volume. Of the over 200 fetuses with bilateral hydronephrosis we evaluated, only five met the prenatal diagnostic criteria for open fetal surgery.⁵ Three fetuses had return of normal amniotic fluid dynamics, and all three survived the neonatal period and had adequate pulmonary function.

Our prenatal diagnostic and selection criteria have greatly improved, and fetal intervention for congenital bilateral hydronephrosis is capable of reliably decompressing the obstructed urinary tract, restoring amniotic fluid dynamics, and preventing otherwise fatal pulmonary hypoplasia. Whether in utero decompression reverses or arrests dysplastic changes in the kidneys and prevents long term renal failure remains an open question.

MICHAEL T LONGAKER
N SCOTT ADZICK
MICHAEL R HARRISON

Division of Pediatric Surgery and
the Fetal Treatment Program,
University of California,
San Francisco Medical Center,
San Francisco, California 94143,
United States

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Screening for antibodies to anaesthetics

SIR,—Based on some 3.5 million general anaesthetics given each year and the figures quoted by Drs D W Noble and P L Yap of serious anaphylactoid reactions (1 in 5000 to 1 in 20 000),¹ 175-700 reactions would be expected each year. Last year we examined 144 serious (operation disrupting and life threatening) reactions in detail, including laboratory investigation, and gave advice on twice that number. We estimate that we assay only 5-10% of all the reactions in the United Kingdom, suggesting an overall morbidity problem of perhaps 5000-10 000 serious reactions each year.²

Drs Noble and Yap also use the data of Harle *et al.*³ to discredit the test on the grounds of false positive response. Harle *et al.* advanced reasons for such presensitisation. These are valid and would still imply the non-use of suxamethonium in these patients. With the more specific tests developed in collaboration with Pharmacia and with Dr E Assem we found very few positive responses in patients who had not received suxamethonium previously.² This we attribute to a test specific to the whole molecule and not to the "universal" quaternary ammonium group common to this group of compounds and to a wide range of foodstuffs and cosmetics. Results of radioallergosorbent tests must be expressed in quantitative and not qualitative terms, and though by analogy to conventional allergen testing with this technique (for pollens or dust mite) clinical significance would be expected only for high titres, it is possible that intravenous drug administration increases the risk even at low titres. Indeed it may be more analogous to food allergens than to the airborne allergens.

With the usual polypharmacy necessitated by modern anaesthesia, who would attempt to distinguish the sensitive drug in a reaction by

taking an "adequate drug history"? We have just examined a reaction thought to be due to propofol, that was almost certainly due to acute blood loss and shock from hypovolaemia. We have no doubt that the case resides (to ICI's cost) on the files of the Committee on Safety of Medicines as a reaction to propofol.

We agree with Drs Noble and Yap that prescreening would be an expensive exercise, but there is nothing to prevent the Association of Anaesthetists taking the initiative and organising trials. The greatest danger to the patient undergoing general anaesthesia is probably not the drugs but the erroneous belief that anaphylactoid reactions are rare—they are an uncommon phenomenon but not rare.

There are no data as yet on the effectiveness of prescreening, but already radioallergosorbent testing is used retrospectively in cases of severe reaction to determine the causative drug. All that we ask is that prescreening is considered in a balanced way rather than on the initial cost of the test itself. The cost of examining a high risk group may well be £10-£20 per patient, but this must be viewed against the cost of disrupted lists, overnight stays in intensive care, and, at worst, brain damage. It is worth remembering that the courts are currently awarding between £250 000 and £500 000 for brain damage.

JOHN WATKINS
A MILFORD WARD

Supraregional Protein Reference Unit,
University Department of Immunology,
Royal Hallamshire Hospital,
Sheffield S10 2JF

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Enteropathy induced by non-steroidal anti-inflammatory drugs

SIR,—The editorial by Dr A K Banerjee¹ is based to a large extent on our research at Northwick Park. With one major exception, all the manifestations described are subclinical: this exception is better described in a definitive article than in the abstract cited.^{2,3}

The strictures of the small intestine are rare but are of immediate importance to the practising surgeon. The history of small intestinal obstruction—acute or subacute—in a patient who has regularly been taking non-steroidal anti-inflammatory drugs for months or years should alert the surgeon. No obvious cause of obstruction will be found at laparotomy; no strictures can be felt. Previous or subsequent barium studies, either a small bowel meal or enema, are likely to be normal. The surgeon may or may not remove a length of small bowel but is unlikely to remove enough, and the natural instinct to conserve small bowel may commit the patient to a second or even a third laparotomy. Small bowel strictures have now been diagnosed prospectively in several patients at Northwick Park, and similar patients are being referred to us for study before returning to the referring hospital for surgery. At the time of surgery the small bowel needs to be inflated with carbon dioxide through a nasogastric tube, and the strictures will then become visible as areas of narrowing of the small intestine.⁴ The strictures do not show transmural inflammation and are less distinct from those of Crohn's disease.

In all other patients the problems are subclinical for 90% of the time and may surface, like an iceberg, showing different facets. Differentiation from Crohn's disease is easy by scanning using

leucocytes labelled with indium-111 as patients with Crohn's disease have a normal scan within four hours, whereas the other patients show abnormal results 18 hours after the ¹¹¹In labelled leucocytes have been given. Without such scans precise diagnosis is not possible, and a mild form of Crohn's disease is the most likely diagnosis. Patients with either condition will respond to treatment with sulphasalazine.

We do not recommend withdrawal of non-steroidal anti-inflammatory drugs even in proved cases, as most patients continue to need these drugs. Even when drugs are withdrawn the inflammation may take up to 16 months to improve, though the response to sulphasalazine is more rapid⁵ and may be associated with a reduced need for non-steroidal anti-inflammatory drugs.

I BJARNASON
J M GUMPEL

Northwick Park Hospital,
Harrow HA1 3UJ

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Inhibin and hydatidiform mole

SIR,—Dr T Yohkaichiya and colleagues reported interesting new data on the placental protein inhibin, particularly with respect to high serum values in seven patients with hydatidiform mole (before evacuation).¹ The possibility that such measurements may have a useful role in diagnosis despite the general reliability of ultrasound and equivocal value of measurements of other trophoblastic proteins will require further investigation because of the small numbers of patients so far studied.

The suggestion that inhibin measurements will contribute to the management of sequelae after hydatidiform mole will also require more data. We note the absence from Dr Yohkaichiya and colleagues' paper of the criteria used for diagnosing persistent trophoblastic disease requiring therapeutic intervention. These can be important: whereas four of the seven patients in their series were treated by chemotherapy, only about 8% of patients in the United Kingdom require chemotherapy.² If measuring inhibin were to result in many patients receiving chemotherapy unnecessarily this would be questionable progress.

K D BAGSHAWE R H J BEGENT
E S NEWLANDS G J S RUSTIN

Department of Medical Oncology,
Charing Cross Hospital,
London W6 8RF

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Correction

Fluconazole and phenytoin: a predictable interaction

A production error occurred in the graph accompanying this drug point by Mr A S Mitchell and Dr J T Holland (13 May, p 1315). The y axis should have been labelled "Plasma phenytoin concentration (μmol/l)," and the graph should not have included the point indicating a plasma phenytoin concentration of 100 μmol/l on 15 May.