Mr. Wilfrid G. Mills (*Birmingham*): There are three clinical features associated with the syndrome of postmaturity, which do not appear to have received adequate notice. They are:

(1) The disappearance of the liquor amnii.

(2) The apparent failure of the baby to gain in weight which may actually progress to a loss of weight *in utero*.

(3) The increased tendency of the baby to develop cerebral hæmorrhage in labour.

(1) *The disappearance of the liquor amnii.*—I believe that when a pregnancy is prolonged beyond whatever is the optimum for that particular baby (that is to say beyond maturity) one of two things may happen. So long as the placenta has an adequate reserve of function, fœtal growth will continue unimpaired, and a large, heavy, hard-headed baby will eventually be born. But when placental sufficiency is exceeded, and the work of Sir Joseph Barcroft has shown that in animals postmaturity is always associated with progressive reduction in oxygen in the fœtal circulation, the child is at once in danger from asphyxia, which may become fatal during delivery. I have induced labour in many women by high rupture of the membranes for no other reason than that 42 weeks had elapsed since the last period, and when there has been an adequate amount of clear liquor, the baby has never been in danger. Occasionally, however, instead of liquor there has been some viscid mucus, often stained with meconium, and in these cases, the state of the child has caused anxiety.

I instance 2 examples. A patient was induced seventeen days over her calculated date having previously had 2 uneventful deliveries of 8 lb. babies. There was no liquor, but thick black meconium flowed out. Labour started at once and the fœtal heart was carefully observed. Its rate remained at 120 but beats were coupled and irregular. Labour was easy and lasted only two hours, when a 9 lb. baby was delivered in profound asphyxia from which it was resuscitated with difficulty. The other case was a primigravida in early labour four weeks beyond her time. The cervix was only 2 fingers dilated but the fœtal heart-rate varied between 92 and 116 and was very irregular. Diagnostic rupture of the membranes showed a viscid fluid stained with meconium, and at Cæsarean section the baby was found covered in meconium, moderately asphyxiated and weighing only 6 lb.

(2) My second contention, that *in extreme postmaturity fœtal growth may cease* and rarely a fœtus may actually lose weight *in utero*, is difficult to prove. The common signs of postmaturity on the baby after birth, the dry cracked skin and the long finger nails, are often associated with good skeletal length but astonishing paucity of subcutaneous fat. Support is given by Hosemann (1948) whose graphs correlating fœtal weight and maturity show an actual decrease in the average weights of babies when gestation is prolonged over three weeks. The most extreme example I have seen was a baby born at least three weeks and probably five weeks over its time. The placenta weighed 1 lb. 5 oz. but the child was only 4 lb. 13 oz. It was gravely asphyxiated at birth, and showed the typical dry cracked skin, long nails and wasted body. It was 19 in. (47·5 cm.) long, and the head had a biparietal diameter of 10 cm. measured on the fourteenth day when the child had just regained its birth weight.

(3) Regarding the *increased tendency to intracranial hæmorrhage* in cases of postmaturity, I was taught that it was a well-recognized occurrence and was due to the hard head which failed to mould in labour. I have seen ample confirmation of the fact, and I recognize that a difficult forceps extraction in a case of postmaturity is more likely to result in cerebral hæmorrhage than an equally difficult case when the child is at term. This bears out the figures published by Clayton (1941) in which he quotes a higher fœtal mortality in postmaturity but finds it due largely to cerebral hæmorrhage after a traumatic delivery. The ætiology might well prove to be the venous congestion which seems to be associated with all forms of asphyxia, and which presumably results in engorgement of the cerebral venous sinuses. This tendency to unexplained hæmorrhage was shown in a young primigravida who was induced when twenty-four days over her time. The fœtus seemed large, but her pelvis was ample and she objected to the induction on the grounds that her mother (whom she resembled closely) had had 10 children and was always three weeks over her time. On questioning the mother this fact was confirmed, but it also appeared that 3 of her babies had been stillborn. The patient was induced, and had a quick and easy labour, delivering a 7½lb. baby that died from cerebral hæmorrhage. As the placenta weighed exactly 2 lb., more than one-quarter of the fœtal weight, it would seem that here again was a baby that had failed to put on weight during its extra time in the uterus. The entire labour took only six and a half hours and an ample episiotomy was performed, so it is difficult to see how an extra hard head could have been a liability to the child.

Conclusion

In postmaturity the danger to the fœtus is primarily the increasing asphyxia that must affect every case of postmaturity if labour does not supervene. I do not anticipate serious trouble so long as there is clear liquor in the amniotic cavity. I will accept postmaturity even when the baby is still small, and I will incriminate postmaturity for death of a baby from intracranial hæmorrhage when delivery has not been unduly traumatic.

