ORIGINAL ARTICLE

Recent trends in admissions and mortality due to peptic ulcer in England: increasing frequency of haemorrhage among older subjects

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Background: Although overall admission rates for peptic ulcer in England declined from the 1950s up until the mid 1980s, perforations among older women increased, possibly due to increasing use of non-steroidal anti-inflammatory drugs (NSAID). Since then, proton pump inhibitors, antibiotic treatment for *Helicobacter pylori*, low dose aspirin, and selective serotonin reuptake inhibitors (SSRI) have been introduced

Aims: To determine time trends for hospital admissions for peptic ulcer from 1989 to 1999 (England), mortality from 1958 to 1998 (England and Wales), and prescriptions for ulcer healing drugs, aspirin, NSAID, oral anticoagulants, and SSRI from 1990 to 1999 (England).

Methods: Hospital episode statistics for admissions and mortality were obtained from the Office of National Statistics: community prescription data from Statistics Division 1E of the Department of Health.

Results: Between 1989/90 and 1998/99, there was a marked rise in admissions for haemorrhage in older patients, particularly from duodenal ulcer. Perforations from gastric ulcer declined but perforations from duodenal ulcer increased among men at older ages. Since the mid 1980s mortality has declined in all age groups except for older women with duodenal ulcer. The number of prescriptions for histamine H_2 receptor antagonists remained constant but those for proton pump inhibitors increased by 5000%, aspirin 75mg by 460%, oral anticoagulants by 200%, and NSAID by 13% between 1990 and 1999. Since the introduction of SSRI in 1991, prescriptions have increased 15-fold.

Conclusions: Admission rates for gastric and duodenal ulcer haemorrhage and duodenal ulcer, but not gastric ulcer perforation, increased among older subjects, over a time when prescriptions for proton pump inhibitors, low dose aspirin, oral anticoagulants, and SSRI increased.

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T he story of peptic ulcer over the last 200 years has been one of profound changes in frequency and clinical presentation. In 1857, Brinton described gastric ulcer as a disease of young women[1]: duodenal ulcer seemed to be rare.[2] In the beginning of the 20th century however the prevalence of duodenal ulcer increased to several times that of gastric ulcer before starting to fall in the second half of the century.[2] Between the 1950s and 1980s, hospital admissions and mortality from peptic ulcer declined in Britain for most age groups.[3–5] In contrast with this general trend, admissions for perforated peptic ulcer and mortality from duodenal ulcer increased among older women in the 1970s and 1980s.[5–7] Similar patterns have been noted in other Western countries[8–10] although a recent Danish study showed that hospitalisation and mortality from peptic ulcer complications have increased, especially among the elderly.[11] This general decline in hospital admission rates and mortality from peptic ulcer has been attributed to a reduced prevalence of *Helicobacter pylori* infection,[10] and a recent review stated that: "Peptic ulcer seems to be on the way out".[9] The rise in mortality and admissions among older women in the 1970s and 1980s has been attributed to increased prescribing of non-steroidal anti-inflammatory drugs (NSAID), the other major cause of peptic ulcer, commonly used in older subjects.[5 6 12]

The introduction of effective antisecretory treatment with histamine H_2 receptor antagonists in the mid 1970s was associated with a reduction in elective surgery for peptic ulcer disease.[6 7 13] Since then two other major advances in peptic ulcer treatment have been introduced: proton pump inhibitors and antibiotic treatment to eradicate *H pylori*. A link has also been proposed between serotonin reuptake inhibitors (SSRI) and upper gastrointestinal bleeding.[14] We have therefore examined the statistics for mortality, admissions, operations,

and drug prescribing relating to peptic ulcer, to determine the latest time trends of these indices of peptic ulcer disease.

METHODS

Hospital admission rates (finished consultant episodes)

Although most patients with uncomplicated peptic ulcer would probably self medicate or be treated by their general practitioner, patients with ulcer complications, in particular perforation and severe haemorrhage, are admitted to hospital. Hospital admission rates can therefore be used as an index of peptic ulcer frequency although it is uncertain if the proportion of ulcers which perforate or bleed is constant while the threshold at which hospital admission occurs may vary according to the prevailing medical practice at the time.[2]

The Department of Health provided hospital episode statistics for England for 1989/90–1998/9. For the period 1989/90–1994/5, we identified finished consultant episodes with a primary diagnosis of ICD 9 codes 531, 532, 533, and 534. For the period 1995/6–1998/9 when ICD 10 codes were in use, we identified finished consultant episodes with a primary diagnosis of codes K25, K26, K27, and K28. Average annual age standardised rates for 1989/90–1991/2, 1992/3–1995/6, and 1996/7–1998/9 were calculated. These were standardised against the European standard population using the mid year population estimates for each year and 10 year age bands. Trends for finished consultant episodes in the age groups 25–44 years, 45–64 years, 65–74 years, and 75 years and over

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were examined. Day cases were excluded. Data for peptic ulcer site unspecified (ICD 9 code 533) was combined with gastric ulcer (ICD 9 code 531) to allow comparison with mortality rates. Peptic ulcer site unspecified made up less than 10% of the gastric ulcer total at the beginning of the period and less than 5% at the end, probably reflecting improvement in the accuracy of coding. Gastrojejunal ulcers (ICD 9 534 and ICD 10 K28) make up approximately 1% of all peptic ulcers and are not considered further in this analysis. We also studied admission rates for perforation and haemorrhage. Ulcers which both perforated and haemorrhaged were grouped with those which just perforated.

Finished consultant episodes relating to operations performed for peptic ulcer disease (OPCS 4 codes A27 (extracranial vagotomy), G28 (partial excision of stomach), G35 (operations on ulcer of stomach) and G52 (operations on ulcer of duodenum)) were examined for the period 1990/91–1998/99.

Mortality rates

Peptic ulcer is a disease of low mortality and most deaths occur as a result of complications in elderly patients with significant comorbidity. Mortality for peptic ulcer is therefore influenced not only by the frequency of ulcer disease but also by its severity, as well as the effectiveness of medical treatment.[2]

Deaths due to ICD 7 codes 540, 541, and 542 (1958–1967) and deaths due to ICD 8 and 9 codes 531, 532, 533, and 534 (1968–1998) were identified for England and Wales. These were standardised against the European standardised population using the mid year population estimates for the year and 10 year age bands. Trends for mortality in the age groups 45–64 years and 65 years and over were examined. Data for peptic ulcer site unspecified (ICD 8 and 9 codes 533) were combined with gastric ulcer (ICD 8 and 9 codes 531) to correspond with gastric ulcer and peptic ulcer site unspecified (ICD 7 code 540).

Prescribing

To explore the possible impact of drug prescribing on mortality and hospital admissions, we analysed information on the number of items dispensed in the community between 1990 and 1999, supplied by Statistics Division 1E of the Department of Health. Values for 1990 are based on fees and on a sample of 1 in 200 prescriptions dispensed by community pharmacists and appliance contractors only. Values from 1991 onwards are from the Prescription Cost Analysis system and are based on a full analysis of all prescriptions dispensed in the community— that is, by community pharmacists and appliance contractors, dispensing doctors, and prescriptions submitted by prescribing doctors for items personally administered in England. The data do not cover drugs dispensed in hospital or private prescriptions but most prescribing for chronic diseases in the UK is carried out by general practitioners and is picked up by the Prescription Cost Analysis system.

Information was provided for the number of items dispensed for ulcer healing drugs (BNF section 1.3), NSAID (BNF section 10.1.1), aspirin (BNF section 2.9 and 4.7.1), oral anticoagulants (BNF section 2.8.2), and SSRI (BNF section 4.3.3).

RESULTS

Hospital admissions

Finished consultant episodes for gastric ulcer and peptic ulcer not otherwise specified (England 1989/90–1998/9)

Overall, there was a slight rise in admission rates over this period (table 1). For age groups less than 65 years there was little change but admission rates increased among older groups. There was a 29% rise among women and a 40% rise among men in those aged more than 74 years. Similarly, admissions for haemorrhage remained stable for younger patients but increased for older age groups. For those aged 75 and over, admission rates rose by 30% among women and by 41% among men.

There was a general decline in admission rates for gastric ulcer perforation. The decline was up to 31% among elderly men (aged 75 and over) over a period when gastric ulcer admissions overall and admissions for gastric ulcer haemorrhage increased.

