

FOETAL DISTRESS

BY

CARL WOOD, M.B., F.R.C.S., M.R.C.O.G.

AND

JOHN H. M. PINKERTON, M.D., F.R.C.O.G.

From the Institute of Obstetrics and Gynaecology, Queen Charlotte's Hospital, Goldhawk Road, London, W.6

WITH the decline of maternal mortality and morbidity over the past 30 years the attention of the obstetrician has turned increasingly to the welfare of the foetus. The detection of an abnormal foetal heart-rate or rhythm or the presence of meconium in the liquor is said to indicate foetal distress which may result in the birth of an asphyxiated or stillborn foetus. By careful observation and management of these cases the obstetrician has endeavoured to reduce the number of stillbirths, and with the diminished risks of Caesarean section in recent years he has often performed this operation for foetal distress. Since the stillbirth rate has remained unchanged since 1948, success in the diagnosis and management of foetal distress has apparently been limited.

As more than half of all stillbirths are due to asphyxia the problem of foetal distress must therefore be considered in conjunction with that of foetal anoxia. Certain aspects of the physiology and clinical significance of foetal distress will be considered using the case histories of patients treated at Queen Charlotte's Hospital in the period from 1957 to 1959.

PHYSIOLOGY

Autopsy shows that more than 50 per cent of stillbirths are associated with anoxia. In recent years a considerable amount of work has been done in an attempt to understand the mechanism of oxygen transfer from the mother to the foetus. Although the anatomical pathway of oxygen

from the mother to the foetus is simple, the large number of factors involved in the system, the inaccessibility of the foetus and the difficulties of measuring the oxygen content of the foetal blood limit the functional assessment of this transfer.

The relation between the amount of oxygen in the atmosphere breathed by the mother and the supply of oxygen to the foetus has been studied by Dieckmann and Kramer (1944) and Kinch (1952) and they concluded that the oxygen content of cord blood was increased if the mother breathed pure oxygen before the delivery of the infant. Prystowsky (1959) studied the effect of prophylactic oxygen on the oxygen pressure gradient between maternal ("intervillous space") blood and foetal blood in normal and abnormal pregnancies. When oxygen had been given to the mother during labour in abnormal pregnancies there was a marked increase in oxygen tension and saturation in the umbilical vein and artery. While considerable criticism of oxygen estimations in the blood of the newborn can be made, there is sufficient evidence to support the administration of oxygen to patients in labour when there is a risk of foetal anoxia.

Huggett (1960), in studying the effects of maternal inspiration of 10 per cent oxygen on foetal function in the sheep, demonstrated a slight fall in oxygen saturation of foetal blood, a rise in lactate and a diminution of umbilical blood flow. The oxygen content of the 50 per cent nitrous oxide and air mixture used for

obstetrical analgesia is only 10 per cent and, although inhalation of this gas mixture is intermittent, its further use requires critical evaluation.

The effect of cardiac disease in the mother on the oxygenation of the foetus has not been adequately studied. The occurrence of only 4 stillbirths in 213 patients with cardiac lesions in pregnancy suggests that heart disease does not usually cause significant interference with the oxygen supply to the foetus. This is not surprising as severe maternal anoxia due to cardiac disease only occurs when cardiac failure is present—a relatively rare event during pregnancy.

The effect on the foetus of lowering maternal blood pressure has been studied by Hingson and Hellman (1956), who showed that foetal bradycardia developed within five minutes if the maternal systolic pressure fell below 80 mm. Hg, and that there was a direct relationship between the drop in maternal systolic pressure and delayed crying time following birth. Their work was primarily concerned with the effects of anaesthesia on the foetus and, while it is possible that the changes in the foetus may be due to placental transfer of the anaesthetic agent used, Hon (1959) has shown a similar foetal bradycardia following maternal syncope. Hon (1960) also demonstrated that a mild foetal tachycardia may precede or follow the foetal bradycardia produced by maternal hypotension. He believes that the foetal bradycardia produced by lowering the mother's blood pressure is anoxic in origin and the bradycardia commences late in the phase of uterine contraction when oxygen deficit is most likely to manifest itself.

While maternal hypotension may well cause foetal bradycardia it is unlikely that it often causes foetal death. Hingson and Hellman (1956) were unable to show an increased perinatal loss associated with hypotension produced by anaesthesia. Clinicians quote individual cases where foetal death has occurred following a sudden drop in maternal blood pressure, but many cases of hypotension have been seen following a vaso-vagal attack in pregnancy or the use of hypotensive agents without the occurrence of foetal death. It is probable that foetal death

from hypotension only occurs when this is severe or prolonged or the foetus is already jeopardized from some other cause.

