ABNORMAL POSITIONS OF THE UMBILICAL CORD

A discussion of some perinatal hazards

IAN A. DONALDSON, M.B., B.S., F.R.C.S., M.R.C.O.G., D.A.

*Consultant Obstetrician & Gynaecologist
City of London Maternity Hospital, London, N.A.*

ABNORMAL positions of the umbilical cord are difficult to define anatomically. The normal or usual positions are not demonstrable at present. At Caesarean Section the cord is usually in the upper segment and only seen in the lower segment in the minority of cases; however, that is the impression given by memory and not by a statistical survey.

Functionally an abnormal position could be defined as one in which the cord is liable to compression, torsion, stretching or other mechanical alteration which might interfere with the free passage of blood through the vessels. Like a diver's air-pipe there may be certain positions which constitute a hazard. On the one hand the hazard may remain potential but on the other hand it may become dangerous or even fatal. The flow of blood may be impeded by compression of the vessels or by spasm in their walls, and is normally or abnormally altered by the pumps and their associated blood vessels at either end. It is clear that here is an enormous subject for research.

Some Abnormal Positions of the Umbilical Cord

(1) Prolapse of the cord.

This is the grossest, most obvious and well-documented abnormal position.

Frank prolapse of the cord is not always so lethal to the baby. The cases occurring from 1957 to 1962 at the City of London Maternity Hospital are shown in Table 1.

In this small series 68.4% of the babies survived. If the babies dead before delivery was commenced together with the one neonatal death of a premature baby are subtracted from the series, then the baby survival rate would be 86.6%. All the babies delivered by Caesarean Section survived. Two babies died during delivery by forceps.

Prolapse of the cord may be discovered for the first time on examination prior to a forceps delivery, the baby crying at birth, with no previously recorded alteration of the foetal heart.

The duration of the prolapse is not always known.

In 1958 a prolapsed cord occurred in a gravida 3 in early labour at home. Pulsation in the cord was reported as absent and the foetal heart not heard. On admission the foetal heart was heard and pulsation was present. The baby was delivered by Caesarean Section and cried at birth some 3 hours after the declared prolapse.

As has long been known, it is the degree of pressure on the cord which matters and determines the prognosis, and the cervix which controls the treatment. Possibly the best, though impracticable, emergency treatment would be to suspend the patient vertically by the ankles and so allow the extruding contents to fall back into the uterus, which so resembles a purse string bag being carried strangely, mouth downwards, even when the contents are most valuable. The acceptable postural methods all invoke the aid of gravity to relieve cord pressure. Something else may be achieved simultaneously:— the uterine contractions may become temporarily weaker. This can be seen in normal labour after favourable progress in the second stage when in the dorsal position, the head may regress when the patient is put in the left lateral position. There then may be a delay before the head reaches its previous station. The delay may seem interminable if there had been some foetal bradycardia. An alternative emergency measure frequently employed for prolapse of the cord does not evoke the life-saving gravitational effects but consists of pushing the presenting part away from the cervix to relieve the pressure on the cord. However, the hand in the vagina may suffer from cramp, partly due to the increased force of the contractions produced by the manipulation. This method could make matters worse for the baby and should be replaced whenever and as soon as possible by effective postural methods. In certain circumstances it may be the only method conveniently available.

An example occurred in 1964. A safe gravida 2 was booked for home confinement. In early labour the cord prolapsed. Pressure on the cord was relieved by the midwife by continuous vaginal manipulation. As the patient was being put in the ambulance a message

TABLE 1

CITY OF LONDON MATERNITY HOSPITAL

CASES OF PROLAPSED CORD AND CORD PRESENTATION PUBLISHED IN REGISTRARS' REPORT.

	1957	1958	1959	1960	1961	1962	Total
NO. OF CASES	2	3	3	1	3	7	19
Booked	1	Not	3	Not	2	3	9
Emergency	1	Noted	0	Noted	1	4	6
INCIDENCE	0.14%	0.20%	0.20%	—	0.18%	0.4%	
TREATMENT							
Normal delivery	1	0	0	0	0	1	2
Breech extraction	0	0	1	1	0	2	4
Forceps delivery	1	1	1	0	0	1	4
Caesarean Section	0	2	1	0	3	3	9
Stillbirth	2	1	1	0	0	1	5
Stillbirth of these 5 those dead before treatment	2	0	0	0	0	1	3
Live birth	0	2	2	0	3	6	13
Neonatal death	0	0	0	1	0	0	1
				2 lbs. 9 oz.			
Perinatal deaths				6 = 31.5%			
Of these, possible avoidable perinatal deaths				2 = 10.5%			
Surviving babies				13 = 68.4%			

was received at hospital that a hand was prolapsing. On admission the patient was put in the Trendelenburg position whilst preparations for abdominal delivery were completed. The cervix was barely half dilated. The baby was delivered by Caesarean Section and cried at birth.

