FETAL MONITORING in Practice

FOURTH EDITION

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PREFACE TO THE FOURTH EDITION

We were delighted to be asked to produce the fourth edition of our book, which was originally published 24 years ago. According to the recent NHSLA report the number of unfortunate obstetric incidents has reduced but the value of claims has increased and currently more than one million pounds is paid out each day in compensation in the UK. The NHSLA report reveals avoidable factors such as difficulty with cardiotocograph interpretation, failure to incorporate the clinical situation, delay in taking action and poor communication as key factors, the same ones that were revealed in the 1990s.

Attempts have been made to use computer technology for assistance in decision making. However, neither the recent INFANT study, with 46 000 pregnancies that involved computer analysis of CTG, nor the FMALERT study, which used computer technology to help with CTG and ECG interpretation, have shown any improvement in clinical outcome. This then leaves us to focus on more systematic education and assessment of practitioners on a regular basis if they are to practice in delivery units. Those who do not reach adequate standards on assessment could use the 'second eye' or 'fresh look' principle and ask a colleague to review the suspicious or abnormal CTGs at regular intervals till such time they are assessed to be competent. NHS England has recommended education and competency assessment in CTG interpretation to avoid stillbirths.

We have retained most of the case examples and basic principles in interpretation as readers have found them useful. The recently released NICE and FIGO guidelines on CTG interpretation have been incorporated to provide readers with information that could be useful. We are grateful to the distinguished authors who have contributed valuable chapters for this edition of the book. They have covered issues on litigation, incorporation of clinical scenarios with CTG interpretation and the need for education and assessment. A book or day of lectures is not adequate to maintain knowledge and skill of CTG interpretation and clinical action. This will need continuous learning by case reviews. We hope that this book will provide the basic knowledge to assist front-line staff in the task of CTG interpretation in different clinical scenarios.

We must continue to climb that mountain together to achieve better care for mothers and babies. We always welcome feedback and suggestions from readers.

September 2016

Donald Gibb
Sabaratnam Arulkumaran

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The editors are most grateful to our distinguished colleagues who contributed chapters for this edition of the book and to the publishers who have helped us enormously to bring out this edition. Our families deserve special mention for letting us continue with our passion of editing this book.

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INTRODUCTION

Donald Gibb

No written records of the detection of fetal life exist in western literature until the 17th century. Around 1650 Marsac, a French physician, was ridiculed in a poem by a colleague, Phillipe le Goust, for claiming to hear the heart of the fetus 'beating like the clapper of a mill'. It was not until 1818 that Francois-Isaac Mayor of Geneva, a physician, reported the fetal heart as audibly different from the maternal pulse heard by applying the ear directly to the pregnant mother's abdomen. Laennec, a physician working in Paris around 1816, was the father of the technique of auscultation of the adult heart and lungs. Le Jumeau, Vicomte de Kergaradec (Fig. 1-1), also a physician working with Laennec, became interested in applying this technique to other conditions including pregnancy. John Creery Ferguson, later to become first Professor of Medicine at the Queen's University of Belfast, visited Paris and met with Laennec and Le Jumeau. On his return to Dublin in 1827, Ferguson was the first person in the British Isles to describe the fetal heart sounds. He influenced Evory Kennedy, assistant master at the Rotunda Lying-in Hospital in Dublin, who wrote his famous work entitled *Observations on Obstetric Auscultation* in 1833.[1] There was much argument over the technique of listening, some demanding the use of the stethoscope for reasons of decency only. At that time, some doctors examined pregnant women through their clothing and this respect for the modesty of the woman must have inhibited the spread of obstetric auscultation. In 1834 Anton Friedrich Hohl was the first to describe the design of the fetal stethoscope (Fig. 1-2). Depaul modified this (Fig. 1-3) describing both in his *Traite D'Auscultation Obstetricale* in 1847.[2] Although Pinard's name is most commonly associated with the stethoscope, his version followed several others, appearing only in 1876. Many papers were subsequently published in a variety of languages elaborating the technique. In 1849 Kilian proposed the 'stethoscopical indications for forceps operation' – stating that the forceps must be applied under favourable conditions without delay when the fetal heart tones diminish to less than 100 beats per minute (bpm) or when they increase to 180 bpm or when they lose their purity of tone.[3] Winkel, in 1893, empirically set the limits of the normal heart rate at 120 bpm to 160 bpm. This has been carried forward for many years and reviewed

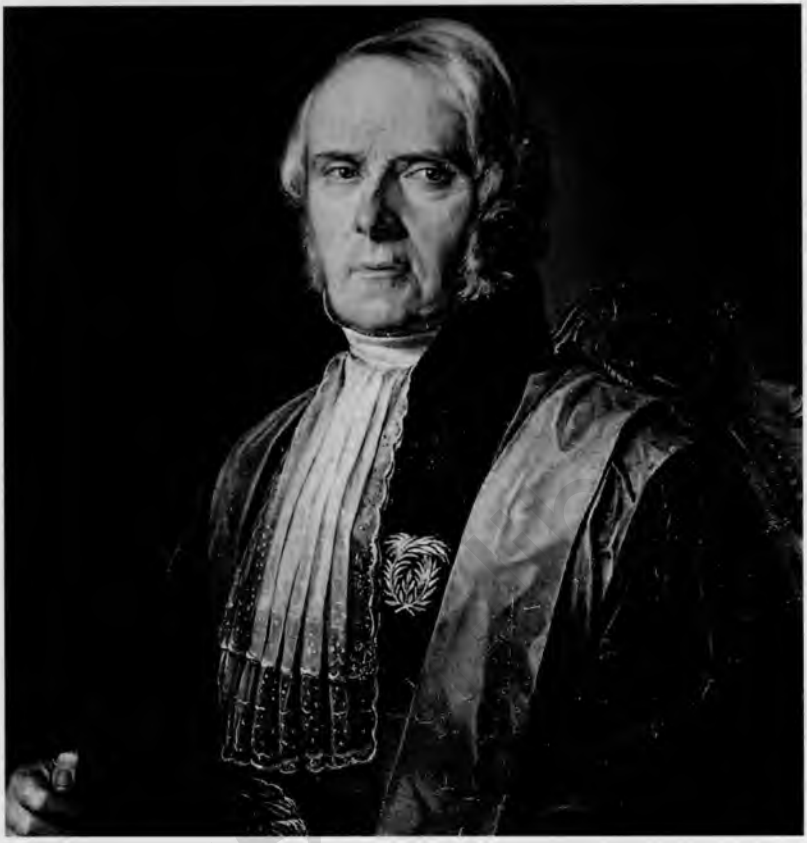

Figure 1-1. Jacques Alexandre de Kergaradec, robed as a Membre de l'Academie de Medicine Paris. *(With thanks to late Professor J. H. M. Pinkerton, Emeritus Professor of Midwifery and Gynaecology, Queen's University of Belfast.)*

in the light of the large amount of material produced by electronic recording.

If hearing the fetal heart was of any value then it was recognized that this was based on a very small sample of time and subject to considerable observer variability. Listening for 15 seconds in 1 hour is to sample only 0.4% of the time. More continuous monitoring may be desirable. The advent of audiovisual techniques associated with the development of the film industry in the early 20th century set the scene for technological developments that led to the equipment we have today. In 1953, while working in Lewisham Hospital, south-east London, Gunn and Wood reported 'The amplification and recording of foetal heart sounds' in the *Proceedings of the Royal Society of Medicine*.[4] In 1958, Hon pioneered electronic fetal

Figure 1-2. The Hohl fetal stethoscope. *(Wellcome Institute Library, London.)*

Figure 1-3. The Depaul fetal stethoscope. *(Wellcome Institute Library, London.)*

monitoring in the USA. Caldeyro-Barcia in Uruguay and Hammacher in Germany reported their observations on the various heart rate patterns associated with so-called fetal distress. This set the scene for the production of the first commercially available fetal monitor by Hammacher and Hewlett-Packard in 1968, soon to be followed by Sonicaid in the UK. It is notable that Saling in Berlin had reported the use of fetal scalp blood sampling to study fetal pH 2 years prior to this in 1966. Fetal scalp blood pH assessment was developed in parallel with electronic monitoring, not as a sequel to it as might be assumed from our current practice.

The early equipment used phonocardiography, simply to listen and record sounds coming from the maternal abdomen as well as generating the fetal heart rate from the fetal electrocardiograph (ECG) from a fetal scalp electrode. Phonocardiography produces inferior traces because of the other extraneous sounds that confuse the picture. This problem was solved very quickly by the introduction of Doppler ultrasound transducers. When the Doppler transducer is applied to the maternal abdomen a Doppler signal is reflected from the moving fetal heart, the location of which has already been determined by auscultation. The signal is altered by a moving structure according to the Doppler shift principle and received by the transducer in its altered form. The moving structure is usually the moving heart and the blood flowing through it. Ultrasound Doppler technology has improved considerably in recent years and the latest generation of monitors produces excellent-quality external traces, comparable to those generated by direct ECG. The previous justification – that rupture of the membranes and application of a fetal electrode are necessary in order to generate a good-quality trace – is no longer valid. This improvement can be largely attributed to the technique of autocorrelation or dual autocorrelation and the use of wide beams. Monitoring of both twins externally has presented problems because of interference between the two Doppler beams. That has been solved in the latest equipment by the use of two different frequencies, or the same frequency but distinguished by position using ultrasound 'windows' in the two ultrasound transducers so that the beams do not interfere with each other. The direct fetal ECG can be obtained by an external or internal technique. The external technique is used only in a research situation because the signal has to be electronically cleaned so as to remove the maternal ECG and electrical activity from the anterior abdominal wall. Direct detection of the fetal heart rate from a fetal electrode applied to the fetus at vaginal examination is used in clinical practice. This is commonly called a scalp electrode, but is better termed a fetal electrode in view of its possible application to the breech. All machines

provide an external tocography facility through a relatively simple strain gauge transducer. It should be appreciated that this provides only an indirect assessment of the uterine contractions. It indicates the frequency and duration of contractions, but little about actual pressure or basal tone. In the unusual situation of requiring direct data about the intrauterine pressure, an intrauterine catheter is necessary with the relevant option in the machine. However, the climate of childbirth has retreated from the excessive use of invasive technology and the role of internal monitoring has become much more limited.

The clinical needs should be assessed and the specification of the machine required determined accordingly. A monitor to be used for antenatal monitoring does not require the intrapartum options and is therefore less expensive. Most modern monitors have similar specifications. The specification of a top-of-the-range intrapartum monitor is shown in Box 1-1.

Some hospitals have introduced electronic monitors including ECG waveform analysis (STAN technique). These have produced interesting data, but it is essential that staff using them should be fully trained in the technique. One effect of their use has been a reduction in the need for fetal scalp blood sampling.

Antepartum monitors are smaller and less costly. Monitors used for antenatal assessment need not have all the specifications of an intrapartum monitor. There is some evidence that a computerized interpretation system may assist in antenatal assessment. Even if this is the case, we should not give up on the best computer by far: the human brain!

Box 1-1. Specification of intrapartum monitor

- Reliable
- User friendly with operating manual and video
- Robust with customized trolley
- Fetal heart rate by external Doppler ultrasound (US) with autocorrelation
- Fetal heart rate by fetal electrode (ECG)
- Twin monitoring US and ECG
- Twin monitoring US and US
- Maternal heart rate
- Event marker
- External tocography
- Internal tocography as an option
- Mode, date and time printout
- Keypad as an option
- Automatic blood pressure, pulse and SaO_2 facility (an option selectively for high-risk labours)

Hand-held Doptones now include a digital display of the heart rate. Some models are waterproof for use in the water-labour scenario. Low-cost printers that can be attached to such devices are being developed. Such systems offer exciting possibilities to countries that have not yet started on the troublesome journey of extensive electronic fetal monitoring. They must be helped to avoid the costly mistakes made by the more developed countries. Technology should be appropriate, low cost and high quality.

Telemetric transmission of the cardiotocograph (CTG) has become more practicable with improved technology in recent years, allowing the woman to remain mobile in labour. However, more selective use of the technology has meant that some of the women who had telemetric monitoring do not actually require continuous electronic monitoring. The use of mobile epidural anaesthesia and of water pools has rekindled interest in this kind of technology. It is very reassuring during a labour in water to be able to listen to and record the fetal heart after a contraction using telemetric technology. We can increase our confidence in water birth.

A solid trolley is an important investment to protect the machine during its busy life in the clinical area. Servicing, back-up and supplies of paper and electrodes must be assured. Modern machines have been factory tested to ensure proper functioning in any climate in the world. They are designed to be used 24 hours a day, 7 days a week. Although the concept of rest and recovery is valid for human beings, it is not necessary for such machines! The electronic clock timings are battery dependent and require adjustment with time changes in the autumn and spring. CTG timings are important in record keeping.

An important step is to identify the midwifery and technical staff who will be responsible for day-to-day supervision and maintenance of this equipment. It is uncommon for such equipment to develop technical faults, and defects will more often be user related. Simple housekeeping and in-service education will pay dividends. Fairly simple instructions sometimes not given due attention include not putting jelly on the tocograph transducer, not breaking the plugs by using push–pull rather than screw action, being careful that transducer cables are not run over and broken by trolley wheels, and ensuring the use of the correct paper the right way round. The less frequent need to use a scalp electrode should not be forgotten, and these electrodes should remain readily available in the delivery area. An expensive piece of equipment requires common-sense care. It is a pity if equipment is out of action because of user errors.

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CLINICAL ASSESSMENT AND RECORDING

Donald Gibb, Sabaratnam Arulkumaran

The journey of birth is a challenging one for any individual. We should think of the baby in the womb as an individual to be observed and examined, not just as a bump in the mother's abdomen that we feel and listen to. We must develop an appreciation of fetal life and existence. When we perform an ultrasound scan we see the baby moving its limbs, yawning, smiling, hiccoughing and passing urine to make amniotic fluid. When we examine the mother's abdomen and listen to the fetal heart we must think this way. What is the baby doing: is it awake or asleep, is it sick or healthy, is it happy or sad? A moving baby, a hiccoughing baby, a big baby is generally healthy and happy. We must consider the reasons why the baby may not be healthy using the fetal heart pattern as contributory evidence, not as an end point. The process of labour and birth is a challenge for the fetus. The fetus, particularly its head and sometimes its cord, is going to be squeezed by contractions every few minutes increasingly until birth. This is similar to an adult or child having their head pushed below the water when swimming every few minutes. An individual needs to be healthy with good reserve in order to do this. At the beginning of labour the healthy fetus is like a child running in a field and throwing a ball. We must not allow it to become sick and damaged. Much attention has been focused recently on the number of late stillborn babies in the United Kingdom. All are tragic; some occur before labour and some during labour.

The part of the birth journey with which we are particularly concerned is that of labour and delivery. The concept of preparation is an important one and, for our purposes, we consider this journey to start with admission to the labour ward. When we prepare for a journey we ensure that we are in good health, our vehicle is in good condition, the roads we will drive on are safe and that we have a good insurance policy. Admission to the labour ward is the time for such a review of the pregnant mother. Intrapartum events are a continuum of antenatal events. Many babies who get into difficulty during labour have already become compromised in the antenatal period and our surveillance system must be designed to find those fetuses and ensure their safe delivery. Assessment on admission helps us to look carefully for high-risk factors that were previously undetected or new factors that have since appeared.

On admission to the labour ward the history is summarized taking particular note of high-risk factors. These may be socioeconomic such as young age, poor socioeconomic status and substance abuse, or individual such as previous perinatal loss, previous or existing intrauterine growth restriction (IUGR), bleeding in pregnancy, diabetes mellitus, reduced fetal movements and a variety of other markers. Breech presentation and multiple pregnancies are obvious high-risk factors. Listen to the pregnant woman; in studies of stillbirth the woman will often feel that something is wrong but complain that she has not been listened to by the staff. On examination, general features such as height, weight, blood pressure, temperature and signs of anaemia are reviewed. Before proceeding to vaginal examination, abdominal examination must be complete. We still miss the breech baby. Is the 'deeply engaged' head actually a breech? We hope to avoid the embarrassment of removing the mother from the water pool when the baby is found to be breech in mid labour. Abdominal examination includes a measurement of abdominal size, an estimate of fetal size, lie, presentation and station of the presenting part. The nature of the contractions, amniotic fluid volume estimation and auscultation of the fetal heart complete this procedure. Traditionally the size of the abdomen and fetus is assessed subjectively. The value of formalizing this with an objective value has been suggested in recent years.[1] A measure of the fundosymphysis height (FSH) in centimetres (Figs 2-1 and 2-2) provides a guide to fetal size so long as the observers have been trained in the technique.[2,3] The fundus should not be actively pushed down during the palpation, and the height from the top of the fundus (without correcting the uterus to the midline) to the upper margin of the symphysis pubis should be measured. Ideally a blinded measurement using the blank side of a tape measure is desirable. Due attention should be paid to the possible confounding factors of

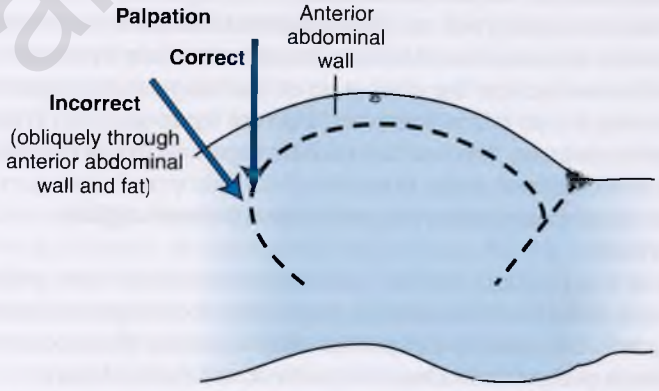

Figure 2-1. Detecting the fundus for FSH measurement.

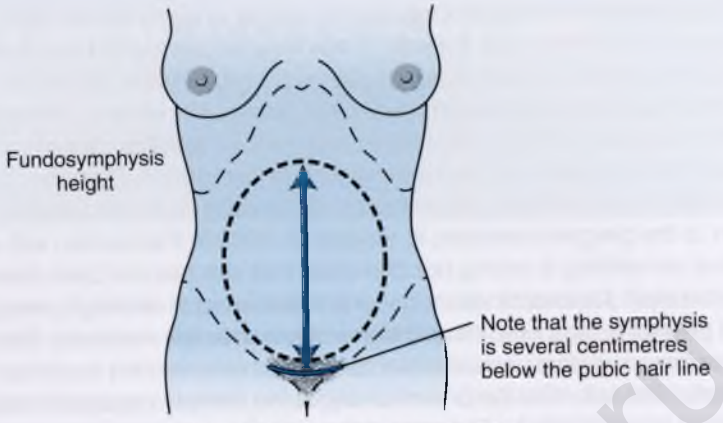

Figure 2-2. Measurement of FSH.

obesity, polyhydramnios, fibroids or unusual physical characteristics of the mother. After 20 weeks' gestation the FSH should be equivalent to the gestational age in centimetres ±2 cm up to 36 weeks, and ±3 cm after 36 weeks. No test should be subjected to unrealistic expectations. A tape measure is cheap, available and reasonably reliable with little inter- or intraobserver variation.[4] We are not good at identifying small babies in utero; this is obvious from studies of adverse perinatal outcome. The reduced fundosymphysis height may indicate a small fetus who may be suffering from chronic asphyxia (intrauterine growth restriction; see Ch. 7). Such a fetus is more likely to develop an abnormal heart rate pattern before, and particularly during, labour. A suspicion of a large fetus is also important so that we can anticipate and prepare for mechanical problems. A history of big babies, shoulder dystocia and diabetes mellitus are all important indicators. A rewarding exercise is recording the estimated fetal weight on the partogram. With experience and regular practice this becomes reliable. Management may be altered if abnormal labour progress becomes manifest and there is a likelihood of cephalopelvic disproportion. Marking 'beware shoulder dystocia' in the 'special features' box on the partogram of women carrying large babies, and especially those with a history of shoulder dystocia, is an important preventative measure. Medical help will be organized to be readily available in the second stage of labour.

Abdominal examination is performed before vaginal examination.

Vaginal examination may be undertaken after abdominal palpation. Progressive changes in the uterine cervix permit a diagnosis of labour to be made in the presence of painful uterine contractions occurring at least once every 10 minutes (min) with or without a show or spontaneous rupture of the membranes. This is an important diagnosis.

Without it the mother will not be kept in the labour ward with the likelihood of ill-advised intervention. In this situation the best decision is often to do nothing rather than to do something. Inexperienced medical staff members sometimes seem to feel an irrational pressure to intervene. Antenatal education should include an objective of the mother not admitting herself to hospital too early in labour. Some hospitals send a midwife to perform an assessment at home. At this stage the contractions are likely to be at least one in every 5 min and are quite painful. If there has been spontaneous rupture of the membranes without labour being present (prelabour rupture of the membranes) then digital examination should not be performed unless a decision has already been taken to proceed to delivery. Umbilical cord compression can be excluded by running a strip of fetal heart rate (FHR) tracing without recourse to digital examination. A speculum examination may help to identify leaking amniotic fluid; however, taking relevant swabs for microbiological examination does not require speculum examination. The colour of any amniotic fluid should be recorded.

In all cases, whether labour is becoming established or not, an admission assessment of the fetal health should be considered. It can be done by an 'admission cardiotocogram (CTG)' (see Ch. 8) or by 'intelligent auscultation'. The mother should be asked about her wellbeing, followed by that of her fetus by enquiring about fetal movements. The last time she felt the fetal movements should be recorded. Palpation for fetal movements by the mother and midwife should be undertaken following the auscultation to determine the baseline FHR. The time at which the mother and the observer felt the fetal movements should be recorded and the opportunity taken to listen to the FHR to observe FHR acceleration. Palpation should be continued to recognize a contraction and to listen soon afterwards to detect any FHR deceleration. The advantage of auscultation as described is that a fetus that had accelerations can be monitored subsequently by auscultation of the FHR, as the baseline rate is likely to rise with fetal hypoxia, and decelerations may be heard soon after contractions suggesting the possibility of fetal hypoxia. The admission CTG may be of benefit if the mother did not have adequate antenatal care or one-to-one midwifery care is not possible. The mother may ask, 'is my baby alright?', and this is best answered with an admission CTG. After this clinical review a decision can be taken about the application of appropriate technology for the rest of the labour. This may consist of mobilization and intermittent monitoring by auscultation (low risk), continuous electronic monitoring (high risk) or, most commonly, a sequential combination of both. Full information should be provided to the woman and her wishes carefully considered. It should be emphasized that, in every case when electronic monitoring

is not being performed, then skilled, careful, intermittent auscultation is undertaken every 15 min for 1 min in the first stage and every 5 min in the second stage soon after a contraction.

All observations are then plotted on a partogram, as shown in Figure 2-3. These should be tailored to the individual case. Maternal temperature should be checked 4-hourly when the previous recording has been normal. Pulse rate and blood pressure are recorded every hour when the previous observations have been normal with no protein

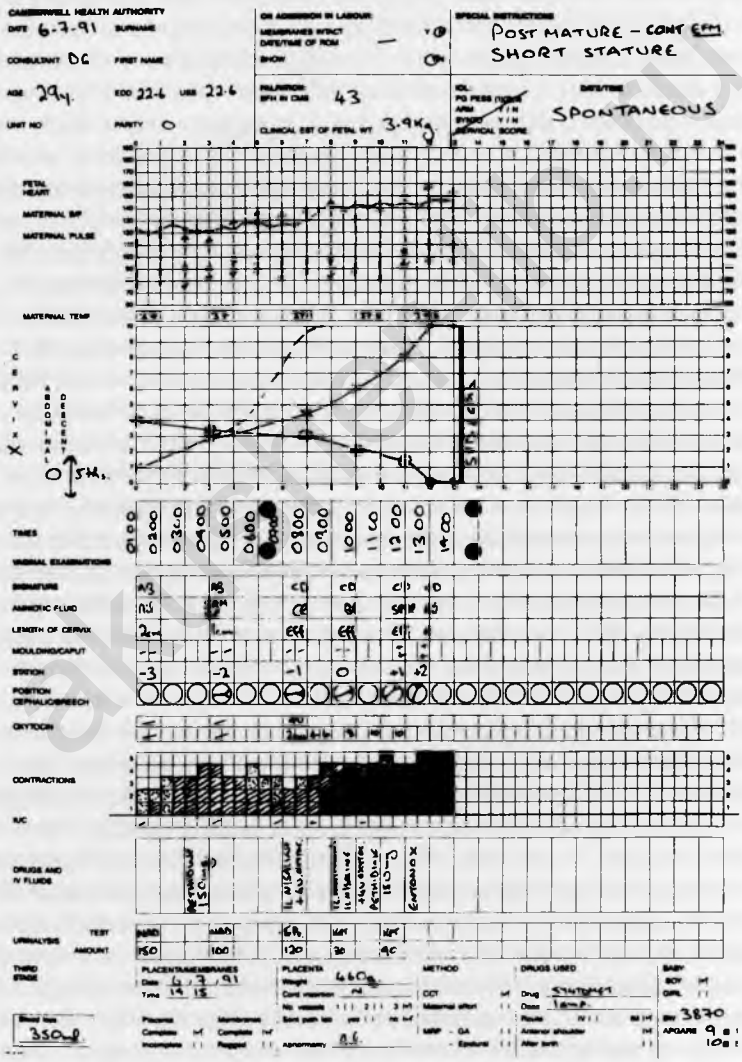

Figure 2-3. Partogram. *(Courtesy of King's College Hospital.)*

detected in the urine. Abdominal palpation is recorded every 4 hours prior to a vaginal examination. Each new observer should ensure the baby is not an undiagnosed breech. Expect the unexpected: breech babies are sometimes diagnosed only after the cervix has opened and previous examiners have thought that the baby was cephalic. Urine is tested for ketones, protein and glucose whenever it is produced. The programme of observations should not be rigid and will vary depending on the clinical situation.

The admission assessment is particularly important with a view to undertaking safe intermittent, limited electronic monitoring. The mother's degree of risk may change from low to high; however, indications will usually be present. A normal admission CTG in a mother who, on history and examination, is low risk assures a healthy fetus for the next 4 hours unless one of four events supervenes:

1. placental abruption
2. umbilical cord prolapse
3. injudicious use of oxytocics
4. imprudent application of instruments.

Placental abruption is characterized by pain, anxiety, tachycardia and often bleeding; a good midwife or doctor should suspect and detect it. It is estimated that one in five cases may have minimal or no symptoms and the condition is diagnosed retrospectively.[5] Umbilical cord prolapse occurs after rupture of the membranes with a high presenting part. Good midwifery and medical practice should detect this early on when it occurs in the labour ward and the outcome for this condition is excellent when properly treated. The proper use of oxytocics and appropriate electronic monitoring (see Ch. 10) and the proper use of instruments are promoted by education and training. Death of a normally formed term fetus within 4 hours of a normal CTG is a rare event, but certainly can occur with a serious placental abruption for which there may be no warning sign. A fetus can die of placental abruption within 15 min of a normal CTG.

The importance of clinical sense cannot be overemphasized. Figure 2-4 shows a 'complete' CTG machine including an accompanying tape measure and fetal stethoscope. Why is the fetal stethoscope needed? The CTG shown in Figure 2-5 was undertaken in a mother admitted to hospital complaining of reduced fetal movements. The fetal stethoscope was not used and the ultrasound transducer was applied directly to the maternal abdomen. The mother was reassured that the baby was healthy; however, a macerated stillbirth occurred 1 hour later. The heart rate that had been picked up was the maternal pulse from a major vessel, the ultrasound beam having passed through the dead fetus. The mother had a tachycardia on account of her anxiety. Figure 2-6 is the trace obtained when the mother was

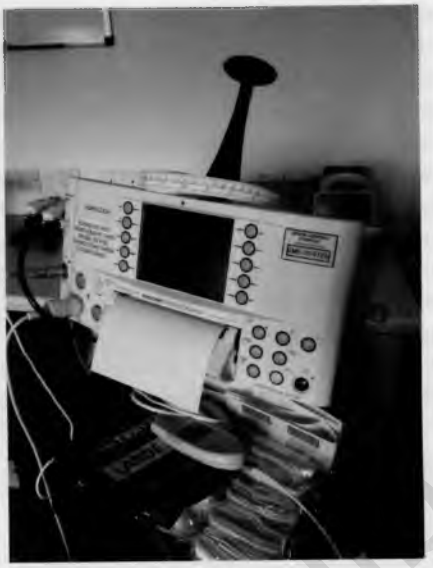

Figure 2-4. 'Complete' fetal monitor.

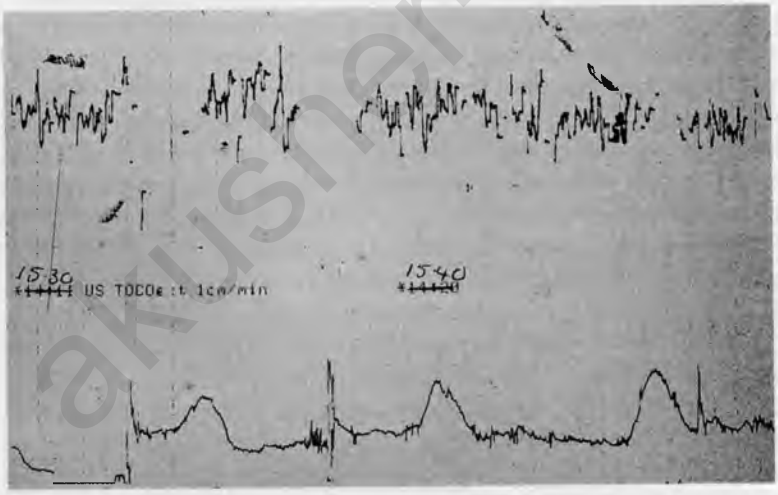

Figure 2-5. CTG of dead baby – ultrasound.

admitted draining thick meconium and a scalp electrode was applied with some urgency. The midwives were reassured by the trace, but the baby was born shortly thereafter as a macerated stillbirth. It was growth restricted and had died of hypoxia some time before. On account of oligohydramnios the fetal buttocks were in contact with the fundus, which in turn was in contact with the diaphragm and the path of transmission of the maternal electrocardiograph (ECG) through the fetus is clear. The scalp electrode may therefore capture the maternal

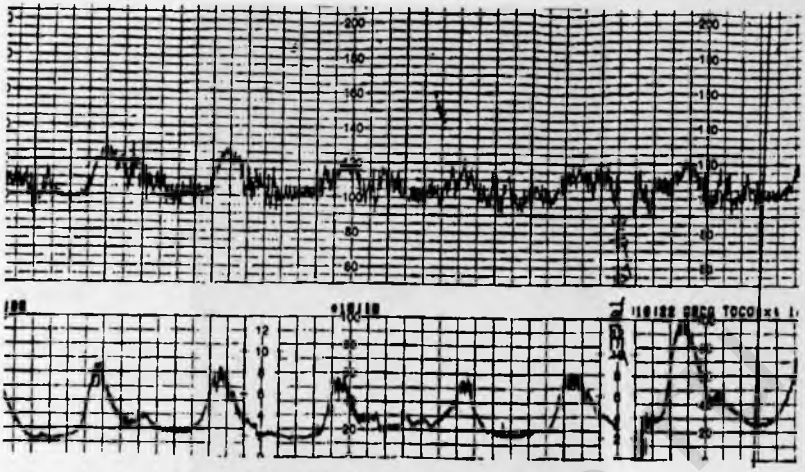

Figure 2-6. CTG of dead baby – fetal electrode.

ECG when the fetus is dead. The stethoscope must always be used to establish a fetal pulse different from the maternal pulse, although this advice is now superseded if the mother's heartbeat is also routinely monitored electronically. This is facilitated by modern monitors that have this ability. Some companies have introduced a mechanism that incorporates infrared sensors in the tocograph transducer ('smart pulse' – Fig. 2-7), which can detect the superficial vessel pulsations and provide a trace of the maternal heart rate (Fig. 2-8).

Figure 2-9 is the trace obtained from another mother who attended not in labour but rather complaining of reduced fetal movements. The midwives applied a Hewlett-Packard 1350 fetal monitor, which included a fetal movement detector in the ultrasound transducer. The black lines in the middle of the trace indicate movements. The mother returned some hours later and delivered a macerated stillbirth. The ultrasound had again picked up the mother's pulse but, more worryingly, the movements detected were not fetal movements but rather maternal intestinal activity or some other maternal movement. It should be noted that adult heart rate recordings also show baseline variability and accelerations. Prolonged accelerations at the time of uterine contractions and increased variability are characteristic of the maternal heart rate in the second stage when the mother has uterine contractions and she is bearing down.[6] An increasingly recognized mistake occurs when the monitor records the maternal heart rate with accelerations (Fig. 2-10) instead of the FHR with decelerations that should be seen with head compression. This may obscure a trace that would be showing a prolonged deceleration requiring delivery. Proper clinical application and relating the FHR patterns carefully to contractions should help us to avoid these tragic pitfalls. The mother's pulse rate

Figure 2-7. The tocographic transducer ('smart pulse'), which consists of two infrared sensors to detect superficial maternal vessel pulsations.

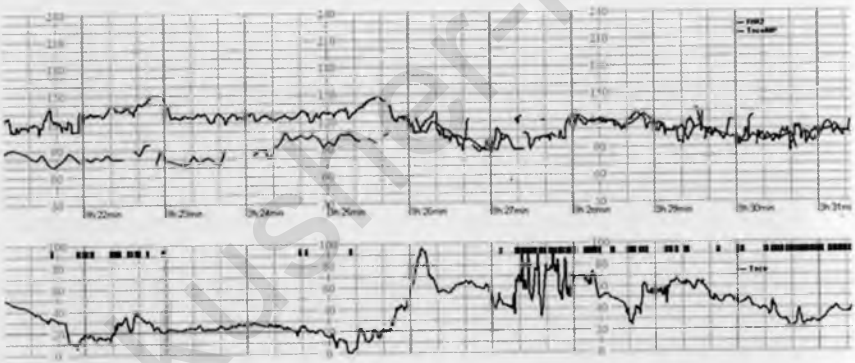

Figure 2-8. A CTG tracing that shows the maternal and fetal heart rate simultaneously using the 'smart pulse' tocographic transducer.

should be correlated to the FHR and annotated at the beginning of the trace. The Medical Devices Agency in the UK advise that the fetal heart should be auscultated prior to the monitor being used, because the maternal heart rate may be detected. The maternal heart rate may be the same as the FHR, or it may be doubled and at times increased by 50% based on the number of maternal pulses picked up and whether they are picked up continuously or intermittently.

Figure 2-11 illustrates the correct use of the 'kineto cardiotocograph' on the Hewlett-Packard 1350 monitor, showing the physiological truth of the relationship between true fetal movements and acceleration of the fetal heart rate. Figure 2-12

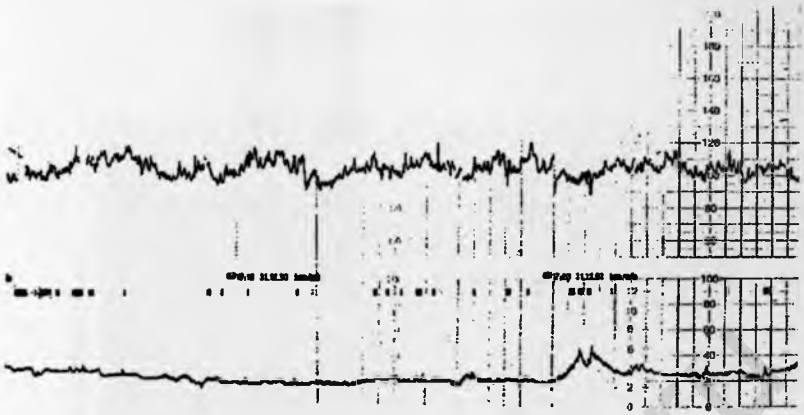

Figure 2-9. CTG of dead baby – ultrasound with fetal movement profile.

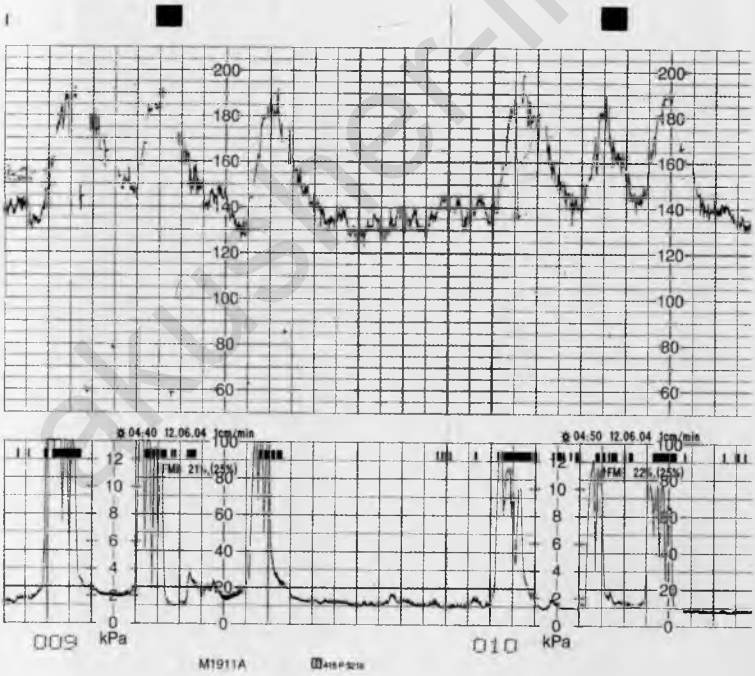

Figure 2-10. Recording in the second stage of labour – the monitor is recording the maternal heart rate, which is increasing with uterine contractions and bearing-down efforts with increased baseline variability, instead of exhibiting the typical head compression FHR decelerations.

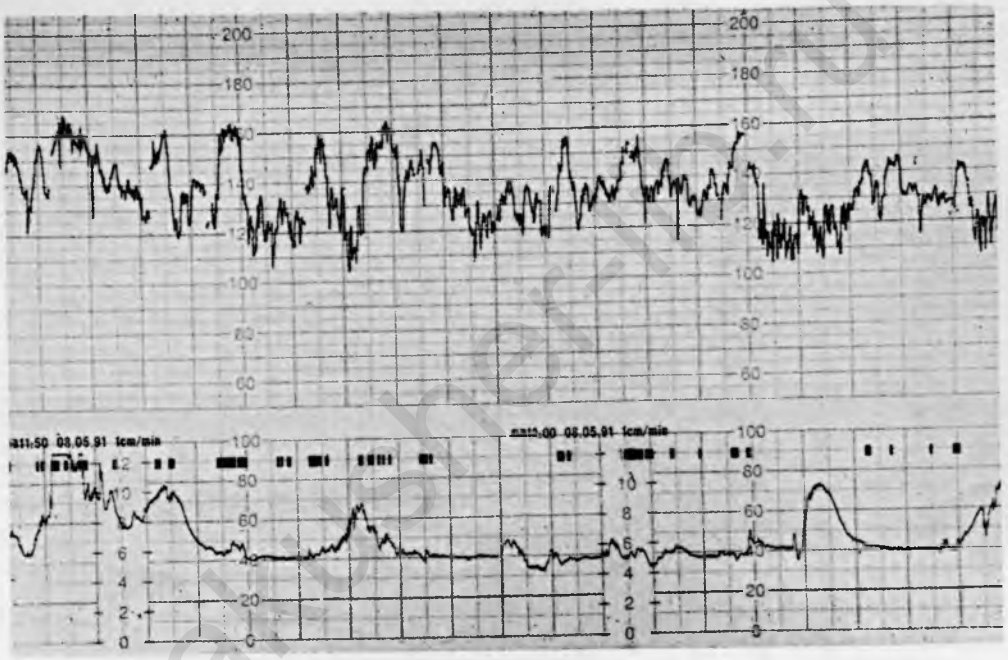

Figure 2-11. Kineto cardiotocography – relationship between fetal movements and accelerations of FHR.

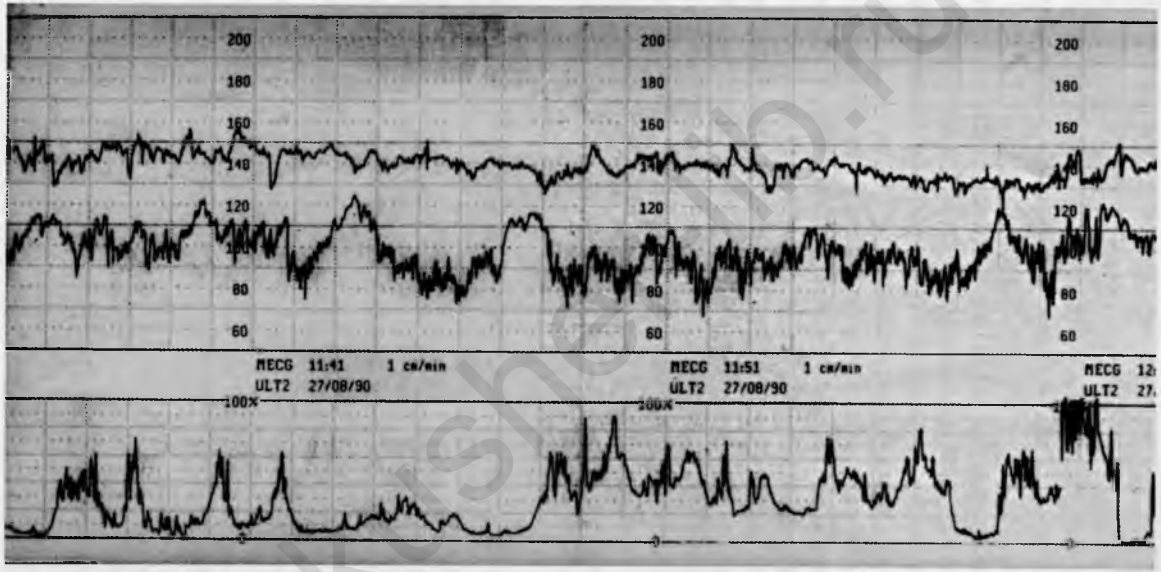

Figure 2-12. Maternal continuous heart rate showing accelerations and good baseline variability along with FHR.

shows the correct use of maternal continuous heart rate recording, as is now available on all intrapartum fetal monitors, demonstrating clearly that, unsurprisingly, the adult heart shows accelerations and baseline variability. Understanding this should reduce confusion in distinguishing one heart rate from the other. The use of such a facility is ideally suited, but much underused, in the scenario of managing preterm labour with beta-sympathomimetic therapy.

