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Author's reply

EDITOR,—The clinical evaluation of the acute scrotum in children is difficult, but I have yet to see a study correctly evaluating the value of radionuclide isotope imaging solely in this population. All of the studies quoted by C J Luscombe and colleagues have been performed predominantly in adults. Children are uncooperative, and their testes are smaller, which must increase the diagnostic error. For instance, in a large series of children with an acute scrotum Lewis *et al* reported a sensitivity of 95.4% and a specificity of 85.7% for radionuclide imaging,¹ which is not as clear cut as Luscombe and colleagues imply. Fenner *et al*, whom Luscombe and colleagues cite, stated, "We believe its [radionuclide imaging's] routine use in clinical practise is limited" and "would result in needless delays and unjustifiable expense."² This is from advocates of the technique.

There is no doubt that delay in surgery results in ischaemia, infarction, and testicular loss. Any diagnostic strategy has to be designed with this as the bottom line. If a nuclear medicine department can perform and report such an investigation within an hour of a request being made by the accident and emergency department, presumably at any time of the day or night, then the investigation may have a role. If the department cannot do this then rapid exploration is the option that is safest (to the testicle). The economic argument is spurious: one false negative scan leading to legal action will wipe out any pecuniary advantage.

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Don't count on urinary white cells to diagnose childhood urinary tract infection

EDITOR,—Because pyuria is common in febrile children,¹ screening for urinary tract infection in children by detecting increased urinary white cells (with leucocyte esterase strips or standard microscopy) will produce false positive results. Because urinary white cells may disappear rapidly,² such screening will also produce false negative results. We report on a child with a devastating urinary tract infection who had very few urinary white cells for another reason.

An extremely ill 8 month old boy presented after 36 hours of diarrhoea, vomiting, and fever. He was drowsy and dehydrated but did not have any localising signs. On the ward, phase contrast microscopy of a suprapubic urine specimen showed $>10^{11}$ rods/l but $<20 \times 10^6$ white cells/l; 12 hours later the presence of *Escherichia coli* was confirmed in urine and blood. A culture of cerebrospinal fluid was sterile. Creatinine and electrolyte concentrations indicated established oliguric acute renal failure, with a fractional excretion of sodium of 2.0%. Ultrasonography showed a solitary, non-obstructed, right hydronephrosis and hydronephrotic. His haemoglobin concentration

(109 g/l) and platelet count ($175 \times 10^9/l$) were in the low normal range, but his neutrophil count was very low at $0.9 \times 10^9/l$, and most cells seemed immature. At 13 hours he had a cardiorespiratory arrest (despite a normal potassium concentration of 4.8 mmol/l) and could not be resuscitated. Postmortem examination showed haemorrhagic adrenal glands and severe acute pyelonephritis but no urothelial inflammation.

The fact that this boy had few urinary white cells was probably related to the low number of circulating blood neutrophils, a pattern of blood film that is well recognised with overwhelming infections.³ To avoid the diagnostic pitfalls of counting white cells when screening for childhood urinary tract infection we now use phase contrast microscopy of fresh, unspun urine to identify bacteria.² This is done routinely in our wards and clinics by permanent nursing and medical staff and is taught to all new junior doctors. Its most obvious advantage is that the result is instantly available, allowing antibiotics to be given immediately when the result is positive (as in this case). Contamination is also identified immediately, allowing a repeat sample to be collected without delay. Samples yielding negative results (the majority) can be discarded, which saves laboratory costs. A minor advantage is that, when a maintained instrument is available, studying a urine specimen by microscopy is quicker than filling in a laboratory form.

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Cerebral oedema after ingestion of MDMA ("ecstasy") and unrestricted intake of water

EDITOR,—3, 4-Methylenedioxymethamphetamine (MDMA or "ecstasy") is a synthetic derivative of amphetamine and a popular recreational drug among young people. It is known to cause severe hyperpyrexia, dehydration, and rhabdomyolysis.¹ Unrestricted ingestion of water is commonly believed to be important in combating some of the side effects of ingestion of the drug. We report on two patients who developed clinical effects due to the syndrome of inappropriate secretion of antidiuretic hormone after ingestion of MDMA. These adolescents had been "left to sleep it off" and presented late.

