OBSTETRICS FOR PAEDIATRICIANS

The management of term labour

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Introduction

Labour is the process of regular uterine activity and associated cervical dilatation. More than 90% of mature babies are born after a period of labour; most deliver vaginally, either spontaneously or with operative assistance; some require emergency caesarean section.

The two main reasons for emergency caesarean section during labour are 'failure to progress' and 'fetal distress'. The baby's condition at birth depends on placental function at the onset of labour, duration (and other aspects related to management) of labour, and mode of delivery.

The first two of these factors relate, primarily, to fetal oxygenation while the third factor relates both to fetal oxygenation and potential physical trauma at the time of birth. Resuscitation is required when the baby fails to breathe after delivery, and the term 'birth asphyxia' is often used when apnoea is associated with other features of a low Apgar score, such as bradycardia and flaccid tone.

This article discusses the management of term labour and relates events to fetal and subsequent neonatal condition. It begins by describing how different concepts of the process of labour influence management and subsequently describes interventions. The second half of the article discusses fetal monitoring and concludes with a brief section on birth asphyxia.

The objective is to convey an understanding of labour management to help the attending neonatal paediatrician decide the appropriate diagnosis and management when resuscitation is required. In particular, aspects of labour which contribute to the present concept of birth asphyxia must be understood, recognised, and correctly interpreted when considering the aetiology of early onset neonatal encephalopathy.

Diagnosis of labour

Recognition of the onset of labour determines all subsequent management objectives. Evidence of cervical dilatation is required to be sure of 'progress', but early graphical descriptions of labour¹ indicated that 'early' labour (defined as cervical dilatation of less than 3 cm) could last many hours. This led to the idea that there is a slow, or latent, phase of labour when the cervix is less than 3–4 cm dilated, after which there is a faster, or active, phase of labour. This conservative approach is

favoured by most midwives in the United Kingdom because they believe this represents the physiological norm. The problem with this approach is the potential for a discrepancy between the symptoms of labour (painful uterine contractions) and cervical dilatation. Clearly, this approach makes it very difficult to recognise abnormal labour before 3 cm of dilatation, and some women are allowed to continue in 'early' labour for many hours, resulting in maternal exhaustion and reduced fetal reserve.

Publication of the experiences of labour management at the National Maternity Hospital, Dublin, has greatly influenced current practice in some units in recent years.² Their policy, described as 'active management' of labour, has resulted in the delivery of 98% of first labours within 12 hours of the diagnosis of labour. This is a major challenge to the idea that prolonged labour is a normal and inevitable consequence if women present 'early' in labour and have yet to get through the latent phase. However, the problem with this approach is the rather low threshold for interventions.

The diagnosis of labour requires recognition of uterine activity that is effecting a change in the cervix. Mothers usually present when uterine contractions become regular and cause major discomfort. Pain is a subjective experience and is always important to the mother, especially when bad enough for her to ask for analgesia. Regularity, an objective observation, is usually accepted when contractions occur more frequently than one every five minutes and have done so for more than one hour.

When regular contractions are considered painful by the mother the question of 'confirming' the diagnosis of labour is appropriate. If the cervix is 3 or more cm dilated then labour is usually diagnosed with certainty. If the cervix is less than 3 cm dilated then this is when conservative and active approaches differ in subsequent management. The former would consider labour 'not to be established' and describes the situation as 'early'. Rarely is a time limit put on the situation and the mother is unlikely to be given any idea of how long the situation will last. A further examination is usual after four hours. On the other hand, the active approach considers it appropriate to confirm or refute the onset of the labour process, despite the symptoms, and looks for other features to confirm the onset of labour. Effacement or dilatation of the cervix, a 'show'

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(the physical extrusion of mucus from the cervical glandular epithelium), and ruptured membranes are all features that are used to 'confirm' the onset of labour in such circumstances. In the absence of any of these the woman is told she is not in labour. The only prospective randomised controlled trial of 'active management' of labour used either complete effacement or dilatation of the cervix, or ruptured membranes, in the presence of regular painful contractions.³

