid reflux.[7]

It is difficult to know how to avoid the dismal prognosis of advanced cancer in patients with Barrett's oesophagus. Earlier reports from prospective studies showed that about one adenocarcinoma developed for every 100 patient years, representing a 30-fold to 125-fold increase in the risk of cancer compared with the general population.[2 8] It is also believed that in patients with Barrett's oesophagus the development of adenocarcinoma is preceded by a continuum of dysplasia, from low to high grade, that can be readily identified by

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random biopsies. For these reasons, most patients with Barrett's oesophagus have undergone surveillance endoscopy every 1-2 years.[8] However, more recent studies have shown that the risk of cancer risk in patients with Barrett's oesophagus is lower, perhaps 1 case for every 200 patient years.[9–11] Also, the focal nature of dysplasia in Barrett's oesophagus may limit the effectiveness of endoscopic surveillance.

In this issue of the journal Macdonald et al (p 1252) present their observations on endoscopic surveillance for Barrett's oesophagus at the Leicester General Hospital.[12] Altogether 143 patients with Barrett's epithelium were offered endoscopic surveillance. An additional 266 patients with Barrett's oesophagus were deemed to be ineligible for surveillance because they were too old or had comorbid conditions. Five cases of oesophageal adenocarcinoma were diagnosed, but only one was detected as a result of participation in the surveillance programme. There were 33 deaths in the group under surveillance, but the great majority of these were caused by non-oesophageal diseases. These findings are in agreement with a report from the Netherlands which showed that oesophageal carcinoma is an unusual cause of death among patients with Barrett's oesophagus.[13] Both of these studies questioned the effectiveness of endoscopic surveillance in patients with Barrett's oesophagus.

What is a reasonable course of action? There are three issues to consider. Firstly, the absolute risk of developing cancer is quite small. Secondly, Macdonald et al showed that many patients were not suitable for surveillance. Thirdly, many of the patients in the surveillance groups were dropped from surveillance because they developed a comorbid illness or were generally frail.

Thus, only surgically fit patients with specialised columnar (intestinal) epithelium should be enrolled on surveillance programmes. It is unclear what proportion of the patients in Leicester who were under surveillance had specialised intestinal metaplasia. Although it is not a foregone conclusion that patients with dysplasia will develop cancer, dysplasia remains the best indicator of cancer risk.[14] Methylene blue staining of specialised columnar epithelium using an established biopsy protocol may increase the yield of dysplasia at lower cost.[15] A confirmed diagnosis of high grade dysplasia should be followed by serious consideration of oesophagectomy. The risk of unsuspected carcinoma in patients with high grade dysplasia at the time of oesophagectomy is 43-73%. Selected patients may be subjected to a vigorous biopsy protocol every three months or considered for ablative endoscopic treatment.[16 17] The progression of low grade dysplasia to cancer is less well defined, but it is estimated to occur in 18% of patients during 1.5 to 4.3 years of follow up.[10] For patients with low grade dysplasia, surveillance at six months, one year, and then yearly is recommended. Several retrospective studies have shown that patients with Barrett's oesophagus who are under regular surveillance and develop cancer have the diagnosis made at an earlier stage of the disease and have less nodal involvement and improved survival when compared with patients who are not undergoing surveillance.[18–20]

For the large group of patients who have Barrett's oesophagus without dysplasia, the risk of subsequently developing cancer is quite low. A recent decision analysis of patients who had Barrett's epithelium without dysplasia suggested that endoscopy surveillance every five years is as adequate and as cost effective as mammography screening for carcinoma of the breast.[11 19] Combining histopathology with flow cytometry may better allow us to define which patients are at high risk. In a recent study of 215 patients with baseline biopsies that showed no dysplasia, indefinite dysplasia, or low grade dysplasia with no abnormalities on flow cytometry, the cumulative, five year risk for carcinoma was 0%.[21]

The cumulative data support endoscopic surveillance in patients who are surgically fit and who have Barrett's oesophagus with specialised columnar epithelium. In future there is likely to be less frequent but better endoscopic surveillance of patients with Barrett's oesophagus.

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