The importance of clinical sense cannot be overemphasized.

Incidentally, Figure 2-5 also shows a common day-to-day error: the incorrect setting of the clock mechanism recording the time on the trace. This may be user error, which is particularly frequent after a seasonal time change, or the batteries in the machine may be running low. It should be very simply corrected.

Good communication with the mother and her partner is vital. Obstetric cases are unique in that patients are not sick, as are those in all other departments of the hospital. On the contrary, they are experiencing one of the most important events in their lives with enormous emotional impact. The intimacy of this should not be compromised except in the 'genuine interest' of safety for mother and child. This book should help us to recognize this genuine interest. We are in a position of great privilege to assist families in one of the most important events in their lives. We must fulfil our role with diligence, care and compassion.

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AUSCULTATION OF THE FETAL HEART RATE

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Auscultation of the fetal heart refers to the technique of listening and determining the fetal heart rate (FHR) as beats per minute (bpm). Determination could be either by listening and counting the heartbeats for 1 minute or by listening for a shorter period and then multiplying by a factor to calculate the heart rate. Counting for one full minute provides a more accurate rate compared with a shorter period and multiplying to get the rate for a minute. Though modern and sophisticated systems are available to monitor the FHR and its pattern, auscultation of the FHR is still an integral part of antenatal and intrapartum monitoring of a pregnant woman and is a necessary skill for carers (healthcare providers/healthcare professionals).

HISTORY OF FETAL HEART SOUND AUSCULTATION

Though in the 1600s the sounds of the fetal heartbeat were recognized, the initial interest and use of ausculatation was supposedly to determine the viability of the fetus.

This interest gradually grew and resulted in publication of a book on *obstetric auscultation* by Evory Kennedy in 1833 (see Ch. 1).[1]

The initial need for an instrument to listen to the fetal heart through the maternal abdomen was to overcome the embarrassment of placing the ear on the abdomen. The rolled sheet used to listen to the adult heart was soon modified into a wooden instrument and used to listen to the fetal heart through the maternal abdomen.

TECHNIQUE OF FETAL HEART AUSCULTATION

Auscultation of the fetal heart either before labour or in labour involves direct contact with the abdominal wall. The examiner explaining the method and its purpose and requesting permission to auscultate will put the woman at ease.

Compared with the adult, the precordium is not so easily accessible on a fetus owing to the attitude it adopts within the uterus, which is folded or bent upon itself. The marker location to hear the heartbeat would be the back of the fetus, between the two scapulae (Fig. 3-1).

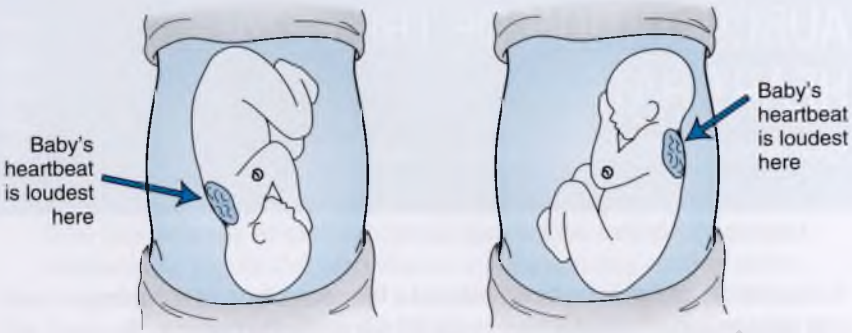

Figure 3-1. Locating the fetal heartbeat.

An abdominal palpation following the modified Leopold's manoeuvre will determine the lie, presentation, position and attitude of the fetus, which will help the examiner to determine the location where the fetal heartbeat is best heard. If the fetal heart cannot be definitely identified, ultrasound could be used to establish the optimal location for auscultation.

The fetal heartbeat produces a distinct sound comparable to a galloping horse, which has to be distinguished from vascular soufflé produced by uterine as well as fetal vessels. The fetal heartbeat is counted for at least 60 seconds to calculate the rate. Evaluation of the maternal pulse simultaneously confirms that the FHR is being monitored.

Placing the hand on the uterine fundus during auscultation provides additional information regarding uterine contractions and fetal movements.

In addition to the heart rate other information, namely FHR characteristics of acceleration and deceleration, could also be obtained, especially if the examiner listens at the time of fetal movements for accelerations and soon after a contraction for decelerations. As with any clinical skill it takes time to develop expertise and there is a slow learning curve for correct identification of accelerations and decelerations.[2] Its degree of reliability may be unacceptable in modern obstetrics owing to a higher rate of failure to recognize these changes.[3]

INSTRUMENTS USED FOR FETAL AUSCULTATION

The widely used instruments in current clinical practice are the Pinard stethoscope, the De Lee stethoscope and the hand-held Doppler monitor.

Pinard Stethoscope

This is a modification of the tool used to listen to the adult heartbeat by Laënnec in 1816. In its current form, the Pinard stethoscope was

Figure 3-2. Pinard stethoscope.

invented in 1895 by Adolphe Pinard, a French obstetrician. It is also referred to as a 'Pinard horn' or fetoscope (Fig. 3-2).

It is an inexpensive tool readily available in most countries and no consumables are needed. However, compared with other instruments used for auscultation it is difficult to use in certain maternal positions.

De Lee Stethoscope

The De Lee stethoscope is available in some countries and is also inexpensive (Fig. 3-3). It is equipped with a head-mount and using a De Lee stethoscope sometimes the examiner can hear the heartbeat by 16 weeks if he/she has practised with it. It is easier to hear from about 20 weeks, at which time the mother can feel the baby moving.

Figure 3-3. De Lee stethoscope.

Figure 3-4. Hand-held Doppler FHR monitor.

Hand-held Doppler Fetal Heart Rate Monitor

The hand-held ultrasound transducer uses the Doppler effect to provide an audible simulation of the fetal heartbeat (Fig. 3-4). Auscultating the fetal heart with a Doppler device is more comfortable for the woman and it is audible to all present in the room including the pregnant woman, which serves to reassure her and her family. Abnormalities in FHR are also more reliably detected by a Doppler FHR monitor than with a Pinard stethoscope and its use has been shown to be associated with good perinatal outcome.[4]

The current machines usually calculate and display FHR values, which reduces errors of counting and calculation by the user.

The hand-held Doppler can be used in various maternal positions and locations including birthing pools. Due to its sensitivity, however, it can inadvertently pick up the maternal heart rate, which must be verified by palpating the maternal pulse simultaneously.

These monitors are more costly than the other two types of equipment and need batteries to function. Also the probe is very sensitive to mechanical damage and needs appropriate handling.

OBJECTIVE AND INDICATIONS/PURPOSE

Auscultation of the FHR is performed during the antenatal period as well as during labour. The main purpose of auscultation of the FHR is to identify a fetus at risk and to take appropriate action to prevent fetal hypoxic injury.

Prenatal Period

The FHR can be usually auscultated by the healthcare provider once the woman perceives fetal movement, or by approximately 20 weeks of gestation. It can be picked up from 14 weeks using a hand-held Doppler.

During antenatal care for uncomplicated pregnancies, auscultation of the fetal heart may confirm that the fetus is alive but its value in predicting fetal health is low; hence routine listening is not recommended by some sources. However, auscultation of the fetal heart when audible using a Doppler device provides reassurance to the mother and her partner.[5]

If a woman reports decreased fetal movements, auscultation of the fetal heart is an important step to confirm fetus viability before further actions are taken.

Intrapartum Period

Low-risk women

There is general agreement in the professional literature that auscultation is an appropriate technique for fetal surveillance when a woman experiences a healthy pregnancy and birth (i.e., low-risk pregnancy and labour).

During the *first stage* of labour, intermittent auscultation is carried out immediately after a contraction for 1 minute, at least every 15 minutes, and recorded as a single rate with a record of accelerations and decelerations if heard. The woman's pulse is taken every 15 minutes to differentiate between the two heart rates. If intermittent auscultation indicates possible FHR abnormalities, this is explained to the woman and cardiotocography is offered.[6] The method of observation can be reverted to intermittent auscultation if the trace is reassuring after 20 minutes.[7]

During the *second stage*, intermittent auscultation of the FHR should be continued in a similar way immediately after a contraction, and auscultation is recommended at least every 5 minutes or after every other contraction. As during the first stage, if auscultation indicates possible FHR abnormalities then cardiotocography is offered. The FHR monitoring method can be returned to intermittent auscultation if the trace is reassuring after 20 minutes.[7]

High-risk women

The benefits of monitoring FHR by auscultation compared with continuous CTG monitoring in pregnancies at high risk has not been proven scientifically.[8]

Currently cardiotocography is offered to women at high risk with complications during pregnancy and the intrapartum period.

In low-resource settings where electronic fetal monitoring is not available, intermittent auscultation should be undertaken as in low-risk women.

CONCLUSION

On admission in labour it is prudent to ask the mother about her health and about fetal movements and to note the time when she felt the baby moving last. Then the baseline FHR should be measured. Following this the mother and the caregiver can feel for fetal movements with a hand on the maternal abdomen, and the FHR is auscultated when the fetus moves – this should show a rise in the FHR (i.e., an acceleration) – if needed this can be done a couple of times within a short period as the baby moves a few times during the active epoch of fetal behaviour. Then the mother could be asked to let the caregiver know when the contraction starts. The contraction can be palpated and at the end of the contraction the FHR can be recorded. If there is a deceleration it is likely to be an 'atypical' variable or late deceleration. Should a deceleration be heard, continuous auscultation would be valuable to see how long it takes to recover to the baseline rate and whether the same occurs with the next contraction, and if so the woman needs to be transferred for continuous monitoring. A FHR that shows acceleration is likely to exhibit decelerations and a gradual rise in the baseline rate or a sudden deceleration for a prolonged period before fetal demise. If fetal movements are not

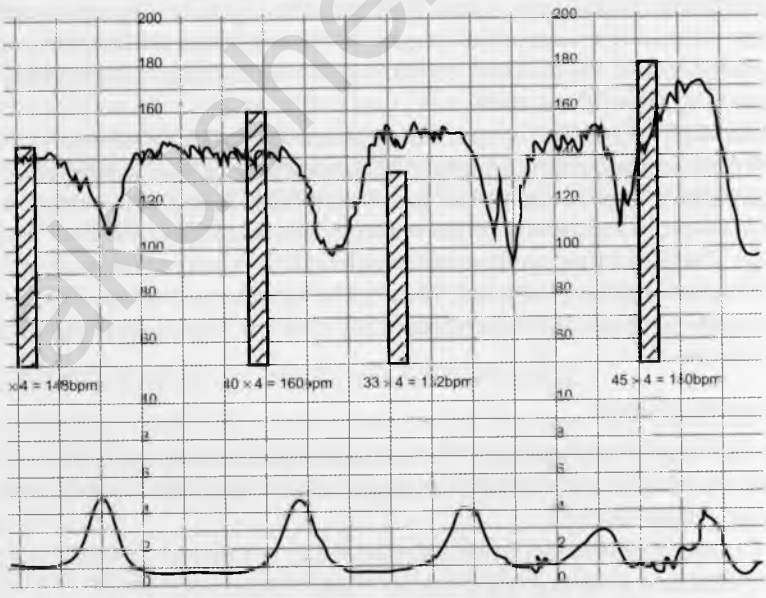

Figure 3-5. CTG showing a rise in baseline rate, progressive increase in depth and duration of deceleration and shortening of interdeceleration intervals. Counting for 15 seconds and multiplying by four provides erroneous FHR (as shown in the shaded bars) and hence the recommendation to count for one full minute.

felt for 90 minutes after admission, the examiner should search for fetal compromise such as reduced/absent fetal movements prior to admission, intrauterine growth restriction (IUGR), infection, bleeding, prolonged pregnancy and meconium at rupture of membranes to institute appropriate surveillance.

The CTG in Figure 3-5 shows a rise in baseline rate, progressive increase in depth and duration of deceleration and shortening of interdeceleration intervals and finally reduction of baseline variability before it becomes hypoxic/acidotic. Except for a reduction of baseline variability, all of these can be identified by continuous auscultation once the abnormality is detected.

One could therefore conclude that intermittent auscultation is an efficient method if used appropriately.

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ELECTRONIC FETAL MONITORING: TERMINOLOGY AND INTERPRETATION – THE BASICS

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Even when we all speak one language there remain difficulties in communication because of differing use of terminology. This may be resolved by better understanding and consideration of terms and definitions agreed by the International Federation of Obstetrics and Gynaecology (FIGO) Committee on Safe Motherhood and Newborn Health. These recommendations were published in 2015 in the *International Journal of Gynecology and Obstetrics*.[1] Recently the National Institute for Health and Care Excellence (NICE) has published guidelines on fetal monitoring and these are discussed in Chapter 6.[2] Without consistency of terminology we cannot have consistency of interpretation.

Monitoring is first of all clinical and then is complemented by technological methods. No cardiotocograph (CTG) can be interpreted without careful appraisal of the clinical situation. The following list illustrates particularly high-risk factors: prematurity, postmaturity, poor fetal growth, reduced fetal movements, meconium-stained amniotic fluid, bleeding in pregnancy, high blood pressure, breech presentation, multiple pregnancy and diabetes mellitus. This list could be extended indefinitely and yet would still account for only a minority of women delivering babies in most labour wards. Recognition of these factors is critical.

In the UK, we refer to antepartum CTGs and intrapartum CTGs. In the USA, antepartum CTGs are referred to as non-stress tests (NSTs). These are therefore distinguished from contraction stress tests (CSTs), where the contractions are stimulated by exogenous oxytocin. In the UK, CSTs are not performed and reliance is placed on other biophysical tests of fetal wellbeing. The admission test (CTG) is a natural contraction stress test using the contractions of early labour.

A fetal heart rate (FHR) tracing should be technically adequate to warrant analysis. The length of the CTG strip depends on the paper speed. In the UK and Europe it is usually 1 cm/min, whereas in the

USA it is 3 cm/min. As the pattern of the trace is dramatically altered by a change in paper speed, this can lead to confusion. It should, therefore, be standardized. A paper speed of 1 cm/min for each vertical division on the paper is 1 cm and therefore 1 min. A tracing should be annotated fully. At the beginning of the trace the mother's name, reference number and pulse rate should be recorded. Modern machines automatically annotate the time and date; however, a human being has to ensure that these are correctly set in the software and changed with seasonal time changes. The newest monitors have keypads or bar-code readers with which any other information may be recorded on the trace. It is important to relate vaginal examination, change of posture, epidural and other transient events to the fetal heart rate pattern, which could have medico-legal implications at a later date. The vertical scale on the paper is usually standardized to display between 50 and 210 beats per minute (bpm) in order for visual perception and interpretation to be consistent.

A tracing should be annotated fully.

The *baseline fetal heart rate* is the mean level of the fetal heart rate when this is stable, with accelerations and decelerations excluded. It is determined over a time period of 10 min and expressed in bpm. The rate may gradually change over time; however, for one particular period it normally remains fairly constant. NICE has defined the normal range of the baseline fetal heart rate at term as 100–160 bpm, whereas FIGO defines it as 110–160 bpm.[1,2]

Rates between 100 and 110 bpm are classified as baseline *bradycardia* and as a suspicious feature. There is little concern if it is an uncomplicated baseline bradycardia defined as a trace that has accelerations, normal baseline variability and there are no decelerations. Close involvement in the labour ward shows us that this is a relatively frequent finding and that the outcome is excellent (Fig. 4-1). One should confirm that the recording is indeed that of the fetus by auscultation and by cross-checking with the maternal pulse. Hypoxia should be suspected if the rate is below 100 bpm.

A range between 160 and 180 bpm is called a baseline *tachycardia* and is considered a suspicious feature. The outcome is good if it is an uncomplicated baseline tachycardia with accelerations, normal baseline variability and no decelerations. However, fetuses at term with a baseline heart rate of between 160 and 180 bpm should be carefully evaluated (Fig. 4-2A). A baseline rate of 150 bpm may fall within the normal range but is of major concern if the fetus had a heart rate of 120 bpm at the beginning of labour. Such a situation occurs in the late first stage and second stage of a prolonged labour when the mother is tired, dehydrated and ketotic. If corrective measures are not undertaken the rate will rise to 160–170 bpm (Fig. 4-2B).

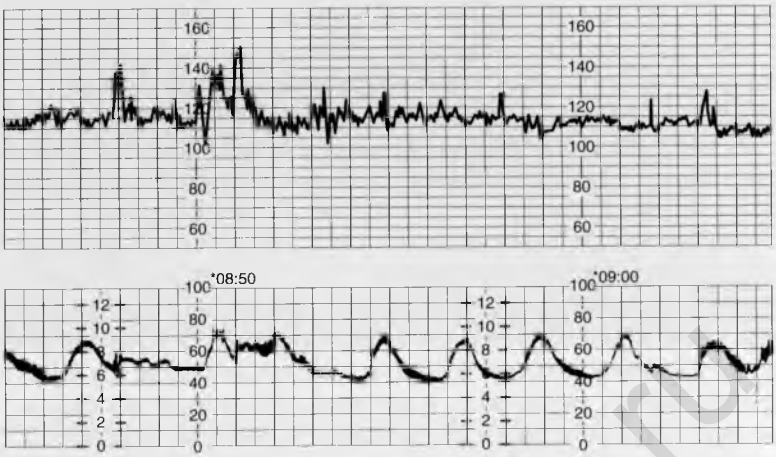

Figure 4-1. CTG – baseline fetal heart rate of 105–110 bpm.

This represents progressive compromise and is not an ideal scenario for a difficult instrumental vaginal delivery. Asphyxia is more likely to develop with a baseline rate of 160 compared with 110 bpm. This statement must be qualified before 34 weeks' gestation when the baseline fetal heart rate tends to be higher and a rate of up to 160 bpm is acceptable, provided accelerations are present and baseline variability is normal. Difficulties with identifying the baseline are considered later in this chapter.

An *acceleration* is defined as a transient increase in heart rate of 15 bpm or more and lasting 15 seconds (s) or more. The recording of at least two accelerations in a 20-min period is considered a reactive trace. Accelerations are a good sign of fetal health: the fetus is responding to stimuli and displaying integrity of its mechanisms controlling the heart. Accelerations are absent in situations of no fetal movements (e.g., fetal sleep), influence of some drugs, infection and intracerebral haemorrhage – hence the need for clinical correlation with the CTG findings.

A *deceleration* is a transient episode of slowing of the fetal heart rate below the baseline level of more than 15 bpm and lasting 15 s or more. Decelerations may be greater than this but not significant when other features of the heart rate are normal. When there is a reduced baseline variability (less than 5 bpm) in a non-reactive trace, decelerations may be very significant even when they are less than 15 bpm in amplitude (see below). A deceleration immediately following an acceleration recovering within 30 s is considered normal.

Baseline variability is the degree to which the baseline varies within a particular *bandwidth*, excluding accelerations and decelerations

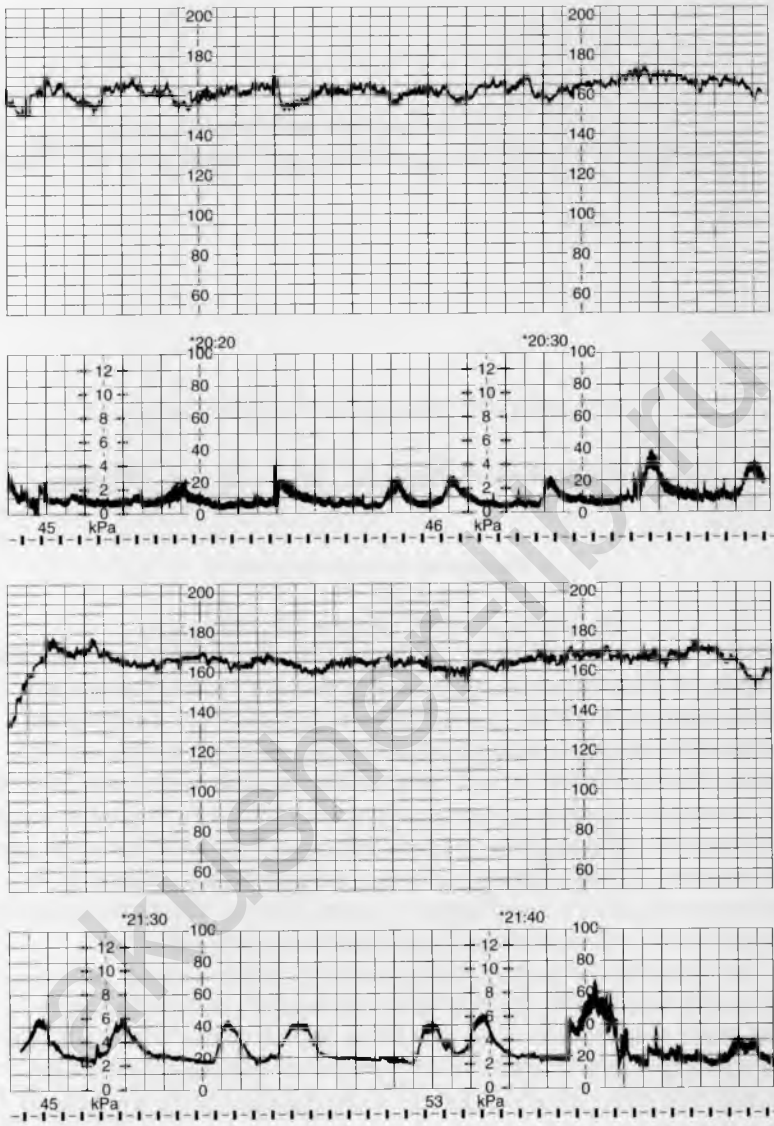

Figure 4-2. (A) Baseline rate 155–160 bpm; (B) rising to 165–170 bpm.

(Fig. 4-3). This is a function of the oscillatory amplitude of the baseline. For the purposes of research, oscillatory frequency and oscillatory amplitude may be quantified and scored. However, this is too complex for routine clinical use and bandwidth is preferred. Figure 4-4 shows bandwidths classified as reduced (<5 bpm), normal (5–25 bpm) and

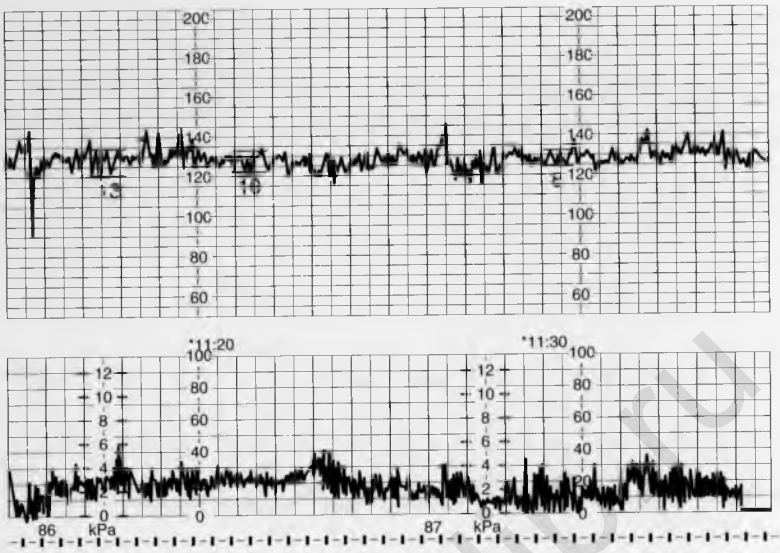

Figure 4-3. Normal bandwidth.

saltatory (more than 25 bpm).[1,2] The baseline variability indicates the integrity of the autonomic nervous system. It should be assessed during a reactive period in a 1-min segment showing the greatest bandwidth. Strictly speaking, beat-to-beat variation is not seen on traces. The equipment is not designed to analyse every beat interval and it uses an averaging technique. In a 1-min interval one cannot see 140 discrete dots. In a research situation, beat-to-beat variation can be analysed and is proportionally related to baseline variability. An understanding of the mechanism of production of baseline variability is crucial to an understanding of fetal heart rate interpretation.

Decelerations are *early*, *late* or *variable*. Early decelerations are synchronous with contractions, are usually associated with fetal head compression and therefore appear in the late first stage and second stage of labour with descent of the head. They are usually, but not invariably, benign. Late decelerations are exactly what their name implies with respect to the contractions: onset of deceleration is >20 s after the onset of contractions. As shown in Figure 4-5 the onset, nadir and recovery are all out of phase with the contraction. They are usually, but not invariably, pathological. Variable decelerations vary in shape and sometimes in timing with respect to each other. They may or may not indicate hypoxia. It is critical to evaluate the fetal condition between decelerations and its evolution with time. The integrity of the autonomic control system of the fetal heart must be evaluated (see Ch. 5).

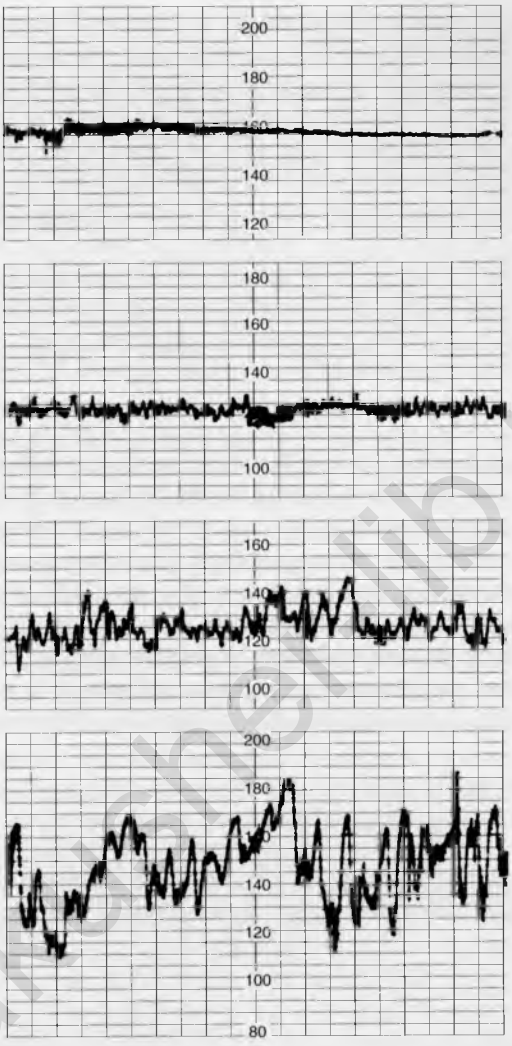

Figure 4-4. Bandwidth classification (reading downwards): silent, hardly any baseline variability (not in the NICE classification, but is not a reassuring sign); reduced, <5 bpm; normal, 5–25 bpm; saltatory, over 25 bpm.

So-called 'fetal distress', as implied from a CTG appearance, is not always indicative of hypoxia. Many fetuses are stressed and the challenge is to recognize when this progresses to hypoxic distress. Many babies are delivered operatively for 'fetal distress' (abnormal CTG) and are in excellent condition. This is the crux of the matter in considering the increased caesarean section rate after the introduction of electronic fetal monitoring. We do not *see* fetal distress on a strip of CTG paper. We *see* a fetal heart rate pattern and should describe

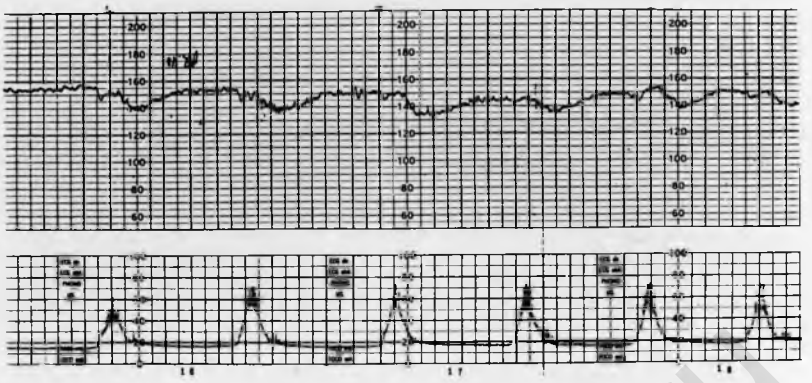

Figure 4-5. Late deceleration.

and classify it as such. It should then be interpreted with respect to the probability of it representing fetal compromise. Anaemia (a low haemoglobin concentration) is not treated rationally without further consideration being given to its aetiology. The same should apply to a fetal heart rate pattern that is not normal. In the light of the clinical situation the likelihood of hypoxia and/or acidosis can be evaluated.

Accelerations are the hallmark of fetal health.

Features of a reactive trace are shown in Figure 4-6. In looking at this trace think of a child playing in a field. The child has a normal

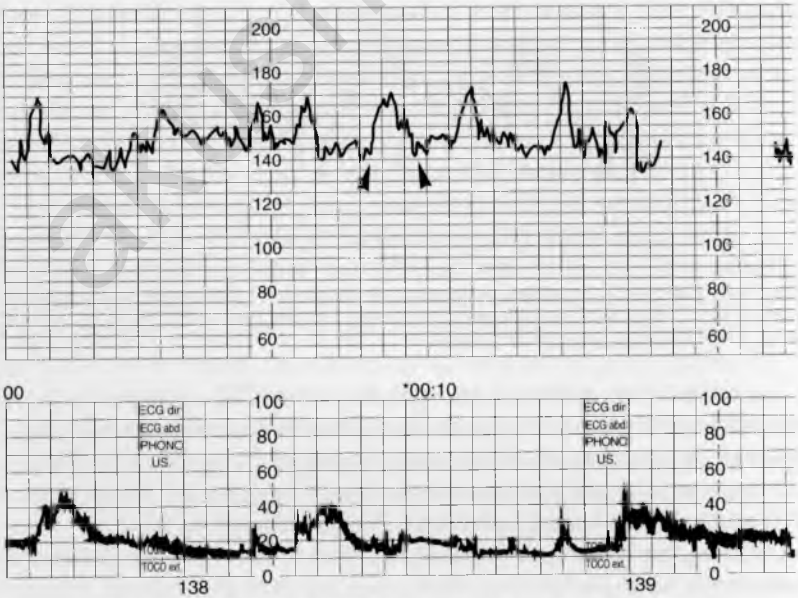

Figure 4-6. Reactive trace – two accelerations in 20 minutes.

pulse rate (baseline rate), minor movements of the limbs suggestive of activity (good baseline variability) and is tossing a ball up and down (accelerations). If the child is tired or is unwell it will start restricting its activity and stop tossing the ball (absence of accelerations is the first thing to be noticed when hypoxia develops suggesting that the child either is not well or is tired). Then the child would either sit or lie down to rest. In such a situation it is difficult to differentiate healthy tiredness from impending sickness. A persistently raised pulse rate after a period of rest would suggest the latter (baseline tachycardia). The fetus has limited capacity to respond to hypoxia by increasing its cardiac stroke volume and so has to increase its cardiac output by an increase in heart rate. Reduction in baseline variability and finally a flat baseline are the progressive features with increasing hypoxia. This is analogous to a rapid, thready pulse in a sick person and should be borne in mind when analysing traces. The baseline variability is due to the sympathetic and parasympathetic activities. An injection of atropine to the mother will increase the FHR and abolish the variability owing to the abolition of the parasympathetic activity.

Figure 4-7 shows a reactive trace with accelerations, normal rate and normal variability but a section of the trace was not registered. In the segment after the missing portion there are no accelerations, a normal rate but reduced baseline variability. A child who was in good health a few minutes ago cannot suddenly become sick without an obvious reason. The absence of accelerations and reduced baseline variability suggest that the fetus is in the quiet phase. This interpretation is further strengthened because there is no increase in

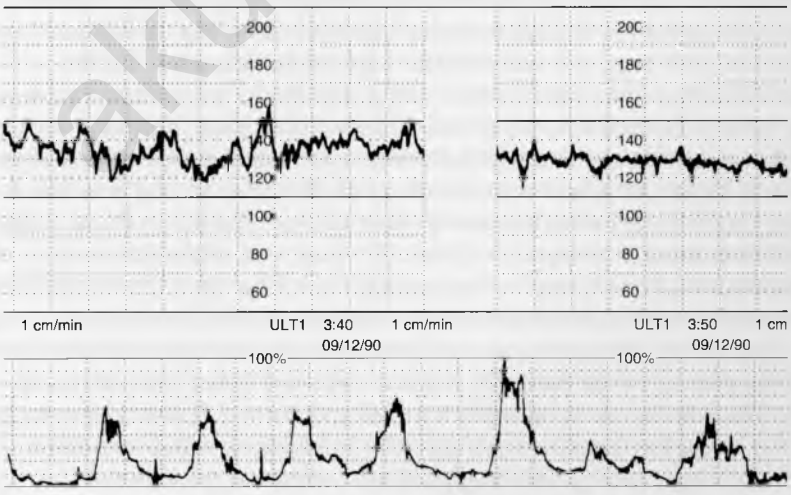

Figure 4-7. Reactive trace with a blank section.

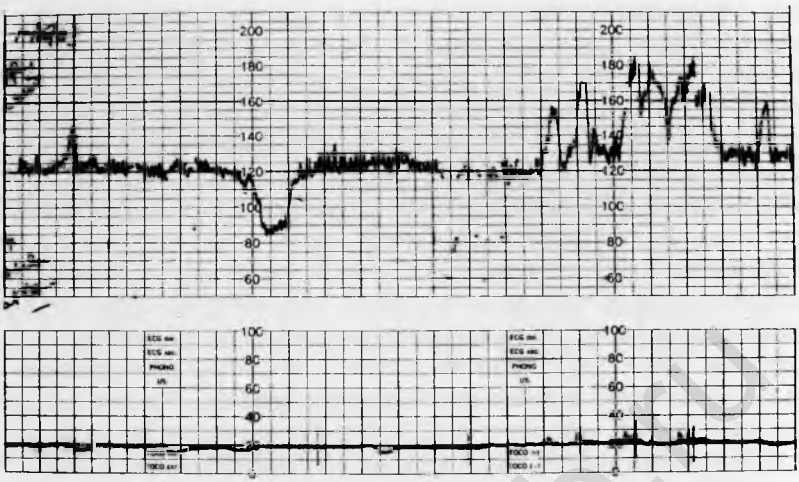

Figure 4-8. Reactive trace with isolated deceleration.

the baseline FHR. There are contractions present but no corresponding decelerations. This indicates that there is no stress to the fetus, such as cord compression or reduction in the retroplacental pool of blood, which may cause hypoxia. In labour, evolution of hypoxia is 'unlikely' without decelerations.

Figure 4-8 shows a trace with a baseline FHR of 120 bpm with normal baseline variability and an isolated deceleration followed by marked accelerations. The normal baseline rate and variability with marked accelerations (tossing the ball up and down) suggest that the fetus is not hypoxic. The isolated deceleration may be due to brief cord compression associated with fetal movement. In the intrapartum situation this may be accounted for by fetal movements, uterine contractions or reduced amniotic fluid due to the membranes having ruptured. This is not an immediate threat to the fetus, although further continuous electronic fetal monitoring is indicated. In the antenatal period the possibility of reduced amniotic fluid has to be considered; it may be due to intrauterine growth restriction, prelabour rupture of the membranes or prolonged pregnancy. Ultrasound evaluation should be undertaken. If the amniotic fluid volume is normal then the deceleration may be caused by pressure on the cord due to fetal movement.

Figure 4-9 shows a trace with repetitive variable decelerations. At the beginning of the trace the baseline rate is 120 bpm, there are no accelerations and the baseline variability is normal. Towards the end of the trace the baseline rate has risen to 160 bpm with a decrease in baseline variability. This suggests an attempt to compensate in response to the evolving hypoxia.

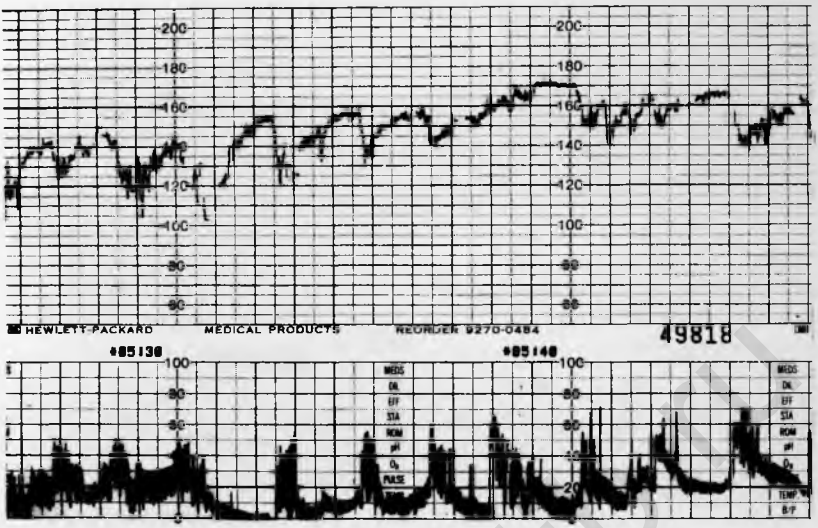

Figure 4-9. Repetitive variable decelerations – developing asphyxia.

PAPER SPEED

It is important to check the paper speed of any cardiotocograph (CTG) tracing before interpretation. It is not easy for someone who has been trained to interpret traces at 1 cm/min to then interpret a trace recorded at a speed of 3 cm/min. With current fetal monitoring technology, the paper speed is annotated automatically on the trace. If the paper speed is not annotated on the trace, scrutiny of the contraction duration would give a clue that the paper speed is more than 1 cm/min as the contraction duration on the trace would be 2–3 min, which is an unlikely event in normal labour. Figure 4-10 shows the effect on the trace by changing paper speed during the recording. Figure 4-11 shows comparative traces recorded at different paper speeds. At the faster paper speed, features such as baseline variability, accelerations and decelerations are altered. The baseline variability appears more reduced than is actually the case, accelerations are difficult to identify (Fig. 4-11A and B), and the decelerations appear to be of a greater duration (Fig. 4-10). To a trained eye the paper speed does not matter, but for day-to-day interpretation it is better to have the paper speed at the rate the staff is used to – failure to appreciate this has led to confusion and serious error. Current fetal monitors have their paper-speed switch mechanism either behind the paper-loading tray, which has to be removed to alter the paper speed, or in a position such that it is difficult to alter the speed accidentally. Although the discussion on paper speed may appear trivial, failure to recognize the

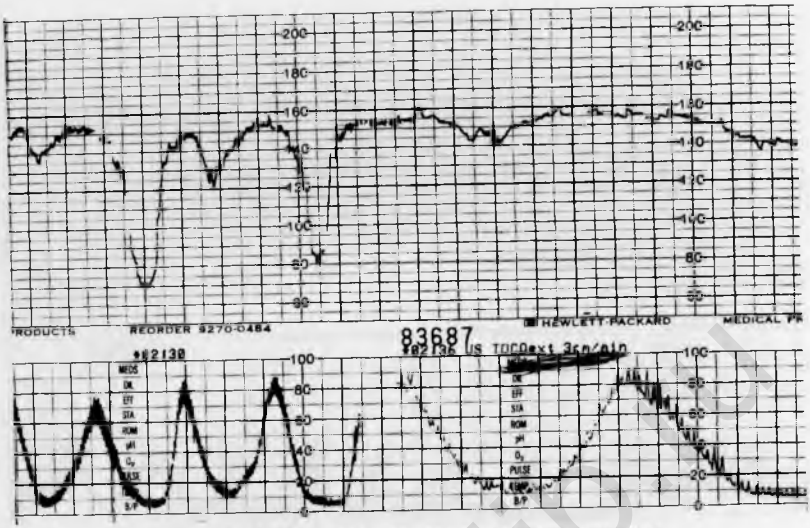

Figure 4-10. Changing paper speed during recording.

difference has resulted in unnecessary caesarean sections in both the antenatal and the intrapartum periods. Such simple mistakes expose the mother to an unnecessary anaesthetic and surgical risk and put her at high risk in her next pregnancy.

PROBLEMS ASSOCIATED WITH THE INTERPRETATION OF BASELINE VARIABILITY

Any FHR tracing has periods of high and low baseline variability cycles both in the antenatal and intrapartum periods. These periods of 'silent phase' with low baseline variability can be as short as 7–10 min in the antenatal period and 25–40 min in the intrapartum period.[3,4] Although baseline variability can be referred to at any given point in the trace, the health of the baby is best judged when the trace is reactive (i.e., when the baby is active and 'playing with the ball' rather than when the baby is sleeping). It is similar to our being judged at an interview when we are active and awake rather than inactive and sleeping.

Reduced Baseline Variability

The commonest reasons for reduced baseline variability are:

1. the 'sleep' or 'quiet' phase of the FHR cycle (Fig. 4-12)
2. hypoxia
3. prematurity
4. tachycardia (>180 bpm – due to technical issues)
5. drugs (sedatives, antihypertensives acting on the central nervous system [CNS] and anaesthetics)

6. congenital malformation (of the CNS more commonly than the cardiovascular system)
7. cardiac arrhythmias
8. fetal anaemia (Rhesus disease or fetomaternal haemorrhage)
9. fetal infection.

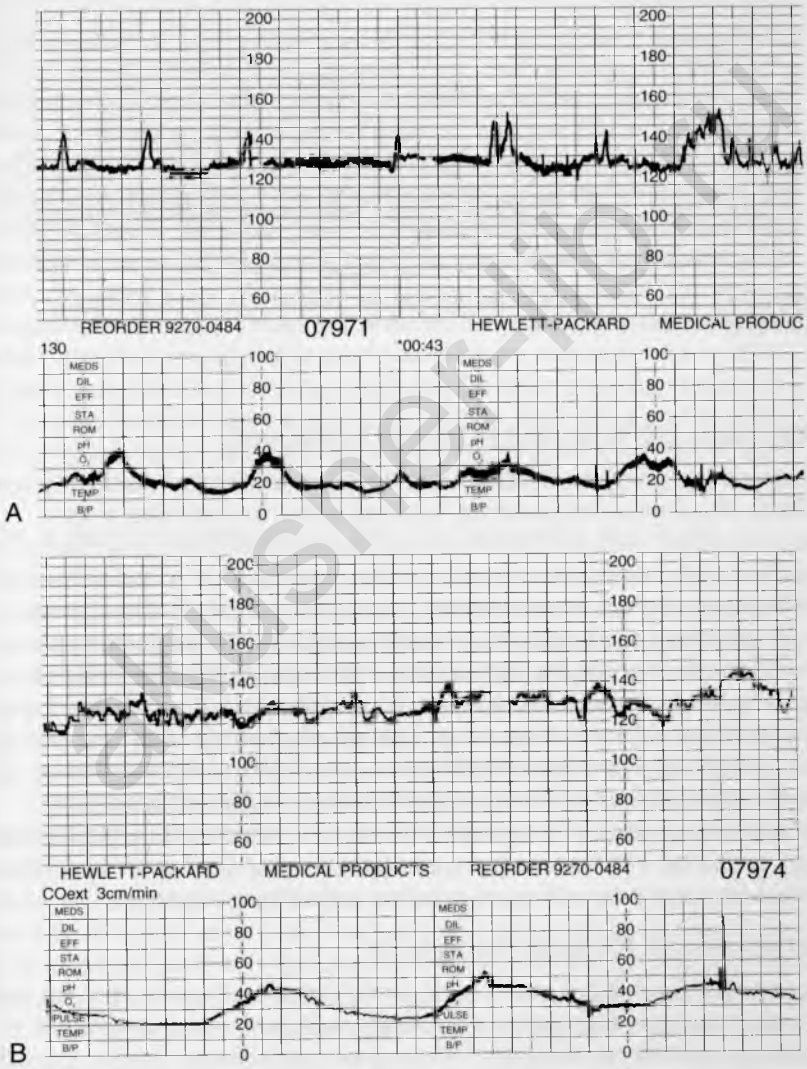

Figure 4-11. Two recordings from the same patient at: (A) 1 cm/min; (B) 3 cm/min.

