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The importance of the umbilical circulation in preventing respiratory distress syndrome following premature Caesarean delivery, 1957 – 1973*

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My first talk to the Paediatric Visiting Club was in October 1969 when I spoke about the significance of neonatal polycythemia to a meeting hosted by John Apley in Bath[1]. John had founded the PVC ten years earlier in 1957 with a small group of senior paediatric colleagues.

By chance 1957 was the year that I started a ten year clinical apprenticeship in perinatal medicine commencing at the Birmingham Maternity Hospital. It was in fact a remarkable institution. As the University teaching hospital for Birmingham, it took problem obstetric cases from all over the Midlands. As a result, it had a Caesarean section rate of 20% when the national average was 2.5%. The provision for newborn care was primitive: no rooming-in, no incubators or equipment, no special care baby unit, and no paediatric houseman. Just one paediatric registrar, myself, who was also responsible for the Queen Elizabeth Hospital obstetric unit, four miles away.

When I arrived the perinatal mortality was very high – over 70 per 1,000 births. After birth the main cause of neonatal death was respiratory distress syndrome of the premature infant (RDS) (Fig 1). Some 40% of these infants died within three days of birth. As I was able to show, both the incidence of the disease and its mortality rose steadily with decreasing gestational age
(Fig 2)[2].

In those days I spent a great deal of my time in the labour wards and in the Caesarean section theatre, as one of my main responsibilities was neonatal resuscitation. My studies revealed that Caesarean born infants were twice as likely to develop RDS than those delivered vaginally and were several times more likely to die[3] (Fig 3). Indeed, in 1961, 32% of all neonatal deaths in the hospital took place among the 2.6% of infants delivered prematurely by Caesarean section – a twelve-fold increase. Furthermore, I was able to show that this increased mortality was due to the Caesarean section itself rather than to the indication leading to the need for that method of delivery[2].

Among a number of clinical observations that I made at that time was that babies developing RDS were usually in fairly good condition at birth but slow to breathe, usually due to maternal sedation or anaesthesia. Their still pulsating cords were then clamped and cut (Fig 4) so that they could be moved to the resuscitation trolley.

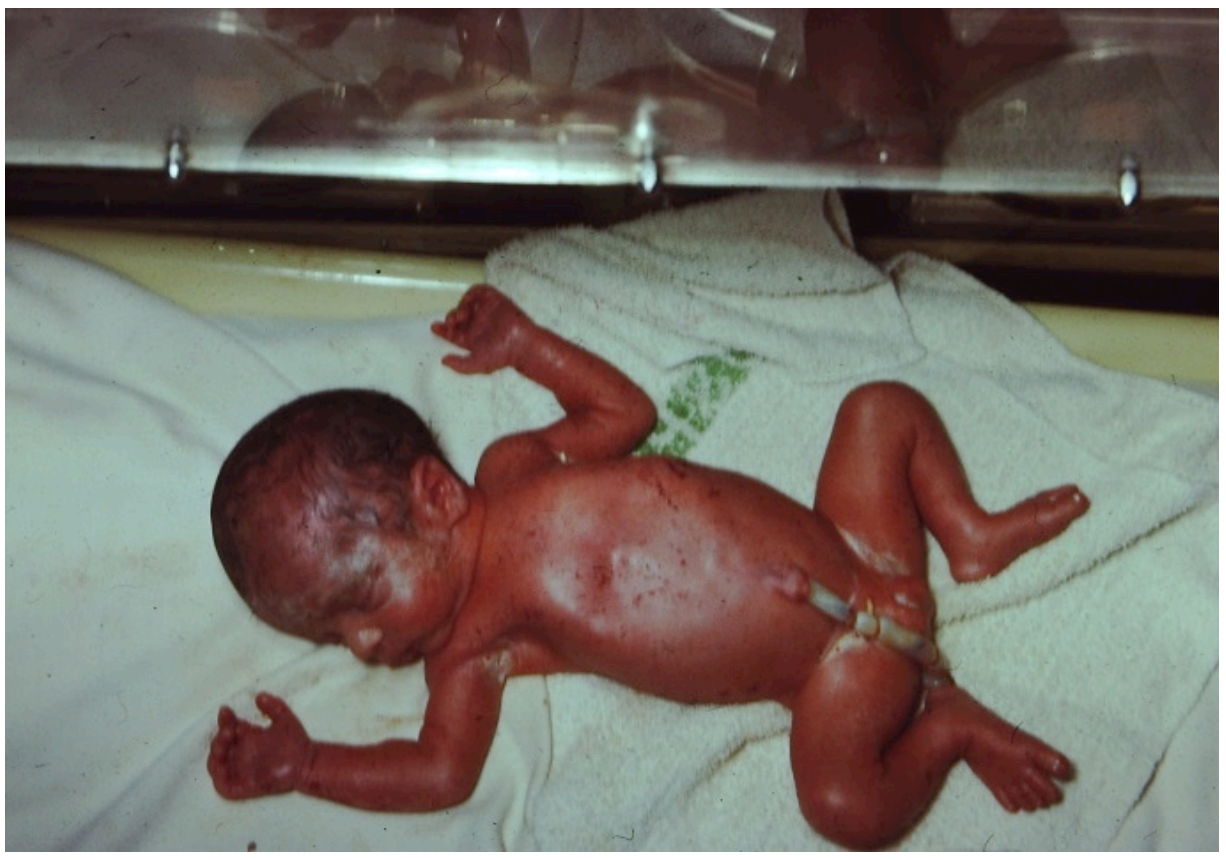

Fig 1.
Premature infant with respiratory distress syndrome on the first day of life.

