higher than that reported from such units. To compare with his Wessex neighbours is unjustifiable—clearly they also need better intensive care facilities, and to compare with England and Wales for the first *month* is again quite unsatisfactory. In addition there is much evidence to show that the overall standard of perinatal and neonatal care in this country is a disgrace.

Secondly, I find his Table 3 unsatisfactory. Prematurity is not a diagnosis I recognise between 1001 and 1500 g. I suspect all those 7 babies died from hyaline membrane disease (HMD)—untreated. Furthermore, column one in Table 3 only contains 12 babies, whereas 26 babies of this birthweight died. Some may have had lethal deformities, but presumably the majority died of intrapartum asphyxia. We have shown (Robertson and Tizard, 1975) that it is such small babies who suffer intrapartum asphyxia that suffer the most severe HMD, but many of these are salvagable in a neurologically intact state by modern techniques in neonatal intensive care.

Thirdly, I would hope that all paediatricians would now regard it as a completely unsatisfactory standard of care to provide face mask oxygen for very low birthweight babies without control of arterial oxygen tension.

One very important factor to consider when such data are provided is what are the obstetricians in his unit doing with women with major complications of pregnancy and seriously compromised fetuses at gestations of 28 to 30 weeks? Do they feel they have no alternative but to try to keep the pregnancy going for another week or two with subsequent intrauterine death, or do they in fact send such women to be delivered in a perinatal unit providing modern standards of intensive care? Many of us who run neonatal intensive care units know the latter practice is increasingly common where the local standards of neonatal care are inadequate.

Neonatal intensive care does save lives. The Table shows that neonatal mortality in Oxford was reduced by half over a 3-year period during which an adequate programme of intermittent positive pressure ventilation (IPPV) was instituted. The standards of care described by Dr Hughes-Davies may have been satisfactory in Salisbury, they may be satisfactory in the Solomon Islands, but I hope that all readers of the *Archives* will reject them as being unsuitable for the UK in 1979.

Table Numbers of neonatal deaths, and infants surviving IPPV over 3-year period

Year	Neonatal deaths	Infants surviving IPPV
1971	41	1
1972	28	9
1973	21	21

Reference

Robertson, N. R. C., and Tizard, J. P. M. (1975). Prognosis for infants with idiopathic respiratory distress syndrome. *British Medical Journal*, 3, 271–274.

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Dr Hughes-Davies comments:

Some of the points made by Dr Robertson are covered in my reply to Dr Dunn. I agree that prematurity is an unsatisfactory diagnosis at any weight. Five of our 1001–1500 g babies so labelled were born in 1970 when I was in Saigon and I have no other details except that 3 were 28 weeks or less, and 2 were second twins; it is likely that babies were disturbed more than in other years. Of the other 2, one, born in 1974, weighed 1020 g supposedly at 25 weeks and died at 4 hours after repeated apnoea; the other died at 24 hours in 1971 and weighed 1420 g; histology showed only atelectasis, and neither had HMD. Of the 26 babies dying in this weight range, 9 had prolonged apnoea at birth, 6 in 1971 or before, one in 1972, and 2 in 1974. They did not have HMD. One of the most striking features was the mildness of respiratory distress in babies handled gently in Salisbury (and in the Solomon Islands where I was pleased to meet a very fit baby now 14 months old who weighed 750 g at birth and who was cared for conservatively by Dr Oगतuti). Perhaps babies prefer pastures to battlefields—certainly their need for high concentrations of oxygen is rare, and its risks may be balanced by those of an indwelling arterial catheter which I am sure Dr Robertson would agree is the only worthwhile method of monitoring yet available. Even this is vitiated by a 10% leak from the ductus arteriosus.

No baby to my knowledge was transferred elsewhere either before or after birth except for surgery, and the distribution of weights matches the country as a whole. Patterns of intervention varied as they did elsewhere over the period, but our relations with the obstetricians were good, and I do not think our practices influenced theirs.

Of course intensive care saves lives; and of course it demands a price which can perhaps be justified in hospitals lucky enough to enjoy the skills of Dr Robertson. But I hope he will read my paper again. I have erred if he reads it as an attack on intensive care. It was simply an account of my reaction to practices current 13 years ago, which he suggests are still common today; and I hope he will regard the results as a control group for his own if he cannot bring himself to try the method. Dr Robertson's Table shows a fall in neonatal deaths about the same proportionately as occurred in Southmead and Salisbury and probably elsewhere in the first half of this decade; and, in the absence of an adequate library, I cannot comment on the number surviving IPPV which must depend on the number submitted to it. Again I would ask first for reliable figures; and second for a demonstration, perhaps in a single region, that Dr Robertson's methods can be as effective in less fortunate hands.

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Hypopituitary dwarfism and breech delivery

Sir,
Recent investigations by Rona and Tanner (*Archives*, 1977, 52, 197) show that breech delivery is a common occurrence in patients with idiopathic hypopituitary dwarfism.

We conducted a survey on the type of delivery in 118 patients with idiopathic growth hormone deficiencies (age range 4–18 years) and in 121 healthy control children.

Among the cases of hypopituitary dwarfism, 43 had isolated and 'total' growth hormone deficiency, 64 had isolated and 'partial' growth hormone deficiency (Preece and Tanner, 1976; Boscherini *et al.*, 1978), and 11 had multiple pituitary trophic hormone deficiencies.

The incidence of breech delivery was much higher in patients with hypopituitary dwarfism (19.6%), than in the control group (1.6%) (Fisher's exact probability test: $P < 0.01$). Moreover we observed that breech deliveries were slightly more common in patients with isolated and 'partial' growth hormone deficiencies, certainly more common in patients with isolated and 'total' growth hormone deficiencies, and were very common in patients with multiple pituitary trophic hormone deficiencies (Figure). The trend was significant when analysed by Armitage's χ^2 test ($P < 0.01$).

Figure Incidence of breech delivery in patients with hypopituitary dwarfism. (A) Control group, (B) partial growth hormone deficiency, (C) total growth hormone deficiency, (D) multiple pituitary trophic hormone deficiency.

The high frequency of breech delivery in our patients and the close relationship between the frequency of

podalic presentation and the severity of functional pituitary impairment support the view that breech delivery plays an important pathogenetic role in idiopathic hypopituitary dwarfism.

References

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Paediatric Ophthalmology Study Group

Sir,
This interdisciplinary study group on Paediatric Ophthalmology was founded by the late Dr Ronnie Mac Keith. It includes ophthalmologists, paediatricians, neurologists, psychologists, teachers, social workers, and others involved in the care of visually handicapped children. Its objectives are to further the knowledge of the subject, exchange ideas, and stimulate interest in this field to the eventual benefit of children who are visually handicapped. The group meets twice a year; in the autumn at the Newcomen Centre, Guy's Hospital, when formal papers followed by discussion are presented, and early in the year when visits are made to research units, special schools, or to any department concerned with the problems of paediatric ophthalmology. The present Convener is Dr Neil Gordon, Booth Hall Children's Hospital, and the organisation of the group is supported by the Spastics Society.

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