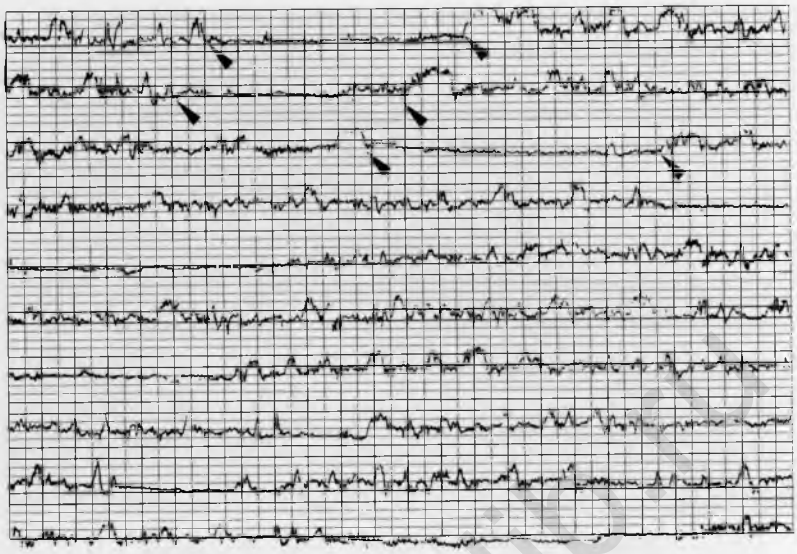

Figure 4-12. Composite trace – reduced baseline variability: period of fetal 'sleep' alternating with 'active' periods.

High and Low Variability Cycles ('Cycling')

When a trace is seen with reduced baseline variability (bandwidth 5 bpm), the previous segments of the trace must be reviewed. If the preceding trace was reactive with good baseline variability, then the segment being reviewed is probably in the 'quiet phase' of the baby's FHR cycle and there is no cause for alarm. The start of another active cycle can be awaited especially if there have been no decelerations or increase in the baseline rate, which might indicate the possibility of hypoxia. If there was no previous segment of the trace to consider, the clinical picture must be reviewed to identify whether the fetus is at risk (e.g., small fundosymphysis height, post-term, thick meconium, no or scanty amniotic fluid at the time of membrane rupture, reduced fetal movements, or other obstetric risk factors) or is influenced by medication (e.g., pethidine, antihypertensives, etc.) at the same time continuing the trace when reactivity with good baseline variability may appear.

Pethidine and Baseline Variability

Sometimes there is concern about giving pethidine to women in labour in case it reduces the baseline variability and obscures the reduced baseline variability of hypoxia. Before giving pethidine it is important to ensure that the FHR trace is reactive and normal with no evidence of hypoxia. Once the pethidine is given the accelerations may not

be evident and the baseline variability may become reduced as in the 'quiet' or 'sleep' phase. The period of this quiet phase following pethidine in some fetuses can extend beyond the natural quiet phase expected and thus leads to anxiety. In labour, if the trace has been reactive and the fetus was not hypoxic, hypoxia can develop only gradually owing to regular uterine contractions cutting off the blood supply to the placenta, unless acute events such as abruption, cord prolapse, scar dehiscence or oxytocic hyperstimulation occur. Alternatively, it can be due to cord compression with each contraction. The reduction of blood supply to the retroplacental area due to regular uterine contractions will present with late decelerations, and that due to cord compression will present with variable decelerations. If these are affecting the fetus and causing hypoxia, the fetus tends to compensate for the hypoxia by increasing the cardiac output, which it does by increasing the FHR as it has limited capacity to increase the stroke volume. Therefore, if the FHR pattern after pethidine does not show any decelerations and there is no increase in the baseline rate then, despite the fact that there are no accelerations and the baseline variability is reduced, these features are likely to be due to pethidine rather than to hypoxia. When the baby is born, the baby may not cry and may need stimulation or reversal of drug effect by naloxone or assisted ventilation because of the effect of the drug on the CNS causing respiratory depression, but the neonate will have good cord arterial blood status indicating that there was no intrauterine hypoxia.

False Baseline Variability Due to Technical Reasons

Modern machines have autocorrelation and do not pose technical problems related to baseline variability, but the older machines did not possess autocorrelation and gave a false impression of exaggerated baseline variability when the FHR was recorded using an ultrasound transducer. Although one may not encounter this in current practice, traces from several years ago may be brought up to you for medico-legal reasons and hence the explanation for this problem is offered in this section.

The baseline variability seen on the trace is produced by the time differences between individual heartbeats. One segment of the serration or undulation, that is, one upswing which contributes to baseline variability, is only a few millimetres but is representative of a number of beats, as was outlined earlier. The machine calculates the beat intervals from the impulses coming back to the transducer, which arise from the movements of the fetal heart. However, there may be extraneous impulses from other sources (caused by movement of the bowel or of the anterior abdominal wall of the mother), which may be misinterpreted and a falsely exaggerated baseline variability produced (Fig. 4-13). When the fetus becomes hypoxic, usually the first feature

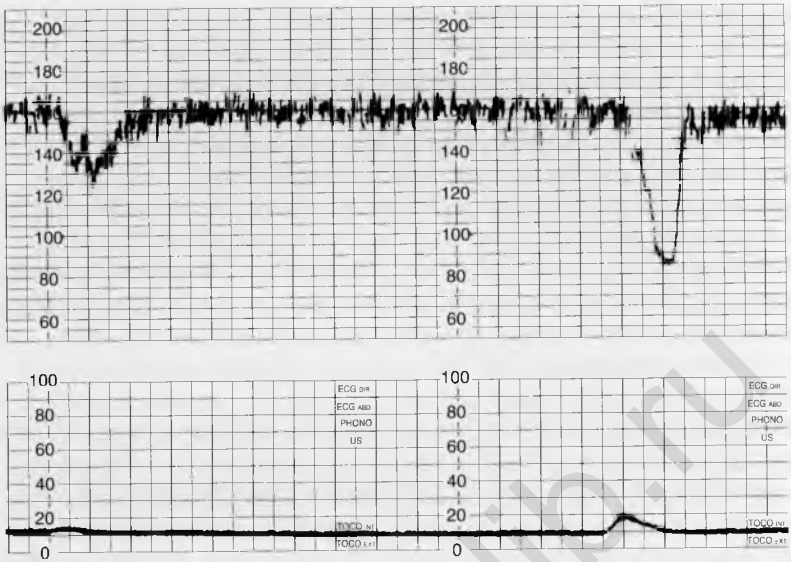

Figure 4-13. Artefactual variability due to old machine without autocorrelation facility.

to be observed is the disappearance of the accelerations, followed by an increase in baseline FHR and a reduction in the baseline variability. In Figure 4-13 there is tachycardia, with a FHR of 160 bpm, there are no accelerations and there are variable decelerations suggestive of possible fetal compromise. This trace was from a growth-restricted fetus with little amniotic fluid surrounding it. Its other features (absence of accelerations, tachycardia and decelerations) are not consistent with the 'good baseline variability' observed on the trace. The problem is that the trace was obtained on a fetal monitor without autocorrelation facilities. The baseline variability obtained on the ultrasound mode with the old fetal monitors is not reliable and in labour it is best to use a scalp electrode with these machines. Figure 4-14 shows an abnormal trace with tachycardia, no accelerations and with reduced variability. The switch from ultrasound to direct electrocardiograph (ECG) mode gives the markedly reduced (flat) true baseline variability of the sick fetus. Use of modern machines should obviate this problem (Fig. 4-15).

Poor contact of the scalp electrode

'Picket fence' artefact is not an uncommon problem with the use of scalp electrodes (Fig. 4-16). The vertical deviation of the baseline, unlike the undulations, suggests that it is an artefact. Figure 4-17 shows a baseline tachycardia with a rate of 150 bpm. There are no accelerations and careful attention reveals that the baseline variability is markedly reduced (less than 5 bpm) and is masked by artefact.

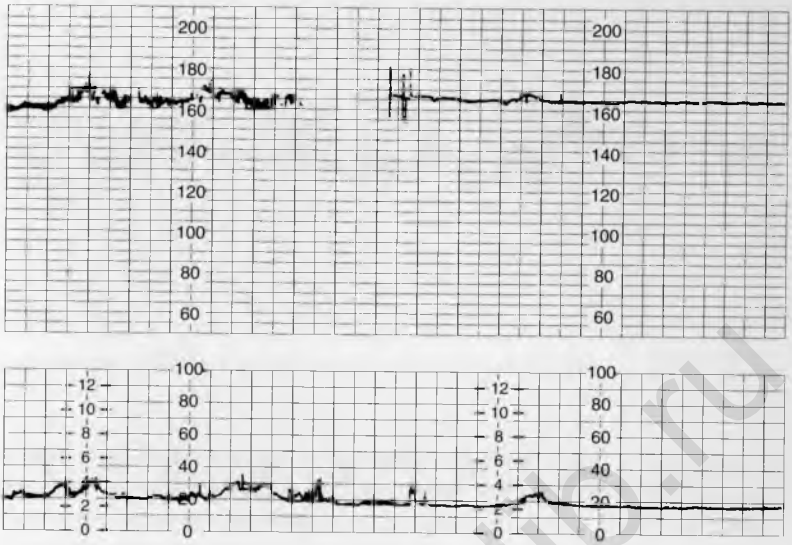

Figure 4-14. Artefactual variability obscuring pathological trace rectified by using scalp electrode.

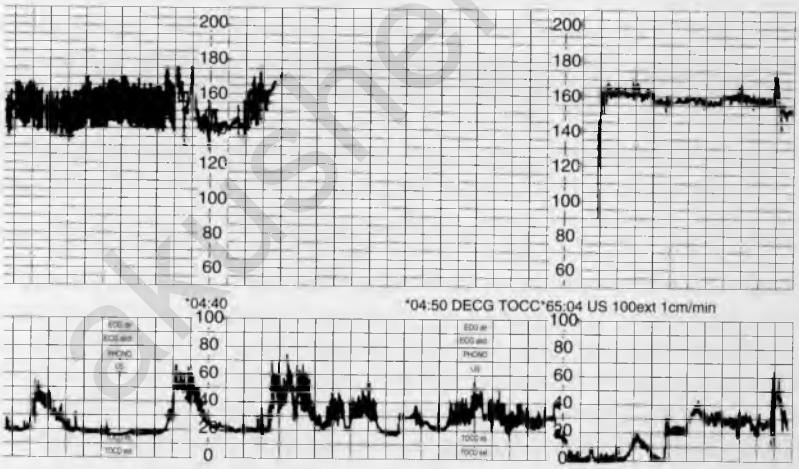

Figure 4-15. Effect on variability of changing monitoring mode from fetal electrode to ultrasound in a machine with autocorrelation facility.

This is usually thought to be due to poor contact of the electrode with fetal tissue or the absence of proper contact of the reference electrode (a metal piece at the base of the scalp electrode) with maternal tissue. Although replacing the electrode and applying an adhesive skin electrode to the maternal thigh as a reference electrode may be of some help, usually these manoeuvres do not markedly

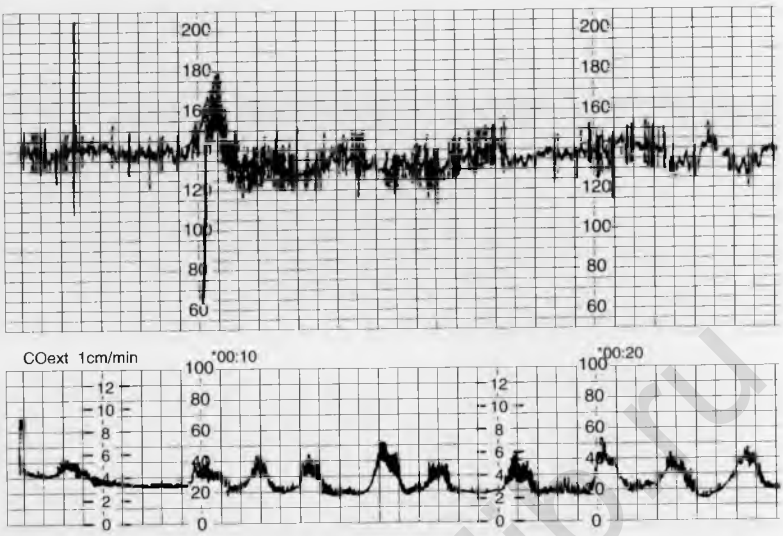

Figure 4-16. 'Picket fence' artefact due to poor contact of fetal electrode.

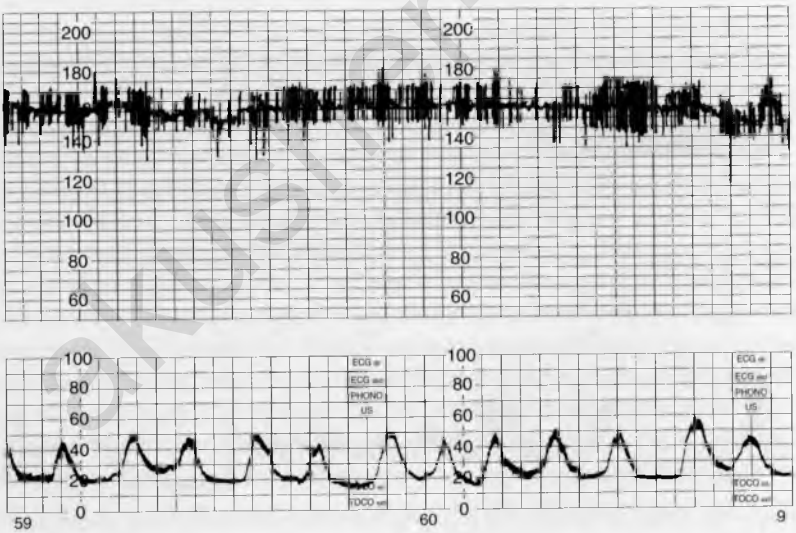

Figure 4-17. Abnormal trace with no accelerations and reduced variability being hidden by 'picket fence' artefact.

improve the quality of the recording. In these situations, it is better to record the FHR tracing with an external ultrasound transducer that has autocorrelation facilities, as most modern equipment has. These fetal monitors give a good-quality trace with a baseline variability equivalent to that obtainable with a scalp electrode. In the past, when a good-quality

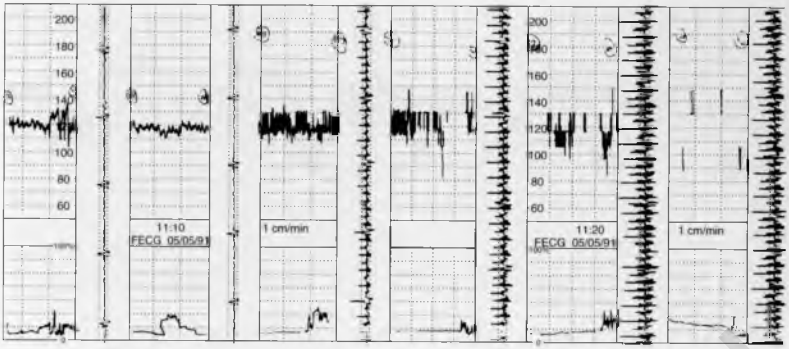

Figure 4-18. Effect of TENS on the trace as the TENS frequency rate is increased.

trace was not obtained with external ultrasound transducers, the use of internal electrodes was advocated, whereas currently the use of external ultrasound transducers is indicated when the FHR trace with an internal electrode is unsatisfactory (see Fig. 4-15). Because of the good-quality tracing obtained with fetal monitors using modern technology, there is no necessity to rupture the membranes during labour in order to place an electrode. The indications for artificial rupture of the membranes are during augmentation of slow labour and to inspect the colour of the amniotic fluid when a trace is abnormal. If the 'picket fencing' has a regular pattern and the distance above and below the baseline is nearly equal throughout the trace then it may be due to cardiac arrhythmia. If not, it is likely to be a problem with disturbance in the signal-to-noise ratio caused by the electrode.

Other interference

Extraneous electrical influences can produce artefact in the baseline variability; if the disturbance exceeds the frequency of signals obtained from the FHR using a scalp electrode it can completely confuse the FHR signals with no FHR tracing. The use of transcutaneous electrical nerve stimulation (TENS) or the obstetric pulsar used for pain relief can produce this problem; Figure 4-18 illustrates this with FHR tracing and the corresponding ECG signals.

With TENS, external ultrasound monitoring is preferable.

CORRECT IDENTIFICATION OF BASELINE HEART RATE

Persistent accelerations may lead to confusion such that some traces have been termed 'pseudodistress' patterns. When the fetus is very active it may show so many accelerations that it is misinterpreted as tachycardia with decelerations (Fig. 4-19). This situation can arise

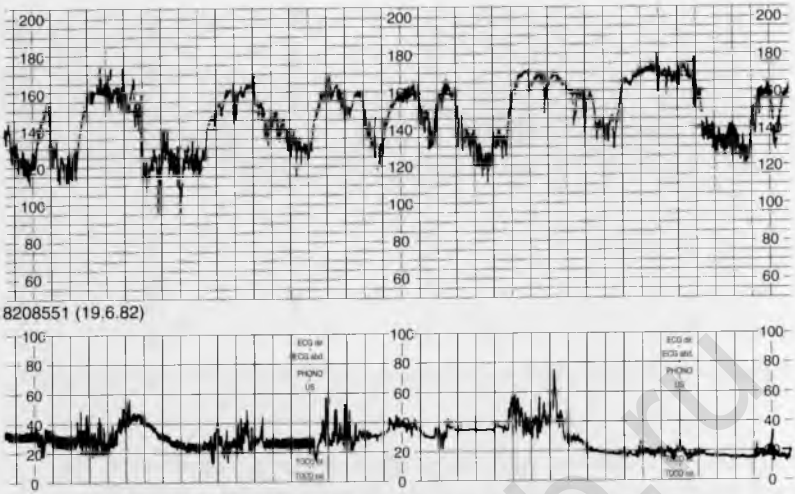

Figure 4-19. Very reactive trace – pseudodistress pattern.

in the antenatal period or during labour. Certain clues aid correct interpretation. The clinical picture and risk assessment will indicate the probability of true compromise. Figures 4-20 and 4-21 show greater degrees of the same phenomenon and are more difficult to interpret. The trace may appear to show a long period of tachycardia and confluent accelerations. In the antenatal period, it is easier to recognize these patterns as non-pathological if the fetus

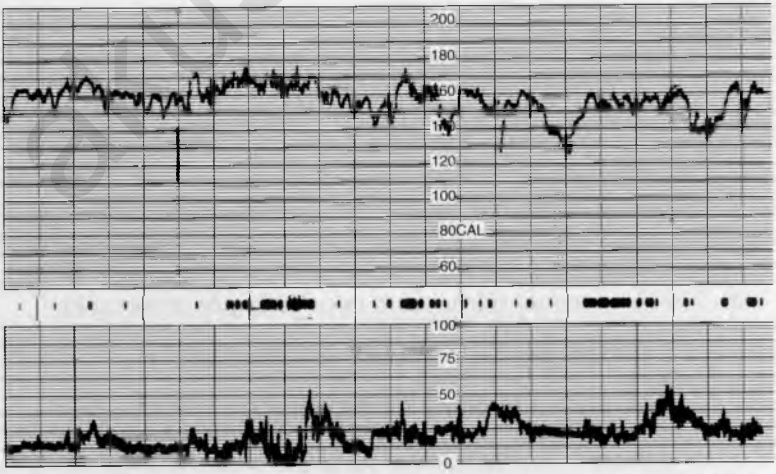

Figure 4-20. Continuous accelerations – very frequent use of event marker.

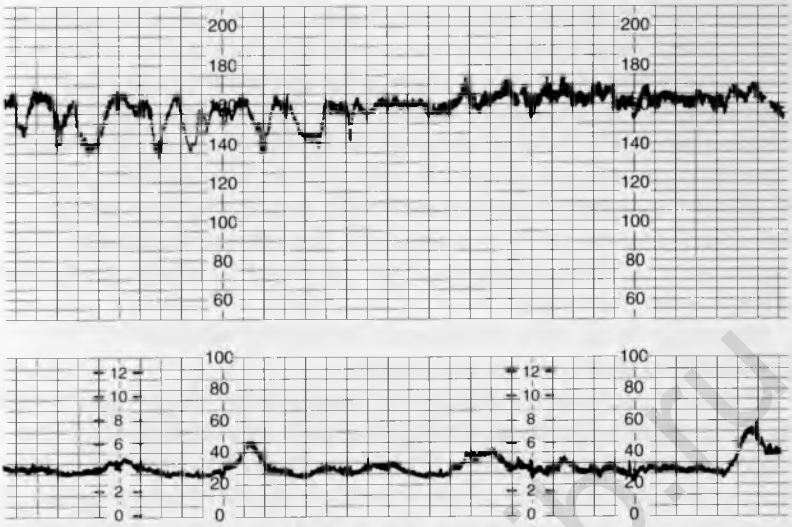

Figure 4-21. Confluent accelerations.

is well grown, has a normal amniotic fluid volume and is moving actively during the recording of the trace. This will be most obviously demonstrated by frequent use of the event marker by the mother or by evidence of fetal movements on the tocography channel (see Fig. 4-20). Many fetal monitors detect fetal movements automatically (Fig. 4-22). Such traces should have good baseline variability both at

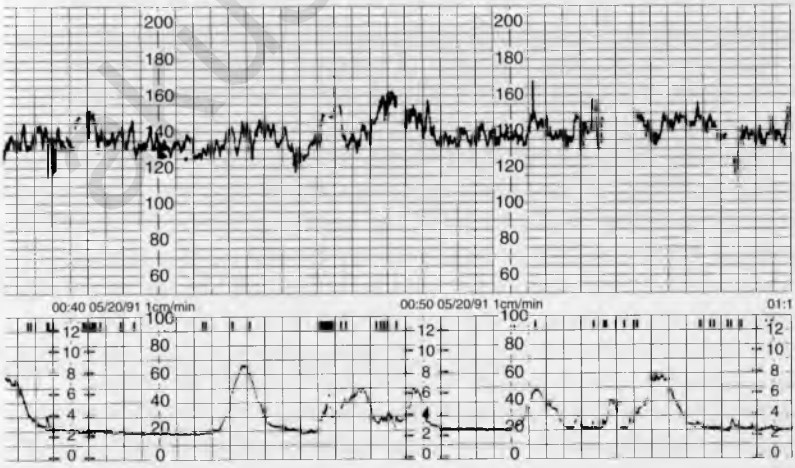

Figure 4-22. Hewlett-Packard 1350 with a combi transducer; automatic fetal movement recording is done through the ultrasound channel.

the true rate and at the higher rate. The true baseline rate on these traces is not below 110 bpm (the lower limit of normal for a healthy fetus). Inspection of the trace prior to the segment where there is doubt as to the true baseline rate would provide evidence of the true baseline rate. If such a segment is not available, continuation of the trace for a longer period should provide it. In clinical practice this pattern is repeatedly misunderstood, resulting in unnecessary intervention and the birth of a vigorous neonate behaving after delivery as it did before: Apgar scores of 9 and 10 after a caesarean section for 'fetal distress'.

A hypoxic fetus with a tachycardia with or without decelerations does not move actively.

At times there may be difficulty in resolving this issue. Figure 4-23A may be considered to show either stress or a very active fetus. The tocography channel suggests rather frequent contractions, and after the reduction in the rate of oxytocin and contraction frequency a more understandable picture emerges (Fig. 4-23B). Further evaluation may be necessary with ultrasound assessment antenatally or fetal scalp blood sampling intrapartum. If an oxytocin infusion is in progress its rate should be reduced.

Importance of Recognition of the Baseline Heart Rate for Each Fetus

When a fetus is in good health the baseline FHR tends to vary by 10–15 bpm in an undulating manner, slowing slightly in the sleep phase and after maternal sedation. It rises slightly during the active phase when the fetus moves, exhibiting a number of accelerations. Gradually increasing hypoxia causes the FHR to rise gradually to a tachycardia. During the evolution of persistent repetitive decelerations it is important to recognize the steadily rising baseline rate due to compensation, potentially leading to compromise. Each fetus has its own baseline rate and, although it may still be within the normal range, for that individual fetus it can represent a significant rise. It is important to take note of the baseline rate at the beginning of the trace and to compare it with the current rate. In the antenatal period, comparison of the baseline heart rate of sequential traces has the same relevance. Priority should be given to the revised definition of normal baseline FHR, 110–160 bpm, bearing these considerations in mind. Any tracing with a baseline rate of greater than 160 bpm should be carefully scrutinized for other suspicious features. Traces within the normal range for baseline rate may be abnormal or ominous on account of other features (Fig. 4-24).

A normal baseline rate can be associated with hypoxia and an ominous trace.

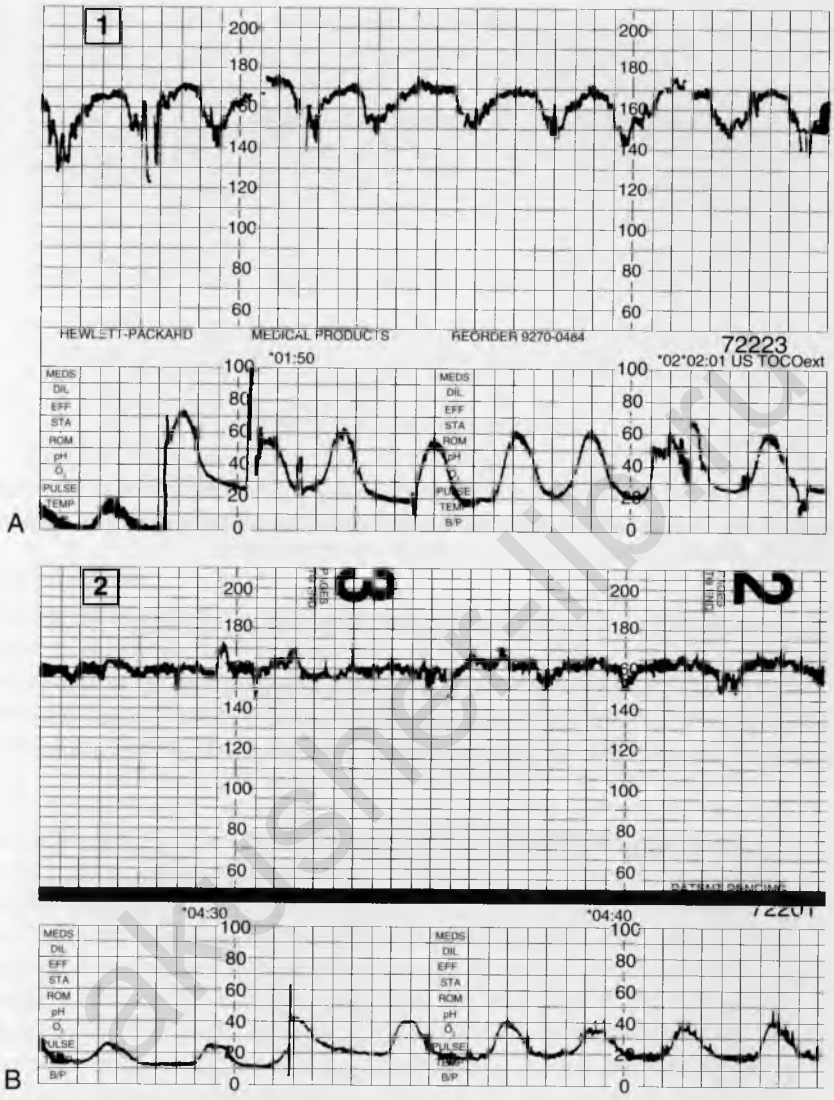

Figure 4-23. Trace showing: (A) hyperstimulation and tachycardia; (B) followed by reduction of oxytocin and resolution.

Baseline Tachycardia and Bradycardia

A range of 160–180 bpm is termed a *baseline tachycardia* and a range of 100–110 bpm is called *baseline bradycardia*. Although they are categorized in the 'suspicious' category in various guidelines, provided there is good baseline variability, accelerations and an absence of decelerations, these FHRs do not generally represent hypoxia.

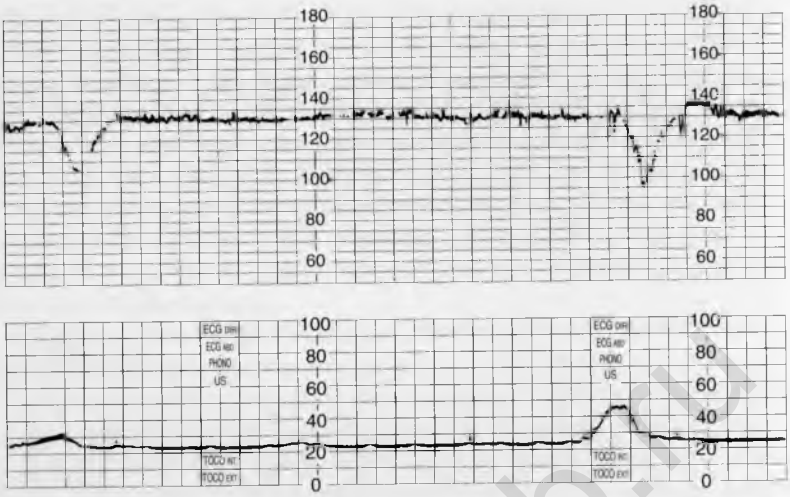

Figure 4-24. Normal baseline rate – pathological trace, with no accelerations, reduced baseline variability ('silent pattern') and shallow late decelerations.

Figure 4-25 shows a moderate baseline tachycardia although other features are reassuring.

Figure 4-26 is a rare trace showing sinus bradycardia at 80 bpm with a trace that is otherwise remarkably normal. The baby was born in good condition with a good outcome. The mother had had a renal

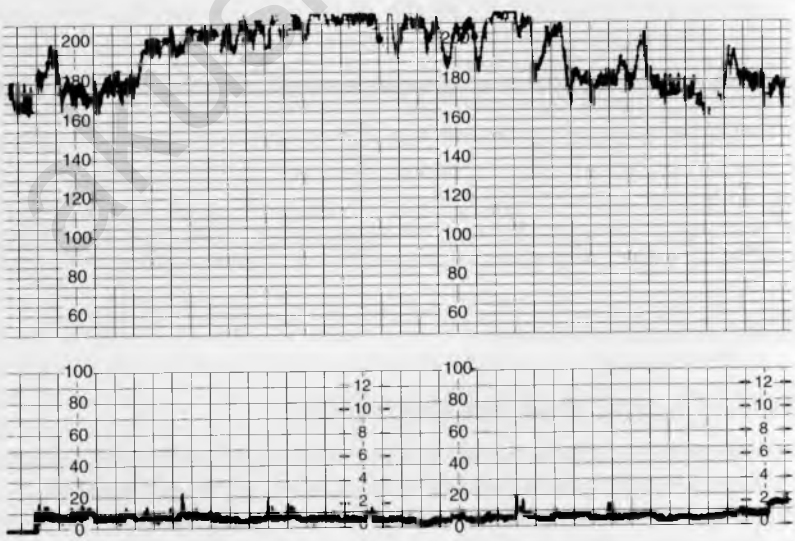

Figure 4-25. Moderate baseline tachycardia (150–170 bpm); other features are reassuring.

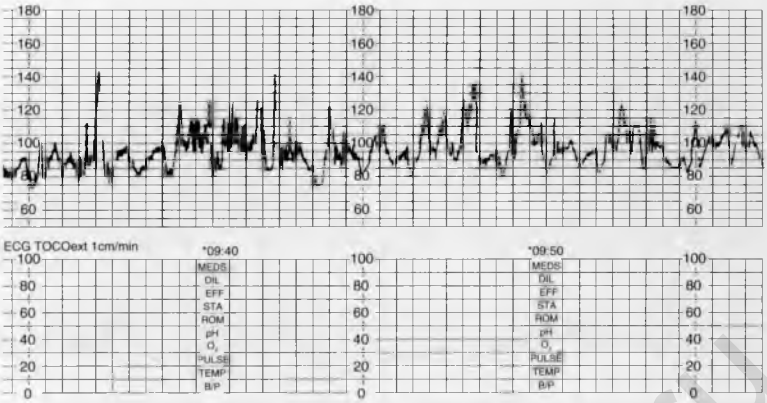

Figure 4-26. Sinus bradycardia.

transplant and was taking various medications including beta-blockers for hypertension, which is contraindicated in pregnancy.

Tachycardia

Tachycardia with a baseline rate greater than 160 bpm should prompt a search for other suspicious features such as absence of accelerations, poor baseline variability and decelerations. Tachycardia is not uncommon in preterm fetuses owing to the earlier maturation of the sympathetic nervous system. With increasing maturity of the fetus the baseline heart rate gradually falls and at term is often between 110 and 140 bpm. Fetal tachycardia may be due to fetal movement or increased sympathetic tone caused by arousal associated with noise, pain or acoustic stimulation. Fetal hypoxia, hypovolaemia and anaemia are pathological causes of tachycardia. Maternal sympathomimetic activation due to pain or anxiety may lead to fetal tachycardia, as can dehydration leading to poor uterine perfusion. Pain relief, reassurance and hydration may be expected to reverse this. Administration of betamimetic drugs to inhibit preterm labour increases sympathetic activity, whereas anticholinergic drugs such as atropine abolish parasympathetic activity through the vagal nerve, resulting in tachycardia.

False or Erroneous Baseline Because of Double Counting of Low Baseline FHR

In normal circumstances the atrium and ventricle beat almost simultaneously followed by the next complete cardiac movement of the atrium and ventricle. The reflected ultrasound from these two chambers, or even from one of the walls (atrium, ventricle or the valves), is used by the machine to compute the FHR. When the FHR

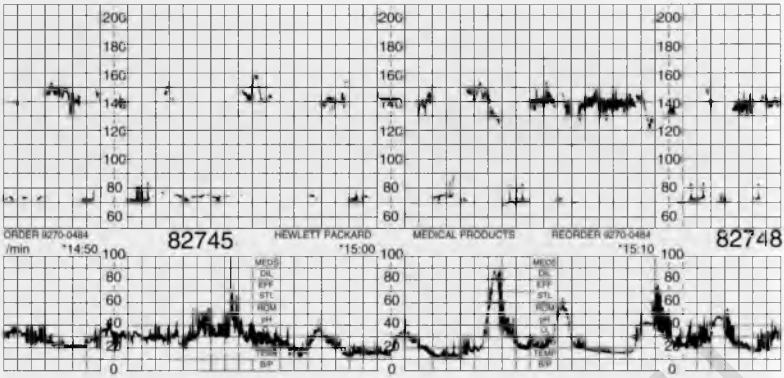

Figure 4-27. Intermittent double counting – heart block in maternal systemic lupus erythematosus.

is slow, at 70–80 bpm, there is a longer time interval between the atrial and the ventricular contractions. The machine recognizes each of the reflected sounds (one from the ventricle and the other from the atrium) as two separate beats and computes the rate, which may mimic the FHR as it will be in the expected range for a normal fetal heart. For most observers the sound generated will also give an impression that the FHR is within the normal range; this is because the heart sounds from the machine are always the same for every baby – they are electronic noise. During the false counting or 'doubling' of the FHR episode, listening with a fetal stethoscope will reveal the true situation. The suspicion that something is amiss will be aroused by the FHR tracing, which may show a steady baseline of 140 bpm but at times will be 70 bpm. Because it is a double-counting phenomenon the upper rate on the recording paper will be exactly double that of the lower rate and can be easily checked by auscultation. Such a trace can also be due to the machine recognizing an atrial rate of 140 bpm and a ventricular rate of 70 bpm at different times in a case with complete heart block (Fig. 4-27). The mother may have an autoimmune disorder. Doubling the rate is a phenomenon dependent on the use of ultrasound monitoring. A fetal electrode will not show this effect and should therefore be used if in doubt. A situation of bradycardia with the doubling effect may be observed in a sick fetus as an acute episode and a preterminal event.

Beware of double counting.

Bradycardia: Fetal or Maternal?

A record of the maternal heart rate made by using the external ECG mode of the monitor by applying maternal skin electrodes, supplied

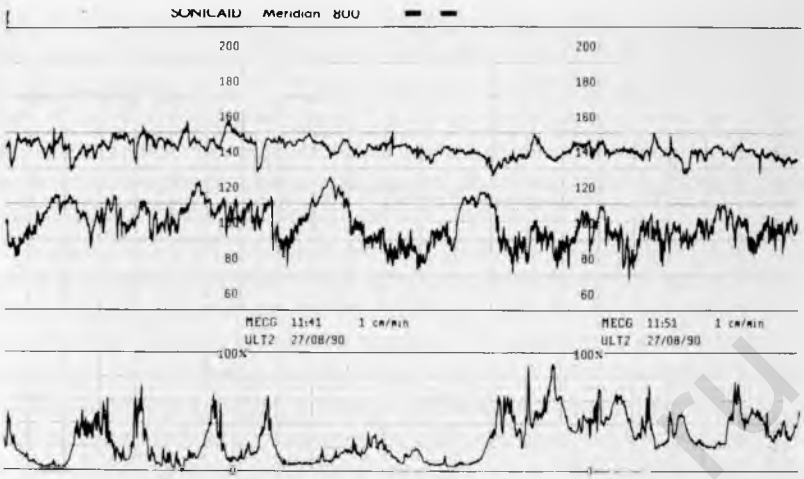

Figure 4-28. Fetal (upper) and maternal (lower) trace recording.

with the equipment, is shown in Figure 4-28. This is identical to fetal recordings (Fig. 4-28, upper trace), and mimics FHR when there is maternal anxiety or with betamimetic therapy for preterm labour, which results in a maternal tachycardia. With current fetal monitors that have facilities for maternal pulse oximetry the maternal heart rate can be recorded, and better still if 'smart pulse' facility with the external transducer is available (see Fig. 2-8). If the woman reports with reduced fetal movements she may have a tachycardia due to anxiety, and this may be mistaken for the actual FHR while the fetus is dead. Note that the lower trace, which is maternal, accelerates and has variability, as does the fetal trace.

If you do not have a modern monitor that displays a reliable maternal as well as a fetal pulse then always use the fetal stethoscope before applying the machine. This is the advice of the Medical Devices Agency of the UK.

When the fetus is dead the ultrasound may be inadvertently directed at maternal vessels. The technical quality of this trace is usually poor with incomplete continuity. In such circumstances it is prudent to verify the presence of the fetal heart activity by auscultation, confirming it with an ultrasound scan if there is doubt.

If two baseline rates appear that do not show the 'doubling' phenomenon the transducer may be picking up the fetal heart at one time and the maternal pulse at another time. The trace in Figure 4-29 was recorded in preterm labour treated with betamimetic drugs showing fetal and maternal tachycardia. This should be verified by counting the maternal pulse at the wrist and by auscultating the fetal

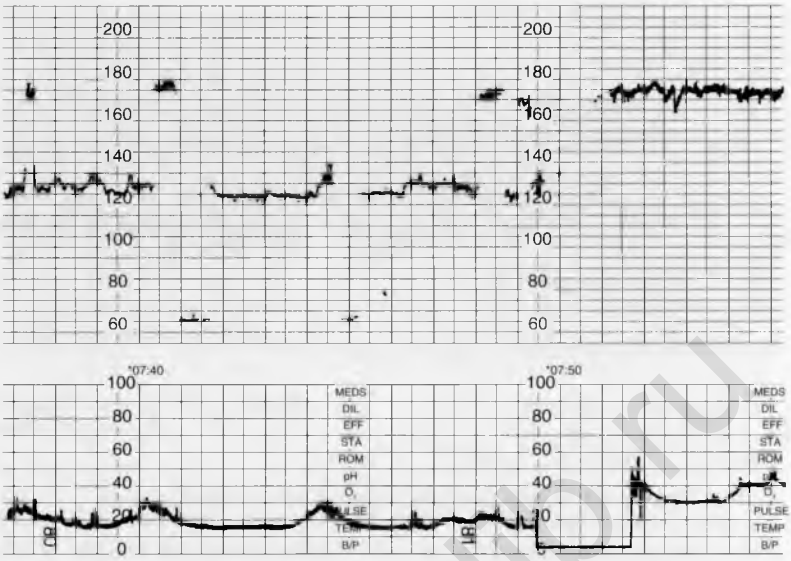

Figure 4-29. Maternal (120 bpm) and fetal (170 bpm) tachycardia – ultrasound mode.

heart simultaneously. The findings should be documented on the CTG paper for clinical and medico-legal purposes.

Fetal Arrhythmia

Complete fetal heart block may be recorded as a stable bradycardia and may give a trace as shown in Figure 4-27. Incomplete heart block is more of a dilemma. Both diagnoses should be substantiated by a detailed B-mode ultrasound scan and further investigation. A heart block must be a proportion of the actual rate (2:1, 3:1) and this should be analysed. Confirmed heart block should prompt a search in the mother's blood for autoimmune antibodies even if she is asymptomatic. Fetal heart block compromises intrapartum surveillance and alternative methods to electronic fetal monitoring should be used (e.g., clinical sense, fetal blood sampling, Doppler blood-flow study).

Occasional dropped beats or ectopic beats are a relatively common phenomenon in normal fetuses; however, more persistent arrhythmias can be associated with hypoxia.