The uterine blood flow has been estimated by measuring the clearance of radioactive sodium from the uterus, by the nitrous oxide method and by the use of an electromagnetic flowmeter. Browne and Veall (1953) and Payling Wright *et al.* (1958) studied the uterine circulation by injecting radioactive sodium (Na^{24}) into the myometrium on the assumption that the time taken for half the injected dose to pass from this depot gives a measure of the effective uterine blood flow. In patients with severe pre-eclamptic toxæmia there was a reduction in uterine blood flow and following rest in bed the flow increased in these cases. During labour a reduction in blood flow was found when uterine contractions were rapid, and also in three cases of foetal distress. Assali *et al.* (1953, 1960), using the nitrous oxide method and the electromagnetic flowmeter, measured the uterine blood flow from the 28th week of pregnancy until the onset of labour. The total uterine blood flow increased progressively during the pregnancy, although when the blood flow was calculated on the basis of the unit weight of the pregnant uterus this remained constant. There is as yet no information bearing on the important question of whether the uterus can adjust its blood flow to local requirements; this occurs in skeletal muscle, dilatation of arterioles increasing the local blood supply in the presence of anoxia.

The decidual vessels at the site of attachment of the placenta have been studied by Dixon and Robertson (1958, 1960). Vascular degeneration was demonstrated in patients with hypertension and pre-eclamptic toxæmia and the extent of these lesions was proportional to the severity of the clinical condition. Decidual vessel narrowing would produce a reduction in blood flow through the intervillous space and this could explain the increased risk of foetal death from anoxia in these conditions.

A study of blood flow through the intervillous space has been made by Ramsey *et al.* (1959). Blood enters the space from the endometrial vessels with sufficient pressure for the stream to be directed to the chorionic plate, when it slows, mixes and then returns via orifices in the decidua

basalis to the maternal veins. Intervillous space and amniotic pressures have been measured in the Rhesus monkey (Ramsey *et al.*, 1959) and in the human (Hendricks *et al.*, 1959), and the average intervillous space pressure during uterine contractions was found to be much lower than the systolic and diastolic pressure in the uterine artery. This system of pressures favours an unimpeded flow of blood from the uterine artery to the intervillous space and from the intervillous space to the uterine vein except when the latter is occluded by a myometrial contraction. Uterine contractions raise the intervillous space pressure so that, when the uterus relaxes, rapid drainage of the intervillous space ensues. If uterine contractions were rapid the time for blood to flow from the intervillous space would be diminished and a stagnant type of anoxia might be produced. When the uterine contractions are strong and frequent the development of a stagnant anoxia would explain the onset of foetal bradycardia late in the phase of uterine contraction (Hon, 1959). When studying amniotic pressure recordings in labour Turnbull (1957), however, was unable to correlate the signs of foetal distress and the presence of strong and frequent contractions, and it must be concluded that any constant relationship between the type of uterine contractions and foetal oxygenation has not been clearly established.

The wide variations in the appearance and histology of individual placentae make it difficult to estimate from a study of these features the ability of the placenta to transfer oxygen to the foetus. Large areas of calcification, fibrin deposition and intervillous thromboses may occur when there is no clinical evidence of foetal distress. Little (1960) in a careful study of villous necrosis of the placenta found that the extent of this lesion could be correlated with the condition of the foetus. When villous necrosis involved more than 10 per cent of the placenta the perinatal mortality was 25 per cent.

Oxygen studies of the blood in the umbilical vessels of the newborn have been made in an attempt to estimate the influence of various obstetrical conditions on foetal oxygenation. Considerable difficulty has been found in the interpretation of the results of these oxygen

estimations because of the conditions under which the blood is obtained, the use of umbilical vessel blood for this work and the methods used in determining the oxygen levels. Blood taken from the newborn following Caesarean or vaginal delivery may not reflect the condition of the foetus *in utero* prior to the birth as delivery is associated with complex circulatory adjustments in the foetus, cord and placenta. The presence of oxygen deficit in the umbilical vessels may not indicate the condition of foetal tissues as the foetus may respond to anoxia by means of increased anaerobic metabolism and local circulatory changes, and the efficiency of these mechanisms may vary for each foetus. The measurement of intervillous space oxygen overcomes the difficulty of changes occurring during birth but the estimation indicates the oxygen in the blood delivered to the foetal surface of the placenta and ignores the transfer of oxygen to the foetus and the state of the foetal circulation.

