Original articles

Impact of improved perinatal care on the causes of death

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SUMMARY A total of 440 perinatal deaths occurring in a maternity hospital over a 6 year period have been analysed clinically and pathologically. The decline in mortality could be attributed to a reduction in asphyxial deaths, lethal malformations, and macerated stillbirths. The establishment of a neonatal intensive care unit seemed to have been more successful in combating birth asphyxia than respiratory distress syndrome. Although the greatest reduction in perinatal mortality was in babies with birthweights between 1 and 1.5 kg, there was no decline in the deaths from hyaline membrane disease or intraventricular haemorrhage, or both. The most striking change was the drop in asphyxia as a cause of perinatal death which was independent of birth trauma. Earlier diagnosis of fetal distress with obstetric intervention and the establishment of the intensive care unit were seen as the main factors in this change, and their mutual dependence and evolution are emphasised.

To a perinatologist the documentation of the causes of death in adults seems enviably simple. For example, an obstetrician may recognise placental abruption as the cause of a premature infant's death, whereas the paediatrician may see this as a death from respiratory distress syndrome, and the pathologist as death from intraventricular haemorrhage. All three specialists are in some sense correct but none feels at ease with the classification adopted by the other two. Of the three the obstetrician is usually the least dependent on the pathologist's opinion, often opting to categorise perinatal deaths in a way that may be supplemented by necropsy findings but not be wholly dependent on them.¹⁻⁴ Thus death is assigned to clinically diagnosable conditions such as pre-eclampsia, antepartum haemorrhage, and major malformation. The difficulty with such a system lies in its lack of specificity. Thus the commonest cause of death in the Scottish perinatal mortality survey² was 'birthweight below 2500 g with no maternal complication'. This is broadly equivalent to saying that most babies die because they are born too soon, and is just as unsatisfactory as having to attribute most adult deaths to old age.

On the other hand a purely morbid anatomical analysis of perinatal deaths has its own difficulties.

The specialised experience required for good perinatal necropsies is not available in every centre.^{5 6} Pathological studies almost always list the incidence of disease in a hospital population⁷⁻¹⁰ rather than an incidence among births in the community. The clinical relevance of the pathology is further undermined by the limitations inseparable from specialisation. The pathologist's difficulties in assessing the role of maternal disease in perinatal death are exactly comparable to the obstetrician's difficulties in the interpretation of fetal morbid anatomy.

To overcome some of these problems Wigglesworth¹¹ suggested a two stage analysis of perinatal deaths in which all deaths were placed in one of five categories and then examined in body weight groupings. The classification was proposed as a simpler and more effective form of hospital audit to the confidential enquiries into perinatal deaths that have been advocated.^{4 12} We have been able to analyse a far larger number of perinatal deaths over a longer period than that originally published by Wigglesworth, and most were supported by necropsy information. The survey covered a 6 year period after the establishment of an appropriately staffed intensive care unit for the newborn and the appointment of an obstetrician specifically to supervise labour ward management. The effect

of these changes on the causes of perinatal death has been examined.

Patients and methods

All the deaths in the survey occurred among the 26 596 births in St Mary's Hospital between 1976 and 1981. Liveborn infants referred from other hospitals in the region to the intensive care unit were excluded, as were referred perinatal deaths. A small number of deaths were included where a mother had been transferred antenatally from another hospital. During this period there were 228 stillbirths and 212 deaths in the first week of life, making a total of 440 perinatal deaths. The legal definition of perinatal death in the United Kingdom was used—that is, all infants requiring death certification on the basis of being classified clinically as stillborn over 28 weeks' gestation or as liveborn irrespective of gestation, but dying during the first postnatal week.

Of the 440 perinatal deaths, 361 (82%) had had a full necropsy examination by, or under the supervision of, a senior pathologist (AJB). This examination included routine histology of all major organs, whole body radiology, and bacteriology. Cytogenetic, virological, and biochemical investigations were performed only when indicated by circumstance.

