of a *chronic* intrauterine condition within the next 72 hours.

But I should like to mention three points: Firstly, the maternal impression of fetal movement seems to vary between one pregnancy and another even in the same woman. So diminution of movement must be assessed against a background of what the patient has been feeling over the previous several weeks, taking into account the slight reduction that is normally noticed as term approaches.

Secondly, it is necessary to ascertain that the mother is describing and estimating limb or "kicking" movements. A mother with an ill fetus often describes her baby as "just turning over." I believe this means that the fetus is very lethargic or perhaps immotile, with the patient confusing Braxton Hicks uterine contractions with fetal movement.

Thirdly, it must be realised that *acute* intrauterine catastrophes occasionally occur in seemingly normal antenatal patients. Here fetal movement ceases abruptly, and after stillbirth postmortem examination reveals no pathological changes other than those of asphyxia. The baby and placenta are not small for dates. Apart from severe abruptio placentae an obscure cause such as cord strangulation may have to be postulated; or could there be a condition analagous to "cot death" in utero? I do not believe that a fetal movement chart would help predict these rare acute deaths and I should be reluctant to request charts routinely from all apparently normal antenatal clinic patients, because of the work involved to themselves and the introspection and anxieties which could be engendered.

But for the fetus suspected to be "at risk" in utero I have found careful assessment of movements to be very helpful. May I suggest asking the mother for a report on her baby before asking the laboratory? And if there is real cause for anxiety ask both.

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SIR,—Mr J F Pearson and Miss Judith B Weaver (29 May, p 1305) have added to the growing literature underlining the importance of obtaining the maternal history of fetal activity in pregnancies at risk from chronic uteroplacental circulatory insufficiency and it is gratifying to note that their findings are broadly similar to those previously reported from this department in the small-for-dates syndrome[1-3] and from Professor Sadovsky's department in Israel.[4 5]

At this hospital I have followed my previous studies on the maternal history of fetal activity with an attempt to answer a common clinical question which will be highlighted by Mr Pearson and Dr Weaver's publication—namely, whether there is any serious connotation to a history (often volunteered) of a marked reduction in fetal activity at about term in a pregnancy which is otherwise completely normal. To do this I compared indices of fetal wellbeing in 25 patients at term with a history of a marked reduction in fetal activity of about a week's duration with those of a control group of 25 patients matched with regard to age, parity, and duration of pregnancy but reporting instead unabated vigorous fetal activity. In all cases the pregnancies were entirely normal by conventional standards. The fetal scalp blood samples were collected at the time of surgical induction of labour by low amniotomy during the 40th week.

	Reduced movements group	Vigorous movements group
Urinary oestriol (μmol/24 h), mean ± SD	180·1 ± 79·8	191·5 ± 76·3
Serum human placental lactogen (mg/l), mean (range)	5·9 (1·5->8·0)	6·3 (2·7->8·0)
Hydrogen ion (H^+) concentration in fetal scalp blood (nmol/l), mean ± SD	45·0 ± 6·0	45·0 ± 4·0
Haemoglobin concentration in fetal scalp blood (g/dl), mean ± SD	18·0 ± 1·2	17·4 ± 1·3
Liquor creatinine concentration (μmol l), mean ± SD	238·7 ± 26·5	229·8 ± 53·0
Birth weight of babies (kg), mean ± SD	3·44 ± 0·33	3·60 ± 0·38

Conversion: SI to traditional units—Urinary oestriol: 1 μmol/24 h ≈ 0·3 mg/24 h. Liquor creatinine: 1 μmol/l ≈ 0·01 mg/100 ml.

The results of these investigations, summarised in the accompanying table, show that there is generally no cause for alarm when reduced fetal movements are reported at term, assuming of course that the pregnancy is otherwise normal. Reassuring explanations such as that the baby is resting before coming can now be given in better faith.

