complicating factor. The effect of opiates on respiration in healthy cats (references 5 and 6 in Dr Woodcock's letter) is hardly comparable to responses in an ill man with chronic lung disease. But the observation reinforces our point that when this man was well after his discharge from hospital he, indeed, showed no response to naloxone. He responded only when he was severely ill, and the greatest response in terms of ventilation was with the bolus study on the day before the infusion study.

Since our paper was accepted for publication, we have given naloxone to more patients in respiratory failure and have found one further patient with a convincing response in rate and depth of ventilation and in blood gases while severely ill but no response after recovery.

Dr Webb's point about the use of a cuirass was one that we discussed. Because of his palsied diaphragms he was provided with a rocking bed, which greatly helped him subjectively both at home and in hospital, where it was also shown to improve his night-time desaturation. He had no evidence of upper airways obstruction. The role of naloxone is not irrelevant as the response in oxygen saturation occurred only during naloxone infusion. We are not suggesting that naloxone is the treatment of choice in acute respiratory failure, but we do believe that our conclusion that endorphins may play a part in some patients with acute respiratory failure is valid, and that this case report should act as the basis for a formal study of this response both in the investigation of the mechanism involved and therapeutically.

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Mapping cancer mortality

SIR,—We would like to comment on recent correspondence (17 April, p 1190) following our paper on variations in cancer mortality (13 March, p 784). With regard to the main point made by Dr M A Sydney, we would like to emphasise that we are not using the death returns for each area, where we would agree that deaths from rare tumours may tend to be lost among a group of "other cancers." Rather, as described in our paper, we have information on each individual death, and so can look at separate ICD codes (even using the fourth digit where applicable) in all local authority areas however small or sparsely populated. With the statistical criteria we have used, the latter type of area will appear on the map only if the excess risk is very high —to guard against a multitude of false-positive findings. On the other hand, it is possible that true-positives may be covered over too, and local informed epidemiology of the kind Dr Sydney mentions will always be important.

Dr J K Howard is concerned about false-positive findings. He is not correct, however, to dismiss Bexley as a north-Kent dormitory suburb of London—the area we mapped was the London Borough of Bexley, which is more extensive. It is known to have contained within its boundary factories which employed workers subject to the asbestos industry regulations, and mesotheliomas have been reported.[1] Areas neighbouring Gillingham, in particular Chatham and Rochester, are in fact high for pleural mesothelioma in men but not sufficiently to satisfy the criteria we used for mapping.[2] As regards Rushden, an exceptionally high proportion of the male workforce (27% in 1971) is employed in the boot-and-shoe industry, and many of them have been involved in a particular type of manufacture—the welted process—which is more dusty than other processes. We are now studying Tower Hamlets in detail to ascertain whether there are further risk industries, but we do know that it contained substantial furniture and leather industries.

One of the objects of the atlas which we are preparing is to supply clues for further investigation rather than definitive answers. We are aware of some at least of the difficulties involved in the presentation and interpretation of these data and will aim to avoid them.

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[1] Health and Safety Executive. *Selected written evidence submitted to the Advisory Committee on Asbestos 1976-77*. London: HMSO, 1977.
[2] Gardner MJ, Acheson ED, Winter PD. *Br J Cancer* 1982 (in press).

Explosive bullets: a new hazard for doctors

SIR,—We disagree that explosive bullets are a new development. Professor Bernard Knight's leading article (13 March, p 768) does not emphasise sufficiently the differences between mechanical methods of making bullets expand on impact and truly exploding bullets. The former attempts to increase the energy transfer in the target by mechanical deformation of the bullet, usually by exposing the lead core of the bullet as in certain hunting ammunition or altering the shape of the bullet as in hollow point bullets. It was these types of bullets that were forbidden by the Hague convention, which tried to exclude from military use any bullet that did not have a complete metal jacket. Designers of military rounds of low velocity may attempt to circumvent the convention by deliberately designing the bullets to tumble in the target as is the Russian 5·45 mm and the British ·303 Mk VII, 7, VIIz, and 7z. This effect can also be achieved in handgun ammunition—for example, Arcane, which is solid copper produced in France. The trend towards high-velocity military rounds has introduced the effect of "tissue flaking" to add to the tumbling effect. All these bullets are safe to handle at operation or necropsy. The exploding bullet has been available for many years; currently it is found in the Russian 7·62 mm × 54 R machine gun ammunition. An internal striker sets off a charge of tetryl and phosphorus, the resulting flash can be used for ranging of the explosion or for incendiary effect. The internal striker is held in a collar and often may be heard to rattle. Such ammunition is quite safe to handle once it has been discharged and is inside the target.

In the quest for more destructive bullets some further systems are now marketed, and these are fragmenting bullets, available commercially in the United States for rifle, handgun, or shotgun ammunition. Their general design is of a central cavity filled with black powder or Pyrodex, with or without mercury additive, with a ball-bearing behind the charge and a magnum primer in front. The quantity of explosive is small, and once again if the bullet has failed to detonate on impact with the target, it is unlikely that handling by forceps is going to cause an explosion. The suggestion, however, that goggles should be worn when exploring a wound or handling these bullets is a sensible one.