All perinatal deaths were assigned to one of five categories according to a classification suggested by Wigglesworth:¹¹

- (1) Normally formed macerated stillbirths.
- (2) Lethal congenital malformation, still or liveborn.
- (3) Livebirths dying from conditions associated with immaturity.
- (4) Intrapartum hypoxia; fresh stillborn or liveborn.
- (5) Specific conditions other than above, still or liveborn.

In order to use this classification consistently certain rules had to be followed:

(a) The presence of a malformation which could be expected to be lethal if it occurred in isolation, overrode any other consideration, such as hypoxia or immaturity that might in fact have caused death in individual infants.

(b) Deaths from immaturity were composed almost wholly of infants with pathological hyaline membrane disease or intraventricular haemorrhage, or both, or at least a clinical history of respiratory distress syndrome. Preterm infants were excluded from this category if there was profound hypoxia at birth that was unresponsive to resuscitation.

(c) Death was attributed to hypoxia only where this developed during labour and resulted in a fresh

stillbirth or a deeply asphyxiated infant unresponsive to resuscitation. Deaths due to birth trauma were included in this group.

(d) Deaths from specific conditions were almost wholly limited to rhesus haemolytic disease and identifiable intrauterine infections. Necrotising enterocolitis and meningitis were regarded as specific conditions but preterm infants dying from other forms of sepsis such as pneumonia were classified as deaths from immaturity.

Results

The total annual number of births in St Mary's Hospital showed little fluctuation during the 6 years of the survey (minimum 4238, maximum 4603 births each year). The stillbirth and perinatal mortality rates/1000 total births and the neonatal mortality/1000 livebirths are shown in Table 1. All three rates declined over this period but the neonatal deaths increased for the first five years. A sharp fall in the deaths among liveborn infants in 1981 lowered noticeably both the neonatal and perinatal mortality rates.

Table 2 shows the breakdown of the 440 perinatal deaths into the Wigglesworth classification (abbreviated to 'macerated', 'malformed', 'immature', 'asphyxial', and 'other') and then further classifies them according to body weight (0.5 kg categories). The largest group were the macerated stillbirths (127) followed by deaths from malformation (109), immaturity (107), and asphyxia (72). The more specific conditions ('other') were in a minority of 25. The macerated and malformed deaths showed no clear relation to body weight. Not surprisingly, preterm deaths predominated in babies weighing below 1 kg, and the asphyxial deaths were most common above 2.5 kg.

The relation between body weight and perinatal mortality is shown clearly in Fig. 1, where the annual perinatal mortality has been plotted in 0.5 kg body weight categories, and in Table 3 which

Table 1 Stillbirths and perinatal mortality rates/1000 liveborn infants between 1976 and 1981

	1976	1977	1978	1979	1980	1981
Total births	4238	4433	4603	4482	4455	4385
Stillbirths	55	41	35	32	38	27
Perinatal deaths	87	80	78	69	75	51
Neonatal deaths	38	42	46	46	48	24
Stillbirth rate	13.0	9.3	7.6	7.1	8.5	6.2
Perinatal mortality rate	20.5	18.1	17.0	15.4	16.8	11.6
Neonatal mortality rate	9.1	9.6	10.1	10.3	10.9	5.5

Table 2 Perinatal deaths classified by cause and birthweight

Birthweight (kg)	Macerated No (%)	Malformation No (%)	Immaturity No (%)	Asphyxia No (%)	Other No (%)	Total No (%)
<1	19 (15.0)	15 (13.8)	67 (62.6)	5 (6.9)	2 (8.0)	108 (24.5)
1-1.5	29 (22.8)	23 (21.1)	27 (25.2)	11 (15.3)	7 (28.0)	97 (22.0)
1.5-2	28 (22.0)	19 (17.4)	12 (11.2)	9 (12.5)	8 (32.0)	76 (17.3)
2-2.5	18 (14.2)	23 (21.1)	1 (0.9)	11 (15.3)	5 (20.0)	58 (13.2)
2.5+	33 (26.0)	29 (26.6)	0 —	36 (50.0)	3 (12.0)	101 (23.0)
Total	127 (100)	109 (100)	107 (100)	72 (100)	25 (100)	440 (100)

Fig. 1 Annual perinatal mortality in St Mary's Hospital between 1976 and 1981 plotted in 0.5 kg body weight categories.

