DIAGNOSIS OF FOETAL ASPHYXIA IN LABOUR

BY

R. W. BEARD AND E. G. SIMONS

For many years intermittent recording of the foetal heart rate with a stethoscope and the detection of meconium in the liquor have been the only signs used for diagnosing intrapartum foetal asphyxia. Although the system has the virtue of simplicity, the unexpected disappearance of foetal heart sound when the foetal heart rate was being recorded as normal, or the frequent delivery of a vigorous baby when a firm diagnosis of foetal distress had been made, have served as constant reminders that more precise tools are needed.

Recently, with the technical improvement in electronic recording, it has become possible to obtain a continuous record of the foetal cardiac activity. At the same time the assessment of the significance of abnormalities of the foetal heart rate has been improved by measuring the pH of foetal blood obtained by the technique of foetal blood sampling (Saling, 1962). In this paper the advantages and limitations of these two techniques for diagnosing foetal asphyxia will be described.

FOETAL BLOOD SAMPLING

The technique.

Blood is usually obtained from the skin overlying the skull of the foetus, but may equally well be collected from the buttocks if the breech of the foetus is presenting.

A conical endoscope is introduced through the cervix until it is firmly against the foetus. A small area of foetal skin can then be visualized and isolated from the surrounding amniotic fluid and debris. The skin is cleaned with a cotton wool swab, sprayed with ethyl chloride to produce reflex hyperaemia and covered with a thin film of silicone gel to allow the blood to form into discrete globules. One or two small incisions are made into the skin by means of a guarded blade.

Immediately a drop of blood appears it is collected into a glass capillary tube containing dried heparin. If required, up to 0.5 ml blood can be collected from the foetus.

Reliability of the pH of scalp blood.

The pH of capillary blood of adults lies somewhere between that of arterial and venous blood. Thus if it can be shown that the pH of scalp blood lies between foetal arterial and venous blood, it is evidence that the scalp sample is satisfactory. Gare, Whetham and Henry (1967) obtained scalp blood at the same time as blood from the carotid artery and jugular vein of sheep foetuses. They showed that the pH of the scalp sample lay between the arterial and venous pH, tending more to the venous value as the animal became hypoxic. Adamsons et al. (1968) confirmed these findings in monkeys after the infusion of oxytocin to simulate labour. They found that over a pH range 6.80–7.40 the pH of the scalp blood was 0.17 above the venous pH and 0.028 below the arterial pH. For obvious reasons such a comparison is more difficult in humans. Kubli et al. (1967) came as close as is possible to a simultaneous collection of samples by obtaining scalp blood within 5 minutes of delivery. Comparison of all acid-base values confirmed the results from animal studies and demonstrated that even in the second stage the flow of blood through the foetal scalp was not sufficiently impaired to alter the pH significantly. The formation of a caput succedaneum is usually considered to be the result of more severe head compression than usual, and would be expected to cause some fall in the pH of capillary blood collected from it. Saling (1965) noted that occasionally the pH of scalp blood from a caput had an increased acidosis, whereas Teramo (1969a) observed very little alteration in pH.

The reproducibility of pH values in scalp blood has been further investigated by the serial collection of samples over a 2-hour period before the onset of labour (Beard, 1970). Although the


* Present address: Harari Hospital, P.O. Box ST 14, Southerton, Salisbury, Rhodesia.
foetal pH variation was as great as 0.10 of a pH unit during this period, it was found to be due to the influence of alterations in maternal acid-base balance, principally from hyperventilation.

The effect of uterine contractions in foetal acid-base values has also been investigated. Saling (1966) has suggested that the improvement in pH that is observed with the onset of labour is due to an enhanced exchange of gases across the placenta. Although this may be the explanation, an alternative one does exist. With each contraction, haemorrhage from the scalp incision increases suggesting an improved blood flow. This change alone will result in an improvement in capillary pH. The changes of pH of foetal blood samples collected during and between contractions has also been investigated. A small but significant fall in \( P_{CO_2} \) during the contraction phase was found by both Kubli et al. (1967) and Beard (1970) although Renou et al. (1968) could find no consistent change.

Errors arising from the collection of blood.

If foetal blood sampling is performed correctly, foetal blood is exposed to air for less than a second before it enters the collecting tube. Both Zernickow (1966) and Kubli et al. (1967) have investigated this potential source of error by determining the fall in \( P_{CO_2} \) of blood exposed to air. The loss of carbon dioxide after 5 seconds of exposure to air resulted in a fall of \( P_{CO_2} \) of less than 1 per cent.