Progress in labour

Once labour has been diagnosed then progress is expected within certain time limits. Regular examinations of the cervix are made, at least every four hours with conservative management, and every one or two hours with active management, so that the rate of dilatation can be plotted against time. Representation of labour progress in graphic form was first described in Germany between 1930 and 1950. Then, following the work of Friedman in the USA, the use of a cervicograph and its incorporation into a 'partogram' was reported from Africa by Philpott.⁴ Such composite records of labour began to be used in the United Kingdom from the early seventies and were soon complemented by a nomogram of expected progress as a result of work by Studd.⁵ The most widespread expectation for progress in labour is a rate of at least 1 cm dilatation per hour which derives from the slowest 10% of women reported by Philpott. With conservative management this expectation begins after the cervix has reached 3 cm; with active management this expectation begins when labour is diagnosed (even if only 1 cm dilated). Interventions to augment progress are more frequent in active management.

Cervicography should begin when the diagnosis of labour has been made. Friedman used the mothers' histories of the onset of contractions and so obtained the graphical impression of a 'latent' phase. Philpott and Studd used the time of admission to hospital. Active management uses their strict definition of labour once confirmed after admission. The higher numbers of vaginal examinations used in active management is because of the belief that early identification, and correction, of slow progress (dystocia) is fundamental in maximising the possibility of achieving a vaginal delivery.

Recognition of abnormal progress in 'early' labour continues to be confounded by the problem of identifying the onset of labour. The increasing use of active management by obstetricians interested in improving the outcome of labour management has resulted in it being considered medical 'interference' with the physiological, or 'natural', process of labour. Conservative management results in some 4–6% of women having regular and painful uterine contractions for more than the mean of eight hours, described by Friedman, before there is concern that the cervix has not dilated more than 3 cm.⁶ Recognition of an abnormal

latent phase requires a definition of labour which does not depend on reaching a cervical dilatation of 3 cm before labour can be diagnosed. A recent study of labours managed by the observational criteria of Friedman found that some (albeit less than 5%) nulliparous women experience a latent phase of more than 20 hours.⁶ However, with active management, less than 2% of primigravidae take longer than 12 hours to deliver.⁷ Active management also reduces by almost 50% the likelihood of a caesarean section due to dystocia (ineffective uterine activity).³ However, despite the evidence of benefit for women, active management of labour continues to be regarded by many midwives as unnecessary 'medical' intervention and forms the basis of a perceived power struggle between some midwives and obstetricians for 'control' of labour management.

Management of the second stage has undergone a change in recent years, particularly since the more widespread use of lumbar epidural. Once the cervix has dilated around the widest part of the fetal head (fully dilated; usually equated with 10 cm) progress is defined as descent of the presenting part through the birth canal. This is usually judged by hourly vaginal examinations. As the fetal presenting part passes the ischial spines and exerts pressure on the pelvic floor (levator ani muscles), the mother feels increasing pressure on the rectum and an increasing desire to push. This commonly occurs before full dilatation in multiparous women when a soft, compliant cervix dilates rapidly with each contraction. In such cases she will be asked to 'breathe through' the contractions until the head is visible at the introitus.

In the absence of an epidural the usual limits of duration for pushing are between 60 and 90 minutes for first labours, and 30 to 60 minutes for multiparous women. These recommendations have some support in that fetal hypoxia and acidaemia increase progressively during the second stage.⁸ However, in recent years, with the increasing use of epidural analgesia, more time in the second stage before pushing has been shown to improve the chance of vaginal delivery⁹ without a major deterioration in fetal pH.¹⁰ Nevertheless, the need for operative assistance remains significantly increased¹¹ and many consider one additional hour sufficient to minimise this possibility. Whether the ventouse or forceps are used, there is an increased risk of bruising and minor lacerations to the baby's head and face with operative delivery. However, a cephalhaematoma is more often the result of moulding of the fetal cranium during its passage through the pelvis.

