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,—Symonds[1] 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 recorded[2] 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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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 indicative parameter in indentifying 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 family[10] of a man who died of malignant hyperpyrexia and whose daughter developed malignant hyperpyrexia on exposure to nitrous oxide[11] 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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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 multidose vial.

Although a recent article on neonatal resuscitation[1] 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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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. Green[1] 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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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