The question is often asked how long foetal blood can be kept before there is a significant change in pH. If the blood is stored in polyethylene tubing there is a progressive loss of carbon dioxide and an increase in the oxygen content of the blood due to diffusion across the wall of the tubing (Kubli et al. 1967). For this reason glass is preferred to polyethylene for the capillary tubes. Studies on foetal blood stored in sealed glass capillary tubes at room temperature show that there is no significant change in pH over the first 30 minutes. From that time there is an increased tendency for the pH to fall (Beard, 1970). It is necessary to mix the blood before measuring the pH, by agitating a small metal rod with a magnet. If the blood is not mixed the pH value tends to be lower.

**Measurement of pH.**

The error will depend on the experience of the individual who measures the pH and the equipment used. In a series of routine pH determinations on 16 blood samples the methodological error of an experienced technician using an Astrup pH meter is shown in table I. It is clear from the figures that error from this source is likely to be small in comparison with the potential error from other aspects of the technique.

**Table I**

<table>
<thead>
<tr>
<th>Determination</th>
<th>Error</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>±0.005</td>
</tr>
<tr>
<td>Base deficit</td>
<td>±0.6</td>
</tr>
<tr>
<td>( P_{CO_2} ) (mm Hg)</td>
<td>±1.2</td>
</tr>
<tr>
<td>( P_O_2 ) (mm Hg)</td>
<td>±1.2</td>
</tr>
</tbody>
</table>

**Normal foetal acid-base values.**

Serial collection of foetal capillary blood during labour has shown that the foetal pH varies very little until shortly before delivery when it tends to fall (Brethes and Saling, 1967; Wulf, Kunzel and Lehmann, 1967; Kubli, 1968; Beard and Morris, 1965). The acidaemia that develops at this time is primarily metabolic and is derived from the mother. Brethes and Schmidt in unpublished work have recently shown that throughout labour the increase of lactic acid in the mother is paralleled by an equivalent increase in the foetus. Table II shows the mean acid-base and blood-gas values of 18 foetuses who had serial blood samples collected through labour and into the first 24 hours of extra-uterine life. There was a fall in pH during labour, and it was only at 24 hours after birth that the pH of the newborn achieved adult values. There was a fall in oxygen saturation of capillary blood to values similar to those found in the umbilical artery at birth suggesting that shortly before delivery there is an increase in oxygen consumption by the foetus.

**Abnormal foetal pH.**

A critical pH, which distinguishes normal from abnormal foetal pH values, is usually accepted as being two standard deviations below the normal mean. Values obtained in this way from various
Acid-base and blood-gas values derived from a study in labour of 18 normal foetuses. Neonatal values during the first 24 hours of life were determined on 10 of these foetuses.

<table>
<thead>
<tr>
<th></th>
<th>Labour</th>
<th>Delivery</th>
<th>Neonatal period (hr)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1st stage</td>
<td>2nd stage</td>
<td>Umbilical vein</td>
</tr>
<tr>
<td>pH</td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td></td>
<td>7.335</td>
<td>0.052</td>
<td>7.301</td>
</tr>
<tr>
<td>PCO₂ (mm Hg)</td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td></td>
<td>41.0</td>
<td>8.2</td>
<td>41.8</td>
</tr>
<tr>
<td>Base deficit (m.equiv/L)</td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td></td>
<td>4.75</td>
<td>2.55</td>
<td>6.65</td>
</tr>
<tr>
<td>PO₂ (mm Hg)</td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td></td>
<td>50.8</td>
<td>16.9</td>
<td>29.5</td>
</tr>
<tr>
<td>So₂ (%)</td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td></td>
<td>37.3</td>
<td>14.2</td>
<td>35.9</td>
</tr>
</tbody>
</table>

studies are compared in table III. These pH values range between 7.17 and 7.23 and their close agreement confirms that 7.20, the pH originally proposed by Saling (1964) is acceptable. However, too close an adherence to a critical level of pH has definite disadvantages in practice.