Finished consultant episodes for duodenal ulceration (England 1989/90–1998/9)

Although there was little overall change in admission rates for duodenal ulcer, there was a decline for younger patients and a substantial rise for older individuals (table 2). Admission rates increased by 33% among women aged more than 74 years and by 49% among elderly men. Much of this rise related to admissions for haemorrhage. For the 65–74 year age group there was a 48% rise among women and 45% rise among men. In the age group more than 74 years, rates rose by 50% and 65%, respectively. Overall, hospitalisation rates for haemorrhage increased by 25% among women and by 13% among men.

Overall perforation rates remained stable among women but declined slightly among men. The latter was due to a

Table 1 Average annual age standardised rate for finished consultant episodes for gastric ulcer and peptic ulcer not otherwise specified, in England from 1989/90 to 1998/9. Age standardised within age bands to European standard population. Finished consultant episodes per 100 000

		Women				Men			
	Age group (y)	1989–91	1992–5	1996–8	% Change	1989–91	1992–5	1996–8	% Change
Gastric ulcer and peptic ulcer NOS	25–44	8.4	8.3	8.6	2	14.6	14.5	14.4	−1
	45–64	35.4	30.9	32.1	−9	50.0	49.2	47.5	−5
	65–74	95.5	95.2	106.8	12	96.2	108.5	122.7	28
	75+	107.2	129.7	138.1	29	95.1	116.3	133.6	40
	All ages	22.8	22.6	24.2	6	28.5	30.0	31.1	9
Gastric ulcer and peptic ulcer NOS: haemorrhage	25–44	2.0	1.9	2.2	9	4.7	5.2	5.1	8
	45–64	11.7	11.6	11.0	−6	19.7	21.4	18.9	−4
	65–74	36.0	39.6	41.0	14	40.4	51.1	55.2	36
	75+	43.1	57.0	56.1	30	41.9	54.0	59.2	41
	All ages	7.9	8.7	8.7	10	11.1	13.0	12.8	15
Gastric ulcer and peptic ulcer NOS: perforation	25–44	0.7	0.5	0.6	−8	0.8	1.0	1.0	20
	45–64	2.9	2.7	2.5	−15	3.9	3.4	3.3	−17
	65–74	9.0	8.5	8.7	−3	7.7	7.7	7.0	−9
	75+	12.2	12.1	10.7	−12	10.4	9.3	7.2	−31
	All ages	2.1	1.9	1.9	−10	2.3	2.1	2.0	−13

NOS, Not otherwise specified.
% Change, percentage change from 1989–91 to 1996–8.

Table 2 Average annual age standardised rate for finished consultant episodes for duodenal ulceration in England from 1989/90 to 1998/9. Age standardised within age bands to European standard population. Finished consultant episodes per 100 000

		Women				Men			
	Age group (y)	1989–91	1992–5	1996–8	% Change	1989–91	1992–5	1996–8	% Change
Duodenal ulcer	25–44	10.7	8.6	8.3	–22	33.4	29.8	26.1	–22
	45–64	29.9	26.7	24.6	–18	71.9	65.9	61.7	–14
	65–74	65.2	72.2	81.7	25	118.7	133.3	150.1	26
	75+	84.0	105.1	111.4	33	112.4	149.6	167.1	49
	All ages	19.1	19.0	19.3	1	44.1	43.3	42.6	–3
Duodenal ulcer: haemorrhage	25–44	1.9	2.1	2.0	7	10.3	11.3	9.6	–6
	45–64	9.2	8.9	9.3	1	27.6	28.6	26.7	–3
	65–74	22.2	27.9	33.0	48	49.4	63.4	71.8	45
	75+	30.8	41.4	46.3	50	48.4	71.7	80.0	65
	All ages	5.7	6.5	7.2	25	16.3	18.8	18.5	13
Duodenal ulcer: perforation	25–44	2.0	1.8	2.0	1	5.3	5.2	5.1	–4
	45–64	6.9	6.5	5.9	–14	13.9	12.3	11.4	–18
	65–74	18.0	20.0	20.6	14	25.6	26.5	24.2	–6
	75+	25.5	28.8	26.2	3	24.3	29.0	29.5	21
	All ages	4.7	4.8	4.7	0	8.4	8.2	7.8	–7

% Change, percentage change from 1989–91 to 1996–8.

decline in admissions for perforation among younger men despite a 21% rise in the 74 years and over age group.

Operations
Finished consultant episodes for operations (England 1989/90–1998/9)

The main indication for vagotomy (A27) is duodenal ulcer. Partial gastrectomy (G28) in contrast is performed both for gastric and duodenal ulcer disease as well as for gastric neoplasms. Vagotomy (A27) rates fell by about 83% between 1990/1–1992/3 and 1996/7–1998/9) (table 3). Over the same period, partial gastrectomy rates declined by about 30%. The frequency of simpler operations, including oversewing of bleeding or perforated gastric or duodenal ulcers (G35 and G52), increased, particularly for duodenal ulcer in younger ages. Overall operation rates changed little when the four categories were taken together.

Mortality rates
Mortality from gastric ulcer and peptic ulcer site unspecified (England and Wales)

Mortality rates from gastric ulcer were highest in the elderly, being about 20 times greater among patients aged 65 years and over compared with those aged 35–64 years (fig 1). There was a general decrease in mortality from 1958 among most age groups. A 2:1 male predominance in mortality rates among those aged 65 years and over in 1958 declined to near unity by the mid 1980s.

Mortality from duodenal ulcer (England and Wales)

Mortality among women aged 65 years and over doubled between 1970 and 1986 but has since stabilised (fig 2). For men aged 65 years and over, mortality steadily declined, the decrease slowing after the mid 1980s. Mortality from duodenal ulcer declined by 75% in the younger age groups over this period.

Table 3 Average annual age standardised rate for finished consultant episodes for operations in England from 1989/90 to 1998/9. Age standardised within age bands to European standard population. Finished consultant episodes per 100 000

	Age group (y)	Women				Men			
		90/1–92/3	93/4–95/6	96/7–98/9	% Change	90/1–92/3	93/4–95/6	96/7–98/9	% Change
A27 Vagotomy	25–44	1.0	0.4	0.1	–87	3.0	1.1	0.4	–88
	45–64	2.3	0.9	0.4	–83	4.8	2.2	0.8	–84
	65–74	3.6	2.0	0.9	–76	5.9	3.3	1.6	–73
	75+	2.8	2.0	0.5	–82	3.5	2.7	0.9	–73
	All ages	1.3	0.6	0.2	–83	2.8	1.3	0.5	–84
G28 Partial gastrectomy	25–44	1.1	0.9	0.8	–30	1.6	1.3	1.0	–40
	45–64	7.0	5.3	4.5	–36	12.5	9.9	7.5	–40
	65–74	16.4	14.6	13.3	–19	26.9	24.0	20.9	–22
	75+	11.9	11.1	9.1	–24	16.4	15.0	13.0	–21
	All ages	3.7	3.1	2.6	–29	6.2	5.2	4.1	–33
G35 Gastric ulcer operations	25–44	0.4	0.4	0.4	24	0.8	0.8	0.9	12
	45–64	1.7	1.6	1.8	2	2.6	2.4	2.6	1
	65–74	5.2	5.9	6.1	17	5.4	5.8	5.7	5
	75+	6.2	6.9	6.7	8	5.0	5.8	4.4	–12
	All ages	1.2	1.2	1.3	11	1.5	1.6	1.6	3
G58 Duodenal ulcer operations	25–44	0.9	1.2	1.6	73	2.5	3.7	4.1	61
	45–64	3.5	4.3	4.3	22	6.4	8.6	8.8	38
	65–74	9.1	11.5	14.0	55	11.8	18.0	18.6	58
	75+	11.2	16.6	15.9	42	11.7	17.1	18.4	57
	All ages	2.3	3.0	3.2	43	3.9	5.6	5.9	52

% Change, percentage change between 1990/1–1992/3 and 1996/7–1998/9.
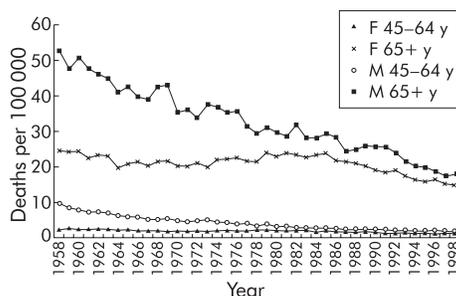
Figure 1 Age specific mortality rates for males (M) and females (F) for gastric ulcer and peptic ulcer site unspecified in England and Wales (standardised to European standard population).
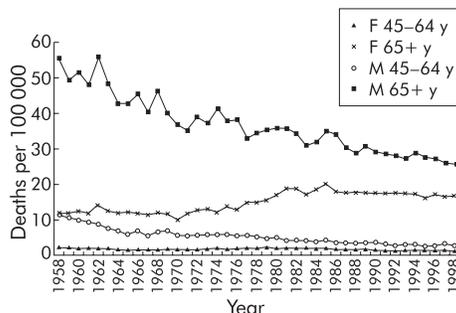
Figure 2 Age specific mortality rates for males (M) and females (F) for duodenal ulcer in England and Wales (standardised to European standard population).