Interventions during labour

There are two interventions designed to improve the rate of progress during spontaneous labour. These are rupture of the fetal membranes and intravenous infusion of oxytocin. Both procedures are widely used as methods for induction of labour and their use in spontaneous labour varies according to

definitions and expectations of normal progress. Their use began with the increasing desire to find methods of correcting slow progress in order to prevent prolonged labour.

RUPTURE OF MEMBRANES

One of the aspects of active management is the use of amniotomy within two hours if cervical dilatation is not evident following the diagnosis of labour.² Friedman¹ reported that the latent phase of labour was sensitive to change. It could be lengthened by sedation and shortened by stimulation. Rupture of the membranes stimulates labour and became a common procedure in early or mid labour to ensure that active labour, once began, continued.¹² However, routine rupture of membranes to prevent slow progress in labour was criticised by Caldeyro-Barcia *et al* after their large collaborative study on rupture of the membranes during labour.¹³ They showed that, in normal labour, membranes rupture spontaneously in only 66% of women before the second stage. They argued that normal, progressive, labour did not require routine rupture of the membranes – a point well accepted today. In half of the women in normal labour in their study membranes were ruptured at 4–5 cm cervical dilatation and, as expected, this significantly shortened the duration of labour. However, this was associated with:

- a higher incidence of caput succedaneum (34% compared with 5% in cases with intact membranes at delivery)
- more cases with disalignment between both parietal bones at the sagittal suture, and between parietal and occiput bones at the lambdoid suture (44% compared with 35% if membranes ruptured later)
- increased incidence of early uniform (type I) fetal heart rate decelerations (16% before and 46% after engagement of the head compared with 4% and 9%, respectively, if membranes remained intact)
- a significantly lower median umbilical artery pH at birth (7.25 compared with 7.31).

The delicate interaction of all aspects of labour was emphasised by Caldeyro-Barcia *et al*.¹³ The shortened duration of labour implied greater uterine pressures during contractions and, sometimes, an increase in the frequency of uterine contractions. Recent studies of fetal brain oxygenation have confirmed that uterine contractions of more than 1 in 2 increase the likelihood of desaturation of fetal cerebral blood.¹⁴ The changes in fetal heart rate may be associated with greater head compression, as evidenced by the greater degree of caput and moulding, or cord compression secondary to loss of amniotic fluid. Finally, the greater tendency to acidaemia, albeit within the normal range in their study, indicates the degree to which oxygen supply to the fetus may be interrupted by the increased uterine activity, cord compression, and a possible reduction in the intervillous space volume resulting from rupture of the membranes.

In a more recent, but smaller, study rupture of the membranes at 4 cm or less was

compared with rupture at 10 cm in normal labour.¹⁵ The effect of significantly shortening labour was confirmed (mean admission to delivery interval 4.9, SD 2.6, compared with 7.0, SD 3.7 hours). There were also fewer labours requiring oxytocin and there was a slightly greater number of normal vaginal deliveries. They found no effect on mean umbilical pH (7.22, SD 0.07) or numbers admitted to the neonatal unit. The data suggest that the effect of rupture of membranes on the fetus is a balance between the results of augmentation of the labour process and the reduced duration of labour.

EFFECTS OF OXYTOCIN

The role of natural oxytocin during spontaneous labour was not clear until it was recently shown that the pulse frequency increases. However, use of an intravenous oxytocin infusion became popular after work during the 1950s and 1960s showed that it was effective in increasing the rate of progress in slow labour and reducing the caesarean section rate for failed induction.² Induced labours, both in primigravidae and multigravidae, were shown to be significantly shorter than spontaneous labours but this was associated with a risk of uterine hypertonicity.¹⁶ The risk of uterine hyperstimulation with induction of labour relates to maintenance of the high dose of oxytocin necessary to initiate uterine activity compared with the lower dose necessary to maintain labour (less than 8 mU/minute) once established.¹⁷

Augmentation of spontaneous labour by oxytocin infusion aims to achieve optimal uterine activity which is usually assessed as a frequency of '3 in 10' minutes, and no more than '1 in 2'. Slow progress, assessed according to a nomogram of expected progress, is the usual indication provided there is no evidence of obstruction (malpresentation) or fetal hypoxia. Active management of labour uses a high dose oxytocin regimen (increasing from a rate of 6 mU/minute to 40 mU/minute every 15 minutes) to increase spontaneous labour in primigravidae who fail to progress at least 1 cm per hour within three hours of admission, but limits the total possible dose to 10 IU (diluted in 1000 ml 5% dextrose).