Lower limit of normal pH values, i.e. the value of pH 2 SD below the mean, in different studies.

|Authors                      | 1st stage | 2nd stage
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Bretscher and Saling (1967)</td>
<td>7.20</td>
<td>7.20</td>
</tr>
<tr>
<td>Paterson et al. (1970)</td>
<td>7.23</td>
<td>7.17</td>
</tr>
<tr>
<td>Kubli (1968)</td>
<td>7.21*</td>
<td>7.23*</td>
</tr>
<tr>
<td>Paul, Gare and Whetham (1967)</td>
<td>7.20</td>
<td>7.20</td>
</tr>
<tr>
<td>Beard and Morris (1965)</td>
<td>7.20</td>
<td>7.17</td>
</tr>
</tbody>
</table>

Lower limit of pH range.

Most foetuses that develop intrapartum hypoxia have a normal pH at the beginning of labour (Beard, Morris and Clayton, 1967). Hypoxic acidosis usually develops slowly during labour (Saling and Schneider, 1967). As it does so the pH falls until the critical value is reached. Thus, if foetal hypoxia is to be detected early, pH values which are in the lower range of normal should be regarded with considerable suspicion. Bretscher and Saling (1967) have proposed a range of pH from 7.20 to 7.24, termed "pre-acidosis", which they regard as suggestive of foetal hypoxia.

Clinical application.
Considerable experience in the clinical use of foetal pH to determine the significance of abnormalities of the auscultated foetal heart rate and meconium in the liquor has been gained since the technique was first described by Saling in 1962. In this country the management of patients presenting with foetal distress by foetal blood sampling has been described by Coltart, Trickey and Beard (1969). In this study a predetermined procedure was adhered to. As soon as any abnormality of the foetal heart rate or the appearance of meconium was noted, a foetal blood sample was collected. If the pH was normal (>7.25), labour was allowed to continue. A further blood sample was only collected if the foetal heart remained abnormal. If the pH was between 7.20 and 7.25, a further sample was collected within 30 minutes of the first. If the pH was less than 7.20 a further sample was collected immediately. In the meantime preparations were made for operative delivery. The management of these patients and their delivery was determined by the foetal pH. When the foetal pH was within the normal range (>7.25) labour was allowed to continue. A further blood sample was collected if the foetal heart remained abnormal. If the pH was between 7.20 and 7.25 the majority of patients were allowed to continue in labour unless, on repeated sampling, the pH remained low and a persistent abnormality of the foetal heart rate was present. If the pH was below 7.20 (this group constituted 45 per cent of all acidotic foetuses), delivery was by Caesarean section in the first stage or by forceps in the second stage. If the cervix was not quite fully dilated and the
foetal pH was less than 7.20 delivery by Caesarean section was usually preferred to vacuum extraction or forceps. This was because it was felt that a difficult vaginal delivery would add further insult to a foetus whose response to stress was already diminished. Maternal acidosis was not always excluded as a possible cause of foetal acidosis because of the lack of availability of a technician to perform the necessary measurements. Beard (1968b) found an incidence of only 10 per cent of infusion acidosis* and, for reasons previously stated, it seems unlikely that this condition was often a cause of a mistaken diagnosis of foetal distress.

The change in the clinical management of foetal distress was reflected in the frequency with which Caesarean sections were performed. At Queen Charlotte's Hospital between 1961 and 1964, in the four years preceding the introduction of foetal blood sampling, the number of Caesarean sections rose sharply as a result of attempts to reduce intrapartum stillbirth. The failure of this policy was evident from the unchanged perinatal mortality over these years. Following the introduction of foetal blood sampling the number of Caesarean sections fell progressively and in 1966 and 1967 only 27 operations were performed as compared with 86 in 1963 and 1964.

Beard and Morris (1969) reported on the perinatal mortality among babies weighing more than 1500 g, without evidence of rhesus sensitization or major congenital anomaly, dying during labour or within 72 hours of birth. They showed that in the four years preceding the clinical use of foetal blood sampling the mortality among these babies remained virtually unchanged, between 27 and 36 perinatal deaths each year. In the five years that followed the introduction of foetal blood sampling into the practice of the hospital, the perinatal deaths were 14, 31, 19, 19 and 11, showing a significant fall in mortality as compared with the previous years.