PROBLEMS ASSOCIATED WITH INTERPRETATION OF TRACES

In the past much time and effort has been spent on categorizing decelerations into 'early', 'late' and 'variable', rather than interpreting the trace as a whole in relation to the clinical situation. A given trace

may be acceptable as normal in the late first stage but not in the early first stage of labour. At times it is difficult to classify the decelerations as early, variable or late. Often they may have mixed features of variable and late decelerations. It is far more important to categorize any trace as normal, suspicious or pathological. The FIGO[1] and NICE[2] recommendations for the classification of the features of the CTG and the CTG as a whole are described in Chapter 6.

Figure 4-30 shows a trace with tachycardia, no accelerations, reduced baseline variability and repetitive decelerations. Clinically the fetus is post-term and the mother is in early labour. This is a grossly abnormal trace demanding intervention. The decelerations may be analysed as variable because of the precipitous fall in the baseline rate characteristic of cord compression and because the decelerations vary in shape and size. They may be considered to be late because of the lateness in recovery. However, even when the decelerations are ignored the trace is abnormal because there are no accelerations, the baseline rate is greater than 160 bpm and the baseline variability is less than 5 bpm. There should be no hesitation in classifying this trace as pathological. Those who have limited knowledge of the pathophysiology of FHR may spend time arguing about the nature of the decelerations without concentrating on the whole trace and the clinical picture.

Intervention is mandatory.

Figure 4-31 shows a pathological trace but this is difficult to recognize unless one is aware of the exception to the rule of interpreting FHR traces. The rate can be within the normal range (110–160 bpm) but with reduced baseline variability (<< 5 bpm) and repeated

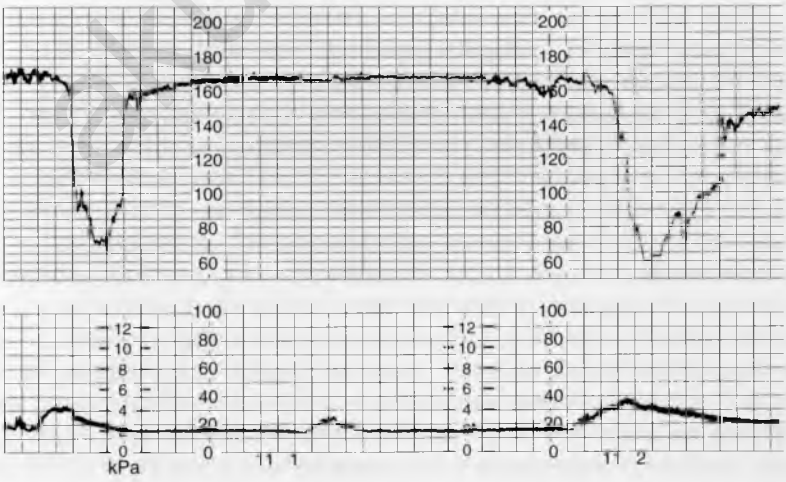

Figure 4-30. Grossly abnormal features – pathological trace.

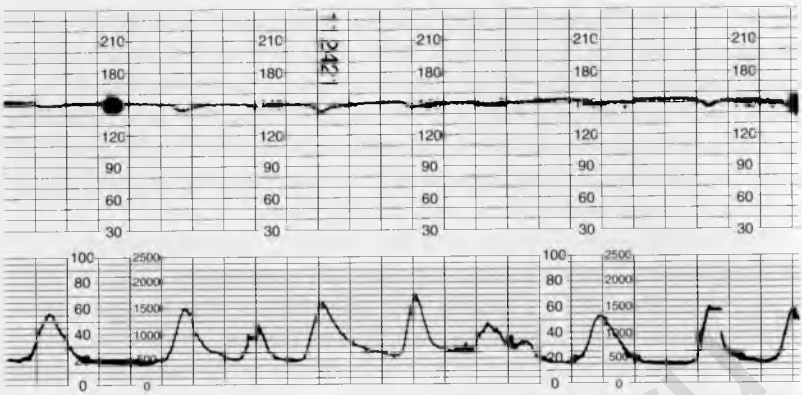

Figure 4-31. Grossly pathological trace.

late decelerations less than 15 bpm. This is an ominous picture unless the trace has shown recent reactive segments. The clinical picture has to be considered and, at times, an immediate delivery is opted for on clinical grounds. All the features of a given trace must be considered before it is categorized as normal, suspicious/non-reassuring or pathological/abnormal. The subsequent management of patients depends on this.

Shallow decelerations with reduced baseline variability in the 'quiet epoch' following an 'active epoch' with accelerations have been found to be associated with fetal breathing episodes.[5] If, on admission or commencement of the CTG, there is reduced baseline variability and shallow decelerations, one should look for clinical symptoms and signs that might suggest possible hypoxia or other insults (e.g. reduced or absent fetal movements, infection, intrauterine growth restriction, prolonged pregnancy or vaginal bleeding). If no such symptoms or signs are evident but mother is at, or close to, term and is in early labour, artificial rupture of membranes may reveal thick meconium with scanty fluid highlighting possible compromise and the need for delivery. In the absence of the above and if the baseline variability is at least 3–5 bpm, it may be acceptable to wait for up to 40 and a maximum of 90 min for the next active epoch with accelerations to become evident. In early labour, if facilities permit, use of ultrasound to observe the quantity of amniotic fluid, fetal body or breathing movements and fetal tone may be useful. If it is in the antenatal period and the fetus is preterm, it may be prudent to undertake the above biophysical assessment and also to determine fetal growth and examine the blood flow in the umbilical arteries and the fetal vessels. If these facilities or expertise are not available then consideration should be given to delivery based on the clinical features and fetal maturity.

- Accelerations and normal baseline variability are the hallmarks of fetal health.
- A hypoxic fetus can have a normal baseline rate, other features being abnormal.
- In the absence of accelerations, repeated shallow decelerations (below 15 bpm) are ominous when baseline variability is less than 5 bpm.

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PATHOPHYSIOLOGY OF FETAL HEART RATE (FHR) PATTERNS

Donald Gibb, Sabaratnam Arulkumaran

Control of the fetal heart rate and the associated features of accelerations, decelerations and baseline variability is complex (Fig. 5-1). The baseline fetal heart rate is determined by the spontaneous activity of the pacemaker in the sinoatrial (SA) node in the atrium. This specialized area of the myocardium initiates the fastest rate and determines the 'baseline' rate in the normal heart. The atrioventricular (AV) node situated on the atrioventricular septum has a slower rate of activity and generates the idioventricular rhythm seen in complete heart block. Under the circumstances of complete heart block the ventricle beats at 60–80 beats per minute (bpm).

The fetal heart rate (FHR) is modulated by a number of stimuli. Central nervous system influence is important with cortical and subcortical influences not under voluntary control. We cannot alter our heart rate at will. The cardioregulatory centre in the brain stem also plays a part. Other physiological factors regulate the heart rate, such as circulatory catecholamines, chemoreceptors, baroreceptors and their interplay with the autonomic nervous system.[1]

The efferent component of the autonomic nervous system is composed of the sympathetic and parasympathetic systems. There is a constant input from these systems varying from millisecond to millisecond. Sympathetic impulses drive the heart rate to increase while parasympathetic impulses have the opposite effect. If we are confronted with a frightening situation our heart rate involuntarily increases. This puts us under stress, sometimes distress; however, it is an adaptive mechanism preparing us for fight or flight – the sympathetic response. On the contrary, if we are feeling very relaxed and happy at home in the evening after a busy day then our heart rate will decrease on account of parasympathetic stimulation.

Electronic FHR monitors compute the heart rate based on averaged intervals between beats extrapolated to what the rate would be if that beat interval remained constant. The machine produces a rate recording after being applied for only a few seconds. However, autonomic impulses immediately and constantly take effect, changing the beat intervals and immediately altering the heart rate. This is

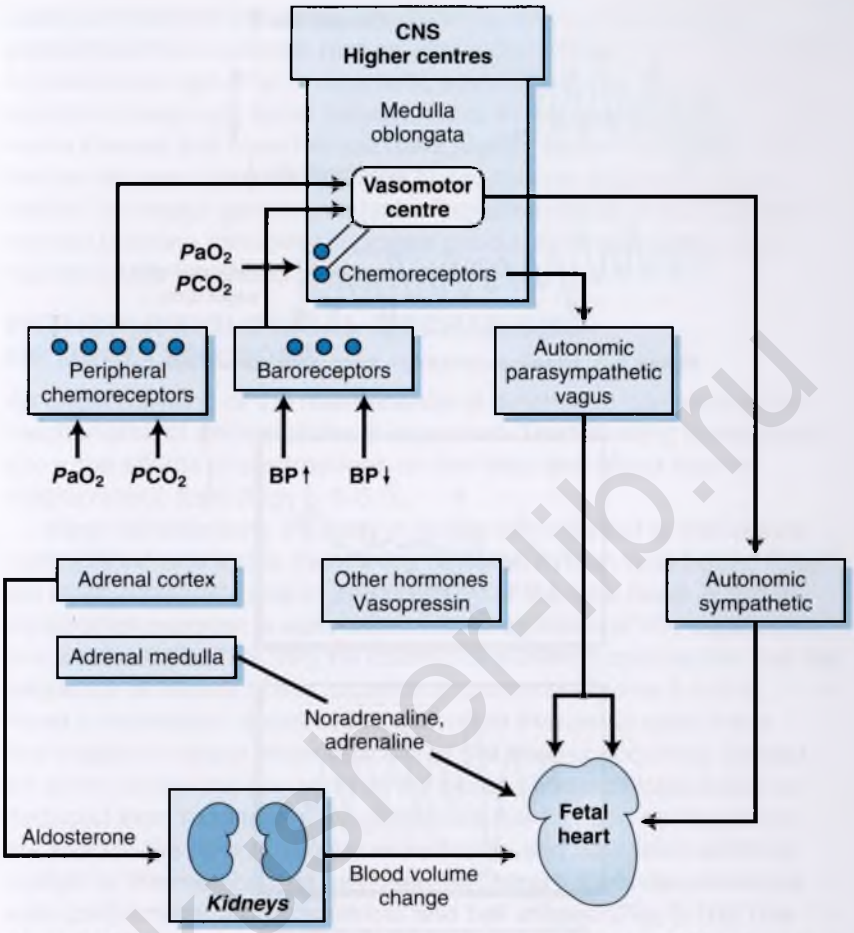

Figure 5-1. Control of the fetal heart. CNS, central nervous system; BP, blood pressure.

how baseline variability is generated and it indicates integrity of the autonomic nervous system (Fig. 5-2). Baseline variability is actually seen on the tracing. If it is greatly magnified, individual beats, and beat-to-beat variation, can be seen with special equipment used for physiological studies (Fig. 5-3). In practice though, baseline variability is the preferred term. The sympathetic nervous system and the parasympathetic or vagal system have the specific effect of generating baseline variability. Suppression of vagal impulses by a drug such as atropine causes tachycardia and reduces baseline variability. Physiological mechanisms are complex and incompletely understood. The autonomic nervous system is sensitive to hypoxia at a critical level for the fetus and changes in this response are therefore

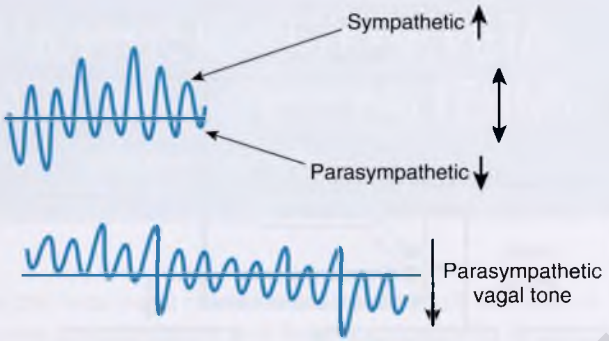

Figure 5-2. Baseline variability – autonomic modulation.

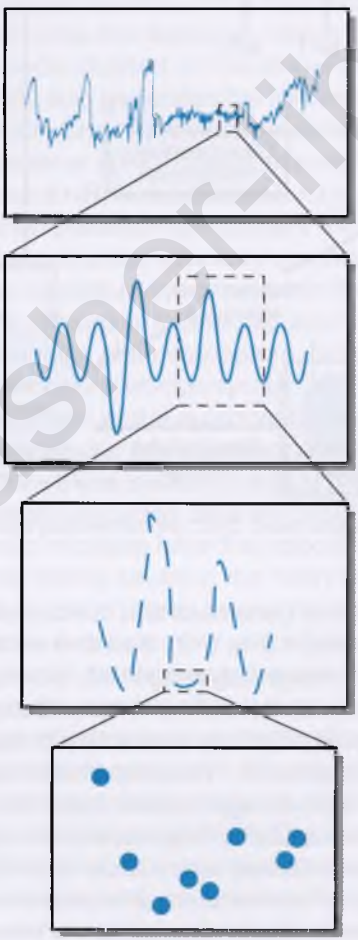

Figure 5-3. Baseline variability – beat-to-beat variation.

used as important indicators of wellbeing. The sympathetic and parasympathetic systems mature at slightly different rates with respect to gestational age. The sympathetic system matures faster and this results in marginally faster baseline rates in the preterm period. It is of some interest that male fetuses have slightly faster heart rates than female fetuses; however, this is of absolutely no diagnostic value. Before 34 weeks' gestation a higher baseline rate is to be expected. Normal baseline variability suggests good autonomic control and therefore little likelihood of hypoxia.

PATHOPHYSIOLOGICAL MECHANISMS OF DECELERATIONS

An understanding of the maintenance of autonomic control and the mechanisms of decelerations is important. The following illustrations show the effects of contractions on the fetus and blood flow in diagrammatic form (Figs 5-4–5-9).

Early decelerations are early in timing with respect to the uterine contractions and this is therefore a better term than type I dips. They are most commonly due to compression of the fetal head. A rise in intracranial pressure is associated with stimulation of the vagal nerve and bradycardia. This may be caused by a uterine contraction and the sequence of events in this situation is shown in Figures 5-5–5-9. Head compression decelerations are most frequently seen in the late stages of labour when descent of the head is occurring. Indeed, on some occasions the onset of the second stage of labour can be deduced from the tracing. Decelerations due to head compression are seen at the time of vaginal examination and also when artificial rupture of the membranes has been performed. Early decelerations with contractions are symmetrical and bell shaped (Fig. 5-10). The clinical situation should be reviewed to ensure that head compression

Figure 5-4. Diagrammatic representation of fetus, placenta and blood flow.

Figure 5-5. Early deceleration – start of contraction.

Figure 5-6. Early deceleration – increasing contraction.

Figure 5-7. Early deceleration – peak of contraction.

Figure 5-8. Early deceleration – decreasing contraction.

Figure 5-9. Early deceleration – end of contraction.

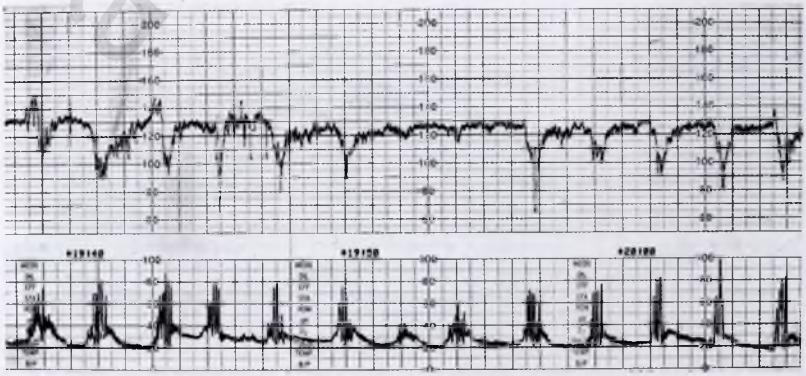

Figure 5-10. Example of early decelerations.

is a likely explanation at that time. If not, and if the trace is atypical, then an apparently innocuous early deceleration may be an atypical variable deceleration and may be pathological. In one case the obstetric registrar reported that a young West Indian nullipara suffering from sickle cell disease at term but with abdominal size and scan suggesting intrauterine growth restriction was 'niggling' but not yet in established labour. The fetal head was unengaged. He reported the trace (Fig. 5-11) as showing early decelerations. He wished to proceed to induction of labour but the consultant suggested he proceed directly to caesarean section. The registrar was surprised but learned an important lesson on delivering a significantly growth-restricted baby covered in meconium with Apgar scores of 5 and 6 who made a satisfactory recovery. Review of the trace the following day showed that although the decelerations might be described by some as early they do show poor recovery of the second one (atypical variable deceleration), no accelerations and a suggestion of reduced variability after the second deceleration. What is more important is that this fetus had no reason to have head compression and also had a background of risk.

Late decelerations are late in timing with respect to the uterine contraction and are therefore best described as such rather than as

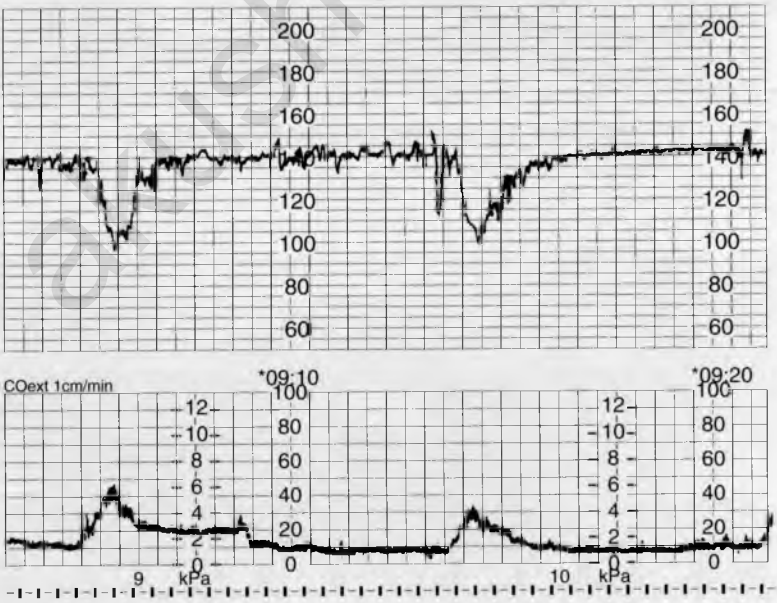

Figure 5-11. Pathological 'early' deceleration (more likely to be atypical variable) – head 4/5 to 5/5 palpable.

type 2 dips. The suggested pathophysiological mechanism of such decelerations is shown in Figures 5-12–5-14. There is a reservoir of oxygenated blood in the retroplacental space. The size of this space varies and is smaller in intrauterine growth restriction. Poor blood flow to the uteroplacental space is characteristic of fetuses with intrauterine growth restriction. As a contraction begins the fetus uses up the reservoir of oxygen in the retroplacental space. Due to the restricted supply of blood a hypoxic deceleration begins (usually 20 seconds after the onset of the contraction), it continues through the contraction and it does not recover fully until some time after the contraction when full oxygenation has been restored. The speed of recovery on the ascending limb may reflect the blood flow and the resilience of the fetus. In a non-hypoxic fetus there is increased variability during a deceleration on account of autonomic response. When hypoxia develops there is a tendency to reduced variability.

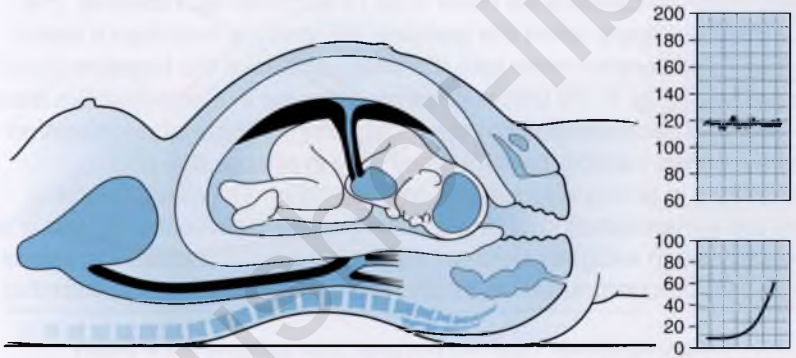

Figure 5-12. Late deceleration – start of contraction.

Figure 5-13. Late deceleration – after peak of contraction.

Figure 5-14. Late deceleration – end of contraction.

Baseline Variability and Decelerations – Exception to the Rule

A deceleration is defined when the FHR decelerates by more than 15 beats from the baseline for more than 15 seconds (s). However, this rule does not apply when the baseline variability is less than 5 beats and any deceleration even less than 15 beats from the baseline could be ominous (Fig. 5-15) unless otherwise proven in a non-reactive trace.

Variable decelerations are the most common type of deceleration and are called variable because they vary in shape, size and sometimes in timing with respect to each other. They vary because they are a manifestation of compression of the umbilical cord and it is compressed in a slightly different way each time. On some occasions it may not be compressed at all and there is no deceleration with that

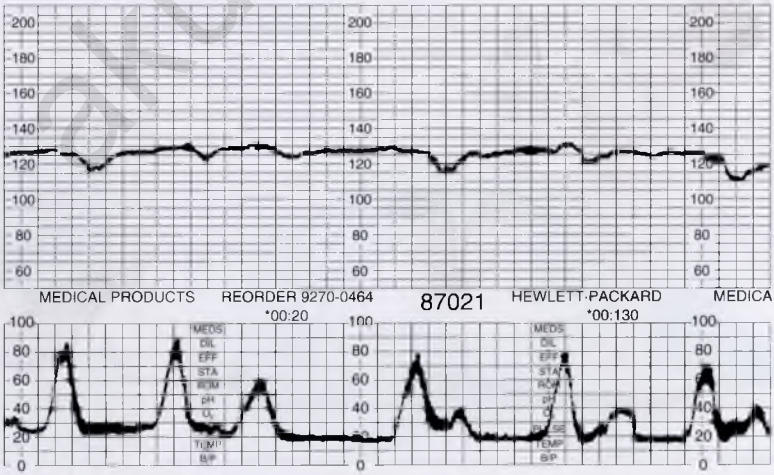

Figure 5-15. Ominous shallow deceleration with baseline variability <5 bpm.

particular contraction. Variable decelerations are more often seen when the amniotic fluid volume is reduced. In North America they are referred to as cord compression decelerations.

The mechanism is illustrated in Figures 5-16–5-20. The umbilical vein has a thinner wall and lower intraluminal pressure than the umbilical arteries (Fig. 5-16). When compression occurs the blood flow through the vein is interrupted before that through the artery. The fetus therefore loses some of its circulating blood volume. When a healthy individual or fetus loses some of its circulating blood volume the natural response effected by the autonomic nervous system is a rise in pulse rate to compensate. A small rise in the FHR therefore appears at the start of a variable deceleration when the fetus is not

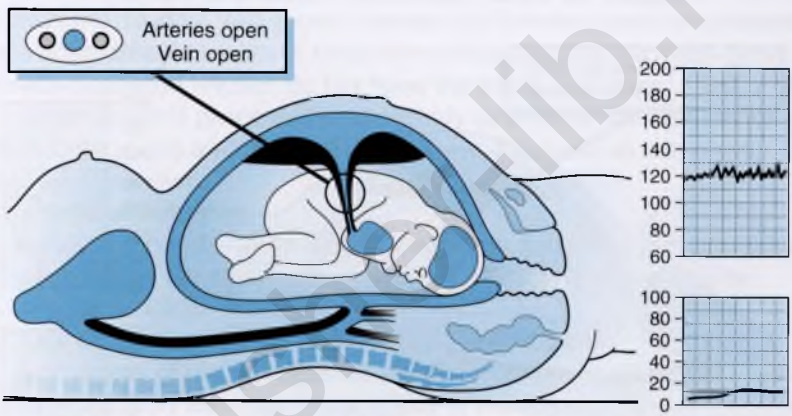

Figure 5-16. Umbilical cord, fetus and placenta – normal circulation.

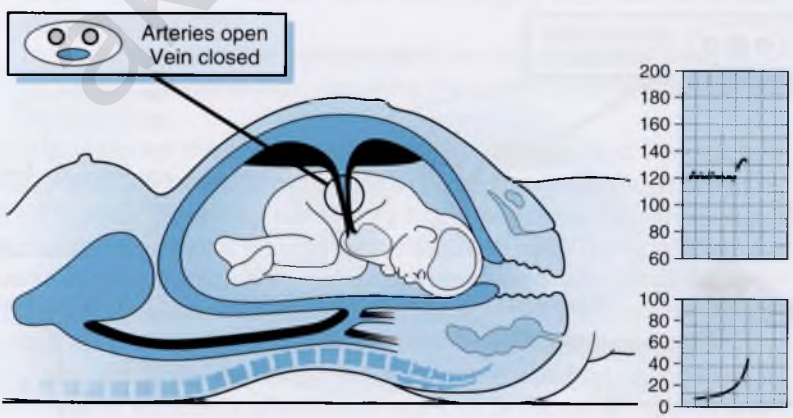

Figure 5-17. Variable deceleration – start of contraction.

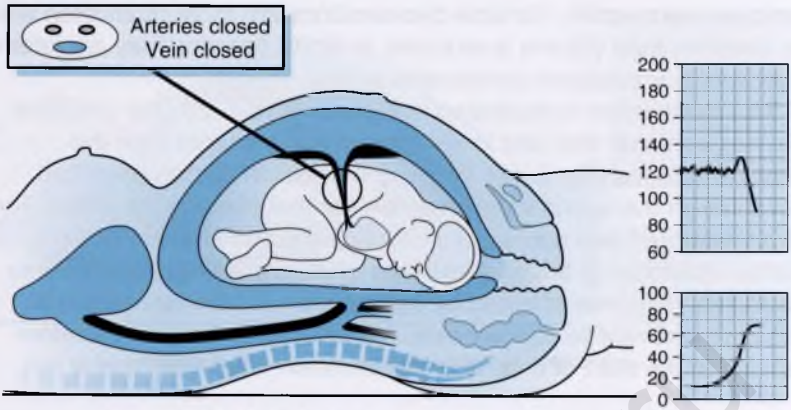

Figure 5-18. Variable deceleration – increasing contraction.

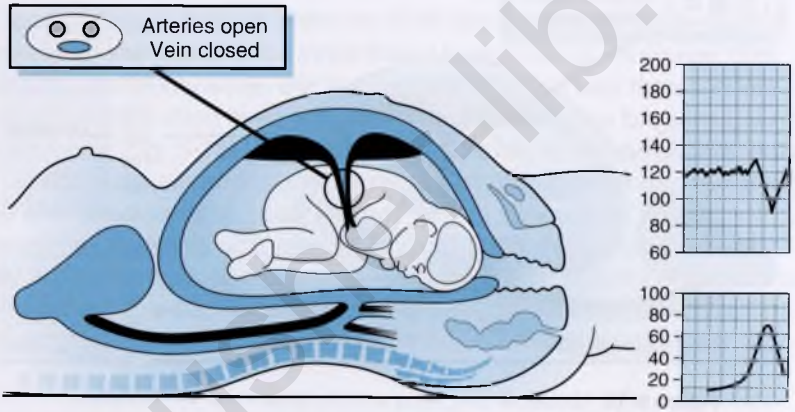

Figure 5-19. Variable deceleration – decreasing contraction.

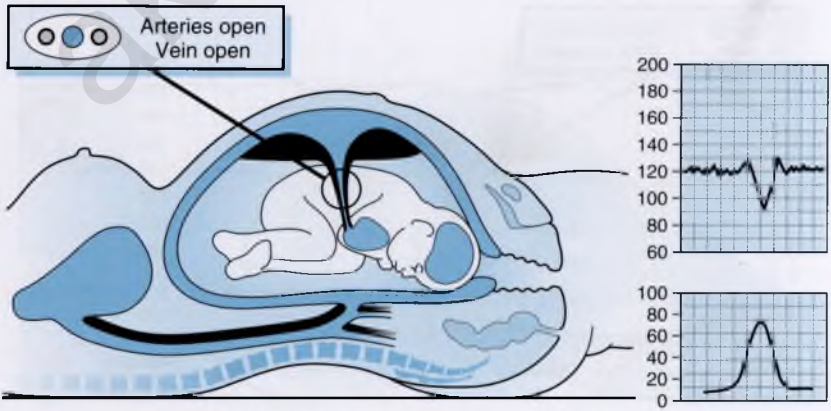

Figure 5-20. Variable deceleration – end of contraction.

compromised (Fig. 5-17). After that the umbilical arteries are also occluded, the circulation is relatively restored followed by an increase in systemic pressure, the baroreceptors are stimulated and there is a precipitous fall in the FHR (Fig. 5-18). The deceleration is at its nadir with both vessels occluded. During release of the cord compression, arterial flow is restored first with a consequent autonomically mediated sharp rise in heart rate (Fig. 5-19) due to systemic hypotension of blood being pumped out culminating in a small rise in FHR after the deceleration (Fig. 5-20). These rises in FHR before and after decelerations are called shouldering. Whatever they are called, they are a manifestation of a fetus coping well with cord compression. The way the cord is being compressed will vary depending exactly on how it is positioned with respect to the structure compressing it. On the same basis, variable decelerations may change if the posture of the mother is changed. Normal well-grown fetuses can tolerate cord compression for a considerable length of time before they become hypoxic. Small growth-restricted fetuses do not have the same resilience.

To assess this process it is necessary to analyse the features of the decelerations and also the character of the trace as it evolves. Figure 5-21 shows:

1. normal shouldering
2. exaggeration of shouldering or overshoot (indicates that additional circulations are needed to normalize) – which is thought to be prepathological
3. loss of shouldering – pathological
4. smoothing of the baseline variability within the deceleration – which is associated with loss of variability at the baseline and is therefore pathological
5. late recovery (variable and late deceleration components merged together) – which has the same pathological significance as late deceleration
6. biphasic deceleration (variable and late decelerations seen as separate components) – requiring the same consideration as a late deceleration.

If the duration of the deceleration is more than 60 s and the depth is greater than 60 beats, progressive hypoxia becomes more likely.

At times a fetus may have more than one stress operating (e.g., a fetus with intrauterine growth restriction may have cord compression due to oligohydramnios and late decelerations due to reduced amount of retroplacental blood behind the small and partially infarcted placenta). The most critical feature, however, is the evolution of the trace with time. A change in the baseline rate and change in the baseline variability are the key signs of developing hypoxia and acidosis.

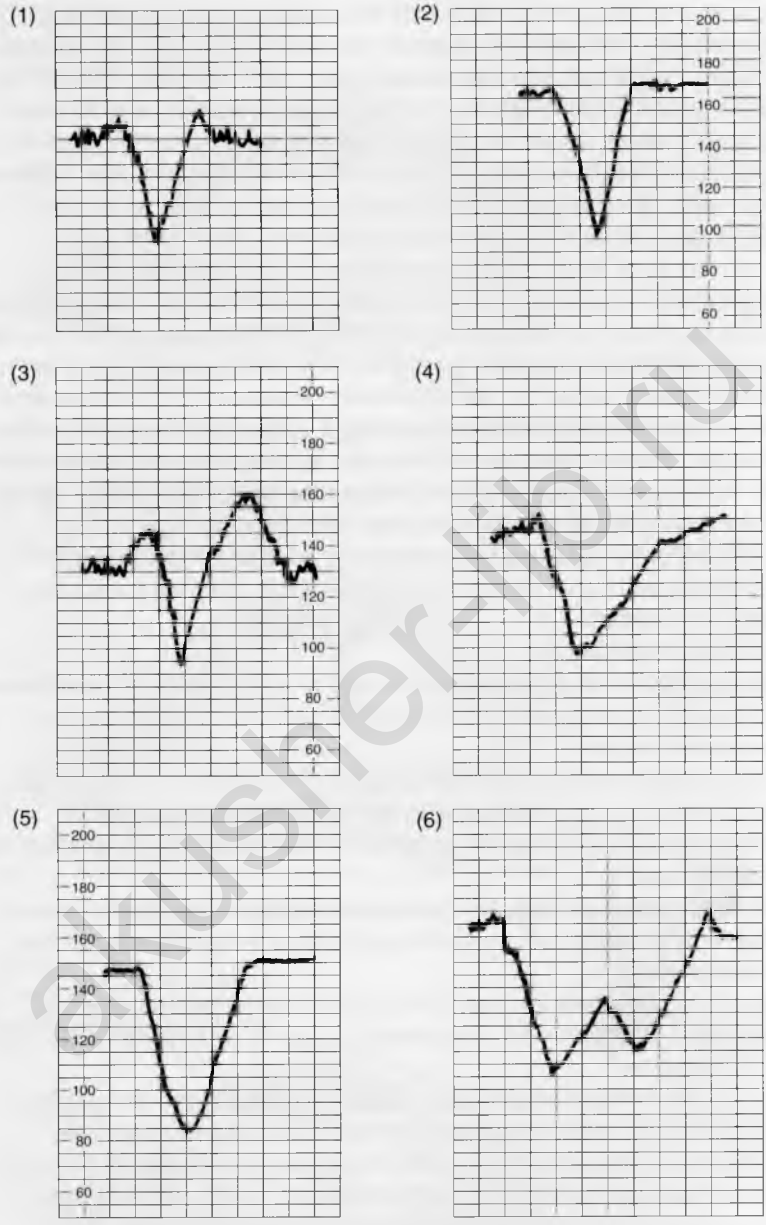

Figure 5-21. Features of variable decelerations.

Figure 5-22 shows two strips of CTG 60 min apart. In spite of marked variable decelerations, the baseline rate and baseline variability are maintained. So long as adequate progress is being made towards delivery this trace need not cause concern. Figure 5-23 also shows

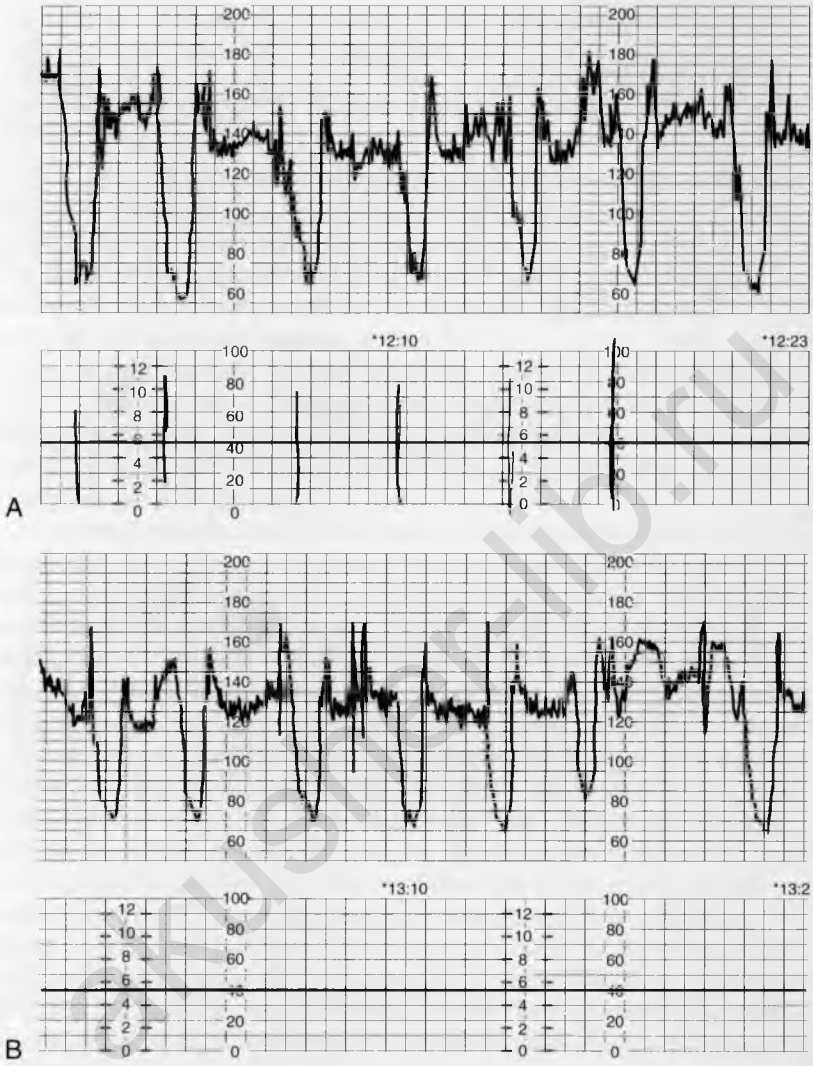

Figure 5-22. Two CTGs recorded 60 min apart, showing variable decelerations. There is no rise in the baseline rate or variability. Occasional decelerations with beat loss >60 and duration >60 s need close observation.

two strips of trace 20 min apart but with quite different features. The progression to a tachycardia with reduced variability suggests developing hypoxia. The time required for a fetus with a previously normal trace to become acidotic related to different patterns of the FHR has been studied.[2] In many cases it may take over 100 min.

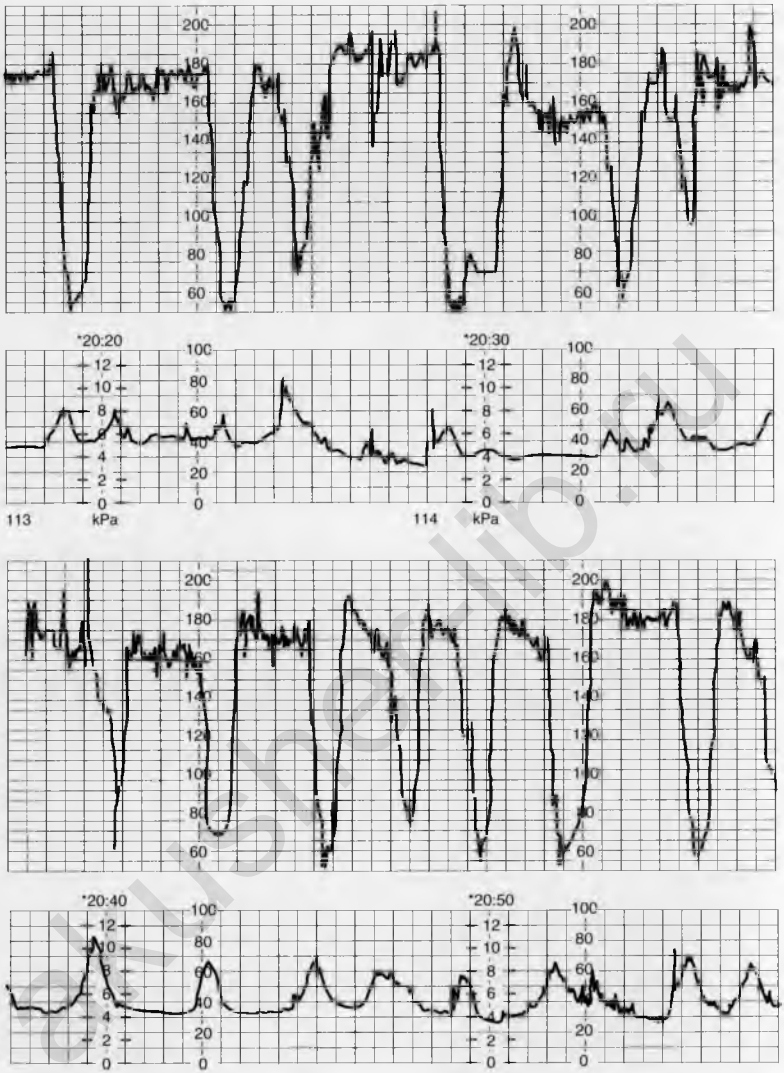

Figure 5-23. Two CTGs recorded 20 min apart: (A) variable decelerations with abnormal features (duration >60 s, depth >60 beats; tachycardia) (abnormal trace); (B) suggestive of distress (tachycardia and reduced baseline variability).

Medical staff should have time enough to identify the problem and act effectively.

At times it may be difficult to decide whether the decelerations are early, late or variable. It is not only the deceleration itself that is critical but also the evolution of the trace with time and the clinical features.

The baseline rate between decelerations, the baseline variability and the presence or absence of accelerations are all critical.

CLASSIFICATION OF FETAL HEART RATE PATTERN

The NICE and FIGO guidelines recommend classifying the individual features of baseline rate, baseline variability, decelerations and accelerations as reassuring, non-reassuring or abnormal (see Ch. 6). The whole CTG trace is then classified as normal or reassuring, non-reassuring or suspicious, and abnormal or pathological. A summary of the above is given in Box 5-1.

A sinusoidal pattern is a regular heart rate with cyclic changes in the FHR baseline like a sine wave, the characteristics of the pattern being that the frequency is less than 6 cycles per min, the amplitude is at least 10 bpm and the duration should be 10 min or longer. Additional details are given in Chapter 6.

A *normal* classification of the trace implies that the trace assures fetal health. *Suspicious* indicates that continued observation or additional simple tests are required to ensure fetal health. *Pathological* warrants some action in the form of additional tests or delivery depending on the clinical picture. If one of the features of the CTG is abnormal, possible remedial action should be taken and, at times, a short period of observation of the trace may be appropriate if there are no clinical risk factors like intrauterine growth restriction, meconium or infection. If there are clinical risk factors or two abnormal features, additional testing such as fetal scalp blood sampling to elucidate the fetal condition, or delivery, may be more prudent if remedial action does not correct the abnormal features of the trace. If three features of the CTG are abnormal, one should consider delivery unless spontaneous delivery is imminent, or perform fetal scalp blood sampling to elucidate the fetal condition. The degree of abnormality of the CTG, clinical risk factors, parity, current cervical dilatation and the rate of progress of labour should determine the decisions to observe, perform a fetal scalp blood sample or deliver promptly. Table 6.1

Box 5-1. Cardiotocograph Classification

Normal/reassuring – A CTG where all *four* features fall into the reassuring category

Suspicious/non-reassuring – A CTG whose features fall into *one* of the non-reassuring categories and the remainder of the features are reassuring

Pathological/abnormal – A CTG whose features fall into *two or more* non-reassuring categories or *one or more* abnormal categories

describes the recommended actions with different categories of FHR patterns observed.

The expression *fetal distress* should be reconsidered. A trace that is not normal may result from physiological, iatrogenic or pathological causes. The clinical situation and the dynamic evolution of features of the trace with time will clarify the situation.

The underlying principle is to detect fetal compromise using the concept of 'fetal distress' very critically. In all situations, it is consideration of the overall clinical picture that will provide the clues as to whether true fetal compromise is present. Many suspicious CTGs are generated by healthy fetuses demonstrating the ability to respond to stress. For the purposes of clinical decision making, so far, scoring systems or computer analysis have not been found to be useful, particularly in the intrapartum period. Results of a large randomized control trial consisting of 45 000 women (INFANT study) are awaited to decide whether computer-assisted interpretation of CTG would be useful.