Prescribing

Number of items prescribed in England from 1990 to 1999

The number of prescriptions for ulcer healing drugs increased two and a half times between 1990 and 1999 (table 4). Prescriptions for H_2 receptor antagonists rose early in the period but subsequently declined to about the same level as in 1990. Proton pump inhibitors started to be prescribed from 1990 and now make up about 60% of drugs prescribed.

Between 1990 and 1999, prescriptions for NSAID increased by about 13%. Prescribing of oral anticoagulants increased by 200% and aspirin 75 mg increased by 460% between 1990 and 1999. In contrast, there has been little change in prescriptions for aspirin 300 mg. SSRI were introduced in 1991 and prescriptions increased 15-fold by 1998.

DISCUSSION

We examined hospital admissions and mortality due to peptic ulcer in England for the period 1989 to 1998 to see if previously described trends had continued. In particular, we were interested to see if there was any change that might be attributable to the introduction of proton pump inhibitors and antibiotic treatment for *H pylori*. Although there was a continuing general decline in hospital admissions for peptic ulcer among younger individuals, the pattern has changed for the elderly. The rise in perforation in women aged 65 years and over has not continued. Instead there has been a general rise in haemorrhage in both sexes, particularly for duodenal ulcer. In contrast, mortality rates have declined in all groups since 1986 except for women aged 65 years and over with duodenal ulcer, among whom the rates have remained stable. This may reflect improvement in medical treatment.

During this period, there has been a general decline in definitive surgery for peptic ulceration. Partial gastrectomy rates have decreased by 30% and vagotomy rates by 83%. There was a corresponding rise in simpler procedures for haemorrhage and perforation. Presumably, surgeons are now oversewing ulcers but relying on subsequent medical treatment to actually heal the ulcers and cure the ulcer diathesis.

Earlier studies of hospital admissions for peptic ulcer in England and Wales between 1958 and 1982 showed a general decline except among women aged 65 years and over. In this group there was a rise in perforations, especially for duodenal ulcer.[3–5] In Scotland between 1975 and 1990, overall admissions for duodenal ulcer declined although again there was an increase in perforated duodenal ulcers among women aged 65 and over.[6] There was a general decline across all ages in admissions for duodenal ulcer haemorrhage.

The advent of effective medical treatment for peptic ulcer since the 1970s may have been expected to reduce the frequency of hospital admissions. However, these advances have occurred at a time when drugs which may cause ulcers are increasingly used—for example, NSAID from the 1970s and low dose aspirin in the 1990s. The effect of H_2 receptor antagonists, the first effective ulcer healing drugs to be introduced, on hospital admission rates for peptic ulcer is uncertain. In the Trent region in England, there was a reduction in waiting list admissions and operations for uncomplicated duodenal ulcer in the five years following their introduction.[7] However, overall emergency admissions for uncomplicated, bleeding, and perforated duodenal ulcer remained unchanged. Indeed, admission rates for bleeding and perforation increased among the elderly.[7] In Tayside, Scotland, long term continuous therapy with H_2 receptor antagonists for peptic ulcer was used much more widely than in the rest of the country. Hospital admissions for peptic ulcer and for ulcer haemorrhage declined significantly in the 1980s while little change was seen in Scotland as a whole.[13] A composite analysis of various indices of peptic ulcer disease in six Western countries in relation to the introduction of histamine H_2 antagonists showed an effect on work loss and disability rather than hospitalisation and mortality.[15] In Denmark, hospitalisation and mortality rates from peptic ulcer complications increased, especially among the elderly, from 1981 to 1993, even with the introduction of histamine H_2 antagonists and proton pump inhibitors.[11] We have also demonstrated an increase in hospital admissions for ulcer haemorrhage among older patients in England, despite the introduction of proton pump inhibitors and curative treatment for *H pylori*, as

Table 4 Number of items prescribed in England from 1990 to 1999. Prescription Cost Analysis system (Department of Health)

	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999	% Change
Aspirin 75 mg	1930	2851	3776	4691	5896	6472	7737	8638	9781	10 848	462
Aspirin 300 mg	1219	822	797	764	1548	1517	1501	1452	1367	1203	−1
Oral anticoagualants	1153	1347	1477	1665	1958	2246	2589	2938	3312	3698	221
Non-steroidal anti-inflammatory drugs	16 387	17 397	17 973	18 080	17 894	17 543	17 722	18 047	18 297	18 534	13
Selective serotonin reuptake inhibitors	—	510	1178	1885	2681	3808	5136	6556	7582	8929	1651
H_2 receptor antagonists	6201	7149	7534	7765	7864	7659	7547	7208	6846	6455	4
Proton pump inhibitors	182	465	1163	2095	3230	4484	5691	6812	8143	9527	5126

% Change, percentage change between 1990 and 1999.

well as a 2.5-fold increase in prescriptions for ulcer healing drugs between 1990 and 1999. The relationship of acid suppressant drug prescribing to the frequency of peptic ulcer is complex. In addition to healing peptic ulcers, they are also coprescribed with NSAID for ulcer prophylaxis and widely used for functional dyspepsia and for gastro-oesophageal reflux.

This was an ecological study based on aggregate data—that is, using groups rather than individuals as the unit of analysis. Observations of groups may not allow causal inferences to be drawn about the individual, a problem known as the "ecological fallacy".[16] The number of tablets of, for example, an NSAID which are prescribed may not be directly translated into its biological activity because of differences in dosage schedules, patient compliance, patient selection for drug prescription, coprescription of gastroprotective drugs, and so on. On the other hand, a therapeutic modality may have an effect on subjects other than those for whom the drug was prescribed. For example, widespread use of anti-*H pylori* treatment may reduce the reservoir of disease in the community and secondarily reduce infection rates among those not previously exposed to the pathogen. Although aspirin, anticoagulants, and NSAID drugs are known to promote peptic ulcer disease and its complications, while anti-*H pylori* treatment is known to have a beneficial effect, our ability to draw inferences regarding cause and effect on temporal trends is necessarily limited in an ecological analysis such as this.

However, our finding of a decrease in hospital admissions for peptic ulcer among younger individuals but an increasing frequency among older individuals would be consistent with the effect of a decline in *H pylori* infection concurrently with an increase in the use of ulcerogenic drugs. The former may be expected to affect younger subjects while older individuals would be more likely to receive NSAID, aspirin, and antidepressant drugs. The increase in peptic ulcer admissions in the elderly is unlikely to be due to a greater prevalence of *H pylori* infection in older patients as the birth cohort with the highest prevalence of *H pylori* infection was those born around 1910.[17] *H pylori* prevalence in cohorts of subjects born after that time would therefore be expected to be progressively lower.

It has been estimated that for every 1000 patients on vascular prophylaxis with aspirin, one or two per year will have a gastrointestinal bleed.[18] There were approximately nine million more prescriptions for 75 mg tablets of aspirin in 1999 compared with 1990. If each prescription was for 30 tablets and one 75 mg tablet was taken each day, there would have been 900 000 more person years of exposure in 1999. The 900–1800 more episodes of gastrointestinal bleeding which would be expected approximates the 1000 excess admissions actually observed.

Hospital episode statistics (started in 1987) are based on finished consultant episodes rather than admissions. Rates derived from them cannot therefore be compared directly with earlier studies that were based on the 10% sample of admissions from the hospital inpatient enquiry, the collection of which ended in 1985.[3–5] However, these data are robust, including all finished episodes rather than the 10% sample in the hospital inpatient enquiry. Ninety five per cent of admissions generate a single finished consultant episode although a minority generate multiple consultant episodes during an admission. We feel therefore that the trends demonstrated within the period 1989–1999 can be compared with those observed in previous studies based on data from the hospital inpatient enquiry. Furthermore, the diagnoses recorded on hospital episode statistics have been shown to be accurate, thus reducing the risk of misclassification errors.[19] Any coding inaccuracies should even out in a study of trends. There has been no major change in diagnostic modalities for peptic ulcer disease over the period under study. According to Rockall and colleagues,[20] the incidence of upper gastrointestinal haemorrhage as a reason for admission (not including haemorrhage occurring in hospital) is approximately 103/100 000 adults/year, or 82/100 000 population/year for males. Peptic ulcer accounted for approxi-

mately 35% of these, or 29/100 000/year. This is very close to our values of 31.8/100 000/year for 1992–5 (duodenal ulcer 18.8, gastric ulcer 13.0). For women, our values were 15.2/100 000/year compared with 20.0/100 000/year based on Rockall's data. We were unable to assess the impact of changes in lifestyle factors such as alcohol intake and smoking habits, which have been shown to be risk factors for peptic ulcer complications.[21] However, these would have changed relatively little during the period of this study.

