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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1 Denis L, Niijima T, Prout G Jr, Schroder FH, eds. *Progress in clinical and biological research*. Vol 221. *Developments in bladder cancer*. New York: A R Liss, 1985.
2 Schroder FH, Klijn JGM, Kurth KH, Pinedo HM, Splinter TAW, de Voogt HJ, eds. *Progress and controversies in oncological urology II*. New York: A R Liss, 1987.

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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1 Ficat P, Arlet J. *Ischémie et nécrose osseuses*. Paris: Masson, 1977.
2 Ficat RP. Idiopathic bone necrosis of the femoral head. *J Bone Joint Surg [Br]* 1985;**67**:3-9.

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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1 Ellis H. Pyloric stenosis complicating duodenal ulceration. *World J Surg* 1987;**11**:315-8.

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
Meningitis	2	Scarlet fever	3
Tuberculosis	3	Pertussis	10
Hepatitis B surface antigen	2	Tuberculosis	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.

The pharmaceutical industry has however remained commercial. There is little doubt that if you require the best in innovation, production, and distribution this is as it should be, as some of the world's larger economic experiments have shown nationalisation to be rather inefficient in all these aspects. If we want the commercial system to function well the best must be highly rewarded, which can be expressed only in high sales with adequate profit margins.

Pharmaceutical marketing staff stand therefore on the frontier between two systems of thought, which if not incompatible are in a state of considerable tension. The job is strange enough anyway. Selling is restricted to doctors who rarely consume and in most cases do not sell the product, whose price to the consumer has been divorced from its market value. When the generally anticommercial stance of the prescriber is added it is not surprising that frustration occasionally leads to some sort of cross border incident.

I believe that the Code of Practice Committee do a good job in trying to prevent conflict and by rapping knuckles when the industry oversteps the mark.

There seems no likelihood of British medicine returning to the small business ethic or of the pharmaceutical industry becoming non-profit making. The only way to avoid conflict is to increase understanding. As a doctor in the industry I have the discomfort of being occasionally held guilty by both sides.

I should like to see my colleagues in industry be more constantly aware that they are not selling a simple commodity and my professional colleagues temper their not entirely justified sense of moral superiority with a little more humility. Both sides have reason to respect and be grateful for the achievements of the other.

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Points

Postperinatal mortality in a health district with a garrison town

Dr J A SHAPIRO (Nuneaton, Warwickshire CV12 9LX) writes: For three years, ending in March of this year, I looked after most of the army families—that is, the civilian relatives of army staff—at a garrison near our practice, and I can confirm anecdotally the evidence of Dr Mala Rao and Ms Elizabeth Hoinville (10 September, p 662) that the mothers were young, most smoked, and fewer breast fed their babies compared with the general practice population. There also seemed to be a high incidence of marital stress. The most interesting finding that we noted was the extremely high rate of consultation in the army camp. Our normal practice population has a rate of consultation of about three consultations for each patient a year. The figure when we first started a branch surgery in the army camp was as high as 10 consultations for each patient a year, though this dropped during our time at the camp to a value of five or six. Our explanation for this high rate concurs closely with that of Dr Rao and Ms Hoinville—namely, that there is little support from the statutory services or from peer and family groups. The army families also form an itinerant population that never builds a real bond with the primary health care team, so there is no time to carry out any effective health education. Moreover, the army authorities themselves seem to us to lack the motivation to provide an appropriate level of support.

Dr KEVIN JONES (Primary Medical Care, Faculty of Medicine, Aldermoor Health Centre, Southampton SO1 6ST) writes: Dr Mala Rao and Ms Elizabeth Hoinville (10 September, p 662) found higher rates of postperinatal mortality and death from the sudden infant death syndrome among army babies. They speculated that several social factors contribute to this finding, but in my opinion they did not emphasise enough the higher rate of smoking. This was noted in their study, although it was not significant. Previous work has highlighted the importance of high rates of smoking among soldiers in the development of coronary heart disease.[1] I have been a regimental medical officer and army medical registrar and my own analysis of data on births in service families based in Hong Kong showed that army babies were significantly lighter and shorter and had smaller heads when compared with babies from civilian expatriate families.[2] Higher rates of smoking were probably partly associated with the aetiology of this difference, although other demographic influences may have applied. I believe that this new report provides yet more evidence for the harm caused to servicemen and their families by the indirect promotion of smoking implicit in the subsidised prices of cigarettes available to service staff, especially overseas. For the sake of our troops and their offspring this must be reconsidered.

1 Lynch P, Ineson N, Jones KP, Scott AW, Crawford IC. Risk profile of soldiers aged under 40 with coronary heart disease. *Br Med J* 1985;**290**:1868-9.
2 Jones KP. Ethnic variations in birth weight—a study of British, Chinese and Gurkha babies. *J R Army Med Corps* 1987;**133**: 146-7.

Occult biochemical pregnancies

Dr GUTTORM BRATTEBØ (Department of Surgery, Hammerfest Hospital, N-9601 Hammerfest, Norway) writes: Minerva (30 July, p 368) raised the question of the incidence of early pregnancy losses, referring to a recently published British study in which no evidence of occult pregnancies was found.[1] A far larger investigation in the United States estimated the rate to be 22% during 707 menstrual cycles among 221 women who were trying to conceive.[2] The American study used a highly sensitive and specific immunoradiometric technique, enabling measurement of human chorionic hormone concentration with no cross reaction with human luteinising hormone.[3] Therefore research does indicate that medical folklore is correct when stating that many women become pregnant only to abort before recognising it.

