SIR,—Your leading article (21 September, p. 701) highlights the problems of Negro patients who may have repeated screening tests for sickle-cell disease.

Now that the existence of sickle-cell disease in Britain has been recognized and well publicized I feel that the need for screening should be put in the right perspective on a par with other potentially dangerous problems associated with anaesthesia and surgery. It is advisable to perform routine screening for haemoglobinopathies on hospital attendance, but should the absence of a sickle-cell test in fit and non-anaemic adults and older children of possible Negro descent be a contraindication to general anaesthesia? This is certainly not the practice anywhere in Africa.

The suggestion that medical cards be carried by these patients is the obvious solution, but logically medical cards should be advisable for most patients and should include not only haemoglobinopathies but other relevant information, such as blood group, drug allergy, therapy with steroids, anticoagulants, etc. for the same reasons as suggested in the article.—I am, etc.,

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Familial Trends in Low Birth Weight

SIR,—I should like to make some comments on the excellent paper of Mr. F. Johnstone and Mrs. Lesley Inglis from Aberdeen (14 September, p. 659). Our theory of the maternal regulation of fetal growth, to which their data conform, was developed from the classical cross-breeding experiments of Sir John Hammond and his colleagues.[1 2] These studies and our own data indicated that the regulator acts by means of *constraining* fetal growth. There is no opposite and equal accelerating mechanism. When maternal constraint is relaxed other biological factors such as maternal stature, weight, and parity make a larger contribution to the individual case.

Analyses of pedigree data[3] demonstrated a clear sequence of mean birth weights on the distaff side of families ascertained through a small-for-dates proband. They ranged in a precise sequence from previous liveborn siblings (2,676 g) and mothers (2,921 g), first cousins through maternal aunts (3,062 g), maternal aunts themselves (3,230 g) and uncles (3,262 g), to first cousins through maternal uncles (3,375 g). When the proband was large for dates a more complex pattern emerged. The mean birth weight of each class of relative was above average, but the differences between relatives were small. The two classes of first cousin did not differ. The mean birth weight of the fathers in our small-for-dates series did not differ from that of the general population, whereas the fathers of our large-for-dates probands had been heavy at birth, with a mean of 3,806 g. These findings support the notion that constraint at its extreme is transmitted through mothers only.

Our male and female probands were originally ascertained on a sex-mixed grid. Since boys grow faster than girls in utero a greater degree of constraint was needed for boys to attain the small-for-dates criterion than for girls. We used Carter's method of the sex of the proband[4] to test our theories and also the validity of our data in a quantitative manner. The predictions were that ascertainment through a small-for-dates boy should reduce the mean birth weight of all distaff relatives except first cousins through maternal uncles, leaving the sequential order in mean birth weights of kin unchanged. Proband sex should not affect the mean birth weight of distaff relatives of large-for-dates probands, because when maternal constraint is relaxed other factors take up more of the variance. Our findings are shown in tables I and II and it will be seen that these predictions are fulfilled. It is pertinent to note in the small-for-dates data that the amount of difference in mean birth weight of relatives affected by the sex of the proband increases in the same sequence.

TABLE I—*Small-for-dates Series. Mean Birth Weights (g) of Relatives According to Sex of Proband*

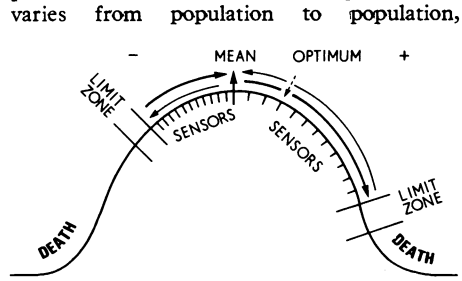
Relative	Total No.	Female Proband	Male Proband	Total
Liveborn siblings	198	2,699	2,617	2,676
Mothers	148	2,971	2,862	2,921
First cousins through maternal aunts	123	3,166	2,957	3,062
Maternal aunts ..	114	3,343	3,084	3,230
Maternal uncles ..	96	3,357	3,116	3,262
First cousins through maternal uncles	67	3,384	3,343	3,375
Fathers	71	3,334	3,334	3,334

TABLE II—*Large-for-dates Series. Mean Birth Weights (g) of Relatives According to Sex of Proband*

