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 analogous 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 syndrome¹⁻³ 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

	Reduced movements group	Vigorous movements group
Urinary oestriol ($\mu\text{mol}/24\text{ h}$), mean $\pm$ SD	180.1 $\pm$ 79.8	191.5 $\pm$ 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 $\pm$ SD	45.0 $\pm$ 6.0	45.0 $\pm$ 4.0
Haemoglobin concentration in fetal scalp blood (g/dl), mean $\pm$ SD	18.0 $\pm$ 1.2	17.4 $\pm$ 1.3
Liquor creatinine concentration ($\mu\text{mol/l}$), mean $\pm$ SD	238.7 $\pm$ 26.5	229.8 $\pm$ 53.0
Birth weight of babies (kg), mean $\pm$ SD	3.44 $\pm$ 0.33	3.60 $\pm$ 0.38

Conversion: SI to traditional units—Urinary oestriol: 1 $\mu\text{mol}/24\text{ h} \approx 0.3\text{ mg}/24\text{ h}$. Liquor creatinine: 1 $\mu\text{mol/l} \approx 0.01\text{ mg}/100\text{ ml}$.

fetal scalp blood samples were collected at the time of surgical induction of labour by low amniotomy during the 40th week.

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 Pampiglione¹ 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 afebrile 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 Professor 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.