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NICE AND FIGO GUIDELINES FOR INTERPRETATION OF FHR PATTERNS

Edwin Chandraharan, Diogo Ayres de Campos

Evidence-based medicine is defined as 'the conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients'. Clinical guidelines refer to a series of 'evidence-based statements' that are intended to avoid variation in clinical practice so as to ensure the best clinical care based on the current 'best evidence'.

Clinicians caring for women and babies during labour need to appreciate the fact that human labour is a rapidly evolving dynamic process that involves changes in the frequency, duration and strength of the uterine contractions. This may result in varying degrees of compression of the umbilical cord and reduction in the oxygenation of the uteroplacental venous sinuses, with each uterine contraction leading to a normal CTG trace rapidly becoming 'pathological' and vice versa. Therefore, irrespective of the classification of the CTG into 'normal, suspicious or pathological' (FIGO) or 'normal, non-reassuring or abnormal' (NICE) or 'Cat 1, Cat 2 or Cat 3' (American Congress of Obstetricians and Gynecologists, ACOG), intrapartum healthcare providers should always improve the intrauterine environment (i.e., changes in maternal position to relieve ongoing umbilical cord compression, intravenous fluids to correct maternal hypotension and reducing or stopping oxytocin infusion to improve uteroplacental circulation) prior to undertaking any invasive procedures (e.g., fetal scalp blood sampling) or an operative intervention (e.g., operative vaginal birth or an emergency caesarean section) based on the classification of the CTG trace.

Irrespective of the classification of the CTG trace, the 'wider clinical picture' should be considered. This includes the rate of progress of labour, physiological reserve of the individual fetus in question (e.g., IUGR, postdates, pre-eclampsia), and presence of meconium staining of amniotic fluid or intrapartum bleeding.

Scientific studies have suggested that deep variable decelerations, bradycardia and loss of baseline variability (i.e., indicating fetal asphyxia) are associated with meconium aspiration syndrome and poor

perinatal outcomes.[1] Therefore, in the presence of meconium staining of amniotic fluid, the presence of oxytocin-induced deep, variable decelerations may lead to poor perinatal outcomes, even if the CTG trace does not meet the threshold to be classified as 'pathological' or 'abnormal'. Similarly, in clinical chorioamnionitis, the pathway of fetal neurological damage is 'fetal systemic inflammatory response' (FIRS) and not intrapartum hypoxia. Therefore, even in the absence of significant decelerations, fetal neurological damage may still occur if labour is allowed to continue with the use of oxytocin (i.e., additional hypoxic insult) despite the CTG trace being classified as 'normal' according to guidelines. A rise in baseline fetal heart rate (FHR) from 110 beats per minute (bpm) to 150 bpm secondary to an evolving hypoxia or ongoing fetal infection is significant to the individual fetus, even though the baseline may be still within the 'normal range' (i.e., 110–160 bpm) as stipulated by the guidelines.

NICE and FIGO guidelines are an addition to an obstetrician's and a midwife's intrapartum toolbox. However, they should not be *blindly* applied in every case without considering the wider clinical picture and the fact that fetal hypoxia may evolve rapidly during the second stage of labour.[2] Hence, 'judicious' use of guidelines on CTG classification is essential. In this chapter, the authors have critically appraised both the NICE and the FIGO guidelines on CTG classification. However, the crux of the matter is not what clinical guideline one uses or the superiority of one specific CTG classification system over the other, but rather its interpretation. The aim is to distinguish a fetus who is normally oxygenated or one who is exposed to intrapartum hypoxic (i.e., late decelerations) or mechanical stress (i.e., early or variable decelerations) and is compensating well from one who has exhausted all the means of compensation and is showing signs of decompensation on the CTG trace.

The role of a clinical guideline on CTG is to ensure that it is not only 'evidence based', but also that it is simple, objective and avoids errors of inter- and intraobserver variability and aids midwives and obstetricians, even at 02:00 am, to appropriately identify a fetus who needs an urgent intervention and, conversely, allows continuation of labour without any unnecessary operative interventions in a fetus who shows no evidence of decompensation.

CRITICAL APPRAISAL OF THE NICE GUIDELINES

The updated NICE guideline 'Intrapartum care for healthy women and babies' (December 2014) has a detailed and impressive analysis of 'evidence base' on fetal monitoring during labour.[3] However, its classification system (see Table 10 in Ref. 3) appears very complicated, with several arbitrary time limits. One needs to appreciate the fact that a growth-restricted fetus may not

withstand variable decelerations for 50% of contractions for 30 or 90 minutes (min) as proposed by the NICE guideline to avoid hypoxic damage. In the authors' personal opinion, the more complicated a clinical guideline, the more likely it is that it will lead to inter- and intraobserver variation in interpretation, in turn leading to either an unnecessary intrapartum operative intervention or poor perinatal outcomes. This is an important criticism.

The biggest flaw in the NICE guideline is the single number (>5 bpm) for baseline FHR variability. All biological parameters (baseline FHR, haemoglobin, white cell count, etc.) should have a 'normal range' with lower and upper limits. Similarly, the baseline FHR variability should also be a range (5–25 bpm) because when there is a rapidly evolving intrapartum hypoxic stress (e.g., with use of oxytocins or maternal active pushing) a fetus may not have sufficient time to increase the baseline heart rate to compensate for this, and therefore would demonstrate an increase in baseline variability (i.e., the 'saltatory pattern') on the CTG trace, which would require immediate measures to improve fetal oxygenation (e.g., stopping oxytocin, use of tocolytics, stopping active maternal pushing). Scientific research suggests that acutely evolving hypoxic stress is associated with an increase in baseline FHR variability.[4,5] Therefore, clinicians should be very cautious as a baseline variability of 50 bpm may be normal according to NICE guidelines (>5 bpm), but it may signify fetal autonomic instability secondary to a rapidly evolving hypoxia.

NICE guidelines have also used confusing terminologies such as 'bradycardia' to describe a single prolonged deceleration that persists for more than 3 min. The term *bradycardia* should be used only for a baseline change (i.e., baseline FHR <110 bpm) that persists for more than 10 min. Similarly, the term 'non-reassuring' variable decelerations may also be confusing to midwives and junior doctors.

In our opinion, the 'management' table proposed by NICE (see Table 11 in Ref. 3) may also lead to confusion because it appears very complicated, crowded and not user friendly. The recommendation that 'fluids and paracetamol' should be offered when baseline fetal tachycardia is observed may result in missing those fetuses with subclinical or clinical chorioamnionitis. This is because chorioamnionitis is a fetal and not a maternal disease and one may not observe clinical signs in the mother (i.e., maternal tachycardia and pyrexia) until very late in the fetal disease process. In addition, some fetuses who are at term may have a very low baseline FHR owing to vagal dominance, and therefore they may increase their baseline heart rate from 110 bpm to 155 bpm. Hence they may be classified as 'normal' because the baseline FHR is still within the NICE guideline range (i.e., 110–160 bpm).

NICE guideline recommends fetal scalp sampling for a 'pathological' CTG trace after concluding that the use of CTG with fetal blood sampling (FBS) is associated with *significantly more* instrumental vaginal births and emergency caesarean sections.[2] The physiological[6] and the clinical[7] role of FBS has recently been questioned. In view of rare but potentially very serious fetal complications of FBS, clinicians should cautiously re-examine its anatomical, physiological and scientific evidence before performing this historical test.[8] Considering that fetal digital stimulation is superior to other invasive tests such as scalp puncture[9] and reduces the subsequent need for FBS in 73% of cases,[10] healthcare providers should consider digital stimulation of the fetal scalp prior to considering other additional tests of fetal wellbeing if there are concerns regarding fetal wellbeing on the CTG trace.

The overall care proposed by the NICE guidelines offers some guidance for management in general, except for the section on FBS. Digital stimulation of the fetal scalp is non-invasive and provides good information compared with FBS and therefore, contrary to what is stated by NICE guidelines, this should be performed first if there are any concerns about fetal wellbeing.

Irrespective of the individual guideline on the CTG trace (NICE, ACOG or FIGO), if a fetus continues to demonstrate a stable baseline heart rate and a reassuring variability then the risk of acidosis is low. In addition, if a fetus spends more time on the baseline as opposed to during decelerations (i.e., no evidence of a subacute hypoxic pattern), it indicates that sufficient time is available to obtain fresh oxygen from the placenta and to expel metabolic wastes, including accumulated carbon dioxide and acid, into the maternal circulation. Persistent fetal tachycardia, even as a part of fetal catecholamine response to hypoxic stress or inflammatory response to ongoing chorioamnionitis, may lead to a reduction in myocardial 'diastole' and the resultant failure of the myocardium to oxygenate itself, despite an increase in metabolic demands. This may lead to progressive myocardial decompensation resulting in a 'step-ladder' pattern to death. Therefore, even if the CTG is merely 'suspicious', persistent fetal tachycardia may lead to poor perinatal outcomes.

A recent study has shown that less than half of the midwives and obstetricians tested (sample size 810) correctly answered questions on NICE guidelines even after 5 years of its implementation.[11] This demonstrates the flaws of depending mainly on pattern recognition whilst classifying the CTG traces using 'guideline boxes' in labour. The knowledge of fetal pathophysiology[12] and rational use of CTG whilst understanding the types of intrapartum hypoxia[13] and fetal response to stress is essential to improve perinatal outcomes and to reduce

unnecessary operative interventions during labour. The role of the clinical guidelines on CTG interpretation should be to aid and facilitate this process.

2015 FIGO GUIDELINES ON INTRAPARTUM FETAL MONITORING

Guideline Development

The 2015 FIGO consensus guidelines on intrapartum monitoring[14–18] were developed with the purpose of updating the previous ones published in 1987,[19] using an accessible language as well as simple, objective and easy concepts to remember. The aim was to promote a common terminology that would be useful for research, and for the improvement of clinical care throughout the world. By including management options, the ultimate goal was to contribute to a reduction in perinatal mortality and long-term sequelae, while at the same time avoiding unnecessary obstetrical intervention.

The process involved a total of 50 experts, 34 nominated by FIGO national member societies, and 16 invited based on their publication record in the field. The process also involved the contribution of chapter authors appointed by ACOG, the Royal College of Obstetricians and Gynaecologists (RCOG), and the International Confederation of Midwives (ICM). A geographical representation of the members of the consensus panel is presented in Figure 6-1.

Figure 6-1. Geographical representation of the panel members of the 2015 FIGO consensus guidelines on intrapartum fetal monitoring.

The consensus process was conducted by email, and involved three rounds of agreement for each chapter, followed by written consent to be included in the panel list. The process involved no internal or external funding, and took 10 months to prepare and a further 18 months to conclude. The guidelines have been endorsed by the European Association of Perinatal Medicine (EAPM) and the European Board and College of Obstetrics and Gynaecology (EBCOG), and supported by ACOG.

In this chapter we provide a brief overview of the concepts presented in the cardiotocography chapter of these guidelines. The full document can be accessed at http://www.ijgo.org/article/S0020-7292(15)00395-1/pdf. Cardiotocography, from the Greek *kardia* meaning heart, and *tokos* meaning labour/childbirth, was judged to be the term that best describes the continuous monitoring of FHR and uterine contraction signals.

CTG ANALYSIS

CTG analysis starts with an evaluation of basic CTG features followed by overall tracing classification.

Evaluation of Basic CTG Features

Evaluation of basic CTG features comprises assessment of the FHR baseline, variability, accelerations, decelerations and other patterns, fetal behavioural state and contractions, and this process requires a solid comprehension of the underlying physiology.

BASELINE – the mean level of the most horizontal and less oscillatory FHR segments, estimated in periods of 10 min (can vary between different periods) and expressed in beats per minute (bpm). Care must be taken to identify the fetal behavioural state of active wakefulness (see below), which can lead to an erroneously high baseline estimation (see Fig. 6-8).

Normal baseline – between 110 and 160 bpm.

Tachycardia – baseline above 160 bpm for more than 10 min. Maternal pyrexia is the most frequent cause, but it may also be associated with epidural analgesia, the initial stages of fetal hypoxemia, administration of beta-agonist drugs, parasympathetic blockers, and fetal arrhythmias such as supraventricular tachycardia and atrial flutter.

Bradycardia – baseline below 110 bpm for more than 10 min. May be caused by acute fetal hypoxia/acidosis, maternal hypothermia, administration of beta-blockers, and fetal arrhythmias such as atrial–ventricular block.

VARIABILITY – oscillations in the FHR signal, evaluated as the average bandwidth amplitude of the signal in 1-min segments. There is a high degree of subjectivity in visual evaluation of this parameter, and therefore careful re-analysis is recommended in borderline situations.

Normal variability – between 5 and 25 bpm.

Reduced variability – below 5 bpm for more than 50 min in baseline segments (Fig. 6-2), or more than 3 min in decelerations. It may be caused by central nervous system hypoxia/acidosis, previous cerebral injury, infection, administration of central nervous system depressants or parasympathetic blockers. During the fetal behavioural state of deep sleep (see below – Fig. 6-7), variability is usually low, and on visual analysis may be classified as reduced. This state seldom exceeds 50 min, so waiting for its reversal will clarify the situation.

Increased variability (**saltatory pattern**) – exceeding 25 bpm for more than 30 min (Fig. 6-3). This pattern has been associated with hypoxia/acidosis of rapid evolution.

ACCELERATIONS – abrupt increases in FHR above the baseline, with >15 bpm in amplitude and >15 seconds (s) duration. Most accelerations coincide with fetal movements and are a sign of a neurologically responsive fetus without hypoxia/acidosis. The amplitude and frequency criteria before 32 weeks' gestation are lower (10 s and 10 bpm of amplitude).

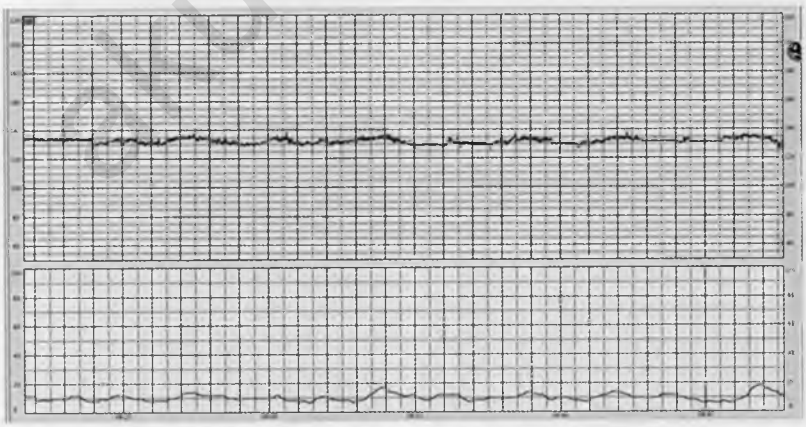


Figure 6-2. CTG displaying reduced baseline variability.

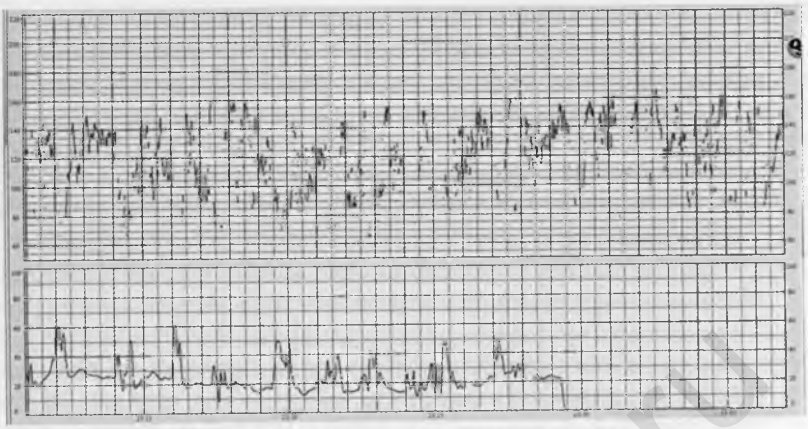
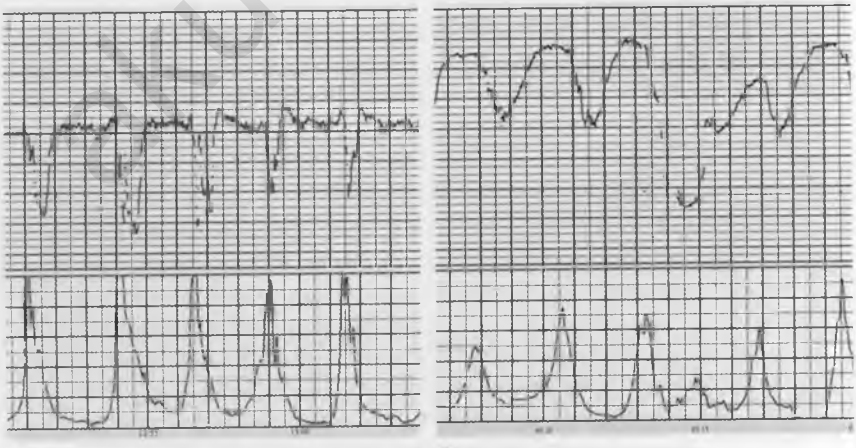


Figure 6-3. CTG displaying increased variability.

DECELERATIONS – decreases in FHR below the baseline, with >15 bpm in amplitude and >15 s duration.

Early decelerations – shallow, short-lasting, normal variability within the deceleration, and coincident with contractions. These are believed to be caused by fetal head compression, and do not indicate fetal hypoxia/acidosis.

Variable decelerations (V-shaped) – rapid drop, rapid recovery, good variability within the deceleration, varying size, shape and relationship to uterine contractions (Fig. 6-4A). These constitute the majority of decelerations, and translate a



A B

Figure 6-4. (A) CTG displaying variable decelerations; (B) CTG displaying late decelerations.

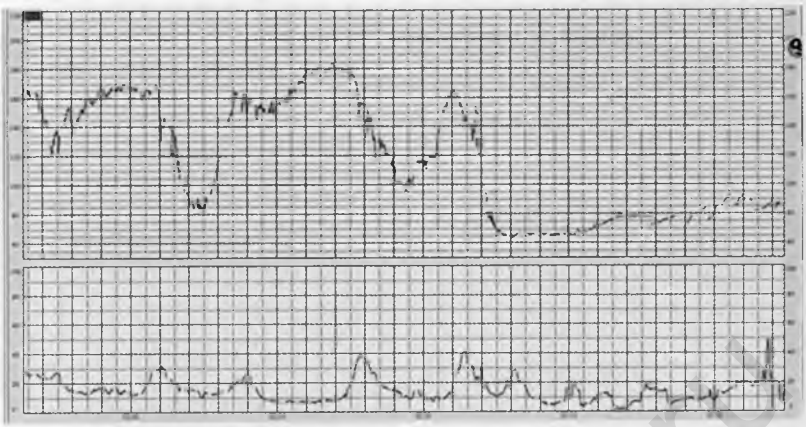


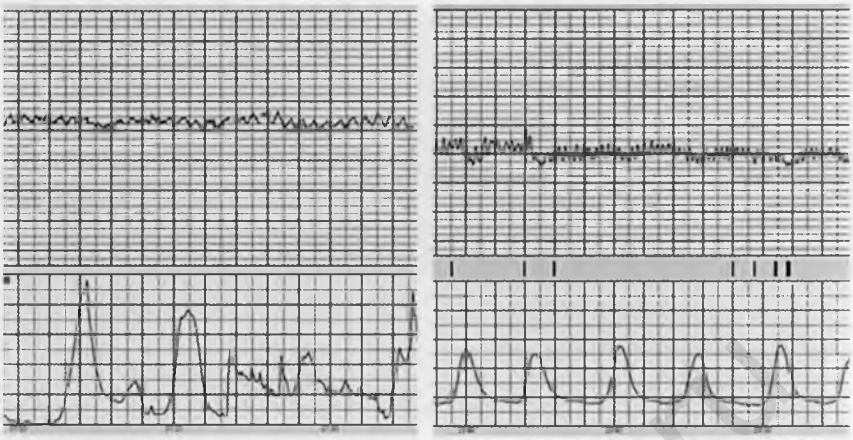
Figure 6-5. CTG displaying a prolonged deceleration with more than 5 min duration, requiring urgent intervention.

baroreceptor-mediated response to increased arterial pressure, as occurs with umbilical cord compression. When isolated, they are seldom associated with an important degree of fetal hypoxia/acidosis.

Late decelerations (**U-shaped** or with **reduced variability**) – gradual onset, or gradual return, or reduced variability within the deceleration (Fig. 6-4B); starting more than 20 s after the onset of a contraction, nadir after the acme, and recovery after the end of the contraction. These are indicative of a chemoreceptor-mediated response to fetal hypoxemia. In a tracing with no accelerations and reduced variability, late decelerations also include those with an amplitude of 10–15 bpm.

Prolonged decelerations – lasting more than 3 min. These are likely to include a chemoreceptor-mediated component and thus to indicate hypoxemia. If they exceed 5 min, with FHR <80 bpm and reduced variability (Fig. 6-5), they usually indicate acute fetal hypoxia/acidosis and require urgent intervention.

SINUSOIDAL PATTERN – a regular, smooth, undulating signal, resembling a sine wave, with amplitude 5–15 bpm, and frequency 3–5 cycles per min, lasting more than 30 min (Fig. 6-6A). This pattern occurs more frequently with fetal anemia, but has also been associated with acute fetal hypoxia, infection, cardiac malformations, hydrocephalus and gastroschisis. A similar pattern, but with a more jagged 'saw-tooth' signal, is called pseudosinusoidal (Fig. 6-6B). The latter is not associated with fetal



A B

Figure 6-6. (A) CTG displaying a sinusoidal pattern; (B) a CTG displaying a pseudosinusoidal pattern.

risk, and has been described after analgesic administration, and during periods of fetal sucking and other mouth movements. It lasts less than 30 min, and has normal patterns before and after.

FETAL BEHAVIOURAL STATES – refers to fetal periods of deep sleep, alternating with active sleep, and wakefulness. The occurrence of different behavioural states is a hallmark of neurological responsiveness and absence of hypoxia/acidosis. Deep sleep can last up to 50 min and the CTG displays a stable baseline, rare accelerations and low variability (Fig. 6-7). Active sleep is the most

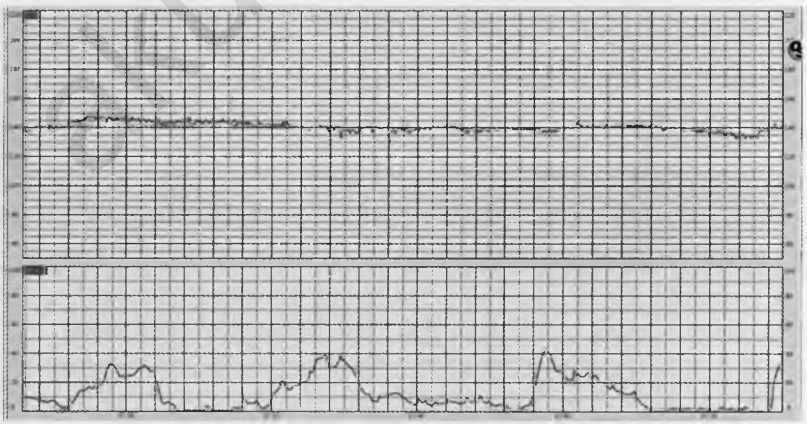


Figure 6-7. CTG representing the fetal behavioural state of deep sleep, which can last up to 50 min. This pattern is difficult to distinguish from that of reduced variability (Fig. 6-2). Only waiting for the end of this state will clarify the situation.

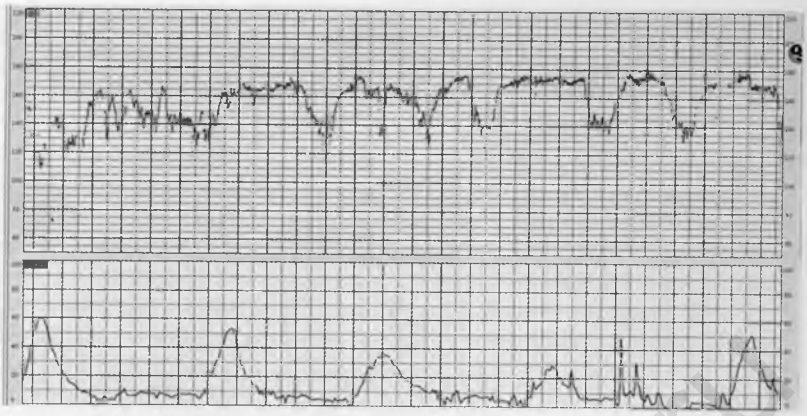


Figure 6-8. CTG representing the fetal behavioural state of active wakefulness. An erroneously high baseline may be identified, if judged to be at the top of accelerations. In this case the baseline is around 145 bpm.

frequent behavioural state, and is represented by accelerations and normal variability. Active wakefulness is rarer and is represented by many accelerations and normal variability (Fig. 6-8). Transitions between the different patterns become clearer after 32–34 weeks of gestation.

CONTRACTIONS – bell-shaped gradual increases in the uterine activity signal followed by roughly symmetric decreases, with 45–120 s in total duration. With the tocodynamometer, only the frequency of contractions can be reliably evaluated. The presence of more than five contractions in 10 min, in two successive 10-min periods, or averaged over a 30-min period, is called tachysystole.

Tracing Classification

Tracings should be classified into one of three classes: normal, suspicious or pathological, according to the criteria presented in Table 6-1. Because of the changing nature of CTG signals during labour, tracings should be re-evaluated for classification at least once every 30 min.

MANAGEMENT

When tracing characteristics (either basic CTG features or overall classification) are suggestive of an impending or already established fetal hypoxia/acidosis, action is required to avoid adverse neonatal outcome, but this does not necessarily mean immediate caesarean section or instrumental vaginal delivery. When a suspicious or worsening CTG pattern is identified, the underlying cause for this

Table 6-1 CTG Classification Criteria, Interpretation and Recommended Management[a]

	NORMAL	SUSPICIOUS	PATHOLOGICAL
Baseline variability	110–160 bpm 5–25 bpm		<100 bpm Reduced variability Increased variability Sinusoidal pattern
Decelerations	No repetitive[b] decelerations	Lacking at least one characteristic of normality, but with no pathological features	Repetitive[b] late or prolonged decelerations for >30 min (or >20 min if reduced variability). Deceleration >5 min
Interpretation	No hypoxia/acidosis	Low probability of hypoxia/acidosis	High probability of hypoxia/acidosis
Clinical management	No intervention necessary to improve fetal oxygenation state	Action to correct reversible causes if identified, close monitoring or adjunctive methods	Immediate action to correct reversible causes, adjunctive methods, or if this is not possible expedite delivery In acute situations, immediate delivery should be accomplished

[a] The presence of accelerations denotes a fetus that does not have hypoxia/acidosis, but their absence during labour is of uncertain significance.

[b] Decelerations are repetitive in nature when they are associated with more than 50% of uterine contractions.

should be sought, preferably before a pathological tracing develops. If a reversible situation is identified then measures should be taken to correct it, with subsequent recovery of fetal oxygenation and the return to a normal tracing. If the situation does not revert or the pattern continues to deteriorate, consideration needs to be given for further evaluation or rapid delivery if a pathological pattern ensues.

Reversible Causes of Fetal Hypoxia/Acidosis

Excessive uterine activity is the most frequent cause of an abnormal CTG, and this can be detected by documenting tachysystole on the tracing or by palpating the uterine fundus. It is usually reversed by reducing or stopping oxytocin infusion, and/or by starting acute tocolysis. During the second stage of labour, maternal pushing can also contribute to decreased placental perfusion, so the mother should be asked to stop pushing until the situation is reversed.

Aortocaval compression is frequent in the supine position and results in reduced placental perfusion. Turning the mother onto her side is frequently followed by normalization of the CTG pattern.

Sudden maternal hypotension may occur after epidural or spinal analgesia, and is usually reversible by rapid fluid administration and/or an intravenous ephedrine bolus.

Some maternal respiratory or circulatory complications may also be reversible in nature (e.g., severe asthma, haemorrhagic shock, cardiorespiratory arrest, pulmonary thromboembolism, generalized seizures). Depending on the severity, duration and presumed reversibility of these situations, waiting for the normalization of fetal oxygenation after their occurrence may be indicated.

Occult and Irreversible Causes of Fetal Hypoxia/Acidosis

Other causes may not be immediately identifiable (e.g., occult cord compression, fetal haemorrhage) or may not be reversible in nature (e.g., uterine rupture, placental abruption, cord prolapse). Prompt delivery is required for all irreversible causes.

Good clinical judgement is required to diagnose the underlying cause of an abnormal CTG, and to judge the severity and speed of installation of fetal hypoxia/acidosis. The fetal capacity to withhold the insult, the reversibility of the situation and the probability of recurrence also need to be taken into account. All of these aspects are important for the objective of avoiding an adverse outcome, balanced against the risks of unnecessary obstetric intervention. When there is doubt about the occurrence of fetal hypoxia/acidosis, adjunctive technologies may be used to clarify the situation further, and these are considered in a separate chapter of the guidelines, which can be accessed at www.ijgo.org/article/S0020-7292(15)00396-3.pdf.

CONCLUSION

The aim of intrapartum FHR monitoring is to differentiate fetal compensation from decompensation so as to institute interventions in time to improve perinatal outcomes without increasing inappropriate operative interventions. Cardiotocography is associated with significant inter- and intraobserver variation as it involves a degree of 'pattern recognition'. National and international guidelines on CTG interpretation, such as the NICE and FIGO guidelines, are aimed at improving communication amongst midwives and obstetricians so as to reduce variation in clinical practice and to identify adverse features that may be associated with poor perinatal outcomes. However, no guideline is perfect or 'foolproof' and it is the responsibility of the midwife or doctor to understand the pathophysiology of intrapartum

hypoxia as well as the 'wider clinical picture' such as the fetal reserve, presence of meconium, intrapartum bleeding, chorioamnionitis and the rate of progress of labour whilst applying these clinical guidelines during the management of individual cases so as to optimize the outcomes.

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ANTEPARTUM FETAL SURVEILLANCE

Donald Gibb, Sabaratnam Arulkumaran

In 2016 NHS England launched a care bundle approach to reduce stillbirths.[1] The care bundle has four components based on the major contributors to stillbirths: reducing smoking in pregnancy, risk assessment and surveillance for growth restriction, raising awareness of reduced fetal movements and effective fetal monitoring during labour. This chapter deals with antenatal fetal surveillance and includes sections on detection and surveillance of growth restriction and management of reduced fetal movements.

Low-risk mothers will be seen largely by midwives in community antenatal clinics. Higher-risk mothers will be seen in hospital antenatal clinics, often by doctors. Those at risk require access to antenatal testing facilities. Maternofetal assessment units have become a standard feature in most large maternity services. The benefits of this include the gathering together of the various tests with the compilation and review of results. Daily outpatient assessment and review may be undertaken where previously admission to hospital was the norm. However, easy access may result in excessive testing with largely normal results. Protocols of referral should therefore be formulated and audit undertaken. An assessment unit should be located near the ultrasound department because most testing in the antenatal period depends on ultrasound examination. The simplest method of fetal assessment is the antenatal cardiotocograph (CTG). Computerized interpretation of the CTG is available from various manufacturers. These systems also provide electronic storage of the CTG; this is very useful because CTGs tend to get 'lost' and the original fades within a relatively short period of time. If a CTG is not electronically stored it should be photocopied, as this lasts much longer. Caution should be exercised in depending on computerized trace analysis with consequent risk of the loss of human skills of interpretation. The best computer is the human brain! The unit should be staffed by motivated midwives, who work with dedicated clinicians to assess those cases suspected to be at high risk. The individual requesting the test should be aware of the result in order to plan and justify further management. This should not be delegated by default to a junior member of staff.

IDENTIFICATION OF THE FETUS AT RISK

There are two groups of women who may require fetal assessment:

1. women with previously recognized historical risk factors such as previous stillbirth and neonatal death, or medical disorders such as diabetes mellitus, hypertension or other conditions
2. lower-risk women who develop obstetric complications during pregnancy, such as antepartum haemorrhage, hypertension, reduced fetal movement, intrauterine growth restriction, cholestasis or prolongation of pregnancy.

Adverse outcomes due to acute events like cord occlusion or placental abruption cannot be predicted by existing tests of fetal wellbeing. Fetal testing on account of the above markers within the past history can only be for maternal reassurance and should be minimized; excessive testing may generate anxiety and *consume much-needed resources.* Chronic compromise due to placental insufficiency operates through growth or nutritional failure of varying degrees. Some of these adverse results might be prevented by identification of the fetus at risk and appropriate intervention. Hypoxia is not the only mechanism of compromise. Other conditions such as diabetes mellitus, Rhesus isoimmunization and maternal or fetal infection may present a different threat. Selection of tests appropriate to the condition is important. There should be a protocol for testing that is related to the condition.

Cases are referred for fetal assessment for a variety of reasons. The most common indications are an abdominal size inappropriate for gestational age and reduced fetal movements. Vaginal bleeding, preterm labour, prolongation of pregnancy and hypertension are also common.

FETAL GROWTH

The abdomen may be judged to be a different size from that expected from the dates. More commonly this is smaller rather than larger. The importance of detecting small babies in utero has been emphasized in Chapter 2.

The clinical scenario may indicate a risk of hypoxic intrauterine growth restriction (IUGR) in well-recognized situations: previous IUGR baby, malnourished mother, smoking, alcohol, drug abuse, medical conditions, gestational hypertension, multiple pregnancy and other conditions. The algorithm suggested by the Royal College of Obstetricians and Gynaecologists (RCOG) for screening and surveillance of fetal growth in singleton pregnancies is given in Figure 7-1[2] and should be used to identify mothers at risk of growth restriction. Those at low risk should have a symphysis height chart plotted and those at high risk should have serial ultrasound

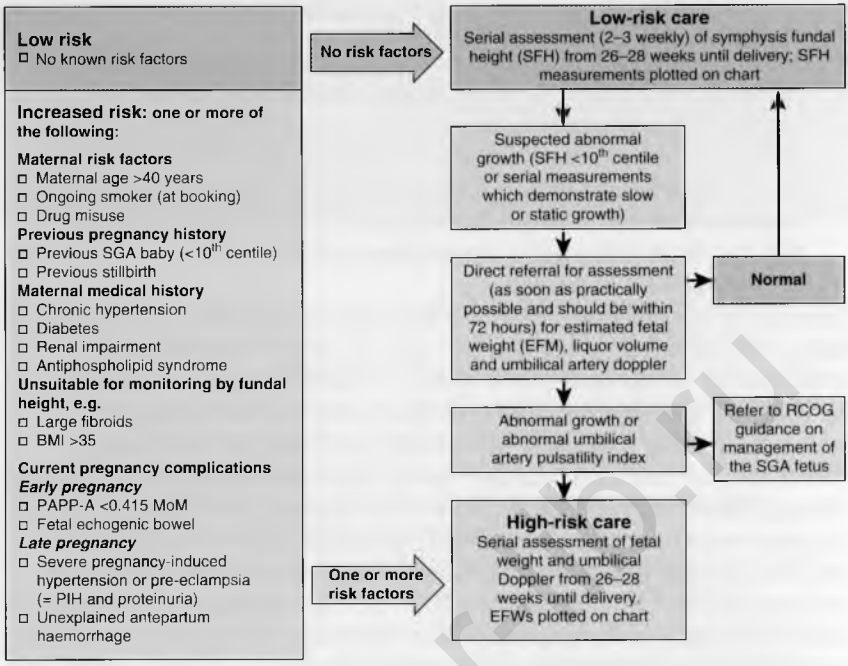


Figure 7-1. Index algorithm and risk assessment tool – screening and surveillance of fetal growth in singleton pregnancies. *(Reproduced with permission from: NHS England document 'Saving Babies' lives – a Care bundle for reducing still births' – 2016 under Open Government License 3.0 and that of the Royal College of Obstetricians and Gynaecologists.* The investigation and management of small for gestational age fetus. *Green-top Guideline No. 31. London: RCOG; 2013.)*

measurements that will provide estimated weight calculation, which should be plotted on a chart.

The measurement of the fundosymphysis height (see Figs 2-1 and 2-2) in centimetres, given that the fetus is a single fetus in a longitudinal lie, is plotted on a chart. If it is more than 2 cm smaller than the gestational age-based uterine fundal height before 36 weeks or 3 cm thereafter, then it is clinically small for dates. The confounding effects of abnormal lie, obesity, fibroids, multiple pregnancy and polyhydramnios must also to be considered.

Clinically small for dates is an indication for an ultrasound scan.

On ultrasound examination measurements of head circumference (HC), abdominal circumference (AC) and femur length (FL) are taken and plotted on a growth chart (Fig. 7-2). The AC reflects fetal weight most accurately and if it falls below the 5th centile this is ultrasonically small for dates. Customized fundosymphysis fundal growth charts based on ethnicity, parity, height and weight of the mother are freely

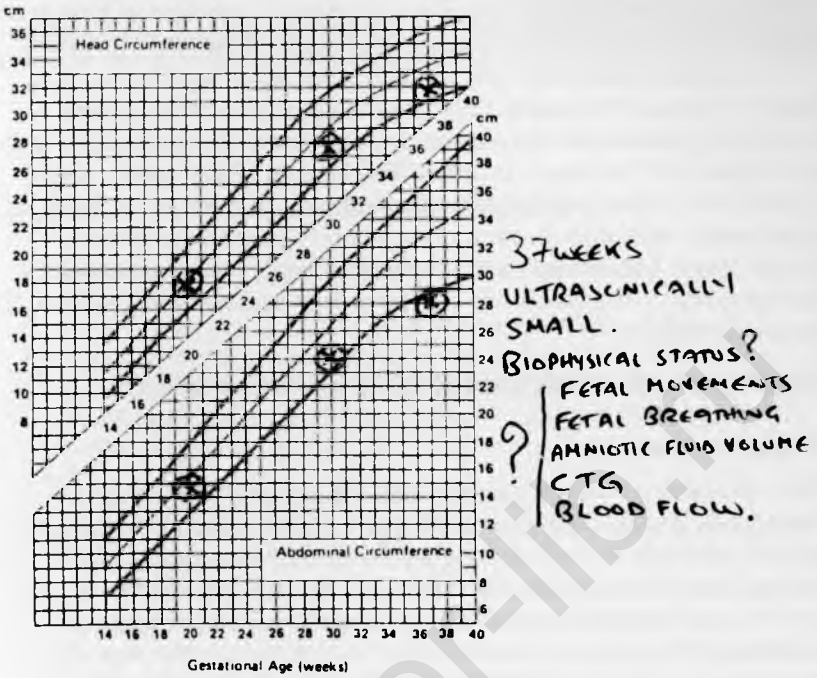


Figure 7-2. Ultrasound chart – small for dates.

available.[3,4] They are recommended by the RCOG as they may help to identify more cases of IUGR than does conventional measurement with the tape.[2] Similarly customized growth charts are available to plot estimated fetal weight based on ultrasound measurements.[3,4] A fetus that is ultrasonically small may be an expected small but healthy baby due to small parents (i.e., genetic smallness). Alternatively, a small fetus may be pathologically small due to an abnormal process. To distinguish one from the other the following should be taken into account:

- risk factors
- amniotic fluid volume
- subjective and objective fetal movements
- CTG
- other biophysical elements: fetal breathing, fetal tone, blood flow velocity waveform in the fetal vessels by Doppler ultrasound.

Pathological smallness is what is generally referred to as intrauterine growth restriction. This term carries an implication of a likelihood of a hypoxic process being present. The pathology of growth restriction is defined by the size, but function is more important.

Not all small fetuses are suffering from IUGR.

A growth-restricted baby is one that has not realized its own intrinsic growth potential.

The growth-restricted baby identified before or on admission in labour is flagged for special care with continuous electronic fetal monitoring, careful use of oxytocic therapy when needed and no undue prolongation of the labour process. The final proof of hypoxic IUGR comes from the neonatologist's observations of weight (in relation to expected weight for gestational age) and neonatal behaviour. Usually these babies have a scaphoid abdomen, little subcutaneous fat deposition in the limbs and can be recognized by measurement of ponderal indices.

BIOPHYSICAL ASSESSMENT OF FETAL HEALTH

Fetal Movements

From 24 weeks onwards, awareness of fetal movements and its importance should be explained to every mother. Reduced or absent fetal movements should be carefully investigated using the RCOG advice:[5] perform a CTG and if there has been no scan within the last 2 weeks then also a growth scan and amniotic fluid volume assessment to exclude missed IUGR. Fetal activity in the form of fetal movement perceived by the mother is a reliable indicator of fetal health. Women should be encouraged to be aware of this. An appropriate abdominal circumference and normal amniotic fluid volume on ultrasound are reassuring and often the fetus is seen to be active during the scan. The woman will also see this and be reassured. Commonly, after or even during assessment the fetus recommences normal movements and there is no need for further assessment.

In a randomized study involving 68 000 women the routine use of fetal movement charts was not beneficial compared with more selective use.[6] Reduced or no movements predicted poor perinatal outcome but this could not be prevented. This may be partly to do with different reporting times in the study and inadequate surveillance, possibly late surveillance or being dependent only on the CTG. In order to avoid such occurrences, NHS England[1] has formulated a checklist and this is given in Box 7-1.

The commonly used chart in the UK is the Cardiff fetal movement 'Count to ten' chart. Sadovsky, who studied fetal movement extensively, suggested that there should be four fetal movements in a 2-h period each day, of which one movement has to be strong.[7] The expectation of four fetal movements in 2 hours (h) or 10 in 12 h is arbitrary and correlates with good perinatal outcome.[8–10] A single fetal movement felt by the mother may not be recorded by the ultrasound movement detection devices. However, when a mother feels clusters of

Box 7-1. Checklist for Required Management of Reduced Fetal Movements[a]

Based upon RCOG guideline 57.[5]
For women ≥28 weeks' gestation.
Keep in guidance notes about Fetal Medicine Unit referral for women <24 weeks' gestation.

Attendance with Reduced Fetal Movements (RFM)

- Ask
  Is there maternal perception of reduced fetal movements?
- Assess
  Are there risk factors for fetal growth restriction (FGR) or stillbirth?
  Consider – multiple consultations for RFM, known FGR, maternal hypertension, diabetes, extremes of maternal age, primiparity, smoking, obesity, racial/ethnic factors, past obstetric history of FGR or stillbirth and issues with access to care.
- Act
  Auscultate fetal heart (hand-held Doppler/Pinard)
  Perform cardiotocograph to assess fetal heart rate in accordance with national guidelines.
  If risk factors for FGR/stillbirth, perform ultrasound scan for fetal growth, liquor volume and umbilical artery Doppler within 24 hours.
- Advise
  Convey results of investigations to the mother.
  Mother should re-attend if further reductions in fetal movements at any time.
- Act
  Act upon abnormal results promptly.

