the forceps." As Kielland's forceps are likely to be used less frequently in the future,[2] surely lack of experience in their use must diminish the degree of finesse.

His second point, disliking "pulling babies out by sucking on their skin" is, dare I say it, somewhat emotive, and could easily be countered by those who dislike the idea of applying steel forceps capable of exerting considerably greater force than the normal forces of labour to a baby's head in order forcibly to rotate and extract it.

As to being put off by vacuum lovers' bias, I can but quote Mr Drife himself: "Feelings for and against Kielland's forceps are in danger of becoming entrenched (as often happens when firm data are lacking)."[2]

His last and most serious point is the risk of fetal trauma if the vacuum is applied above the level of the ischial spines. Guidelines for the safe use of the vacuum could prohibit its use in such circumstances; there are no fewer than seven strictures surrounding the use of Kielland's forceps.[3]

By all means let's have a trial, with perhaps a caesarean section arm into which only multigravidas would be randomised. Recent work has shown that the latter group are more likely to suffer pudendal nerve damage with forceps intervention for second stage delay,[1] and their risk of developing distressing anorectal or urinary incontinence is greater than that of primigravidas. But not, please, as Dr Edward Morris suggests (p 1823), an exclusive London teaching hospital trial, but rather a multicentre trial to include district general hospitals, where the variation in experience and supervision of the operators will reflect a more accurate picture of the country as a whole.

SHEILA SHEERIN

Princes Risborough,
Bucks HP17 9AS

1 Vacca A, Grant A, Wyatt G, Chalmers I. Portsmouth operative delivery trial: a comparison of vacuum extraction and forceps delivery. *Br J Obstet Gynaecol* 1983;**90**:1107-12.
2 Drife JO. Kielland or Caesar? *Br Med J* 1983;**287**:309-10.
3 Cardoza LD, Gibb DMF, Shidd JWW, *et al*. Should we abandon Kielland's forceps? *Br Med J* 1983;**287**:315-7.
4 Snooks SJ, Setchell M, Swash M, Henry MM. Injury to the innervation of the pelvic floor musculature in childbirth. *Lancet* 1984;ii:546-50.

Frozen shoulder

SIR,—Dr N A Watson's criticism (22 June, p 1904) of my leading article "Time for a new name for 'frozen shoulder'" (27 April, p 1223) deserves an answer. The article was intended to show how confusing the terminology has become on shoulder pain. Frozen shoulder has become an exceedingly lax term, which is now applied in a blunderbuss fashion to all types of shoulder pain and is as helpful as saying that someone has a limp.

I agree with Mr Watson that the selective tissue tension technique of Cyriax is useful for coning down pathology to one part of the rotator cuff, but Mr Watson well knows (from his recent leading articles) that it is not possible to mention all contributions to a subject. I had to content myself with discussing terminology and not diagnostic methods. As far as diagnostic methods go Cyriax is only one facet—for instance, pain ablation studies are just as important,[2] but that is another story.

In fact I was much more anxious about upsetting the disciples of Codman, rather than Cyriax, for Codman was not only a great surgeon but an exceptionally original mind who was pioneering audit at the turn of the century and was the first to investigate the uses of the new Roentgen rays in the diagnosis of bone disease in America (hence Codman's triangle).

However, I have to disagree with Mr Watson's statement that there is one paper showing a "clear advantage" for selective steroid injections in frozen shoulder. Hollingworth's paper shows nothing of the kind.[3] Hollingworth actually states in his paper that he had "poor results" for selective steroid in the frozen shoulder group, only 6 out of 23 patients having any benefit and none of 20 getting any relief from steroid injection to the tender site. Hollingworth concludes that these results confirm the work of Bruckner[4] and Cyriax,[5] who also got poor results in the "frozen" group—certainly not a "clear advantage" for steroids.