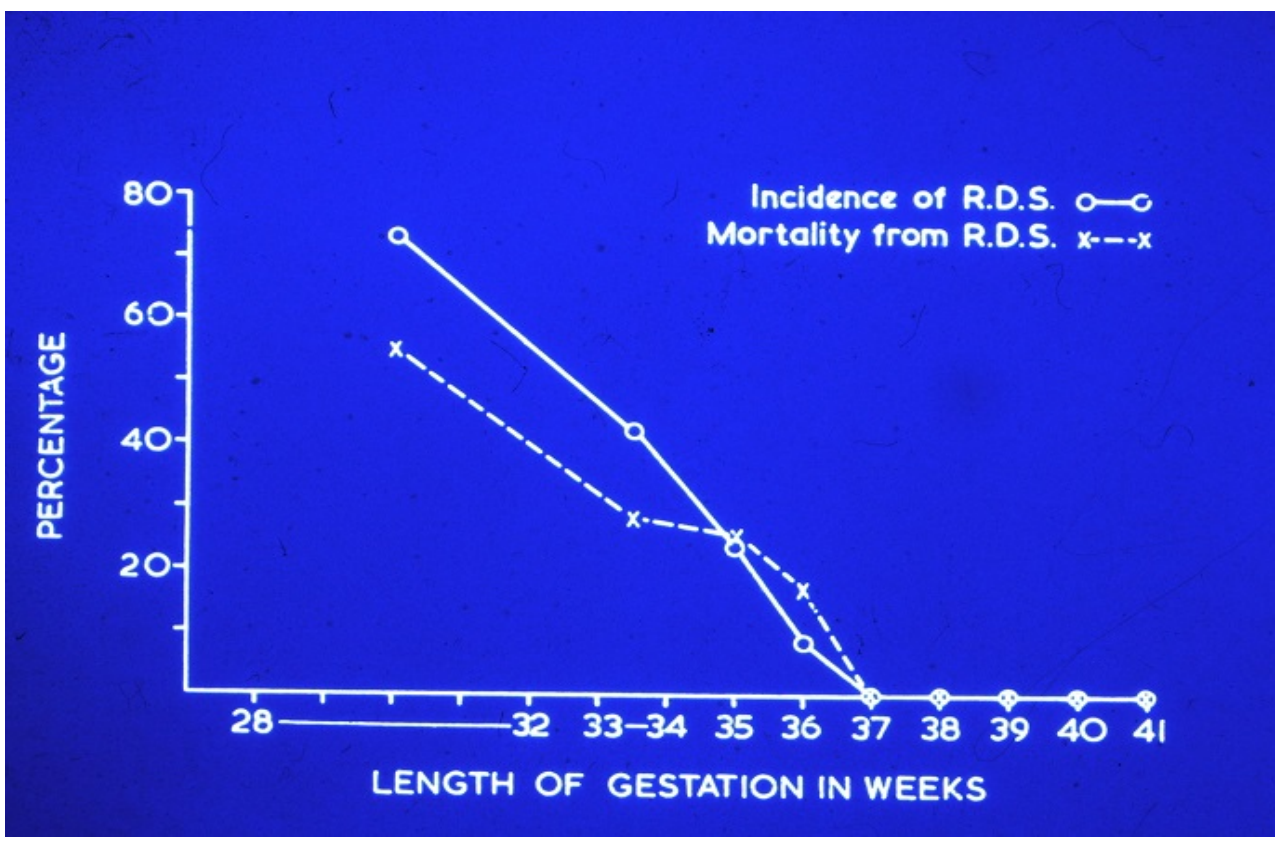

Fig 2.
Incidence of the respiratory distress syndrome (RDS) and of neonatal mortality in relation to length of gestation among premature infants born in the Birmingham Maternity Hospital in 1961[2].

*** Based on a talk to The Paediatric Visiting Club, Taunton, September 2016**

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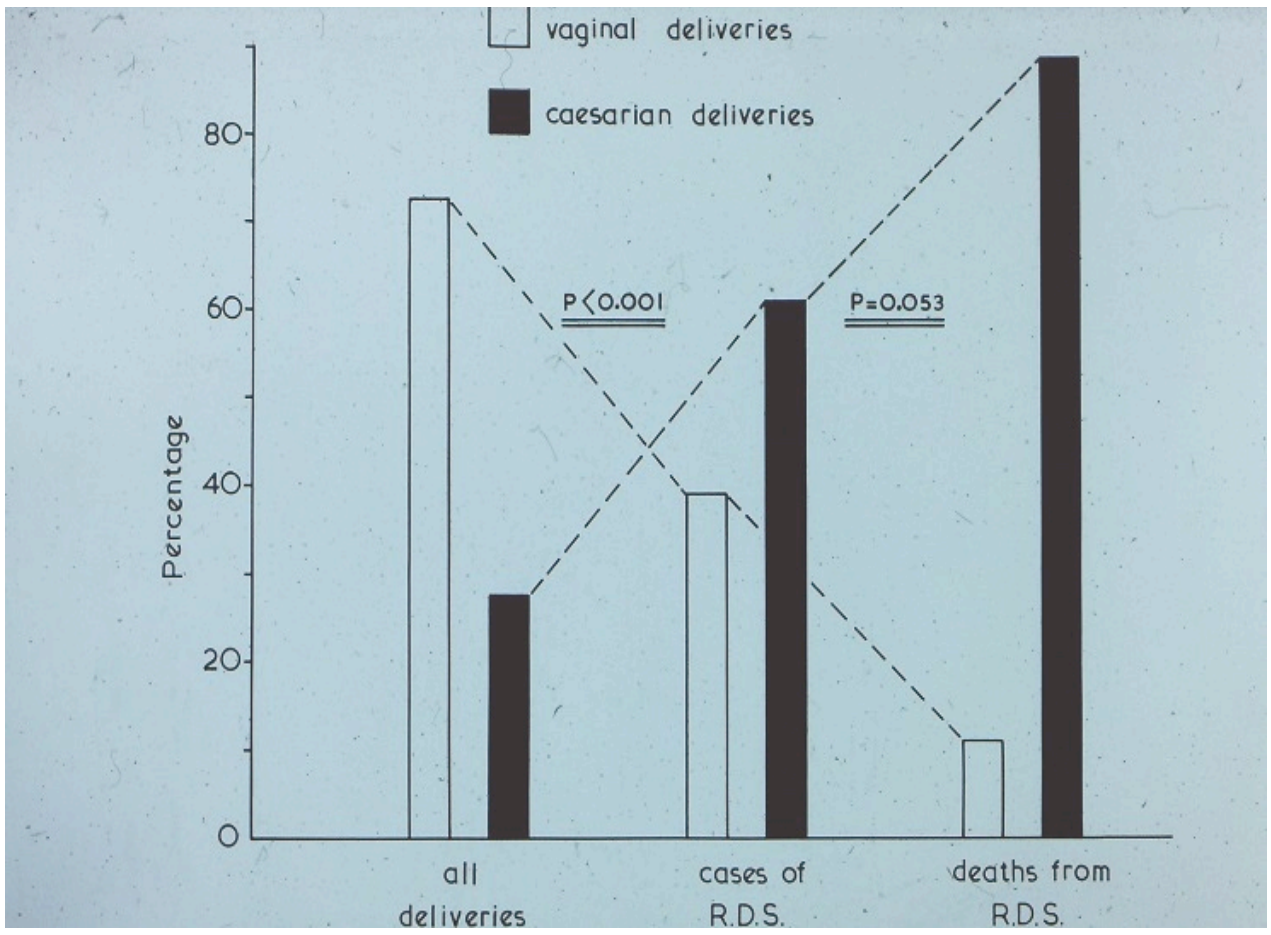

Fig 3.
Mode of delivery among 164 infants born alive after a gestation of 33-36 week and the influence of this on the incidence of RDS and neonatal mortality (Birmingham Maternity Hospital, 1961)[3].