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The Oxygen Saturation of Umbilical Artery and Vein Blood at Birth, with Special Reference to Cord Obstruction [*Abridged*]

By C. A. B. Clemetson, M.A., B.M., B.Ch.

Fœtal distress in labour is largely due to fœtal anoxia. There are other causes, such as diabetes mellitus, fœtal trauma, congenital abnormalities, infections and toxins or drugs, but the majority of distressed fœtuses are simply suffering from oxygen-lack, which is loosely termed anoxia.

There is no accurate method of estimating human fœtal oxygen consumption, as this requires a knowledge of the volume of blood flowing in the umbilical circuit per minute, as well as the umbilical artery-vein oxygen difference.

It is, however, possible to obtain some information concerning fœtal oxygenation by analysis of umbilical cord blood samples at birth.

Eastman (1932) has described a type of fœtal anoxia in which the oxygen saturation of the umbilical vein blood is reduced to a very low level. This is true anoxæmic anoxia, where the blood flowing towards the fœtus is poorly oxygenated. I have seen this type of fœtal anoxia on several occasions, but it is another common type of fœtal anoxia occurring in cord obstruction that I wish to describe.

In a study of the oxygen and carbon dioxide content of human umbilical artery and vein blood at birth, the umbilical cord was digitally occluded at each end to arrest the blood flow before the first gasp; Spencer Wells forceps were then applied on either side of the fingers, and a length of cord was isolated by cutting between the clamps.

Umbilical artery and vein blood samples were collected anaerobically over mercury from this isolated segment of cord using a modification of the anaerobic blood sampler described by Austin *et al.* (1922).

Duplicate estimations of the oxygen and carbon dioxide contents of the blood samples collected in this way, were carried out in the Van Slyke and Neill (1924) manometric apparatus, and a specimen of umbilical vein blood was used for oxygen capacity determinations.

In a series of normal vertex vaginal deliveries of non-toxæmic mothers breathing ½% trilene in air, or air alone, there were some babies born with the umbilical cord coiled round the neck, and some where this complication did not arise.

A comparison of the results of the cord blood analyses in these two groups, enables us to assess the effects of "cord round the neck".

Fig. 1. While the umbilical vein oxygen saturation was materially the same in the two groups, 63% and 62%, the umbilical artery oxygen saturation fell from 31% in the normal cases to 6% where the cord was round the neck.

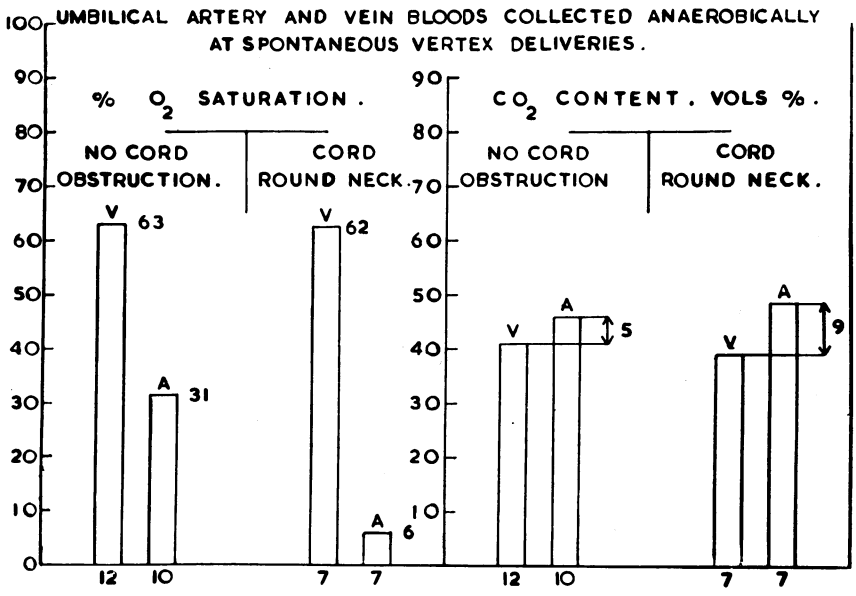

FIG. 1.—Showing the effect of cord obstruction by "cord round the neck" on the oxygen saturation and carbon dioxide content of umbilical artery and vein blood. V = umbilical vein. A = umbilical artery. The number of cases comprising each mean value is shown below the blocks.