1 Walker EM, Lewis M, Cooper W, Marnie M, Howie PW. Occult biochemical pregnancy: fact or fiction? *Br J Obstet Gynaecol* 1988;**95**:659-63.
2 Wilcox AJ, Weinberg CR, O'Connor JF, *et al.* Incidence of early loss of pregnancy. *N Engl J Med* 1988;**319**:189-94.
3 Wehmann RE, Harman SM, Birken S, Canfield RE, Nisula BC. Convenient radioimmunoassay for urinary human choriogonadotropin without interference by urinary human luteotropin. *Clin Chem* 1981;**27**:1997-2001.

Drug rationalisation programme in hospital

Professor K B SAUNDERS (Department of Medicine, St George's Hospital Medical School, London SW17 0RE) writes: At St George's we noted with astonishment the comment in Editor's Choice that "as yet no other London teaching hospital has followed suit" in using a drug rationalisation programme similar to that described by Mr John A Baker and others (13 August, p 465) at Westminster Hospital. A restricted drugs policy was introduced here in 1980 and a description of the first five years' experience was published by Collier and Foster in 1985,[1] which is given in the Westminster paper. Savings in Wandsworth since 1980, calculated in a different way from that used by the Westminster group, are about £1m. The restricted list was originally developed for Wandsworth but was subsequently adopted by two neighbouring districts and used as a basis in developing lists in several others. May I protest, gently, on behalf of my colleague Dr Collier, who is of course too modest to do so.

1 Collier J, Foster J. Management of a restricted drugs policy in hospital: the first five years' experience. *Lancet* 1985;i:331-3.

Treatment of night terrors

Drs A J HOWAT and L HOWAT (Sheffield S10 4LN) write: We agree wholeheartedly with the paper by Dr Bryan Lask (3 September, p 592) on the treatment of night terrors. We came to the same conclusion after personal experience. Our second child began to have classic night terrors when aged 2½ years. They occurred two to three times a week and were characterised by initial mumbling and jibbering followed by screaming in terror and staring unseeingly at a corner of the bedroom. Attempted consolation by cuddling and soothing was ineffectual; the attack would last five or so minutes, whence she would then quickly return to sleep. She had no recollection in the morning, and when asked how she had slept the reply was always "very well, thank you." These experiences were, however, shattering for us, making us housebound and loth to go out leaving a babysitter in charge of the children. It became apparent that these attacks occurred one and a half to two hours after falling asleep. So we disturbed and woke her about one to one and a half hours after her going to sleep. The night terrors promptly stopped. We continued to wake her like this for a few weeks and then stopped. The terrors returned a few months later but were quickly treated by the waking regimen. She is currently 5½ years old and no longer affected. We have since told general practitioner friends of this treatment, and they have used it with success in other families whose children experience these terrifying (for the parents) experiences.

Drug Point

Haemolytic anaemia associated with fenbufen

Dr T MARTLAND and Mr W D STONE (District Hospital, York YO3 7HE) write: Several non-steroidal anti-inflammatory drugs cause haemolytic anaemia.[1] We report a case caused probably by fenbufen, an association that has been noted only once before.[2]

A retired gardener aged 66 was admitted with a three day history of lethargy, anorexia, and mild jaundice with dark urine. His only previous illness was polymyalgia rheumatica, which had resolved without treatment. He had been taking his wife's fenbufen 300 mg twice daily for 10 days for mild arthritic symptoms. He was not taking other drugs and had not been exposed to environmental hazards.

On admission his temperature was normal, but he was pale and sweating, with mild jaundice but no lymphadenopathy, hepatosplenomegaly, or bruising. Initial results showed a haemoglobin concentration of 43 g/l, mean corpuscular volume of 95 fl, and packed cell volume of 0·12; a blood film showed spherocytosis and normoblasts with a reticulocyte count of 18%. A direct Coombs test was strongly positive. The erythrocytes were coated with IgG and C3d component of complement. Red cell eluate tests at 20°C and 37°C showed no irregular antibodies. Tests for antinuclear factor, rheumatoid factor, anti-DNA antibody, mycoplasma agglutination, and complement fixation gave negative results. A chest radiograph was normal and results of biochemical tests showed no abnormalities, except for bilirubin (74 μmol/l) concentration and alkaline phosphatase (107 IU/l) and serum alanine transaminase (45 IU/l) activities. Dipstick testing of his urine showed protein and blood with no bilirubin. He was treated initially with high dose intravenous and oral steroids but continued to deteriorate, becoming breathless, confused, and paranoid. He was transfused with six units of blood during 24 hours and improved appreciably. He was discharged home nine days after admission entirely well, taking prednisolone 20 mg a day. At follow up four months later he was without symptoms, except for his mild arthritis, and was not receiving any treatment; his haemoglobin concentration was 142 g/l and reticulocyte count 2%.

This patient's haemolytic anaemia was probably caused by fenbufen, and his subsequent wellbeing supports this. There are close similarities between this case and the previous report of haemolytic anaemia associated with fenbufen, especially the short time for which the drug was taken and the sudden and severe onset of anaemia. Fenbufen is closely related pharmacologically to other anti-inflammatory agents that cause similar reactions, such as ibuprofen, naproxen, and mefenamic acid. Perhaps all drugs of this class should be considered as potential causes of haemolytic anaemia?

1 Sanford-Driscoll M, Knodel LC. Induction of haemolytic anaemia by non-steroidal anti-inflammatory drugs. *Drug Intell Clin Pharm* 1986;**20**:925-34.
2 Beucler A, Rezean H, Castot A, Guillemain JL, Angellier JF. Anémie hémolytique au fenbufene. *Presse Med* 1986;**30**:1426.