In conclusion, while peptic ulcer admissions and mortality have declined in frequency among young individuals, admission rates for gastric and duodenal ulcer haemorrhage, and duodenal ulcer perforation, have increased among older patients. Further work is needed to establish the reasons for these different time trends for haemorrhage and perforation, for the young and old, for gastric and duodenal ulcer, and the relative contribution of various drugs, both ulcer causing and ulcer healing, to the pattern of peptic ulcer disease in our population.

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PostScript ....

Osteoporosis and liver disease: additional reasons for coeliac disease screening

We read with great interest the recently published guidelines on the management of osteoporosis associated with chronic liver disease (*Gut* 2002;**50**(suppl I):i1–9). However, we would like to add a few words of comment. Associations between coeliac disease (CD) and primary biliary cirrhosis in particular and other autoimmune liver diseases in general have been reported.[1–3] In addition, it has been suggested that these individuals should be considered as an at risk group for whom serological testing for CD is indicated.[1] Patients with CD are at high risk of developing low bone mineral density and bone turnover impairment,[1] and it has been shown that adherence to a gluten free diet has a significant positive impact on these parameters.[4] Thus we suggest that physicians caring for patients with the above mentioned liver diseases should screen them for CD in the presence of signs and symptoms suggestive of malabsorption such as osteoporosis. This seems a reasonable strategy as detection of CD will allow for a more rational therapeutic approach to the risks determined by this association. Complications due to the presence of CD, such as malnutrition, anaemia, and osteoporosis, may have a considerable impact on liver disease management and the need/success of transplantation.[5 6]

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Caerulein induced pancreatitis

We have read with interest the article by Frossard *et al* (*Gut* 2002;**50**:78–83) entitled "Both thermal and non-thermal stress protect against caerulein induced pancreatitis and prevent trypsinogen activation in the pancreas". We have a few comments with regard to the interpretation of the data that were obtained in this experiment.

Previous experimental work by Frossard and others have implicated HSP70 as playing a protective role in caerulein induced acute pancreatitis. In the present study Frossard *et al* showed that thermal and non-thermal stress induced by injection of the β agonist isoproterenol upregulated HSP70 in the pancreas which is associated with amelioration of subsequently induced caerulein pancreatitis. The authors hypothesise that the protective effects on pancreatitis severity caused by thermal and non-thermal stress may by mediated by HSP70. We believe however that both heat shock stress and non-thermal stress can stimulate several other anti-inflammatory pathways which were not discussed in this study, all of which could be alternative explanations for the observations that were made.

It is widely established that catecholamines, both endogenously released during heat shock stress or by injection of isoproterenol, can influence activation of inflammatory pathways during inflammation and infection[1] (reviewed by van der Poll[2]). Evidence exists that catecholamines exert anti-inflammatory effects on a number of host mediator systems, such as the cytokine network and neutrophils, all of which are implicated in the pathogenesis of acute pancreatitis and the pancreatitis associated systemic inflammatory response syndrome. Catecholamines, either endogenously produced or exogenously administered, may act to dampen excessive pro-inflammatory pathways by mechanisms not related to enhanced production of heat shock proteins. Firstly, catecholamines exert anti-inflammatory effects on the cytokine network by inhibiting the production of proinflammatory cytokines such as tumour necrosis factor (TNF), interleukin (IL)-1β, IL-12, and interferon γ (IFN-γ), of which TNF and IL-1β have been implicated as mediators that play a proinflammatory role in acute pancreatitis.[3 4] Secondly, in animal models of endotoxaemia, pretreatment with isoproterenol enhances the production of the anti-inflammatory cytokine IL-10 which has been shown to be protective in acute pancreatitis.[5 6] Thirdly, in endotoxaemia models, β adrenergic stimulation results in reduction of levels of CC chemokines.[7] Fourthly, neutrophil migration to the pancreas, one of the hallmarks of acute pancreatitis, towards chemotactic stimuli such as C5a and lipopolysaccharide (LPS) is reduced by administration of β agonists but also affects LPS induced neutrophil degranulation in vivo. Fifthly, with regard to the hypothesis that HSP70 prevents the activation of trypsinogen in the pancreas, it must be noted that recent evidence suggests that neutrophils and possibly cytokines can also influence trypsinogen activation. Therefore, the reduction in trypsinogen activation shown in their study might be unrelated to HSP expression and may be explained by the reduction of inflammation due to β adrenergic effects.[8 9]

Therefore, we believe that the conclusion by Frossard *et al* that the protective effects of thermal and non-thermal stress might be mediated by HSP70 is only one possible explanation and that their observations might also be explained by the immunomodulatory effects of catecholamines.

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Authors' reply

We thank van Westerloo *et al* for their interest in our paper and their comments on the interpretation of our data. They are of the opinion that besides heat shock proteins both thermal and non-thermal stress can stimulate several other anti-inflammatory pathways that in turn could be responsible for the protective effects observed in the study. Secondly, catecholamines can exert anti-inflammatory effects independent of heat shock proteins.

When we embarked on this project, we were also concerned that all the stresses that result in the induction of HSP70 may have other non-HSP related effects and did mention this in our discussion. At that point we did not have the tools to show the crucial protective role played by HSP70.

To prove that a cause-effect relationship exists between HSP70 expression and protection against pancreatitis, we adopted the antisense oligonucleotide approach in another recently published experimental study[1] to indicate unequivocally that the thermal stress induced protection of intrapancreatic trypsinogen activation and protection against caerulein induced pancreatitis are mediated by HSP70. Furthermore, our studies have shown that HSP70 induction that occurs during the evolution of pancreatitis in non-thermally stressed rats acts to limit the severity of pancreatitis.

Using antisense oligonucleotides to HSP70, Nisoli and colleagues[2] have also shown that the protective effects of noradrenaline against tumour necrosis factor α induced apoptosis in cultured rat brown adipocytes is due to nitric oxide induced HSP70 expression. In fact, catecholamines have been used in the past to induce heat shock proteins in several experimental systems.[3] [4]

Westerloo *et al* have cited examples wherein exogenous or endogenous catecholamines inhibit the production of inflammatory cytokines and enhance the production of interleukin 10 (IL-10), an anti-inflammatory cytokine that has been shown to limit the severity of pancreatitis. Unfortunately, in the studies cited expression of HSP70 was not monitored. It is entirely possible that prior thermal or non-thermal stress induce HSP70 which may in turn lead to the enhanced production of anti-inflammatory factors and attenuation of proinflammatory cytokines. Indeed this has been shown to be the case in many experimental systems, including animal models of sepsis (reviewed by Bruemmer-Smith and colleagues[5]). Moreover, mycobacterial HSP70 has been shown to prevent adjuvant arthritis and induce IL-10 producing T cells.[6]

The mechanism(s) by which HSP70 might protect against caerulein induced pancreatitis is not yet known. Experiments examining the relationship between HSP70 and the inflammatory cascade induction during caerulein induced pancreatitis are currently underway.

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Pathology and cost effectiveness of endoscopy surveillance for premalignant gastric lesions

We read with great interest the article by Whiting *et al* (*Gut* 2002;**50**:378–81). The Birmingham experience shows how the prevalence of gastric cancers detected at an early stage is significantly higher in the endoscopically surveyed population than in non-surveyed patients. As a result, this study demonstrates that, in the secondary prevention of gastric malignancy, the "once in a lifetime" strategy (suggested for colorectal cancer) is not cost effective while repeated endoscopies (in selected patients) seem most appropriate. This conclusion however raises two cardinal questions. Firstly, are there "special" requirements (that is, a protocol of gastric biopsy sampling) to be satisfied when carrying out the upper endoscopy procedure? Secondly, are there evidence based criteria for selecting patients to be included in surveillance programmes?