The artery-vein carbon dioxide difference was increased from 5 vol.% in the normal, to 9 vol.% where the cord was round the neck; this was due to a rise in the umbilical artery and a fall in the umbilical vein carbon dioxide content. Clearly, both the oxygen and the carbon dioxide results indicate that the fœtal umbilical circuit was slowed by cord obstruction when the cord was round the neck.

No attempt was made to decide whether the cord was tightly or loosely coiled, although this may be important; the cases were simply classed as "cord round the neck" and "no cord obstruction".

We do not know the fœtal oxygen consumption, but we do know something about the oxygen tension of the fœtal tissues; their oxygen tension must be lower than that of the blood which has given up oxygen to them. Since umbilical artery blood consists mainly of that returning from the

upper half of the fœtus, its oxygen saturation is an index of the oxygen tension of the tissues in that part of the fœtus. The umbilical artery oxygen saturation is decreased when the cord is round the neck, so one must conclude that this complication decreases the oxygen tension of the upper half of the fœtus.

The lower half of the fœtus will have an even lower oxygen tension, as it uses blue blood exactly like that in the umbilical artery as arterial blood. This does not mean that all babies born with the cord coiled round the neck will suffer from asphyxia neonatorum. Most of them do not, for they escape in time, but any prolongation of the period of cord obstruction will lead to asphyxia, and may even be fatal.

Harrar and Buchman (1951) attributed 14 out of 38 unanticipated fœtal deaths in the perineal stage of labour to "cord round the neck" alone, so one must not let familiarity with this common complication breed contempt.

So far, I have only mentioned "anoxæmic anoxia" where the umbilical vein blood is poorly oxygenated, and "cord obstruction anoxia" where the flow of normally oxygenated blood is impeded. "Anæmic anoxia" and "histotoxic anoxia" may also occur in the fœtus, but these are less common. In anæmic anoxia there will, of course, be a low oxygen capacity, so that blood with a normal oxygen tension does not carry sufficient oxygen.

In severe histotoxic anoxia one would expect a rise in the umbilical artery oxygen saturation and a fall in the artery-vein oxygen difference, owing to decreased fœtal oxygen consumption. I have not seen this type of anoxia, probably because narcotics were always used sparingly in labour.

It is thus evident that a study of the umbilical vein oxygen content alone gives a very incomplete picture of fœtal oxygenation. The umbilical artery oxygen content and the oxygen capacity of the fœtal blood must also be known before a diagnosis may be reached. Many factors may interfere with fœtal oxygenation, and while our knowledge is far from complete, it is possible to list certain factors according to the type of anoxia they cause (Table I).

TABLE I.—CAUSES OF FŒTAL ANOXIA

Anoxæmic anoxia	*Cord obstruction anoxia*
1. Retro-placental hæmorrhage	1. Cord round fœtus
2. Maternal anoxia	2. Prolapse of cord
3. Severe maternal anæmia	3. True knot of cord
4. Maternal blood pressure drop	4. Breech delivery
5. Cæsarean section factor	5. Short cord
Anæmic anoxia	*Histotoxic anoxia*
1. Erythroblastosis fœtalis	1. Narcotics
2. Fœtal hæmorrhage	

Other factors to be considered

Vascular anastomosis of homozygous twins. Postmaturity.
Placental inadequacy. Uterine hypertonus. Uterine retraction.
Placenta prævia. Placental infarction. Pre-eclampsia.

Factors such as postmaturity, long labour, uterine hypertonus, placenta prævia, placental infarction and toxæmia of late pregnancy are incompletely understood, but there is evidence (Clemetson and Churchman, 1953) that pre-eclamptic toxæmia simulates the cord obstruction type of anoxia: there may be slowing of the umbilical circuit in this condition due to spasm of the fœtal placental veins; this has indeed been described by Bartholomew *et al.* (1951) as the mechanism of placental infarction.

Fig. 2 shows the results of analysis of umbilical artery and vein blood from three cases which illustrate "normal fœtal oxygenation", "cord obstruction anoxia", and "anoxæmic anoxia".

The first two cases were spontaneous vertex vaginal deliveries, with and without the cord being coiled round the fœtal neck at birth; the third was delivered by Cæsarean section (under cyclopropane and 80% oxygen anæsthesia), performed for hæmorrhage and fœtal distress immediately after a high rupture of the membranes; I have labelled this one placental detachment, for there was little doubt that this had occurred.

At present we rely almost entirely on changes in the rate or rhythm of the fœtal heart sounds for diagnosis of fœtal distress. Intermittent auscultation is a useful guide, but it is not altogether reliable.