Fig 4.
An early clamped umbilical cord at birth.

Gradually I became convinced of the importance of achieving alveolar ventilation prior to imposing circulatory change on the baby by clamping the pulsating umbilical cord. Unfortunately as a paediatrician I had no control of either the use of maternal sedation or the timing of cord division. Obstetric interest in the latter was minimal. Sir Eardly Holland's great text book, *'Obstetrics, 1958'*, rather dismissed this important obstetric intervention in two lines:

'Opinions differ about the time at which the cord should be tied. Many obstetricians, including myself, do it without delay ...'

Looking back, by 1960 my written instruction to the obstetric houseman at the BMH read as follows:

'Whenever possible the umbilical cord should not be clamped until the infant has taken at least two breaths. Provided respiration has become established, the cord should be clamped 20-30 seconds after delivery. If ergometrine has been administered intravenously to the mother, cord clamping should not be delayed more than 30 seconds after delivery.'

Furthermore, in 1961 I gave a presentation on the management of the umbilical cord at birth to the Birmingham Children's Hospital[4]. This is an extract from my talk:

'At normal delivery, the cord is allowed to cease pulsating before being clamped and divided. This usually takes 3-4 minutes ....
It is important to allow the cord to stop pulsating before clamping. This allows more normal and physiological transition from fetal to adult circulation. I believe sudden clamping of a strongly pulsating cord to be a very important factor in the aetiology of respiratory distress syndrome of the premature infant ...'

I was of course particularly concerned about the premature babies delivered by Caesarean section as they had the highest mortality from RDS. I was always summoned to such Caesarean sections in order to be available to resuscitate the baby. The problem was how to ensure the baby breathed before the cord was clamped.

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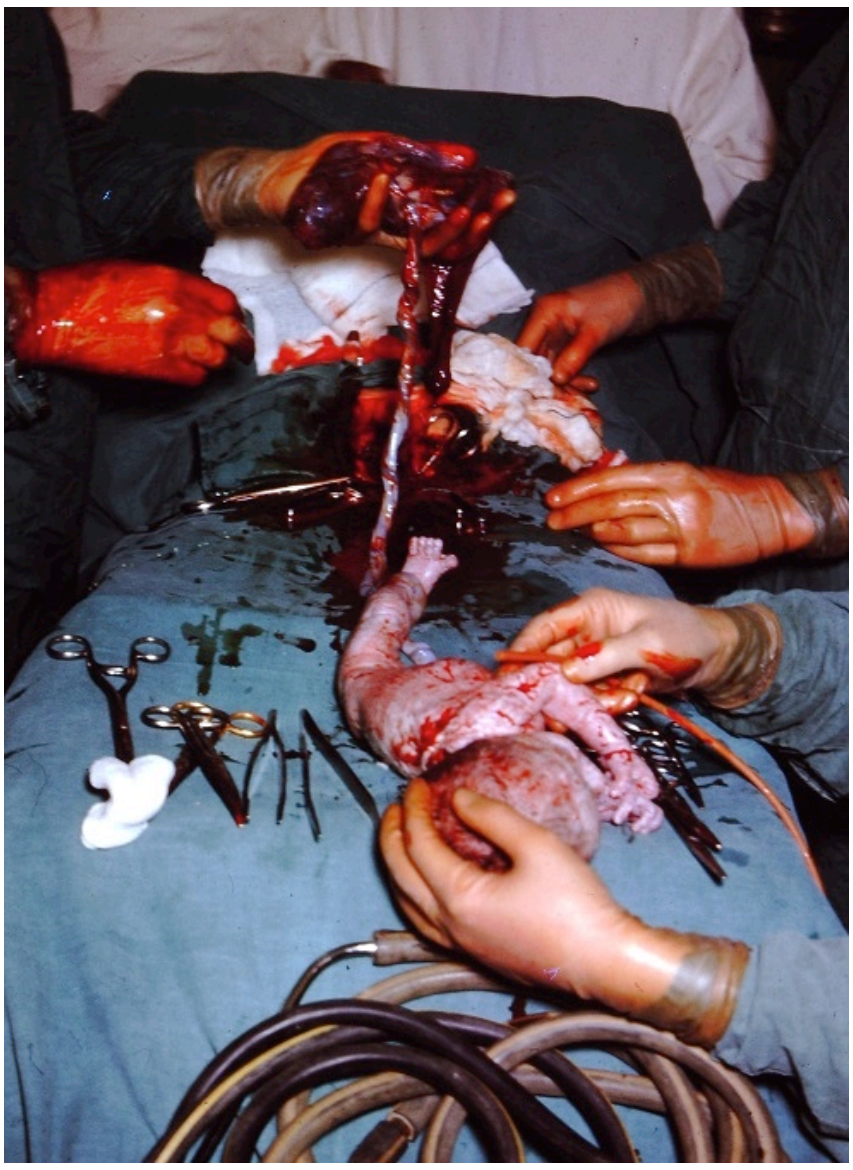

Fig 5.
Delivery of a premature infant at Caesarean section with placenta and unclamped umbilical cord.