J. Walker (1959) believes that oxygen saturation in the umbilical vein is a direct index of the supply of oxygen available to the baby at the maternal placenta and that oxygen tension, while reflecting the oxygen available to the foetal tissues, does not indicate the efficacy of its source of supply at the placenta as the baby can achieve a good oxygen tension when the maternal supply is grossly impaired. Kaiser (1959) emphasizes the difference in results between a number of workers using oxygen saturation measurements and believes that the discrepancy in oxygen saturation estimations lies not only in the difficulty of obtaining foetal blood samples under standard conditions but also that oxygen saturation is an inaccurate measure of oxygen. He quotes the Krogh-Erlang equation to support the importance of measuring oxygen tension, not saturation. Rooth, Sjöstedt and Caligara (1960) have studied the oxygen tension in the cord blood in normal deliveries and state that, in order to understand the oxygen supply of the foetus, it is necessary to know the oxygen capacity, the oxygen saturation, the oxygen tension, the pH of the foetal blood and the dissociation curve of foetal haemoglobin. The circulation rate of the foetus, which is so far unknown, is also of importance.

DIAGNOSIS

Foetal Heart-rate

The foetal heart-rate is usually recorded by auscultation but this method has the disadvantage that counting is inaccurate at rapid and variable heart-rates and during uterine contractions. Mechanical methods of foetal heart-recording have the advantage that continuous and accurate recording of all types of foetal heart-rate is possible without disturbing the patient. The foetal phonocardiograph and electrocardiograph have been used by a number of obstetricians but technical difficulties have prevented their common use in obstetric practice.

Although the foetal heart is counted at least every 15 minutes in this hospital, foetal distress was diagnosed in only 29 per cent (20/69) of anoxic stillbirths. It is likely that foetal distress would be diagnosed more often if the foetal heart-rate were recorded more frequently, particularly in patients where the risk of foetal death is increased, i.e., at least every 10 minutes in early labour and every 5 minutes from the latter half of the first stage.

Hon (1959) has studied the foetal heart-rate during contractions by the use of a recording ratemeter attached to the foetal electrocardiograph. A short period of foetal heart-slowness is present in the middle of a contraction during the latter part of the first stage of labour; this is seen as a "V"-shaped slowing on the recording ratemeter and there is some evidence that it is due to an increase in the intracranial pressure during the contraction of the uterus. Hon describes prolonged foetal heart-slowness during the contraction as abnormal; the onset of this slowing occurs early in the contraction phase when it is due to prolapse of the umbilical cord and late in the contraction when due to anoxia from other causes. The abnormal slowing of the foetal heart during a uterine contraction is seen as a "U"-shape on the recording ratemeter and Hon has shown that this change preceded slowing of the foetal heart between uterine contractions.

Further study of the foetal heart-rate during and immediately after the uterine contraction is needed.

Meconium Staining of the Liquor

The presence of meconium in the liquor amnii is diagnosed by the appearance of particles of black, brown, green or yellow material in this fluid. The appearance of clear green liquor has been stated to result from the passage of meconium into the liquor earlier in the pregnancy but it has not been possible to produce this appearance by incubating liquor and meconium at body temperature for periods of up to two months.

There is no way of knowing that the foetus has passed meconium into the liquor until the membranes rupture. In 163 out of 200 patients with meconium-stained liquor this was present at the time of membrane rupture and may have been present for a considerable period prior to this. Fourteen out of 55 stillbirths had meconium liquor but in only 3 was it detected when the foetus was still alive. If the passage of meconium is to be diagnosed during the early development of foetal distress artificial rupture of the membranes should be performed early in labour in patients with an increased risk of foetal anoxia or when the foetal heart rate is abnormal.

