p 591) reported that even a fairly high single dose, 2000 mg, in a suicide attempt had no effect on blood pressure or pulse rate. They concluded that "the effect of propranolol on the healthy heart needs to be reconsidered." It is also well known that propranolol may be given in very high doses in the treatment of hypertension. Prichard¹ has been using doses up to 4000 mg daily and Kincaid-Smith and her co-workers² up to 2000 mg daily, but with a gradual titration of the optimal dose. It is therefore of interest to note that even a relatively small "massive" dose of propranolol can give rise to severe cardiac disturbances.

A woman aged 24 was recently treated in our department after a suicide attempt with 25 40-mg tablets of propranolol taken as a single dose together with some beer and 100 ml of whisky. On admission to hospital about two hours later she was comatose, without measurable blood pressure, and had pronounced sinus bradycardia with a pulse rate of about 35-40/min. Over the lungs sibilant rhonchi were auscultated. Atropine was given intravenously, but soon after admission asystole developed. External cardiac compression was immediately started, endotracheal intubation was performed, assisted ventilation was started, and bicarbonate was given intravenously. This, however, did not have any effect on cardiac activity, but after adrenaline had been given intracardially spontaneous sinus rhythm was restored, with adequate peripheral circulation. The patient was observed for five days and discharged without any signs of cardiac or cerebral damage. The concentration of propranolol in the serum was not measured.

It was suggested by Drs A J Boakes and B H Boeree (15 December 1973, p 675) that since a healthy subject resting in the supine position maintains an adequate cardiac output and blood pressure in the absence of sympathetic drive, beta-blockers in massive doses are not likely to represent a hazard. This case, however, shows that such a hazard does exist, though it is possible that the effect was potentiated by the simultaneous consumption of alcohol. One can, however, expect that the potential risk of cardiac arrest should be lower with those beta-adrenergic blocking drugs which have an intrinsic sympathomimetic activity.

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¹ Prichard, B N C, *British Journal of Hospital Medicine*, 1973, 10, 45.

² Kincaid-Smith, P, Fang, P, and Laver, M C, *Clinical Science and Molecular Medicine*, 1973, 45, 758.

IUD and congenital malformation

SIR,—Attention is drawn by Dr H Barrie (28 February, p 488) to the possible teratogenic effect on the developing fetus of copper released from intrauterine devices (IUDs) for contraception.

It has previously been shown¹ that copper released from such devices results in a significant increase in the local copper concentration in the intrauterine fluid. However, this does not result in a significant rise in serum copper or caeruloplasmin concentrations.^{2 3} Even if a significant rise did occur it is to be expected that the placenta would form an effective barrier to the transfer of the large (molecular weight 151 000) caeruloplasmin molecule to the fetus. As about 93% of copper in the maternal blood exists in the form of this enzyme only a small amount of free copper is

available to diffuse across the placenta to the fetus. This relative isolation of the developing fetus from copper is supported by the apparent normality of the majority of animal and human infants^{4 5} delivered from mothers using copper IUDs. As Dr Barrie points out, however, it is likely that such pregnancies are seriously under-reported, in addition to which closer examination of those fetuses which do not go to term but abort at an earlier stage might well reveal a different pattern.

In a recent case (reported in full elsewhere⁶) the rare opportunity was taken to estimate the cord blood copper and caeruloplasmin concentrations from a neonate conceived in the presence of a Gravigard IUD. The serum copper concentration was 2.5-5 times higher than the normal range⁷ for neonates of equivalent gestational ages and the serum caeruloplasmin (measured as copper oxidase by the same technique as by Canzler *et al.*) was 3.5-8 times the normal range. The maternal serum copper and caeruloplasmin concentrations were within the normal range. In this instance the neonate was apparently normal, although he died shortly after delivery, probably from immaturity.

Presumably if significant quantities of copper do not cross the placenta they must diffuse across the embryonic membranes and liquor to reach the fetus. Whichever route is taken we must conclude on present evidence that a fetus conceived in the presence of a copper IUD is incompletely isolated from its copper-containing environment. Until more is known of the effect of copper on human organogenesis it would be prudent to exercise caution in the use of copper IUDs.

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¹ Oster, G K, *Fertility and Sterility*, 1972, 23, 18.