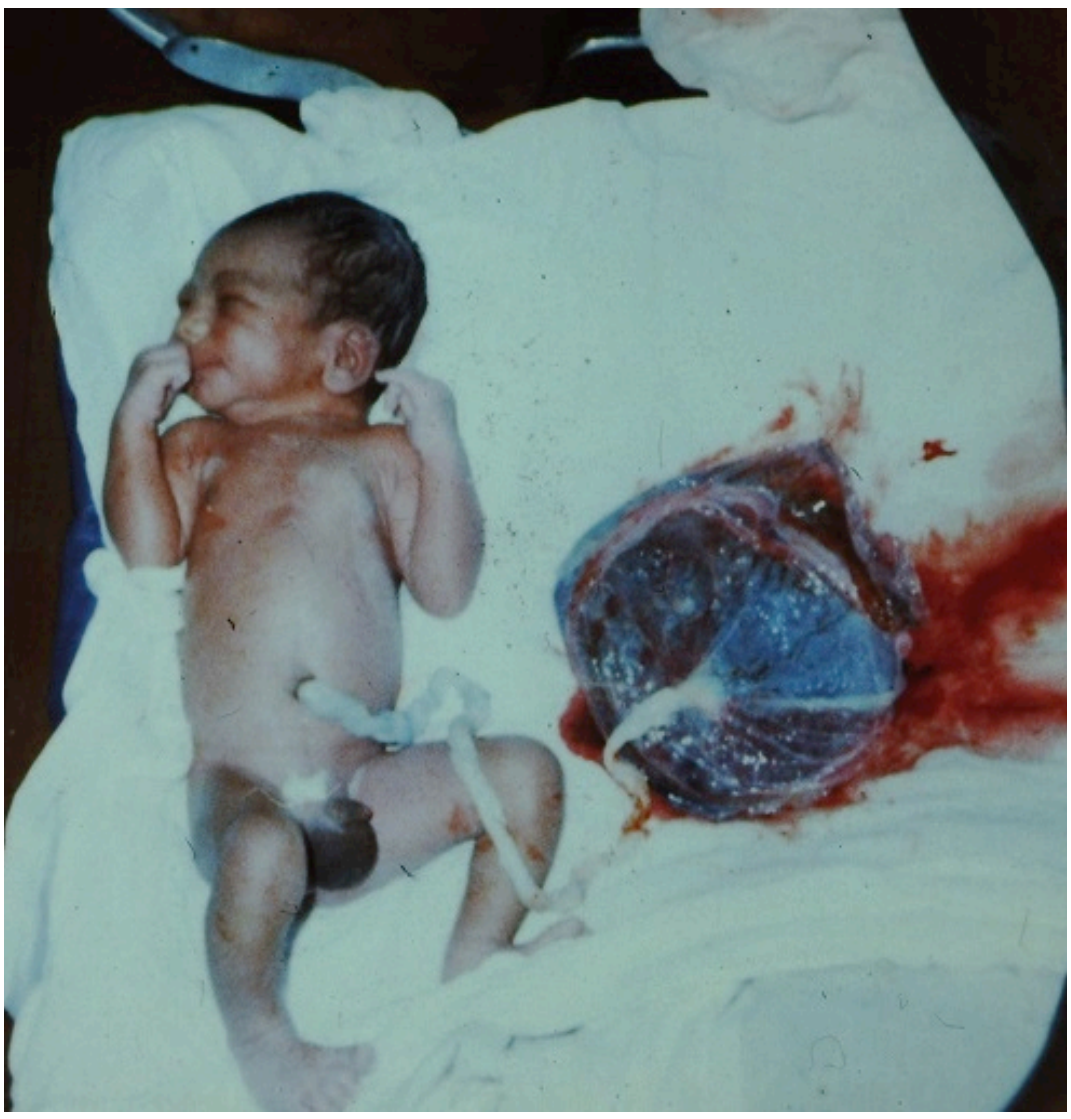

Fig 6.
Premature infant delivered by Caesarean section with unclamped umbilical cord and placenta lying at same level.

Towards the end of 1961 I developed a method of delivering the baby at Caesarean section with the umbilical cord and placenta intact. In brief, I scrubbed up and stood alongside the obstetrician. Once the baby's head was delivered, intravenous ergometrine was given into the mother's arm.

Fifty seconds later the uterus contracted and the obstetrician completed the delivery of the baby which was placed on a warm towel between the mother's legs (Fig 5).

At this time, my main function was to ensure the cord was not clamped or pulled. I also gently cleared the baby's upper airway. Then the whole unit – baby, cord and placenta was removed to the resuscitation area.

There the baby was placed in a gentle head-up position with the placenta and cord lying alongside at the same level (Fig 6).

Provided the infant was breathing with steadily improving air entry and that cord pulsation diminished and finally ceased, then the cord might be clamped and divided. If, on the other hand, there was no or inadequate respiratory effort, air entry was poor and the cord continued to pulsate, then gentle positive pressure respiration was instituted. I usually used a face mask and bag. The aim of this was not to blow up the lungs but to gently milk alveolar fluid into the pulmonary lymphatics and hence into the central venous pool. I must stress that anything but very gentle pressure is counterproductive and likely to cause rupture of the fluid filled alveolar ducts. Placing the infant in a head-up position, of course, aids the drainage of lymph from the lungs.

I shall always remember vividly the first occasion when I used this new technique. The late Mr. Leonard Deacon was the obstetrician and the mother, a doctor's wife, had very severe pre-eclampsia at thity-three weeks gestation. Up to that time, there had been no survivors during the previous two years in the hospital following Caesarean delivery before thirty-four weeks. To my delight, this baby, delivered with its placenta, showed absolutely no signs of respiratory distress and had an uneventful neonatal period. During the following year I attended all ten pre-term Caesarean deliveries using my technique with 100% survival.

In summary, I advocated avoidance where possible of maternal sedation, general anaesthesia and Caesarean delivery; avoidance of clamping the pulsating cord and of excessive placental transfusion; nursing the newborn infant in a head raised position with the placenta alongside and trying to ensure alveolar expansion using very gentle insufflations when necessary.

At this point, 1963, I moved from Birmingham to Bristol to research the influence of umbilical cord management on fetal adaptation to extrauterine life. For the next three years in a research post, I studied on the labour wards and in my small laboratory many aspects of the delivery process including the feto-placental transfusion during the second stage of labour, uterine activity during the third stage, the placental transfusion at delivery, and the postnatal plasma shift.

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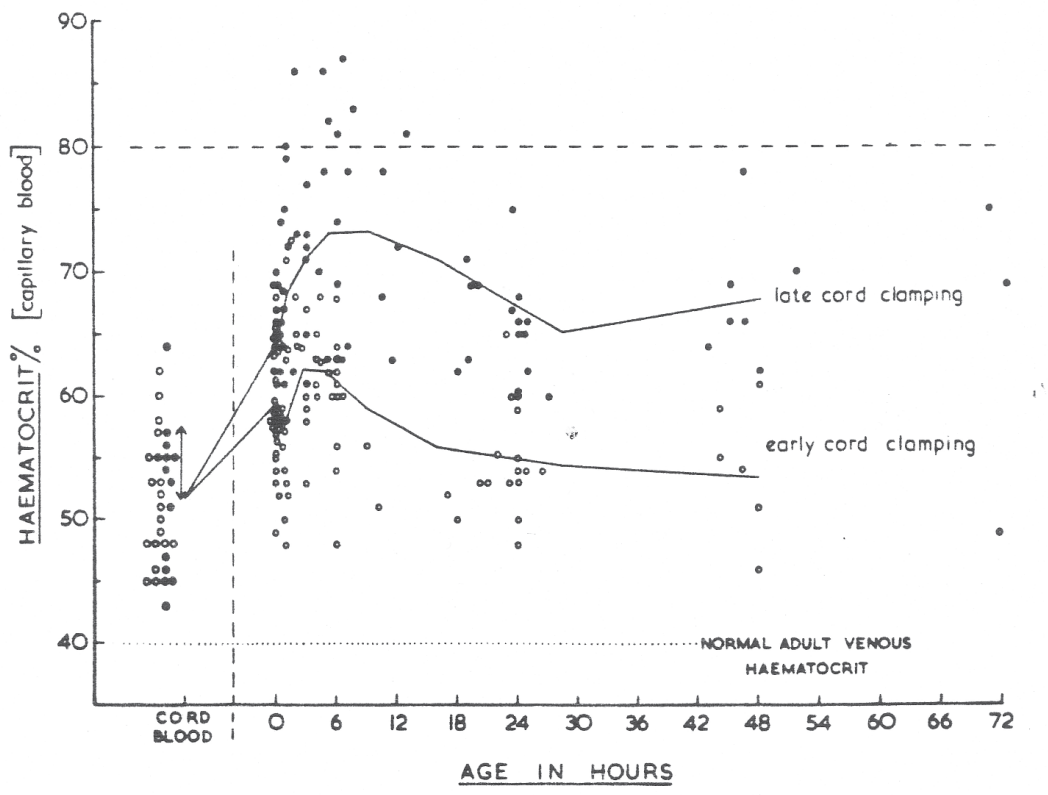