What Hollingworth did show was that if careful diagnostic methods are applied to patients with shoulder pain some are found to have referred pain from the neck, some have acromioclavicular joint pain, and most have rotator cuff "tendinitis" (which I take to mean irritation of the rotator cuff, either from subacromial impingement or from a small cuff tear). Only 25 of 92 patients with shoulder pain were left in the "frozen" group and these were the poor responders. In other words, careful diagnosis leads to better results than a blunderbuss approach.

So, Mr Watson, we appear to agree, let us get rid of the blunderbuss "frozen shoulder" and by coning down on real pathology help our patients more than 6 out of 23 times.

TIM BUNKER

Exminster,
Devon

1 Tulloch Brown J. Early assessment of supraspinatus tears. Procaine infiltration as a guide to treatment. *J Bone Joint Surg* 1949;**31B**:423.
2 Neer C. Impingement lesions. *Clin Orthop* 1983;**173**:70.
3 Hollingworth G, Ellis R, Hattersley T. Comparison of injection techniques for shoulder pain. *Br Med J* 1983;**287**:1399.
4 Bruckner F. Frozen shoulder (adhesive capsulitis). *J R Soc Med* 1982;75:688.
5 Cyriax J, Troisier O. Hydrocortisone and soft-tissue lesions. *Br Med J* 1953;ii:966.

Unrecognised depression in general practice

SIR,—Freeling and his coauthors conclude that their most important finding was that the doctors they studied failed to recognise depression in more than half the depressed patients they saw during the sessions screened (22 June, p 1880). While strictly true, this statement is misleading in that it suggests that the diagnostic "sensitivity" of their doctors for depressive illness was less than 50%. This latter conclusion could not be supported by their method, and their results suggest that it is not the case.

The potential confusion arises from the nature of the sampling frame and method. Their sampling frame seems to have been the consultations held by a set of GPs over a period of time. From this frame consultations were selected by a method of choosing clinics at representative times of the day and week.

The problem with this is that patients who consult their GP frequently will have a greater chance of appearing in the sample. If a GP has n consultations in the sample frame period and 100 of his consultations are selected for study, then the probability of selecting a patient who consults 10 times in the sample frame period will be roughly $(10\times100)/n$, while the probability of selecting a patient who consults only once will be roughly $(1\times100)/n$. ("Roughly" because I am assuming they would have excluded anyone who had already been sampled.)

How does this affect the interpretation of the results? If it were the case that on average unrecognised depressives consulted their GPs no more or less often than recognised ones it would mean that the sample could be taken to be representative of patients consulting. If, on the other hand, unrecognised depressives consult more often then the results need modifying for conclusions to be drawn about the diagnostic "sensitivity" of the GPs. For example, if the unrecognised depressives consult on average five times as often as the recognised ones then the "sensitivity" of the GPs would be a little better than 75%. The authors report that the unrecognised depressives tended to have had a longer history of illness and also a higher chance of concurrent physical illness than the recognised ones. Both these features would be likely to make them more frequent consulters, as would the GPs' failure to recognise their depression.

It is important to recognise this type of sampling bias since if data are collected about the precise degree of bias for each patient—in this case the number of times each patient selected had consulted in a representatively long period (say a year)—then by simple statistical weighting the bias can be eliminated.

GYLES R GLOVER

London E3 5DB

Acute appendicitis and infectious mononucleosis

SIR,—Dr Hugh Trowell (1 June, p 1660) debates with Professor D J P Barker (13 April, p 1125) the "narrow lumen hypothesis" in acute appendicitis. Appendicitis is not a recognised complication of infectious mononucleosis.[1,2] The most characteristic histological feature of the normal appendix is the lymphatic tissue and this closely resembles the nodules that surround the crypts of the palatine tonsils.[3] Why the Epstein-Barr virus, a pathogen of lymphocytes par excellence, does not invade the appendix and produce obstruction to the lumen leading to appendicitis more often than published reports suggest remains one of the many unsolved questions concerning the virus. Burkitt's lymphoma, another condition in which Epstein-Barr virus is implicated,[2] also affects the appendix very rarely.[4]