In all cases of prolapse of the cord it must be remembered that this is the last stage of a journey and we have no idea when the journey commenced nor how many incomplete journeys occur.

(2) *Forelying cord and occult prolapse of the cord.*

Sometimes a loop of cord may lie beside the head. My attention was drawn to this fact dramatically at Oxford in 1940. I was giving an anaesthetic for a forceps delivery. The experienced general practitioner had known the patient all her life. This child born after an orthodox, leisurely and easy extraction was dead. The feeling was conveyed that the inexperienced anaesthetist was somehow to blame. On examining the cord closely I noticed two areas of flattening fairly close together. Illustrations in DeLee's book (1940) of a forelying cord and of occult prolapse, where the cord is higher, gave the answer. About a week later I was performing a forceps delivery and discovered an occult prolapse and was careful in the application of the blades and delivered the baby in a shorter time. It cried at birth.

In my mind I formulated the dictum that it was safer for the cord to be round the neck rather than dangling beside the baby's head.

An occult prolapse of the cord is sometimes seen at Caesarean Section, either lying lateral to the baby's head or less commonly posterior to

the baby's head. It may be an incidental finding with no prior alteration of the foetal heart or be suspected as in the following case in 1964.

The patient was a primigravida, 20 years old. She was admitted on 25th April, 1964 at the forty-first week for induction of labour because of oedema. Vague uterine contractions commenced 22 hours after artificial rupture of the hindwaters. A Syntocinon infusion was given. After ten and a half hours despite strong contractions, the cervix was thick, only admitting one finger, the blood pressure had been rising and was 160/190 mm hg, and the foetal heart which had been varying between 144 and 150 a minute developed a bradycardia of 110 after contractions and only slowly recovered. The head was in the left occipitolateral position and was felt easily per abdomen; pressing the head into the pelvis evoked bradycardia. The baby was delivered by Caesarean Section, there was a little liquor; there was a loop of cord confined between the left side of the head and the lower uterine segment. The baby boy cried at birth and weighed six pounds seven ounces. (2.91 kg.). The cord was 20 inches (50.8 cm.) long. If the cord had been found frankly prolapsed there would be no debate about the mode of delivery. As it was, it could be argued whether baby would have been healthy if expectancy had been adopted, but as the definitive treatment had been taken the question could not be answered scientifically.

How often in obstetrics would we like a problem all over again, treated differently to evaluate scientifically the results! An occult prolapse of the cord could be more common than generally accepted, it could kill a baby during labour without leaving a trace of evidence at vaginal delivery—unless the whole

cord was carefully examined possibly microscopically. Could any of the unexplained intra-partum foetal deaths be due to unsuspected disturbance of cord function rather than to placental insufficiency? Experienced labour ward staff at the City of London Maternity Hospital support this view from cases seen over the years, though no statistical records have been kept. In some cases a Caesarean Section has rescued a baby whose cord was felt high up beside the head in the first stage of labour, in other cases it was believed that a baby died from nipping of an undiagnosed loop of cord beside the head.

This occurred on the 28th February, 1965 when the foetal heart stopped during a protracted trial of forceps in a young primigravida of 18, a procedure which is not always in the best interest of the baby and was the subject of correspondence (Donaldson, 1963a). When this dead baby was delivered a loop of cord was beside the head just above the ear.

(3) *Nuchal cord.*

The umbilical cord encircling the neck is such an obvious and common condition at birth that it has long been described. It may occur early in pregnancy, Vayssire and Dor (1938) described an abortus with the cord 4 times round the neck. I have seen the cord encircling the neck of a 14-week foetus at hysterotomy. Cases tend to be reported where the cord has caused an abnormality or tragedy: Frigyesi (1942) quoted 9 cases of intra-uterine death and emphasised that torsion with relative shortening of the cord could alter the progress of labour. He stressed the need for earlier recognition and preventive treatment. He cited 13 cases treated successfully by Caesarean Section, the cord being round the neck once or twice. Hamilton (1947) described the ways in which a cord round the neck could cause the death of the foetus. The recognised diagnostic signs were mentioned. In addition, pressing the head into the pelvis produced marked bradycardia. This sign led to prophylactic Caesarean Section on three patients described. The following case demonstrated this sign, preparations were made for Caesarean Section which fortunately proved unnecessary.