Table 3 Ratio of perinatal deaths/total births by year and birthweight

Birthweight (kg)	1976	1977	1978	1979	1980	1981
<1	14/15	15/22	24/33	16/21	22/35	17/31
1-1.5	20/30	20/35	16/49	15/64	13/56	13/59
1.5-2	14/76	18/100	14/89	13/89	12/117	5/84
2-2.5	15/262	6/271	9/283	10/285	11/269	7/258
2.5+	24/3855	21/4005	15/4149	15/4023	17/3978	9/3953

records the actual numbers of births and deaths by body weight. The decline in perinatal mortality detailed in Table 1 must be attributed to the improved prognosis among the minority of babies weighing less than 1.5 kg. Improved mortality during the study period was greater for infants weighing between 1 and 1.5 kg (667 to 220/1000 births) than for those weighing less than 1 kg (933 to 548/1000 births) and those weighing between 1.5 and 2 kg (184 to 60/1000 births). The improved prognosis for infants weighing 1 to 1.5 kg was achieved during a period when the number of

births in this category had nearly doubled (from 30 to 59).

Fig. 2 illustrates the lives which were being saved in 1981 that 6 years previously had been lost. It shows the four main categories of perinatal death expressed as an annual incidence/1000 total births. It is evident that the decline in perinatal mortality resulted from a reduction in macerated stillbirths, congenital malformations, and in asphyxial deaths; the latter being the most dramatic (5.0 to 1.1/1000 births). Deaths caused by immaturity increased between 1976 and 1978 (1.9 to 5.2/1000 births) and showed minimal evidence of decline thereafter.

Further insight can be gained by more detailed analysis of the asphyxial and immature deaths. It has been the practice in previous mortality studies^{8 13} to group deaths from asphyxia with those from birth trauma on the grounds that the two are often found together, each predisposing to the other. One might therefore anticipate that the decline in asphyxial deaths between 1976 and 1981 was attributable in part to a decline in birth injury. Tentorial tears represent a common, easily identifiable, and specific form of intracranial trauma. Fig. 3 shows the annual incidence of tentorial tears found at necropsy among

Fig. 2 Incidence between 1976 and 1981 of the four main categories of perinatal death—macerated stillbirth, malformation, asphyxia and immaturity.

Fig. 3 Annual incidence of asphyxial, perinatal deaths without evidence of birth trauma compared with the incidence of tentorial tears found at necropsy.

the perinatal deaths, and compares this with asphyxial deaths in whom there was no evidence of birth trauma. It seems that the decline in asphyxial deaths was not only largely independent of birth injury but that the two conditions, far from potentiating each other, show a reciprocal relation.

Since the greatest decline in perinatal mortality was in those infants weighing between 1 and 1.5 kg, this group was examined separately. Fig. 4 depicts the annual incidence for the four major categories in perinatal deaths/1000 births among infants weighing between 1 and 1.5 kg and the actual figures are given in Table 4. The numbers of macerated, malformed, and asphyxial deaths all declined among this group of infants, and indeed no asphyxial death was recorded in 1981. Deaths from immaturity showed

Fig. 4 Annual incidence of the four main categories of perinatal death in infants weighing between 1.0 and 1.5 kg from 1976 to 1981.