The effects on fetal oxygenation of an intravenous oxytocin infusion to the mother relate to the effect on uterine contractions. Uterine perfusion decreases during uterine contractions, resulting in a decrease in blood flow to the intervillous space.¹⁸ As long as the relaxation periods between contractions remain adequate, and sustained uterine tone is avoided, fetal bradycardia is not seen¹⁶ and fetal cerebral oxygenation remains unaffected.¹⁴ Nevertheless, in clinical practice use of oxytocin is associated with a greater incidence of fetal heart rate decelerations.¹⁹

Fetal monitoring in labour

The fetus responds to many aspects of labour and its management. Monitoring of fetal

wellbeing requires an understanding of placental function and, in particular, knowledge of the influence of pregnancy complications and labour on maternal–fetal gas exchange. During labour, observations are made on the amount and colour of the amniotic fluid, but the mainstay of fetal monitoring is the recording, either intermittently or continuously, of the fetal heart rate. Use of the fetal heart rate during labour requires an understanding of the physiological mechanisms which control fetal heart rate so that changes may be interpreted appropriately. Optimal interpretation of some changes in the fetal heart rate requires fetal blood sampling to determine the fetal acid base level.

AMNIOTIC FLUID VOLUME

A reduction in the volume of amniotic fluid is important for two reasons. Firstly, oligohydramnios present before postdates labour is a predictor for intrapartum fetal distress²⁰ and has come to be regarded as an indication of reduced placental function. Less effective placental gas exchange between contractions would therefore explain the risk of a more rapid development of fetal hypoxaemia and acidaemia during labour. Thus, continuous fetal heart rate monitoring is advocated and decelerations are more readily interpreted as an indication of clinically important fetal hypoxia.

Secondly, a normal volume of amniotic fluid offers some degree of protection against the many effects of uterine contractions. Maintenance of a fixed intrauterine volume during contractions means that the volume of the intervillous space is maintained and is not compressed against the fetus. Similarly, compression of the umbilical cord is avoided. Any reduction in uterine perfusion may be smaller during contractions with intact membranes as a result of isometric contractions of myometrium muscle fibres. However, as previously discussed, the stimulating effect of ruptured membranes on uterine activity inevitably means that subsequent contractions are likely to be more intense. A reduced volume of amniotic fluid found in early labour is associated with an increased likelihood of subsequent fetal heart rate decelerations and the greater possibility of fetal distress being diagnosed.²¹ The infusion of saline into the amniotic cavity during labour has been shown to decrease the incidence of fetal heart rate decelerations and may reduce the likelihood of caesarean section being considered necessary.

MECONIUM

The passage of fresh meconium (fetal bowel contents) into the amniotic fluid during labour is one of the traditional indicators of fetal distress and is associated with increased perinatal morbidity and mortality.²² However, meconium stained amniotic fluid before labour is found increasingly after 37 weeks of gestation, reaching an incidence of about 15–20% of pregnancies after 41 weeks. Meconium is often

found in the absence of other evidence of fetal distress at the time of caesarean section after prolonged labour, and yet it is rarely seen in situations of acute fetal distress such as placental abruption and cord prolapse. The mechanism by which the fetal bowel is stimulated to pass meconium is unknown.

Meconium is sterile but the importance of meconium in the amniotic fluid is the risk of neonatal meconium aspiration syndrome. The precise aetiology of this remains obscure although any associated fetal distress is more likely to predispose to perinatal morbidity if meconium is present. Oligohydramnios predisposes to meconium remaining thick and undiluted and may be an important determinant of any subsequent fetal distress in such circumstances. The passage of prelabour meconium may represent a previous transient fetal stimulation, possibly hypoxic, although infection and thyrotoxicosis are rare causes.