This patient was a primigravida, 19 years old. At the 30th and the 35th week she had developed excessive weight gain with oedema and this was controlled by short courses of chlorothiazide added to dietetic advice. At the 39th week the oedema had recurred, the patient was admitted on 28th April, 1964. Fourteen hours after artificial rupture of the hindwaters, the foetal heart was commencing to slow and become irregular, within an hour the foetal heart was 92 to 95 per minute between contractions and 80 per minute during contractions with considerable irregularity. Oxygen was administered to the mother

with no improvement, arrangements were made to perform Caesarean Section, the patient appeared to be fully dilated on being moved to the theatre 20 minutes later. The foetal heart was then dropping to 60 per minute and was grossly irregular. Immediate forceps delivery produced a girl who cried at birth and weighed seven pounds two ounces (3.23 kg.). The umbilical cord measured 24 inches (61 cm.) and was coiled tightly round the neck twice and was looped round the right leg.

Walker (1956) drew attention to the important question of breech presentation being caused by the cord round the neck and to the obvious danger of attempted external cephalic version in such a situation. He recorded 6 cases. As was well taught by the older obstetricians persistent bradycardia after external version demanded reversal of the process because of probable cord entanglement.

The following case demonstrates how cord entanglement could occur during external cephalic version.

This patient, a doctor's wife, had her first baby in 1962. The pregnancy was normal. The labour lasted seven hours. The second pregnancy occurred in 1964. At 29 weeks a breech presentation persisted, external cephalic version was easily performed. At 31 weeks the breech presentation had recurred, external version was followed by transient foetal bradycardia. The breech presentation recurred, attempted version a week later was followed by foetal bradycardia which was not relieved until the baby was returned to breech presentation. At 35 and again at 36 weeks version was attempted whilst listening to the foetal heart; bradycardia commenced before the baby was a transverse lie. I assumed that there was umbilical cord entanglement, probably a nuchal cord. On balance I decided to deliver by Caesarean Section. I found the cord free but coiled anterior to the baby's arms. Obviously turning the baby the only way she would go caused the arms or even the head to become entangled in the cord. The baby was a pound heavier than her sister and has made even better initial progress. This raises an entirely different subject, worthy of study, the observation of intelligent mothers that there may be a link between emotional development and facility of delivery.

Death of babies in successive pregnancies associated with multiple coiling of the cord round the neck was described by Vovor and Sudre (1960), the seventh baby was delivered by Caesarean Section and again the cord was round the neck. Reiss (1958) described a case where the cord was five times round the neck and quoted the literature, where up to nine coils round the neck had been recorded.

Baltzer (1958) and Temperini (1959) both stressed the hazard to the baby of the cord round the neck: the latter pointed out that it was of no hindrance in breech deliveries. A rare occurrence in which the cord of the second twin was coiled round the neck of the first twin was described by Bender and Prebble (1961) and by Irvine (1963).

There have been statistical surveys of large series of cases to determine the effect on the baby of the umbilical cord coiling round the neck. Kan Pun Shui and Eastman (1957) concluded that it was not related to stillbirth nor to neonatal death, but Crawford (1962) felt it was associated with increased foetal distress and neonatal depression. Dippel (1964) surveyed a series of cord entanglements and felt that these were rarely responsible for foetal or neonatal death, but there was more foetal heart irregularity, meconium staining and need for resuscitation.

These statistical surveys and their conclusions emphasise day-to-day clinical observations. The coil or coils of cord around the neck, which occur in 20 to 30% of cases, may be of less importance than the remainder of the cord. Does the coiling make the cord relatively short or cause it to take unusual pathways, thus inviting tensions or pressure? Are the loops around the neck so slack that the cord can develop an occult prolapse? A recent case in 1965 demonstrates this unseen hazard.

This patient lost her first baby in 1963 when she was 36 years old, in late pregnancy increased weight gain and slightly raised blood pressure occurred. She was admitted at term. Labour was induced at the forty-first week. During a weak Syntocinon infusion the foetal heart stopped. The liquor became meconium stained. Delivery was completed by forceps because of delay in the second stage. The stillborn female baby weighed seven pounds one ounce (3.2 kg.). The cord was coiled twice round the neck and its total length was 22 inches (55.9 cm.). During this second pregnancy excessive weight gain occurred at the thirty-fifth week, this was temporarily controlled by a short course of chlorothiazide. There was no rise in blood pressure. Caesarean Section was performed at the thirty-ninth week. There was an undiagnosed anterior placenta praevia, extending half way down the lower uterine segment, the liquor was stained with meconium, the cord was loosely coiled twice round the neck and the slack portion of one loop was lying beside the face on the left side of the pelvis. No alterations of the foetal heart had been noticed. The child, a male weighing seven pounds fifteen ounces (3.6 mg.) responded rapidly after removal of meconium from the respiratory passages.

Clearly, abdominal delivery would have saved the first baby and in retrospect it would have been justified.

