“Squelch sign” in detection of small herniae

Sir,—It may sometimes be difficult to detect the presence of a small inguinal rupture, particularly if it is hard to be convinced of the presence of a cough impulse. The following method has been found to be valuable.

The patient is examined standing. If the clinician is right-handed he examines the patient while standing on his right. He places his hand flat over the patient’s inguinal canal and applies pressure with a quick, jerky movement of the fingers (similar to that used in “dipping”) to detect the presence of ascitic fluid. If a hernia is present the contents of the sac are felt sliding back into the abdominal cavity, producing a “squelching” sensation.

A similar sensation can sometimes be felt with a large lipoma of the cord. Nevertheless, when one is in any doubt about the presence of a hernia and a cough impulse cannot be elicited with certainty a positive “squelch sign” may be a useful aid to diagnosis.

W G EVERETT
Addenbrooke’s Hospital, Cambridge

Papilloedema in patients taking perhexiline maleate

Sir,—We were interested in the report of “Raised intracranial pressure due to perhexiline maleate” by Dr W P Stephens and others (7 January, p 21), but we question the conclusion reached in their title. The three patients described had bilateral papilloedema; investigation excluded an intracranial mass or hydrocephalus and the fundal appearances improved when perhexiline was stopped. Measurement of cerebrospinal fluid (CSF) pressure was not made during the initial evaluation, but one man had a pressure of 130 mm Hg (presumably 130 mm H2O) six weeks after the perhexiline was stopped, when his fundus had returned to normal.

Papilloedema has been noted previously in three patients who presented with a peripheral neuropathy caused by perhexiline.1 The CSF pressures were not included in this report, but a high CSF protein concentration was noted, up to 10 g/l. Although there was no clinical evidence for a neuropathy among Dr Stephens’ patients, nerve conduction studies might have revealed as one patient (case 3) complained of numbness and weakness of the limbs and all had ataxia of gait.

We recently investigated a 52-year-old man with a peripheral neuropathy and papilloedema; he had taken perhexiline maleate for one year. No structural intracranial lesion was defined and his CSF pressure was normal. The CSF protein level was elevated at 1.46 g/l. Four months after stopping perhexiline there had been substantial improvement in his neurological state and fundal appearances.

We feel that the cause of papilloedema in patients taking perhexiline needs to be identified for there is as yet no proof of elevated intracranial pressure. (We accept that intermittent pressure elevation might explain the findings in our patient.)

Papilloedema is sometimes seen in association with the Guillain-Barré syndrome. Abnormalities in CSF protein and changes in CSF pressure have been implicated in this context, but the mechanism for papilloedema remains unknown.2

W M HUTCHISON
JOHN WILLIAMS
JEFFREY GAWLER
Department of Neurological Sciences
St Bartholomew’s Hospital,
London E1

Thyroid disease and asthma

Sir,—Your leading article (5 November, p 173) omits discussion of the possibility that the link between these two types of disease might be the iodine contained in the medicines given to asthmatics.

The syndrome of iodine-induced thyrotoxicosis (iod-Basedow) is well recognised. A recent review by Weaver et al1 suggests that the increase in a variety of thyroid abnormalities may be related to the iodine supplements added to our diet. Those who take large and continuing doses of potassium iodide, among them many asthmatics, are the more likely to show evidence of toxicity from this potentially harmful element.

ELINOR D U POWELL
Victoria, BC, Canada

Pathogenesis of acute appendicitis

Sir,—I would like to endorse the findings of Mr L W L Horton (24-31 December, p 1672) that related to the pathogenesis of acute appendicitis. At the suggestion of Mr Denis Burkitt we also have studied the histological findings in 30 consecutive cases of acute appendicitis. Appendices were opened longitudinally when fresh, as described by Mr Horton. In our series the inflammation was total or confined to the distal part of the appendix in 90%, of cases. Inflammation of the proximal part of the appendix occurred in only one case.

Similarly, in our series, faecoliths were found in only 10%, of cases, but an obstructed lumen due to faeculent or purulent material was found in 60%, or. These findings would tend to confirm the low-fibre theory of obstruction causing acute appendicitis suggested by Mr Horton.

J R JOHNSON
Royal Berkshire Hospital,
Reading, Berks

Vasovagal shock after insertion of intrauterine device

Sir,—With reference to your expert’s answer to a question on this subject (24-31 December, p 1645), while it is to be hoped that all clinicians will use care and gentleness when dealing with patients and while this approach would do not reduce patients’ anxiety, it remains a fact that inhibition of action (unless unpleasant experience can at times be dangerous. This matter was dealt with by you in 1970 in a leading article! which was followed by a letter from me2 pointing out the increased risk during the insertion of intrauterine devices. To attempt to remove a device while a patient is suffering from vasovagal shock could result in more serious complications. The correct treatment is to inject atropine intravenously. (Unless premedication has included the use of atropine even light anaesthesia will not prevent this potentially fatal complication.) The value of the atropine administration can be confirmed by observing that the pulse rises to a normal value some 60 s after the injection. The answer to the question put to your expert should have been: “A syringe and solution of atropine suitable for intravenous administration.” No clinic at which intrauterine contraceptives are inserted ought to be without this drug, and other resuscitation equipment should be available. As your expert writes, “Heaven forbid that any other resuscitative measures should need to be considered,” but this cannot mean that the possibility is to be ignored.