Despite these encouraging results the major limitation of foetal blood sampling used in the manner described was that a foetal pH determination was performed only when the midwife recorded some abnormality of the foetal heart rate or recognized the presence of meconium in the liquor. If, as is the case in many obstetric centres, the foetal heart sounds are only auscultated every 30 minutes and the membranes are left intact foetal asphyxia is often not detected for some time with a consequence that the foetus may be subjected to prolonged asphyxia. An analysis of intrapartum stillbirths over a 30-month period during which 10,000 babies were delivered was made. In this time there were 13 stillbirths due to intrapartum asphyxia. In these babies there was no abnormality of the auscultated foetal heart rate or meconium in 10 of them to indicate the need for the collection of a foetal blood sample (Beard, 1968a). These findings clearly point to the need for more sensitive and reliable diagnostic criteria of foetal asphyxia.

**CONTINUOUS RECORDING OF THE FOETAL HEART RATE**

*Theoretical considerations.*

The principle underlying the detection of foetal asphyxia by continuous foetal heart rate monitoring is that uterine contractions serve as intermittent stressful stimuli to the foetus. With each contraction there is a temporary reduction in the flow of oxygenated blood through the intervillous space. If placental function was impaired before the onset of labour, or the contractions are too frequent, then the foetus is liable to become asphyxiated. Equally, if the umbilical cord is entangled with the foetus (around the neck, shoulder or a limb) it is most likely to become occluded during the contraction phase. In the early stages of hypoxia abnormalities of the foetal heart rate (decelerations during contractions and tachycardia between contractions) precede acidosis (Lumley and Wood, 1967). It is only in the latter stages that foetal heart rate decelerations become more prolonged, extending into the resting phase between contractions and the foetal pH falls. Because auscultation is confined to the interval between contractions the detection of foetal asphyxia is likely to be delayed longer than if continuous recording of the foetal heart rate is used for monitoring.

Various techniques have been employed to record the instantaneous foetal heart rate, most of which are commercially available in one form or another. A brief description of these will be given.

---

*Infusion acidosis in the foetus is secondary to a primary metabolic acidosis which may develop (in the mother) during labour—usually shortly before the second stage.*
All are dependent on the conversion by a cardiotachometer, of paired beats into an instantaneous rate expressed on a dial and on recording paper as the number of beats per minute.

Abdominal Methods.

(1) Phonocardiography (Hewlett-Packard Ltd, 8020A monitor).

Foetal heart sounds are detected by a microphone strapped to the maternal abdomen. The system is dependent on the heart sounds being of sufficient intensity to trigger the cardiotachometer. The value of this method is limited in obese patients or if the foetus moves. In addition, extraneous sounds are also picked up by the microphone and an otherwise satisfactory record may disappear at the peak of uterine contraction just when it is most useful.

(2) Ultrasound (Sonicaid Ltd, FM2 monitor).

Movements of the foetal heart are detected by a beam of ultrasound of low acoustic energy (less than 10 mw/cm²) on the maternal skin, transmitted by a transducer attached to the maternal abdomen. Because of the wide angle of the beam of emitted sound a certain amount of foetal movement can occur without interruption of the record. Maternal movement and uterine blood flow do not interfere with the foetal signal. Some anxiety has been expressed about the safety of prolonged exposure of the foetus to ultrasound (Editorial, 1970a; Macintosh and Davey, 1970) but recent work has not supported this view (Hellman et al., 1970; Boyd et al., 1971).

(3) Indirect foetal electrocardiography.

Electrodes attached to the maternal abdominal wall pick up the foetal e.c.g. (Larks and Dasgupta, 1958; Southern, 1957; Bergman and Hall, 1958). The poor signal to noise ratio and interference from the maternal e.c.g. has limited the usefulness of this technique.

Intra-uterine Methods.

Direct foetal electrocardiography (Hewlett-Packard Ltd; Corometrics Ltd; Sonicaid Ltd).

A unipolar electrode attached directly to the foetus and insulated from the mother picks up the foetal e.c.g. A separate electrode lying free in the vagina of the mother picks up the maternal e.c.g. which can then be filtered off to prevent interference. The cardiotachometer is triggered by the foetal R wave. At the time of writing this is the best method available for obtaining a reliable record of the instantaneous foetal heart rate throughout labour. The drawback is that the cervix must be sufficiently dilated and the membranes must be ruptured before the electrode can be applied. However, the fact that the signal is unaffected by maternal or foetal movement or by the intensity of uterine contractions outweighs this disadvantage. No single instrument is better than another, but the Sonicaid FM2 is the most versatile since the foetal heart rate can be recorded either by ultrasound or foetal e.c.g.