What the practising clinician wants to know is the degree of risk to the individual baby. Is the position of the cord one of hazard and if so of major or minor degree? We want to avoid delivering a dead or damaged baby due to the cord round the neck and from obstetric histories we know that this has happened repeatedly and the mothers so informed.

(4) *Coiling round the body or limbs.*

This occurs not infrequently and probably in

the majority of instances has no deleterious effect on the baby. The true incidence *in utero* is unknown and at present statistics cannot be produced. However, in the literature are examples where the foetus or baby has been killed or was at risk. Luraschi (1957) described and illustrated a dead 16-weeks-old foetus where there were tight loops of cord round the right thigh. Robaczynski (1963) described two cases of defects of the foetal extremities due to winding of the umbilical cord around them. Durand (1953) published photographs of a girl who had had a groove at the waist since birth, due to a coil of the umbilical cord. She also had a right hydronephrosis which it was surmised might have been caused by the constriction.

The cord was found to be coiled three times round the wrist in the following case, seen in 1964.

This primigravida was 26 years old, the pregnancy was completely normal until the thirty eighth week when episodes of foetal bradycardia—120 to 115 per minute—with irregularity were noted especially after palpating the baby. She was admitted for observation. On vaginal examination the head was in the pelvis and no cord was palpable. On the sixth day after admission the foetal heart became irregular, the rate varying between 100 and 136 per minute, this did not last very long. Three days before term on the 10th March 1964, I decided to induce labour. The foetal heart was irregular at the beginning of the procedure. The forewaters were ruptured, clear liquor obtained, no cord was felt and the foetal heart was regular at the end of the examination. Six hours later, labour was established and the foetal heart had again become irregular; there were periods when it was only 72 per minute between contractions. Oxygen was administered. The cervix only admitted two fingers. I diagnosed intermittent cord compression and thought there was a nuchal cord or occult prolapse. At the Caesarean Section I was surprised to find no cord in the lower uterine segment. On delivering the baby I found the cord twisted tightly three times round the left wrist. The baby boy required resuscitation, regular respirations were established in three minutes. He weighed five pounds six ounces (2.44 kg.). Unfortunately, the length of the cord was not recorded, but the impression obtained at the operation was that foetal movements could have affected the circulation in the cord but desire to deliver the baby expeditiously prevented a leisurely detailed survey of the intrauterine conditions.

I have already described a baby whose cord was coiled around the neck and around the right leg. In the following case history the baby had its cord coiled round the neck and round the right arm.

This patient had her first baby when she was 30 years old in 1962. The baby was born at the forty-second week. She weighed eight pounds four ounces (3.74 kg.). The mother was given oxygen for hours because of foetal distress and was told after delivery that she would have lost the baby if labour had lasted longer. The second pregnancy in 1964 was normal

apart from a persistent occipito-posterior position and a raised blood pressure near term which might have been related to her increasing anxiety about labour. I induced labour at term and after 17 hours during which there was no abnormality of the foetal heart tones, delivered a baby girl weighing six pounds twelve ounces (3.6 kg.). The umbilical cord was loosely coiled round the neck and coiled round the right arm; the baby cried at birth and the maternal anxiety vanished.

Mechanically one would expect a cord loosely coiled around an extremity to uncoil during the process of delivery. The umbilical cord coiled loosely around the body or extremities may not be in an abnormal but in the normal position. This may be the means whereby it is retained in the more roomy upper segment, instead of descending to the more dangerous lower segment. The means whereby the growth in length of the cord is controlled is completely unknown. Could coiling round the embryo early in pregnancy somehow encourage growth in length? Doubtless there is some controlling formula or factor yet to be discovered.

(5) *Short cord*

The umbilical cord may be relatively short because it has coiled round some part of the baby; a tragic example occurred in 1960.

A primigravida of 20 had a persistent breech presentation. Repeated attempts at external cephalic version in late pregnancy were unsuccessful. Labour was induced at 39 weeks. The first stage was very slow and abdominal delivery was discussed; however, after 36 hours the cervix became fully dilated, the buttocks approached the vulva, the foetal heart dropped suddenly from around 140 per minute to 96 per minute and continued to fall to 56 per minute. Breech extraction was commenced. The cord was taut and not pulsating when the umbilicus appeared; there was no loop of cord available to pull down. The cord was over the right shoulder but not round the neck and it had to be divided to permit delivery of the head. The dead child weighed seven pounds ten ounces (3.34 kg.) and the length of the cord was 14½ inches (36.8 cm.).

Anatomical shortness of the cord may cause it to be in an abnormal position *in utero*. It may well function normally during pregnancy but be the cause of trouble or even tragedy during labour. This possibility threatened the baby of a safe gravida two booked for home confinement in 1964.