Relative	Total No.	Female Proband	Male Proband	Total
Liveborn siblings	241	3,992	3,905	3,937
Mothers	126	3,760	3,583	3,647
First cousins through maternal aunts	144	3,425	3,633	3,570
Maternal aunts ..	110	3,460	3,674	3,596
Maternal uncles ..	118	3,629	3,629	3,629
First cousins through maternal uncles	131	3,347	3,520	3,438
Fathers	96	3,783	3,819	3,806

The physiological paths by which human fetal growth is constrained are unknown. Csapo *et al.*[5] made elegant experiments in rats which identify oestradiol 17β (E2) as the inhibitor of the growth of the conceptus. The mechanisms by which fetal growth is regulated must be relatively subtle, since they are presumably sensitive to continuing information from the conceptus about the amount of growth achieved. There should be a set-point, with sensitivity finer on the minus as opposed to the plus side. Such systems have been established for the control of glucose, temperature, and steroids.[6] The units are hypothalamic integrating neurones with sensitivities specific to each parameter. In each case the numbers of positively sensitive and negatively sensitive neurones are unequal. This provides plus and minus sensors which are able to control feedback loops on either side of the set-point, but with unequal strength.

The regulation of fetal growth is more complex than simply keeping the level of a physiological variable within limits at a given time; the distinction is between maintenance of steady growth and maintenance of a steady state. Nevertheless, these systems provide a model for our concept of the maternal regulator of fetal growth (see fig.). At both extremes of growth rate the system is non-viable. Within these there is a grey area where survival is possible but not probable. In the normal range the regulator is more sensitive below the set-point than above it. The set-point itself varies from population to population, pedigree to pedigree, woman to woman, but only occasionally from pregnancy to pregnancy.



Model of maternal regulator; c.f. Schade's glucostat, thermostat, and steroidstat.[6]

The great variation in mean birth weight between different ethnic groups may well be adaptive. But for any given population or group optimum birth weight is above mean birth weight. There is thus a selection pressure towards higher birth weight. This system is in a state of evolutionary change; it is dynamic, not static, over generations. Our present theory parsimoniously suggests that a quantitative shift in the numbers of plus and minus sensor neurones by constraint imposed on the female fetus would be adequate to explain the data so far to hand.—I am, etc.,

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3 Ounsted, M., and Ounsted, C., *On Fetal Growth Rate*, Clinics in Developmental Medicine, No. 46. Heinemann, London, 1973.
4 Carter, C., and Evans, K. A., *Journal of Medical Genetics*, 1969, **6**, 233.
5 Csapo, A., Dray, F., and Erdos, T., *Lancet*, 1974, **2**, 51.
6 Schade, J. P., in *The Hypothalamus*, ed. L. Martini, M. Motta and F. Fraschini, p. 69, New York. Academic Press, 1970.

Experts and Child Abuse

SIR,—Evil always fascinates; and by concentrating our attention on itself can lead to the neglect of related evils of equal importance. If we hope to improve our care of battered babies we must not forget what we know about the dangers of separating mothers from their infants. The physical health of the baby is of immediate and obvious concern, but its emotional health is quite as important in the long term.

If, as Prof. S. D. M. Court and others suggest (28 September, p. 801), we should admit the child to hospital for "medical and psychiatric as well as social diagnosis of the family whenever there is suspicion" of abuse, I submit that it is equally important that mother should be admitted *with* the child—that is, to a mother and baby unit. The bond between mother and infant which is the foundation of the child's later emotional health is probably already impaired in such cases; and separate admission of the baby can only confirm and exacerbate the impairment. *Primum non nocere.*

As well as the advantages this would offer to the precision of the "psychiatric as well as social diagnosis of the family," social workers (who, however young and inexperienced, are daily required to make decisions which would perplex most doctors) might feel less reluctant to refer their cases to a medical team if they were assured in this way that doctors, besides being expert in dealing with physical damage, respected the long-term emotional health of the child as well as they themselves have been taught to respect it.—I am, etc.,

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Drug-induced Red Cell Aplasia

SIR,—Cephalothin has been associated with neutropenia,[1] thrombocytopenia,[2] and anaemia with a positive direct Coombs test.[3]