² Hagenfeldt, K, *Contraception*, 1972, 6, 119.

³ Daunter, B, and Elstein, M, *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1973, 80, 644.

⁴ Chang, C C, and Tatum, H J, *Contraception*, 1973, 7, 413.

⁵ Tatum, H J, *American Journal of Obstetrics and Gynecology*, 1973, 117, 602.

⁶ Alderman, B, *Proceedings of the Royal Society of Medicine*. In press.

⁷ Canzler, E, Brosch, G, and Schlegel, C, *Zentralblatt für Gynäkologie*, 1972, 94, 646.

SIR,—In recent weeks the intrauterine device (IUD) as a method of fertility regulation has been receiving something of a bad press in daily and weekly newspapers. Some of this can be attributed to misreporting or to sensationalism for its own sake, but the article by Dr H Barrie (28 February, p 488), relating to fetal abnormality associated with IUD use is, I believe, even more damaging.

I found Dr Barrie's article difficult to understand as I could find no evidence in it to support the conclusions he draws. The claim that "the possibility of a causal association [between IUD use and congenital abnormality] is consistent with the known hazards of intrauterine devices" is puzzling. What evidence is there for this claim? No published study that has assessed the outcome of pregnancy among IUD users has revealed evidence of a causal link between IUD use and congenital malformations.

A further puzzling aspect of the article is the suggestion that it was the copper content of the IUDs that was to blame. The two devices described were the Gräfenberg ring and the Dalkon Shield, both of which carry

only trace copper. In the United Kingdom at the present time approximately one-third of all IUDs fitted are devices which carry very large amounts of surface copper. For example, the Gravigard (Copper-7), the Gyne-T (Copper-T), and the Copper Omega. These devices carry 200 mm² or more of surface copper yet no reports of congenital abnormalities associated with the use of these devices have been received.

The second case cited by Dr Barrie is based almost entirely on conjecture and certainly does not fit with his belief that this is "experimental evidence." There are uncertainties relating to the device model—the Dalkon Shield was named by the mother after being shown illustrations and samples three months after the delivery. The device was not found at delivery and it would be equally reasonable to assume that the device had been expelled before conception occurred.

The family planning research unit in the University of Exeter has been collecting data on IUDs fitted in 20 centres situated in various parts of the United Kingdom for a number of years. At present over 100 000 medical records relating to 25 000 IUD fittings are held in Exeter. All pregnancies reported are followed up to assess the outcome of that pregnancy. Since April 1972 317 completed pregnancies have been recorded and details of the pregnancy outcome published.¹ In summary, 33% of these pregnancies ended in spontaneous abortion, 35% were terminated, 3% were ectopic pregnancies, and 21% proceeded to full term (the outcome of the remaining 8% is not known). No congenital abnormalities were reported among these full-term births.

At the present time it is estimated that about 400 000 women in the United Kingdom are using IUDs. For almost all IUD models the pregnancy rate during the first year of use lies between 2.0 and 4.0 per 100 users.² Taking the lower figure, this yields approximately 8000 pregnancies among women wearing IUDs. According to evidence collected in this unit and summarised above approximately 1 in 5 of these pregnancies will go to term. If 5% of all babies are born with congenital abnormalities, as Dr Barrie states, one would expect approximately 80 malformed infants to be born to mothers wearing IUDs in the United Kingdom as a whole. It would be double this figure if the higher pregnancy rate (4.0%) was taken. From the above evidence it is possible to argue that the incidence of congenital abnormality is reduced in infants born to mothers wearing an IUD. This is clearly a dubious hypothesis, but at the present time there is more evidence to support it than there is to support the contrary hypothesis put forward by Dr Barrie.

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¹ Snowden, R, *FPA Medical Newsletter*, No 59, 1976.

² Snowden, R, *Journal of Biosocial Science*, 1975, 7, 367.

Immunisation against whooping cough

SIR,—I wonder if Professor G T Stewart's evidence has all been published or whether I shall upset him again by writing too soon—his published article¹ has little in common with the "raw data" he sent me. He asks (6 March, p 583) why I associate the new vaccine with the recent decline in whooping cough, whereas a "greater decline . . . occurred before it began to

be used in 1968." The answer is already published^{2,3} (with references): the vaccine is "once again highly effective, as it was in the 1950s against the then-prevalent strains." The decline of the 1950s followed the initial introduction of vaccine,⁴ acceptance of which neared its peak by 1957. Eradication seemed imminent. Then serotype 1,3 emerged in the 1960s: his own graphs¹ show how the decline decreased—until the vaccine was modified.