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[1] Mathews, D D, *British Medical Journal*, 1972, **1**, 439.
[2] Mathews, D D, *Lancet*, 1973, **1**, 1315.
[3] Mathews, D D, *Obstetrics and Gynecology*, 1975, **45**, 488.
[4] Sadovsky, E, and Yaffe, H, *Obstetrics and Gynecology*, 1973, **41**, 845.
[5] Sadovsky, E, *et al*, *International Journal of Obstetrics and Gynaecology*, 1974, **12**, 75.

Febrile fits

SIR,—In 1973 Pampiglione[1] directed attention to the confusion associated with the term "febrile convulsions," stating that "it covers a wide range of circumstances and it is not uniformly defined." We completely agree with this statement and believe that inconsistency in nomenclature and classification is undoubtedly responsible for some of the contradictory and perplexing reports relative to the diagnosis, treatment, and prognosis of seizures associated with an elevation of temperature in young children. In our opinion the article by Dr Sheila J Wallace (7 February, p 333) does little more than compound and perpetuate the existing confusion, primarily because the term "febrile fit" is not adequately defined throughout the paper. Without specific description, amplification, or elaboration the appellation "febrile seizure" is essentially meaningless and therefore the reader cannot be certain in most instances whether Dr Wallace's data pertain to "complicated" febrile fits or "simple febrile convulsions."

Our current concepts concerning the diagnosis and prognosis of childhood "febrile convulsions," which are based upon three independent investigations (one retrospective and two prospective) that we conducted over the past 37 years at the Johns Hopkins Hospital Epilepsy Clinic,[2-4] are at variance with those presented by Dr Wallace. We classify convulsions associated with pyrexia in children, excluding those which occur with intracranial infections or with diseases that adversely affect the central nervous system or other disorders known to be toxic to the brain, into two groups: (*a*) simple febrile convulsions and (*b*) epileptic seizures precipitated by fever.

We make a diagnosis of a simple febrile convulsive disorder in children who present: brief, generalised seizures, seldom lasting longer than a few minutes, occurring soon after a rise in temperature; no clinical or laboratory evidence of cerebral infection or intoxication; and a normal electroencephalogram (EEG) after the patient has been afebrile for at least a week. A family history of simple febrile convulsions is very common in patients who have similar "febrile seizures." The prognosis in these children is excellent. The seizures tend to recur for several years during early childhood, and in our series there was no recurrence after six years of age. Afebrile seizures developed in only 2·9% of our patients with simple afebrile convulsions. There was no evidence that simple febrile convulsions had caused brain damage in any of our patients and we remain unconvinced that a simple febrile disorder is a precursor of psychomotor (temporal lobe) epilepsy.

A diagnosis of epilepsy precipitated by fever is strongly indicated by one or more of the following: prolonged seizures, focal convulsions of any duration, convulsions associated with fever in a child over six years of age, or EEG abnormalities such as are observed in overt epilepsy. This type of epileptic seizure may initially occur at any age and may take place at any time during a febrile episode. In our series 97% of children with epilepsy triggered by fever subsequently developed afrebile seizures.

Dr Wallace classifies a "complicated" febrile fit as one that is "prolonged [over 30 minutes], repeated within the same illness, or unilateral." We disagree with this definition, particularly with respect to the duration of the attack. We do not believe that a "febrile convulsion" must persist for "over 30 minutes" in order to be classified as "complicated." We would not, for example, regard a 20-25-minute "febrile fit" as anything other than "complicated" and would definitely consider such an episode as "epilepsy precipitated by fever." Acceptance of Dr Wallace's criterion for duration of a "complicated" febrile fit would most assuredly lull a physician whose patient experienced, for example, a 20-minute "febrile convulsion" into a false sense of security relative to prognosis.

We emphasize that proper management of the child who experiences seizures in association with fever depends upon accurate diagnosis and classification of the initial convulsion in accordance with the criteria designated in this communication.

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[1] Pampiglione, G, *Lancet*, 1973, **2**, 1035.
[2] Livingston, S, Bridge, E M, and Kajdi, L, *Journal of Paediatrics*, 1974, **31**, 509.
[3] Livingston, S, in *Advances in Pediatrics*, vol 10, ed S Z Levine, p 114. Chicago, Year Book Publishers, 1958.
[4] Livingston, S, *Comprehensive Management of Epilepsy in Infancy, Childhood and Adolescence*, p 16. Springfield, Thomas, 1972.

