

colleagues that routine pelvimetry should be performed only postpartum or after a caesarean section, difficult forceps delivery, or perinatal death. Is it not possible that if we had known about that nasty pelvis before we would not have traumatised or killed the baby delivering it vaginally but have made the correct decision to deliver it abdominally? Surely for a risk of only 1 in 30 000 it is well worth while having this extra piece of information with which to program our personal computer.

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Oral dihydroergotamine in management of cluster headache

SIR,—Symonds¹ made a significant advance in the management of cluster headache when he introduced the prophylactic injection of ergotamine as mentioned in your leading article (22 November, p 425). However, we would advocate a trial of oral dihydroergotamine before resorting to parenteral injection.

We recently recorded² the case of a 55-year-old woman who had suffered intermittently from classical migraine since the age of 16. Her symptoms always began with a flickering light over the left visual field. This persisted for about an hour and overlapped with a right-sided throbbing headache. The headache lasted usually for a few hours but had persisted for up to 24 hours. A Cafergot suppository (ergotamine tartrate 2 mg, caffeine 100 mg, belladonna alkaloids 0.25 mg, isobutylallyl barbituric acid 100 mg) would usually relieve her headache within about two hours. She occasionally noticed at the height of her headache that the right eye would start to water and that her right nostril felt blocked. After 27 years of these very intermittent symptoms a cyclical pattern began to emerge. Her headaches would occur almost daily for spells of up to two months, with periods of freedom for up to a year. It proved possible to control her symptoms without recourse to injections. She has been well maintained on dihydroergotamine 1 mg twice daily.

We would agree with you that the distinctive clinical features of the condition "still often escape recognition." We suspect that this is especially true in women, in whom the time relationships are often less precise, the cluster of headaches may last longer, and the whole symptomatology may be less clearly defined than in men.³ It is nevertheless an eminently treatable condition. As you say, "in few conditions are patients so grateful for relief of their symptoms."

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¹ Symonds, C P, *Brain*, 1956, 79, 217.

² Herzberg, L, et al, *Journal of Neurology, Neurosurgery and Psychiatry*, 1975, 38, 648.

³ Bickerstaff, E R, in *Handbook of Clinical Neurology*, ed P J Vinken, and G W Bruyn, vol 5, p 111. Amsterdam, North-Holland Publishing Co, 1968.

Serum creatine phosphokinase and malignant hyperpyrexia

SIR,—We must take issue with Dr M A Deborough (15 November, p 408) in his continued assumption that serum creatine phosphokinase (CPK) levels are an indica-

tive parameter in identifying patients susceptible to malignant hyperpyrexia.

Abnormal CPK levels have been found in a wide range of variegated disorders, including acute psychosis and epilepsy,¹ tetanus,² myocardial infarction and cardiac disease,³ prolonged coma or cerebrovascular disease,⁴ and muscular dystrophy and musculoskeletal disorders,⁵ and with muscular exercise.^{6,7} The administration of suxamethonium causes a marked and significant rise in serum CPK, which is potentiated by halothane in normal patients.^{8,9}

We have been studying the family¹⁰ of a man who died of malignant hyperpyrexia and whose daughter developed malignant hyperpyrexia on exposure to nitrous oxide¹¹ although she had a normal serum CPK level. Little attention has been focused on the problems of normal serum CPK levels in susceptible families and it seems unwise to place too much emphasis on the measurement of this enzyme as a prognostic guide. Muscle biopsy and in-vitro testing remains the surest form of screening.

Until a simple diagnostic test is available unexpected cases will occur during anaesthesia and we must rely on the routine monitoring of temperature during anaesthesia and the availability of a standard treatment pack.

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¹ Meltzer, H Y, et al, *Archives of General Psychiatry*, 1970, 22, 398.

² Brody, I A, and Hatcher, M A, *Archives of Neurology*, 1967, 16, 89.

³ Dreyfus, J C, et al, *Revue Française d'Études Cliniques et Biologiques*, 1966, 5, 386.

⁴ Wright, N, et al, *British Medical Journal*, 1971, 3, 347.

⁵ Walton, J N, *Disorders of Voluntary Muscle*. London, Churchill, 1964.

⁶ Vejjiava, A, and Teasdale, G M, *British Medical Journal*, 1965, 1, 1653.

⁷ Rose, L I, *Journal of Applied Physiology*, 1970, 29, 355.

⁸ Innes, R K R, and Strømme, J H, *British Journal of Anaesthesia*, 1973, 45, 185.

⁹ Tammisto, T, and Airaksinen, M, *British Journal of Anaesthesia*, 1966, 38, 510.

¹⁰ Ryan, D W, and Appleyard, T N, *British Journal of Anaesthesia*, 1975, 47, 1001.

¹¹ Ellis, G R, et al, *British Medical Journal*, 1974, 4, 270.

Neonatal-strength ampoules of nalorphine

SIR,—In correspondence with the makers of Lethidrone (nalorphine) Neonatal I have attempted to persuade them that they should discontinue production of the present multi-dose vial.

