place in Hull on 12-15 May 1989 and will be accompanied by the first consensus conference on testicular cancer. At this meeting representatives of all the principal clinical research organisations will again be present and with their help a meaningful consensus on the most appropriate treatment in individual patients can be clearly defined. Only then can a sensible decision be made on which treatment is the most cost effective and sensible guidelines laid down for oncologists and other clinicians concerned in the care of patients with cancer.

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The frozen hip

Drs M D Chard and J R Jenner (3 September, p 596) suggest that a capsulitis of the hip may lead to painful restriction of movement comparable to adhesive capsulitis of the shoulder. The three cases they describe seem to provide little evidence to support this idea. Although frozen shoulder may reasonably be diagnosed from normal results in an arthrogram and increased uptake in a bone scan, to apply these criteria to the hip—a joint of entirely different structure and function—seems to be an imaginative extrapolation. The cases described could all have been episodes of intraosseous hypertension associated with venous stasis—stage I osteonecrosis of the femoral head.[1] No mention is made of osteonecrosis as a possible diagnosis and no attempt seems to have been made to exclude it by measuring intraosseous pressure and performing phlebography or magnetic resonance imaging.

Stage I osteonecrosis of the femoral head may resolve spontaneously but has the potential to progress to structural damage and secondary osteoarthritis. Forage decompression of the femoral head in the early stages of osteonecrosis carries a good prognosis for relief of symptoms and preservation of articular structure.[2] Even if no intervention were contemplated it would be a mistake to confuse osteonecrosis with so called frozen hip as osteonecrosis does not carry the good prognosis that Drs Chard and Jenner say can be expected for frozen hip.

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Endoscopic balloon dilatation of benign gastric outlet obstruction

Drs P I Craig and P E Gillespie (6 August, p 396) describe an interesting technique but the design of their trial and their methods of assessing gastric outlet obstruction raise questions.

That most of their patients were elderly and many had associated serious illnesses may be important in addition to the fact that surgery is best avoided in such patients. There is no disputing the diagnosis of gastric outlet obstruction in three of their 14 patients—the two with postbulbar duodenal obstruction and one with stomal stenosis after Billroth 2 gastrectomy. But the basis for diagnosis in the remaining cases is debatable as it is not uncommon at endoscopy to find a "tight" pylorus in the elderly without evidence of peptic ulceration, whether active or past, and, more importantly, without any symptoms of gastric outlet obstruction.

Drs Craig and Gillespie state that all but one of their patients had symptoms but do not state their nature. Instead they cite barium retention or the failure to pass an endoscope of 11 mm in diameter through the pylorus as objective indicators of gastric outlet obstruction. A detailed description of symptoms would have been more reliable, and if an objective test was required gastric emptying could have been measured simply and more accurately by scintiscanning. Symptoms are important, as alluded to by the authors in methods and results when they state that one patient without symptoms was found to have outlet obstruction at endoscopy and again in the comment when they mention "asymptomatic patients with endoscopic evidence of restenosis." Also, was the associated serious illness diabetes in any of their patients? For if so, here is a recognised cause of delayed gastric emptying irrespective of the calibre of the pylorus at one instant in time. That passage of a bolus through the gastrointestinal tract is dictated by both motility and the size of the lumen is clearly shown by the patient with scleroderma with the merest of a stricture having intense dysphagia because oesophageal peristalsis is poor while a patient with normal oesophageal peristalsis has to develop a large stricture before experiencing dysphagia.

The authors also do not state how many patients had received a trial of medical treatment before dilatation, yet all were started on ranitidine after dilatation. It might have been interesting to determine the proportion of patients who settled on medical treatment alone.

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AUTHORS' REPLY,—Preoperative criteria for diagnosing gastric outlet obstruction lack uniformity but traditionally have included appropriate symptoms and signs, x ray and endoscopic evidence of obstruction, and an increased fasting gastric residue.[1] Undoubtedly scintiscanning has recently become a useful adjunct to diagnose delayed gastric emptying due to either outlet obstruction or motor disorders, yet the wide normal range of the test reduces its usefulness.

Dr Mughal disputes the diagnosis of gastric outlet obstruction in 11 of our 14 subjects. Before dilatation 13 of the 14 patients had typical symptoms of gastric outlet obstruction (postprandial vomiting 12, weight loss 12, early satiety 9, and cramping abdominal pain 8) and 8 had appropriate signs (muscle wasting 8, epigastric fullness 4, and gastric splash 3). The one asymptomatic patient, with recurrent cholangitis, was found to have pyloric stenosis during a procedure indicated for endoscopic sphincterotomy. Seven patients were restricted to either a fluid or soft diet while another tolerated nothing orally and required total parenteral nutrition. Of the selected patients therefore, 13 had typical clinical features; a small diameter endoscope failed to pass through the stricture in all cases, and each of the 10 barium meals performed were consistent with gastric outlet obstruction. After dilatation symptoms resolved completely in all but two patients who had persisting early satiety.

Certainly one of our patients with pyloric stenosis had well controlled maturity onset diabetes mellitus, which we agree can be associated with delayed gastric emptying. Nevertheless, she also had typical symptoms and barium meal and endoscopic evidence of pyloric stenosis, which all improved after successful dilatation. Finally, only two patients with active ulceration contributing to stenosis were dilated without having received a full course of H_2 receptor treatment.

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Notification of tuberculosis

I fear that the experience of Dr B L Bradley and others (3 September, p 595) is not limited to notification of tuberculosis but may be seen across the spectrum of notifiable diseases.

In September-November 1987 the notification of infectious disease by the laboratories and general practitioners in west Dorset was reviewed. There was significant under reporting, as shown in the table.

Numbers of laboratory reports of infectious diseases and numbers of notifications received

Laboratory reports of infectious disease (n=77)		Notifications received (n=57)	
Salmonella	38	Salmonella	15
Campylobacter	21	Campylobacter	3
Other food poisoning and diarrhoeal episodes	11	Food poisoning	5
		Scarlet fever	3
Meningitis	2	Pertussis	10
Tuberculosis	3	Tuberculosis	2
Hepatitis B surface antigen	2	Measles	6
		Scabies	11
		Hepatitis A	2

This information was circulated to all general practitioners with a resulting 300% increase in notifications in the following two months. Regular audit of this kind has ensured a much higher notification record in the district and consequently a better overall picture of infectious disease in the community.

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The code for promoting drugs

Professor Michael Langman's editorial (20-27 August, p 499) seemed to have an air of resignation. I agree with what he said, and if indeed he is resigned I share that also. Perhaps the reasons for this are worth exploring a little.

Since 1948 the British medical profession has become less and less commercial in experience and sympathies. Before that time our fathers and grandfathers contrived to lead a reasonably respectable middle class life on the shillings and half crowns paid to them directly for their services by their patients. The payment of ancillary staff and the purchase and running costs of their surgeries were entirely at their own risk. They were small businessmen. But that was 40 years ago. Noncommercialism has gone a long way towards frank anticommercialism. Though the debate still rambles on—pay beds, private practice, private hospitals—the voice of the profession is mainly anticommercial.