Fig 7.

The umbilical cord and heel capillary blood haematocrit values of 32 infants delivered normally at term, according to whether the umbilical cord was clamped at the instant of birth (<2 s) or after a delay of at least 3 min.

o = early clamping; ● = late clamping (Dunn, 1985[13])

I also conducted a randomised controlled trial on normal vaginally delivered term infants comparing immediate versus delayed cord clamping[(5-10)]. My research convinced me of the importance of an intact umbilical circulation during fetal adaptation to extrauterine pulmonary respiration for these reasons:

Avoidance of a premature rise in the baby's systemic blood pressure.

Provision of a safety valve for excessively raised central venous pressure.

Provision of the best opportunity for achieving circulatory normovolaemia.

Allow me to illustrate from those early studies the effect on the haematocrit of the baby of immediate versus delayed cord clamping (Fig 7). Indeed it was this study that alerted me to the dangers of neonatal polycythemia about which I spoke to the PVC in 1969[(1)].

These two babies from that study (Fig 8) were each photographed at 30 minutes following delivery. Both had had identical cord blood haematocrits at birth, namely 52%. The baby above with delayed cord clamping had received a large placental transfusion. Due to hypervolaemia and a massive postnatal plasma shift, his haematocrit rose to 82%. In contrast, the infant with immediate cord clamping (below), reveals the pallor due to hypovolaemia, hypotension and peripheral vasoconstriction.

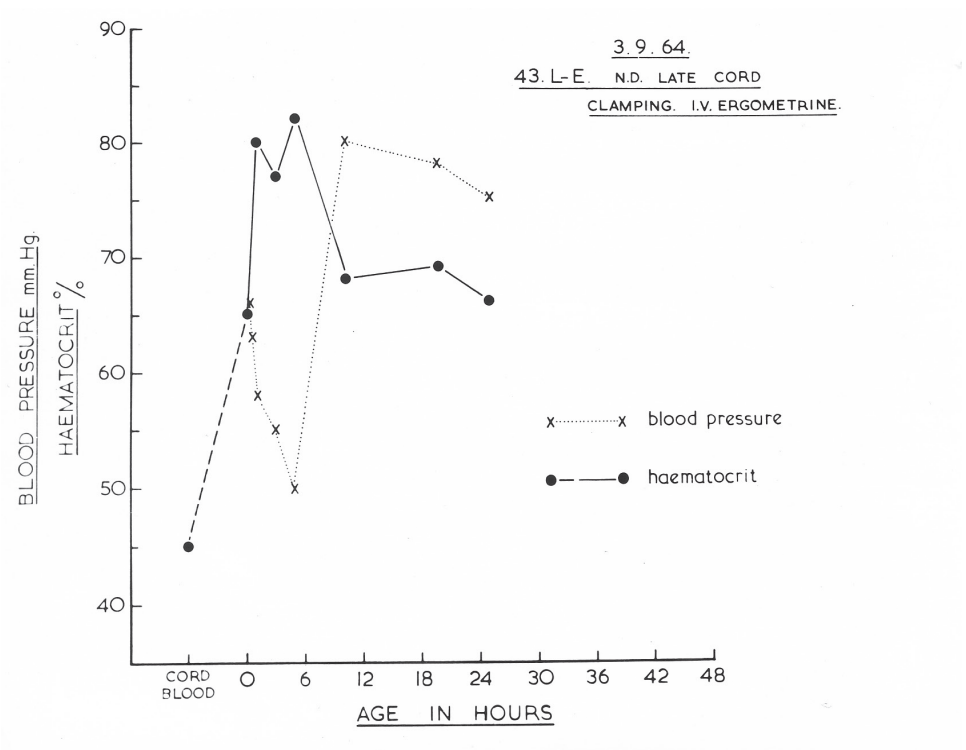

Fig 9. Serial haematocrit and blood pressure levels of the immediately cord clamped plethoric infant shown in Fig. 8.

