are metabolised, and the serum sodium drops to less than 150 mmol/l. Intravenous nutrition may then be restarted at a level indicated by the carbon dioxide production (calories) and the nitrogen loss (amino acids). Do not increase water intake.

Case 2 has a different aetiology. The positive sodium balance would be expected to give a rise in serum sodium of 25 mmol/l. The observed rise suggests a shift of water from intracellular to extracellular compartments of 9% of extracellular volume as a result of excessive sodium intake. The water shift is in the reverse direction from that seen in cases 1 and 3. Diagnosis: Sodium overload. Management: The sodium intake should be restricted and water intake increased, since the intracellular compartment is depleted. There is no indication for withdrawing intravenous nutrition.

The "Lessons of the Week" are therefore: (1) A high serum sodium concentration may be due to dehydration, carbohydrate calorie overload, or sodium overload. (2) Careful attention to glucose metabolism, sodium and water balance, and transcellular shifts can differentiate the causes and hence delineate the management of the problem.

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[1] Allen SC, Balfour IC, Wise CC. *J Clin Path* (in press).

SIR,—The problem of "inexplicable" hypernatraemia in very sick patients undergoing intensive therapy is all too familiar, but I found the explanation offered by Drs R W G Prescott and J C Stoddart in their article (22 March, p 847), on this topic less than convincing.

Two of their patients were shown to have positive "corrected" water balance with a negative sodium balance, which seems to me to compound rather than explain the problem. I endorse their statement that parenteral nutrition can induce dehydration but believe that this is usually due to osmotic diuretic mechanisms in cases where the blood sugar is inadequately controlled or the administered amino-acids are poorly utilised. While hypertonic glucose solutions do have a fractional water content considerably less than unity, 1 g of glucose when metabolised produces 0·6 ml of water, so that 1 litre of a 40% glucose solution (for example, Glucoplex 1600) although only containing 764 ml of water, produces an additional 240 ml of metabolic water. Whether or not this was taken into account, if we assume an accurate sodium balance these patients must have been considerably more dehydrated than even the "corrected" fluid balances indicated.

Perhaps the real "lesson of the week" is that it is almost impossible to assess water balance accurately in very sick, febrile hypermetabolic patients, because of the inevitable errors incurred in estimating metabolic water production and water losses via skin and lungs. Accurate weighing, which is now a simple procedure in the intensive care unit, reduces many of these errors but there remains the problem due to changes in body cell mass, which really can only be resolved by radioisotope techniques.[1]

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[1] Shizgal HM, Spanier AH, Kurtz RS. *Am J Surg* 1976;**131**:156.

Bacterial infection in the newborn

SIR,—Dr H B Valman (15 March, p 772) relegates chlamydial neonatal conjunctivitis to a mild condition. The spectrum of clinical symptoms is wide, from severe conjunctivitis indistinguishable from that produced by gonococcus (which may coexist) to little more than a "sticky eye." In an unpublished study at this hospital *Chlamydia trachomatis* was responsible for over 40% of purulent conjunctivitis. Up to 50% of the babies born to women harbouring *C trachomatis* in the cervix will develop chlamydial conjunctivitis.[1]

In-vivo antibacterial studies have shown that chloramphenicol is unreliable in eradicating *C trachomatis*.[2] The organism will be suppressed, but the frequency of relapse is high, often after the baby has been discharged from hospital. A mild conjunctivitis may persist, which while usually self-limiting may result in impairment of sight.[3] A further effect of chloramphenicol therapy will be the failure to isolate the organism in the laboratory if a diagnosis of *C trachomatis* infection is considered after therapy has commenced. The use of neomycin for the early treatment of ophthalmia will be as effective as chloramphenicol against staphylococci and coliforms, but will not impair the ability to make a firm diagnosis of chlamydial infection subsequently. If the conjunctivitis has not improved at 36 hours, a presumptive diagnosis of chlamydial ophthalmia should be made, and in the absence of a laboratory diagnostic service treatment with chlortetracycline locally plus erythromycin systemically should be commenced.[4] Treatment should be continued for three weeks. Investigation and treatment of the parents are mandatory.

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Symptomatic treatment of primary pneumatosis coli with metronidazole

SIR,—We were interested to read the report by Mr B W Ellis of the treatment of primary pneumatosis coli with metronidazole (15 March, p 763). As he points out, there is increasing evidence to suggest that gas-producing bacteria are responsible for the maintenance of these gas cysts, and the work of Van der Linden and Hoflin[1] and ourselves[2] on breath hydrogen excretion in these patients supports this hypothesis. Despite this, bacteriological studies have so far failed to identify any causative organisms and the underlying cause of the initiation of cyst formation remains obscure.

Treatment of symptomatic pneumatosis coli with antibiotics is an attractive prospect since oxygen therapy requires hospitalisation and is somewhat inconvenient for the patient. We also have occasionally observed clinical improvement in patients given antibiotics, one case of which[3] is quoted by Mr Ellis, and have therefore been attempting a more systematic appraisal of such treatment. As yet we have no consistent evidence of benefit. Indeed, we have had to abandon a course of metronidazole in one of our patients because of rapid worsening of symptoms.

There are theoretical reasons for caution in the use of antibiotic therapy in pneumatosis coli. The maintenance or resolution of pneumocysts may be critically dependent on the partial pressure of hydrogen within the colon and this in turn is determined by the balance between hydrogen-producing and hydrogen-catabolising colonic bacteria. Net hydrogen production may be increased or decreased by antibiotic therapy so a variable response of pneumatosis coli to antibiotics may be expected. It is even possible that the association of pneumatosis with chronic pulmonary disease arises as a *consequence* of the repeated antibiotic therapy these patients receive.

Until more information is available we would urge that antibiotic therapy, including metronidazole, be used with caution in these patients, possibly being restricted to those in whom oxygen therapy has failed or is contraindicated. While we share Mr Ellis's desire for a simple form of therapy, we believe that more experience is needed to establish the effectiveness and safety of antibiotic regimens for pneumatosis coli.

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Volkmann's ischaemic contracture

SIR,—Following your recent leading article on Volkmann's ischaemic contracture (16 February, p 430) and the unhappiness about this condition in Toronto to which you refer,[1] it seems worth reporting the experience of supracondylar fractures in the Royal Hospital for Sick Children, Edinburgh, over a period of almost 30 years, during which the condition appears to have been eliminated with respect to this fracture.

In the three years 1947-9 inclusive 71 fractures required surgical intervention. Eight were explored because of suspected arterial damage at presentation. One of these patients developed an ischaemic contracture, but he had been manipulated no less than five times at another hospital before referral to Edinburgh. Open exploration of the newly presenting supracondylar fracture was abandoned following the realisation that the circulation was almost always restored by gentle manipulation of the fracture in order to relieve an entrapped or compressed artery (rather than to coapt the bone fragments). Stabilisation after reduction in as flexed an attitude as is compatible with adequacy of the peripheral circulation and the avoidance, unless absolutely essential, of an encircling plaster of paris cast are most important also. From January 1950 to June 1975 28 out of 486 patients with closed fractures presented with vascular problems. None was explored and none developed an ischaemic contracture. Four other patients developed signs of vascular compromise during their first manipulation, but suspension of the limb to minimise oedema and prevent extrinsic pressure on the artery allowed restoration of the circulation. One patient developed a temporary partial median nerve palsy and autonomic disturbance in the hand, but no contracture. Of the 23 open operations in this period, 21 were for persistent unacceptable deformity after manipulation. The