Recording of Uterine Contractions.

This can be done by internal or external methods. If a measure of the intensity of contractions and the resting uterine tone is required an internal method must be used—usually a fluid-filled catheter lying inside the uterus and attached to a strain gauge, but recently telemetric methods have shown promise. For clinical purposes internal methods have some disadvantages. The external method using a strain gauge is easier because all that is necessary is to strap it to the maternal abdomen. Although this method does not record intra-uterine pressure, an indication of the onset, peak and end of the contraction is provided, which is all that is required for assessing the significance of changes in the foetal heart rate. Hopefully a method which combines the advantages of both internal and external systems will be developed.

THE SIGNIFICANCE OF CHANGES IN THE CONTINUOUS FOETAL HEART RATE RECORD

Our knowledge about the regulation of the circulation of the mature foetus, human or otherwise, is still far from complete. It seems likely that there is a well-developed autonomic nervous system which is capable of mediating chemoreceptor and baroreceptor reflexes (Dawes, 1968). Thus relatively small changes in blood-gas and hydrogen ion concentration are likely to result in changes in the foetal heart rate. The problem for the clinician is complicated by the intermittent type of stress to the foetus which results from
DIAGNOSIS OF FOETAL ASPHYXIA IN LABOUR

879

uterine contractions. If the stress were continuous, such as under experimental conditions when the foetus can be asphyxiated by partial constriction of the umbilical cord, then the changes in foetal heart rate and acid-base would be predictable. However, in labour the severity of foetal asphyxia induced by uterine contractions is dependent on such variable factors as the intensity and duration of the contractions and the position of the patient. The problem is not made simpler by trying to decide at what stage of intermittent hypoxia the foetus is liable to suffer permanent neurological damage. In practice, although abnormalities of the continuous foetal heart rate record are considered as evidence of foetal asphyxia, operative delivery of the foetus is not contemplated unless there is an associated fall in foetal pH (Beard et al., 1971a).

The two pioneers of foetal heart rate monitoring, Hon (1962) and Caldeyro-Barcia et al. (1966), assessed the significance of continuous foetal heart rate patterns from the condition of the baby at birth. This work showed that babies were often born in poor condition if birth was preceded by decelerations appearing near the end of or after contractions (late decelerations of Hon, or type II dips of Caldeyro-Barcia). In contrast, when its heart rate showed no tendency towards slowing with contractions, the baby was usually born in good condition. These observations were complicated by the fact that decelerations can be caused by non-hypoxic stimuli such as head compression (Hon, 1959; Schwarcz et al., 1969), that foetal heart rate abnormalities frequently do not follow every contraction, and that a baby born in poor condition is not necessarily asphyxiated. A more objective assessment of the significance of foetal heart rate patterns has been possible with the advent of foetal blood sampling which has allowed a simultaneous comparison of foetal heart rate and foetal pH. Figure 1 shows the terminology that is used for describing continuous foetal heart rate changes.

**Table IV**

Mean pH and base deficit values found in relation to specific heart rate patterns (Kubli et al., 1969; Hon and Khazin, 1969).

<table>
<thead>
<tr>
<th></th>
<th>Mean foetal pH (± SD)</th>
<th>Mean foetal base deficit (± SD) (m.equiv/l.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal pattern</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Early deceleration</td>
<td>7.30 ± 0.042</td>
<td>6.98 ± 0.16</td>
</tr>
<tr>
<td>Mild variable deceleration</td>
<td>7.30 ± 0.041</td>
<td>6.97 ± 0.41</td>
</tr>
<tr>
<td>Moderate variable deceleration</td>
<td>7.29 ± 0.046</td>
<td>7.84 ± 0.19</td>
</tr>
<tr>
<td>Mild late deceleration</td>
<td>7.26 ± 0.044</td>
<td>8.98 ± 0.44</td>
</tr>
<tr>
<td>Moderate late deceleration</td>
<td>7.22 ± 0.060</td>
<td>9.29 ± 0.49</td>
</tr>
<tr>
<td>Severe variable deceleration</td>
<td>7.21 ± 0.054</td>
<td>10.79 ± 0.43</td>
</tr>
<tr>
<td>Severe late deceleration</td>
<td>7.15 ± 0.069</td>
<td>10.17 ± 0.93</td>
</tr>
<tr>
<td></td>
<td>7.12 ± 0.066</td>
<td>12.88 ± 0.77</td>
</tr>
</tbody>
</table>

Early deceleration: early onset relative to peak of uterine contraction.