This patient had a first baby in hospital, surgical induction was performed 11 days after term and the delivery was normal. Next year when she was 23 years old she commenced her second pregnancy and was booked for home confinement. Again she went over her dates and was admitted 15 days after term. Buccal oxytocic tablets were used for induction. One hour later the foetal heart was reported as fading, the rate was regular around 140 per minute, then the tones improved. Six hours later the foetal heart was varying between 160 per minute and 130 per minute, there were irregular contractions and the cervix

admitted one finger. The membranes were ruptured. Clear liquor was obtained and no cord felt. Fifty hours after the onset of uterine contractions, the cervix only admitted a finger despite strong contractions during a syntocinon infusion, the head remained in the brim, though there was no disproportion, the foetal heart continued to vary between 120 per minute and 160 per minute. There had been no recorded foetal bradycardia and no gross irregularity. It was decided to deliver abdominally. The head was not engaged and the cord appeared to be short. The baby cried at birth. The cord was 9 inches (22.8 cm.) long.

It was felt that the relatively short cord was responsible for the altered pattern of the labour and might well have damaged or killed the baby if expectant treatment had been continued, hoping for a vaginal delivery. However, intervention destroyed the evidence which could have proved or disproved that opinion.

Diagnosis of a Hazardous Position of the Umbilical Cord

The best method would be to visualise the umbilical cord *in utero* and see if it was in a position which could constitute a hazard to the foetus; that is not, at present, practicable. This is not the same as saying that the problem is insoluble. A technique awaits discovery and in addition from working models the varying and possible cord positions and lengths could be worked out and further information obtained. As it is, the diagnosis depends on direct observation in the case of a nuchal or prolapsing cord and some forelying cords and on indirect evidence for the majority of hazardous positions of the umbilical cord. Early diagnosis, so essential in obstetrical abnormalities if results are to be improved, is neither easy nor certain. The evidence has to be weighed carefully. It can be considered under three headings which overlap.

Evidence from manipulation

During pregnancy persistent foetal bradycardia or irregularity of the foetal heart following attempted or completed external cephalic version or pushing the presenting part into the pelvis may be indicative of distortion of the cord rather than due merely to pressure on the head as one used to think. Hamilton (1947) and Walker (1956) stressed these observations in relation to the nuchal cord. During labour the sequence of events may be so accelerated that a tragedy may have occurred before a warning diagnosis has been made; Maxwell (1958) wrote on this question and he pointed out the value of the Trendelenburg position as an aid to diagnosis where cord compression is suspected as the cause of foetal bradycardia. He also

stressed the need for more detailed pathological examinations where there was a tragedy unexplained by clinical observations.

Study of the foetal heart

Intermittent auscultation of the foetal heart is invaluable but it is intermittent and depends on the observer. Hon (1958) demonstrated the human error in auscultation in counting the foetal heart rate. The results obtained by 15 observers counting recorded pulses of known rate were shown graphically and the variations seen. The value of a reliable electronic method of recording the foetal heart is obvious. The first reported foetal electrocardiograph was taken in 1906. Hon described his apparatus and the results on 80 patients of recordings throughout labour. Transient bradycardia during contractions became more marked as the cervix dilated. It was considered to be due to head compression, as bradycardia was repeated as the head passed through the vaginal outlet, could be produced by pressing on the head between contractions, but was absent in breech presentation during contractions. The mechanism of production of the bradycardia, whether an alteration in the brain stem or in the haemodynamics, was not determined. Hon (1959) used the electronic equipment to distinguish physiological from pathological bradycardia. He pointed out that Von Winckel in 1893 had drawn attention to the poor foetal outcome following foetal bradycardia. Yet an undamaged baby could be born after periods of bradycardia. Studying in detail 7 out of 500 tracings, certain features emerged. Physiological bradycardia commenced 30 seconds from the onset of the contractions, was short lived and V-shaped on the tracings. Bradycardia due to cord compression commenced within 10 to 15 seconds from the onset of the contractions, the duration was proportional to the degree of compression and was U-shaped on the tracings. Experimental work on lambs subjected to cord compression showed that the initial bradycardia was due to vascular reflexes responding to acute hypoxia and prolonged bradycardia was related to persistent anoxia. Bradycardia associated with frequent strong contractions developed 25 to 30 seconds from the onset of the contraction and it usually took 50 seconds before the heart rate recovered. Further work in lambs demonstrated that slowly developing hypoxia was followed by tachycardia then bradycardia, acute hypoxia was followed by bradycardia. Hon, Bradfield and Hess (1961) and Mendez-Bauer, Poseiro, Arrellano-Hernandez, Zambrana and

Caldeyro-barcia (1963) studied the vagal factor in foetal bradycardia. Atropine given to the mother relieved mild foetal bradycardia considered to be due to cord compression. These studies suggested that some foetal bradycardia might be a compensatory mechanism and not necessarily evil, though it indicated a compromised foetal environment. These studies of the foetal heart by the newer foetal electrocardiograph are giving more precise information and earlier evidence of changes in the foetal heart. They also largely confirm earlier routine clinical observation and the research work based on older equipment.