Meconium aspiration syndrome occurs in about 1% of cases with meconium present. Many labours with meconium present will remain uncomplicated and have a normal outcome. The risk for perinatal morbidity increases if the fetal heart rate becomes abnormal in the presence of meconium. Thus, meconium remains an indication for continuous monitoring of the fetal heart rate during labour, and the presence of meconium lowers the threshold for making a diagnosis of fetal distress if fetal heart rate abnormalities occur.

FETAL HEART RATE MONITORING

The value of fetal heart rate monitoring during labour is highly controversial. Differences of opinion are the inevitable result of the fact that there are few well conducted studies of this widespread technique. Many are still reluctant to accept that obtaining more data (by continuous, as opposed to intermittent, fetal heart rate monitoring) does not guarantee a better outcome for the baby. This is a reflection of the interpretation of the data and indicates limited understanding of the control of the fetal heart rate. Randomised trials have suggested that continuous fetal heart rate monitoring confers no advantage over intermittent auscultation for normal, uncomplicated, labours,²³ but this is more a condemnation of the way it is currently used. 'Pattern recognition' of changes in the fetal heart rate, as advocated by many early enthusiasts,²⁴ is no longer acceptable as the means of interpreting fetal heart rate changes during labour. Future studies of the value of fetal heart rate monitoring must await further understanding and acceptance of a more physiological approach to interpretation.²⁵

A prime example of the problem that exists because of previous lack of knowledge and understanding of fetal heart rate control is interpretation of changes in fetal baseline heart rate variability. It was not until the 1980s that the influence of fetal rest-activity behavioural cycles on intrapartum variability became fully recognised.²⁶ Low variability had generally been considered an indication of fetal distress

because of its association with suboptimal outcome in complicated pregnancies. As the use of continuous fetal heart rate monitoring became more widespread, associations between altered fetal heart rate patterns and suboptimal outcomes, initially described in complicated and high risk labours, were found not to be sufficiently strong to be helpful in normal and uncomplicated cases.

Episodes of low variability, often with an amplitude of less than five beats a minute, are associated with the quiet fetal behavioural state and the absence of movement related fetal heart rate accelerations.²⁶ Such physiological episodes do not normally last more than 45 minutes and so further tests of fetal wellbeing are appropriate after this. It has been known for some time that the only pattern not associated with fetal acidaemia is the presence of fetal heart rate accelerations during labour.²⁷ However, accelerations were also thought to be an abnormal pattern and no comment was made about this finding. It was not until later in the 1970s that normal fetal behaviour, during pregnancy was found to be associated with fetal heart rate accelerations. Recent interest in fetal stimulation has confirmed the absence of fetal acidaemia associated with a fetal response in the form of fetal heart rate accelerations.²⁸ However, the absence of fetal heart rate accelerations does not necessarily imply fetal acidaemia.

Fetal tachycardia, bradycardia, and excessive variation have traditionally been regarded as signs of fetal distress. Even in high risk pregnancies, none of these was found to be particularly predictive of fetal acidaemia.²⁷ Acute rises in fetal heart rate in labour probably reflect an increase in catecholamine secretion and indicate an early adaptive response to fetal hypoxia before the development of fetal acidaemia. Although chorioamnionitis should always be suspected if the maternal temperature increases during labour, recent studies have shown that epidural analgesia is a benign cause of maternal fever and associated fetal tachycardia.²⁹

Other associations with the use of epidural analgesia are fetal heart rate decelerations and a consequent increase in the frequency of fetal blood sampling. However, labours with an epidural which reach the second stage of labour were found to have a similar outcome in terms of mean umbilical artery pH and Apgar score at five minutes of greater than 7. Thus, fetal heart rate decelerations in such cases are probably more a reflection of adjustments to fetal cardiovascular control rather than an indication of the development of fetal acidaemia.³⁰