Variable deceleration: variable in onset and shape.

Late deceleration: deceleration with definite lag-time.

Table IV is taken from a study by Kubli et al. (1969) and Hon and Khazin (1969). It shows that the greater the amplitude and duration of deceleration, and the later the deceleration relative to the contraction, the more likely is the foetus to have a metabolic acidosis. Studies adopting a similar approach by Wood et al. (1969) and Caldeyro-Barcia et al. (1968) have resulted in similar conclusions being reached.

Attempts have been made to distinguish by heart rate changes the asphyxiated from the well-oxygenated foetus without resort to foetal pH determinations. Shelley and Tipton (1971) have suggested that a good assessment of the foetus can be made by measuring the area of the decelerations on the foetal heart rate record that appear over the period of an hour. If the presence or
absence of meconium in the liquor is also considered, a very reliable forecast of the condition of the baby at birth can be obtained. Sureau et al. (1970), who have measured the area of deceleration occurring after the end of a contraction with the aid of a computer, consider that there is a direct relationship between the residual bradycardia and the severity of foetal asphyxia.

Both these approaches have the advantage that the assessment takes into account both the duration of the foetal heart rate abnormality and the severity of the deceleration with each contraction. At the moment the problem is to discover how this can be applied to the management of the foetus during labour.

There is clearly a place for regarding the continuous foetal heart rate as a screening process for foetal asphyxia, and then using the foetal pH for confirming or rejecting the diagnosis. In a recent study, Beard et al. (1971a) have shown that 32 out of 37 acidotic foetuses (P≤7.25) had an abnormality of the continuous foetal heart rate record. In contrast of the 138 foetuses with an abnormality of the foetal heart rate only 32 were acidotic. The logic behind the use of a combined continuous record and pH in this assessment was summed up by Goodlin (1971) when he stated that a foetus subjected to chronic hypoxia may be unable to tolerate that minimal degree of hypoperfusion associated with type I dips (early decelerations of small amplitude), while a well-nourished foetus may easily tolerate severe uterine hypoperfusion associated with type II dips.

Recent studies (Goodlin, 1971; Low et al., 1971; Beard et al., 1971b) have all tended to show that certain features of the continuous foetal heart record are indicative of foetal asphyxia:

1. loss of the normal fluctuation of the heart rate—sometimes, referred to as "loss of beat-to-beat variation" or "smoothing";
2. baseline tachycardia with lag time;
3. delayed decelerations—referred to variously as late decelerations or type II dips;
4. deep decelerations exceeding 50 beats per minute.

An example of an abnormal trace which has these features is shown in figure 2. For comparison a normal trace is shown in figure 3.

**Effects on the Foetus of Drugs Administered to the Mother.**

Drugs administered to the mother in labour are known to affect the foetal heart rate record. Diazepam (Valium) when given intravenously results in a smooth trace, or loss of beat-to-beat variation (Beard et al., 1971b; Goodlin, 1971). A similar effect, although not so constant, is observed after the administration of pethidine. These changes are liable to confuse the interpretation of the con-

---

**Fig. 2**

Abnormal continuous foetal heart rate record.
Continuous foetal heart rate record, but if the foetal heart rate is within the normal range and there are no decelerations with uterine contractions it can be assumed that the loss of beat-to-beat variation is due to the drug and not to asphyxia (Beard et al., 1971b). Intravenous atropine crosses rapidly from mother to foetus, resulting in a baseline tachycardia (presumably due to blocking of the foetal vagus) and loss of beat-to-beat variation. It is of interest that even after atropinization, decelerations due to asphyxia persist. Propranolol causes a small but significant fall in foetal heart rate (Renou et al., 1969).

CLINICAL ASPECTS OF COMBINED FOETAL HEART RATE AND pH MONITORING

The clinical application of monitoring techniques to the care of the foetus is still in its infancy. Nevertheless it is likely that over the next few years we shall see considerable changes brought about by monitoring in the management of patients before and during labour.

Antepartum monitoring.