Perhexiline maleate in angina pectoris

SIR,—The reports by Drs D J Howard and J Russell Rees (17 January, p 133) and by Professsor F Lhermitte and his colleagues (22 May, p 1256) prompt us to give preliminary information about a safety and efficacy assessment of perhexiline maleate recently completed in the British Isles.

An open multicentre monitored release study was conducted by 85 cardiologists and physicians in order to assess the safety and efficacy of perhexiline maleate under conditions of ordinary wide-scale use in hospital clinical practice. Patients with angina pectoris were treated for periods of up to six months and liver function tests (serum aspartate aminotransferase, serum alanine aminotransferase, alkaline phosphatase, serum bilirubin) were carried out at monthly intervals. Angina attack rate, trinitrin tablet consumption, side effects, and concomitant treatment were also recorded.

A total of 363 patients received treatment with perhexiline maleate for a mean duration of 4·2 months. Their average age was 59·8 years and the mean number of anginal attacks recorded before treatment began was 95 attacks per month. Concomitant treatment during the study with beta-adrenoreceptor blocking agents was received by 32% of patients, while 34% received a wide variety of other drugs.

Thirty patients showed some abnormalities of liver function tests during perhexiline maleate therapy and as a result seven were withdrawn from further treatment. Variable increases of serum transaminases were evident in all these 30 patients, which reverted towards or to within the normal range when perhexiline maleate was withdrawn. No case of jaundice was observed.

Side effects, predominantly nausea and dizziness, were frequent during the first month of treatment, leading to withdrawal of 49 patients, but most of these effects persisted only for one month or less. Significantly more patients experienced side effects following a dose of 400 mg perhexiline maleate daily than 200 mg daily. No case of peripheral neuropathy was observed during the study, at a time when 100 patients had completed six months' treatment.

The efficacy of perhexiline maleate was demonstrated by a mean reduction of 60% in the number of anginal attacks per month after one month's treatment, and in subsequent months by a reduction of 70-75%. Complete suppression of angina was reported by 24% of patients and 73% reported a decrease of 50% or more of their attacks. The response of patients receiving concomitant treatment with beta-adrenoreceptor blocking agents was similar to that of those receiving only perhexiline maleate. There was no evidence of any interaction phenomena between perhexiline maleate and any other drug. Trinitrin tablet consumption correlated closely with the angina attack rate.

The detailed results of this study will be published in full elsewhere.

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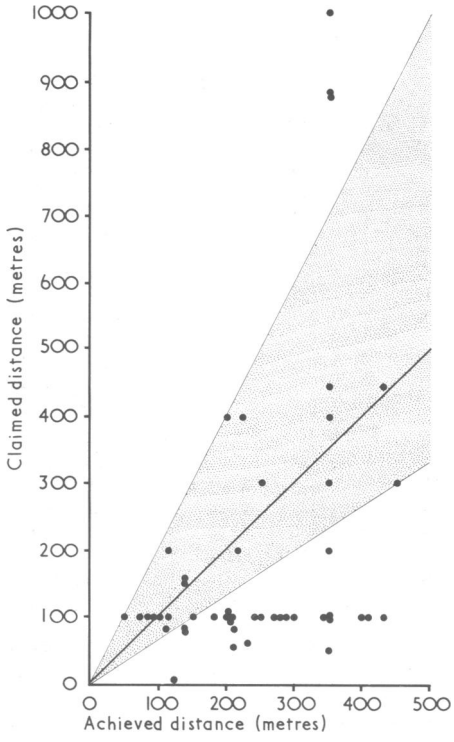
Intermittent claudication

SIR,—We were interested to read your leading article on this subject (15 May, p 1165), which emphasised that most patients who suffer from this symptom can be treated conservatively and kept under observation. We would like to make the point that simple clinical follow-up is often inadequate because a patient's own assessment of his disability, especially his walking distance, is unreliable. The walking distance can be measured objectively on a treadmill under standard conditions.