[a]Reproduced with permission from: NHS England document 'Saving Babies' lives – a Care bundle for reducing still births' – 2016 under Open Government License 3.0 and that of the Royal College of Obstetricians and Gynaecologists. *Reduced Fetal Movements*, Green-top Guideline No. 57. London: RCOG; 2011.[1]

fetal movements for 15–20 seconds (s) it is detected by the ultrasound transducer and is almost always associated with fetal heart rate (FHR) accelerations.[11] Women should be encouraged to be reassured by clusters of fetal movements.

The commonest answer to the question 'Is the baby moving?' is 'Yes, a lot'. We have to be prepared for the next question 'Can it move too much? Can this be bad?' There are many anecdotal reports by experienced clinicians of excessive fetal movements followed by death in utero. This must be due to an acute event and cord accidents or abruption could be postulated. In utero convulsions may occur, whether due to pre-existing brain abnormality or to another mechanism, and may be reported by the mother as excessive fetal movement followed by death. In any event it must be extremely rare and this should not compromise our general reassurance of the

mother that a lot of fetal movement is a healthy phenomenon. When a woman complains of excessive fetal movements a reversion to normal movements is reassuring, but if there is subsequent absence of fetal movement she should attend urgently for review.

Increased fetal activity can lead to confluence of accelerations mimicking a fetal tachycardia, and the synchronous automatic recording of fetal movements as done by the newest monitors will help to clarify this situation.[12] There are monitors using actograms that attempt to record fetal movement and fetal breathing in addition to the FHR. The clinical application of this principle remains to be proven, however.

Antepartum Electronic Fetal Heart Rate Monitoring

Non-stress test

The recording of the FHR for a period of 20–30 min without any induced stress to the fetus (like oxytocin infusion or nipple stimulation) to produce uterine contractions is called the non-stress test (NST). In the UK this is commonly referred to as an antenatal CTG. The duration of this test should be until reactivity is observed – that is, until there are two accelerations in a 10-min period. The sleep phase with no fetal movement and no FHR accelerations does not usually exceed 40 min in the vast majority of healthy fetuses and almost all healthy fetuses show a reactive trace within 90 min.[13] This forms the framework for extending the NST for 40 min when the trace is not reactive in the first 20 min.

A summary of the interpretation of the NST based on the International Federation of Obstetrics and Gynaecology (FIGO)[14,15] and the actions that are recommended with each type of trace is given below.

Antepartum cardiotocograph (NST)

Normal/reassuring

- At least two accelerations (15 beats for >15 s) in 10 min (reactive trace), baseline heart rate 110–150 beats per min (bpm), baseline variability 5–25 bpm, absence of decelerations.
- Sporadic mild decelerations (amplitude <40 bpm, duration <30 s) are acceptable following an acceleration.
- When there is moderate tachycardia (150–170 bpm) or bradycardia (100–110 bpm), a reactive trace without decelerations is reassuring of good health.

Interpretation/action: Repeat according to clinical situation and the degree of fetal risk.

Suspicious/equivocal

- Absence of accelerations for >40 min (non-reactive).
- Baseline heart rate 150–170 bpm or 110–100 bpm, baseline variability >25 bpm in the absence of accelerations.
- Sporadic decelerations of any type unless severe as described below.

Interpretation/action: Continue for 90 min until trace becomes reactive, or repeat CTG within 24 h, amniotic fluid index (AFI) or single vertical pocket of amniotic fluid/biophysical profile (BPP)/Doppler ultrasound blood velocity waveform.

Pathological/abnormal

- Baseline heart rate <100 bpm or >170 bpm.
- Silent pattern <5 bpm for >90 min.
- Sinusoidal pattern (oscillation frequency <2–5 cycles/min, amplitude of 2–10 bpm for >40 min with no acceleration and no area of normal baseline variability).
- Repeated late, prolonged (>1 min) and severe variable (>40 bpm) decelerations.

Interpretation/action: Further evaluation (ultrasonic assessment of amniotic fluid volume, BPP, Doppler ultrasound blood velocity waveform). Deliver if clinically appropriate.

The antepartum cardiotocograph (NST) is usually applied for diagnostic purposes; its value for screening has not been proven.[14,15] Pooled results of four studies of NSTs involving 10 169 patients revealed a satisfactory outcome with a false-negative rate of 7 per 10 000 cases.[16–19] The NST may be abnormal not only due to hypoxia but also due to other causes associated with reduced baseline variability such as infection, medication, congenital malformation, cerebral haemorrhage and cardiac arrhythmia. A review of the history with further evaluation will be helpful to clarify the cause.

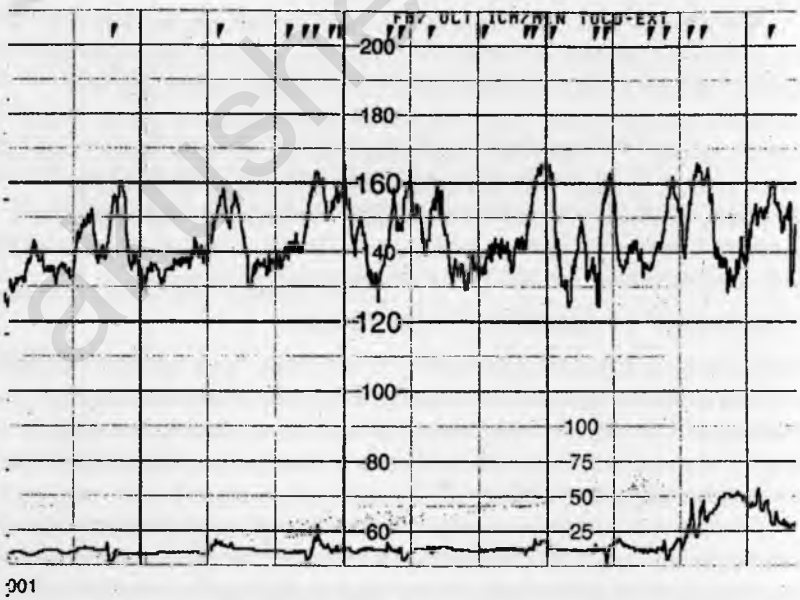
Assessment of Amniotic Fluid Volume

Fetal urine contributes significantly to amniotic fluid volume. Fetuses with no kidneys have severe oligohydramnios. With diminished placental function and reduced renal perfusion the amniotic fluid volume decreases. Perinatal outcome is poor when the amniotic fluid volume is reduced at delivery.[20–22]

Clinical evaluation of amniotic fluid volume by abdominal palpation can be deceptive. The impression of the amniotic fluid volume gained on ultrasound examination is fairly reliable. Objective assessment of the vertical depth of the largest pocket of amniotic fluid after excluding loops of cord or the sum of the vertical pockets in the four quadrants of the uterus (AFI) is used in practice. Recent review of the literature

suggests that either AFI or the single largest vertical pocket can be used as an objective measure.[23] Reduced amniotic fluid by either method is associated with poor fetal outcome[20–22] and delivery should be considered, assuming a reasonable gestational age. If only one vertical pocket is measured, a value of <3 cm in the largest pool is an indication for delivery.

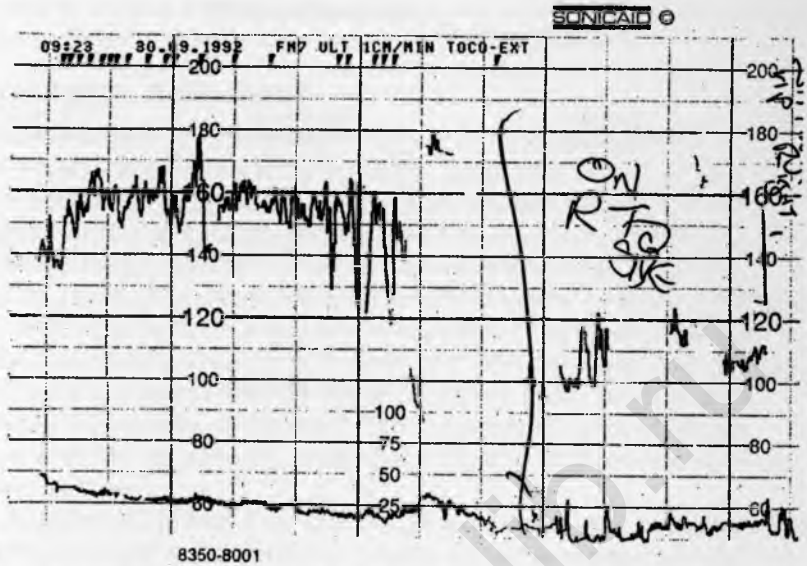
In post-term pregnancy or that complicated by severe growth restriction, the decline in fluid volume can be up to one-third every week, and twice-weekly assessment is advisable. Combining amniotic fluid assessment and NST could be the first-line assessments in high-risk pregnancies and is adequate for most women. Antepartum fetal death within a week of a reactive NST may occur for those who have an AFI below 5.[24] It is quite possible for a fetus with a reactive NST and good fetal movements to die suddenly in the presence of marked oligohydramnios (Fig. 7-3A–C). This may be due to umbilical cord compression. Most centres now recognize that, for high-risk pregnancies where a reduction of amniotic fluid volume is suspected (e.g., IUGR, post-term, etc.), it is desirable to perform an amniotic fluid estimation. A schematic diagram incorporating AFI and NST as the first-line assessment is shown in Figure 7-4.



A

Figure 7-3. (A) NST in a post-mature fetus: variability and fetal movements seen;

(Continued)



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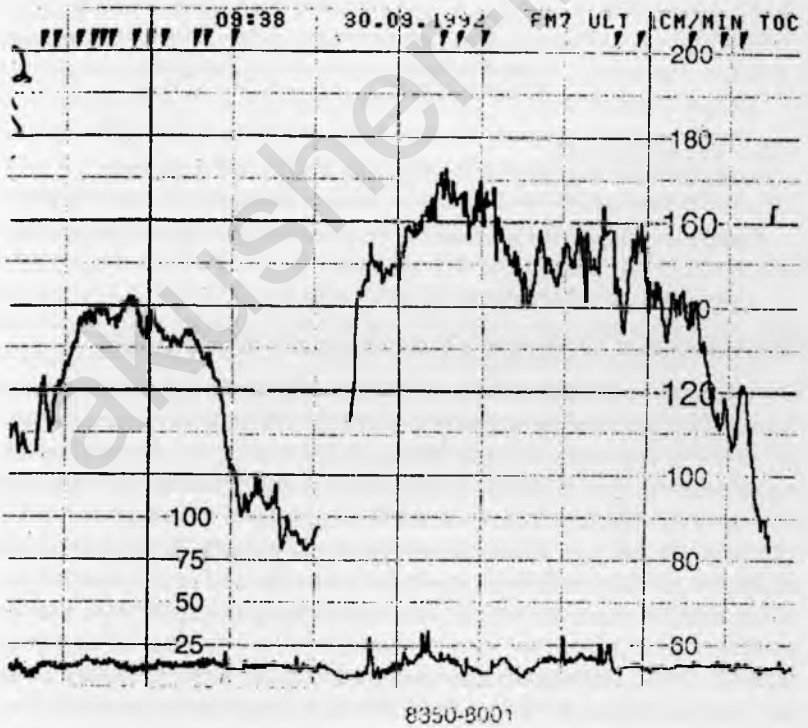
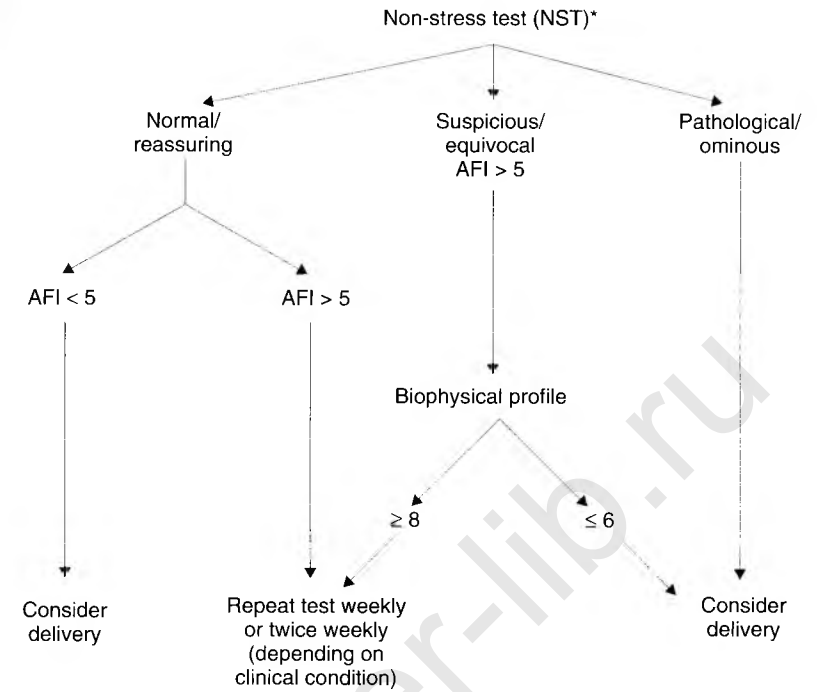


C

Figure 7-3. (*Continued*) (B) sudden decelerations; (C) bradycardia and fetal death within minutes.



AFI – Amniotic fluid index
*Repeat NST and AFI weekly or more often acording to clinical situation.
In preterm situations additional tests (e.g., Doppler velocimetry) may be of value

Figure 7-4. Suggestion for antepartum fetal monitoring in high-risk pregnancies.

Measurement of Blood-Flow Velocity Waveforms

Sometimes it is not possible to deliver a fetus at risk of progressive hypoxia because of prematurity. There is difficulty in interpreting the NST at early gestations. Measurement of blood-flow velocity waveforms in the umbilical artery, fetal aorta, middle cerebral artery and ductus venosus may give additional useful information for the timing of delivery in these circumstances. Initially there is increased resistance in the umbilical artery followed by reduced resistance in the middle cerebral artery. With increasing compromise, the umbilical artery Doppler may show absent or reversal of end-diastolic flow. With absent end-diastolic flow, if the maturity does not pose a major challenge then delivery may be undertaken. If more prolongation of the pregnancy is required then additional testing by computerized CTG or ductus venosus Doppler may be of value, the latter being preferred based on long-term outcome studies.[25] If there is reversal of end-diastolic flow in the umbilical

artery, or ductus venosus flow or computerized CTG does not meet the set criteria, then it is an indication for delivery.

Biophysical Assessment

Fetal responses to hypoxia do not occur at random but rather are initiated and regulated by complex, integrated reflexes of the fetal central nervous system. Stimuli that regulate the biophysical characteristics of fetal movement, breathing and tone arise from different sites in the brain. There is some evidence that the first physical activity to develop is fetal tone at 8 weeks' gestation. It is also the last to cease functioning when subjected to increasing hypoxia.[26] Fetal movements develop at 9 weeks and fetal breathing at 20 weeks. FHR activity matures last, by about 28 weeks, and is the first to be affected by hypoxia. In hypoxia, FHR characteristics may become abnormal first, followed by breathing, body and limb movements, and finally by tone.

Evaluation of more than one biophysical parameter to assess fetal health has been suggested but it may not be necessary if the NST is reactive and amniotic fluid assessment is normal. In the assessment of biophysical profile fetal movements, tone, breathing and amniotic fluid volume assessed by the scan and NST are considered, and for each a score of 2 or 0 is given, there being no intermediate score.[27] When the NST is not reactive, as is more common in the preterm period, it might be useful to assess the fetal biophysical profile. A score of 8 or 10 indicates a fetus in good condition. Retesting should be performed at intervals depending on the level of risk. In situations where the compromise may develop faster, as in prolonged pregnancy, IUGR and prelabour rupture of membranes, it is best performed twice weekly. If the score is 6, then the score should be re-evaluated 4–6 h later and a decision made based on the new score. When the biophysical profile is ≤ 2 on one occasion, or ≤ 4 on two occasions (6–8 h apart), delivery of the fetus is indicated if the fetus is adequately mature and has a good chance of survival.[28] Further evaluation with fetal blood-flow velocity waveform measurement may be considered if the fetus is so premature that deferring delivery by even a few days is considered beneficial. Good perinatal outcome has been reported with biophysical profile scoring in high-risk pregnancy[29] and as a primary modality of testing in prolonged pregnancy.[30]

A modified biophysical profile where only the ultrasound parameters are evaluated (without NST) has been found to be equally reliable.[31] Due to the time and expertise needed to perform a biophysical profile, many centres perform an NST and amniotic fluid assessment. Biophysical scoring should be reserved for fetal medicine units and research protocols.

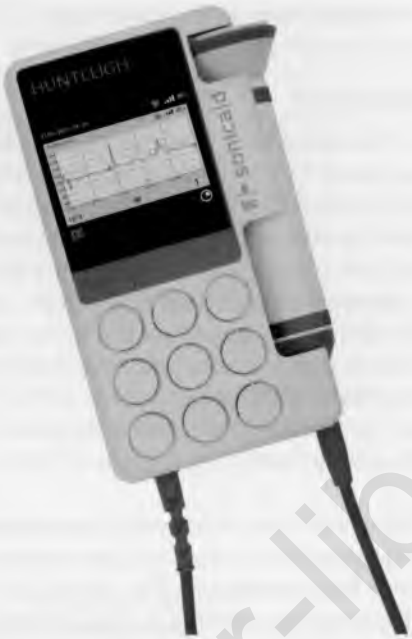


Figure 7-5. Digital hand-held Doppler monitor with cardiac tracing. *(Courtesy of Huntleigh/Sonicaid.)*

Assessment of the Fetus in an Outpatient Clinic with Limited Facilities

A hand-held Doptone with a digital display will give a baseline FHR. New Doppler machines give a CTG in the LED screen and it is possible to identify FHR accelerations and decelerations (Fig. 7-5).

NST is usually used for diagnostic purposes and has not been proven to be of value as a screening test. The ability of the test to identify the problem being investigated should be known. A normal NST indicates fetal health/wellbeing. However, with chronic placental dysfunction, fetal adaptation occurs and normal (reactive) NST does not indicate the degree by which placental function may be reduced. Thus, the predictive value of a normal NST is governed by the clinical situation.

CASE ILLUSTRATIONS

The CTG may not be normal owing to a variety of causes other than hypoxia: cardiac arrhythmias, heart block, brain abnormality (congenital or acquired), chromosomal abnormality, anaesthesia, drug effects and infection.

Hypoxia

Severe IUGR is seen in the preterm period. It has been suggested that decelerations are a normal feature of the preterm CTG. There is

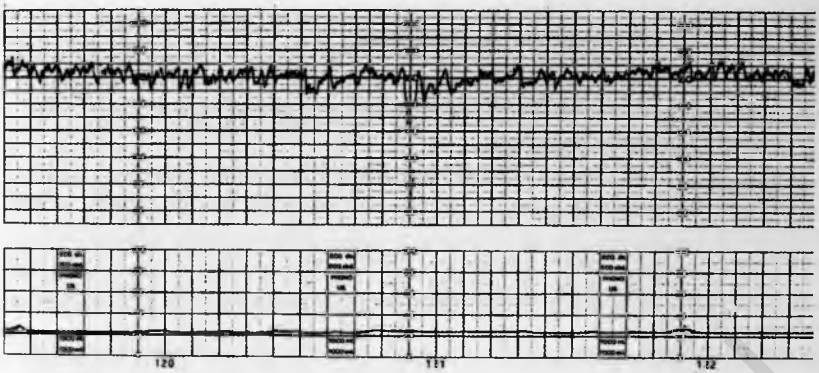


Figure 7-6. CTG in preterm baby – low-amplitude accelerations and short sharp decelerations.

a reduction in variability, and lower-amplitude accelerations are seen in the preterm CTG (Fig. 7-6); however, major decelerations are not a normal feature. In the preterm period, short sharp decelerations of <15 s may be seen. They are often seen with change of state from sleeping to waking and may follow immediately after the acceleration. When major decelerations occur the clinical situation should be considered. Figure 7-7 is from a known case of severe IUGR at

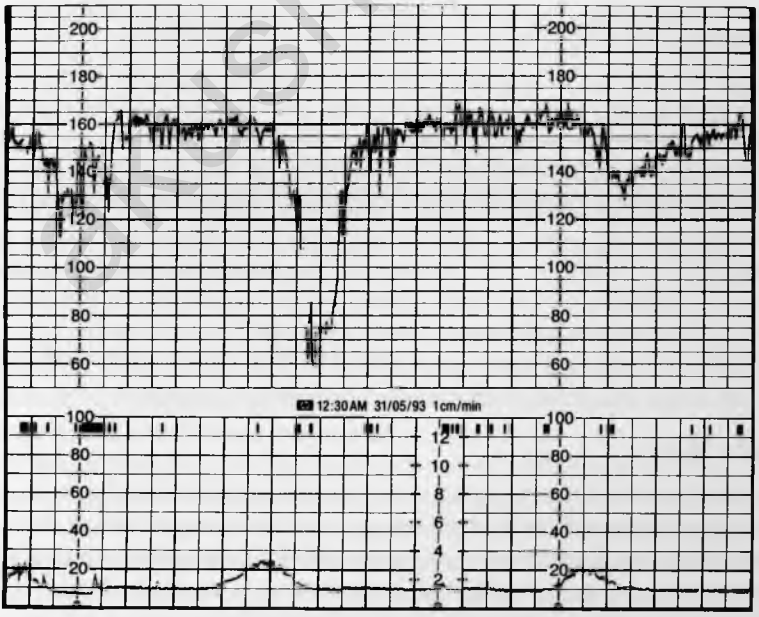


Figure 7-7. NST in a case of severe IUGR, oligohydramnios, poor fetal movement and abnormal fetal and poor maternal blood flow.

25 weeks' gestation. There was oligohydramnios, poor fetal movement and very abnormal fetal and maternal blood flow. On account of a very small fetal weight estimate and early gestation, the couple, with the advice of the obstetrician, opted for conservative management. The fetus died in utero 3 days later.

Given a bigger weight estimate and later gestation, delivery would have been appropriate. There will be no guarantee that the baby is not already damaged; however, there is a good chance such a baby will do well with good neonatal intensive care.[31] Leaving a fetus to die in utero is not ethically justifiable in the face of reasonable weight and gestation.

Cardiac Arrhythmias

Fetal arrhythmia may give rise to an abnormal trace, although the fetus is not hypoxic. Figure 7-8A was obtained from a case where the midwife auscultated the fetal heart in the antenatal clinic and heard a tachycardia. She noted that the multiparous woman was classically low risk and that the fetus was well grown and moving. This was confirmed by ultrasound scan after referral to hospital. Twenty hours later the CTG was repeated and was essentially unchanged. Advice was sought from a specialized unit, a diagnosis of fetal supraventricular tachycardia was made and the administration of double the adult dose of digoxin was recommended. Fetal echocardiography was normal. Figure 7-8B was recorded the following day. The pregnancy continued, culminating in normal labour, normal intrapartum CTG and normal delivery of a healthy baby 2 weeks later. The baby had a structurally normal heart and no further problem with the heart rhythm. Figure 7-9 is a similar case but the observation of supraventricular tachycardia was made in early labour. Advice was sought and the administration of digoxin considered inappropriate because the drug would not have taken effect until after the baby had been born. The baby was noted to be moving and continued to do so during labour. The amniotic fluid was clear and the woman was low risk. The CTG remained unchanged during the 6 h of labour until the second stage. At this time the features changed, possibly due to vagal stimulation with descent of the head. Although technically imperfect, there appeared to be a normal rate, variability and second-stage decelerations (Fig. 7-9B). After delivery the baby had a normal heart rate and no further problem!

Heart Block

This can be complete or partial, and continuous or intermittent. Occasional dropped beats are frequent and of no significance: they generally do not interfere with the appearance of the trace or persist after delivery. A case of maternal systemic lupus erythematosus with fetal heart block has been shown in Figure 4-27.

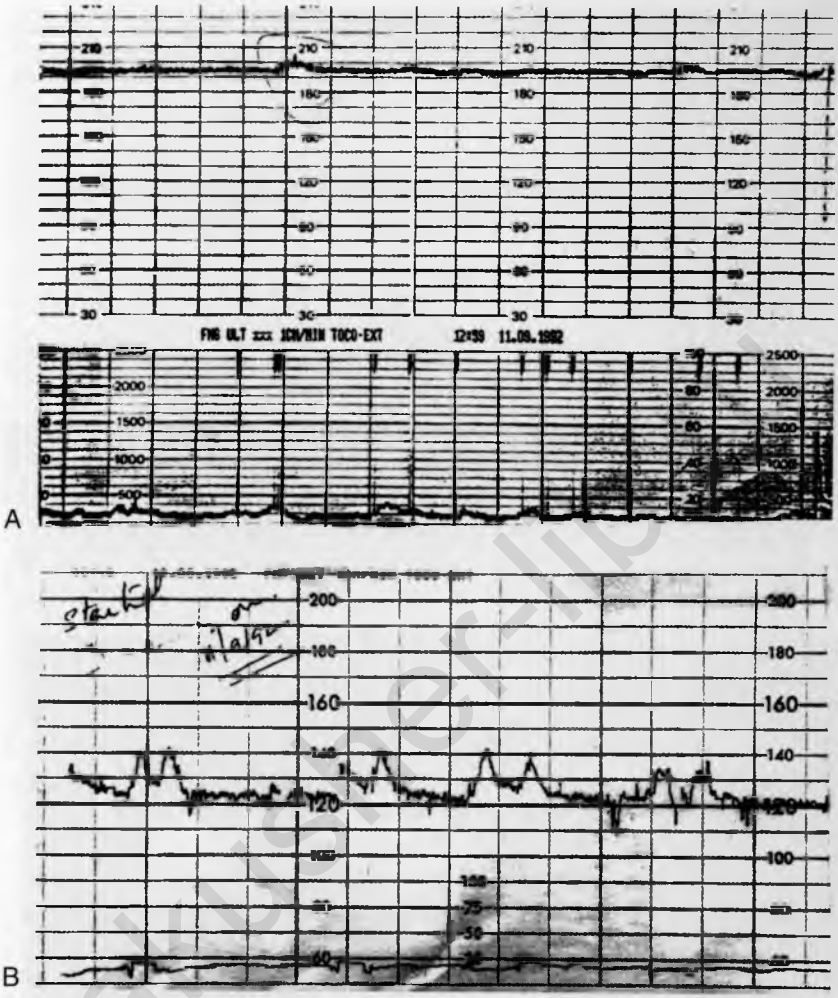


Figure 7-8. (A) Fetal supraventricular tachycardia; (B) reversal to normal rate after maternal administration of digoxin.

Brain Abnormalities – Acquired

Physiological mechanisms controlling the fetal heart require the integrity of the central nervous system.

An abnormal CTG with no accelerations or decelerations and markedly reduced baseline variability was recorded (Fig. 7-10A) when a high-risk woman on antihypertensive medication presented with a sudden cessation of fetal movement. The fetus was well grown and the amniotic fluid volume was normal on ultrasound scan. During a prolonged scan the fetus did not move. There was

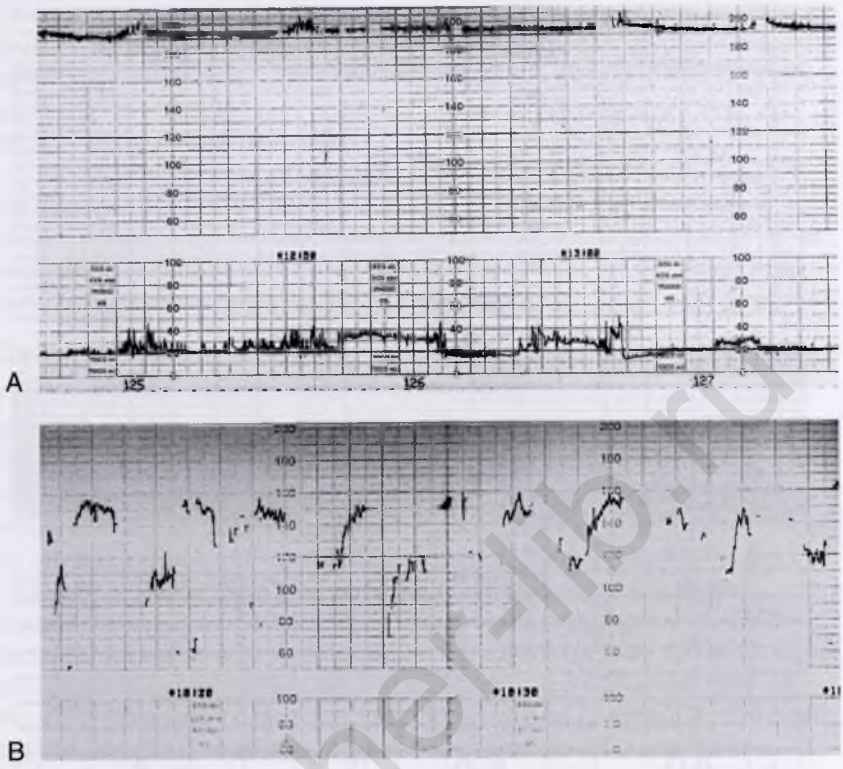


Figure 7-9. (A) Supraventricular tachycardia diagnosed in labour; (B) reversal to normal heart rate with decelerations in the second stage of labour.

a collapsed stomach and an atonic dilated bladder with evidence of a large cerebral haemorrhage (Fig. 7-10B). In view of the unusual findings a fetal blood sample was obtained from the umbilical vein for karyotyping, fetal haematology and cytomegalovirus screening. The fetal blood gases were normal and the fetal haemoglobin was 8 g/dl consistent with the intracranial haemorrhage. While the karyotype results were awaited the fetus did not move and died 24 h after the procedure. Postmortem confirmed the cerebral haemorrhage. This severely 'brain-damaged' fetus was not hypoxic and, if delivered, would have had a very poor prognosis. The mother accepted and understood the outcome; she has since had a living child. Intracranial haemorrhage may occur in cases of alloimmune thrombocytopenia or when the woman is on warfarin therapy. When a CTG becomes abnormal despite good growth and good amniotic fluid volume, such unusual causes must be considered before deciding to deliver. Delivery will not lead to an improved outcome in these circumstances. In twin-to-twin transfusion syndrome when one fetus dies, the 'second

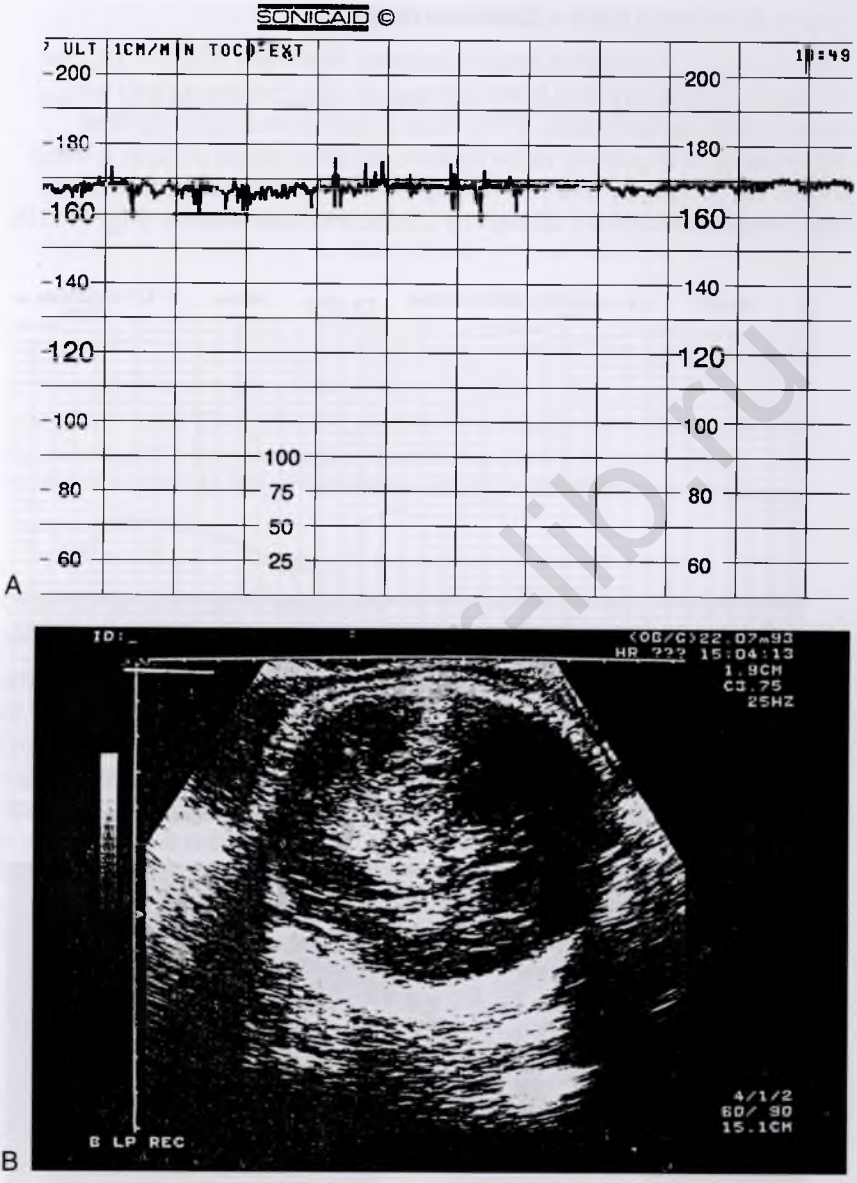


Figure 7-10. (A) CTG with a 'silent pattern' (baseline variability <5 bpm), no accelerations or decelerations; (B) scan showing evidence of fetal intracerebral haemorrhage.

fetus' may suffer from the consequences of sudden haemodynamic changes that may affect the brain and then manifest as a non-reactive CTG. No changes in blood gases on fetal blood sampling or obvious ultrasonic morphological change in the brain are seen immediately, but vacuolation in the brain may follow.

Brain Abnormalities – Congenital

The inability to maintain a steady baseline heart rate (Fig. 7-11A) can be due to severe hypoxic brain damage or may be associated with severe brain malformation. If the fetus is active, indicated by fetal movements, it is unlikely to be hypoxic and the cause of such a trace should be sought by further investigation. The associated pathology was holoprosencephaly, shown by ultrasound examination (Fig. 7-11B).

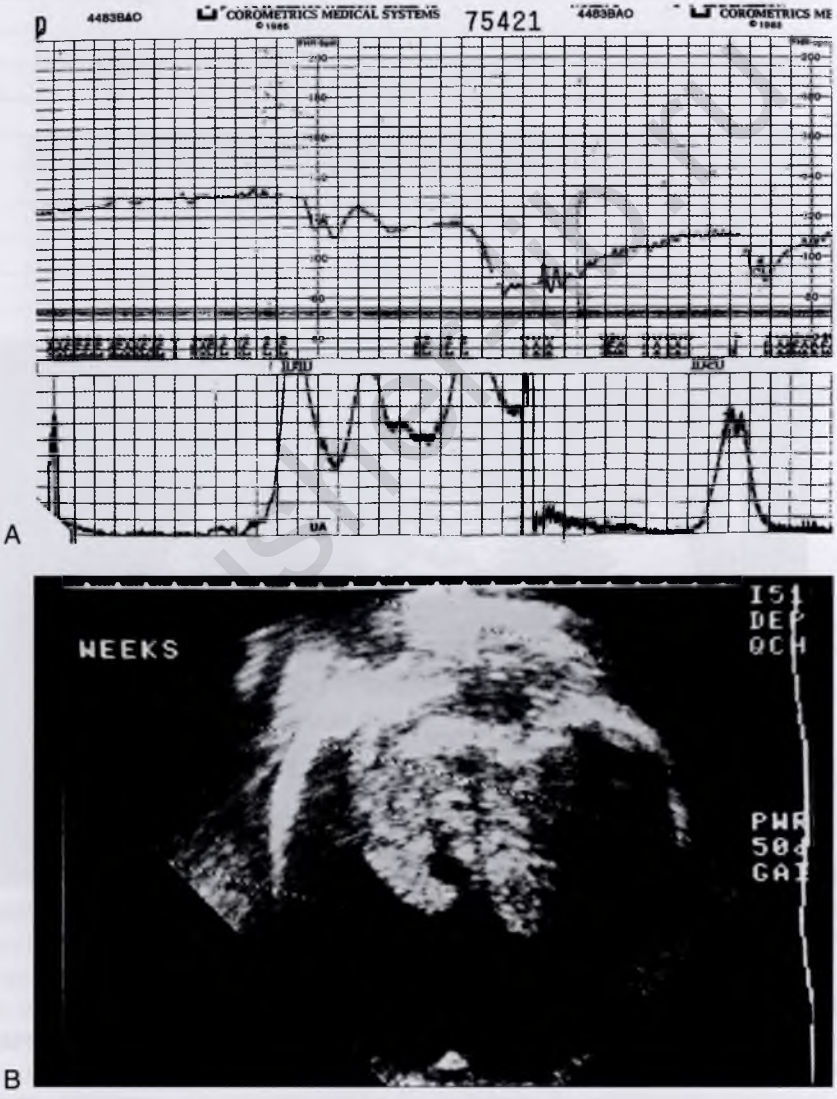


Figure 7-11. (A) CTG: unsteady baseline but with plenty of fetal movement; (B) ultrasound scan showing the fetus with holoprosencephaly.

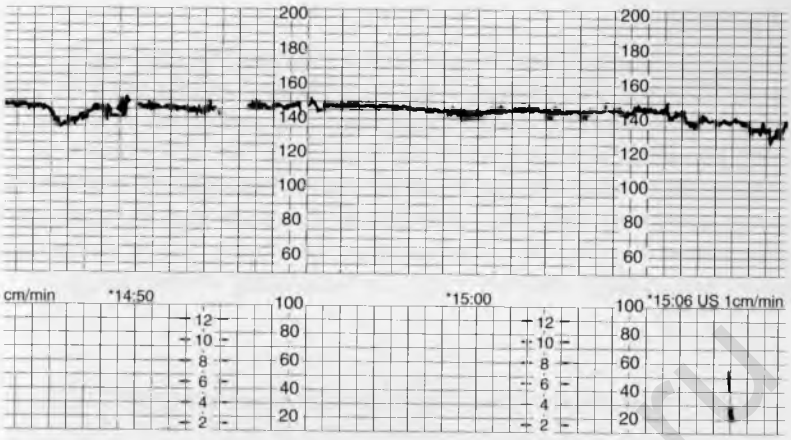


Figure 7-12. CTG with poor baseline variability, no accelerations and isolated decelerations. Misfit of fetal wellbeing tests; abnormal karyotype.

Chromosomal Abnormality

A 39-year-old multiparous woman was referred from another hospital with a well-grown fetus, reduced fetal movements and a good volume of amniotic fluid, and yet an abnormal CTG (Fig. 7-12). The Doppler blood-flow studies in fetus and mother were normal. There was a slightly reduced femur length and slight hydronephrosis. Delivery was deferred until the result of karyotype from a fetal blood sample was known. The fetus died in utero the day before the result, showing Down's syndrome, became available. The mother had been counselled of this strong possibility and requested that the baby not be delivered without the karyotype result.

In chromosomally abnormal fetuses, especially trisomies, the central neural pathway may be disorganized resulting in an abnormal CTG,[32] although the fetal growth, amniotic fluid volume and fetal movements may be normal. In trisomy 13 and 18 the fetus might be growth restricted with an increased amniotic fluid volume. In a proportion of these cases the CTG shows a steady baseline but with poor baseline variability, reduced or absent accelerations and isolated decelerations. The disorganized neural development may manifest after birth as mental retardation.

A misfit of fetal function tests suggests the need for further investigations.

Fetal Anaemia

This may show a sinusoidal or sinusoidal-like pattern and is discussed in Chapter 11.

Anaesthesia

The fetus is anaesthetized as well as the mother! The fetus may excrete the drugs more slowly than the mother. A multiparous woman fractured her tibia at 29 weeks' gestation. She was given a general anaesthetic in order to insert a pin and plate. A CTG performed on her return from the operating theatre 2 h after induction of anaesthesia showed a dramatic reduction of baseline variability and the absence of accelerations (Fig. 7-13A). The inexperienced junior doctor suspected hypoxia and thought delivery might be necessary. Ultrasound scan

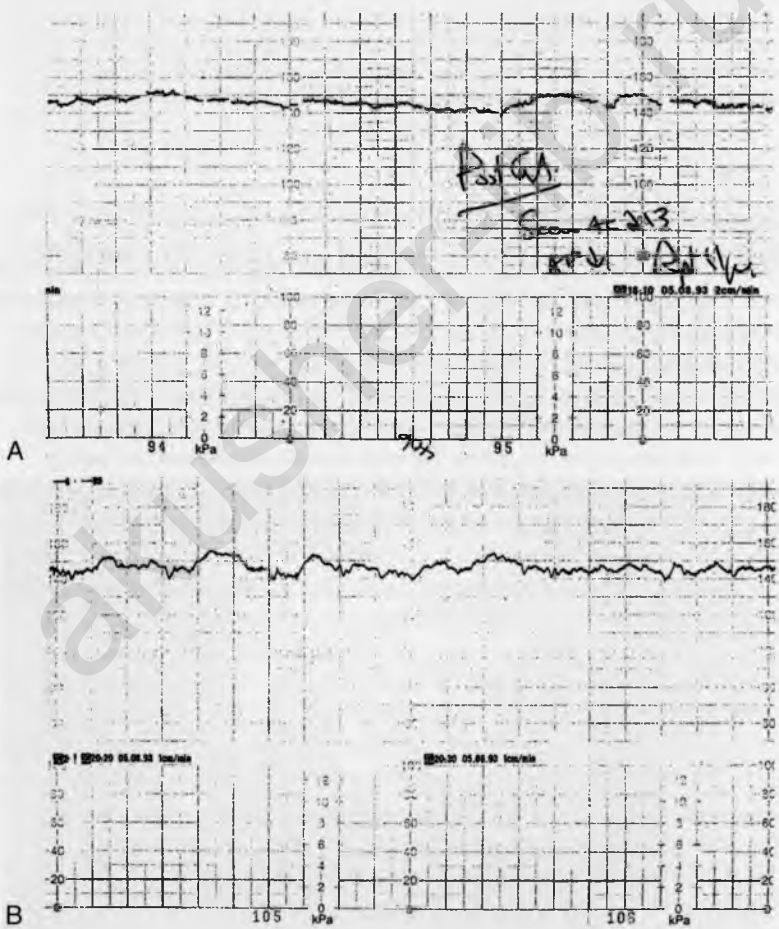


Figure 7-13. (A) CTG performed 2 h after induction of anaesthesia: no accelerations and reduced baseline variability; (B) CTG 4 h after induction of anaesthesia;

(Continued)

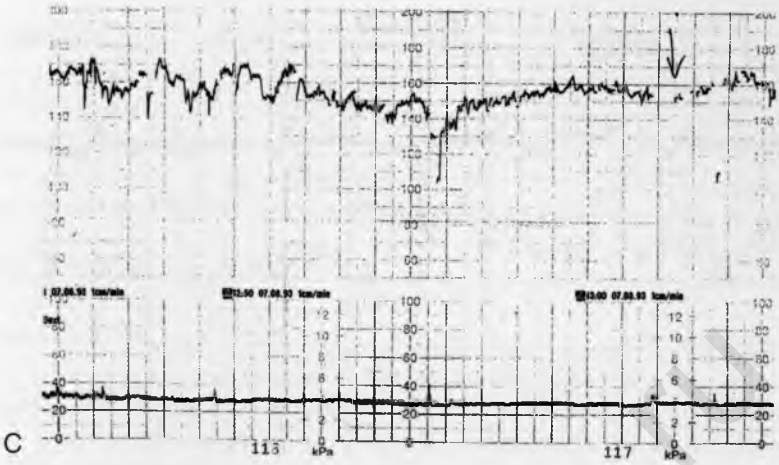


Figure 7-13. (*Continued*) (C) CTG 24 h later – reactive trace after the effect of anaesthesia has worn off.

confirmed a well-grown fetus and reasonable amniotic fluid volume. The consultant recommended a repeat CTG 2 h later (Fig. 7-13B) and another 24 h after that (Fig. 7-13C). The pregnancy progressed normally to term without further complication.