Foetal Movement

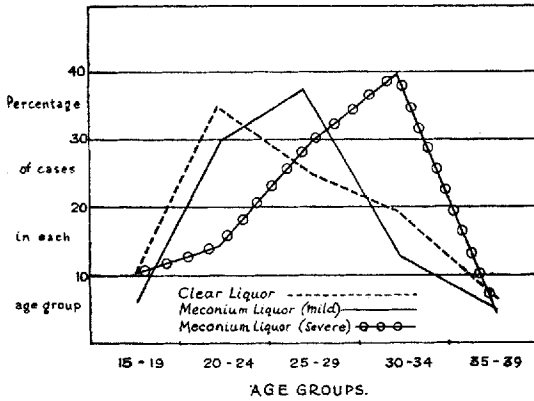
The occurrence of tumultuous foetal movements is said to indicate the presence of foetal distress. Excessive foetal movement may occur during normal pregnancy and labour and sometimes prior to foetal death. Diminution or absence of foetal movement is often recorded prior to foetal death *in utero* when the foetal heart can still be heard. A similar absence or lessening of foetal movement may occur during pregnancy and labour when there is no evidence of foetal distress. However, after questioning a number of patients concerning the type and time of foetal movements, it was concluded that the wide variation in pattern of foetal movements during normal pregnancy markedly detracts from the value of alteration of foetal movement as a sign of foetal distress.

SIGNIFICANCE OF MECONIUM-STAINED LIQUOR

A prospective study of 200 cases where meconium was present in the liquor was made in patients delivered at Queen Charlotte's

TABLE I

Relation Between Age of Patients and Type of Liquor



Hospital in 1959. These cases occurred in 1,400 hospital deliveries, an incidence of one in seven of all confinements (13 per cent). The amount of meconium in the liquor was termed mild or severe, the presence of lumps of meconium or a thick appearance of the liquor indicating severe staining. The cases with meconium in the liquor were compared to 1,200 patients with clear liquor delivered during the same time period.

The age of patients with different types of liquor is shown in Table I, and it is apparent that the age of patients with severe meconium-staining in the liquor was approximately 10 years older than patients with clear liquor. When the gestation period at the time of delivery was analyzed, the average gestation period with severe meconium was 41 weeks and with clear liquor 40 weeks. The age and gestation of the patients with mild meconium-staining of the liquor did not differ significantly from those with clear liquor. The presence of meconium in the

liquor of premature infants is uncommon; the incidence of premature labour in patients with clear liquor was 8 per cent and in patients with meconium liquor 3 per cent.

The relation between the type of liquor and the incidence of obstetrical abnormalities during the pregnancy has been studied. In a comparison of patients with clear liquor and meconium-stained liquor there was no significant difference in the incidence of pre-eclamptic toxæmia, hypertension, ante-partum hæmorrhage and external version.

Labour was more often longer than 24 hours in primigravidae with meconium-liquor, 25 per cent (26/103), than in primigravidae with clear liquor, 8 per cent, while the incidence of labour less than 2 hours was similar in these two groups of patients.

In patients with meconium in the liquor the foetal umbilical cord was more often around the baby's neck at birth than in patients with clear liquor. If the presence of the cord around the neck causes the foetus to pass meconium it was thought that this would occur most commonly during the second stage of labour when advance of the presenting part would tighten the cord. A comparison of the time of onset of foetal distress in patients with the foetal cord normally situated and with the cord around the baby's neck is shown in Table II, and in the latter circumstance the onset of foetal distress was usually in the second stage.

The incidence of severe congenital foetal abnormalities was 1 per cent in the patients with meconium-liquor. In a study of central nervous system abnormalities at Queen Charlotte's Hospital, meconium was passed by 11 of 40 hydrocephalic infants in labour and by none of 42 anencephalic infants. Passage of meconium by an anencephalic infant has been

TABLE II

Relation of Cord Around Neck to Diagnosis of Foetal Distress in Second Stage

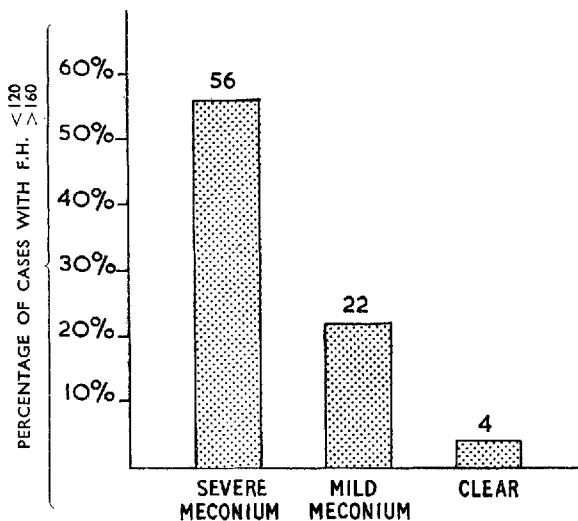
	Cord Around Neck	Cord Normal
Percentage of cases where meconium appears in second stage ..	74% (18/27)	41% (67/164)
Percentage of cases where abnormal foetal heart-rate appears in second stage	85% (11/13)	52% (34/65)

recorded by other authors but the low incidence of this occurrence in the anencephalic may be related to the absence of medullary influence on the vagus nerve which controls intestinal peristalsis.