Distance claimed plotted against distance achieved on a treadmill at 4 KPH up an incline of 2·5 .

We have found that when the maximum walking distance attained by patients on the treadmill is plotted against their claimed distance there is no correlation (see figure). It is interesting to note the number of patients who quoted 100 m. We have also found that when patients are tested several times over a period of a few months the results show a variation of only 10%.

Long-term observation is the mainstay of management for most claudicants; the maximum walking distance achieved on a treadmill together with ankle pressures measured using ultrasound are simple objective methods of following up these patients. Genuine deterioration in lower limb ischaemia can be measured and the surgeon's decision on who needs reconstructive surgery and when is made easier.

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Laparoscopy explosion hazards with nitrous oxide

SIR,—We should like to reply to the criticisms by Professor J S Robinson and others (22 May, p 1277) of our letter about laparoscopy explosion hazards with nitrous oxide (6 March, p 586).

The method that we used was as follows: samples were taken, as stated, at the end of 12 laparoscopic procedures from the gas port of the laparoscopy cannula, with the telescope in place. The effective dead space of the gas passage is about 5 ml. At least 50 ml of gas was withdrawn and discarded from the gas port before the samples were taken, in duplicate, into glass syringes lubricated with a thin film of silicone grease. Such syringes are commonly used for sampling blood for blood gas analysis. The syringes were sealed with the sterile disposable polyethylene tap that was used to connect them to the gas port of the cannula. All the gas samples were analysed for hydrogen and methane within 48 hours of collection, a National Coal Board gas chromatograph, model 3, with a 180 cm × 6 mm copper column packed with 13X molecular sieve being used. Five-ml samples were introduced through a rubber septum 15 cm from the column, argon was used as the carrier gas, and a hot wire detector was used. Calibration was carried out with a standard cylinder-stored mixture of 0·5% hydrogen and 1% methane in nitrogen. Such concentrations are appropriate if gas compositions approaching explosive proportions are anticipated. This analysis system has been used by the National Coal Board Area Laboratories in Edinburgh for 15 years for routine analysis of hydrogen and methane. A sample syringe was filled from the cylinder of standard gas and stored at room temperature for 10 days. At the end of this time no difference could be detected between the composition of the sample in this syringe and that of a sample freshly drawn from the cylinder of standard gas.

On analysis of the gas samples from patients no methane was detected. In one syringe hydrogen was detected at a concentration of 20 ppm, but none was detected in the duplicate.

Our intention was to assess the assertion by Professor Robinson and his colleagues (27 September 1975, p 764) that "inevitably the abdominal cavity itself must contain large concentrations of hydrogen and, probably in many cases, methane." They suggested that gas mixtures of explosive concentrations must be present in the abdominal cavity during laparoscopy with nitrous oxide. These assertions, which are based only upon deductions from the physical properties of the gases and not from any actual measurements, are not supported by our results.

It is impossible to prove, without analysis of gas from each and every case, that explosive concentrations of bowel gases will never occur during laparoscopy with nitrous oxide, nor did we suggest that our data support this contention. We are in agreement with Mr P C Steptoe (3 April, p 833) that the only likely cause of significant amounts of intestinal gas in the peritoneal cavity would be consequent upon accidental puncture of the bowel, and this is what we suggested in our original letter. However, our experience is that the incidence of explosion, whatever the incidence of bowel puncture may be, is so far zero. Laparoscopy using nitrous oxide as the inflating gas has been carried out in one unit of this hospital since 1972.[1] More than 400 procedures are carried out each year and about 70% of these have been for tubal diathermy. No explosion or fire has occurred so far. We would continue to suggest that explosion is not a significant hazard in laparoscopy of short duration when nitrous oxide is used.

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[1] Scott, D B, and Julian, D G, *British Medical Journal*, 1972, **1**, 411.

**This correspondence is now closed.—ED, *BMJ*.