illness for the couple. Trying out different forms of words for explaining the illness to children may make it easier for the patient or partner to express concerns or expectations not previously shared, and it may be an avenue to better and more supportive communication between the partners.

Children need truthful explanations

We should explain that children do not need every detail: what they crucially need is a truthful and convincing explanation for their parents' distress. We should introduce the possibility of asking children whether they have their own ideas about why this bad thing might have happened. Children (like adults) may need to be relieved from irrational feelings of guilt. We should offer to be there when the children are told if the parents would find this helpful. Such an offer will often be declined if the parents feel adequately prepared. If time has been spent dealing with the issue of communicating the diagnosis to the children this should be mentioned in communications between those who provide primary care and hospitals. Referrals to child psychiatric services should be considered in particularly difficult situations or when children develop overt and important psychological or behavioural problems

Why do we not always do as much of this sort of family support as we might? We have many other demands on our time, but we also find it difficult to witness situations which bring to mind our own fears or memories of pain, separation, and loss. None the less, we should be prepared to offer help to our patients in deciding what to tell their children about a serious illness. And when we do become involved in work of this kind we must be aware of our own needs for support.

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Gastro-oesophageal cancer: death at the junction

Understanding changes at molecular level could lead to screening opportunities

The death rates from cancers of the oesophagus and gastro-oesophageal junction, adjusted for age, have risen steadily since the early 1970s (from 3 to 6 per 100 000 and from 1.5 to 3 per 100 000 population in the United Kingdom respectively).[1] These figures are comparable to those in northern Europe and the United States. The incidence of Barrett's adenocarcinoma in the United States has increased from 0.3 per 100 000 to 2.3 per 100 000 over the past three decades.

Despite improvements in multimodality therapy, especially chemotherapy regimens of combined epirubicin, cisplatin, and fluorouracil combined with surgery, survival has not improved significantly, suggesting that alternative strategies for identifying and treating these conditions are needed.

The incidence of intestinal metaplasia of both the oesophagus (Barrett's oesophagus) and the gastro-oesophageal junction are also increasing. This metaplastic tissue is believed to have a premalignant potential, and Barrett's oesophagus is related to bile and acid reflux disease.[2] About 8% of patients undergoing routine endoscopy and 3% of the adult population have at least 1 cm of Barrett's oesophagus.[3] Furthermore, 17% of patients undergoing routine endoscopy and 6% of the adult population may have intestinal metaplasia of the gastro-oesophageal junction.[3 4] These metaplastic lesions are characterised by mucin-secreting epithelium, containing goblet cells, that replaces the native stratified squamous or transitional zone epithelium.

Metaplastic changes may progress from dysplasia to adenocarcinoma.[2] About 5-15% of people with Barrett's oesophagus and 2-5% of those with intestinal metaplasia of the gastro-oesophageal junction also have dysplasia, which in the case of Barrett's oesophagus increases the risk of cancer between 30-fold and 150-fold. The risk of cancer for people with metaplasia of the gastro-oesophageal junction has so far not been quantified.

This has led many centres to establish surveillance programmes to identify dysplastic changes or early adenocarcinomas.[5] However, although these programmes detect cancers earlier, there is controversy about their cost effectiveness.[6 7] Interest has therefore been rekindled in strategies to prevent the onset of Barrett's oesophagus or intestinal metaplasia of the gastro-oesophageal junction and to find other risk factors that more accurately detect the subgroups of patients who will progress to malignancy.

Rare inherited syndromes of colorectal cancer have given valuable information about tumour initiation. Syndromes of familial gastro-oesophageal cancer are rare and heterogeneous and account for only 1-5% of cases, but they have also provided valuable information.[8] In particular, inherited germline mutations of the E cadherin gene, involved in cell adhesion, leads to loss of E cadherin expression.[8] E cadherin is not only a cell adhesion molecule but also a tumour suppressor gene. Reduced expression of adhesion molecules on the surface membranes of cancer cells makes them far more likely to have invasive properties. Furthermore, analysis of sporadic gastric cancer shows that the stage and invasiveness of gastric tumour is also associated with reduced expression of E cadherin. E cadherin binds with an intracellular protein called β catenin, to form adhesion complexes. β catenin levels are tightly regulated within the cell, and free, unbound β catenin is

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normally completely degraded. If any free, unbound β catenin accumulates it can enter the nucleus and bind with certain transcription factors that help activate target oncogenes such as COX-2 and c-myc that may induce proliferation.[9] The amount of β catenin and transcription complexes in the nucleus is dramatically increased by the release of unbound β catenin in situations where E cadherin expression is reduced. This situation seems to occur during the progression from metaplasia to adenocarcinoma.[10]

A second inherited predisposition to gastric cancer has also been reported. Infection of the gastric body with *Helicobacter pylori* can cause hypochlorhydria, atrophy, and malignancy, whereas infection of the antrum is related to the development of peptic ulcer disease. Evidence now suggests that these different outcomes are related to the host response. Abnormal variants of the interleukin 1β gene (genetic polymorphisms that enhance activity) are associated with an increased risk of developing gastric cancer.[11] Patients possessing such a polymorphism produce higher levels of interleukin 1β in response to *H pylori* infection, and interleukin 1β increases the risk of developing atrophy and malignancy. Furthermore, interleukin 1β can reduce the expression of adhesion molecules, such as E cadherin, further accentuating the tendency to malignancy.[12]

The role of mucosal inflammation

Gastro-oesophageal metaplasia seems to be induced or potentiated by mucosal inflammation**.** Understanding the molecular changes in this process may mean that we can identify people at risk of developing malignancies. Identifying E cadherin mutations and interleukin 1β polymorphisms may make it possible to screen people who have known risk factors such as a strong family history or metaplasia or dysplasia. Now that there is evidence to implicate chronic inflammation in cancer development, the role of anti-inflammatory drugs such as cyclo-oxygenase-2 inhibitors or more specific cytokine inhibitors may provide a new impetus to medical intervention.[2]

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We thank Mr John Fielding, chairman of the NHS/Department of Health Upper GI Cancer Group, for his helpful comments during the writing of this manuscript.

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New system for GP recruitment

Should be fairer and more educationally relevant

It is still less than a quarter of a century since vocational training for general practice in the United Kingdom became compulsory, though informal training is as old as medicine itself. The present arrangements, which require registered medical practitioners to have two years' experience in certain specialties and a year of vocational training in a training practice, were satisfactory for most trainees who graduated from British medical schools. The arrangements lacked flexibility, however, and as new developments in general practice arrived, some cracks started to show. As a result directors of postgraduate general practice education have now taken full responsibility (including budgetary responsibility) for managing vocational training.[1 2]

One of the biggest cracks appeared with the advent of compulsory summative assessment.[3] A registrar who failed the process had to be referred to the secretary of state before an additional period of training time could be allowed—a cumbersome process. The new arrangements delegate this responsibility to directors of postgraduate education in general practice, which is sensible and welcome.

Linked to this is budgetary control. The money for general practice registrar training used to come from General Medical Services funds (the Red Book), but this budget has now been transferred to the medical and dental education levy. This will give directors the ability to enable general practice trainees to train flexibly and will also allow doctors who have acquired rights as European Union general practitioners to have a period of orientation training before starting practice in the United Kingdom. It will also enable periods of refresher training for doctors who have been out of practice for some time. These measures can also only be welcome, particularly if the government hopes to

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BMJ 2000;321:464–5