Thus, far from despising notifications or being in conflict with Colindale, I support Dr N D Noah's view (17 January, p 128) that "notifications . . . reflect trends in the incidence of *Bordetella pertussis* infection." But they are unreliable for evaluating vaccine, as it is difficult to diagnose mild cases clinically. The data of Dr Christine L Miller and Mr W B Fletcher (17 January, p 117) suggest that the new vaccine is very effective against illness that can be diagnosed more accurately—either severe cases or hospital admissions. In these groups only 10% of their children aged 1-2 years had been vaccinated, although there would be about three times as many vaccinated as non-vaccinated at risk. This implies that current vaccine is more than 95% effective, a figure which agrees with my recent (unpublished) data. Also, Professor Stewart pays only lip-service to the "desirability of bacteriological confirmation." His claim¹ that "antigen 3 . . . did not . . . protect against . . . the prevailing serotype 1,3 in 1974" is substantiated by only four cases of type 1,3 infection—and no indication whether even these had received the new vaccine.

He accuses me of asking people to "accept the new vaccine as being non-toxic." On the contrary, I said² that "its safety is rightly being examined." However, I have never seen a vaccine-damaged child, though many severely ill with whooping cough and cultures from some of them post mortem. But those who have recorded possible vaccine-damage^{5,6} admit that they cannot compare the risks of natural infection and vaccination; and even *World Medicine*⁷ now talks of an "occasional—and possibly receding—hazard" with vaccine that "appears to be both good and safe." Mrs Rosemary Fox (21 February, p 458) still picks on pertussis vaccine as the culprit, though only 65% of her cases followed the use of triple vaccine. Moreover, she tells us nothing of the incidence of similar conditions in children who have not received any vaccine. Perhaps we should allow the Subcommittee on the Complications of Vaccination to study the problem as they think best.

Perhaps Professor Stewart may now follow his own advice and await parturition of my data.

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¹ Bassili, W R, and Stewart, G T, *Lancet*, 1976, 1, 471.
² Preston, N W, *Lancet*, 1974, 2, 1138.
³ Preston, N W, and Stanbridge, T N, *Lancet*, 1975, 1, 1089.
⁴ Griffith, A H, *Symposia Series in Immunobiological Standardization*, 1973, 22, 13.
⁵ Kulenkampff, M, Schwartzman, J S, and Wilson, J, *Archives of Disease in Childhood*, 1974, 49, 46.
⁶ Aicardi, J, and Chevrie, J J, *Lancet*, 1974, 2, 894.
⁷ *World Medicine*, 1976, 11, No 8, 5.

A place to be born

SIR,—Dr R Dingwall (14 February, p 396) has done well to remind your readers that the social aspects of childbirth can be the subject of systematic research, albeit in a mode somewhat different from that employed in clinical research. Too often statements relating

to the social and psychological aspects are dismissed as "merely subjective," "just emotion," or matters of (pigheaded) opinion. As a sociologist, I believe that there may be more hazards in the way we handle childbirth than can be measured by perinatal and maternal mortality rates and that we should take account of these.

At a recent seminar at Warwick University funded by the Nuffield Foundation, where obstetricians and social scientists met together to discuss mutual problems, Dr Iain Chalmers produced evidence, using conventional obstetric outcome measures, which suggested that the active management of labour did not result in the benefits claimed for it. If this is so there can be no doubt that social and psychological variables should be taken into account, as should the views of the women themselves. The predominant impression with which most of us emerged from that day of talking at Warwick was that there was a long way to go before mutual understanding was achieved. The correspondence in your columns confirms this impression. For example, it is suggested that hospitals should be made more home-like, that women there should be given more choice and be more involved in the birth—all proposals which I personally would applaud. But there seems no recognition of just how difficult such goals are to achieve. A hospital is a totally different sort of social organisation from a home; it is not possible for a woman to be treated there as she is at home. Social scientists can help by analysing and explaining these differences so that the problems may be better understood.