A 15 year old girl was admitted to hospital with a two day history of colicky abdominal pain, vomiting, and diarrhoea. Four days previously she had complained of sore throat; septic tonsillitis was diagnosed and treated with oral erythromycin. On examination her temperature was 39°C, pulse was regular at 100 beats/min, and blood pressure was 110/70 mm Hg. Her throat was inflamed with tonsillar enlargement and exudate. Cervical lymph nodes were enlarged with fading macular skin rash. Abdominal examination showed guarding and tenderness of the lower abdomen with rebound tenderness. Liver and spleen were not palpable. Chest radiograph was normal. Haemoglobin concentration was 154 g/l and white cell count $10{\cdot}2\times10^9$/l with 60% neutrophils, 35% lymphocytes, and numerous atypical mononuclear cells. A Monospot test was positive. Blood culture and throat swab grew no bacteria.

At laparotomy an acutely inflamed, gangrenous appendix, which was adherent to the pelvic wall, was removed (Mr D Gatehouse). There was perforation of the mid-portion of the appendix and thin pus in the peritoneal cavity. Intravenous cephradine and metronidazole were given and she recovered uneventfully.

Histological examination of the appendix showed acute suppurative inflammation, ulceration, and peritonitis with virtual "gangrenous" change in the wall, particularly near the tip. A number of reactive lymphoid follicles were present, but in view of the severe inflammatory change these could not be associated with infectious mononucleosis with certainty (Dr I B Porteous).

To confirm the diagnosis of recent infectious mononucleosis further tests were done at the Public Health Laboratory, Newcastle upon Tyne. Serial serum titres for cytomegalovirus, leptospira, *Rickettsia burneti*, *Mycoplasma pneumoniae*, mumps, and adenovirus were all negative. She was positive on fluorescence antibody test for Epstein-Barr virus and also for Epstein-Barr virus specific IgM. These serological and

haematological findings confirmed a current infection with Epstein-Barr virus producing acute infectious mononucleosis.

The diagnosis of infectious mononucleosis was established by clinical, haematological, and serological features, which included the presence of specific antibody to Epstein-Barr virus.[2] Advances in virology and immunology now permit us to recognise increasing numbers of diseases caused by Epstein-Barr virus.[2] Infectious mononucleosis and appendicitis are two common acute illnesses which affect the same age group. Many primary Epstein-Barr virus infections are probably missed because the full spectrum of illness is not yet known. Although isolation of the virus adds weight to any causal relation based on serological reactions, recovery of Epstein-Barr virus from throat swabs or blood has not yet been attempted on any large group of sick children with ill defined diseases. I suggest that the time has come to look at the relationship of Epstein-Barr virus to appendicitis.

B THALAYASINGAM

Paediatric Unit,
Shotley Bridge General Hospital,
Consett,
Co Durham DH8 0NB

1 Finch SC. Clinical symptoms and signs of infectious mononucleosis. In: Carter RL, Penman HG, eds. *Infectious mononucleosis*. Oxford: Blackwell Scientific Publications, 1969: 19-46.
2 Purtilo DT. Immunopathology of infectious mononucleosis and other complications of Epstein-Barr virus infection. In: Sommers SC, Rosen PP, eds. *Pathology annual*. Vol 15. Part 1. Norwalk, Connecticut: Appleton-Century-Crofts, 1980: 253-99.
3 Copenhaver WM, Kelly DE, Wood RL. *Bailey's textbook of histology*. 7th ed. Baltimore: Williams and Wilkin, 1978:509.
4 Nanji AA, Anderson FH. Burkitt's lymphoma with acute appendicitis. *Arch Surg* 1983;**118**:1352.

Delayed puberty

SIR,—Dr A J Chapman makes many useful points in his clinical algorithm on delayed puberty (18 May, p 1493) but also perpetuates some popular misconceptions.