The assessment of the condition of the foetus before labour has been based chiefly on the estimation of the 24-hourly urinary excretion of oestriol by the mother (Turnbull, 1970). Recently various forms of “stress” tests have been studied. Physical activity is known to diminish uterine blood flow (Morris et al., 1956), and following the earlier work of Hon and Wohlgemuth (1961), Stembera (1969) has reported on the effect on the foetal heart rate of exercising the mother. He found that in a group of women with complicated pregnancies, 51 with a normal rate throughout the test had no evidence of foetal asphyxia during labour, whereas in 56 in whom late decelerations appeared during the test there were two stillbirths and a high incidence of intrapartum foetal asphyxia.

Uterine contractions stimulated by intravenous oxytocin has been used instead of exercise (Pose et al., 1969). The value of these tests in the management of the patient is, as yet, not altogether clear. It has been proposed by these workers that an abnormal test is an indication for elective Caesarean section. However, before such a conclusion is generally acceptable further studies will have to be done to determine the incidence of acidosis among foetuses with an abnormal stress test who are subjected to labour. If, as seems likely, the foetus is at little risk until pH changes have been present for some time, it is probably best to regard a positive stress test as an indication for continuous heart rate monitoring and serial foetal pH values during labour. Then, as soon as foetal acidosis appears, the foetus can be delivered without delay.
Intrapartum monitoring.

Recently three centres have reported the results of monitoring on a clinical service basis. In two studies Kubli and Ruttgers (1971) monitored 300 patients, and Goodlin (1971) monitored 500 patients. In both these studies attempts to monitor every patient admitted in labour were unsuccessful, a rate of only 40 and 48 per cent respectively being achieved. In the study by Beard et al. (1971a) 392 high-risk pregnancies were monitored. These patients were selected and in consequence a success rate of 72 per cent was achieved, but it is clear with either approach that monitoring in its present form cannot be applied to all women in labour.

Perinatal mortality has traditionally been used as the yardstick against which to measure the success or otherwise of any change in clinical practice. The disadvantage of this approach is that mortality in labour is low anyway, and that any improvement in practice is liable to escape unnoticed (Editorial, 1970b). Nevertheless in the study by Goodlin (1971) he reported a perinatal mortality of 15.6 per 1000 births as compared with figures of 22.8, 18.0 and 24.9 for the preceding years when monitoring was not used. Beard et al. (1971a) had no stillbirths among the patients in whom monitoring was satisfactory. However, their study did reveal some of the difficulties of eliminating stillbirth by this approach. Failure to recognize high-risk indications for monitoring accounted for two stillbirths, and it suggests that to eliminate intrapartum stillbirth, monitoring should be made available to all patients. Goodlin (1971) found the highest incidence of foetal heart rate abnormalities among his obstetrically “normal” group of women.

There seems little doubt that foetal asphyxia can be detected at an earlier stage by continuous monitoring than by auscultation of the foetal heart. Examination of the records of babies born in poor condition with biochemical evidence of asphyxia usually show an abrupt change from a normal to an abnormal pattern of foetal heart rate record. The earlier diagnosis of foetal asphyxia is shown in two comparable studies. In one the indication to collect a foetal blood sample was an abnormality of the auscultated foetal heart rate (Coltart et al., 1969). In the other a continuous record was made (Beard et al., 1971a). The incidence of severe foetal acidosis (pH<7.15) was considerably less in the group monitored continuously.

Reliance on monitoring to detect foetal asphyxia at an early stage has resulted in a considerable fall in elective Caesarean sections at King’s College Hospital (Beard et al., 1971a). During the study year only 21 out of 137 operations were elective. The annual incidence of 6.9 per cent remains unchanged, and it could be argued that women who eventually had a Caesarean section were unnecessarily being subjected to labour. In fact when individual cases are scrutinized few of the women who had Caesarean sections in labour had any indication for an elective operation. Thus there appears to be a tendency to resort more readily to Caesarean section among women who are being monitored when there is a combination of an abnormal continuous foetal heart rate record and delayed progress, for example. A similar effect was noted by Goodlin (1971) and Hibbard (1971). Margolis (1971) and Beard, Morris and Clayton (1966) found a decreased incidence when foetal blood sampling was used in conjunction with auscultation of the foetal heart. It seems likely that if foetal acidosis becomes generally accepted as the criterion for a diagnosis of foetal distress, the incidence of Caesarean section should fall.

IATROGENIC FOETAL ASPHYXIA

Monitoring has provided a valuable means of determining some of the avoidable causes of foetal asphyxia.