In 1946, I studied 32 cases of foetal distress, as judged by changes in the foetal heart, for which medical aid was summoned at the North Middlesex Hospital. This study formed the basis of an unpublished commentary on intrapartum foetal distress; the conclusions then from studying the cases and the literature were that progressive foetal bradycardia was the most reliable sign of foetal distress but did not necessarily indicate the degree of distress. Bartholomew (1925) and Freed (1927) had come to the same conclusions. Foetal tachycardia was a fallible sign of foetal distress; studies by King (1940) and later by Lund (1943) using continuous recording of the heart tones had given similar conclusions.

Observation of the liquor

The significance of meconium staining of the liquor may be difficult to interpret from the point of view of the baby's welfare. Leff (1932) felt that intestinal peristalsis was produced by venous congestion of the bowel, part of a general overloading of the foetal circulation; Hon, Bradfield and Hess (1961) suggested that it was due to vagal stimulation rather a sign of hypoxia. Leonard (1962) surveyed the whole subject and studied 100 cases, concluding that such babies were at risk and would require resuscitation, but only a small fraction would be stillborn.

Meconium staining of the liquor occurred in varying degree in 20 of the 32 cases studied in 1946. It may be more an indication of an earlier than the present condition of the baby, which is better assessed by behaviour of the foetal heart.

Differential Diagnosis

Causes other than abnormal positions of the umbilical cord have to be considered when the sole sign is alteration of the foetal heart. Bradycardia, irregularity and fading were all observed in the cases described in this paper;

in 1 or 2 of the other cases the foetal heart stopped between periods of observation with no record about its final behaviour pattern. The whole subject is far from clear and one or many factors may be operative. Looking back this is obvious in the series of 32 cases studied in 1946. Nine patients were under 20 years old, 16 were over 30. Pre-eclamptic toxæmia was present in 9. Fifteen had labours lasting over 24 hours and 9 of these over 48 hours. Fourteen were estimated to be a week or more overdue. Twenty had premature rupture of the membranes but only 8 for more than 24 hours. In 12 patients, 3 or more of these factors were present but were not directly proportional to the condition of the baby at birth. There were 2 stillbirths and 2 neonatal deaths, that is 12.5% perinatal deaths, and 9 other babies were shocked at birth requiring considerable resuscitation, that is 28.1% perinatal morbidity in the series.

The factors or conditions which may cause or possibly predispose to alterations of the foetal heart observed clinically can be placed into 3 broad, perhaps interacting, groups; the general condition of the mother, the utero-placental factors, the foetal factors.

General condition of the mother

This subject will doubtless receive further study. Increasing maternal age is accepted as a predisposing factor. Maternal hypertension particularly when part of the pre-eclamptic toxæmia syndrome is associated with lowered oxygen content in the umbilical artery as reported by Clemetson and Churchman (1953) and decreased uterine blood flow as shown by various studies quoted by Hon and Wohlegemuth (1961). These last workers studied the effect of maternal exercise on the foetal heart; the production or accentuation of tachycardia, bradycardia or irregularity occurred in 6 patients and all had abnormalities in labour.

Maternal hypotension may give rise to foetal bradycardia as noted by Hon and Wohlegemuth (1961). A dramatic fall in blood pressure, the unexpected side effect of an analgesic or sedative drug, can cause fading or even disappearance of the foetal heart and was the subject of correspondence. (Donaldson, 1963b).

Utero-placental factors

Uterine contractions when too frequent may produce foetal bradycardia. This has been known for many years. Leff (1932) maintained that when the uterus contracts blood is squeezed

from the placenta into the baby's circulation, increasing the foetal blood volume and pressure. Marey's law was invoked to explain the bradycardia. After rupture of the membranes and particularly in the second stage, overloading of the foetal circulation could be more marked and even heart failure result. Hon (1959) suggested a possible working hypothesis about the effect of uterine contractions on the utero-placental circulation. Briefly, he suggested that the myometrial contraction closes the maternal vein draining an intervillous space before closing the supplying artery. There results poor intervillous blood flow and so oxygen lack to the foetus. The hypothesis is demonstrated by a series of diagrams. Misrahy, Beran, Spradley and Garwood (1960) studied the question by inserting electrodes into the foetal brain in animals. They felt that the foetal brain might have less physiological reserve than the adult. They found that spastic uterine contractions with poor relaxation between were potentially the most harmful and that giving oxygen to the mother did not help. Lund (1940) had discussed this problem, suggested giving oxygen, expediting delivery or giving a general anaesthetic. Earlier Waters and Harris (1931) had observed the improvement for the baby in some cases of foetal distress by giving oxygen.