The diagnosis of meconium in the liquor was made at the time of rupture of the membranes in 81 per cent of cases. On mixing meconium and liquor in different ways at 37° C. it was apparent that the appearance of meconium in liquor will depend on the amount, colour and consistency of the meconium, the degree of mixing of the liquor and meconium, and, least important, by the duration of time the meconium has been in the liquor. *In vivo* the foetal movements and rapid exchange of amniotic fluid may also affect its appearance but at present there is no evidence that the characteristics of the meconium will accurately reflect the duration of time it has been in the liquor. The presence of pieces of meconium in the liquor usually indicates that it recently has been passed from the foetus but sometimes pieces of meconium have been seen some hours after foetal death. Unless meconium appears in the liquor after the occurrence of rupture of the membranes it is difficult to be sure when the meconium was passed.

TABLE III

Relation of Type of Liquor to the Incidence of Abnormal Foetal Heart-Rate



From Table III it can be seen that the occurrence of a foetal heart-rate above 160 or less than 120/minute is more common when meconium is present in the liquor. When the meconium-staining was severe an abnormal foetal heart rate occurred in more than half the cases and if this type of meconium appeared during the first stage of labour the incidence of foetal heart-rate abnormality was 66 per cent (22/33).

Slowing of the foetal heart occurred more frequently in the presence of meconium in the liquor (57 cases) than quickening (11 cases). Table IV shows the relation between the foetal

TABLE IV

Relation of the Foetal Heart-Rate to the Condition of the Foetus in Patients with Meconium in the Liquor

Foetal Heart-Rate	Number of Cases	Stillbirth	Regular Respiration After 4 Minutes
> 160	11	—	7
160-120	130	1	13 (10%)
< 120	57	1	14 (30%)

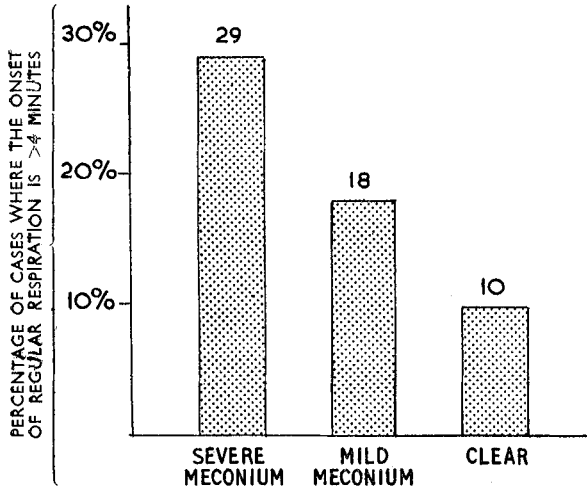
heart-rate and the outcome of labour in patients with meconium in the liquor. Excluding the cases of Caesarean section, delay in the onset of regular respiration was more frequent when the foetal heart-rate was abnormal; there was no evidence that a rapid foetal heart-rate was less significant than a slow rate.

In 7 patients the foetal heart-rate was 60/minute and all the infants were liveborn; in only 2 of these was the onset of regular respiration delayed. The foetal heart slowing preceded delivery in 4 cases and in the remaining 3 it occurred for a short period during the first stage of labour. As no stillbirth occurred in these 7 patients and the foetal heart-rates were the slowest recorded in the series it was not possible to estimate a critical heart-rate below which death of the foetus is imminent.

The accurate timing of the onset of respiration in the newborn is difficult so the onset of regular respiration has been used as a guide to the condition of the infant after birth. Delay in the

TABLE V

Relation of Type of Liquor to the Incidence of Delay in the Onset of Regular Respiration



onset of regular respiration was more common in cases with severe meconium-staining than in those with clear liquor (Table V).