The authors do not provide detailed information on the number of biopsy samples obtained per endoscopy. We belive that a standardised protocol of biopsy sampling is a leading part of any upper endoscopy procedure, and mucosal "abnormalities" should be considered the targets of additional sampling.[1] Taking into account that 46% of the cancers referred to in Whiting's study were discovered within 13 months from the second last procedure, we agree with the authors who considered these cancers endoscopically missed.

The second point of concern is the rationale for a surveillance protocol. Any cost effective strategy of secondary cancer prevention requires the risk of cancer to be higher within subjects undergoing surveillance than in the general population. In the Birmingham study, such a prerequisite does not seem to have been assumed in the study design. Endoscopy surveillance definitively included the whole spectrum of abnormalities described in gastric carcinogenesis,[2] [3] from regenerative changes (with a nearly null cancer risk) to non-invasive neoplasia (which carries, by definition, predisposition for progression to invasion and metastasis). As a consequence of (i) cancers missed at endoscopy and (ii) shortness of the follow up period, the results shown in their table 3 may be misleading. The association of intestinal metaplasia (regardless of its histochemical phenotype) with the highest risk of cancer evolution is not only biologically questionable but, and this is even worse, it may result in inappropriate patient management. While intestinal metaplasia represents the most common background of stomach cancer,[4] [5] "gastric intestinalisation" per se does not carry the phenotypic and genotypic alterations pre-currying invasive neoplasm. Most importantly, the high prevalence of metaplastic lesions within subjects who will never develop adenocarcinoma exclude (non-extensive) intestinal metaplasia as the proper target of surveillance programmes.

In the natural history of epithelial tumours, the term dysplasia identifies a lesion that carries biological alterations comparable with those of full fledged cancer but lacking stromal invasion.[2] Recently, the term "dysplasia" has been replaced by "non-invasive neoplasia", which more clearly identifies such a lesion as the most advanced alteration antecedent to invasive adenocarcinoma.[6] Since 1985, we have prospectively followed up a

Table 1 Invasive cancer detected during follow up of non-invasive gastric neoplasia

Histology at enrollment	Gastric cancer detected after follow up longer than 12 months				Gastric cancer detected within 12 months from initial diagnosis			
	Follow up (months)*	EGC	AGC	GC-nos	Follow up (months)*	EGC	AGC	GC-nos
Low grade non-invasive neoplasia (99 cases)	48 (38–80)	5	1	2	1.5 (1–2)	1	1	0
High grade non-invasive neoplasia (25 cases)	30 (13–72)	7	1	3	2 (1–4)	6	3	0
Total	35 (13–80)	19			1.7 (1–4)	11		

EGC, early gastric cancer (that is, UICC pathological stage I); AGC, advanced gastric cancer (that is, UICC pathological stages II-II-III); GC-nos, gastric cancer of unknown pathological stage.
*Mean (range).

series of patients with low and high grade gastric non-invasive neoplasia.[4] The follow up schedule was differentiated a priori, depending on the cancer risk presumably associated with each grade of lesion.[7] The number of patients enrolled in each diagnostic category, follow up time, and number of cancers detected are shown in table 1. It is worth emphasising the high prevalence of early gastric cancers (77%) among the 30 cases of cancer detected in our prospective follow up study. The 19 cases of cancer detected in the long term follow up support the premalignant significance of non-invasive neoplasia while the 11 cases detected within one year from the original endoscopy fully demonstrated that non-invasive neoplasia frequently coexists with advanced cancer. Both of these observations could represent valid foundations in drawing a surveillance programme aimed at secondary gastric cancer prevention.

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Adult height in patients with early onset of Crohn's disease

Alemzadeh *et al* (*Gut* 2002;**51**;26–9) reported that adult height, compared with the general Dutch population, was reduced by a mean of −0.9 SDS (95% confidence interval −1.55 to −0.28) in 15 Crohn's patients with prepubertal onset of symptoms. However, the calculated deviation from "target" height (based on parental height) did not reach statistical significance and the authors have speculated that familial short stature, and not Crohn's disease, may be a factor in this group. Furthermore, no height deficit was found in those with postpubertal onset of symptoms.

We are currently undertaking a review of those with childhood onset diseases attending our paediatric and adult IBD clinics. In the majority of cases parental height was measured by trained auxologists, although in some, details were not available in the case notes to discern the method of measurement and may therefore have included self reported parental heights. We calculated SDS scores from the revised British Longitudinal standards[1] using the method described by Alemzadeh *et al* (mean British male adult height of 176.0 cm (SD 6.3) and female adult height of 163.6 (SD 5.7)). "Target height" was calculated for male patients by (paternal height+(maternal height+13))/2 and for female patients by (maternal height+(paternal height−13))/2 (cm). "Prepubertal" children were defined as males and females with onset of symptoms at <13 and <11 years, respectively. We defined the upper limit of "postpuberty" as 16 years, in contrast with Alemzadeh *et al* who used 22 years. The population we serve is ethnically diverse and therefore we have confined this analysis to Caucasians, as per the Dutch study.

There was no deficit in height of the parents (48 mothers and 46 fathers) of children with childhood onset Crohn's disease compared with the general population. Furthermore, there was no deficit in height when examined by onset of their child's Crohn's disease: mean "prepubertal" parental SDS 0.00 (SD 1.11) and mean "postpubertal" parental SDS 0.08 (SD 1.12) (n=70 and n=24, respectively; p=0.34). In addition, we found no significant sex difference: mean paternal SDS −0.20 (SD 0.98) and mean maternal SDS 0.03 (SD 1.22) (n=46 and n=48, respectively; p=0.94).

In 27 cases (18 males, nine females) children of these parents are now aged more than 16 years (mean age 19.3 (SD 2.6) years). An analysis of final height is presented in table 1. A separate analysis was also carried out using the nomogram of the Child Growth Foundation[2] which corrects height until the age of 22, but this did not alter our findings and the data are therefore not presented.

In the majority of patients (85% (23/27); p=0.008, χ^2 test) final height was less than "target height", and in 22% (6/27) the final height deficit was more than 10 cm. For those aged over 18 years the values were 88% ((14/16); p=0.022, χ^2 test) and 25% (4/16), respectively.

In this sample of patients with childhood onset Crohn's disease we found no evidence of a familial basis for short stature. Our data confirm the findings of others that mean adult height of patients with onset of symptoms before the age of 16 is reduced.[3 4] Using age of onset of symptoms as a proxy for puberty, we found no significant difference in final height between those with pre- and postpubertal onset of symptoms. This is in contrast with the findings of Alemzadeh *et al* and may be because of differences in the upper age limit of "postpuberty" (16 versus 22 years).

Growth failure remains a concern to our British Crohn's patients and although the mean deficit of 5–6 cm from target height may be considered by some to be clinically inconsequential this includes a subset with much more significant growth impairment. A better understanding of the mechanisms underlying growth failure is required to determine whether there is an identifiable group of children that may benefit from early and more intensive immunosupression and/or nutritional therapy.[5 6]

We agree with Alemzadeh *et al* that only larger (population based) studies will have the power to determine the effect of factors such as site of disease activity and therapeutic intervention.

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Table 1 Final height of Caucasian Crohn's patients with pre and postpubertal onset of symptoms

	All	Prepubertal	Postpubertal	Pre v post (t test)
>16 years at last height				
SDS	−0.68 (−1.17 to −0.19)	−0.57 (−1.16 to 0.02)	−1.01 (−1.86 to −0.16)	p=0.41
Deficit (cm)	−5.9 (−8.2 to −3.4)	−5.5 (−8.6 to −2.4)	−7.0 (−8.8 to −5.3)	p=0.62
n	27	20	7	
>18 years at last height				
SDS	−0.73 (−1.42 to −0.04)	−0.65 (−1.48 to 0.18)	−1.07 (−2.12 to −0.02)	p=0.58
Deficit (cm)	−5.3 (−8.6 to −2.0)	−5.1 (−8.9 to −1.38)	−6.2 (−11.08 to −1.38)	p=0.84
n	16	13	3	

Data are mean (95% confidence interval).

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Author's reply

I think that this study is clear. We agree with the authors that the differences are possibly caused by the other "end point" of puberty and the small population in both studies. Another difference could be that the authors did not calculate the corrected height SDS (height SDS target height SDS); this may be lower for the prepubertal group compared with the postpubertal group. Furthermore, they used another formula for target height. This formula does not include a correction for secular trend which will underestimate the deficit. Also, there is no information on the effect of corticosteroid use.

