committee has endeavoured to be exhaustive in reviewing the evidence of concealment of crime in the past, and places its emphasis on the likely frustration of a would-be murderer. The committee does not give enough weight to the consideration that a calculating family killer may decide that there is more than an equal chance that his crime could go undetected in the case where he can rely upon the susceptibilities of a single certifying doctor. To our mind, the possibility of successful premeditated murder in a variety of ways still remains.

Having stated that we are opposed to the abolition of the second certificate (Form C) and the post of medical referee, we would have thought that proposals to abolish such safeguards would have included the setting up of a proper pilot scheme in agreed areas of the country so that it could be demonstrated that the safeguards could be relaxed with impunity. In our view, it would show that the possibility of inaccurate certification and the concealment of crime would still remain.

The last paragraph, No. 14, of the conclusion of the Brodrick report is worthy of emphasis—namely, that “goodwill and co-operation between the individuals and the interests involved are essential” if necessary improvements are to be achieved. But this will not come about by abolition of existing safeguards.—We are, etc.,

WILFRID H. PARRY
KENNETH D. MASON
MARGARET W. SEYMOUR

Health Services,
City of Nottingham

¹ Home Office, *Reprt of the Committee on Death Certification and Coroners*. Cmd. 4710. London, H.M.S.O., 1971.

Typhoid Fever in Young Children

SIR,—I read with interest Mr. T. O. Mulligan's article on typhoid fever in young children (11 December 1971, p. 665). We have recently reviewed 265 African patients with typhoid fever who were admitted to Harari Hospital, Salisbury.¹ I would like to comment on a number of points arising from his paper.

Mr. Mulligan states that, especially in endemic typhoid areas, the disease predominantly affects the younger section of the community. This was certainly so in our series, where the average age (excluding a group of 22 children under the age of 3 years) was 20 years. The usefulness of the white cell count in our experience has been of limited value. Of our adult patients 46% presented with a leucopenia (less than 5,000 cells/mm³), but unfortunately this is common here in pyrexial patients who may be suffering from malaria, tuberculosis, or viral infections. More significant was the fact that only 8% of the adult patients had a leucocytosis (greater than 10,000/mm³), and this was usually associated with a complication of the disease. Of the 22 patients under the age of 3 years eight had a white cell count in excess of 10,000, and four of these had a white cell count in excess of 20,000. It would seem that leucocytosis is much more marked in children than in adults.

I was particularly interested to note that Mr. Mulligan found the diazo reaction a useful and effective simple screening procedure. Huckstep² strongly advocated the value of this test in 1962, but little mention has been made of it in the literature since

then. Renny,³ working in Harari Hospital, Salisbury, examined the urine of 20 proven cases of typhoid fever and found a positive diazo reaction in 30%. However, when the test was carried out between the fifth and fourteenth day of the illness, as suggested by Huckstep, the positivity reached 100%.¹ We have been further evaluating the usefulness of this procedure, and preliminary results suggest that it is an effective and simple test which should be especially valuable in district medical hospitals where laboratory facilities are usually limited.

It is interesting that the peak incidence of the disease in Africa appears to vary from country to country. In Rhodesia the majority of cases occur at the beginning and in the middle of the rainy season,¹ whereas in East Africa² the incidence drops dramatically with the onset of the rainy season. In Ghana⁴ most cases occur after the rains have finished.—I am, etc.,

A. C. B. WICKS

Department of Medicine,
University of Rhodesia,
Salisbury,
Rhodesia

¹ Wicks, A. C. B., Holmes, G. S., and Davidson, L., *Quarterly Journal of Medicine*, 1971, **40**, 341.

² Huckstep, R. L., *Typhoid Fever and other Salmonella Infections*. Edinburgh, Livingstone, 1962.

³ Renny, H., Personal Communication, 1969.

⁴ Archampong, E. Q., *British Medical Journal*, 1969, **3**, 273.

Use of Amphetamines by Drivers

SIR,—Sir Ronald Tunbridge (5 February, p. 373) is right to emphasize the risks of prescribing amphetamines to combat fatigue in night driving.