In 25 of the infants it was recorded that meconium was aspirated from the baby's mouth or pharynx but no obvious respiratory disease followed this. Only one case of neonatal pneumonia occurred in the 200 patients with

meconium in the liquor despite the fact that in many of these cases some meconium presumably passed into the foetal lung. There is thus no evidence in this series that aspiration of meconium causes respiratory disease in the newborn.

The perinatal mortality in 200 patients with meconium in the liquor was 3 per cent and in 1,488 patients with clear liquor was 4 per cent; even in patients with severe meconium-staining of the liquor no increase in perinatal mortality was found. Because one-half of the perinatal deaths in patients with clear liquor occur in premature infants and the incidence of Caesarean section is increased in the patients with meconium in the liquor one cannot deduce from these figures that the presence of meconium in the liquor is not associated with an increased risk of foetal death. In other hospital series a high perinatal mortality has been reported when there was meconium in the liquor (Table VI) and at Queen Charlotte's Hospital meconium was present in the liquor of 1 in 4 anoxic stillbirths, which was higher than the incidence of 1 in 7 in all patients delivered in the hospital.

In 68 patients in this series with meconium in the liquor and an abnormal foetal heart-rate one stillbirth occurred. More recent figures obtained from analysis of patients delivered at

TABLE VI

Perinatal Mortality in Foetal Distress

	Abnormal Heart-Rate		Meconium		Meconium + Abnormal Heart-Rate			
	Rapid	Slow	Slow and Irregular	Irregular	Ante-partum	Intra-partum	Slow	Rapid
Walker, J. (1959)	5%	2%	—	—	9%	—	7%	18%
Walker, N. (1959)	—	2.6%	—	—	9%	—	—	11%
Hellman, <i>et al.</i> (1958)	0/15	5%	14%	12%	7%	—	20%	—
Resnick (1955)	—	8%	—	—	—	—	—	32%
Leslie (1959)	—	—	—	—	2.4%	—	—	—
Macafee and Bancroft-Livingston (1958)	—	—	—	—	5%	—	—	—
Lund (1943)	2%	—	—	—	—	—	—	—
Q.C.H.	—	—	—	—	3%	—	—	3%
Ginsburg (1957)	—	—	—	—	3.9%	—	—	—
Fitzgerald and McFarlane (1955)	—	—	—	—	11%	—	—	—

Queen Charlotte's Hospital showed that the perinatal mortality in 138 cases with meconium in the liquor and an abnormal foetal heart-rate was 3 per cent.

The incidence of forceps delivery was similar in patients with meconium-stained and clear liquor, 8 and 11 per cent, while Caesarean section rate was higher in cases with meconium in the liquor, 8 per cent, than cases with clear liquor, 4 per cent. It was difficult to analyze accurately the indication for Caesarean section as the age of the patients, fertility, maturity and other obstetrical factors influenced this decision, but the most significant feature in 13 of the 16 abdominal deliveries was the presence of an abnormal foetal heart-rate and meconium in the liquor during the first stage of labour. There was no perinatal loss in these cases.

After the decision had been made to perform Caesarean section in these patients the foetal heart-rate returned to normal prior to incision of the abdomen in 3 of the 7 cases in which it was recorded. A further 16 patients who had an abnormal foetal heart-rate and meconium in the liquor during the first stage were delivered vaginally of live infants and in 4 of these cases the foetal heart-rate was abnormal throughout labour. Because of these findings we may conclude that we have no accurate way of diagnosing severe foetal distress. The low perinatal mortality and low operative delivery rate in this series also suggests that the presence of meconium in the liquor and an abnormal foetal heart-rate does not usually indicate immediate danger to the life of the foetus.

SIGNIFICANCE OF FOETAL HEART-RATE

Study of the foetal heart-rate is limited by the lack of knowledge of its normal variation. The normal foetal heart-rate can only be established by study of the physiology of heart-rate control in the foetus and by continuous monitoring of the foetal heart during pregnancy and labour. There is no evidence that a change in foetal heart-rate always indicates the presence of foetal distress or that a rise or fall in rate is due to one particular cause.

Anoxia was the post-mortem diagnosis in 60 per cent of stillbirths and a study of the foetal

TABLE VII

Foetal Heart-Rate Prior to Anoxic Stillbirth

	Cases
Normal	11
Normal→Abnormal	7
Normal→Abnormal→Normal	10

heart-rates prior to anoxic death of the foetus during labour has been made in 28 patients (Table VII). The foetal heart-rate was recorded every 15 minutes in the first stage of labour and every 5 minutes if there was meconium in the liquor or if the foetal heart-rate was abnormal it was also recorded every 5 minutes during the second stage. Although an abnormal foetal heart-rate was present at some time during labour in 17 of 28 cases, in only 7 of 28 cases, 25 per cent, did this abnormality persist until the death of the foetus. The duration of the ante-mortem foetal heart change was more than 20 minutes in 4 of these 7 cases.