Drug Effects

Sedatives, tranquillizers, antihypertensives and other drugs that act on the central nervous system tend to reduce the amplitude of the accelerations and suppress the baseline variability. In these situations, other forms of surveillance become necessary. With antihypertensive therapy, fetal activity may be unaffected. Corticosteroids given to achieve fetal lung maturity can also reduce the baseline variability for 24–48 hours.

Infections

A fetal tachycardia associated with a maternal infection is a cause for concern. The mechanism may be direct fetal infection or secondary response of the fetus due to transplacental passage of pyrogens or adrenergic metabolites. When fetal tachycardia occurs with maternal tachycardia due to maternal urinary tract infection, it usually settles with antibiotic treatment. However, when fetal tachycardia persists for a considerable period of time then the fetus may not be able to tolerate it. Consideration of the clinical picture will suggest whether an actual fetal infection is likely. Preservation of baseline variability and reactivity suggests a resilient fetus.

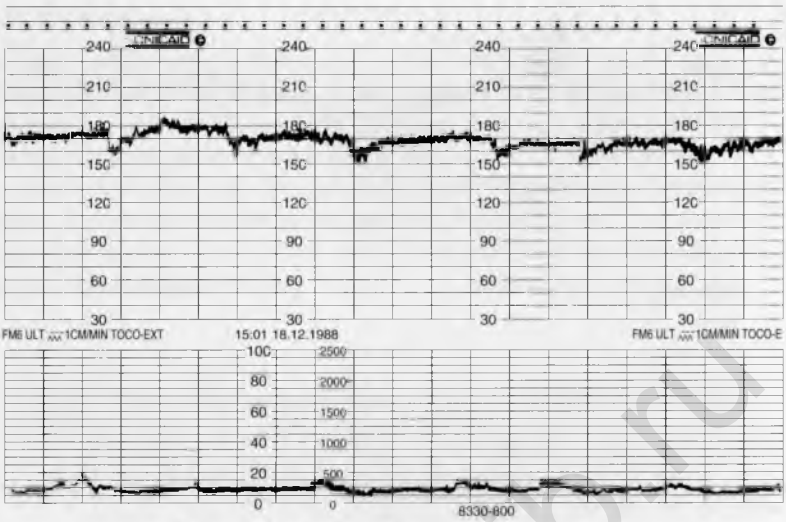


Figure 7-14. Ominous trace – listerial infection.

If there is reduced variability with or without decelerations in the absence of accelerations, the fetus itself is sick. A mother was admitted with a systemic illness at 33 weeks' gestation and tachycardia. On assumption of the diagnosis of urinary tract infection a cephalosporin was prescribed. The trace showed tachycardia with markedly reduced variability and shallow decelerations (Fig. 7-14). The mother's condition did not improve, nor did the fetal heart tracing. Rupture of the membranes with the release of meconium-stained amniotic fluid prompted caesarean section. The baby succumbed within hours of birth to congenital listeriosis; it was heavily infected. This is reflected in the seriously abnormal fetal heart tracing.

Maternal illness and preterm meconium suggest possible listerial infection.

Suspicion of the diagnosis, blood cultures and treatment with ampicillin might have led to a better outcome.[33]

In cases with prelabour rupture of the membranes, a CTG showing tachycardia, lack of accelerations and reduced variability suggests a higher probability of infection even in the absence of clinical signs.

PROLONGED PREGNANCY

This is a common indication for assessment in many hospitals. The clinician will have reviewed the menstrual and ultrasound dating and most cases will have reached 41 weeks. The CTG may be normal but caution should be applied in being reassured by this. Figure 7-3 was obtained in the assessment unit in a case where the maturity

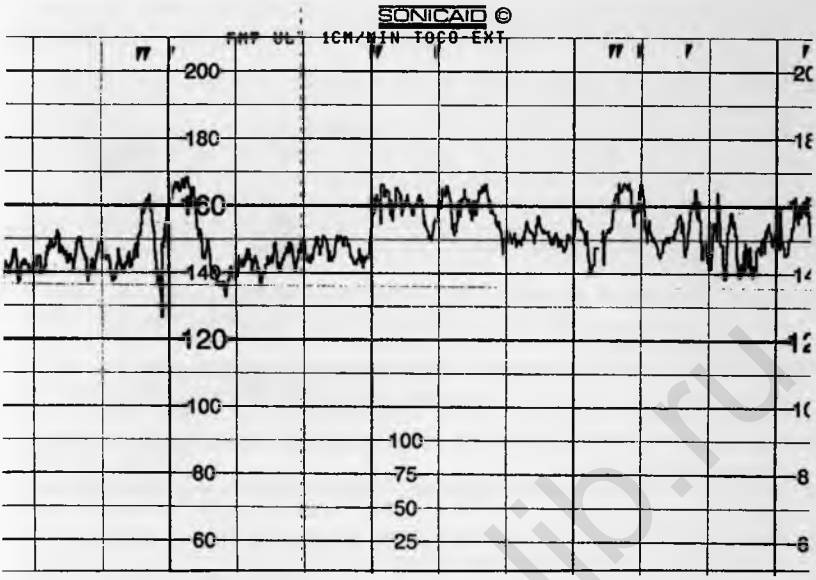


Figure 7-15. Postmature intrauterine death next day.

was 42 weeks and 5 days. Two days previously the deepest pool of amniotic fluid had been 3.2 cm and the CTG was reactive. On the day of assessment the CTG was the first investigation to be performed. Fetal movements are seen on the trace and the first 7 min suggest reasonable baseline variability although a slightly fast rate. Deep decelerations followed and the woman was transferred to the labour ward. In the anaesthetic room 20 min after the end of the trace, ultrasound scan showed a terminal bradycardia. A decision was made not to deliver and the heart stopped within minutes of observation. The baby was found to be otherwise normal at postmortem examination. Again the presentation suggests possible cord compression with oligohydramnios as the mechanism. In another case where intrauterine death occurred 24 h after a CTG in a postmature gestation (Fig. 7-15), the CTG had been normal and a single deepest pool of amniotic fluid had been 2 cm. Since that case we have performed AFI measurement during assessment.

Amniotic fluid assessment should form an integral part of assessment of fetal wellbeing.

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THE ADMISSION TEST BY CARDIOTOCOGRAPHY OR BY AUSCULTATION

Sabaratnam Arulkumaran, Donald Gibb

The 2014 NICE guideline 'Intrapartum care for healthy women and their babies' states (points 1.4.6–1.4.10): 'Auscultate the fetal heart rate at first contact with the woman in labour, and at each further assessment. Auscultate the fetal heart rate for a minimum of 1 minute immediately after a contraction and record it as a single rate. Palpate the maternal pulse to differentiate between maternal heart rate and fetal heart rate. Record accelerations and decelerations if heard. Do not perform cardiotocography on admission for low-risk women in suspected or established labour in any birth setting as part of initial assessment.'[1] The Birthplace UK (2011) study indicates that the incidence of stillbirths after start of care in labour is 0.22/1000, that of the death of the baby in the first week of life is 0.28/1000 and that of neonatal encephalopathy is 1.6/1000.[2] With such low figures of adverse outcome the numbers that need to be studied will run into several thousands and, based on available data, we do not have sufficient data to state that admission CTG should not be performed. In Sweden, with a very low intrapartum fetal death rate, admission CTG is routine. We would like to take the view that the choice should be given to the mother after giving her the available information including that no definitive benefit is known and also that it increases the obstetric intervention rate.

Fetal morbidity and mortality are greater in high-risk women with hypertension, diabetes, intrauterine growth restriction and other risk factors. A greater number of antenatal deaths are observed in this group. In pregnancies that have proceeded to term, morbidity and mortality due to events in labour occur with similar frequency in those categorized as low risk compared with those categorized as high risk based on traditional risk classification.[3,4] This may be because high-risk cases such as intrauterine growth restriction have been missed during antenatal care. To resolve this we have to turn our attention to better screening during the antenatal period and at the onset of labour. As stated above, the FHR is auscultated after

admission and every 15 min for a period of 1 min after a contraction in the first stage of labour and every 5 min or after every other contraction in the second stage of labour. During auscultation the baseline fetal heart rate (FHR) can be measured, but other features of the FHR such as baseline variability, accelerations and decelerations are more difficult to observe and quantify unless the following recommendation about auscultation is adhered to.

ADMISSION TEST BY AUSCULTATION ('INTELLIGENT AUSCULTATION')

If we are to limit our practice to auscultation it may be useful to use a Doptone so that the mother and her partner can listen. On admission the mother must be asked the question as to when the baby moved last and the time noted. A baseline FHR can be taken and recorded. With her permission the midwife or doctor could place a hand on the maternal abdomen and ask the mother to notify the examiner when she feels the baby moving. The care giver can note that he/she felt the fetal movements along with the mother, and auscultation at this time should give a heart rate of 15 beats more than the baseline heart rate as accelerations are expected with fetal movements. Continued palpation of the uterus should reveal a contraction, when the FHR should be auscultated. The presence or absence of obvious decelerations should be noted. If fetal movements were felt, the FHR acceleration was heard with the fetal movement, and there was no deceleration with, or soon after, a contraction, then the examiner could reassure the mother of good fetal health. Subsequent observations could be as recommended – to listen every 15 min for 1 min soon after a contraction in the first stage and after every 5 min in the second stage. Non-technological monitoring is undertaken during home births by competent midwives using this principle.

Figure 4-31 shows an admission CTG of a fetus in serious trouble with a pathological trace. Auscultation after a contraction by a skilled midwife (indicated by black dots) showed a 'normal' heart rate of 150 beats per min (bpm).

Baseline variability is not audible to the unaided ear.

The features that will provide the reassurance of fetal health are the presence of accelerations, normal baseline variability and an absence of decelerations that outlasts the contractions (i.e., late and atypical variable decelerations).

An admission test (AT) should pick up the apparently low-risk woman whose fetus is compromised on admission or is likely to become compromised in labour. This admission test may be performed by a CTG or by 'intelligent' auscultation.

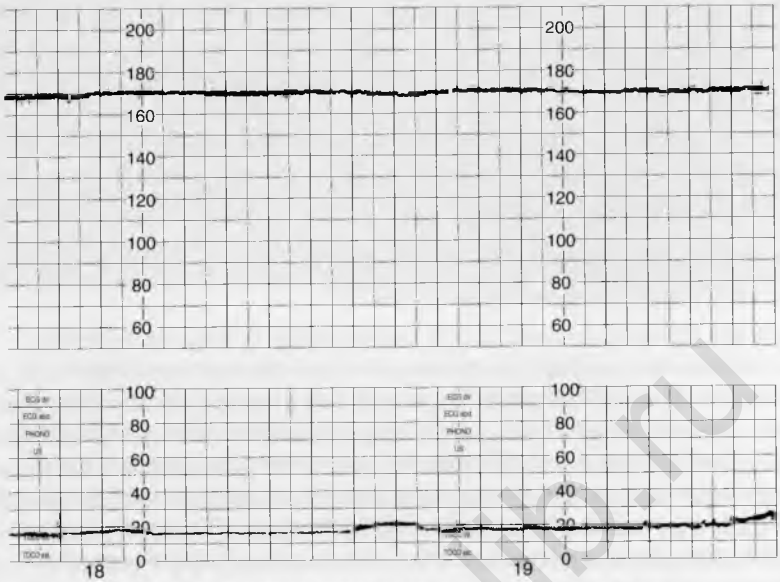

Figure 8-1. Abnormal admission test without contractions.

The AT by CTG is a short, continuous electronic FHR recording made immediately on admission, and gives a better impression of the fetal condition compared with simple auscultation. In many hospitals, electronic monitoring is performed but it is done long after admission. The mother may have waited for a bed, a nightdress, general observations to be noted and other administrative issues to be resolved. In most instances the mother walking into the labour ward is entirely healthy and her main concern is to have a healthy baby. An AT may identify those who are already at risk with an ominous pattern on admission even without any contractions (Fig. 8-1). In those with a normal or suspicious FHR the functional stress of the uterine contractions in early labour may bring about the abnormal FHR changes (Fig. 8-2). These changes may be subtle and difficult to identify by auscultation. Careful review may reveal a reduced FSH and a growth-restricted fetus in such cases. An admission CTG can be considered to function in the same way as a natural oxytocin challenge test.

STUDIES ON ADMISSION CARDIOTOCOGRAPHY

In Kandang Kerbau Hospital in Singapore a blinded AT study was carried out on 1041 low-risk women.[5] A FHR tracing was obtained after covering the digital display of the FHR and the recording paper and turning down the volume so that the research midwife had no information about what the FHR trace was showing. The

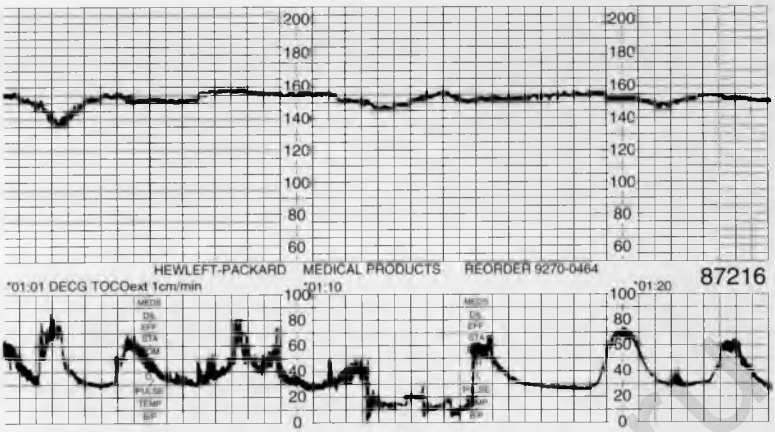

Figure 8-2. Contraction stress is often present in an admission test.

transducer was adjusted based on the green signal light of a fetal monitor (Hewlett-Packard 8040 or 8041), which indicates good signal quality and produces a good tracing. The trace, obtained for 20 min immediately on admission, was sealed in an envelope and put aside for later analysis. These women were a low-risk population based on risk factors and hence were sent to the low-risk labour ward for care by intermittent auscultation. This study was accepted by the departmental ethical committee because the normal practice at that time was that none of the low-risk women had any electronic monitoring.

For this study a reactive normal FHR trace was defined as a recording with normal baseline rate and variability, two accelerations of 15 beats above the baseline for 15 s, and no decelerations. A 'suspicious' or 'equivocal' trace was one that had no accelerations in addition to one abnormal feature such as reduced baseline variability (<5 bpm), presence of decelerations, baseline tachycardia or bradycardia. A trace was classified as 'ominous' when more than one abnormal feature or repeated atypical variable or late decelerations were present. To evaluate the outcome, 'fetal distress' was considered to be present when ominous FHR changes led to caesarean section or forceps delivery, or if the newborn had an Apgar score <7 at 5 min after spontaneous delivery (Table 8-1).

In women with ominous ATs ($n=10$), 40% developed fetal distress compared with 1.4% (13 out of 982) in those with a reactive AT. Of those 13 who developed fetal distress after a reactive AT, 10 did so more than 5 h after the AT. Of the three who developed fetal distress in less than 5 h, one had cord prolapse (baby born by caesarean section in good condition) and the other two fetuses were less than 35 weeks' gestation. They had low Apgar scores at birth 3 and 4 h after the AT but

Table 8-1. Results of Admission Test in Relation to the Incidence of 'Fetal Distress'

	ADMISSION TEST	FETAL DISTRESS
Reactive	$n=982$ (94.3%)	13 (1.4%)
Equivocal	$n=49$ (4.7%)	5 (10.0%)
Ominous	$n=10$ (1.0%)	4 (40.0%)§

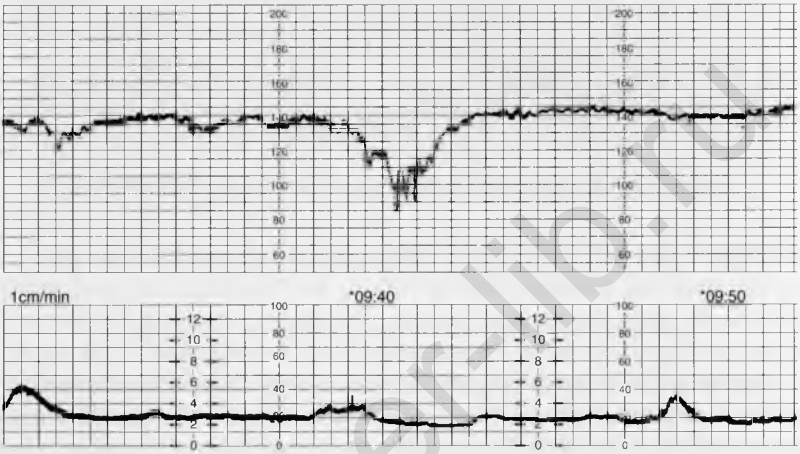

Figure 8-3. Concealed admission test of a fetus who died intrapartum.

needed minimal resuscitation. In those with an ominous AT there was one fresh stillbirth of a normally formed baby with normal birth weight for gestational age at term. The midwife was charting the FHR as 140/min every 20 min for 2 h when she reported that she was unable to hear the FHR. The admission test trace is shown in Figure 8-3. There is no doubt that the midwife's observations were correct; but unfortunately she could not hear the poor baseline variability and the shallow decelerations, which are ominous features, although the baseline rate was normal.

Barring acute events, the AT may be a good predictor of fetal condition at the time of admission and during the next few hours of labour in term fetuses labelled as low risk. If the AT is normal and reactive, gradually developing hypoxia will be reflected by no accelerations and by a gradually rising baseline FHR; the latter could be picked up at the time of intermittent auscultation or electronic monitoring. Figure 8-4A–F shows sequential changes in an 8-h labour showing gradual rise of FHR with absent accelerations and reduced baseline variability. Furthermore, it is known that if a well-grown fetus with clear amniotic fluid and a reactive trace starts to develop an abnormal FHR pattern it takes some time with these FHR changes

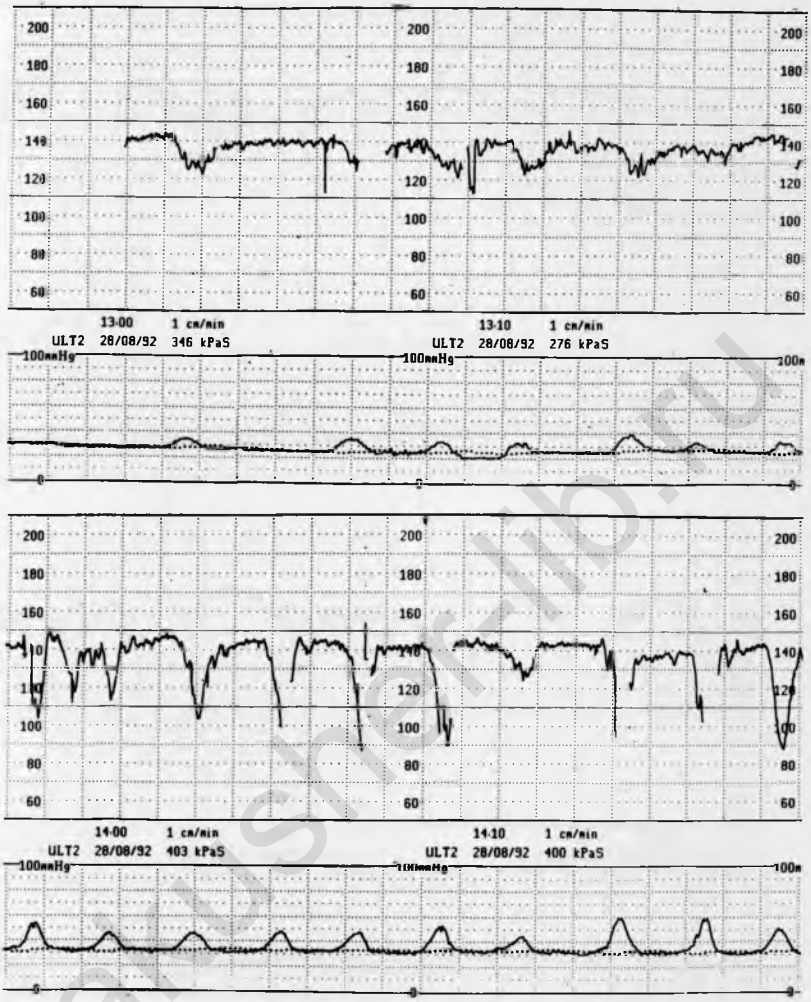

Figure 8-4. (A–F) Sequential CTG changes to abnormal.

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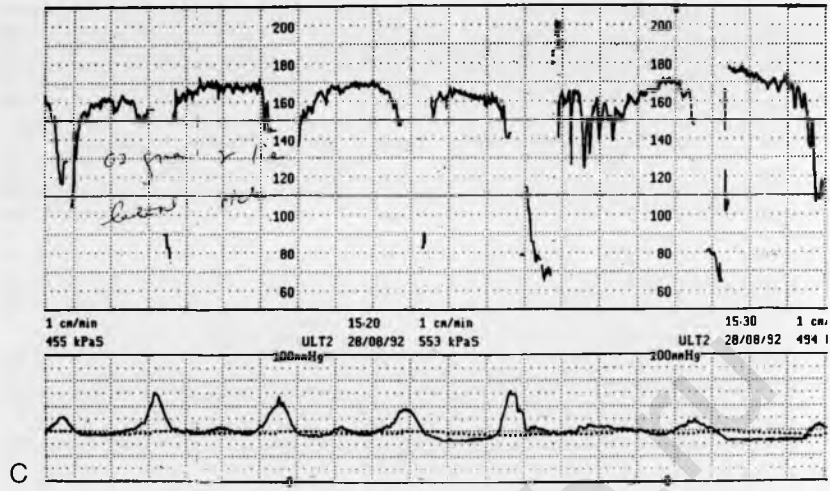

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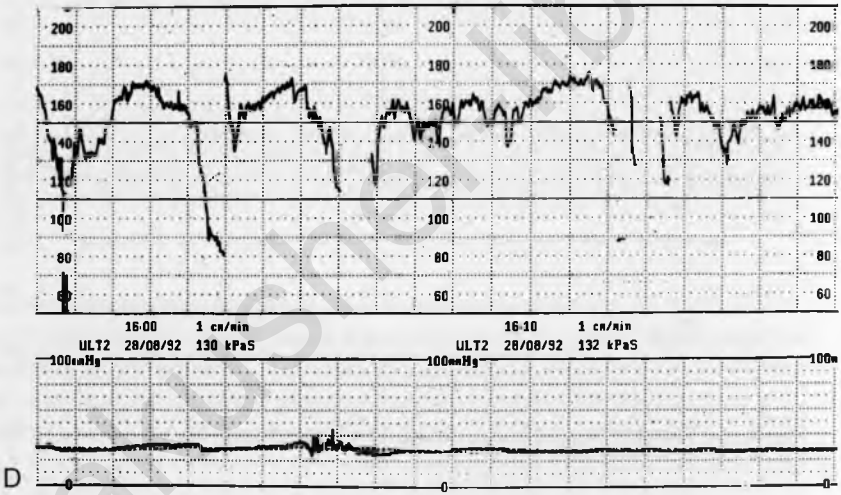

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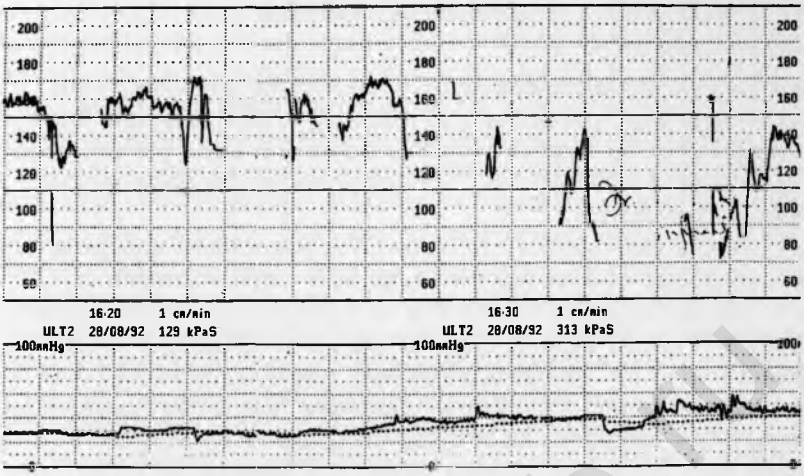
Figure 8-4. (*Continued*)



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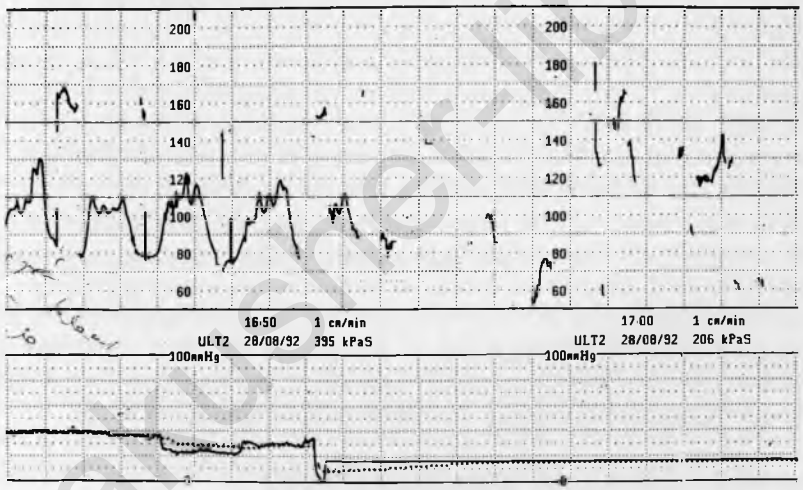


F

Figure 8-4. *(Continued)*

before acidosis develops. It was estimated that, in these situations, for 50% of the babies to become acidotic took 115 min with repeated late decelerations, 145 min with repeated variable decelerations and 185 min with a flat trace.[6] Therefore, it can be safely assumed that if the AT was reactive it is reasonable to perform intermittent auscultation. In some institutions this is further enhanced by 20 min of electronic monitoring 2–3-hourly in low-risk labour.

RANDOMIZED CONTROLLED TRIALS ON ADMISSION TEST

A recent systematic review of three randomized controlled trials ($n = 11\,259$) and 11 observational studies ($n = 5831$) suggests that there is no evidence to support the labour admission test.[7] The two large randomized controlled trials[8,9] did not show any benefit in terms of neonatal outcome.

In those who had the admission CTG, the epidural analgesia rate was increased (relative risk [RR] 1.35; 95% confidence interval [CI] 1.1–1.4), as well as the incidence of continuous electronic fetal monitoring (EFM) (RR 1.3; 95% CI 1.2–1.5) and fetal blood sampling (FBS) (RR 1.3; 95% CI 1.1–1.5). The operative delivery rate was the same in the two groups, suggesting that this may have been due to the increased fetal scalp blood-sampling rate in those who had the admission CTG. The study from Dublin[9] contributed 8580 out of the total of 11 259 cases for this meta-analysis.[7] In the Dublin study,[9] the presence of clear amniotic fluid was a prerequisite to enter the study. In order to achieve this, artificial rupture of membranes was performed at a mean cervical dilatation of 1.2 cm. This may not be an acceptable practice in many centres. We believe that the latter study influenced the outcome of the meta-analysis. There were higher rates of continuous EFM and a higher incidence of FBS, and this may be because, in this study, 32% of admission CTGs were considered suspicious or abnormal – an unexpectedly high percentage in early labour in women with clear amniotic fluid. Despite no definitive evidence to support admission CTG, it is carried out in many units and the CTG not discontinued, and we believe that this may be due to lack of confidence in interpretation of the CTG or to the shortage of midwives to provide one-to-one care, including auscultation every 15 min as recommended by NICE.[1]

OTHER FORMS OF ADMISSION TEST

The amniotic fluid index (AFI) and Doppler indices of umbilical artery blood flow, to assess fetal wellbeing in early labour, have been evaluated as useful screening tests for fetal distress in labour.[10,11] However, these tests need expensive equipment and expertise compared with an admission CTG.

Assessment of Amniotic Fluid Volume

Perinatal mortality and morbidity are increased in the presence of reduced amniotic fluid volume at delivery.[12,13] A reproducible semiquantitative measurement of amniotic fluid volume in early labour could conceivably be used as an adjunct to an admission CTG to triage a fetus to a high- or low-risk status in early labour.[14] In a study

involving 120 women in early labour, it was found that ultrasound measurement of the vertical depth of two amniotic fluid pockets could be easily and rapidly performed by medical and midwifery staff and that the results were easily reproducible.[15] It found that a vertical depth of two pools of amniotic fluid over 3 cm was highly sensitive and predictive when used as a predictor of the absence of significant fetal distress in the first stage of labour. In this study, six women had a vertical depth less than 3 cm; four of these women had a caesarean section in the first stage of labour for fetal distress, and in three of the newborns the cord pH was <7.2. None of the women who had amniotic fluid volume greater than 3 cm required caesarean section for fetal distress. In a study of 1092 singleton pregnancies,[16] amniotic fluid volume was 'quantified' by measuring the AFI, using the four-quadrant technique.[17] An AFI of less than 5 in early labour, even in the presence of a normal admission CTG, was associated with higher operative delivery rates for fetal distress, low Apgar scores, more infants needing assisted ventilation and a higher admission rate to the neonatal intensive care unit. When the admission CTG was suspicious, an AFI of greater than 5 was associated with better obstetric outcome compared with those with an AFI of less than 5. The low AFI of below 5 may indicate incipient hypoxia and the stress of cord compression, or gradual decline of oxygenation with contractions in labour may be the cause of poor outcome.

Umbilical Artery Doppler Velocimetry

Umbilical artery Doppler velocimetry has been used as an admission test. However, it has been shown to be a poor predictor of fetal distress in labour in the low-risk population.[10,18] A larger study of 1092 women has shown Doppler velocimetry on admission to be of little value in the presence of a normal admission CTG. However, in cases with a suspicious admission CTG, normal Doppler velocimetry was associated with fewer operative deliveries for fetal distress, better Apgar scores and less need for assisted ventilation or admission to the neonatal intensive care unit.[16]

RELATIONSHIP OF NEUROLOGICALLY IMPAIRED TERM INFANTS TO RESULTS OF ADMISSION TEST

There is controversy regarding the value of continuous EFM, let alone an admission test. Other than acute or terminal patterns of prolonged bradycardia or prolonged decelerations of a large amplitude and duration, there is little information regarding FHR patterns and neurological handicap at term[19–22] other than some observation of neurological impairment and non-reactivity,[23–25] especially in the presence of meconium. In an investigation of 48 neurologically

impaired singleton term infants, the admission FHR findings and the FHR patterns 30 min before delivery were analysed.[26] Findings of this investigation are shown in Tables 8-2 and 8-3.

Based on the data in Tables 8-2 and 8-3, it is clear that fetuses with a reactive AT (accelerations) will show the following features prior to or when becoming hypoxic: all will exhibit decelerations (100%); almost all will have reduced baseline variability (93%) and tachycardia (93%). The one case where the FHR did not exceed 160 bpm showed an increase in the baseline rate by 25% and decelerations, which can be picked up on auscultation and action taken. On the other hand, if the AT is non-reactive the development of further abnormal features with progress of labour are variable and subtle; this is difficult to recognize by intermittent auscultation. This is because already there might have been hypoxic damage and the fetus is unable to respond. In those with a non-reactive AT, nearly 82% had decelerations on the AT and 64% had reduced baseline variability (below 5 bpm) and many (82%) had a normal baseline rate. The fact that a hypoxic fetus can have a normal baseline rate and shallow decelerations of less than 15 bpm in a non-reactive trace when the baseline variability is below 5 bpm is not widely known (see Fig. 8-3).

Table 8-2. Admission FHR Findings in 48 Neurologically Impaired Term Infants Separated on the Basis of FHR Reactivity[26]

FHR PATTERN ON ADMISSION UP TO 120 MIN	REACTIVE ($n = 15$)	NON-REACTIVE ($n = 33$)
FHR variability (average)	14 (93%)	12[a] (36%)
Decelerations	2 (13%)	27 (82%)
Tachycardia	0 (0%)	6 (18%)

[a] $P < 0.001$.

Table 8-3. FHR Pattern in the Last 30 min Before Delivery, Separated on the Basis of Admission FHR pattern[26]

ADMISSION FHR PATTERN LAST 30 MIN BEFORE DELIVERY	REACTIVE ($n = 15$)	NON-REACTIVE ($n = 33$)
FHR variability (average)	1 (7%)	11[a] (33%)
Decelerations	15 (100%)	5 (15%)
Tachycardia	14 (93%)	9[b] (27%)

[a] $P < 0.05$.
[b] $P < 0.001$.

All fetuses who exhibited a reactive AT had decelerations and a gradually increasing baseline FHR suggestive of developing fetal hypoxia. It is not difficult to identify this increase in baseline FHR on auscultation (see Fig. 13-6A–J). A randomized study compared the obstetric outcome in a group who had intermittent auscultation and 2-hourly 20 min of CTG following the admission test with a group who had continuous EFM.[27] The obstetric outcome, in terms of operative delivery, low Apgar scores and admission to the neonatal unit, was the same in the two groups. The interval between admission to the labour ward to first detected FHR abnormality was the same in the two groups. This finding reassures that FHR can be confidently auscultated for changes that will indicate 'fetal distress' if the AT showed a reactive trace. On the other hand, if the trace was non-reactive with silent pattern (baseline variability below 5) for over 90 min with shallow or no decelerations, the fetus may already be compromised or is likely to become compromised. Action should be taken to establish the acid–base status by FBS, or delivery should be considered. Failure to take action may end in fetal death (Fig. 8-5A–J). It is difficult to know whether the fetus is already hypoxic or acidotic or is suffering from another insult (e.g., infection, brain injury due to haemorrhage, etc.) unless the acid–base status is known prior to or after delivery.

Fetuses with hypoxia may have a normal baseline rate, but with no accelerations, silent pattern (baseline variability below 5) and shallow decelerations (amplitude less than 15 beats) (see Fig. 8-3). Such a fetus may not stand the stress of labour and may die within 1–2 h of admission. Figure 8-6A–D shows an admission test trace with a baseline rate of 140 bpm. With progress of labour the baseline variability is further reduced (less than 5 beats) without rise in the baseline rate and the fetus dies in a span of 40 min. There appears to be some difficulty in identifying the correct baseline rate and some may consider the baseline to be 120 with accelerations. Careful attention to reducing baseline variability would indicate that the correct baseline rate was 140 bpm with decelerations.

PLANNING MANAGEMENT

An admission test is helpful when planning the subsequent care in labour. High-risk women or women with suspicious or abnormal admission tests should have continuous EFM throughout labour. A normal admission test is an insurance policy that permits us to encourage mobilization with no further need to perform EFM for 3–4 h or until signs of the late first stage of labour are apparent. Even in the second stage of labour a few minutes' strip of EFM after a contraction should be enough in the low-risk woman. Alternative delivery positions,

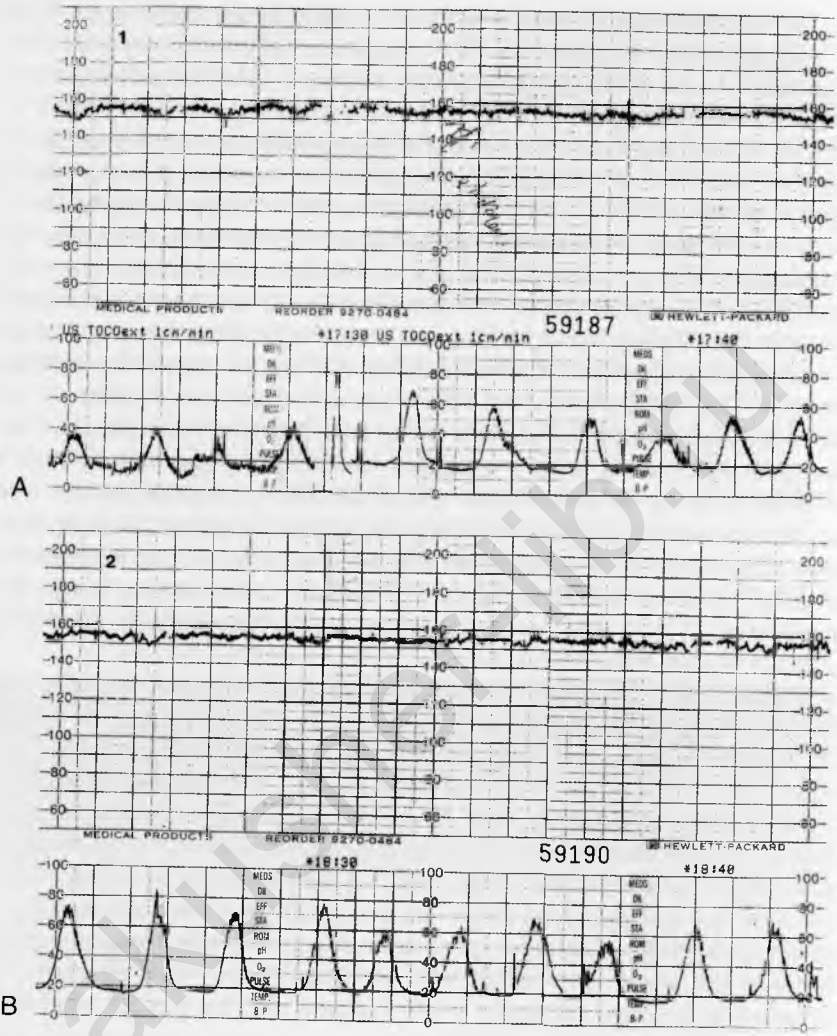
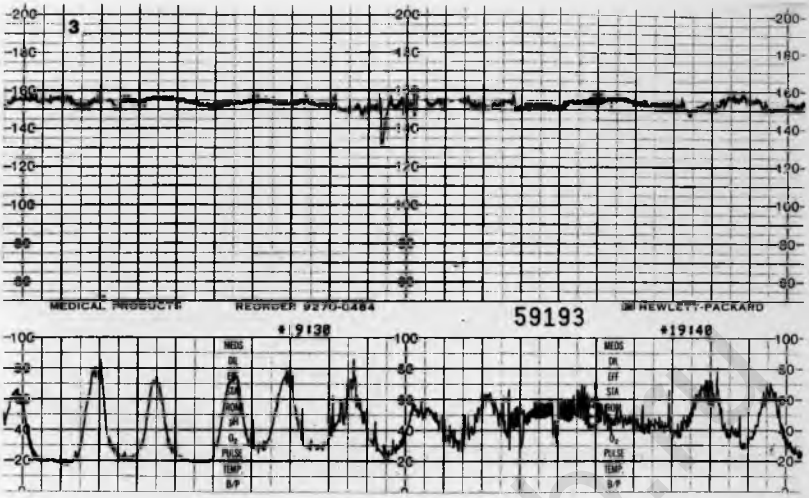


Figure 8-5. (A–J) A trace with reduced baseline variability for >90 min is abnormal, especially in the presence of shallow decelerations in a non-reactive trace. Sequential traces till the baby's demise are shown.