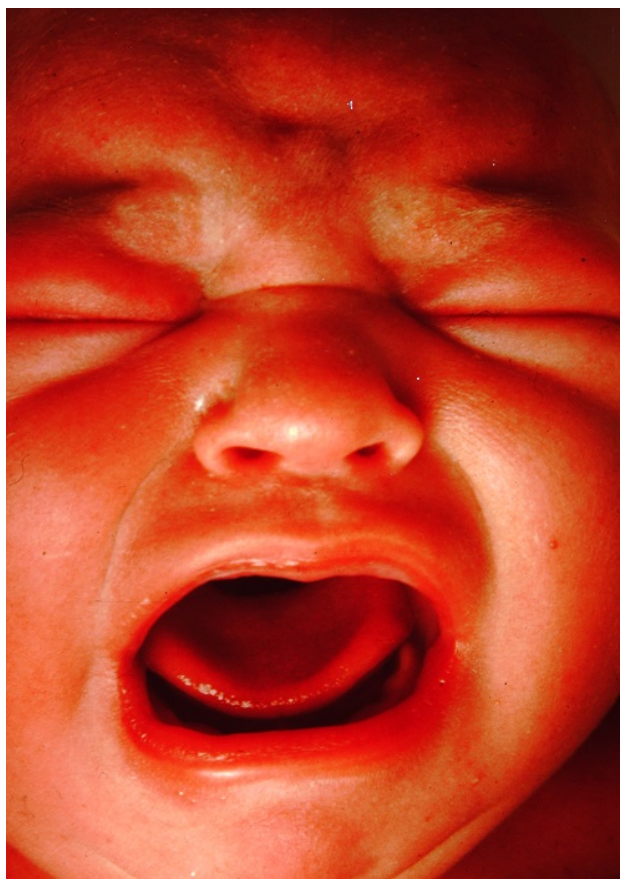

Figure 8a. Delayed clamping

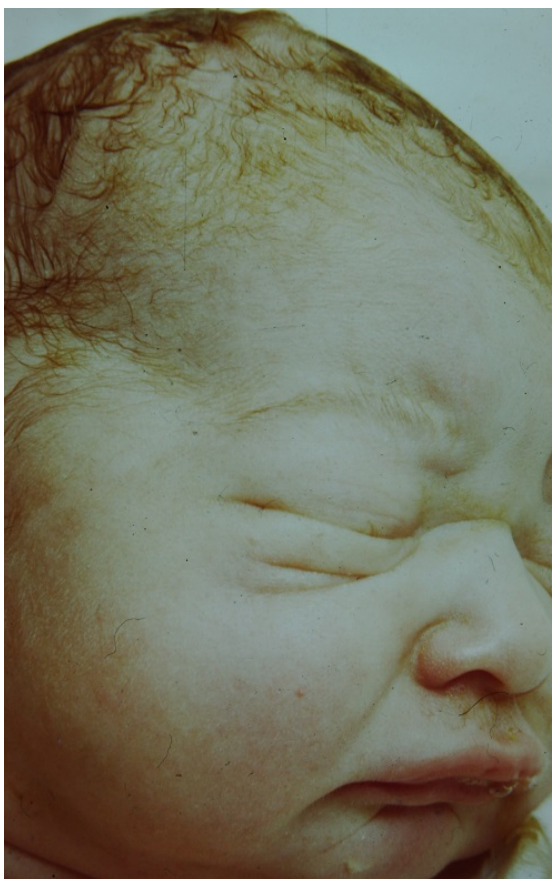

Fig 8b. Immediate clamping

Fig 8. Neonatal facies at the age of 30 minutes of two infants following (a) delayed and (b) immediate clamping of the umbilical cord. Both infants had identical cord blood haematocrits. 200ml of blood was later drained from the placenta of infant on the right.

Figure 9 shows serial haematocrits and blood pressures of the plethoric late clamped infant shown in Fig 8 during his first day of life. Note how the baby's blood pressure inversely falls as the haematocrit rises and visa-versa. This is a dangerous combination even for a term infant. These changes were caused by the hypervolaemia following the placental transfusion leading to a postnatal plasma shift from the circulation into the tissues.

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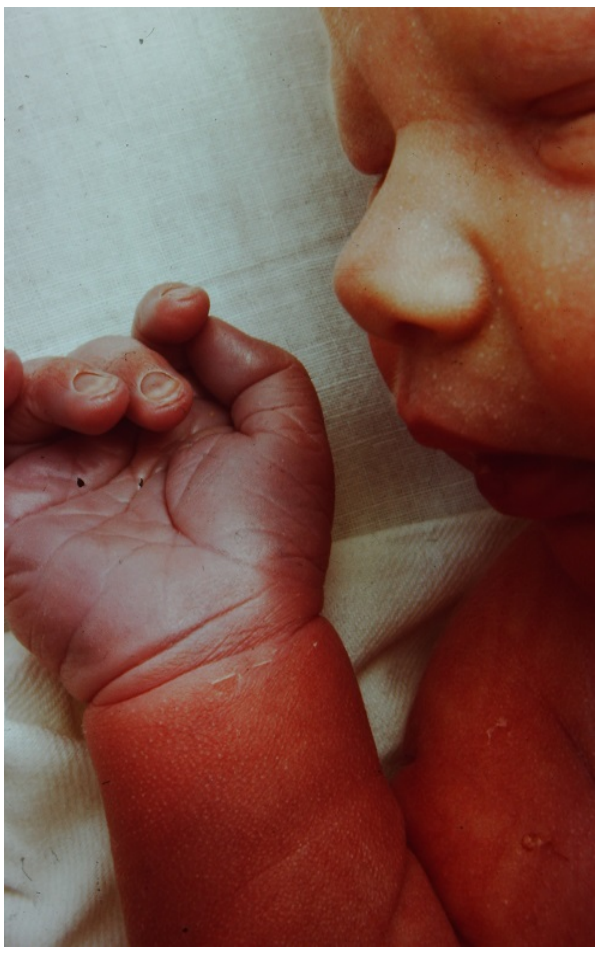

Fig 10a.
The face and hand of the plethoric infant (see Fig 8a) at the age of 3 hours. Note the cyanosis and oedema of the hand.

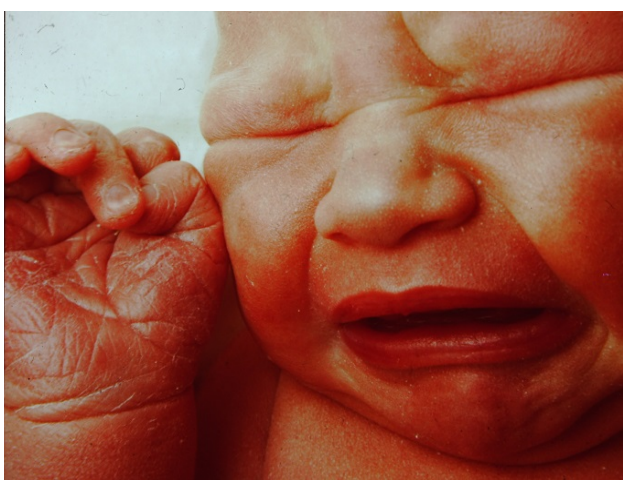

Fig 10b.
The same infant at the age of 10 hours. Note the absence of cyanosis and oedema from the hand.

In Fig 10a, you see that same baby at the age of three hours. Note the peripheral cyanosis and oedema of the hand due to polycythaemia and the plasma shift. Now compare this with the photo of the same baby just seven hours later (Fig 10b) after the plasma shift had been recovered into the circulation and the haematocrit had fallen from 82% to 68%, a fall of 24%. The cyanosis and oedema on the hand have disappeared.

Meanwhile, Gordon Lennon, professor of obstetrics and gynaecology in Bristol, had become convinced of my arguments. He often used to call me to manage the resuscitation of infants following premature Caesarean delivery and the success that I had had in the Midlands continued with very low morbidity and mortality.

In 1966 I spent time in north America and was away for two years. In 1969 I returned to clinical duties at Southmead Hospital. I found that there was a new professor of obstetrics, Geoffrey Dixon, and also that the use of my technique had lapsed. I discussed the matter with him and he challenged me to convince him of my ideas. So, through 1970 and 1971, I asked the obstetric staff to notify me of all planned preterm Caesarean sections in the department. There were in all thirty-seven preterm infants delivered in this way. I was singlehanded and very under-staffed but I was able to attend 35% of them and managed the delivery in the way I have described. The remaining 65% had their cords clamped at delivery in the usual orthodox way.