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Audit of computerised recall scheme for cervical cytology

SIR,—The recent paper by Dr Maureen Reynolds and Dr Clive Richards on the cervical cytology recall scheme (8 May, p 1375) draws attention to the complexity of the bureaucracy which has been built up for the purpose of writing individually to well women who have had previous normal smears, and also reminds us that only those women who have had previous smears are reached by it. Does this effort serve a useful purpose? In these enlightened times why should not the DHSS adopt the simpler and cheaper alternative of placing advertisements in women's magazines or elsewhere to inform all women of the availability of screening? The modern woman does not really need the help of a computer in order to find her way to an Ayre spatula every few years and might, indeed, resent the computer storage of so much personal data with no better justification.

So far as recall of women with dyskaryotic smears is concerned, why should not the present multiple-copy report form be modified to include an abridged report to be posted to the woman herself? This could take the place of the copies hitherto sent to the national recall centre and to the health authority, and would simply tell the woman whether her smear was normal or whether, on the other hand, she should return to her doctor for further investigation. The resulting saving in administrative costs would go some way towards funding the routine screening of younger women, who increasingly seem to be at risk of premalignant dysplasias of the cervix.

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Insulin given intranasally induces hypoglycaemia in normal and diabetic subjects

SIR,—In your periodical you have published a paper from Dr A E Pontiroli and others (30 January, p 303) on the effect of insulin given intranasally. The authors support with their results our previous observations on the same topic published as early as 1958.[1] We studied the resorption of insulin as well as of adrenalin from the nasal mucosa. In animal experiments the resorption was thoroughly followed by making comparisons between nasal application and submucosal injections.

After having finished these experiments, the effect of insulin given by nasal tampons was studied on healthy and diabetic patients. Later

on nasal application of "asthmolysine" (containing adrenalin and posthypophyseal hormone) was measured on patients suffering from asthma. We found that both hormones were resorbed from the nasal mucosa and that about 53% of the effect of the same subcutaneous dose could be achieved by this route.

However, the effect was unstable and variable, and, therefore, we did not propose intranasal insulin treatment for the treatment of diabetes. In contrast to this, in certain cases of asthma the intranasal application of antiasthmatic preparations could enlarge our therapeutic possibilities.

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[1] Hankiss J, Hadhazy CS. *Acta Med Acad Sci Hung* 1958;**12**:107-14.

Extensor digitorum brevis—a predictor of neuropathy in the leg?

SIR,—Dr C T Kirkpatrick (23 January, p 238) suggests that wasting of extensor digitorum brevis is "a reliable clinical predictor of electrophysiologically demonstrable nerve disease," and failure to palpate it, "an excellent predictor of disease in the peroneal nerve."

I report here on a 15-year-old white girl with physical findings of peroneal muscular atrophy and cavus feet, mild weakness of foot dorsiflexion, inversion and eversion, absent Achilles deep tendon reflexes, and diminished sensation in a stocking-glove distribution. Muscle enzymes were normal. Nerve conduction velocity study of the peroneal nerve demonstrated a terminal latency of 8·3 ms, conduction velocity proximally of 18 m/s and distally of 15 m/s, with a response voltage (polyphasic) proximally of 2·0 mV, mid 2·6 mV, and distally 4·5 mV. These findings were consistent with a severe chronic polyneuropathy. The clinical diagnosis was type I—hereditary motor-sensory neuropathy (Charcot-Marie-Tooth syndrome).

This young woman presented for surgical correction of her foot deformity. Examination of the feet revealed observable and palpable hypertrophy of the extensor digitorum brevis (fig 1). During surgery (metatarsal and calcaneal osteotomy) a biopsy taken from this muscle (fig 2) showed the stigmata of chronic denervation (areas of small group atrophy and scattered small angular fibres).[1] A nuclear magnetic resonance carbon

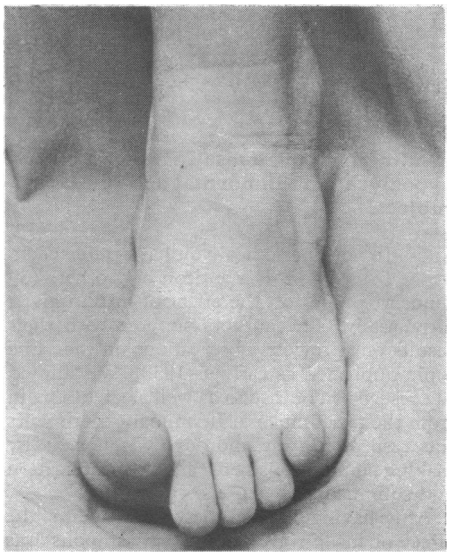

FIG 1—Hypertrophy of extensor digitorum brevis.