Although a recent article on neonatal resuscitation¹ does not mention the use of morphine antagonists, most practitioners in this field in Britain do recommend their use where there is presumptive evidence of depression of the baby due to opiate or pethidine administered to the mother. The ampoules of nalorphine for neonatal use contain 5 ml of solution with a concentration of 1 mg/ml. An individual dose for an infant should be of the order of 0.1 mg/kg body weight intravenously. Thus the present form of the vial exposes the infants to the twin risks of overdosage and infection. It seems irrational that we have largely rid ourselves of multidose ampoules because of the safety hazard to patients and yet we

allow the most vulnerable group at risk—the neonates with low Apgar score—to face the possibility of septicaemia or over-enthusiastic treatment of pethidine-induced respiratory depression.

In common with members of the division of paediatrics I feel that the time is overdue for the removal of multidose vials of nalorphine from the resuscitation trolley. Perhaps those of your readers who agree might care to contact the manufacturers. I have suggested as an alternative single-dose ampoules containing 0.5 mg nalorphine in 0.5 ml.

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¹ Gregory, G A, *Anesthesiology*, 1975, 43, 225.

Nutmeg poisoning

SIR,—Following the report by Dr J A Barrowman and others (5 July, p 11) of the therapeutic use of ground nutmeg (*Myristica fragrans*) in doses of nine teaspoonfuls daily in controlling the diarrhoea associated with medullary carcinoma of the thyroid we would like to add a cautionary note on the dose recommended. Our own patient was given this dose and there was a satisfactory improvement in the number of motions passed daily. Within three days, however, he complained of dry eyes and mouth, blurred vision, dizziness, tingling, and feelings of depersonalisation and remoteness. They gradually resolved as the dose was reduced.

Nutmeg poisoning is not new. Green¹ cites descriptions given by De Lobel in 1576 and Purkinje in 1829. The subject aroused interest in the *BMJ* in 1906,² when the use of nutmeg as an abortifacient was reported; many subjects failed in their original intention and suffered side effects similar to those we have described. After a single dose of 18.5 g another patient reported periods of excitement and fear of impending death alternating with clouding of consciousness.¹ More recent reports^{3,4} cover the side effects intended in its use as a hallucinogen in the hippy subculture.

Should nutmeg or its active component, myristic acid, become a regular part of therapy for the diarrhoea associated with medullary carcinoma of the thyroid we would like to emphasise the side effects and remind people that at least one fatality has been recorded.¹

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¹ Green, R C, *Journal of the American Medical Association*, 1959, 171, 1342.

² Hammond, et al, *British Medical Journal*, 1906, 1, 593; 1906, 2, 778; 1906, 2, 900; 1906, 2, 984; 1911, 2, 269.

³ Unwin, J R, *Canadian Medical Association Journal*, 1968, 98, 402.

⁴ Panayotopoulos, D J, et al, *British Medical Journal*, 1970, 1, 754.

Management of acute asthma

SIR,—I was interested in your leading article on this subject (11 October, p 65) and in Professor C M Fletcher's letter (8 November, p 345). I was particularly interested in his

statement that "no patient with acute asthma should be sent to hospital without first being given 200 mg of hydrocortisone or prednisolone intravenously or intramuscularly." As a consultant paediatrician I have always been intrigued by the fact that the majority of my child patients who come into hospital show very good response in status asthmaticus to a single injection of ACTH gel, given in a dosage equivalent to 40 to 80 units in an adult and scaled down according to the child's weight. What I find particularly surprising is that this treatment seems to work within 20-30 minutes.

I understand from some of my general practitioner colleagues that this treatment also works in the home. It would be interesting to know if other practitioners have found this very safe treatment effective and if any of our more academic colleagues can explain why ACTH gel should produce this effect within such a short space of time. With many of my very small patients it is difficult to believe that the relief of bronchospasm could be psychogenic in these circumstances. We do, of course, recognise the response of asthma to coming into hospital in those who are sensitive to the house dust mite present in their home environments but not present in the cleaner and at least less dusty environment of the hospital ward, but this response is, of course, much slower and takes hours or days.

The other first-aid measure, which seems greatly to help children in severe bronchospasm is what I call "aided respiration." In this procedure the child sits on the adult's knee facing away from the adult. The adult then wraps his arms around the child's chest and, in time, with the child's efforts at expiration, he gently squeezes the chest to encourage fuller emptying. It is most important that this manoeuvre is timed to coincide with the child's attempts at expiration, and the adult must not in any way attempt to dictate the rate of respiration. After such an aided expiration the next breath the child takes gives quite considerable relief, and this seems to restore confidence and reduce the sense of suffocation felt in a severe attack of bronchospasm. There is, of course, considerable psychogenic reassurance for the child, seated and held in this way by a concerned and empathic and helping adult.