Uterine contractions produced by an oxytocic infusion can cause alterations in the foetal heart. Hess and Hon (1960) monitored the foetal heart electronically during such infusions. Too rapid contractions produced tachycardia before or after bradycardia, tetanic contractions produced profound bradycardia while in a small number of cases there was no alteration even with a rapid induction and in 2 cases of pre-eclamptic toxæmia bradycardia occurred even with infrequent and mild contractions. The foetal heart pattern as influenced by an oxytocin infusion may reveal or be an index of foetal reserve. In other words, the conditions inside the incubator may be adequate in the relative static state of pregnancy but once the walls move in the dynamic state of labour a latent and unsuspected weakness inimical to the baby may be revealed. In life it could be advantageous to have warnings of latent lethal situations.

Abnormalities of the placenta can cause tachycardia, bradycardia, fading and cessation of the foetal heart. An attached placenta may develop placental insufficiency because of degenerative changes and so make foetal

distress more likely to occur. Walker (1959) demonstrated that prolonged gestation—particularly when associated with difficult labour, advancing maternal age and induced labour—increased the incidence of foetal distress.

Premature detachment of the placenta due to or possibly causing intra-uterine haemorrhage whether in early or late pregnancy or during labour is a potential threat or even a death-blow to the foetus. Richardson (1936) maintained that the foetal heart rate was related to the area and duration of the detachment of the placenta, tachycardia preceding bradycardia. Examples of premature detachment of the placenta are not uncommon. The factors influencing premature detachment of the placenta require further investigation and research. The effect of premature detachment on the baby and the foetal heart is seen in various obstetrical situations.

In 1946 a calamitous fall in and cessation of the foetal heart was seen during a too tardy breech delivery; the dead baby seemed to have acquired the placenta as a hat.

In 1955 an elderly primigravida had been admitted at the thirty-eighth week with a slight antepartum haemorrhage. The head was engaged. She was allowed home in a few days. The show or mild antepartum haemorrhage recurred at term and on arrival at hospital the foetal heart seemed to be fading away into the distance, bradycardia and irregularity was occurring and was followed by cessation of all foetal heart sounds within 20 minutes. Dark clots and fresh blood followed the dead baby at delivery.

In some cases of the dangerous triad where either foetal tachycardia or bradycardia has occurred I have observed partial separation of the placenta at Caesarean Section but the condition of the baby at birth was satisfactory.

The baby may be rescued in time even from a Couvelaire uterus as reported by Donaldson and Bismillah (1963); on the other hand attempted rescue may be too late as occurred in a gravida 2 in 1961: the foetal heart was 86 per minute on admission and fell to 46 per minute a quarter of an hour later. It quickly improved with a rapid blood transfusion and became 120 per minute. Following an unusual stormy induction of anaesthesia, it fell to 100 per minute, the baby delivered by Caesarean Section was shocked at birth, developed neurological signs and died when 24 hours old.

Foetal factors

There may be biochemical anomalies, quite apart from those induced by anoxia, still to be discovered. Possibly the altered maternal metabolism in a prolonged labour may induce a biochemical illness in the baby and so produce perinatal morbidity. For the present 2 organs of the foetus must be considered.

The foetal heart may be diseased and have

an abnormal rhythm. Redman (1958) suggested that in 50 per cent of cases of foetal bradycardia there may be a cardiac lesion. Hon and Huang (1962) made an electronic evaluation of premature and missed beats in the foetal heart; in 25 cases of foetal cardiac arrhythmias, 22 had disappeared at birth.