Case 1—A 15 year old girl presented semiconscious and having fits 18 hours after ingesting an unknown quantity of alcohol and MDMA. She had been left to sleep by friends after a party. On examination she had a score on the Glasgow coma scale of 5/15, was hypertonic with dystonic movements, and was apyrexial but sweating. The initial serum sodium concentration was 119 mmol/l and plasma osmolality 156 mmol/kg. The urine osmolality was 655 mmol/kg and urine sodium concentration 6 mmol/l when plasma values were 270 mmol/kg and 129 mmol/l, respectively. The creatine kinase level was 3249 U/l (normal 24-195). Severe fluid restriction improved the serum osmolality and corrected the hyponatraemia over 24 hours. Computed tomography showed moderate cerebral oedema. Toxicological examination of urine

confirmed ingestion of MDMA. She recovered fully with no neurological sequelae.

Case 2—A 16 year old girl presented 21 hours after a party at which MDMA, alcohol, and amphetamines had been available. Fifteen hours after being left to sleep she had been found semiconscious with dystonic movements and agitation. Her score on the Glasgow coma scale was 11/15. She was apyrexial. The initial serum sodium concentration was 112 mmol/l and plasma osmolality 142 mmol/kg. Urine osmolality was 184 mmol/l and urine sodium concentration 99 mmol/l. Computed tomography showed mild cerebral oedema. MDMA and methyldeoxyamphetamine were detected in urine and gastric aspirate. The creatine kinase value was 1157 U/l. With fluid restriction her electrolyte concentrations returned to normal over 24 hours. She recovered fully but had some anterograde and retrograde memory loss.

Three recent case reports have linked use of MDMA with the syndrome of inappropriate secretion of antidiuretic hormone.²⁻⁴ Our patients developed cerebral oedema secondary to this syndrome. We suggest that the current practice of encouraging unrestricted intake of water needs to be reviewed. Rhabdomyolysis did not occur in our patients, but creatine kinase values were initially high. It is potentially dangerous to assume that an adolescent with altered consciousness after a party is intoxicated with alcohol and can be "left to sleep it off."

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Managing patients with an absent or dysfunctional spleen

Guidelines should highlight risk of salmonella infection in sickle cell disease

EDITOR,—The guidelines for the prevention and treatment of infection in patients with an absent or dysfunctional spleen recognise sickle cell disease as being an important cause of a dysfunctional spleen.¹ We are surprised, however, that they do not mention the infection that occurs most frequently in patients with this disease.

In districts such as ours, which serves a large population with sickle cell disease (about 620 patients are registered), *Salmonella* spp are the micro-organisms most commonly isolated from blood cultures. In a review of 4884 bacteraemias at our hospital during 1976-91 *Salmonella* spp accounted for 11 of 21 bacteraemias in patients with sickle cell disease, while *Streptococcus pneumoniae* and *Haemophilus influenzae* type b were responsible for only three and one respectively. In addition, if Gram negative bacilli were seen on microscopy of blood samples from patients with sickle cell disease in whom culture yielded positive results, *Salmonella* spp were cultured in nearly 70% of cases.² This high incidence of salmonella bacteraemia may not be universally appreciated by all clinicians. Despite aggressive treatment, salmonella osteomyelitis developed in about a third of cases. The emergence of resistance to antibiotics in *Salmonella* spp presents further problems in the management of osteomyelitis in sickle cell disease.³

The risk and consequences of salmonella infection in sickle cell disease are well established, though patients' susceptibility to this micro-organism remains unexplained. Simple preventive measures, however, may limit exposure to the micro-organism. We recommend that all patients with sickle cell disease who develop diarrhoea are investigated for infection with *Salmonella* spp and given antibiotics. If Gram negative bacteraemia is found to be present an antibiotic that is effective in salmonella infection should be started. Other measures to reduce the risk of salmonellosis—for example, the provision of education about scrupulous food hygiene and information on food sources of salmonella—should also be taken.

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Under half of doctors know that antibiotic prophylaxis should be life long

EDITOR,—Despite several articles in the medical press over the past two years, including communications to all doctors from the chief medical officer, we were dismayed to note that several elderly patients admitted to our wards who had had splenectomies years previously had not been given advice on prophylaxis against infection or relevant immunisation. Deodhar *et al* found that in 184 patients who had had a splenectomy during a 12 year period 58% had not received advice or prophylaxis against infection and only 36% had received pneumococcal vaccination.¹

We recently assessed doctors' knowledge of prophylaxis after splenectomy by means of a questionnaire survey. An anonymous questionnaire was sent to 160 hospital doctors of all grades and 200 general practitioners. A total of 118 questionnaires was returned for analysis (69 (43%) by the hospital doctors and 49 (25%) by the general practitioners). Most of the doctors (116/118) knew that patients who had had a splenectomy were at risk of pneumococcal infection. However, only half (34) of the hospital doctors and a third (16) of the general practitioners knew that patients were at risk of meningococcal infection and malaria. Most of the respondents (50 (72%) of the hospital doctors and 27 (55%) of the general practitioners) knew about the risk of *Haemophilus influenzae* infection. Although there was general awareness about antibiotic prophylaxis, only seven (14%) of the general practitioners and 34 (49%) of the hospital doctors knew that this prophylaxis should be life long.