This, of course, was not a randomised controlled trial but in retrospect the two groups of babies were well matched for indication for Caesarean section, for sex, for birth weight and gestational age.

In terms of outcome, the incidence of RDS and neonatal mortality among those managed in the orthodox manner was some six times greater than among infants delivered with their placental circulations intact (Fig 11).

The outcome was even more dramatic among the infants delivered before thirty-five weeks gestation. Among the nine delivered conventionally with their cords clamped, six developed RDS and five died. In contrast, among the six delivered using my method, only one developed RDS and none died. You should recollect that this was before the days of steroid therapy, the use of surfactant and the availability of effective respiratory support. Professor Dixon stated that he was convinced and adopted my methodology for all preterm Caesarean deliveries in the department from then on.

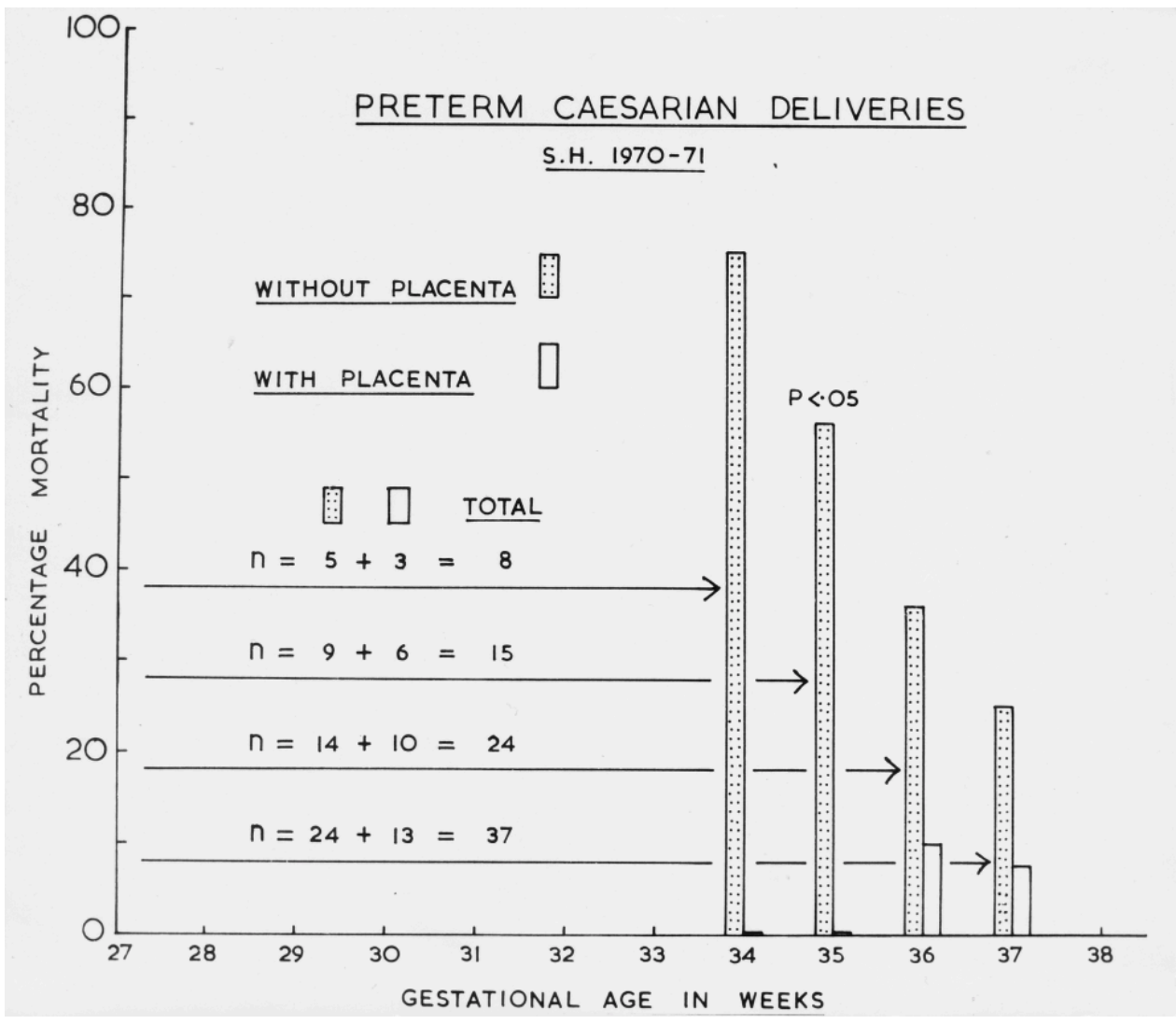

Fig 11.
The neonatal mortality among 37 premature infants delivered in the University Department, Southmead Hospital, 1970-71, according to whether they were or were not delivered with their placentas and intact umbilical circulations.