Fetal heart rate decelerations during uterine contractions undoubtedly represent interruption of maternal-fetal oxygen transfer sufficient to cause a transient bradycardia. In a healthy fetus with sufficient 'metabolic reserve' (a fetus not already adapted to longstanding placental insufficiency with its associated chronic hypoxaemia and possible malnutrition), the most likely mechanism to account for the fetal response is chemoreceptor stimula-

tion.³¹ The transient bradycardia is mediated by the vagal nerve and is associated with an increase in peripheral resistance and a rise in blood pressure. Many studies, mainly using the pregnant sheep model, have shown that fetal acidaemia occurs only after a considerable reduction in fetal oxygen delivery.³² Thus, fetal heart rate decelerations have a low predictive value for fetal acidaemia, particularly in the absence of associated complications or risk factors. However, an abnormal fetal heart rate may be the first sign of unrecognised placental insufficiency and may be associated with a faster rate of development of acidaemia.²⁵

FETAL SCALP BLOOD SAMPLING

Studies in the late 1960s and early 1970s, of the use of continuous fetal heart rate monitoring in the United Kingdom, followed soon after the introduction of the technique of fetal blood sampling during labour. First described in Germany in 1962 by Saling, blood sampling from the fetal scalp during labour allows microsamples of fetal blood to be obtained using capillary tubes. Initially, only pH measurements could be made readily on such small samples but complete blood gas measurements are now possible.

When continuous fetal heart rate monitoring was first introduced into clinical practice it was used in complicated cases in which it was becoming appropriate to consider 'elective' fetal blood sampling. It was shown that fetal blood sampling could be deferred in most high risk cases until an 'abnormal pattern' was seen on the fetal heart rate record.²⁷ The incidence of pH measurements below 7.25 varied with different patterns but was always less than 50%. Most abnormal patterns had a low risk of being associated with fetal acidaemia and this has been confirmed by many other studies. Fetal blood sampling is a difficult technique whereas continuous (electronic) fetal heart rate monitoring is relatively easy. Most obstetric units soon acquired electronic monitors. By the mid 1980s, consultant units were using electronic monitoring in a substantial proportion of labours,³³ although fewer than half the units had facilities for fetal blood sampling. The ease of obtaining a continuous fetal heart rate trace therefore resulted in the rapid uptake of electronic monitoring by most units; its recommended role as a screening test to determine the use of fetal blood sampling was ignored. However, the evidence from clinical trials clearly indicates an increased risk of caesarean section without fetal blood sampling and, recently, this has led the Royal College of Obstetricians and Gynaecologists to recommend that electronic monitoring should not be used without such facilities.³⁴

Fetal pH falls more rapidly during the second stage, associated with the pushing phase.^{8 25} Fetal heart rate patterns bear some relation with the rate of acidaemia: decelerations and prolonged bradycardia are associated with increased rates of fall in the pH. A prolonged bradycardia in the second stage requires delivery within 15 minutes to prevent

acidaemia at birth³⁵ as the pH may fall as fast as 0.1 units per 10 minutes.

UMBILICAL CORD BLOOD SAMPLING

Measurement of pH and blood gas values in blood from the umbilical cord immediately after delivery are an invaluable guide to the interaction between labour and the fetus. Umbilical venous blood values give an indication of the effects of placental gas transfer and correction of buffered arterial metabolic acids. Oxygen saturation and pH values are higher in venous blood which is the best representation of cerebral blood oxygen content. Umbilical arterial blood gas analysis has shown that the fetal response to labour is a varying degree of acidaemia in the blood supplying the lower body.²⁵ The range of fetal adaptation to labour is wide and umbilical artery pH values above 7.05 show no significant association with low Apgar scores³⁶ or adverse neurodevelopmental outcome.³⁷ Correction of such severe acidaemia after birth by healthy babies is rapid and correlates with a low PCO₂ at 1 hour of age.³⁸