Six of the general practitioners had a computerised splenectomy register, and 20 said that they would like more advice and information on managing patients who had had a splenectomy. The general practitioners had (to their knowledge) a total of 107 patients who had had a splenectomy registered with their practices; the commonest reasons for the operation were trauma, idiopathic thrombocytopenic purpura, and lymphoma.

Awareness and implementation of guidelines for preventing and treating infection in patients

with reduced or absent splenic function is essential.² This could be helped by the setting up of computer databases on patients. The guidelines in our district are accessible on the pathfinder system, which is a computerised information system available in our hospital and to a number of general practitioners. Hopefully, this system will become available to all general practitioners; the guidelines and other information would then be easily accessible.

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Patients expect splenectomy in rural Zaire

EDITOR,—The working party of the British Committee for Standards in Haematology's Clinical Task Force has produced comprehensive guidelines on preventing and treating infection in patients with an absent or dysfunctional spleen.¹ Massive splenomegaly is common in north east Zaire, mainly owing to the occurrence of the tropical splenomegaly syndrome in a region holoendemic for falciparum malaria. Patients with this syndrome present in the outpatient department with pain and a large mass extending from the lower left costal margin. They expect the hospital to perform a splenectomy to relieve their discomfort.

Splenectomy is dangerous here because facilities for blood transfusion are limited. The seroprevalence of HIV in our region is roughly 7%,² so the procedure carries an appreciable risk for the surgeon. The risks of overwhelming infection after splenectomy are high as vaccination against *Streptococcus pneumoniae* and *Haemophilus influenzae* infections is not possible. Lifelong prophylaxis with oral phenoxymethylpenicillin and chloroquine is difficult to assure as the population is mobile and patients may live beyond the reach of even basic medical facilities.

In view of these difficulties splenectomy should be performed only if the patient's life is in danger—that is, for splenic trauma with uncontrolled haemorrhage or hypersplenism with severe anaemia (haemoglobin concentration less than 50 g/l with symptoms of anaemia). Splenic tissue should be conserved when possible and the patient informed of the need to take lifelong antibiotic and malarial prophylaxis.

The main role of doctors in this region with regard to splenectomy is to educate patients and dissuade them from having the operation except in life threatening situations. Instead of operating we should be providing simple analgesia for the pain and treating the tropical splenomegaly syndrome with long term antimalarial drugs.

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Data linkage is useful in setting up a register

EDITOR,—We wish that the guidelines on preventing and treating infection in patients with an absent or dysfunctional spleen¹ had included advice on ways of ensuring that all patients at risk are contacted and reminded about the need to carry a splenectomy card and other precautionary measures. One way of doing this is by means of a register.

Grampian Health Board has established a register of patients who have had a splenectomy since 1984. This was coordinated by the department of public health medicine as part of its monitoring of and advice on immunisation. Patients were identified from both routine hospital discharge data and pathology records because of concern about the completeness of these sources of data.^{2,3} Many patients die shortly after splenectomy because of underlying disease. To minimise the work in establishing the register, linkage of hospital discharge data and death certificates⁴ by the information and statistics division of the Common Services Agency, Edinburgh, was used to exclude patients who had died. This excluded 98 of the 315 patients initially identified.

For efficient identification of patients at risk we recommend using discharge data, pathology records, and data linkage supplemented by additional information from general practitioners and hospital clinicians. Local data linkage with the community health index can then be used to help keep such a register up to date.

We thank S Kendrick for supplying the data linked information.

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Uncertainty exists over frequency of blood tests to test antipneumococcal immunity

EDITOR,—I wonder whether the British Committee for Standards in Haematology would comment on the level of antibody to pneumococcus that indicates protection against infection in patients who have had a splenectomy and have received pneumococcal vaccine.¹ General practitioners need further guidance on the frequency of blood tests to assess immunity to pneumococcal infection in these patients. Should we first test six weeks after vaccination, then three years later, and thereafter every year?

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Is there evidence to show that daily antibiotic treatment is best?

EDITOR,—The recent guidelines on preventing and managing infection in patients with an absent or dysfunctional spleen recommend lifelong prophylaxis with oral antibiotics for all