The types of alteration in the foetal heart-rate in cases associated with intra-partum anoxic death of the foetus are shown in Table VIII and it can be seen that no particular foetal heart change can be associated with anoxia.

It is apparent, therefore, that because of the variable response of the foetal heart to anoxia and the short time period that may elapse between the detection of signs of anoxia and foetal death, present methods of diagnosing foetal distress are inadequate.

In 15 of 26 cases where the stage of labour at the time of foetal death could be accurately diagnosed, foetal death occurred during the second stage.

TABLE VIII

Foetal Heart-Rate Prior to Intra-partum Anoxic Stillbirth

Foetal Heart-Rate (per minute)	Number of Cases
>180	2
180-161	3
160-120	11
119-100	3
<100	3
>160 and <120	3
Irregular	3

Rapid Foetal Heart-Rate

A foetal heart-rate above 160/minute has been found in patients attending the antenatal clinic when the subsequent course of the pregnancy and labour was normal. Lund (1943) states that a transitory rise above 160/minute occurs in 20 per cent of labours and is of no significance. While physiological foetal tachycardia certainly occurs, a raised foetal heart-rate may also be due to anoxia, intra-uterine infection and maternal acidosis, especially when the foetal heart-rate is higher than 180/minute. The diagnosis of intra-uterine infection may be made by the presence of maternal pyrexia and tachycardia and the occurrence of offensive liquor, but sometimes the rise in foetal heart-rate precedes the appearance of these signs. Foetal tachycardia due to maternal acidosis will disappear after adequate intravenous therapy to the mother.

In 2 patients the mother's pulse-rate was more than 120/minute and the foetal heart-rate remained at 130 to 140/minute, and it is clear that a rise in maternal pulse-rate does not necessarily affect the foetal heart-rate.

Slow Foetal Heart-Rate

In 1958 Prystowsky showed that the percentage saturation and partial pressure of oxygen in the intervillous space was not diminished at the height of a contraction, and in 1959 Hon, using the foetal electrocardiograph, produced evidence that transitory slowing of the foetal heart during a contraction was due to an increase in intracranial pressure of the foetus. Compression of the foetal head either before or after delivery of the infant will often produce foetal bradycardia. This may explain the transitory slowing of the foetal heart which sometimes occurs when the presenting part enters the pelvis following membrane rupture. In 2 patients in this series there was clinical evidence of subdural haemorrhage in the infant at birth and the foetal heart-rate was less than 100/minute shortly before delivery. There is sufficient evidence of the role of increased intracranial pressure causing foetal heart slowing, but how often this occurs clinically and how dangerous it may be to the life of the foetus is difficult to determine.

Prystowsky (1959) could find no correlation between foetal bradycardia and oxygen determinations in the foetal and maternal intervillous space. The presence of foetal bradycardia in 9 out of 28 anoxic stillbirths at Queen Charlotte's Hospital suggests that anoxia may be associated with foetal bradycardia and the increased incidence of anoxic stillbirth when the foetal heart was slow in the series of Hellman *et al.* (1958) supports this. The low perinatal mortality associated with foetal bradycardia in J. Walker's series (1959) may be due partly to the fact that 82 per cent of the cases occurred during the second stage. The relation of the umbilical cord to the foetal neck may determine the onset of foetal heart slowing, particularly during the second stage. The role of maternal hypotension in causing foetal bradycardia has already been discussed, and rarely a complete heart-block may cause a regular slow foetal heart-rate.

There is little information concerning the foetal heart-rate which varies from less than 120/minute to more than 160/minute, but this pattern was associated with several anoxic stillbirths. As anoxia is the only condition which is known to cause both quickening and slowing of the foetal heart it is probable that the presence of anoxia is more likely if the heart-rate varies from more than 160 to less than 120/minute than if quickening or slowing of the foetal heart occurred alone.

Foetal heart irregularity may be due to extra-systoles, a partial heart-block or to a sinus rhythm which varies markedly over a short period of time. Redman (1958) has reviewed the subject of foetal cardiac arrhythmias and discussed their association with anoxia and congenital cardiac lesions of the foetus. In 2 patients in this series the foetal heart was irregular a few minutes before it became inaudible and in 1 case an irregularity was present for 20 minutes before foetal death.