Meconium staining of the liquor amnii is even less reliable as a sign of fœtal distress; the funic souffle of cord obstruction, and the tests suggested for diagnosing cord round the neck may also be deceptive, so it is with great hopes that we watch the development of methods for recording the fœtal heart.

The radio-active isotope studies of Flexner and Gelhorn (1942) showed that a volume of water equal to that of the amniotic fluid flows into and out of the amniotic sac of the guinea-pig every hour; this led one to wonder whether the oxygen tension of this fluid would be in equilibrium with that of the fœtal or uterine tissues producing it. However, using the method described by Winkler (1924) for estimating dissolved oxygen in water, I find that amniotic fluid, like urine, contains no free oxygen.

The first principle of treating fœtal distress is, of course, to deliver the fœtus as soon as possible. In theory this is quite simple and straightforward, but owing to the difficulty of assessing the severity of fœtal distress, it is often difficult to know when to interfere.

Whatever method of delivery is adopted, oxygen should be administered to the mother by face mask until the child is born.

Dieckmann and Kramer (1944) took repeated blood samples from a loop of umbilical cord delivered through a small wound at Cæsarean section under local anæsthesia and found that the oxygen content of fœtal blood was appreciably raised by the administration of oxygen to the mother.

Of course, complete cord obstruction or extensive placental separation will prevent oxygen from reaching the fœtus, but as cord obstruction is usually intermittent, and as placental separation is usually partial, there is good reason to administer oxygen in the hope that some will reach the fœtus.

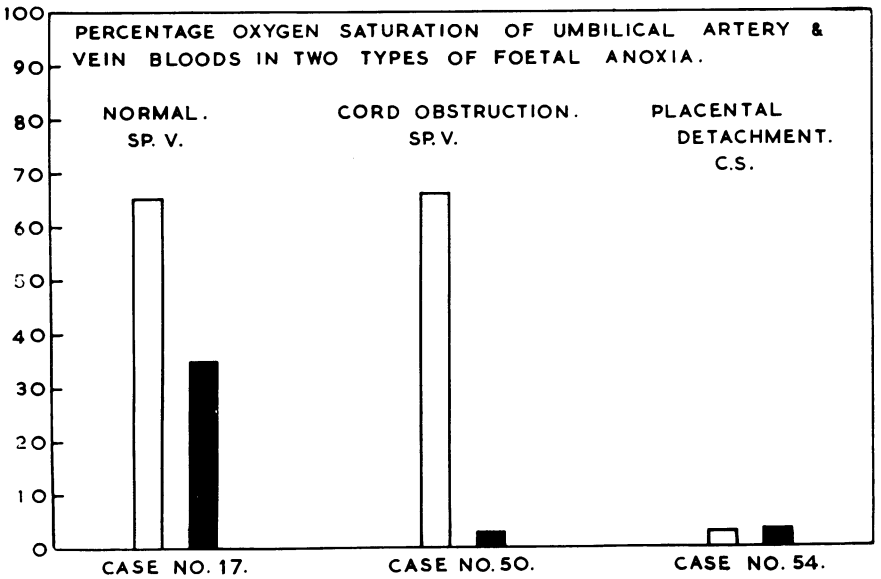

FIG. 2.—Showing the oxygen saturation of umbilical vein and artery blood samples from three cases. The white blocks represent the oxygen saturation of the umbilical vein blood, and the black blocks that of the umbilical artery blood. SP. V. = spontaneous vertex delivery. C.S. = Cæsarean section.

Clinical impressions concerning the value of giving oxygen to the mother in fœtal distress differ widely, but there is no justification for withholding a form of treatment which may do good, and can do no harm.

I wish to thank the Board of Governors of University College Hospital for a grant which made this work possible. Part of this study was carried out while I was a Nichols Fellow of the Royal Society of Medicine.

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Dr. N. R. Butler (University College Hospital): *Prognosis of Fœtal Distress.*

There are several serious dangers, short of fœtal death, which may result from prolonged fœtal distress. Clement Smith (1950) has aptly stated that oxygen-lack may not only stop the machine but also may wreck the mechanism. Even the normal fœtus suffers from oxygen-deprivation in the later months of pregnancy, compensated partly by an increase in the number of red blood cells, which also contain fœtal hæmoglobin with better oxygen affinity than adult hæmoglobin. The fœtal central nervous system is also relatively resistant to hypoxia, which is just as well because the normal developing fœtus probably faces and surmounts a series of asphyxial crises *in utero* which go unrecognized clinically. The fœtal animal can

survive for up to thirty minutes *in utero* with the cord clamped, though Windle *et al.* (1944) have shown that severe fœtal oxygen-deprivation may later give rise to impaired motor and adaptive functions, anyway in the guinea-pig. It is not known how long a human fœtus can survive complete oxygen-deprivation, but there have been well-authenticated cases of survival following fifteen minutes complete anoxia immediately after birth, when anaerobic oxidation of glucose probably provides the central nervous system with sufficient nutrition to ensure survival, if not complete recovery of function.