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Deep vein thrombosis in pregnancy

SIR,—In your leading article on "Venous thromboembolism and anticoagulants in pregnancy" (22 November, p 421) no mention was made of the value of ultrasound in the diagnosis of deep vein thrombosis in pregnancy and the puerperium. Ultrasound fetal heart detectors are widely used in obstetric units in Britain and can be used to test the patency of the iliofemoral venous segment. Minor calf vein thrombi will not be detected, but it has been shown that these do not carry any significant risk of embolism.¹

The technique and its limitations are described very fully in the paper by Dr Jeanette Meadway and others (6 December, p 552). It is clear from this careful study that the technique is not foolproof and that the results must be interpreted with

common sense, but it does represent a great improvement on clinical evaluation. In pregnancy the special techniques of venography and the radioiodinated fibrinogen uptake test cannot normally be used because of the radiation hazard to the fetus. If the ultrasound test is used in pregnant patients with calf pain of uncertain cause most of them will be saved from unnecessary anticoagulants.

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¹ Kakkar, V V, *et al*, *Lancet*, 1969, 2, 230.

SIR,—Your leading article on venous thromboembolism and anticoagulants in pregnancy (22 November, p 421) emphasises that the failure to reduce death from thromboembolism during pregnancy is mainly because clinical diagnosis is inaccurate because invasive techniques such as phlebography and ¹²⁵I-fibrinogen uptake scanning are hazardous.

However, the problem may be overcome by a number of non-invasive techniques which carry no hazard to the fetus; these include thermography,¹ which has a 95% correlation with phlebography, and the measurement of the serum level of fibrin(ogen) degradation fragment E^{2,3} and plasma level of β -thromboglobulin.⁴ Preliminary results of these estimations show a considerable degree of accuracy in the diagnosis of venous thromboembolism. In our experience thermography alone has either confirmed the clinical suspicion or has avoided unnecessary anticoagulation in patients in whom this therapy would otherwise have been indicated.

In the future it may be possible to screen all patients at specific risk using one or more of the above methods, and it will be of great interest to see whether such a programme would serve to reduce the morbidity and mortality associated with venous thromboembolism in pregnancy.

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¹ Cooke, E D, and Pilcher, M F, *British Journal of Surgery*, 1974, 61, 971.

² Gordon, Y B, *et al*, *Lancet*, 1973, 2, 1168.

³ Cooke, E D, *et al*, *Lancet*, 1975, 2, 51.

⁴ Ludlam, C A, *et al*, *Lancet*, 1975, 2, 259.

Inhalation technique in treatment of asthmatic children with steroid aerosols

SIR,—The use of aerosol steroids has become well established as an important part of the management of severe childhood asthma.¹ Their place is well deserved and few would doubt their efficacy. However, there remain a few children in whom control of symptoms is inadequate without resort to alternate-day oral steroids, and we have realised recently that poor inhalation technique has been the probable reason for the inadequate response in some of these.

In the children's asthma clinic at this hospital patients have always been carefully

taught how to take aerosols when they are first prescribed. In addition to this we now ask all children to demonstrate their inhalation technique at each visit. We have found some who do not take their inhalations correctly, and control of symptoms has improved very considerably in some cases when they have been re-instructed.

We therefore suggest that asthmatic children who are receiving inhalation therapy should be asked to demonstrate the use of their inhaler as a routine part of such outpatient visits and that particular attention should be paid to this point in those children with persistent symptoms for whom oral steroid therapy is being considered.

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¹ Godfrey, S, and König, P, *Archives of Disease in Childhood*, 1974, 49, 591.

Cigarette smoking and chest pain

SIR,—I was surprised that your leading article on this subject (15 November, p 368) made no comment about gastro-oesophageal reflux as an important and common reason for anterior chest pain.¹ Reliance on demonstrating a hiatus hernia to confirm this diagnosis is misplaced.^{2,3} Cigarette smoking is a potent cause of gastro-oesophageal reflux⁴ because it causes relaxation of the lower oesophageal sphincter,⁵ and this mechanism should therefore be much more frequently considered as the cause of chest pain in smokers than your article suggests. The oesophageal acid perfusion test^{6,7} is helpful in confirming the diagnosis.

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¹ Bennett, J R, and Atkinson, M, *Lancet*, 1966, 2, 1123.

² Hiebert, C S, and Belsey, R H R, *Journal of Thoracic and Cardiovascular Surgery*, 1961, 42, 352.

³ *Lancet*, 1968, 2, 267.

⁴ Stanciu, C, and Bennett, J R, *British Medical Journal*, 1972, 3, 793.

⁵ Dennish, G W, and Castell, D O, *New England Journal of Medicine*, 1971, 284, 1136.

⁶ Bernstein, L M, and Baker, L A, *Gastroenterology*, 1958, 34, 760.

⁷ Bennett, J R, and Atkinson, M, *Lancet*, 1966, 2, 1150.

SI units

SIR,—I am directed to write to you on this subject by my committee. There is no support from the clinicians in this district, or indeed in the area of East Sussex, for the introduction of SI units at this time. It is our opinion that the use of even relatively small quantities of money to effect this change will be wrong and that absolutely no benefits to the patients will follow such a change.

The argument that it is standardisation is the purest tosh. All doctors and laboratory workers will need to carry two standards in their memories since reference to American and Scandinavian work will be large. We would like to see the change to SI units delayed until such time as the Health Service can afford even these small charges and the