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Correspondence

Trends in birth prevalence of cerebral palsy

Sir,

The paper by Pharoah *et al*[1] is a splendid example of the confusion that can arise when a perinatal problem is examined only through the eyes of a paediatrician. I am prepared to concede that a rise in the incidence of cerebral palsy in low birthweight (LBW) infants before 1977 exonerates intensive care as a cause. But the implication that it lets paediatricians off the hook does not necessarily follow.

Firstly, I think I speak for both perinatal pathologists and ultrasonographers when I say that the diagnosis of brain damage as a cause of cerebral palsy is comparatively rare before birth, but becomes increasingly easier postnatally. For those who believe that anatomy is related to function most cases of cerebral palsy appear to have their origins intrapartum and especially in the immediate postpartum period.

Secondly, and here I speak on behalf of obstetricians, the phenomenon of a rising rate of cerebral palsy in LBW infants cannot be understood without knowing the stillbirth rate in LBW infants during the same period. The authors' figures show a declining birth rate (45 637 to 28 430 from 1966–77) which is unlikely to be due entirely to a declining population. At the same time there was a declining risk of LBW at least among live births and hypothetically also among stillbirths. In 1967 there were 68·2 live births below 2500 g per 1000 births, rising to a peak of 79·8 in 1970 and thereafter reducing to 50·8 per 1000 births in 1977. An obstetrician could be expected to interpret this as reflecting improved living standards, family planning, and more successful antenatal obstetric care. This is difficult to reconcile with an increasing risk of being deprived of oxygen during and after birth. I suspect that immediately before the dawn of intensive care for the newborn, growing enthusiasm for relatively unsophisticated resuscitation of small babies provoked the significant rise in the incidence of cerebral palsy as well as an increased chance of survival. From the authors' figures survival below 1500 g rose from 33% to 49% and for infants between 1500 and 2500 g from 94% to 97% for the period 1967–1977.

On the other hand, without knowing how many LBW infants were stillborn it could be argued that the propensity of the Merseyside mother to go into premature labour remained unchanged between 1966 and 1977. A rising stillbirth rate in LBW infants would then necessarily be accompanied by the recorded decline in LBW live births. In this case the blame would lie more at the obstetrician's than the paediatrician's door, because the fewer liveborn infants who escaped the increasing risk of death in utero would be more likely to be hypoxic at birth and more at risk of cerebral palsy.

There is ample evidence that modern obstetric practice and neonatal intensive care have simultaneously had repercussions for each other.[2] Cerebral palsy is unlikely to have been independent of either of these disciplines before the institution of intensive care. The traditional plea of the community paediatrician to examine 'geographically delineated groups' must be challenged. The study of a disease in a community is justified when (as is usual) the disease has its origins there. This would be acceptable for a study of congenital malformations but not for cerebral palsy. Most infants with cerebral palsy have been admitted to a maternity hospital with a morphologically normal central nervous system whilst in utero, and are subsequently discharged to the community with pathological cavities in their brains. Hospital based figures make sense for cerebral palsy as the disease originates within its walls. Geographically delineated groups merely average the results of differing hospital practices.

References

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2 Barson AJ, Tasker M, Lieberman BA, Hillier VF. Impact of improved perinatal care on the causes of death. *Arch Dis Child* 1984;**59:**199–207.

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Professor Pharoah and Drs Cooke, Rosenbloom, and Cooke comment:

In his criticism of our paper, Barson makes several points that are unsubstantiated by any evidence. The first is that the diagnosis of brain damage as a cause of cerebral palsy is rare before birth but becomes increasingly easier postnatally. This may be so, but that cannot be translated to mean that *most* cases of cerebral palsy appear to have their origins either intrapartum or, more especially, immediately post partum. Merely being unable to diagnose brain damage with currently available techniques, including ultrasound, does not mean that abnormality is not already present. Indeed, all the evidence refutes this view and indicates that in most cases of cerebral palsy brain damage occurs well before labour begins. In the cohort followed up by Nelson *et al* only 9% of cases were damaged perinatally.[1] Even among low birthweight (LBW) infants, Stewart *et al* found that 52 of 68 infants with neurodevelopment disorders had no periventricular haemorrhage or parenchymal densities or had only a small periventricular haemorrhage.[2]

In answer to Barson's second point, national data reveal that the stillbirth rates for LBW babies are also falling.[3] In the Mersey Region similar data are not available for the period before 1974, but from 1974 to 1977 the pattern of

decline in stillbirth rates followed the national one. There is, therefore, no evidence that the decline in early neonatal mortality was achieved at the expense of a rise in stillbirth rate.