TREATMENT

The perinatal mortality associated with foetal distress in several obstetrical centres is shown in Table VI. Perinatal mortality was usually increased when there was meconium in the liquor, particularly when this occurred with

an abnormal foetal heart-rate. The differences in mortality in these series may be accounted for by variation in the definition of foetal distress, the age, parity, nutritional and social status of the patients, the hospital selection of cases, the general obstetrical care, and the management of the foetal distress. This variation in the perinatal mortality of foetal distress emphasizes the fallacy of applying conclusions drawn in one hospital to the obstetrical practice of another. Treatment of foetal distress is unsatisfactory at present and is likely to remain so until clinical investigation can predict more accurately when foetal death is likely to occur.

For the present it would seem reasonable to administer oxygen to the mother as there is evidence that this increases the oxygen supply to the foetus; on the other hand, the use of 50 per cent air and nitrous oxide mixtures (10 per cent oxygen) would seem inadvisable in view of Huggett's observations on the sheep.

N. Walker (1959) suggested that active intervention in labour for foetal distress did not alter the prognosis for the baby; he reached this conclusion by analyzing the results of a series of 350 patients with foetal distress. The management was determined by random selection of cases for obstetrical intervention and non-intervention. The loss of a large number of infants following symphysiotomy, the absence of post-mortem analysis of the cause of foetal death and the relatively high stillbirth rate of 7-8 per cent suggests that his conclusions cannot be applied to current obstetrical practice without further clinical research. His results, however, support the idea that, when foetal distress goes on to foetal death, it usually does so quickly and treatment may be too late to save the foetus. The presence of an abnormal foetal heart-rate for more than 20 minutes in only 4 of the 30 intra-partum anoxic stillbirths at Queen Charlotte's Hospital supports this hypothesis.

As active intervention for foetal distress can only mean an increased survival of a small percentage of cases, other obstetrical factors must be considered in deciding the management of these patients.

In the elderly primigravida or the relatively infertile patient more frequent use of Caesarean section for foetal distress may be justified. The

possibility of major foetal abnormalities must be borne in mind and careful clinical and radiological examination should be carried out before Caesarean section is performed. The presence of pre-eclamptic toxæmia, hypertension or other disease where the risk of foetal death is increased would indicate active intervention for foetal distress. Special significance should probably be attached to a foetal heart-rate above 180/minute or less than 100/minute, especially when meconium is present in the liquor.

It is difficult to determine the relation between the time of occurrence of foetal distress and the risk of foetal death because of the number of variable factors which may influence the perinatal mortality in these circumstances. If labour produces anoxia one would expect it to have an adverse effect on a foetus already distressed. Leslie (1959), however, found that ante-partum meconium-staining of the liquor involved no greater risk of foetal death than did intra-partum staining. Nevertheless, the rate of progress of labour and the expected time of delivery should be estimated as this will often help in deciding the method of delivery of the foetus. If foetal distress occurs in the second stage of labour immediate delivery of the infant is usually indicated although the risk of trauma to the foetus by difficult forceps or breech delivery should also be considered.

SUMMARY

Certain aspects of the physiology, clinical significance and treatment of foetal distress have been discussed.

Foetal distress was diagnosed in only 29 per cent of anoxic stillbirths and therefore an assessment of the methods of diagnosis of the condition was made.

Assessment of the significance of meconium-staining of the liquor was made by a prospective study of 200 cases. The degree of meconium-staining of the liquor was related to the occurrence of foetal heart-rate abnormality and delay in the onset of regular respiration in the newborn.

The cause of foetal heart-rate change was considered, particularly the alteration of the

foetal heart-rate prior to intra-partum anoxic stillbirth.

At Queen Charlotte's Hospital the perinatal mortality in patients with meconium in the liquor either alone or with an abnormal foetal heart-rate was 3 per cent. This low perinatal mortality and operative delivery rate in patients with foetal distress suggests that this condition does not often indicate a grave risk to the life of the foetus.

Until clinical signs or special investigations can determine when foetal death is likely to occur the treatment of foetal distress will remain unsatisfactory. Caesarean section can only slightly reduce the perinatal mortality of foetal distress and therefore other obstetric factors have to be considered in the management of these cases.

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