Intravenous oxytocin.

Uterine activity stimulated by intravenous syntocinon has been shown to have characteristics which differ from those of spontaneous activity and which are more likely to lead to foetal asphyxia (Kubli and Ruttgers, 1971). These workers showed that basal tone and uterine hyperactivity were more frequent when syntocinon was used. The adverse effect on the foetus was demonstrated by the significantly lower pH of foetuses subjected to uterine hyperstimulation. In practice the effect can be recognized during the course of stimulated labour by an abnormal heart rate
pattern. If the oxytocin infusion is reduced or turned off the pattern usually becomes normal, and labour continues.

**Regional anaesthesia.**

In recent years there has been an increasing tendency to use paracervical and epidural anaesthesia for analgesia during labour. The paracervical approach has been an attractive one to obstetricians because it does not require the presence of an anaesthetist. However, in the last few years several reports have appeared showing that paracervical block can lead to foetal asphyxia which is sometimes fatal (Teramo, 1969b; Jung, Dopecky and Klöck, 1970; Gordon, 1968). The reason for this is not altogether clear. The most generally accepted view is that the anaesthetic agent crosses to the foetal circulation and by its quinidine-like effect on the myocardium leads to cardiac arrest. Recently, however, Kubli and Rutgers (1971) have shown that uterine hypertonus may follow the injection of the local anaesthetic, and they have suggested that the diminished flow of blood to the intervillous space could well be a cause of foetal asphyxia.

Epidural, and caudal, anaesthesia are being increasingly used in the routine management of labour. There is no doubt of the benefit to the mother in terms of pain relief of this form of anaesthesia but some anxiety has been expressed about possible adverse effects on the foetus (Morishima et al., 1966; Takahashi, 1969).

Noble et al. (1971), in a prospective trial involving 100 women who had an epidural anaesthetic in labour compared with 102 women given conventional anaesthesia, claim that the anaesthetized group gave birth to babies in better condition. Potter and MacDonald (1971) in a larger study found no significant difference between the Apgar scores of the babies of mothers given epidural anaesthetics and others. In both these studies hypotension following the injection of the local anaesthetic agent was not uncommon—18 and 8 per cent in each study respectively. It is this complication that carries some risk to the foetus; Potter and MacDonald report the death of a foetus after a hypotensive episode following a “top-up.” Noble et al. found that the blood pressure recovered in all their hypotensive patients as soon as they were turned on to their sides, yet Potter and MacDonald report that recovery occurred in only 55 per cent of their patients, the remainder showing no relief from this manoeuvre. The results from these two studies suggest that the safety of epidural anaesthesia largely depends on the technique of administration employed. Maternal blood pressure and foetal heart rate (preferably by continuous recording) should be monitored throughout the administration of the anaesthetic and for some time afterwards. The patient should be encouraged to lie on her side once an epidural has been induced. It is probably preferable to administer an epidural when a patient is not under general anaesthesia.

**Supine hypotension.**

Pregnant women are particularly liable to supine hypotension (Kerr, 1968) and deaths during general anaesthesia have been reported (Courtney, 1970). There is little information on the frequency with which this condition occurs in pregnancy and labour. The supine position is commonly adopted in labour, during the first stage so that the midwife can listen to the foetal heart and in the second stage to facilitate delivery. Unrecognized supine hypotension is occasionally a cause of foetal asphyxia (Beard and Roberts, 1970). The clinical course of events is that initially an abnormal foetal heart rate and foetal acidosis are detected and the decision made to proceed to Caesarean section. However, at this time it may be noticed that the patient is pale and sweating. By turning the patient on her side a dramatic improvement in the foetal heart rate and a slow return of the foetal pH to normal is seen. It is of some interest to speculate on the extent to which the supine position is a cause of foetal asphyxia at Caesarean section, either elective or in labour. Lumley et al. (1970) showed that there was an inverse relationship between the time taken to deliver the baby at Caesarean section and the foetal pH. Supine hypotension may well explain this relationship and account for some of the babies born in poor condition after an apparently uncomplicated Caesarean section.
CONCLUSION

Monitoring the foetus by continuous foetal heart rate recording and foetal pH measurements is now a practical procedure. It seems likely that the application of these techniques to routine clinical obstetrics will lead to the earlier diagnosis of foetal asphyxia and to the more rational management of patients in labour.

REFERENCES