The final point is that hospital rather than geographically delineated groups should be studied on the basis that the causative factors for cerebral palsy originate within the hospitals. This is emphatically not so; it must be restated that for most cases, even those of LBW, cerebral damage occurs well before birth. To use Barson's phraseology, this lets both obstetricians and paediatricians off the hook, but it behoves all of us to seek the nature and timing of the insult(s) which compromise cerebral development. *Pace* Barson, observing a hospital population cannot account for the biases inherent in differential inter hospital transfer rates and clinical management policies which may affect prevalence without any bearing on the incidence of the condition.

References

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[3] Pharoah POD, Alberman ED. Mortality of low birthweight infants in England and Wales 1953 to 1979. *Arch Dis Child* 1981;**56**:86–9.

Prediction and management of nocturnal hypoglycaemia in diabetes

Sir,

Following the report of Whincup *et al*[1] I have analysed my own data on hospital based blood sugar profiles with rather different results.

A group of 118 diabetic children were admitted to the Royal Manchester Children's Hospital for 24 hours for a study of their renal function.[2] Capillary blood was taken before and one hour after meals and at 0300 hours for estimation of plasma glucose concentrations. In 107 of these children an after supper (usually 2100 to 2300 hours), and 0300 hour plasma glucose estimation were available for predictive analysis. Nocturnal hypoglycaemia was defined as a plasma glucose concentration of less than 3 mmol/l. The table shows the results.

Twenty seven children had a plasma glucose concentration <7 mmol/l after supper and of these only five had one of <3 mmol/l at 0300 hours. Eighty children had a plasma glucose concentration >7 mmol/l after supper and of these three had one of <3 mmol/l at 0300 hours. Thus for these data the after supper plasma glucose value was a poor predictor of nocturnal hypoglycaemia, being correct only about one fifth of the time.

I suspect that the difference between these results and those of Whincup *et al* reflects a difference in the per cent of the two populations that were confirmed positive and confirmed negative. The predictive value of a test depends entirely upon the ratio of confirmed positive and confirmed negative results in the population.[3] In this data set,

Table *Predictive value models for nocturnal hypoglycaemia (n=107)*

Plasma glucose concentration after supper (mmol/l)	*Sensitivity*	*Specificity*	*Predictive value*	
			Positive	*Negative*
<4	25	97	40	94
<5	50	91	31	96
<6	50	85	22	95
<7	63	78	19	96
<8	63	72	18	97
<9	63	65	15	97
<10	63	63	14	97

7·4% of the population were confirmed positive compared with the 34% of that of Whincup *et al*. I can only speculate on what percentage of the general population of diabetic children have nocturnal hypoglycaemia, but from clinical practice I would be surprised if it was more than 10% to 20%.

There is an additional problem with predictive analysis. Feinstein comments that such an analysis has to accept the idea that the results can be listed in a fourfold table.[3] Thus the presence of 'nocturnal hypoglycaemia' is expressed as a simple 'yes' or 'no' and the results of the after supper plasma glucose estimations are similarily interpreted. This is a simplification of the realities of glycaemic control and of home blood glucose monitoring in diabetic children, where the results are rarely 'yes' or 'no' but often 'perhaps' or 'maybe'.

Most British paediatricians ask their diabetic children to perform one blood glucose test at home at a different time each day. Acceptance of this predictive model implies two home blood glucose tests each day; one during the course of the day and one at bedtime. Personally I find it difficult establishing a once daily home blood glucose monitoring routine for most diabetic children and I believe that, for many, a twice daily routine would be difficult to achieve.

In conclusion, therefore, despite the results of the intervention study I remain unconvinced by the argument that paediatricians should be encouraging some of their diabetic children to estimate a nocturnal blood glucose concentration to reduce the incidence of nocturnal hypoglycaemia.

References

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