Fetal Damage from Breech Birth

SIR,—Your leading article "Fetal Damage from Breech Birth" (26 April, p. 158) is a timely reminder of the hazards of this type of delivery. As you point out, more frequent recourse to caesarean section in recent years has improved results, and most obstetricians will subscribe to the formula "breech + anything else = caesarean section." However, I feel that your article was incomplete in its failure to point out that caesarean section carries a 10-fold increase in maternal mortality over vaginal delivery. If this fact is not borne in mind, then too frequent use of caesarean section in breech presentation may result in an unwelcome rise in maternal mortality figures.

I had not previously associated breech presentation of a normal fetus with premature labour, except in so far as the frequency of breech presentation decreases towards term as a result of spontaneous version. No doubt admission to hospital, as you suggest, would reduce the incidence of premature labour in breech presentation, but so would it in cephalic presentation. You advise admission "(as in cases of twin pregnancy)... some six weeks before term." In a recent study of 200 consecutive twin pregnancies from this unit premature delivery was defined as delivery before 36 weeks gestation. However, on reviewing the figures it was found that only one normal infant died as a result of premature delivery after 34 weeks, and in this case labour was induced at 36 weeks because of severe pre-eclampsia. May I, therefore, point out that any measure taken to prevent premature labour in multiple pregnancy must be instituted much earlier than you suggest, the most dangerous time being between 28 and 34 weeks? This presupposes an early diagnosis, and any suspicion of multiple pregnancy should be investigated at the earliest opportunity. The availability of ultrasonic diagnostic facilities in most centres means that most such pregnancies should be detected at an early stage and not, as so often happens, when the patient is admitted already in premature labour.—I am, etc.,

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SIR,—I must take issue with the ridiculous suggestion made in your leading article (26 April, p. 158) that "in an attempt to lower the incidence of premature labour it would be reasonable (as in cases of twin pregnancy) to admit the mother to the antenatal ward for complete rest some six weeks before term."

Unfortunately you have confused cause with effect. The association of breech delivery with prematurity is due to the fact that the incidence of breech presentation decreases naturally as term approaches unless there are other complicating factors. Hence a premature labour is more likely to produce a breech birth than is a labour at or near term. Breech presentation (unlike twins) is not in itself a factor in the initiation of premature labour.

I do hope that your suggestion is seen to be illogical by all who give antenatal care. Otherwise many mothers-to-be may be cruelly and needlessly incarcerated in our

antenatal wards for several weeks before the penny drops (tails or heads!).—I am, etc.,

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** Our suggestion that the risk of premature labour in breech presentation might be reduced by admitting those cases which continue beyond the 34th week was a compromise. Earlier admission would indeed lead to large numbers of patients being admitted, but by 34 weeks the numbers should be much smaller and it would be interesting to establish whether, in these high-risk cases, rest and closer observation in an antenatal ward led to a more favourable outcome. Apart from the possibility that this might reduce the risk of premature onset of labour (which is not known at the moment) there could be other bonuses. It is likely that cord presentation or prolapse would be diagnosed earlier and there is the advantage that the obstetrician, away from the pressures of the antenatal clinic, would be better placed to make a more balanced judgement on the management of the case. We had in mind that these mothers might spend one or two weeks in the antenatal ward, but where the presentation converted to a vertex the patient would go home at once.—ED., *B.M.J.*

Warfarin Poisoning in Domestic Animals

SIR,—May we comment on your leading article "Rats Today" (19 April, p. 105) on the basis of our clinical experience of warfarin poisoning in domestic animals?

An animal may be poisoned by warfarin as a result of eating affected rodents or warfarin bait. The warfarin bait is usually prepared by making a 1:20 mixture of a 0.5% or 0.1% commercial warfarin preparation with oatmeal or other suitable food to give a 0.025% or 0.005% warfarin bait. Papworth¹ quotes the weights of 0.005% bait to give different animals the estimated LD₅₀ dose if consumed daily for five days as follows:

Animal	Weight of Bait		LD ₅₀ (mg/kg)
	oz	g	
Rat ..	0.8	22.7	1
Dog ..	2.4	68	3
Dog ..	2.4	68	3
Pig ..	0.1	2.8	0.4
Poultry ..	8.0	227	10
Cattle ..	40.0	1134	200

The inherent danger of warfarin compounds rests with their cumulative effect, whereby a large single ingestion of warfarin is less likely to be harmful than small quantities consumed over a period.

Pigs may be poisoned by eating warfarin bait or affected rodents, whereas dogs are usually poisoned by eating warfarin bait and cats by eating affected rodents. Clinical cases of warfarin poisoning are most commonly seen in dogs, cats, and pigs in that order of frequency, and this is to be expected when both the susceptibility of the animal and the availability of the poison are taken into account.

At the Cambridge Veterinary School we deal only with the more complicated cases

of warfarin poisoning which are referred to us by veterinary surgeons in practice. Our cases are thus but a small proportion of the number actually occurring, but even so we average one or two cases of warfarin poisoning in dogs each week during the time of year that warfarin bait is laid. Not only the amount of blood lost but the site of haemorrhage (intrapleural, intrapericardial, or intrapulmonary) demands that immediate and vigorous steps be taken if the animal's life is to be saved.

To say with regard to warfarin that "ordinary domestic animals... are not particularly sensitive to it" is to perpetuate the myth that warfarin is a safe poison to all but the rat.—We are, etc.,

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¹ Papworth, D. S., *Journal of the Royal Society of Health*, 1958, 78, 52.

Diffuse Pulmonary Disorder Caused by Oxyphenbutazone

SIR,—Lung disorders caused by pyrazolones, of which phenylbutazone is one, are uncommon. Pulmonary oedema has been reported¹ and also two cases in which a syndrome resembling systemic lupus erythematosus developed.² I wish to report a case of reversible diffuse pulmonary disorder which I saw five years ago in a patient who had been taking oxyphenbutazone over a long period.

The patient, a 66-year-old woman, suffered from stiffness of the hands and painful ankles and had radiographic evidence of osteoarthritis. She had been taking oxyphenbutazone for nearly three years when she suddenly developed breathlessness. On examination fine crepitations were heard at the base of both lungs. The erythrocyte sedimentation rate was raised and antinuclear factor was present transiently in the serum. Neither rheumatoid factor nor L.E. cells were found. Oxyphenbutazone therapy was stopped. Diuretics were given initially but no diuresis or loss of weight resulted. During the eight weeks after stopping the oxyphenbutazone the patient's breathing returned to normal. When given a further challenging course of oxyphenbutazone breathlessness and crepitations rapidly returned, despite concomitant diuretic therapy.

A drug-induced lupus diathesis seemed the most probable diagnosis in this case and was supported by the finding of antinuclear factor.^{3,4} The failure of diuretics to produce a diuresis or loss of weight excluded the complication of excessive tubular sodium reabsorption. Fibrosing alveolitis, though associated with rheumatoid arthritis, was unlikely in the absence of rheumatoid factor and also the symptoms were reversible. Systemic lupus erythematosus, unmasked by the drug, was also unlikely since antinuclear factor was present only transiently and no L.E. cells were found. Now, five years later, there is no clinical evidence of the disease.

Drug-induced diatheses are rarely complete or dose-dependent and, apart from those due to hydralazine, they cannot be induced in animals.⁵ They are due either to a delayed immunological response to the drug or a metabolite and possibly a type 3 hypersensitivity reaction or to hyper-