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Underdiagnosis of hereditary haemochromatosis: reflects lack of clinical not biochemical penetrance

In their paper, Ryan *et al* (*Gut* 2002;**51**:108–12) reported that 78% of men (mean age 42 years) and 36% of women (mean age 39 years) who were identified to be C282Y homozygotes by family screening had evidence of biochemical iron overload. They concluded that underdiagnosis of hereditary haemochromatosis may be the result of failure to diagnose the phenotype in patients with iron overload.

In Glasgow, the prevalence of the C282Y homozygous state is high at approximately 1 in 180 of the population, of whom only 5.1% had been diagnosed by August 2001.[1] Of these known cases we identified 42 (20 males) C282Y homozygotes who had been diagnosed by predictive genetic testing of family members of affected probands. At diagnosis, all 20 males (mean age 46) had evidence of biochemical iron overload, defined as a transferrin saturation of ≥45% (transferrin detected immunochemically) or a serum ferritin of ≥300 μg. Both parameters were elevated in 15 (75%) individuals, with three having an isolated elevated transferrin saturation and two an isolated elevated ferritin.

Of the 22 females (mean age 44) identified, 18 (81%) had evidence of biochemical iron overload at diagnosis, with 10 (45%) having raised transferrin saturation and ferritin, as defined above. A further seven patients had an isolated elevation in transferrin saturation

and one had an elevated ferritin alone. Only four (9.5%) C282Y homozygotes identified by family testing had no evidence of biochemical iron overload. All of these individuals were female (age range 17–48 years). Unfortunately, due to the retrospective nature of the analysis, it was not possible to assess symptoms at diagnosis.

The prevalence of biochemical iron overload in our predominantly Celtic population is high and comparable with that reported from Dublin by Ryan *et al*. However, the proportion that will develop clinical "disease" related to hereditary haemochromatosis remains uncertain. Ryan *et al* proposed that underdiagnosis of hereditary haemochromatosis might be due to the non-specific nature of the symptoms early in the disease. They noted that fatigue, arthropathy, and male impotence were common complaints in these C282Y homozygotes identified by family screening. However, they provided no evidence that these symptoms were due to iron excess as they appeared to be as common in their biochemically non-expressing control group. It would be interesting to know whether any of these non-specific symptoms improved with phlebotomy.

In a recent large population screening study from the USA, Beutler *et al* reported the prevalence of biochemical iron overload in C282Y homozygotes to be similar to that observed by the Dublin group and ourselves. However, they found no evidence of more frequent symptoms in C282Y homozygotes compared with controls, even if biochemical iron overload was present.[2] It appears that these individuals have iron overload and a number of unrelated non-specific symptoms, similar to those seen in the general population. Prospective longitudinal studies are required to establish the proportion of C282Y homozygotes who will eventually exhibit the clinical phenotype of hereditary haemochromatosis.

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Gastrointestinal epithelial neoplasia

We read with interest the viewpoint "Gastrointestinal epithelial neoplasia: Vienna revisited" by Dixon (*Gut* 2002;**51**:130–1).

For many years Western gastrointestinal pathologists have followed the recommendations of British gastrointestinal pathologists. We learned that terms such as *carcinoma in situ* should be banned from the diagnostic terminology as it could lead to misinterpretation by surgeons and to unnecessary surgical intervention.

The Vienna classification[1] has introduced new avenues to the understanding of the

process of carcinogenesis in the gastrointestinal tract. For some Western pathologists in the Vienna group who also received histopathological training in Japan, the concept of intraepithelial carcinoma (that is, carcinoma in situ) and of intramucosal carcinoma appeared natural. Although during the first day of discussions other Western pathologists appeared reluctant to accept such controversial notions, the discussion became less intense during the second day, and at the end a consensus was reached, gaining finally the pages of this journal.[1]

The Vienna classification[1] dismembered the concept of dysplasia from that of carcinoma in its earlier forms. After many years of studying adenomas we now know that low grade dysplasia may progress to high grade dysplasia. On the other hand it remains elusive whether carcinoma in situ is preceded by high grade dysplasia or develops without a prodromic phase. By the same token we do not know whether carcinoma in situ antedates intramucosal carcinoma. If those microscopic realities of colorectal carcinogenesis are being ignored, how are we going to learn in a correct sequential fashion the intricate molecular footsteps that telescope from dysplasia to submucosal carcinoma? As that Pandora box is being presented to pathologists we should treasure it by opening it little by little.

One criticism of the Vienna classification[1] may be that although various categories of neoplasia were listed, the histopathological criteria for each one of the lesions were not verbalised, thus postponing the opportunity for its worldwide acceptance. Notwithstanding, some Western pathologists have started to herald the new "doctrine" by providing histopathological descriptions (criteria) for each one of the various categories proposed in Vienna.[2]

"To see or not to see" is not the question, as all lesions are there. As an example, dysplasia can be differentiated from carcinoma in situ.[2] Dysplasia in the glandular gastrointestinal mucosa is characterised by spindle or cigar shaped, elongated, pleomorphic, hyperchromatic nuclei, and regular nuclear membrane whereas carcinoma in situ displays large vesicular nuclei, irregular conspicuous nucleoli, and scalloped nuclear membranes. Bridges of nucleolus associated chromatin reaching irregular chromatin deposits are seen along the nuclear membrane. Bridges of chromatin are also seen connecting angular chromatin clumps. The nuclear polarity is disrupted, and marked cell pleomorphism and aberrant mitosis are present. Structural alterations may occur such as budding or branching crypts or tubules, with epithelial septa and back to back glands, and cribriform growth of epithelial cells in clusters and sheets. Those structures are confined to the basement membrane of the epithelial layer.[2] But surprisingly, despite those differences, high grade dysplasia and carcinoma in situ are still being regarded as synonyms in the Western literature.

The present discussion is beyond the usefulness of the Vienna classification as a tool for proper treatment; the discussion aims to point out our present lack of knowledge regarding the histogenesis of lesions represented by categories 4.2, 4.3, and 5.1 of Vienna[1] and their correct identification for future molecular research.

The viewpoint of Dixon appears to be in concert with the desire of many Western pathologists who are willing to embrace this new "doctrine" in order to acquire accurate information on the histological steps followed

by early neoplastic lesions of the gastro-intestinal tract. Only then will we be able to translate such events into molecular terms.

Perhaps the spirit of Johan Strauss has succeeded in orchestrating not only fiddlers but also workers engaged in the microscopic diagnosis of gastrointestinal epithelial neoplasias.

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BOOK REVIEWS

Dynamic Radiology of the Abdomen: Normal and Pathologic Anatomy, 5th edn

M A Meyers. Germany: Springer-Verlag, 2000. ISBN 0-387-98845-9

This is the fifth edition of *Dynamic Radiology of the Abdomen: Normal and Pathologic Anatomy,* a book that has become essential reading for all those aiming to be expert at abdominal imaging. Previous editions have been published in Italian, Japanese, Portuguese, and Spanish.

The continued aim of the author is to present a systematic application of anatomical and dynamic principles to aid our understanding of the characteristic appearances and modes of spread of intra-abdominal disorders. Dissections and cross sectional views of cadavers are used in conjunction with a full range of imaging modalities, including plain radiographs, contrast studies, computed tomography (CT), ultrasound, magnetic resonance imaging (MRI), and endoscopic, laparoscopic, and intraoperative ultrasound.

This edition has been extensively updated with six new chapters, 180 additional pages, and more than 520 new illustrations. Subjects that are included for the first time include clinical embryology in relation to disorders that become clinically apparent in the adult, TNM staging of gastrointestinal cancers, and the manifestations of free intraperitoneal air. There are now 11 other contributing authors, but Morton Meyers is solely responsible for about three quarters of the book and many of the of the cases illustrated are reproduced from his own numerous publications.

This book is well written, superbly illustrated, and comprehensively referenced. The illustrations, particularly the extensive use of cross sectional spiral CT images, make it easier for the reader to understand the complex anatomical arrangement of the abdominal organs and spaces and how they are modified by disease processes. The normal and pathological anatomy of the different parts of the gastrointestinal tract, the other abdominal organs, and the extraperitoneal spaces is described in detail. There are excellent descriptions of the intraperitoneal spread of infections and of malignancies. There is also a chapter on internal abdominal hernias. Pancreatic disorders and their mode of spread are described in detail; the diverse locations of pancreatic pseudocysts are well illustrated.