There is a substantial amount of evidence that the *onset* of puberty is not delayed in boys with Klinefelter's syndrome, both from the follow up of infants identified by population screening[1-3] and from reports of paediatric endocrinology clinics.[4 5] However, the *progress* of puberty is slower, along with significantly lower levels of circulating testosterone.[6] As regards the intellectual functioning of these boys, all groups find an increased incidence of delayed speech development and difficulty in learning to read at school, but on formal intelligence testing the differences, though statistically significant, are small.[1]

As a point of information for Dr J Burns (8 June, p 1745), it is not necessary to write to Professor Prader to obtain an orchidometer (although those who prefer turned wooden beads may wish to); there are yellow polyethylene ellipsoids of volume 1-25 ml, known as Test-size, which can be obtained from Resimed SA, PO Box 775, CH 1122, Geneva 1, Switzerland.

SHIRLEY G RATCLIFFE
GARY BUTLER

Medical Research Council, Clinical and Population Cytogenetics Unit,
Western General Hospital,
Edinburgh EH4 2XU

1 Stewart DA, Bailey JD, Netley CT, *et al*. Growth and development of children with X and Y chromosome aneuploidy from infancy to pubertal age. In: Stewart DA, ed. *The Toronto study in birth effects*. New York: Alan R Liss, 1982;**18**:99-154.
2 Ratcliffe SG. Klinefelter's syndrome in children: a longitudinal study of 47,XXX boys identified by population screening. In: Bandmann H-J, Breit R, eds. *Klinefelter's syndrome*. Berlin: Springer-Verlag, 1984:38-47.
3 Salbenblatt JA, Bender BG, Puck MH, Robinson A, Fairman C, Winter JSD. Pituitary-gonadal function in Klinefelter syndrome before and during puberty. *Pediatr Res* 1985;**19**: 82-6.
4 Schibler D, Brook CGD, Kind HP, Zachmann M, Prader A. Growth and body proportions in 54 boys and men with Klinefelter's syndrome. *Helv Paediatr Acta* 1974;**29**:325-33.
5 Topper E. Dickerman Z, Prager-Lewin R, Kaufman H, Maimon Z, Laron Z. Puberty in 24 patients with Klinefelter's syndrome. *Eur J Pediatr* 1982;**139**:8-12.
6 Ratcliffe SG, Bancroft J, Axworthy D, McLaren W. Klinefelter syndrome in adolescence. *Arch Dis Child* 1982;57:6-12.

SIR,—Dr J Burn asked for a supplier of the Prader orchidometer. Such an orchidometer made of wood (Testometer) is available from AB Alexander Graf, Scheelevägen, S-302 39 Halmstad, Sweden, at the price of US $43. In each package an instruction is included with a graph of reference values of testicular volumes in Swedish boys.[1]

JOHN TARANGER

Pediatric Outpatient Clinic,
Västra Frölunda Hospital,
S-421 48 Västra Frölunda,
Sweden

1 Taranger J, Engström I, Lichtenstein H, Svennberg-Redegren I. Somatic pubertal development. *Acta Paediatr Scand* 1976; Suppl 258:121-35.

Should all casualty radiographs be reviewed?

SIR,—Mr J Wardrope and Dr P M Chennells (1 June, p 1638) suggest that all casualty radiographs should be reviewed by an experienced radiologist. We recently undertook a retrospective study of casualty radiograph reporting over one month. There were 3392 new attendances over this period and 2448 radiographic examinations were performed. Radiographs are normally reported within 24 hours, except at weekends, by either a consultant or a trainee of at least one year's experience, a copy of the casualty card being available at the time of reporting. Our findings were broadly in agreement with the findings in the article, but we would like to make two points.

Firstly, the Leeds group omitted to study those patients admitted or referred to other departments. This subgroup comprised 18·1% of the accident and emergency patients who underwent *x* ray examination during our study. In this subgroup we found a disagreement rate of 13·2% between the interpretations of the casualty officer and the radiologist. For those patients managed in the accident and emergency department the disagreement rate was 8·2%, which can be compared with the 6·2% figure in the Leeds study. Another important subgroup not mentioned are those patients about whose radiographs the casualty officer expressed uncertainty in his interpretation. In our study uncertainty was expressed in 5·1% of total cases. In this subgroup we found a much higher disagreement rate of 48%. The uncertainty rate for a prospective study might have been higher, and the 10% figure found by de Lacey *et al*[1] would support this.