As a general practitioner, I am not infrequently asked to prescribe “something” for anxious patients who are about to take their driving test. Any tranquillizer that is strong enough to be effective in such a situation would be likely to make the driver a potential menace on the road and could, in theory, render the patient liable to be charged with driving a motor vehicle while under the influence of drugs.

In my opinion, those who cannot drive a car without a sedative or pep pill ought not to be driving at all.—I am, etc.,

STUART CARNE

London W.12

Measuring Placental Function

SIR,—Your leading article (22 January, p. 193) harps aright the fear of many obstetricians that modern placental function tests have proved of little value in clinical practice. In fact, the condition of the fetus during labour can be monitored very effectively indeed¹ and it is only with regard to the monitoring of the fetus during pregnancy that one is entitled to feel disappointed.

In labour it is direct fetal parameters that are measured—fetal heart rate and fetal pH. In pregnancy the only direct parameter of fetal well-being measured is the important one of growth, yet your leading article sandwiches the assessment of fetal growth between tests on the liquor and tests conducted on the maternal blood and urine. Are our research workers also in danger of losing sight of the wood for the trees? Is not the proper study of the fetus the fetus?

In my practice at Sheppey General Hos-

pital and All Saints' Hospital, Chatham, fetal activity as reported by the mother has been studied in all high risk pregnancies and many others for three years. One of the two major reasons why toxæmic patients are not sedated² is the fear that the sedation may mask any naturally occurring change in the spontaneous activity of the fetus. To date there have been nine stillbirths and two neonatal deaths resulting from chronic intra-uterine hypoxia where fetal activity had been recorded weekly prior to death. In six cases death was preceded by a period of one to eight weeks in which fetal activity had been markedly reduced while the fetal heart remained audible with the Doptone fetal pulse detector. Low biweekly oestriol excretion values³ were obtained in only one of these cases (a neonatal death). All the fetuses were judged to be small for dates. In the remaining five cases fetal activity was reported to be vigorous up to the day of death or delivery. In none of these cases was the small for dates syndrome suspected antenatally, but one birth weight was below the 10th percentile for the particular gestation, parity, and sex.⁴

No firm conclusion can yet be reached but these results suggest that the small for dates fetus at risk of dying from chronic intra-uterine hypoxia is much more likely to reduce its activity for a clinically useful time prior to such death than the fetus which has grown more normally ($\chi^2=4.2$; $P<0.05$). Both diminished growth and diminished activity can be seen as fetal adjustments to the threat of a negative energy balance.

Dawes has recently made an authoritative plea for more study of fetal movements as an index of fetal well-being.⁵ This letter is written in support of that plea.—I am, etc.,

D. D. MATHEWS

Sheppey General Hospital,
Sheppey, Kent

¹ Sheppey General Hospital, *Abrideed Maternity Hospital Report*, Medway and Gravesend Hospital Management Committee, 1970.

² Mathews, D. D., Patel, I. R., and Sengupta, S. M., *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1971, **78**, 610.

³ Brombacher, P. J., Gijzen, A. H. J., and Verheesen, P. E., *Clinica Chimica Acta*, 1968, **20**, 360.

⁴ Thomson, A. M., Billewicz, W. Z., and Hytten, F. E., *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1968, **75**, 903.

⁵ Clayton, S., and Beard, R., eds. *Symposium on Methods of Monitoring the Foetus in Pregnancy and in Labour*, London, Royal College of Obstetricians and Gynaecologists, 1971.

Continuous Fetal Heart Rate

SIR,—Your leading article (25 December, p. 764) invites comment. The term “fetal distress” was avoided and “fetal asphyxia” chosen in preference. If “fetal distress” is to be considered an obsolete term surely it would be more accurate to replace it by “fetal hypoxia.”

The third paragraph states that Hon has shown that early decelerations are associated with cord compression. This is untrue. Hon describes early decelerations—type I dips—as due to head compression, and variable decelerations—type O dips—as due to cord compression.¹

The fourth paragraph is also misleading. In our paper on “dip area,”² Shelley and I demonstrated a significant relationship between “dip area” and one minute Apgar score over the whole range of scores. Dip area is not chiefly a measure of amplitude of dips but a measure of duration, frequency, and amplitude. It was never suggested that