CT is now used to show the many places where free intraperitoneal air can collect in the abdomen. In recent years Cho and Baker have used this information to reassess the radiological appearances of free intraperitoneal air on the supine abdominal radiograph and have also described a number of new signs. They have brought this information together in their chapter and the result is an excellent and well illustrated contribution, providing information on an important subject that is not widely available elsewhere.

This edition of *Dynamic Radiology of the Abdomen* lives up the reputation established by previous editions. It should be of interest to all doctors who wish to learn more about the abdomen, particularly radiologists, oncologists, and surgeons. Radiologists who perform any type of abdominal imaging should find this latest edition invaluable and registrars training in radiology should become acquainted with it early in their career. In the preface to the first edition, Lloyd Nyhus commented on the innovative nature of this work and stated that it was an important reading source for surgeons; it likely that he would be equally enthusiastic about the latest edition.

D Nolan

Artificial Nutrition Support in Clinical Practice, 2nd edn

Edited by J Payne-James, G Grimble, D Silk. Greenwich Medical Media, £125. ISBN 1-90015-97-9

The provision of nutritional support for patients in hospital and in the community has derived from a heightened awareness of the impact of malnutrition on patient outcome and quality of life. Attention has focused on the prevalence of disease related malnutrition, both in terms of weight loss and weight for height, but also in terms of micronutrient deficiencies. Drives to enhance awareness of undernutrition in the community have followed efforts to improve nutrition in hospitals through food improvement, supplement provision, and artificial feeding. The need for routine nutritional assessment in clinical practice, especially in chronic disease, and its careful documentation has become ever more appreciated, particularly among gastroenterologists.

It is widely acknowledged that the approach to nutritional care is best made via a multiprofessional team which combines the skill and knowledge of dieticians, nutrition nurse specialists, pharmacists, and doctors. Teams like this are the key to excellent patient centred nutritional care and lead to overall hospital strategies for nutritional support which seamlessly combine, in a cost effective way, the use of food and supplements with artificial feeding to provide nutrients by the simplest safest route acceptable to the patient. Such teams need to develop more in the community too.

As the more enlightened hospitals form teams, a demand develops for a comprehensive textbook. This one is widely seen as one of the best and it now enters its enlarged second edition. It is an edited collation in which British authors dominate but it also includes contributions from key players from continental Europe making it representative of much of what goes on in the more Anglophone corners of ESPEN. As such it unsurprisingly contains some excellent chapters and others which are a little more patchy. But the collations is comprehensive, with a sensible mixture of the practical and the theoretical, the general and the more specialist. I would recommend it as a sound basis and as a useful resource for further reading.

J Powell-Tuck

Dyspepsia

M J Lancaster Smith, K L Koch. UK: Health Press, 2000, $14.00. ISBN 1-899541-92-6

We all have a word of advice and the general practitioner is a handy person to give it to. Getting it packaged right can be a challenge, not least when the front cover states that it is an "Indispensable guide to clinical practice". In this unusual transatlantic collaboration, two distinguished gastroenterologists have made a worthy effort to reach out and have successfully condensed most gastroenterological scenarios faced by the jobbing clinician into an attractive and accessible package. This little book packs a lot—while seeming to be a handy reference it is in fact a repository of facts and information and I confess to dipping into it often to confirm matters or to cull material for a presentation. For example, a map indicating the worldwide prevalence of *Helicobacter pylori* and tracings illustrating lower oesophageal pressures during swallowing enliven concepts glossed over in other publications.

None the less, the pedigree of the authors does tell on them in some of the sections. Hardly has the invisible ink (from the primary care practitioner's viewpoint) dried on the Rome II definitions before the one is being exhorted in the chapter on "Functional dyspepsia" to differentiate, on clinical grounds, ulcer-like and dysmotility-like dyspepsia. This is accompanied by advice on tailored regimens based on acid suppression or dysmotility agents. In real life, successful management, one fears, is more likely to be related to serendipity than acumen but there can be no harm in thinking constructively. The "Functional dyspepsia" chapter did rather throw me: the first line defines it as discomfort or pain centred in the epigastrium; luckily I kept at it and further down the page was informed that this only applies where "common or uncommon structural, biochemical or infectious agents have been excluded". Actually, this angst, and that of further subdividing functional dyspepsia, applies only to those who have heard of Rome II. Most primary care practitioners can thus relax. So can our gastroenterology colleagues who might otherwise be requested to confirm an exact diagnosis of functional dyspepsia in younger patients. Avoiding endoscopy here also avoids opprobrium; alas, the diagnosis of functional dyspepsia must remain in the mind rather than in the investigation suite.

This is the kind of handy book which one needs to receive gratis although I do recommend its purchase if necessary. It is eminently suited to distribution through the good will

and thoughtfulness of a pharmaceutical company and I trust that someone will come forward to do this. I do not plan to part with my copy, despite continuing references to prokinetic drugs which are no longer, or were never, available in the UK.

P Hungin

Reconstructive Surgery of the Esophagus

M K Ferguson. Futura Publishing, 2001, $105, pp 344. ISBN 0-87993-494-8

Oesophageal disease brings together many disciplines within the field of gastroenterology and the book is aimed primarily at the specialist oesophageal surgeon. Reconstruction of the oesophagus following resection for benign or malignant disease is one of the most challenging surgical procedures currently performed, and the oesophagus being such an unforgiving organ increases that challenge. Rather surprisingly, this is the first truly authoritative and evidence based volume to be devoted completely to this topic.

Mark Fergusson was trained at the University of Chicago under doyens of oesophageal surgery including Skinner, Belsey, and De-Meester, and has continued the tradition of that fine school. He is therefore well qualified to write this specialist tome. The historical chapter regales the courage of the pioneers of oesophageal surgery in the first half of the twentieth century, in which great British oesophageal surgeons including Grey Turner, Ivor Lewis, Allison, and Belsey are afforded due prominence. Following general sections on the philosophy of and indications for oesophageal replacement and the choices available of the oesophageal substitute and route to bridge the gap, a chapter is then devoted to each of the principal reconstructive techniques using stomach, colon, and jejunum, as well as the use of prosthetic tubes. Each of the chapters goes into considerable detail about relevant surgical anatomy, physiology, operative technique, and complications and their management.

Reconstructive Surgery of the Esophagus is clearly and succinctly written. While it draws heavily on the author's considerable experience, one of the attractions of this book is that it is clearly evidence based, and as well as being liberally referenced, the key references and their conclusions are highlighted in tabular form in each chapter. Another strong point for its predominantly surgical audience is the wealth of line drawings, which clearly depict surgical anatomy and technique. Overall, this is an excellent book, which takes its place well between existing tomes on oesophageal disease. I can recommend it wholeheartedly as an essential reference volume for both trainees and consultants in oesophageal surgery and indeed gastroenterologists might usefully dip into it occasionally so as to appreciate the many challenges facing their oesophageal surgeons in this fascinating branch of gastrointestinal surgery.

A Watson

The Pelvic Floor: Its Function and Disorders

Edited by J H Pemberton, M Swash, M M Henry. Harcourt, 2002, £79.95, pp 487. ISBN 0-7020-2307-8

In my clinical practice, I have felt for a long time that the knowledge acquired in coloproctology should be more frequently shared with specialists in obstetrics, gynaecology, urology, neurology, etc. Therefore, I enjoyed receiving this book where the contributors range between at least 10 specialities which concern pelvic floor disorders. Each chapter is written by a leading figure or a group expert in this field. The book is a valuable starting point for gastroenterologists who wish to become up to date with data concerning the clinical problems of the pelvic floor, associating the anterior and posterior components, including physiology, anatomy, diagnostic imaging, surgery, nursing, and psychology.

The previously published book by these authors was entitled *Coloproctology and the Pelvic Floor*—coloproctology has disappeared from the title of their new book, indicating that it is no longer possible to approach the posterior pelvic floor disorders without studying the pelvic floor as a whole. In this way, this new title by itself is a very strong message. However, the reader might be a little disappointed if he looks for how to treat, for example, a patient suffering from both anal and urinary incontinence, or a patient complaining of urinary stress incontinence and posterior pelvic floor dysfunction inducing straining at stools. The book contains a number of excellent algorithms concerning pathogenesis, investigations, and treatment of the pelvic floor disorders. However, these algorithms have been constructed to treat either the anterior pelvic floor or the posterior pelvic floor, but not to treat a patient who complains simultaneously of the two parts of the pelvic floor. It was perhaps because the book was initially so promising and the subsequent chapters so interesting that I was hoping for a little more specific detail from the authors!

