close observation and to give them antibiotics only on clinical indications.

Since 1976 no infants have been found dead or moribund in the postnatal wards. Measurement of the respiratory rate in the way described is the only effective method of observing an infant next to his mother in a dark postnatal ward. It has been acceptable to the nurses and reassuring to the mothers.

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Use of local anaesthetic drugs in hospital practice

Major adverse reactions have been produced by intravenous regional anaesthesia,^{1,2} and three deaths have been reported in the national press.³ We report the results of an inquiry into the use of local anaesthetic drugs by a cross section of junior hospital doctors.

Method

A questionnaire comprising seven questions was answered by 57 junior doctors below the grade of senior registrar, drawn from many specialties and trained in various hospitals. The questions were presented to the doctors in the course of one afternoon and all those approached answered the questions.

The doctors were first asked when they had last used a local anaesthetic and for what purpose. Further questions sought to find which drug had been used, its concentration, and route of administration. Finally, the doctors were asked to indicate the recommended maximum safe dose of plain lignocaine in milligrams per kilogram body weight and to state the number of milligrams per millilitre of the drug in a 1% solution.

Results and comment

The results provide some insight into the way local anaesthetic drugs are used in this hospital. Of the doctors questioned, 59.6% had used a local anaesthetic drug during the previous week and 80.6% within the previous month. Only one doctor had not used a local anaesthetic within the past year. Lignocaine was the most frequently used drug (by 94.6% of respondents). The majority (64.3%) used lignocaine by local infiltration for minor surgical procedures. The second most common mode of administration was topical (30.3% of respondents)—mainly for use in bladder catheterisation, bronchoscopy, eye surgery (0.5% proxymetacaine), and ear, nose, and throat procedures (cocaine).

Comment

Although 0.5% lignocaine is an adequate concentration for local infiltration,⁴ 31 out of 36 doctors used a higher concentration. Four out of 16 doctors used topical lignocaine without knowing its concentration and 68.4% did not know the maximum recommended dose of lignocaine for infiltration; 71.4% did not know how to calculate the dose they were giving.

Although many doctors claimed to know the safe dose of lignocaine (in terms of volume of a known percentage solution) for a man weighing

70 kg, there are many instances where more accurate calculations of the dose must be made—for example, when used in the elderly and children. The results are perhaps not surprising since information regarding the correct dose of local anaesthetic is often presented in a way that requires calculations to be made.⁵

We have designed a simple chart (figure) whereby the correct maximum volume of lignocaine solution can be easily calculated from the patient's body weight and the percentage solution to be used. It also shows graphically the undoubted advantage of using the most dilute solution that will effectively produce analgesia.

Maximum safe volume of plain lignocaine according to body weight. (Maximum safe dose of lignocaine—without adrenaline 3 mg/kg, with adrenaline 7 mg/kg; uses of different solutions—0.25-0.5% for infiltration and intravenous regional anaesthesia, 1% for nerve blocks, epidural anaesthesia, and intravenous regional anaesthesia, 2% for nerve blocks.)

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Overdose of trilostane

Trilostane is a new blocker of synthesis of glucocorticoids. We report the first recorded case of trilostane overdose.

Case report

A 29 year old woman was admitted after ingesting lorazepam 50 mg, 40 tablets of a combination diuretic (each containing hydrochlorothiazide 50 mg and amiloride 5 mg), and 60 trilostane capsules (each containing 60 mg). She was taking trilostane for premenstrual tension, which was thought to be a component of her long standing depression. There was no history of hypertension or renal impairment. On examination she was drowsy but

conscious. Her pulse rate was 90 beats/min and blood pressure 90/70 mm Hg. There were no other abnormal physical signs. Plasma urea concentration was 2.7 mmol/l (16.2 mg/100 ml), sodium 136 mmol(mEq)/l, potassium 3.3 mmol(mEq)/l, bicarbonate 23 mmol(mEq)/l, and glucose 6.3 mmol/l (113 mg/100 ml).

Gastric lavage was performed five hours after ingestion of the tablets. As trilostane blocks synthesis of glucocorticoids she was treated with hydrocortisone 30 mg/day by mouth and her pulse rate, blood pressure, and plasma potassium concentration were monitored carefully. Her blood pressure remained in the range 100/60 mm Hg to 120/80 mm Hg; plasma potassium concentration decreased to 3.1 mmol/l but did not fall further.