Secondly, in the absence of 24 hour immediate reporting by radiologists an aid to the reduction of errors in interpretation may be available from the radiographer. One of us has had personal experience, as a casualty officer, of a system in which the radiographer returned accident and emergency radiographs which she considered abnormal in a different coloured envelope. This system was found to be very helpful. Berman *et al* suggest that although radiographers and casualty officers may miss a similar number of abnormalities about 44% of casualty officers' "misses" were detected by the radiographer.[2]

Other studies have also shown a significant number of errors of interpretation of casualty radiographs in the casualty department.[3 4] We would recommend that all casualty radiographs, including those of patients admitted or referred to other departments, should be reported on by an experienced radiologist within 24 hours. When possible an immediate report, by a radiologist, should be obtained in cases of uncertainty.

N DUIGNAN
J T LAWSON

Department of Radiology,
The Ulster Hospital,
Dundonald BT16 0RM

1 de Lacey G, Barker A, Harper J, Wignall B. An assessment of the clinical effects of reporting accident and emergency radiographs. *Br J Radiol* 1980;**53**:304-9.
2 Berman L, de lacey G, Twomey E, Twomey B, Welch T, Eban R. Reducing errors in the accident department: a simple method using radiographers. *Br Med J* 1985;**290**:421-2.
3 Mucci B. The selective reporting of *x* ray films from the accident and emergency department. *Injury* 1983;**14**:343-4.
4 Nolan TM, Oberklaid F, Boldt D. Radiological services in a hospital emergency department—an evaluation of service delivery and radiograph interpretation. *Aust Paediatr J* 1984; **20**:109-12.

Haemoperfusion in chloroquine poisoning

SIR,—There are few indications for the use of haemoperfusion to accelerate the removal of drugs in poisoned patients. The case presented by I Kantola and M Errko and others (11 May, p 1394) was not one of these. Haemoperfusion should be considered only when: (*a*) the toxic substance is readily adsorbed on to charcoal or resin and is present in plasma in large amounts—that is, the plasma level of the drug is high and its distribution volume is small; (*b*) the patient is severely poisoned and his clinical condition progressively deteriorates in spite of aggressive supportive therapy.

Chloroquine has a large volume of distribution (93·6 l/kg) in the blood and is highly concentrated in the red cells.[1] The authors did not measure or calculate the amount of the drug removed through the column. It was probably very low.

The clinical improvement of the patient and the rapid drop of the plasma concentration do not indicate that haemoperfusion is of value in the treatment of chloroquine poisoning. Aggressive supportive measures are sufficient to obtain a complete recovery in most cases, and it has been shown that haemodialysis and haemoperfusion increase the total body clearance of chloroquine only slightly.[2]

No details are given on the blood salicylate concentrations and the biochemical disturbances noted in this case. Consequently, it is impossible to decide if haemoperfusion was indicated for the treatment of the aspirin poisoning. Haemoperfusion (or haemodialysis) is of interest only in severely poisoned patients (salicylate concentration >900 mg/l) when alkalinisation and supportive measures have proved to be ineffective.

R GARNIER
J ELMALEM

Centre Anti-Poisons de Paris,
Hôpital Fernand Widal,
75010 Paris, France

1 Ritschel WA, Hammer GV, Thompson GA. Pharmacokinetics of antimalarials and proposals for dosage regimens. *Int J Clin Pharmacol Biochem* 1978;**16**:395-401.
2 Heath A, Ahlmen J, Mellstrand T, Wickström I. Resin hemoperfusion in chloroquine poisoning. *J Toxicol Clin Toxicol* 1982-83;**19**:1067-71.

Misleading false positives

SIR,—In a recent review of reports on screening for intrauterine growth retardation there were at least four papers[1-4] (two from the *BMJ*) which used false positive rate as a measure in evaluation. It