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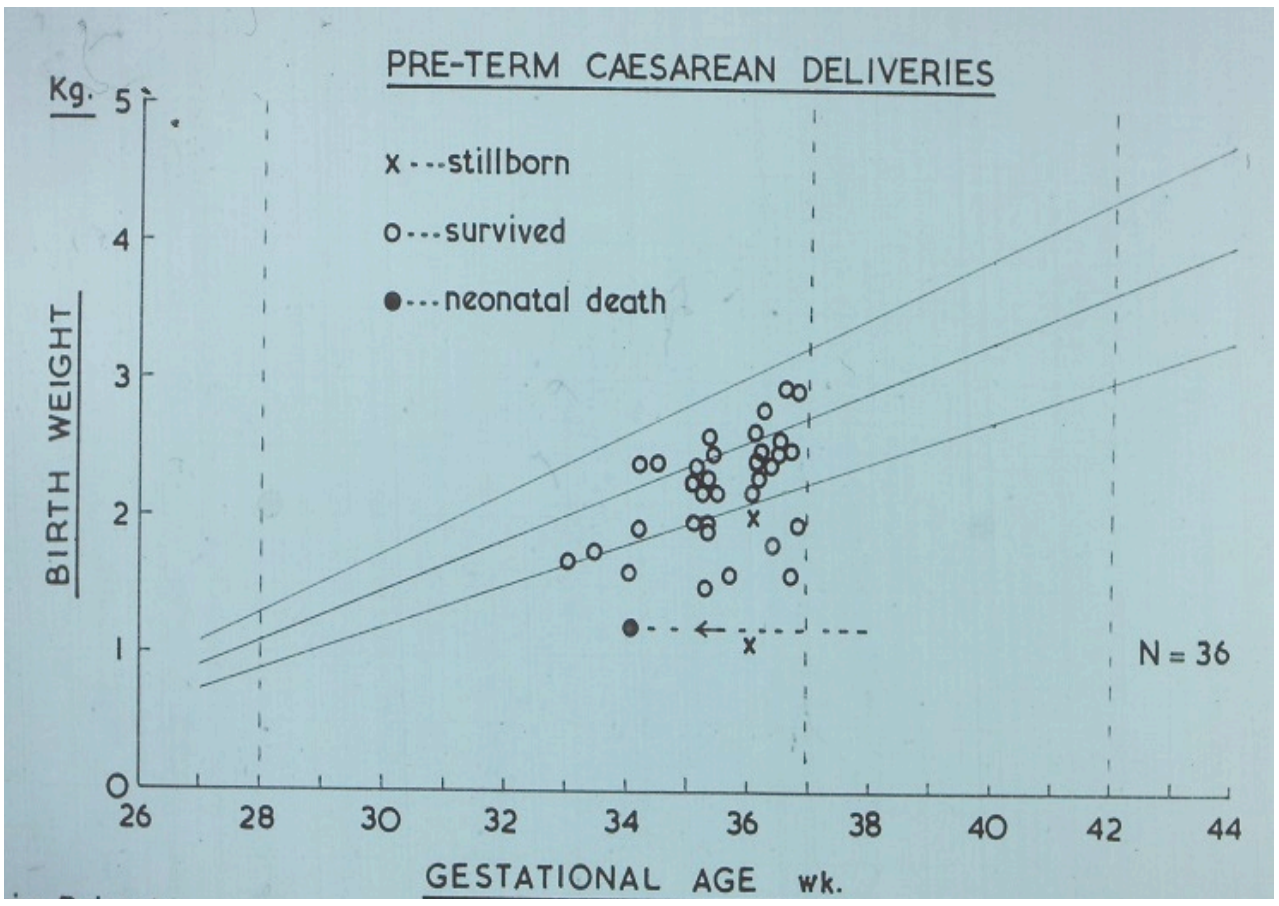

Fig 12.
Outcome of 36 premature infants delivered with their placentas at Caesarean section, 1972-73, in the University Department, Southmead Hospital.

Fig 12 shows the results for the next two years, 1972-3, in the University Department among thirty-six preterm Caesarean deliveries. Apart from two stillborn infants and one baby that was abnormal, there were no deaths among the remaining thirty-three babies. The following year we even had a Caesarean survivor using my technique at twenty-seven weeks gestation.

I reported my findings at the European Congress of Perinatal Medicine in Lausanne in 1972[11] and in many papers and lectures in the years since[12-21] (approx. n=150).

Sadly, my technique did not catch on.

As someone once wrote: '*No human force, not even fear, is stronger than habit.*'

I recognise that premature Caesarean delivery is, of course, always a tense time and my method required very close collaboration between obstetrician, anaesthetist and paediatrician. In addition, the paediatrician, usually an SHO, was often the most junior of the three. However, I understand that recently management of the umbilical cord has become a 'hot' neonatal topic. It was for that reason that I thought it worthwhile to recount the early studies made some fifty to sixty years ago.

Let me close with an abstract from a paper I gave at the World Congress of Obstetrics at Rio de Janeiro in 1988[17].

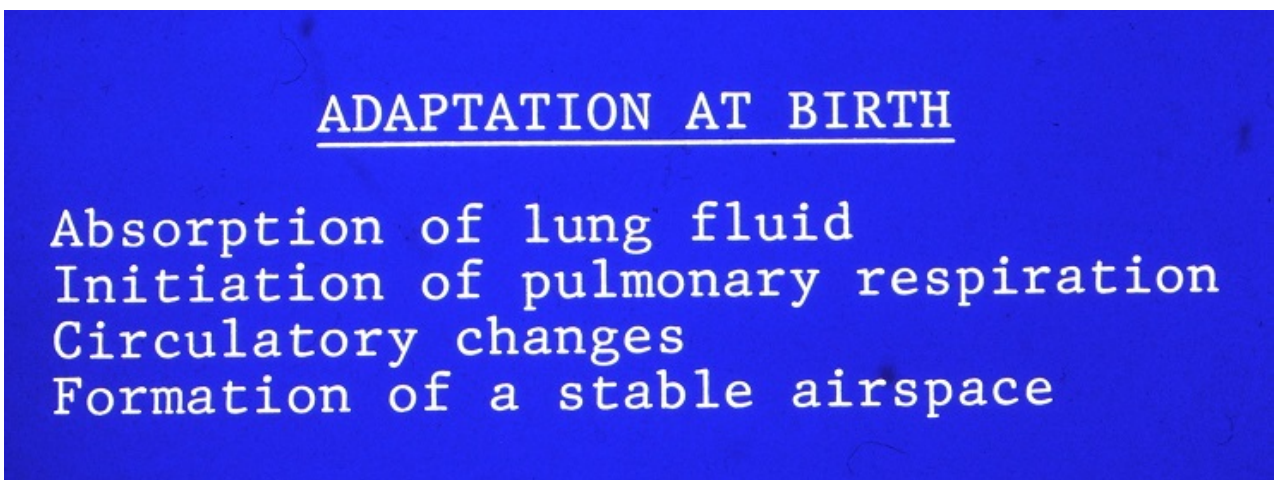

Fig 13.
Normal order of events during an infant's adaptation to extrauterine life.

"Adaptation to extrauterine life depends on the achievement of adequate alveolar ventilation, followed closely by a greatly increased pulmonary blood flow and other profound fetal-to-adult changes in the circulation.

The key to successful adaptation is the replacement with air of the lung fluid which fills the alveoli prior to delivery. Evacuation of this lung fluid is aided by an abrupt rise in blood catecholamines during labour and by thoracic compression during the second stage of vaginal delivery. After birth it is further achieved by pulmonary lymphatic drainage impelled by the 'milking' action of respiration.

The preterm infant is vulnerable to maladaptation and the respiratory distress syndrome because of various disadvantages in respect to the mechanics of respiration which impede the establishment of alveolar ventilation. His difficulties are increased by Caesarean delivery which may anaesthetise him and also deprive him of the vaginal squeeze. Elective section before labour further deprives him of the surge in blood catecholamines.

At delivery his cardio-respiratory haemodynamics may be seriously compromised by premature occlusion of the umbilical cord and also by resuscitation in the head-down position. An understanding of the pathophysiology of maladaptation at birth has led to a method of management that attempts to avoid these iatrogenic factors. This includes delivery of the preterm Caesarean section infant with umbilical circulation intact, and the use of gentle positive pressure ventilation with the infant in a head-up position and the placenta lying alongside the baby."

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