Table 4 Yearly totals by mortality group and perinatal mortality rates/1000 births among babies weighing between 1.0 and 1.5 kg

Yearly totals by mortality group	1976	1977	1978	1979	1980	1981
Total births	30	35	49	64	56	59
Livebirths	20	24	41	59	51	56
Stillbirths	10	11	8	5	5	3
Early neonatal deaths	10	9	8	10	8	10
Perinatal deaths	20	20	16	15	13	13
Macerated	6	7	7	4	2	3
Malformation	7	3	3	3	4	3
Immaturity	2	4	4	5	5	7
Asphyxia	4	5	1	1	1	0
Other	1	1	1	2	1	0
Perinatal mortality rates/1000 births						
Macerated	200	200	143	63	36	51
Malformation	233	86	61	47	71	51
Immaturity	67	114	82	78	89	119
Asphyxia	133	143	20	16	18	0
Other	33	29	20	31	18	0
Immature deaths/1000 livebirths	100	167	98	85	98	125

no decline, however, and were greater at the end of the study period (67 in 1976 to 119 in 1981/1000 births for infants weighing between 1 and 1.5 kg).

These figures and the apparent trends shown in the graphs were subjected to a statistical analysis that is summarised in Table 5. There were significant linear downward trends in the perinatal deaths and stillbirths from 1976 to 1981 but not in the neonatal deaths. This decline in perinatal deaths among babies weighing 1 to 1.5 kg was highly significant ($P < 0.001$) but mortality among the other body weight groups did not show significant trends. Changes in the macerated and malformed deaths failed to realise significance but there was a statistically significant downward trend in the asphyxial deaths (against both other deaths and births) and an upward trend in the immature deaths (against other deaths but not births).

Analysis of the deaths among babies weighing 1 to 1.5 kg showed a linear decline in the stillbirths and in macerated and malformed deaths when assessed against the births but not when assessed against other deaths. There were too few asphyxial deaths in babies of this weight for statistical analysis, although the numbers suggested a decline. Immature deaths in babies weighing between 1 and 1.5 kg as a proportion of all perinatal deaths showed a significant upward trend, although this failed to achieve significance when assessed against the other births.

Table 5 Statistical analysis of perinatal deaths and categories of perinatal death in St Mary's Hospital for the years 1976 to 1981

	Group difference	Linear trend
<i>Deaths of all weights against all births</i>		
Perinatal deaths	P=0.040	Downward
Stillbirths	P=0.013	Downward
Neonatal deaths	NS	—
<i>Deaths by weight against all births</i>		
Perinatal deaths <1 kg	NS	—
Perinatal deaths 1-1.5 kg	P<0.001	Downward
Perinatal deaths 1.5-2 kg	NS	—
Perinatal deaths 2-2.5 kg	NS	—
Perinatal deaths >2.5 kg	NS	—
<i>Deaths in causal categories of all weights</i>		
Macerated against other deaths	NS	—
Macerated against other births	NS	—
Malformation against other deaths	NS	—
Malformation against other births	NS	—
Asphyxia against other deaths	P=0.017	Downward
Asphyxia against other births	P=0.001	Downward
Immature against other deaths	P=0.011	Upward
Immature against other births	NS	—
Immature against deaths in livebirths	P=0.043	Upward
Immature against other livebirths	NS	—
<i>Deaths among infants weighing 1.0 to 1.5 kg</i>		
Stillbirths	P<0.001	Downward
Early neonatal deaths	NS	—
<i>Deaths in causal categories weighing 1.0 to 1.5 kg grouped in paired years</i>		
Macerated against other deaths	NS	—
Macerated against other births	P=0.004	Downward
Malformation against other deaths	NS	—
Malformation against other births	P=0.036	Downward
Asphyxia against other deaths	Insufficient numbers	—
Asphyxia against other births	Insufficient numbers	—
Immature against other deaths	P=0.022	Upward
Immature against other births	NS	—
Immature against deaths in livebirths	NS	—
Immature against other livebirths	NS	—

NS=not significant.

Group difference refers to the χ^2 test for the difference in annual values over the 6 year period.