In the more acute forms of fœtal distress such as that following complete placental separation, rapid fœtal death occurs from sudden asphyxia with the well-recognized post-mortem features of ecchymoses or petechial hæmorrhages on the surface of organs which are usually congested, but may be bloodless if death occurs at once or in the event of cord pressure or occlusion. In the past it was thought that if the fœtus could be delivered rapidly before intra-uterine death, no permanent damage would result, but there is now known to be a short-term danger both to initiation of post-natal respiration and to lung expansion, as well as a potential longer term threat to the development of the central nervous system and mentality. Regarding the lungs, most authorities are agreed that rhythmical though intermittent respiratory movements occur in the normal human fœtus and that there is a circulation of liquor amnii as far down as the alveoli which are thus kept patent following their earlier recanalization. This problem has been clarified by the well-controlled experiments of Davis and Potter (1946) using injections of Thorotrast into the human amniotic cavity before therapeutic abortions and also preceding Cæsarean section at term, proving that the dye became concentrated in the alveoli after absorption of liquor amnii. In the event of fœtal distress, these shallow respiratory movements are replaced by stronger gasps with aspiration into the alveoli of larger quantities of liquor, together with vernix, epithelial debris and even meconium. These may cause an interstitial foreign body reaction difficult to distinguish macroscopically from an intra-uterine bacterial pneumonia. They may also cause cohesion of the alveolar walls with difficulty in initiating respiration, or may even form in some cases a basis for the post-natal formation of a serious so-called "hyaline" or "vernix" membrane. We have analysed 275 cases of fœtal distress occurring in 2,490 live births at University College Hospital in 1950 and 1951 (Table I) and

TABLE I.—CONTRASTING EVIDENCE OF FŒTAL DISTRESS AND POST-NATAL RESPIRATORY COMPLICATIONS IN 2,490 DELIVERIES AT UNIVERSITY COLLEGE HOSPITAL IN 1950 AND 1951

		Post-natal respiratory complication	No post-natal respiratory complication	Total
Fœtal distress	Forceps or Cæsarean section	33	94	127
	Normal delivery	28	120	148
No fœtal distress	Forceps or Cæsarean section	49	190	239
	Normal delivery	82	1,894	1,976
	Total	192	2,298	2,490

have found that 61 (22%) of those babies with fœtal distress also experienced respiratory difficulties at or after birth, either severe enough to require resuscitation or resulting in a delay in establishing regular respiration for a period of five minutes or more after birth. Though less than one in four babies showing fœtal distress actually experienced delay in establishing post-natal respiration, this rate of delayed onset of breathing was nearly three times that occurring in all the live births in the hospital during the same period, emphasizing the need for expert care of such infants immediately after delivery. Of the 275 fœtal distress cases, 148 were delivered spontaneously and the remaining 127 underwent obstetric intervention, by forceps (86) or Cæsarean section (41). It should be mentioned that fœtal distress was not always the primary indication for obstetric intervention in the latter group. Comparing the two groups, the number requiring post-natal resuscitation in the normal delivery group was 28 (19%), as opposed to 33 (26%) in the group delivered by forceps or Cæsarean section. This difference though not enough to be statistically significant may be partly due to a weighting of severely distressed fœtuses in the group undergoing obstetric intervention. It appears that neither the possible adverse effect of general anæsthesia upon an already distressed fœtus nor the fœtal risk inherent in the forceps delivery or Cæsarean section materially worsen the post-natal prognosis in these cases, while early obstetric intervention does undoubtedly reduce the stillbirth rate.

Finally, does perinatal asphyxia carry a serious risk to the ultimate mental or physical development of the child? There are still two schools of thought on this matter. Retrospective studies of children referred for behaviour disorders and abnormalities in the C.N.S. contain a disproportionately high number with a history of birth anoxia. Of the three well-controlled studies so far undertaken Darke (1944) found significant mental retardation in a small series of 19 cases, but Campbell *et al.* (1950) and Keith and Norval (1950) found that no permanent damage occurred in the survivors from perinatal asphyxia. A detailed longitudinal study is needed and with the increasingly accurate methods of diagnosis which have been discussed, babies with fœtal distress should now be given equal prominence with those showing post-natal respiratory delay in studies assessing the long-term prognosis of perinatal asphyxia.

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