D N MENZIES
Liverpool Maternity Hospital, Liverpool


•••Our expert replies: “What Mr Menzies suggests is sensible and reasonable, though what I recommended should really be enough. His suggestion that removal of the device might increase the shock is probably more theoretical than real.”—Ed, BMJ

A case of self-diagnosis

Sir,—I read Dr Eoin O’Brien’s article (24-31 December, p 1648) with enjoyment, but I suspect that he is mistaken in attributing his malaise to the Portuguese man-o’-war Physalia physalis. Strong winds blow physalia from warmer waters to the south and west coasts of the British Isles by acting on its sail-like float. Although occasionally it arrives in great numbers,1 it makes little headway into the Irish Sea, where it is rare.1 I know of no records from the Irish shore as far north as the Martello Tower from which Buck Mulligan gazed out over this “snortgreen scrotum-tightening” sea.2 No unusual influx of physalia was reported in the British Isles in 1977 when, I presume, Dr O’Brien had his unpleasant experience. It is unlikely, therefore, that this species was present at the Forty Foot.


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Moreover, Dr O'Brien's symptoms, which were late in onset and lacked the intense burning pain usually felt at the site of contact with phylalia, are suggestive of a less potent stinger.

The most likely animals to have stung Dr O'Brien are the true jellyfish. They swim through the water column and consequently are less obvious to bathers than phylalia, which is tied to the surface by its iridescent-blue float. The lion's mane jellyfish Cynneospicula, made famous by Conan Doyle in 'The Adventure of the Lion's Mane', is the most probable species. It occurs more sparingly than the other common species (but less so than phylalia) and it was abundant nearby on my only visit to the area in 1977 (September).

In addition, its dense brush of fine tentacles could readily cause the type of lesion described by Dr O'Brien, whereas the long, heavily-armed tentacles of phylalia might be expected to leave streak marks. Other possibilities are C lamanchi, Chrysaora isoecla, and Rhizostoma octopus. On the grounds of distribution, behaviour, and symptoms any of these would be more likely than phylalia.

Interestingly, the man-a-war's more potent stinger might have provided Dr O'Brien with a less grievous diagnostic challenge.

Brian West
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Factor VIII-related protein in renal failure with prolonged bleeding time

SIR,—I thank Dr G Remuzzi and his colleagues for acknowledging the use of our method for immunohistological localisation of factor VIII-related protein in their admirable study in renal disease (14 January, p 70). Of course, protein localised by the method could exhibit normal, increased, or decreased activity relevant to haemostasis. For instance, some patients with von Willebrand's disease with grossly prolonged bleeding times have normal plasma levels of the protein as measured by electromunnoassay although it does not seem to function effectively. I agree with Dr Remuzzi and his colleagues that care should be taken in interpreting immunohistological appearances in functional terms.

A L Bloom
Department of Haematology, University Hospital of Wales, Cardiff

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Arthritis simulating thrombosis on urokinase scan

SIR,—I read with interest the report by Dr G P McNeill and others (14 January, p 81) which demonstrated abnormal uptake of 99mTc-urokinase in arthritis which he was physically ill-treated. It has been shown that urokinase labelled with 99mTc loses the radioactive marker quite rapidly in vitro and in vivo both in experimental animals and in patients.1 In vitro approximately 50% of the technetium label dissociates from urokinase in one hour at room temperature. Free technetium as perchnetate is used as a point-scanning agent, acute forms of arthritis in general showing positive uptake.2 3 I would like to venture the opinion that the abnormal joint uptake was not due to labelled urokinase but to free 99mTc.

For accuracy, I would like to correct an error in the comments of Dr McNeill and his colleagues, who state that 99mTc-urokinase localises in blood clots and quote the work of K Koolwijk et al.1 Their investigator did show that urokinase localises in blood clots, but they certainly did not show that 99mTc-labelled urokinase localised there. Technetium was not "discovered" until several years after that work. It also has been shown that urokinase labelled with technetium does not retain the biological activity of unlabelled urokinase and almost certainly fibrinolytic activity is lost during the labelling process.2

I Ross McDougall
Division of Nuclear Medicine, Stanford University Medical Centre, Stanford, California


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Medicine and apartheid

SIR,—At a time of increasing concern about repressive measures taken by the South African Government we should like to point out some examples of the effect of apartheid on people who practise medicine in South Africa.

The death of Steve Biko has been widely publicised. However, it is not well recognised that Mr Biko was a medical student at the University of Natal until he was expelled after becoming politically active. In the words of Dr Kurt Waldheim, "he suffered constant persecution, but was never charged with any offence, even under arbitrary South African laws." As Professor R Hoffenberg has pointed out (14 January, p 112), one of the doctors concerned "admitted that he wrote out a 'highly incorrect' medical certificate" on Biko following his imprisonment at the request of a security police colonel, in grave violation of professional ethics.

Among other cases, which details have been obtained by Amnesty International, are the following.

Dr Hoen Hoefjeje, a dentist from Durban, was arrested in August 1977 and reportedly found hanged in his cell four hours later. Although the police allege suicide, Dr Hoefjeje's family consider this unlikely, and, being a Muslim, he would have been averse to suicide on religious grounds. Photographs of Dr Hoefjeje's body showed multiple injuries of recent origin, probably inflicted with a blunt instrument shortly before his death.

Dr Matshe Mokopa of the King Edward VIII Hospital, Durban, was arrested in 1974 after an attempt had been made to organise a political rally. After three months' detention without charge, during which he was physically ill-treated, he was brought to trial, convicted, and sentenced to six months' imprisonment. He is now held in the notorious Robben Island prison.

Dr Mamphela Ramphela, superintendent of the Zanezempilo clinic at Umzimcandu's Town, was detained without trial from August to December 1976 after attending a post-mortem examination of a prisoner who was alleged to have committed suicide. Although a charge has never been brought against her, she has been banned and restricted to...