Nevertheless, the psychological characteristics of the pelvic floor disorders are very well described, suggesting that the impact of social factors, such as sexual abuse for example and psychological distress, on the expression of pelvic floor symptoms should be taken into account. To date it has not been very easy to suggest guidelines indicating how to achieve a balance between identifying the pathophysiology of pelvic floor disorders and understanding psychological factors. There is no doubt that the algorithms given at the beginning of the book will be very useful for the reader. However, they would have been even more useful if the experts had suggested at which step(s) of their algorithms they felt the need to investigate the psychological profile of their patients.

As C Norton wrote in the book, "there is a small but growing movement to create multidisciplinary pelvic floor clinics, where urogynecologists, colorectal surgeons, specialist nurses, physiotherapists, neurologists, psychiatrists . . . work together to improve the management of pelvic floor disorders". While we are waiting for these future multidisciplinary clinics of "perineology", it was probably not the time to furnish algorithms in this particular edition of *The Pelvic Floor* concerning investigations and management of associated symptoms of the anterior and posterior pelvic floor, integrating the psychological profiles of the patients. JH Pemberton, M Swash, and MM Henry must be acknowledged and congratulated for bringing together the knowledge of all the specialties involved in the pelvic floor.

P Denis

Clinical Governance in Gastroenterology

G Rubin, R Jones, J Price, *et al*. Radcliffe, 2000, B/W, pp 101. ISBN 1-85775-438-7

Can external control drive clinical standards? In the meantime we have clinical governance. What this actually means, how it is meant to operate, and whether governance guidelines will become yardsticks for judging performance also remain open. But at least it sounds like a good thing, and one glib but hopefully sensible answer is that clinical governance might ensure "uniform standards" across heterogenous NHS practices, reassure the public and, in any case, it seems here to stay—at least for the moment.

It still baffles me as to what differences there are between excellent clinical practice and practice by clinical governance—presumably the latter is not meant to be quite as good, but will do. There seems to be a clamour for " acceptable" standards and this handbook, from a group of eminent UK general practitioners, fills a gap for primary care. The authors are members of the Primary Care Society for Gastroenterology, an organisation that has made significant contributions in knocking down barriers between primary and secondary care and has advocated cogent seamless care. The publication covers a series of problems from dyspepsia to colon cancer and includes an interesting section on horizon scanning, an example of new NHS speak, evidence that the authors have their ear to the ground, shifting though it may be.

It would be deluding to assume that all we know about gastroenterology, and that which matters to patient care can be compressed into 94 pages, but there is sufficient to keep governance types well busy. The information is well accessible but this is, of course, not a textbook. The chapter on stomas begins "A stoma is an artificial opening of the bowel on to the external skin", sheer gold dust to the doctor who forgot to attend the surgery lectures or to the non-clinical manager now anticipating being in charge of ensuring we do our jobs properly and services are commissioned effectively. There are one or two omissions—for example, the lack of mention of varices as a cause of acute bleeding—but each chapter does have a section on health economics for those hopefully traversing the quagmire of costs, effectiveness, care, and quality. Word has it that this book is selling well; the authors have got that aspect of health economics correct and timely.

P Hungin

Gastroenterology Highlights 2001–02

Edited by E Quigley. Health press, 2002, £15.00, colour, pp 84. ISBN 1-903734-12-6

Gastroenterology Highlights 2001–2 is attractively presented in good quality four colour format. This slim volume of 84 pages comprises 10 chapters written by a panel of international experts. Topics covered range from diseases of the oesophagus, liver, pancreas, and small bowel, to complications of liver disease, endoscopy, and colorectal cancer prevention and screening. The aim is to discuss key papers and put them into context. In most chapters, about 20 papers are reviewed but the actual numbers range from 16 to 30. Most chapters also review one or two abstracts. The

vast majority of papers covered were published in 2001 or 2000.

The *Fast Fact Highlights* series aims to "keep its readers abreast of the latest innovations" in each specialty. The flyer states that the information is presented "in an accessible style, comprehensively illustrated and fully indexed". Have these aims been met? Certainly the style is easy to read. However, there are only three figures in the whole book. Two of these are world maps showing geographical variations in colorectal incidence and mortality, while a third figure is a rather pointless flow chart of "preventive steps" for patient groups at average, moderate, and high risk from colorectal cancer. The steps are identical for the first two groups: change in lifestyle, chemoprevention and screening, and early diagnosis. These steps are again repeated for the high risk group, with preventive surgery added. There is no subject index.

I like the table in each chapter stating what are "in", what are "out", what are contentious, and what are still needed. However, it is irritating that many of the items mentioned as "in" or "out" have neither been discussed in the text nor referenced.

In the discussion on endoscopic treatment of gastro-oesophageal reflux, the EndoCinch and implantation of microspheres were discussed, but not the Stretta procedure. Both Freedman's study on the association between cholecystectomy and oesophageal adenocarcinoma as well as Schnell's report on nonsurgical management of Barrett's oesophagus with high grade dysplasia, were reviewed in the oesophagus chapter and again in the chapter on gastrointestinal cancer. Tighter editing could have avoided this duplication as space in this book is clearly at a premium. I was surprised to read that "rectal examination as the only test for colorectal cancer" was "out". This statement was not referenced!

These brief reviews cannot by their nature be comprehensive. While this volume covers more ground than the short literature review booklets sponsored and distributed free by pharmaceutical companies, only about 20 papers are reviewed per topic. This can only represent a small selection of the many advances over a one to two year period, and

falls far short of the excellent reviews in the *Current Opinions in Gastroenterology* series. It is probably unsuitable for a library collection, and is not a book I would myself keep for reference. I am uncertain who may wish to purchase this volume, even though it is modestly priced at £15. While it is an easy read, I suspect that few consultant gastroenterologists would want to buy this book. I doubt if many trainees would either.

J Y Kang

CORRECTION

In the paper by Higham *et al* (*Gut* 2002;**50**:460–4) the heading for table 4 should read "Number of items (thousands) prescribed in England from 1990 to 1999. Prescription Cost Analysis system (Department of Health)".

NOTICES

38th EASL Annual Meeting

The European Association for the Study of the Liver will be holding its 38th annual meeting on 29 March–1 April 2003 in Istanbul, Turkey. Further information can be found on the website www.easl.ch/easl2003.

Falk Workshop—Inflammatory Bowel Disease: Turning New Advances into Practice

This will be held on 3 April 2003 in Berne, Switzerland. Further details: Falk Foundation e.V., Congress Division, PO Box 6529, Leinenweberstr. 5, 79041 Freiburg/Br, Germany. Tel: +49 761 15 140; fax: +49 761 15 14 359; email: symposia@falkfoundation.de; website: www.falkfoundation.de

International Symposium on Viral Hepatitis and Liver Disease

This conference will take place on 6–10 April 2003 in Sydney, Australia. Further information: ISVHLD 2003 Congress Managers, GPO Box 128, Sydney NSW 2001, Australia. Tel: +612 9262 2277; fax: +612 9262 3135; email: isvhld@tourhosts.com.au; website: www.tourhosts.com.au/isvhld

Prague Hepatology Meeting

To be held on 5–7 June 2003 in Prague, Czech Republic. Leading speakers from Europe and the USA will present new ideas and suggestions on pathophysiology, diagnostics, and therapy of liver diseases in ten programmes blocks. Further details: Ms Veronica Revicka. Tel: +420 241 445 759; fax: +420 241 445 806; email: veronika@congressprague.cz

Falk Symposia—New Findings on Pathogenesis and Progress in Management of IBD

Two symposia and a workshop will be held on 10–14 June 2003 in Berlin, Germany. Further details - see Falk Workshop details above.

Gastroenterology and Endotherapy: XXIst European Workshop

This will be held on 16–18 June 2003 in Brussels, Belgium. Further details: Nancy Beauprez, Administrative Secretariat of the Workshop, Gastroenterology Department, Erasme Hospital, Route de Lennik 808, B-1070 Brussels, Belgium. Tel: +32 (0)2 555 49 00; fax: +32 (0)2 555 49 01; email: beauprez@ulb.ac.be

The Association of Coloproctology of Great Britain & Ireland

This annual meeting will be held on 7–10 July 2003 in Edinburgh, UK. Further details: Conference Secretariat, The ACGBI at the Royal College of Surgeons of England, 35–43 Lincoln's Inn Fields, London WC2A 3PE. Tel: +44 (0)20 7973 0307; fax: +44 (0)20 7430 9235; email: acpgbi@asgbi.org.uk; website: www.acpgbi.org.uk