Linear trend refers to statistical testing of these values for a departure from a linear regression of the annual values. A positive trend implies that there is no significant departure from a linear regression. This test is only valid when the initial χ^2 test on the group difference is significant.

Because of the small numbers analysis of the deaths in the 1 to 1.5 kg body weight group was performed on the paired years 1976 to 1977, 1978 to 1979 and 1980 to 1981.

A possible criticism of this analysis is that immature deaths must, by definition, be liveborn, just as those which are macerated must be stillborn. There was, however, a significant upward trend in the number of immature deaths, not only as a proportion of all other perinatal deaths but also in comparison with deaths among liveborn infants. The figures did not reach significance when immature

deaths were compared with all other live births or when analysed specifically against live births in the 1 to 1.5 kg body weight category.

Discussion

The advantage of Wigglesworth's classification¹¹ of perinatal deaths is that it combines simplicity with implications for clinical management. Thus the numbers of macerated stillbirths and asphyxial deaths, especially among large babies, is of particular obstetric concern, while the survival of the immature infant, albeit that many are born electively, is of prime paediatric interest. Wigglesworth confined his asphyxial deaths to those conditions that developed in labour. A basis for intrapartum asphyxia was always evident in the present survey, however, about 45% of macerated stillbirths had no adequate obstetric or pathological explanation. Deaths among liveborn infants invariably had at least one recognisable cause. The importance of a high necropsy rate cannot be overemphasised. About a quarter of deaths, live and stillborn, are due to congenital malformation, many of which go unrecognised without a methodical necropsy. It is by no means a rare event for a preterm infant with respiratory distress to have his symptoms attributed, with the wisdom of hindsight, to congenital heart disease or intracranial haemorrhage from a birth injury rather than the presence of hyaline membranes.

The 440 perinatal deaths reviewed in this survey cover a 6 year period after the introduction of ventilatory support for preterm infants within an intensive care unit that has become progressively better staffed, more experienced, and technologically sophisticated. As antepartum and intrapartum fetal heart monitoring and ultrasound scanning have become common the ability of the obstetrician to assess fetal welfare has increased and there has been a greater readiness to intervene where the pregnancy has seemed to be in jeopardy. This has been associated with an increase in the numbers of preterm births and a caesarian section rate that has risen from 10.2% in 1976 to 18.2% in 1982. It would be an oversimplification, however, to regard the evolution of the obstetric and neonatal services as independent of each other. The very success and availability of a neonatal intensive care unit encourages an obstetrician to deliver a mother prematurely when the prognosis for conservative management is known to be poor.

The relation between improved obstetric and neonatal care is represented diagrammatically in Fig. 5. There are a number of well documented accounts of the effect of neonatal intensive care on

Fig. 5 Relation between improvements in obstetric and neonatal care and perinatal mortality.

mortality.¹⁴⁻¹⁹ In broad terms, infants weighing less than 1.5 kg comprise about 1% of all births, but 50% of neonatal mortality.¹⁴ It is this group, however, that has most to gain from the establishment of a neonatal intensive care unit, and this fact was confirmed by our own experience (Fig. 1). What previous studies have not shown is which infants are being saved by intensive care, not in terms of their body weight, but in respect of their pathology. There is often a tacit assumption that a declining mortality among infants weighing less than 1.5 kg is synonymous with the successful treatment of respiratory distress syndrome. The present survey, perhaps surprisingly, does not endorse this view.

Whether the perinatal deaths are examined as a whole (Fig. 2) or specifically for that body weight category where most success has been achieved (Fig. 4, Table 4), the most dramatic decline has been in the asphyxial deaths among both still and liveborn infants. It is the authors' conviction that a greater awareness of fetal compromise and a willingness to intervene has led to a steady decline in both the macerated births and the asphyxial deaths. It is possible that the drop in the incidence of fetal malformation is in part due to the diagnosis and early abortion of these conditions, but it is more likely that this is merely a local manifestation of the national decline in lethal congenital defects of the central nervous system.²⁰

Not only is early delivery encouraged by the presence of a neonatal intensive care unit but those infants born alive but asphyxiated benefit from the

ready availability of expert resuscitation. The evidence of this survey seems to indicate that this is a major justification for such a unit. The steep decline in asphyxia as a cause of perinatal death between 1976 and 1981 is a product of both pre- and postnatal care (Fig. 5).

