Classification). There were 27 spontaneous deliveries, 8 forceps deliveries, and 9 caesarean sections. In brief, there was a sharp fall in C.V.P. on delivery of the child in every case. During the third stage of labour the C.V.P. ranged from −2 to +9 cm. H_2O (zero level was the sternal angle). Each patient received ergometrine 0·25 mg. or 0·5 mg. intravenously, sometimes before and sometimes after placental separation. Placental separation and the injection of ergometrine produced small and transient rises in C.V.P. (never more than 3 cm. H_2O and never lasting for more than 4 minutes). Blood loss was measured at these deliveries and was of the anticipated magnitude.[1 2] It is believed that the direct effects of ergometrine on the veins were to a variable extent cancelled out by the effects of haemorrhage.

The period after delivery of the child is a dangerous time for the patient with heart disease. Bearing in mind the several and major concurrent alterations in the circulatory state which may occur at this time and the absence of any major rise in the C.V.P. in 44 patients, one wonders whether the deaths which have occurred at this time are due, as is often stated, to the effects of ergometrine. One must weigh the risks of haemorrhage against the risks of ergometrine. I believe that a dose of up to 0·25 mg. of ergometrine is, on balance, less hazardous than a third-stage haemorrhage for the patient with severe heart disease. Pulmonary oedema if it develops may not be due to the ergometrine and is usually treatable. As an obstetric anaesthetist, while not advocating the routine use of general anaesthesia and operative delivery for patients with serious heart disease, I mention the control of oxygenation attainable during modern anaesthesia and the potential benefits of intermittent positive pressure ventilation on pulmonary oedema.—I am, etc.,

DONALD D. MOIR.

Queen Mother's Hospital,
Glasgow C.3.

REFERENCES
1 Wallace, G., *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1967, **74**, 64.
2 Moir, D. D., and Wallace, G., *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1967, **74**, 424.

Errant Genes

SIR,—In your leading article "Errant Genes" (7 February, p. 315) you state: "Thus, in phenylketonuria there is a deficiency of the enzyme phenylalanine hydroxylase, so that phenylalanine cannot be converted to tyrosine. The resulting excess of phenylalanine in the blood is toxic to the growing brain, causing mental retardation and fits."

The evidence that phenylalanine is responsible for the neuropathological aspects of phenylketonuria can only be described as sketchy. Absence of phenylalanine hydroxylase shunts phenylalanine into alternate oxidative pathways, resulting in the formation of phenyllactic, phenylpyruvic, and phenylacetic (converted to phenylacetylglutamine) acids.[1] We cannot yet say with certainty whether it is phenylalanine or the oxidized derivatives which are mainly responsible for the effects of the disease on the nervous system. If we could be sure that phenylalanine alone is responsible, then it may well be indirectly via an inhibitory effect on Dopa-decarboxylase reducing the concentration of transmitter substances in the synopses.[2]—I am, etc.,

R. DUGUID.

Department of Biology,
University of York.

REFERENCES
1 Harris, H., *Human Biochemical Genetics*, London, Cambridge University Press, 1959.
2 Woolf, L. I., In *Biochemical Aspects of Neurological Disorders*, 3rd. Series. Ed. J. N. Cumings and M. Kremer, p. 109, Oxford, Blackwell Scientific, 1968.

Recurrent Virus Meningitis

SIR,—Dr. J. P. Anderson (27 December, p. 786) reports a case of a patient who had two separate attacks of aseptic meningitis with different viruses as aetiological agents. He states that "a review of the literature and correspondence with colleagues of wide experience have not revealed any further virologically proved examples of such double attacks."

However, a paper entitled "Multiple attacks of Aseptic Meningitis in the Same Individual" by K. Lapinleimu and myself was published in the *B.M.J.*[1] Multiple attacks were established in four patients: in two adults, one three times and the other four times, and in two children, twice in each. The intervals between the individual attacks were from four months to eight years. An enterovirus isolation from the stools succeeded in 5 attacks out of 11. Antibody responses gave us strong reason to believe that the attacks were caused by the viruses isolated. In a sixth attack serological evidence of infection with E.C.H.O. 7 was obtained. In two patients different viruses appeared to be responsible for the successive attacks of meningitis.

Since publishing the above-mentioned paper, six further patients who have had more than one attack of aseptic meningitis of proved or probable viral aetiology have been treated at the Aurora Hospital.—I am, etc.,

ERKKI KLEMOLA.

Aurora Hospital,
Helsinki, Finland.

REFERENCE
1 Klemola, E., and Lapinleimu, K., *British Medical Journal*, 1964, **1**, 1087.

Unnecessary X-rays?

SIR,—While having great sympathy with Mr. A. W. Fowler (7 February, p. 632) in his desire to remove all legal considerations from the decision as to whether an *x*-ray should be advised, I feel that he has oversimplified the issues.

It may be true, as he says, that "no fracture in a limb which is not clinically obvious will suffer from delay in diagnosis for three days." When, however, a three-day delay in diagnosis occurs and negligence is alleged, it will be impossible to deny the extra three days' pain and suffering. Very possibly also the plaintiff will allege that the position of the fracture has worsened during the three days, and may produce an expert witness to testify that earlier reduction might have been expected to obtain a better result. This is but one example of the difficulties which may follow the course proposed.

Regarding the recommendation that the number of calls on the duty radiographer after 5 p.m. should be reduced, we think it very much open to question whether any department accepting casualties should be without immediate *x*-ray facilities. Certainly hospital junior staff should not, in our view, be subjected to any pressure whatever to avoid or postpone *x*-rays which they feel to be necessary. If things go wrong it is the casualty officer who will be blamed, and if he is capable of being left in charge of a casualty department he is equally capable of deciding when to order an *x*-ray. In particular, failure to order an *x*-ray because the case is one of "simple" concussion is, we feel, fraught with danger.

The casualty officer is more exposed than is any other doctor to allegations of negligence and is deserving of every support from the ancillary services. Better to press the authorities to provide improved radiographic services than to seek to reduce the calls made upon them.—I am, etc.,

H. A. CONSTABLE,
Secretary,
Medical Protection Society Ltd.
London W.1.

SIR,—There is one particular situation in which I must strongly disagree with the views expressed by Dr. R. M. Scott (13 December, p. 689) and Dr. W. B. James and others (10 January, p. 110). Any injury in a child under the age of two makes a complete skeletal survey mandatory. No matter how plausible the story, such an infant must be considered to be an instance of "battered baby syndrome" until proved otherwise. Only radiological examination of the whole child will reveal old and healing injuries, which are virtually pathognomonic. It is also worth remembering that any subsequent battering carries a 10% mortality.—I am, etc.,

BRUNO GANS.

Department of Paediatrics,
Lewisham Hospital,
London S.E.13.

SIR,—The letter from Mr. A. W. Fowler (7 February, p. 362) is an excellent summary of the considerations which should govern the request for an *x*-ray examination.

This is summarized by the advice which I have always offered to medical students and young residents: "Never ask for an *x*-ray examination if the result will not change the treatment of the case."—I am, etc.,

D. C. STAVELEY.

Alford, Lincs.

Fibrin Degradation Products and Menstruation

SIR,—I read with interest Mr. H. K. Basu's paper (10 January, p. 74) and his contention that higher concentrations of fibrin degradation products in cases of menorrhagia may indicate increased local fibrinolytic activity in the uterus.

That the uterus is a potent source of plasminogen activator to the systemic cir-