The case for routine analysis of umbilical pH and blood gas measurements in clinical practice will not be accepted on cost effective grounds until it can be shown that the information made available has a beneficial influence on clinical management. Some have argued that it may be an appropriate medico-legal precaution that is particularly valuable to show absence of clinically relevant acidaemia at birth. However, the distribution of umbilical arterial and venous pH values is almost normal about a mean of between 7.2 and 7.25. Two standard deviations below the mean is about 7.05, and the possibility that babies delivered with pH values below this may be considered asphyxiated has medico-legal implications. The real difficulty is distinguishing between fetal response to labour and birth asphyxia as a consequence of management, particularly given the predisposition to fetal hypoxia associated with the use of oxytocin. As yet there is no simple diagnostic test for birth asphyxia.

Birth asphyxia

To establish conclusively birth related asphyxia, particularly when considering the possible aetiology of neonatal encephalopathy, it is appropriate to determine whether there is evidence that a greater than normal reduction in oxygen delivery to the fetus has occurred. The development of neonatal encephalopathy is not confirmation of birth asphyxia, but it may be one of the consequences. Birth asphyxia is therefore not a diagnosis but a term which describes the process and consequences of a severe disruption to maternal-fetal gas exchange. To be sure that such a process has occurred it has been suggested that particular criteria should be met³⁹: objective evidence of an interference with fetal oxygenation; and that the infant's condition at birth, neonatal course, and outcome are consistent with a disruption

of cerebral oxygenation. Clinical events such as placental abruption or cord prolapse clearly predispose to severe asphyxia neonatorum. Difficulties arise when labour, although not straightforward, has not shown clear evidence of intrapartum hypoxia (bearing in mind that the fetal heart rate is not diagnostic) and yet the baby develops an encephalopathy.

Many studies have shown the increased likelihood of longterm neurological damage after moderate or severe neonatal encephalopathy,^{40,41} but the absence of clear intrapartum antecedents in many cases suggests an increased vulnerability to labour.⁴² The need for more specific measures of maternal-fetal gas transfer in early labour are required to study this possibility.⁴³ Pre-existing placental dysfunction, particularly if unrecognised by conventional observations, means that normal labour can result in severe neonatal acidaemia. Repeated fetal blood sampling may show a rapid fall in pH in such cases, indicating reduced ability (reserve) to mount further normal adaptations to labour.²⁵ Uterine abruption, cord compression, and uterine hyperstimulation are accepted predisposing factors and an abnormal fetal heart rate in the presence of these factors should be a cause for concern. Fetal blood sampling may be indicated in some circumstances, particularly if the labour is prolonged. An objective measure of the degree of disturbance to cerebral metabolism, such as that obtained using magnetic resonance spectroscopy, is likely to prove helpful in the prediction of longterm neurological damage.⁴⁴

Summary

Although the management of so-called early labour remains a matter of some discussion, it is clear that the identification and correction of slow progress is more successful when undertaken early. Slow progress before reaching a cervical dilatation of 3–4 cm is still considered by some to be natural 'early' labour, despite the evidence that its prevention is associated with a significant reduction in the rate of caesarean sections. Rupture of the membranes is useful when progress is slow. It is also necessary if the liquor is to be seen or if a fetal scalp electrode is considered indicated. Oxytocin is also indicated if progress remains slow, even in the second stage of labour, after rupture of the membranes. Fetal heart rate monitoring during labour remains routine, but continuous, as opposed to intermittent, monitoring is increasingly restricted to complicated cases or labours at high risk of fetal hypoxia. Fetal heart rate abnormalities may indicate an increased risk of fetal hypoxaemia but most changes are not diagnostic. Fetal blood sampling is appropriate when the risk of acidaemia (secondary to clinically relevant fetal hypoxaemia) is, or becomes, significant. However, reliance on the fetal heart rate to determine the need to assess accurately maternal-fetal gas transfer by fetal blood sampling continues to limit the detection of reduced 'fetal-placental' reserve during

labour. The distribution of umbilical pH values at birth clearly represents the wide spectrum of fetal response to the effect of uterine contractions on oxygenation.

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