The satisfying drop in perinatal mortality has not been without its drawbacks. Early intervention for antepartum fetal compromise has necessarily resulted in the birth of more preterm infants (Table 3). In addition, preterm infants born spontaneously have been treated more enthusiastically, even when their birthweight is below 1 kg. It is inevitable that neonatal care which saves the lives of some merely prolongs the survival of others (Fig. 5). An infant who in 1976 died of asphyxia because resuscitation was withheld or came too late, may well in 1981 have been kept alive on a ventilator long enough for the formation of hyaline membranes, with or without intraventricular haemorrhage. How far ventilator care saves lives from the consequences of immaturity as opposed to birth asphyxia is difficult to assess. The figures in the present survey indicate an increase in the number of infants dying of hyaline membrane disease and intraventricular haemorrhage which as a proportion either of perinatal deaths or of deaths among liveborn infants is statistically significant (Table 5). Taking the group of infants weighing 1 to 1.5 kg, there were 6 immature deaths in the years 1976-7 compared with 13 deaths in livebirths from other causes. In 1980-1 this had changed to 12 immature deaths and 6 deaths

from other causes. Although this apparent increase in immature deaths is not statistically significant we can at the very least say that there has been no decline in immature deaths among infants weighing between 1 and 1.5 kg. On the other hand perinatal deaths, stillbirths, and asphyxial deaths have, by any criteria, declined over the period of the survey. One is bound to wonder whether the amelioration of intrauterine asphyxia by elective preterm delivery without labour predisposes the infant to more severe respiratory distress syndrome. Certainly it is easier to show the neonatologist's success in combating birth asphyxia in the immediate neonatal period than the gains he has made in the longer term consequences of immaturity. Even so the figures must be seen against a background in which the number of infants of birthweight below 1.5 kg has risen from 45 with 34 deaths in 1976 (76%) to 90 with 30 deaths in 1981 (33%).

Without expert perinatal necropsies birth trauma is greatly underdiagnosed. Its incidence is now so low, however, that the time is opportune to reassess its relation to asphyxia. Prolonged second stage labour and high forceps delivery of large babies is now a thing of the past. These circumstances a generation ago doubtless predisposed to the coincidence of asphyxia and trauma,¹³ but analysis of traumatic and atraumatic asphyxia in the present survey shows a reciprocal relation between the two (Fig. 3). Currently, it is probably true to say that a hurried delivery demanded by awareness of fetal distress carries with it a slight but appreciable risk of birth trauma. Referring to Fig. 3 the peak in asphyxial deaths in 1980, which was against the overall trend, was caused by an unusual number of intrapartum asphyxial deaths from placental abruption. Rapid intervention was precluded by the knowledge of fetal death, and hence the possibility of birth trauma by precipitate delivery did not arise.

Finally it should be noted that obstetricians located away from perinatal centres often underestimate the viability of the low birthweight infant.²¹ Moreover, the neonatal mortality rate may be reduced without there being any comparable decline in the stillbirth rate.¹⁵ The obstetric and neonatal services which are the subject of the present survey have developed in the same hospital and have continuously and, perhaps to a degree, unconsciously influenced each other's evolution. The year 1976 marked the availability of ventilatory care for preterm infants in St Mary's Hospital but it would not be true to say that technological advance began only in that year. The mortality for low birthweight infants was so high, however, in 1976 (in excess of 660/1000 births for infants weighing below 1.5 kg) that the effect of therapeutic innovation could not

have been so striking before that date than it was in the 6 subsequent years.