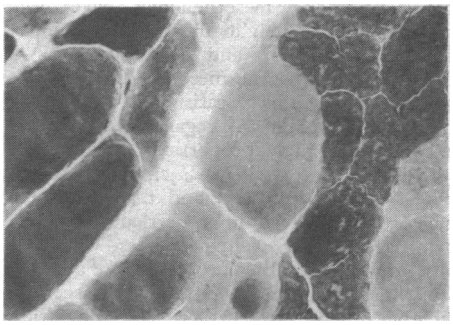

FIG 2—Muscle biopsy—small group atrophy and scattered small angular fibres (ATPase—pH 9·4).

spectrum study of the specimen was compatible with chronic neuropathy.[2]

It would seem that the extensor digitorum brevis muscle attempts to compensate for progressive weakening of the long dorsiflexors of the toes and foot from whatever etiology, neuropathic or myopathic. A true postural work hypertrophy occurs in this muscle, and unless or until the time-intensity product of the primary disease is great enough to cause atrophy, it remains palpable.

We have observed this phenomenon in early chronic neuropathy and early and late in the scapuloperoneal syndrome[3] and so-called limb-girdle dystrophy. Although the exception does not always prove (or disprove) the rule, consideration should be given to stage of the disease when making a clinical judgment such as the one in question.

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[1] Dubowitz V, Brooke M, eds. *Muscle biopsy: a modern approach*. London: WB Saunder:, 1973 123-5.
[2] Barany M, Chalovich J, Burt C, Glonek T. In: Schotland DL, ed. *Disorders of the motor unit*. New York: John Wiley and Sons, 1982:697-714.
[3] Brooke M. *A clinician's view of neuromuscular diseases*. Baltimore: Williams and Wilkins, 1977: 116.

Adrenergic mechanisms in control of plasma lipid concentrations

SIR,—I read with interest the paper by Dr J L Day and others (17 April, p 1145). The results of their study show that selective and non-selective beta-blocking agents and beta-blockers with weak intrinsic sympathomimetic activity increase mean plasma triglyceride and decrease high-density lipoprotein (HDL) cholesterol and free fatty acid concentrations. Pindolol is an unselective beta-blocker with high intrinsic sympathomimetic activity. The concentrations of serum free fatty acids and triglycerides remained about constant during six months' treatment of 20 hypertensive patients with pindolol.[1] The concentration of HDL-cholesterol was increased after the first month of treatment, but there were no significant differences between the concentrations of HDL-cholesterol before the treatment and after three and six months of treatment. The total serum cholesterol concentration was slightly lower after six months than after one month of treatment. The ratio of HDL-cholesterol to total cholesterol increased from 0·18 to 0·20 during treatment, but this increase was not statistically significant.

Dr Day and others propose that unopposed alpha stimulation inhibits adipose tissue lipoprotein lipase with subsequent changes in plasma triglyceride and HDL-cholesterol concentrations. No significant changes of adipose tissue lipoprotein lipase activity occurred during treatment, however, with sotalol[2] or pindolol.[1] Lecithin cholesterol acetyltransferase is an enzyme participating besides lipoprotein lipase in lipoprotein metabolism. Lecithin cholesterol acetyltransferase activity was significantly higher during pindolol treatment than after three weeks' break of treatment.[1] Comparing the results of the study of pindolol with reports of other beta-blockers suggests that pindolol might have fewer untoward effects on lipid metabolism than beta-blockers without high intrinsic sympathomimetic activity.

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[1] Lehtonen A, Hietanen E, Marniemi J, Peltonen P, Niskanen J. *Br J Clin Pharm* 1982;**13**,suppl No 2: 445-8.
[2] Lehtonen A, Hietanen E, Marniemi J, Peltonen P, Nikkilä EA. *Int J Clin Pharm* (in press).

Hypersensitivity to local anaesthetics: a direct challenge test with lignocaine for definitive diagnosis

SIR,—I would like to comment on two points in the paper by Dr M R Barer and Dr M K McAllen (24 April, p 1229) on hypersensitivity to lignocaine. The first point is their doubt that true anaphylaxis to lignocaine has ever been shown and the second their use of chlorpheniramine to "cover" challenge testing.

I was recently referred a patient who had been told that she had had a severe allergic reaction to lignocaine some years previously. From her story I suspected that the reaction was due to overdosage and proceeded to skin testing mainly to reassure her. The case has been reported in detail elsewhere,[1] but in brief she had a systemic reaction to 0·2 ml 0·5% bupivacaine, which was confirmed by serial changes in complement and which responded to treatment with chlorpheniramine.

Hypersensitivity to all amide local anaesthetics is extremely rare, but this proved reaction to one drug of the group together with the history of an earlier reaction to lignocaine would indicate that she has a true hypersensitivity to these drugs including lignocaine. She responded very quickly to chlorpheniramine, and I wonder if pretreatment with that drug might not have masked the reaction. Subsequent injection of a normal dose of local anaesthetic might have proved disastrous.

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[1] Brown DT, Beamish D, Wildsmith JAW. *Br J Anaesth* 1981;**53**:435-7.

Missed injuries of the spinal cord

SIR,—I was most interested to read the report from Bournemouth Hospital of a patient with hypothermia and an undiagnosed spinal cord injury (1 May, p 1334). May I suggest, however, that the principal reason that this patient's injury was not diagnosed was not her hypothermia but the failure of anyone to gather and record an adequate history from the ambulance crew that initially brought the patient to hospital.