I am glad to learn that in various parts of the country social scientists are getting together with obstetricians to try to establish a dialogue so that the latter may come to some understanding of the sociological and psychological hazards of childbirth, while the social scientists learn more of what are seen by obstetricians as medical imperatives. Let us not go blindly forward, perhaps doing unintentional damage, which is no less real because it is not immediately visible or readily measurable.

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SIR,—In your leading article "A place to be born" (10 January, p 55) you make comparisons between the results of the British and the Dutch systems of obstetric organisation. As you brought our country in and we share your interest in promoting all aspects of maternal and child care as much as possible, we feel compelled to make some remarks.

(1) You quote De Haas-Posthuma as follows: "De Haas-Posthuma [*sic*] has shown that those parts of Holland with the highest incidence of hospital confinement have the lowest perinatal mortality rates." According to the list of references this statement is to be found on p 220 of the *Proceedings of the Organisation for Health Research, Series A, No 11*. On that page we cannot find the quoted sentence. On p 211, however, we read: "Although home confinement still strongly predominates an increasing tendency towards institutional confinements is to be found in the Netherlands also: 27½% in 1960 as against 22% in 1952 or a relative rise in hospital confinements of 25% in 8 years. During the same period perinatal mortality fell from 31 per 1000 in 1952 to 25 per 1000 in 1960. Even if a causal relationship were to exist between hospitalisation and falling perinatal mortality—a point that has not been proved—this would not yet mean that the tendency towards hospitalisation should be encouraged indiscrimi-

nately. An excessive degree of hospitalisation should be guarded against."

(2) The decline in the percentage of home confinements in the Netherlands has continued since then. In 1973 this percentage was 51. Perinatal mortality in 1973 went down to 16.3 per 1000. In that same year 84% of the home confinements took place in co-operation with the so-called Organisatie inzake Kraamhulp (Maternity Home Help). Perinatal mortality in that group was 4.5 per 1000. Hospitalisation of the newborn during the first 10 days post partum took place in 2½% of all cases. In 1960 perinatal mortality of the same group of home confinements was 14 per 1000. This means that from 1960 to 1973 hospitalisation went up from 27.5% to 49% (a relative rise of 78% in 13 years); perinatal mortality fell from 25 to 16.3 per thousand (a relative decline of 34%). Perinatal mortality in home confinements with maternity home help fell from 14 to 4.5 per thousand in that same period (a relative decline of 68%).

(3) From the facts now available we can conclude that any correlation between the percentage of hospital confinements and perinatal mortality by region or municipality is very poor or even non-existent. It seems that other factors must be responsible for the differences in perinatal mortality in the various parts of the Netherlands.

(4) In a recent study stimulated by the obstetrical department of the University of Amsterdam one of us (D van A) followed accurately a group of 916 women who were pregnant with their second child and who were selected for normality and home confinement in accordance with the Dutch list of "medical indications for hospital confinements." From this group 24 women (2.6%) were transferred during labour to the hospital. In this group of transferred women there was one artificial delivery (vacuum extraction) and one case of perinatal mortality. Of the whole group of 916 women 892 were delivered at home or in a simple home-like maternity unit at Wormerveer, where only midwives or family doctors were present and where no hospital facilities and no possibilities for artificial delivery or blood transfusion existed. These 892 women gave birth to 893 children (one unrecognised twin pregnancy). Among the 893 children perinatal mortality was zero.

We do not make propaganda for home confinements but we protest against the simplification that total hospitalisation should be the aim of an ideal obstetrical organisation. In the Netherlands for example we can show that improvement of the care in our hospitals will be of considerably more importance than compelling every pregnant woman to have her baby in hospital. Before we are entitled to demand total hospitalisation it is our duty to make every hospital a place for "the best way to be born."

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Dangerous labelling

SIR,—The varicose vein sclerosing fluid called "S.T.D." (a proprietary preparation containing 3% sodium tetradecyl sulphate) is labelled "for intravenous use" on the bottle and the carton, with no indication of the sclerosant nature of the contents. The fluid is available in many operating theatres.

Although this method of labelling has apparently been passed by the Dunlop Committee, I consider it to be extremely dangerous as the fluid could be drawn up into a syringe and handed to an anaesthetist or surgeon in an emergency and injected into an arm vein, in which case it might not only