There have been no major changes in the composition of the population from which this hospital draws its patients during the period of the survey and the decline in mortality specifically in the group of babies weighing 1 to 1.5 kg is unlikely to be attributable to such a change. Antenatal transfers have become more common but only 20 of the 440 deaths during the study occurred in infants whose mothers had been transferred. Unfortunately valid comparisons cannot really be made with adjacent hospital districts because of the lack of necropsy information. With the exception of deaths from malformation, which are largely outside the clinician's control, the improvement in perinatal mortality has been caused by earlier obstetric intervention and expert resuscitation. It is not stretching the truth too far to give some of the credit for the reduction in stillbirths to the availability and proved expertise of the neonatal intensive care unit. The opposing influences on the perinatal mortality depicted in Fig. 5 are mainly towards an overall decline but the price has been an increase in the number of liveborn immature infants. While lethal birth asphyxia has become appreciably less common, the longer term consequences of survival in this group in the form of hyaline membrane disease and intraventricular haemorrhage have actually increased (Table 5). The two main obstacles to a further drop in perinatal mortality lie in reducing the number of women going into spontaneous preterm labour and the prevention of intraventricular haemorrhage among liveborn low birthweight infants. The interaction between obstetrics and neonatology is such that advances in one inevitably have consequences for the other. The purpose of the present study was to examine fully perinatal deaths as a coherent group. The separation of the causes of stillbirths from those of first week deaths is likely to be even more artificial in the future than it has always been in the past.

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Commentary

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We are presented with a conundrum. Obstetricians have more confidence in paediatricians. That confidence is based on a belief that paediatricians are now able to save the lives of babies with hyaline

membrane disease who would in the past have died. It leads obstetricians to deliver more preterm babies whose lives are saved, not because of better treatment of hyaline membrane disease, but because of the prevention or treatment of hypoxia. Is the whole cycle based on a false premise or do obstetricians and neonatal paediatricians believe correctly that intensive care saves the lives of babies with hyaline membrane disease?

Full modern intensive care of the newborn was introduced at St Mary's, Manchester in 1976 and there was a sharp fall in neonatal mortality that year.¹ (First week mortality rate falling from 13 in 1975 to 7 in 1976.) In order to assess the impact of intensive care on causes of death, therefore, we would need to compare data from before 1976 with data after 1976.

Thirty six fewer babies died at St Mary's in 1981 than in 1976. There were three more deaths in babies weighing less than 1 kg, 7 fewer in the 1 to 1.5 kg group, 9 fewer in the 1.5 to 2.0 kg group, 8 fewer at 2 to 2.5 kg, and 15 fewer weighing more than 2.5 kg. In absolute terms, therefore, the major contribution to the improved perinatal mortality over this period was a saving of larger babies. If the change in distribution of birthweights is taken into account, however, comparing the weight specific perinatal mortality in 1981 with that in 1976, the savings become 11 babies in those weighing less than 1.0 kg, 27 in the 1 to 1.5 kg group, 10 in the 1.5 to 2.0 kg group, 8 at 2 to 2.5 kg, and 15 over 2.5 kg.

Deaths from immaturity among babies weighing 1.0 to 1.5 kg did not fall over the period of the study. There are several possible explanations:

(1) Neonatal intensive care, as practised in Manchester, does not save the lives of babies with hyaline membrane disease.

(2) Neonatal intensive care caused a maximal reduction in mortality from hyaline membrane disease in 1976 with no further improvement since then.

(3) Some babies who would previously have died of asphyxia are now saved from asphyxia only to die of hyaline membrane disease or intraventricular haemorrhage.

(4) There has been an increase in the incidence of severe hyaline membrane disease in this birthweight group for some other reason.

(5) Improvement in the management of respiratory failure has been accompanied by an increase in deaths from intraventricular haemorrhage.

(6) Increased sophistication in the diagnosis and assessment of intraventricular haemorrhage has led to the elective withdrawal of intensive care from some babies who might otherwise have survived severely handicapped.