The foetal brain will be subject to increasing study. Leff (1932) maintained that cerebral haemorrhage in the newborn can cause asphyxia but that asphyxia does not produce cerebral haemorrhage. He also stated that pressure on the foetal head did not explain the bradycardia. Hon (1958), quoted earlier, observed that pressure on the head prior to the cervix being dilated to a diameter of 6 centimetres produced only slight bradycardia but after that marked bradycardia. He could not determine the mechanism of the production of the bradycardia. During or after external cephalic version there is commonly foetal bradycardia which may be profound and the foetal heart may not be heard for a short while. With other babies external version, which was discontinued because of bradycardia, can be safely performed another time. In other cases no alteration for the foetal heart rate occurs with external cephalic version. This must surely mean that the degree or position of the pressure on the baby's head must vary or another cause be operative; this could be the position of the cord as was described earlier in one of the patients. Foetal bradycardia during the second stage of labour I had tacitly assumed to be due to pressure on the baby's head: if so, with similar pressures, which are difficult to measure, this should occur invariably. During the second stage of labour some of the utero-placental factors may be operative, particularly in the protracted second stage seen in former years when the baby died in the second stage; on the other hand a loop of cord could be nipped between the foetal head and the lower uterine segment as was described earlier. The space between the lower segment and the foetus may be very small and babies in the past have been cut slightly or severely on opening the lower uterine segment. I have seen the buttocks sutured or adorned by Michel's clips or an ear resutured. The forces in this area can be considerable, enough to mould an unengaged head, as reported a few years back (Donaldson, 1962) and seen subsequently, and so surely enough to squeeze a loop or coil of the umbilical cord if situated in the high pressure area. This subject remains an open question to be elucidated by further observation and research.

Prognosis

The effect of an abnormal position of the umbilical cord on the foetus may vary between harmless or lethal, depending on the degree and duration of the disturbance to the circulation. Such variations have been noted in the patients described earlier. We must be increasingly interested in the long-term development of the baby in relationship to perinatal morbidity. In this respect the studies of Windle (1963) are pertinent. He performed experiments on monkeys which probably illustrate the mechanisms of brain damage in human infants. The foetuses were delivered complete in the amniotic sac by Caesarean Section so that the degree of anoxia could be controlled. The behaviour of the foetus in the amniotic sac with varying intervals of anoxia and the resuscitative measures required were recorded. Detailed examination of the brains at various stages was carried out. With longer asphyxia not only lack of oxygen but also acidosis were the known biochemical abnormalities. Nerve cells were damaged and some died. The areas affected were the thalamic nuclei, basal nuclei, brain stem and possibly the spinal cord. The lesions were discrete, circumscribed and bilaterally symmetrical. They were non-haemorrhagic. The survivors were hypoactive, hyporeactive, possibly ataxic, unemotional and awkward. They seemed to have poor manual dexterity.

Another syndrome was depression on the second or third day; the babies were dyspnoeic, cyanotic and had fits. Later they resembled humans with cerebral palsy. These brains had also neocortical and cerebellar lesions. Strong uterine contractions produced for hours by an oxytocic infusion might produce intrapartum or neonatal death. The brains showed bilateral haemorrhage in the globus pallidus. The survivors appeared to be retarded. Windle felt that petechial haemorrhages are not caused by asphyxia but are traumatic or agonal due to venous congestion. These studies by Windle should rivet our attention and critical faculties on our present obstetric practice to ensure that we are making continual improvements and preventing avoidable damage to the vital organ which seemingly controls extrauterine development over the years.

Treatment

The principles of treatment of abnormal positions of the umbilical cord are clear. Expectancy should be followed if there is no danger to the baby during labour. If expectancy

could result in a dead or damaged baby then it should be discontinued and the baby removed from its inimical environment. In practice absence of precise information about the position of the umbilical cord makes the mode of treatment a matter of obstetrical opinion and guesswork as has already been discussed in the foregoing cases. This will remain so in the borderline cases until techniques have been developed to study more accurately the actual intrauterine conditions. There is a great need for further research to obtain methods suitable for routine clinical practice to demonstrate the position of the umbilical cord *in utero* and to distinguish between harmful and harmless alterations of the foetal heart; only then can the decision between expectancy and intervention become scientific in obstetric practice.

Summary

The normal position of the umbilical cord is not demonstrable but the abnormal positions are those liable to disturbance of the circulations.

Frank prolapse of the umbilical cord is not always so lethal to the baby.

The forelying umbilical cord and occult prolapse are important, not easily diagnosed and could be the explanation of some unexpected foetal tragedies.

The nuchal cord can occur early in pregnancy, causes some breech presentations, can recur in subsequent pregnancies and is only dangerous if relative shortness of or pressure on the umbilical cord is produced.

Coiling of the umbilical cord around the body or limbs is harmless in most cases and may be the normal position of the umbilical cord. Some exceptions are given.

A short umbilical cord may be responsible for an altered pattern of labour and even the death of the baby.

Alteration of the foetal heart rate or rhythm may be the only evidence of interference with umbilical cord function. The results from electronic studies of the foetal heart are advancing knowledge.

The differential diagnosis involves consideration of the general condition of the mother, utero-placental and foetal factors which are discussed.

Work on experimentally produced asphyxia in animals related to subsequent behaviour and minute studies of the brain may well help in understanding the syndromes in the human.

Treatment will only become scientific in borderline cases when the position of the

umbilical cord and degree of hazard for the baby are accurately known.

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