Comment

Trilostane is a new blocker of synthesis of glucocorticoids. It blocks the enzyme 3 β -hydroxysteroid dehydrogenase by competitive inhibition, thus lowering production of cortisol, aldosterone, and deoxycorticosterone.¹ Its major indication is in the treatment of Cushing's syndrome and primary aldosteronism,² and it is being used experimentally in premenstrual tension to alter the sex hormone balance.

There are no reports of overdosage in humans. In rats and mice the lethal dose for 50% is greater than 16 000 mg/kg body weight (Sterling-Winthrop, unpublished observations). The theoretical risk is of an Addisonian crisis and an increase in serum potassium concentration. Trilostane decreases the potassium loss induced by thiazide diuretics,³ which is of interest in this case as a thiazide diuretic was also taken.

We conclude that trilostane overdosage with 3600 mg was safe in our patient and emphasise that in cases of overdosage blood pressure and plasma potassium concentration should be carefully monitored and physiological replacement with glucocorticoids given. This may be important if the use of trilostane for treating premenstrual tension continues.

We are grateful to Dr J Costello for permission to report this case.

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"Claudication" of the sciatic nerve

We report a case of "ischaemic" sciatica that responded dramatically to reconstructive surgery. We believe that this hitherto undescribed syndrome may justifiably be termed "claudication" of the sciatic nerve.

Case report

A 57 year old housewife, a heavy smoker, presented with a history of an episode of acute back pain seven years previously. Eighteen months before admission she had experienced further sudden lumbar pain after lifting, which had radiated down the back of the right leg to the ankle, suggesting a further disc protrusion. Although the initial severe pain had improved, a constant ache persisted at rest, aggravated by sitting, standing, and walking and relieved by lying flat. Its distribution corresponded to the territory supplied by S1. The persisting quality of the pain was unlike that of a disc prolapse. The striking feature was that walking, in particular, worsened the pain in the right buttock and leg in a way that indicated a claudication effect related to ischaemia of the sciatic nerve. Walking also precipitated an additional distinct pain in the muscles of the right calf, thigh, and buttock after a distance of 30 m; this was typical of intermittent claudication and

forced her to stop at 200 m. The second pain subsided after five minutes' rest, although the pre-exercise pain of sciatic distribution remained.

On examination there was normal spinal movement and straight leg raising to 90° was painless bilaterally. Knee jerks were equal and ankle reflexes absent. Only weak ankle pulses were felt on the right. All pulses were palpable in the left leg. A lumbar x ray film showed minor osteophyte lipping at L2 and L3. Doppler studies confirmed a reduced resting ankle systolic pressure index of 0.85 on the right, falling to 0.37 after exercise. The left side was normal. A translumbar aortogram (figure) showed bilateral atheromatous iliac stenoses, more severe on the right. At operation bilateral semiclosed aortoiliac endarterectomy was performed with Dacron patch angioplasty: strong foot pulses were restored. The next morning she reported that the continuous pain in the back and right leg had gone.

Aortograms obtained before (top) and after (bottom) operation, showing how severe occlusive disease of aortoiliac segment (arrowed) was rendered widely patent by arterial reconstruction (arrowed).

Postoperative Doppler studies were repeatedly normal. Further aortography showed a widely patent aortoiliac system (figure), and a selective study confirmed that the right internal iliac circulation was normal. She subsequently remained free of symptoms during 18 months of follow up.

Comment

Cord ischaemia due to lumbar artery occlusion and claudication of the cauda equina as a result of spinal stenosis¹ are well known. Pain in the distribution of the sciatic nerve caused by aneurysm has been described.² Claudication, a term first used by Dejerine³ in relation to the spinal cord, was extended by Blau and Logue⁴ to claudication of the cauda equina. Claudication of the sciatic nerve has not been previously reported. The inferior gluteal branch of the internal iliac artery supplies the sciatic nerve in the gluteal fossa. Impotence and gluteal claudication in Leriche's syndrome⁵ result from low pressure in the internal iliac arteries.

Low back pain and buttock pain are often difficult to interpret on clinical grounds. We considered that, in addition to having pain caused by degenerative osteoarthritis, our patient had pain of sciatic distribution that was ischaemic in origin as it was aggravated by exercise and improved, but not abolished, by resting. More importantly, it was immediately and completely abolished by internal