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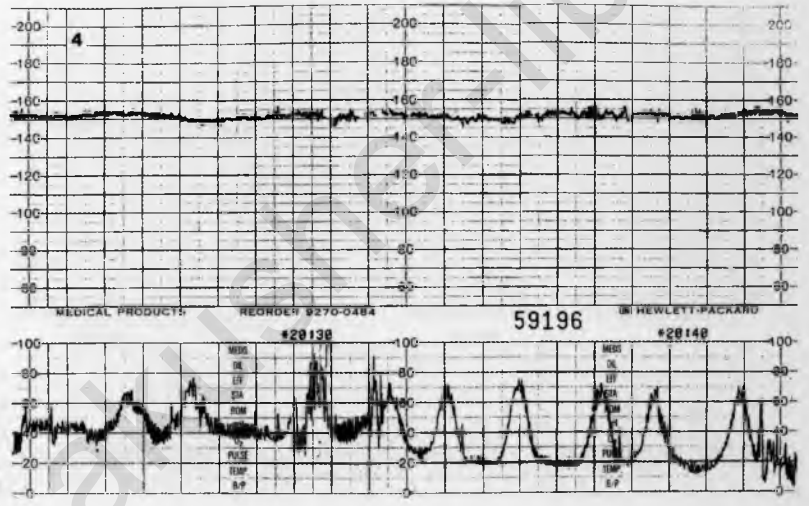

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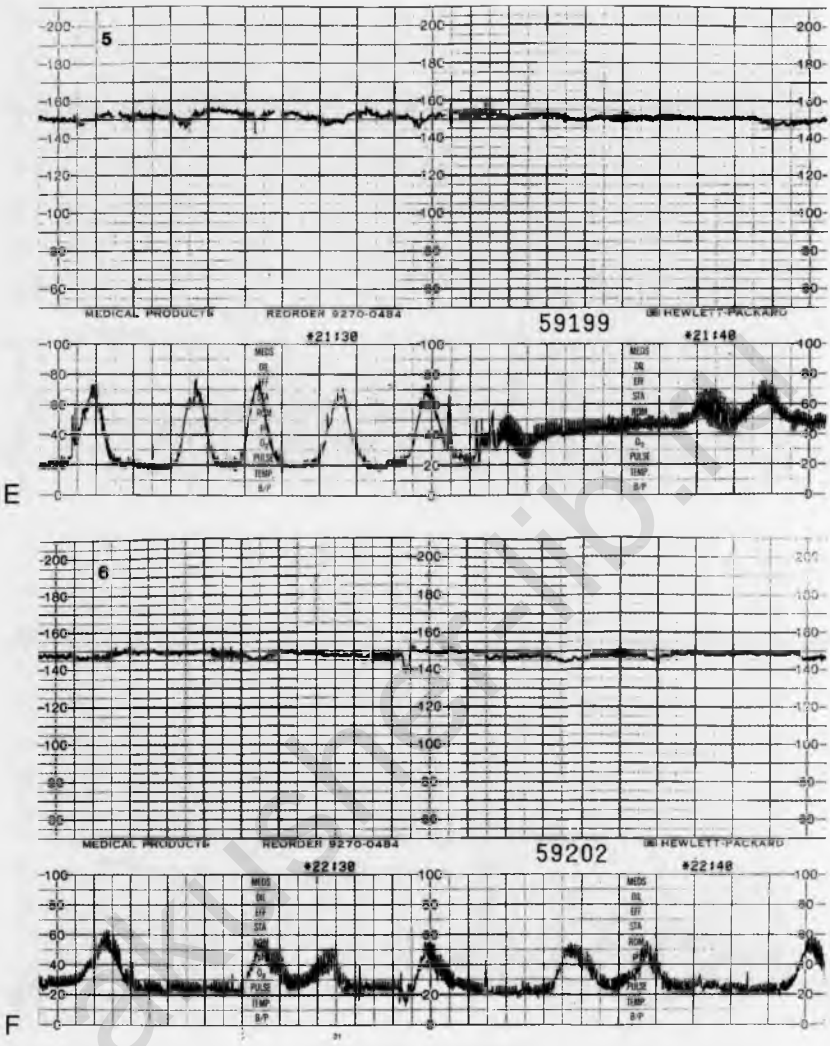
Figure 8-5. (*Continued*)



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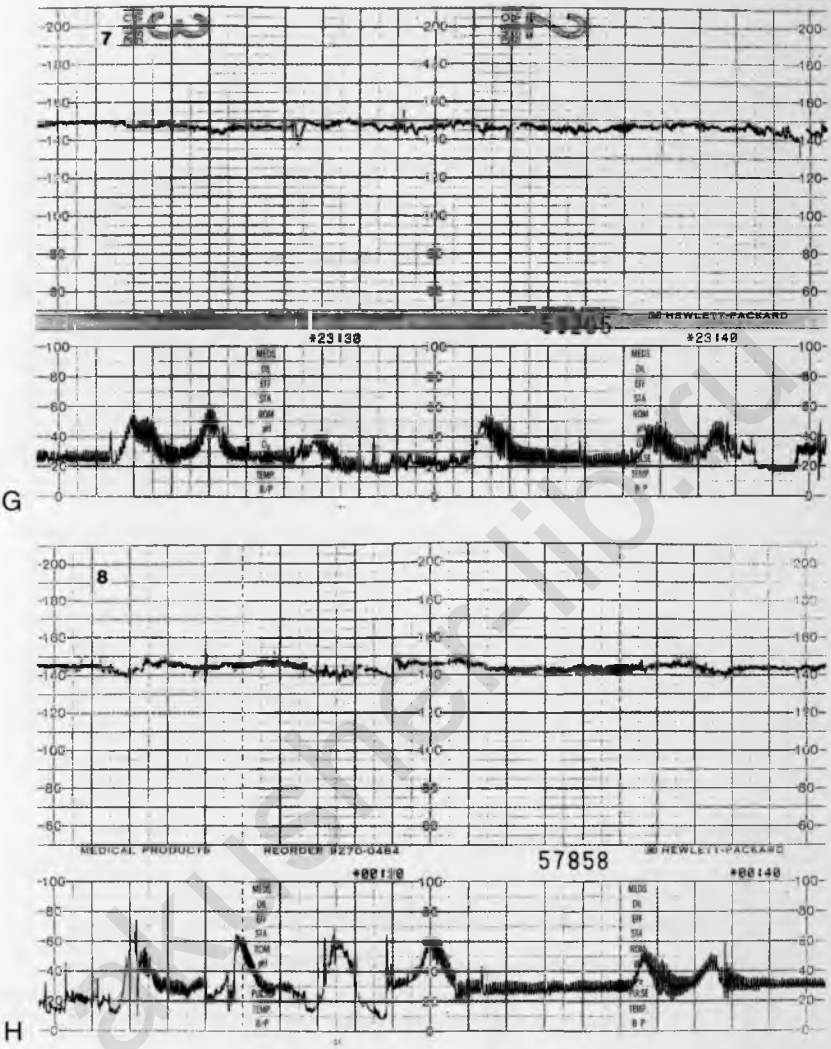

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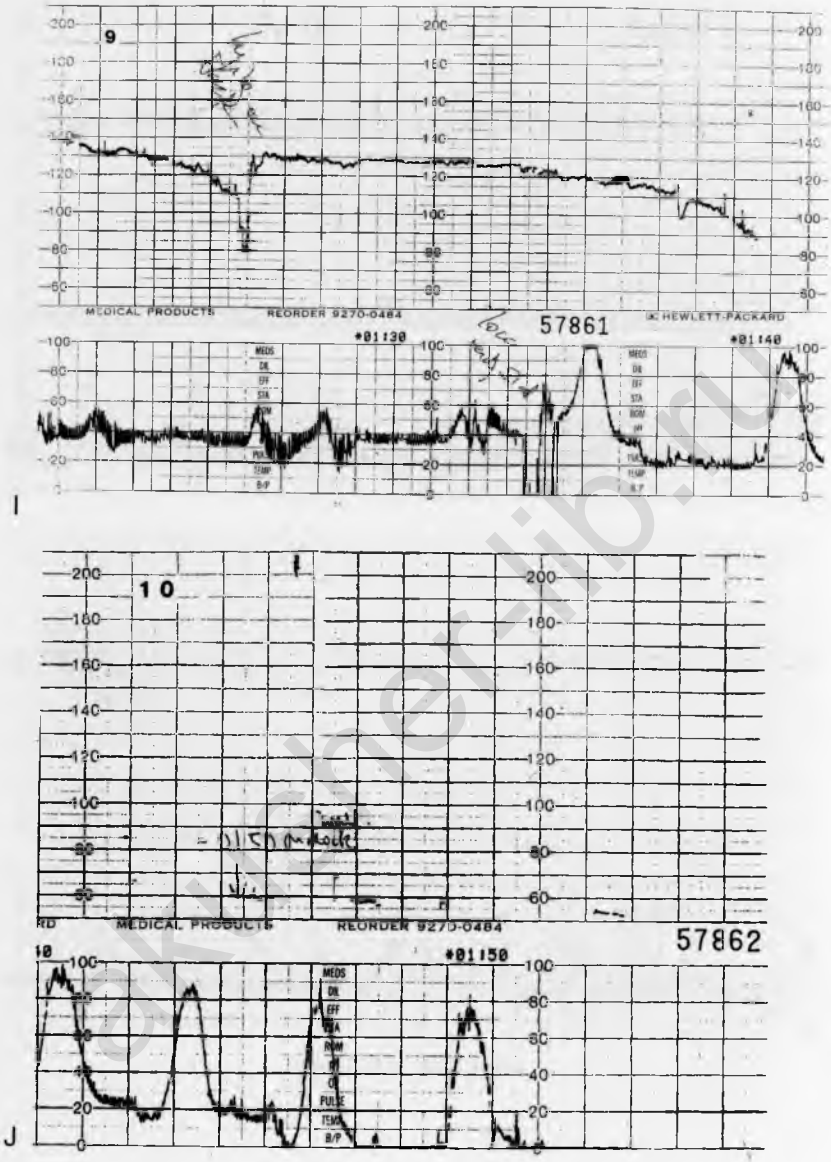

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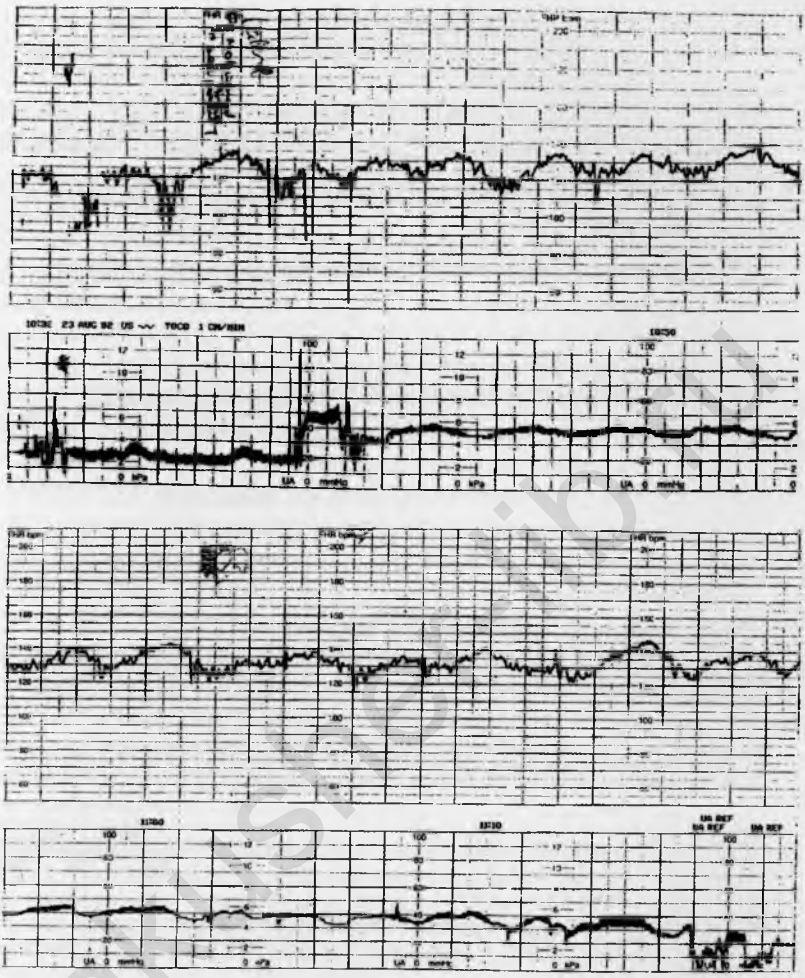

Figure 8-6. (A–D) A non-reactive trace with normal baseline FHR, silent pattern and shallow decelerations. Sudden fetal demise within 50 min of admission.

(Continued)

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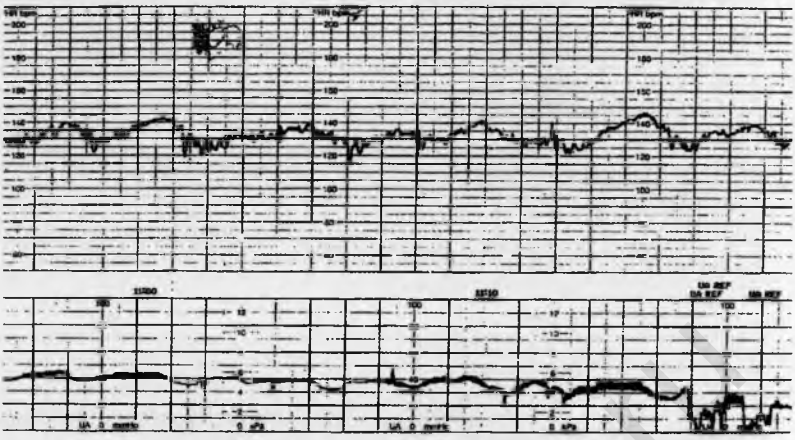

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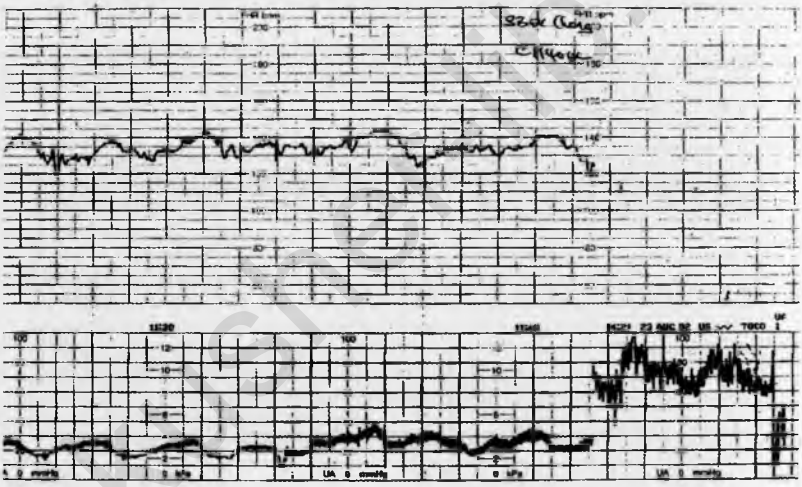

Figure 8-6. (*Continued*)

the use of water immersion in labour and preparations for water birth may be more confidently pursued.

A shortage of paper imposes a discipline requiring careful consideration. An AT followed by monitoring in the late first stage and second stage, the time of greatest stress, appears appropriate.

HOW LONG SHOULD AN ADMISSION TEST LAST?

An AT should last as long as necessary until it is normal. This implies a consideration of fetal sleep and fetal behavioural states. If two accelerations, a normal rate and normal variability are seen in the first 5 min then that is very reassuring. It is useful if two or more contractions are witnessed during this time as this will provide reassurance that there is no stress to the fetus with the contractions. If EFM is commenced at the start of a quiescent phase for the fetus then it will need to be continued until the fetus reawakens. Most ATs should last 15–30 min; however, the mother with a normal trace in 5 min, keen for mobilization and natural labour, should not be monitored unduly. Midwives can gain more confidence in the home-birth situation by applying these principles and using a hand-held Doptone and, if necessary, a connected printer.

The parents should be given a choice, as in every matter; however, the choice provider may find it difficult to offer truly informed choices. It seems to us the simple question that the parents should be asked is 'Would you like us to check that your baby is OK?'

EFM should be appropriate: not too much, not too little.

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ASSESSMENT OF UTERINE CONTRACTIONS

Donald Gibb, Sabaratnam Arulkumaran

Effective contractions (the *powers*) of the uterus are an essential prerequisite for labour and vaginal delivery. The progress of labour, evidenced by dilatation of the uterine cervix and descent of the presenting part, is the final measure of contractions. During the journey through the birth canal (the *passages*), the passenger is intermittently squeezed and stressed by the contractions. Maternal blood flow into the uteroplacental space ceases when the intrauterine pressure (IUP) exceeds the pressure of flow of blood into the retroplacental area, which could be 30–45 mmHg due to the function of the spiral arteries. A well-grown fetus with good placental reserve tolerates this as 'normal stress' and displays no change in the fetal heart rate (FHR). A compromised fetus may show changes with this stress, and reduction of the retroplacental pool of blood due to contractions will be manifested as late decelerations. In a normal fetus, stress can be brought about by cord compression, which will be shown as variable decelerations. The presence of atypical variable decelerations indicates that there is cord compression and at the same time there is reduction of the retroplacental pool of blood (e.g., atypical variable decelerations with late recovery, or a combination of variable and late decelerations). Oxytocics or prostaglandins are given expressly to increase the contractions; when they are given to induce labour the fetus is often already at risk. Particular care should be taken to 'manage' the contractions under these circumstances and to monitor the FHR continuously.

RECORDING

The commonest method of assessing contractions is with the hand placed on the abdominal wall over the anterior part of the uterine fundus. This permits observation of the duration and frequency of contractions. A subjective impression is gained of their strength. This is entirely adequate if performed intermittently in normal low-risk labour.

Continuous monitoring of uterine contractions is performed using external tocography. The tocograph transducer (Fig. 9-1) is a strain gauge device detecting forward movement and change in the abdominal wall contour due to change in shape of the uterus with an anterior thrust on account of the contraction; it records continuously

Figure 9-1. External tocography transducer (Hewlett-Packard 8040). *(Courtesy of Hewlett-Packard.)*

what the hand feels intermittently. The transducer is placed without the application of jelly on the anterior abdominal wall, near the uterine fundus, and secured with an elastic belt. It is important to adjust the tension of the belt for comfort and to secure an adequate recording. Currently tocograph transducers are available that are kept in position with an adhesive instead of a belt, avoiding a feeling of restriction. These transducers are also cordless, thus allowing the mother to move freely. Obesity and a restless mother can compromise uterine contraction recording; in these circumstances, and in other clinical situations, there may be a role for palpation of uterine contractions or IUP measurement using an intrauterine catheter (Fig. 9-2). IUP

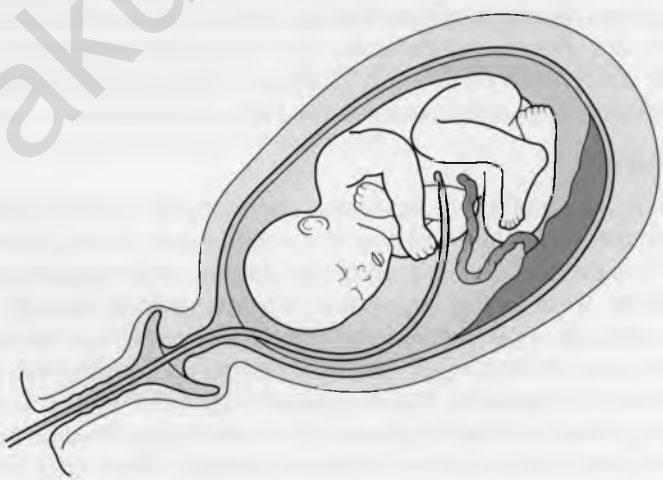

Figure 9-2. Intrauterine catheter in situ.

measurement is the most effective method of recording contractions including a fairly precise measure of the strength in millimetres of mercury (mmHg) or kilopascals (kPa).[1] The technology for this has been developed and several disposable, solid-state devices are available – for example, the Intran II catheter (Fig. 9-3) (Utah Medical Products, Utah). Figure 9-4 shows the change in recording in an obese mother seen after converting from external to internal tocography over a period of 20 min.

Figure 9-3. Gaeltec transducer tipped catheter. *(Gaeltec Ltd., Scotland.)*

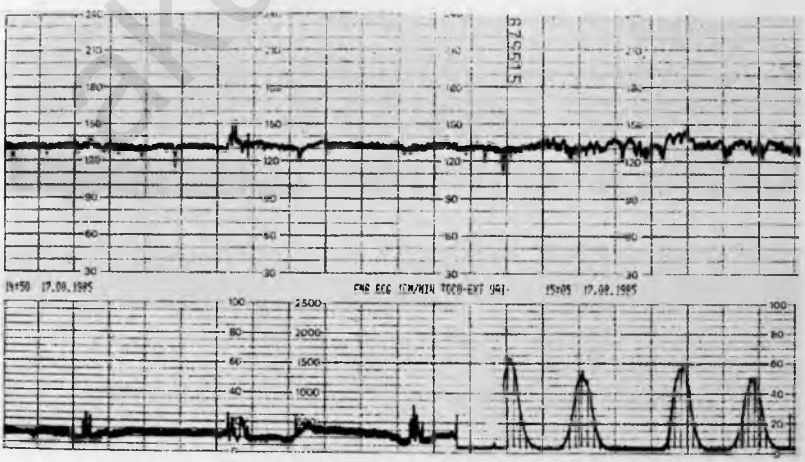

Figure 9-4. External tocography trace followed by internal recording.

MEASUREMENT

The most relevant measure of contractions in labour is their outcome: dilatation of the cervix and descent of the presenting part resulting in spontaneous vaginal delivery. The quality of contractions present is very variable. A simple assessment of the frequency of contractions (number per 10 min), the mean duration (in seconds) and a subjective impression of strength (weak, moderate or strong) usually suffices. The method of recording this is seen on the partogram (see Fig. 2-3). When IUP monitoring is being used the opportunity arises for greater precision. In 1957 Caldeyro-Barcia and colleagues suggested Montevideo units using average pressure multiplied by frequency.[2] In 1973 Hon and Paul introduced the concept of contraction area under the curve: uterine activity units.[3] In 1977 Steer introduced the active contractions area under the curve: kilopascal seconds per 15 min.[4] A simple system based on Systeme Internationale (SI) units has been considered and recommended by the Royal College of Obstetrics and Gynaecologists Working Party on Cardiotocograph Technology.[5]

The appropriate units for IUP quantification are listed in Table 9-1, and the appropriate units for measuring the total activity over a period of time are listed in Table 9-2. The recommended period of measurement is 15 min.

Consistent terminology is essential.

Table 9-1. Units for Intrauterine Pressure Quantification

Mean contraction active pressure (MCAP)	kPa
Mean baseline pressure	kPa
Mean contraction frequency	Number per 10 min
Mean duration of contractions	Seconds
Mean active pressure (MAP): sum of MCAP divided by time	kPa

Table 9-2. Units for Measuring the Total Activity over a Period of Time

Active pressure integral (API)	kPa
Baseline pressure integral (BPI)	kPa
Number of contractions per period	
Total duration of contractions	Seconds
Proportion of active time	Per cent

CLINICAL APPLICATION

What are the indications for continuous tocography? In general, continuous external tocography is performed when continuous FHR monitoring is being performed. This is a pragmatic, practical approach; however, it ignores the rationale that the indication for each is separate although they may be related. If the FHR pattern is normal and the labour progress is normal then continuous tocography does not provide additional useful information, and the woman could be spared the discomfort of the tocography belt. There remains the issue that if the fetal heart pattern or labour progress becomes abnormal then information is already available about the pre-existing contractions, which are of importance. Hence two-channel monitoring – of the heart rate and the contractions – is standard. Whenever the heart rate is abnormal or labour progress is abnormal requiring treatment, the need for continuous contraction recording is clear.

Figure 9-5 shows an admission test performed on a woman with tightenings. Although the tocographic tracing suggests frequent regular contractions, the woman was not experiencing pain and did not go into labour that day. The tocography transducer may detect localized contractions that are not propagating throughout the uterus, as also shown with marked irregularity in Figure 9-6.

The diagnosis of labour is not made from the cardiotocograph.

What are the indications for internal tocography using an IUP catheter? Other than in an obese or restless mother, external

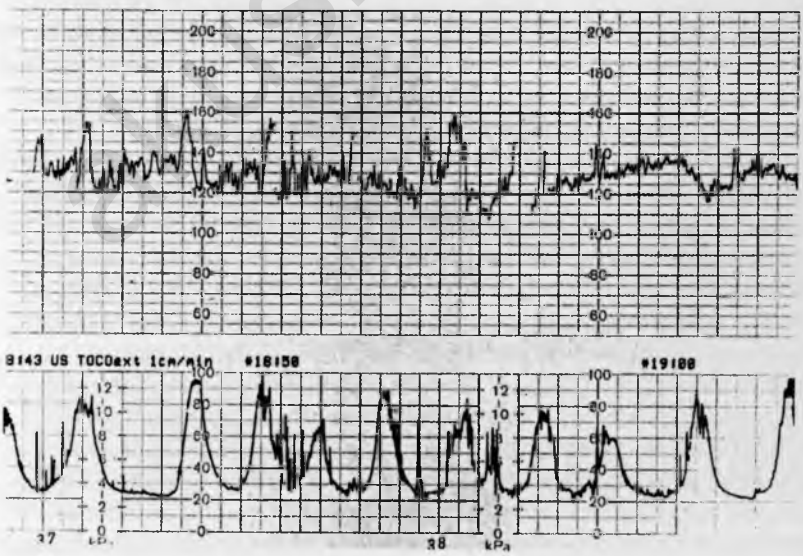

Figure 9-5. Contractions recorded – subject not in labour.

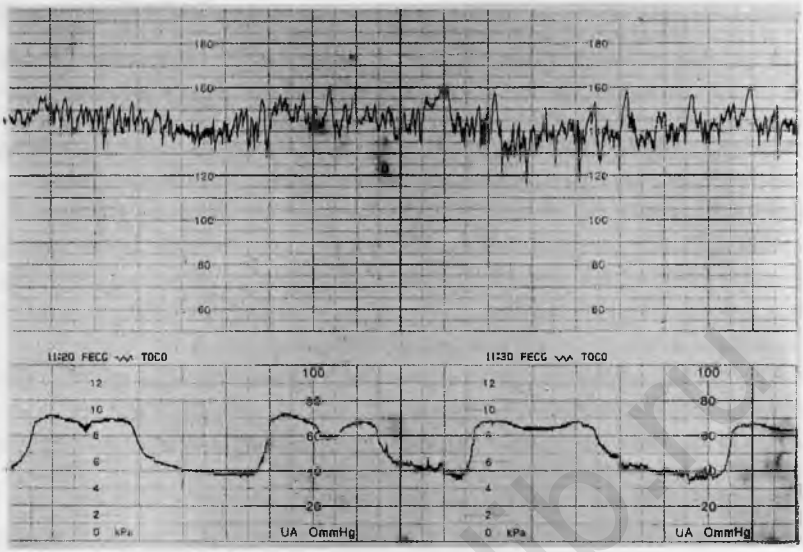

Figure 9-6. Contractions recorded – subject not in labour.

tocography provides enough information to interpret an abnormal fetal heart tracing. The management of the contractions is another issue. If induction of labour or augmentation of slow labour (see Fig. 2-3) is non-progressive then the more complete information derived from an IUP catheter might be useful.[6] However, available data suggest that, in most of these situations, titration of the oxytocin infusion rate based on frequency and duration of contractions recorded by external tocography is adequate.[7,8] The exception might be the obese, restless mother or the nullipara with an occipitoposterior position with poor progress of labour requiring a high-dose infusion of oxytocin.

Breech presentation in labour, which is nowadays very uncommon, and labour with a previous caesarean section scar present specific problems. Some obstetricians do not practise vaginal delivery of a baby presenting by the breech; in those who do there is some reluctance to use oxytocin if labour progress is slow. The anxiety is that the slow progress is a manifestation of fetopelvic disproportion and therefore the sign to terminate the labour by performing a caesarean section. The counter-view is that poor contractions are just as likely (if not more likely) to occur in a breech presentation. If complete assessment of the fetopelvic relationship has shown favourable features and the contractions are shown to be weak then oxytocin augmentation may be safely undertaken. The additional information derived from an IUP catheter may be useful under these circumstances.

A similar rationale applies to poor labour progress in a woman with a previous caesarean section scar. Additionally, there is the further concern for the integrity of the uterine scar. Scar rupture or dehiscence may not manifest scar pain, tenderness, vaginal bleeding or alteration in maternal pulse and blood pressure, or may manifest these some time after the event; FHR or uterine activity changes may be an earlier sign of scar disruption.[9,10] Figure 9-7 shows a case where resiting of the catheter led to an acceptable tocographic tracing in spite of scar dehiscence; presumably the replaced catheter was in a loculated pocket of normal pressure. In some centres there is a link between the indication for internal FHR monitoring with an electrode and internal pressure monitoring with a pressure catheter. There is no logic in this as each addresses separate issues. Excessive use of internal monitoring is invasive psychologically as well as physically.

There is a very limited place for IUP measurement.

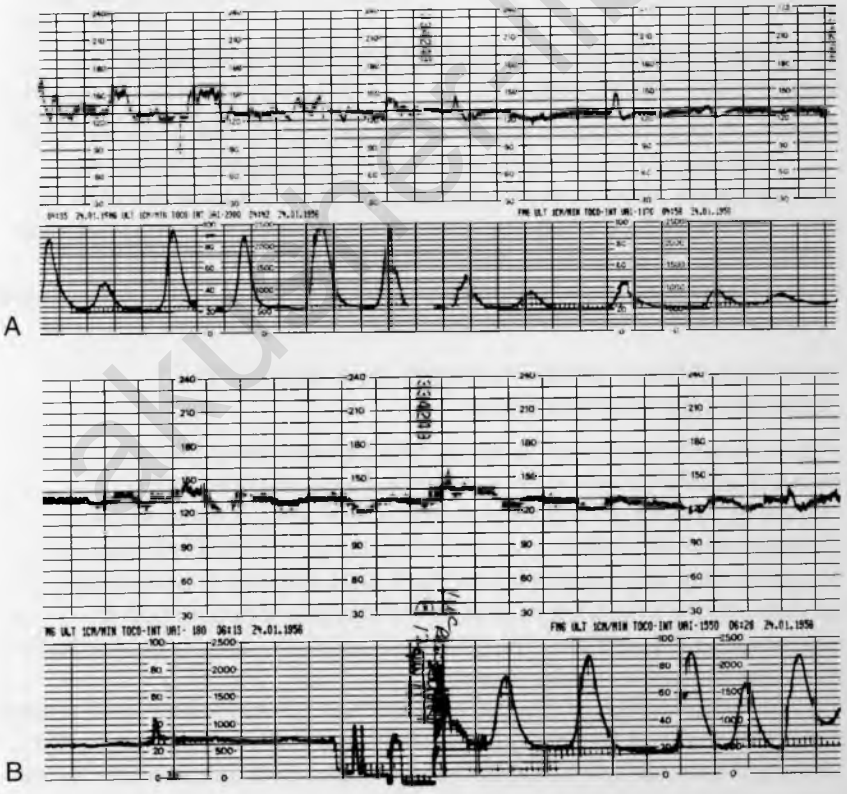

Figure 9-7. Scar dehiscence: (A) reduction in uterine activity; (B) intrauterine pressure catheter replaced in another pocket showing normal uterine activity.

Uterine hyperstimulation and fetal hypoxia are a real possibility when oxytocics are used and, in these circumstances, continuous electronic FHR monitoring is important and this is discussed in Chapter 10.

CONTRACTION MONITORING WITH THE USE OF PROSTAGLANDINS FOR INDUCTION OF LABOUR

It is important to record uterine contractions and the FHR prior to, and soon after, insertion of vaginal prostaglandin (PG) pessaries or gels. The rate of absorption of PG varies from woman to woman based on the pH, temperature and moisture content of the vagina and whether there is infection, inflammation or abrasion in the vagina. Rapid absorption can give rise to tetanic or too frequent contractions, which need not be painful but may cause suspicious/abnormal FHR changes, including prolonged deceleration that may compromise the fetus if prompt action is not taken. Action can be in the form of removing the PG pessary if possible and/or use of tocolytic agents to abolish uterine contractions.

CONTRACTION MONITORING AFTER EXTERNAL CEPHALIC VERSION

A small abruption leading to uterine irritability and FHR changes may occur following external cephalic version (ECV) without much pain, and hence the need to monitor uterine contractions and the FHR for 30–60 min after ECV. If uterine irritability is observed with too frequent contractions (five in 10 min), the FHR may become abnormal and hence the recording should be continued until no, or infrequent, uterine contractions are observed and the FHR pattern is normal.

CONTRACTION MONITORING IN CASES OF SUSPECTED ABRUPTION

In the presence of clinical features suggestive of abruption (i.e., bleeding and/or continuous abdominal pain if there is uterine irritability), uterine contractions and the FHR should be monitored. Consideration should be given for early delivery if the FHR trace is unsatisfactory with uterine irritability, if fetal maturity is not a major concern. In the presence of uterine irritability and suspicious or pathological FHR pattern, the FHR can suddenly deteriorate leading to the need for an emergency delivery.

The development of clinical skills and an educational motive remain important reasons for giving due attention to the contractions. In the USA, and increasingly in the UK, many cases of litigation relate to the misuse of oxytocin. Better understanding of the labour process and contractions should help to counter such misuse.[9,11]

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OXYTOCIN AND FETAL HEART RATE CHANGES

Sabaratnam Arulkumaran, Donald Gibb

Oxytocin is commonly used for induction and augmentation of labour. Many medico-legal cases relate to the misuse of oxytocin. Oxytocin does not have a direct influence on the fetal heart rate (FHR) or on the controlling cardiac centres in the brain, as is the case with some anaesthetic and antihypertensive drugs. Its influence is indirect via increased uterine activity, mostly due to increased frequency of contractions or baseline pressure (hypertonus). Increase in duration or amplitude of contractions can also lead to FHR changes. The 2001 NICE guidelines defined hyperstimulation as more than five contractions in 10 min (some literature defines it as tachy- or polysystole) and if it is associated with FHR changes it is defined as 'hyperstimulation syndrome'.[1] There is a confusion on terminology, which is clarified by Oláh & Steer.[2] They do not use the term 'hyperstimulation syndrome' but rather tachysystole, hyperstimulation (when Syntocinon is being used) and hypertonus (when there is a rise of the baseline). We agree with this as we do not think that CTG changes in this situation are a syndrome in the way that ovarian hyperstimulation is a syndrome. We do not think that we should use the word syndrome as described by NICE. Figure 10-1 shows fetal bradycardia due to 'tetanic' or sustained contractions lasting for 3–4 min, caused by oxytocin hyperstimulation. Because the fetus was healthy with a normal reactive FHR prior to the episode, the transient bradycardia returned to normal once the oxytocin infusion was reduced and the abnormal contractions ceased.

Figure 10-2 shows fetal bradycardia due to 'hypertonic' uterine activity. The baseline pressure was elevated by 15 mmHg for 3 min despite regular contractions. The raised baseline pressure reduced the perfusion in the retroplacental area leading to FHR changes, which returned to normal once the baseline pressure settled to normal levels, restoring normal perfusion.

Figure 10-3 shows a reactive trace with one contraction in 3 min. An oxytocin infusion was commenced 10 min from the start of this segment at a rate of 1 mU/min. This resulted in the late decelerations and changes seen in the latter part of the trace. The contraction recording shows no increase in frequency or duration of contractions,

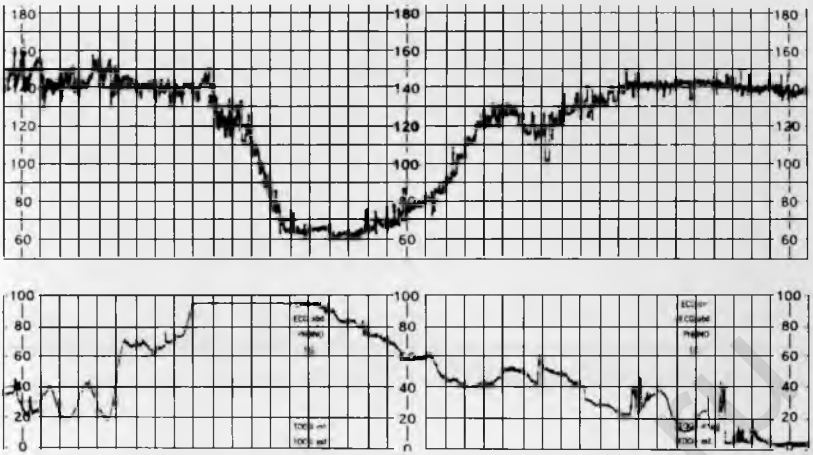

Figure 10-1. Sustained contraction and bradycardia.

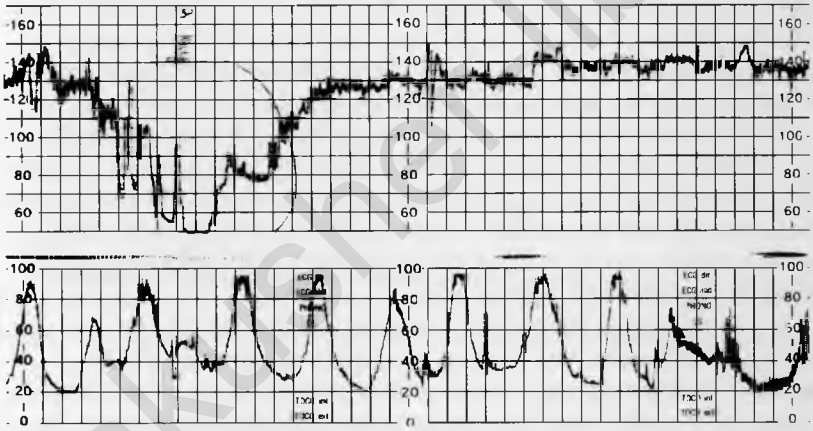

Figure 10-2. Hypertonic contraction and bradycardia.

nor an increase in baseline pressure, but does show an increase in amplitude of contractions. Discontinuation of the infusion resulted in return of the FHR trace to normal.

The FHR changes associated with oxytocin infusion may be caused by compression of the cord with contractions, or by the reduction in placental perfusion due to increased intrauterine basal pressure and frequent contractions cutting off the blood supply to the placenta. Pressure on the head or supraorbital region of the fetus can also give rise to variable decelerations. The rate of increasing hypoxia would be shown by a deteriorating trend of the FHR. The rate of decline of pH

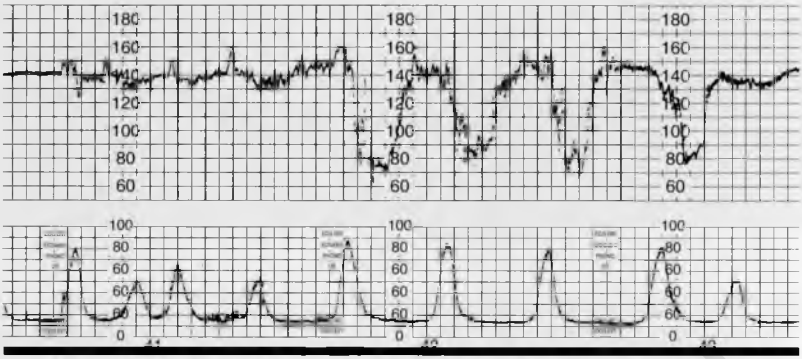

Figure 10-3. Normal trace and subsequent decelerations with oxytocin.

depends on the FHR pattern observed and the physiological reserve of the fetus.[3] A rapid decline would be anticipated in post-term and growth-restricted fetuses and those with reduced amniotic fluid with thick meconium, infection or intrapartum bleeding. Fortunately, in the vast majority of patients who are given oxytocin, FHR changes of a worrying nature are not encountered and most changes, even when they occur, are transient and resolve spontaneously, or with reduction of the dose or transient cessation of the infusion. It is good practice to run a strip of cardiotocograph prior to commencing oxytocin to make sure of good fetal health as reflected by a normal reactive FHR pattern; *if the trace is pathological then oxytocin should not be used*, as it can cause further hypoxia to the fetus by reducing the perfusion to the placenta by additional contractions.

If a pathological FHR pattern is observed in a woman on an oxytocin infusion, the infusion should be stopped, or its rate reduced, and the woman nursed on her side to improve the maternal venous return, and thus her cardiac output, in order to increase the uteroplacental perfusion. Oxygen inhalation by the mother and an intravenous bolus of tocolytic drugs to abolish uterine contractions are given in some centres. Such practice may not be necessary in the majority of cases and its value in other cases is debatable. It is known that oxytocin becomes bound to receptors, and for its action to be reduced to half can take up to 45 min after stopping the oxytocin infusion. A case may be made for the use of a bolus dose of a tocolytic drug in a patient with a grossly abnormal (pathological) FHR pattern.[4,5] There is little merit in performing a fetal scalp blood pH measurement in a patient receiving oxytocin as the FHR changes are iatrogenic. If the test is done soon after a prolonged bradycardia, or after ominous decelerations, it may show acidosis, prompting the performance of an emergency caesarean section (Fig. 10-4A). On the other hand, if a fetal

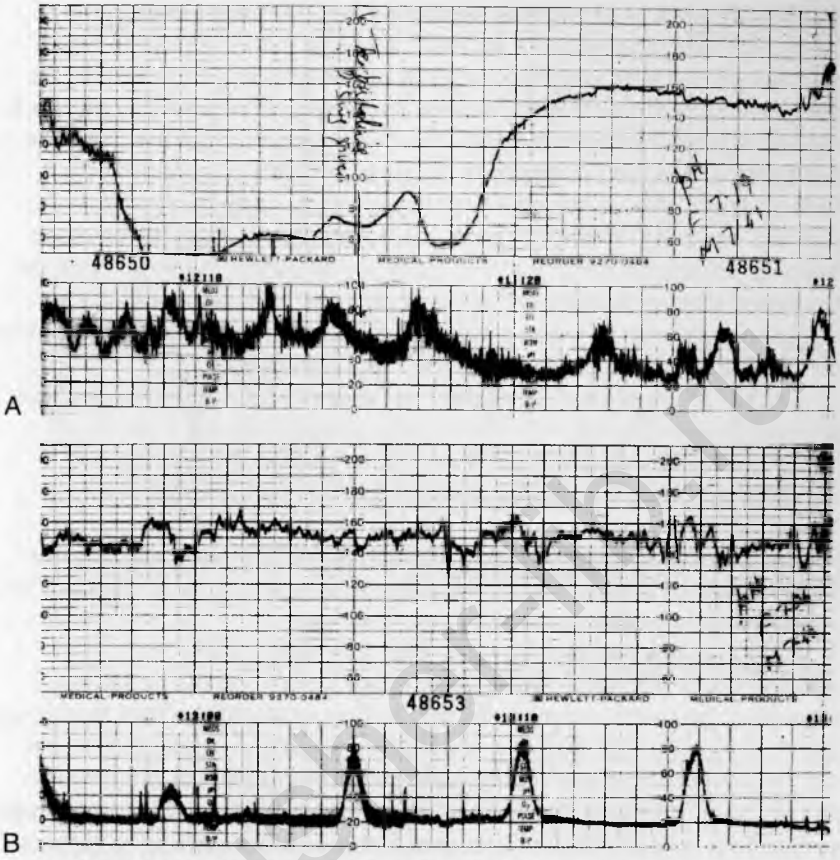

Figure 10-4. (A) Cardiotocograph: bradycardia with acidosis; (B) reversion to normal pH.

blood sample is not taken and time is allowed, the FHR recovers and within 30–40 min the scalp blood pH is likely to be normal (Fig. 10-4B). On many occasions there is no need to measure scalp blood pH and the oxytocin infusion can be restarted after the return of the FHR to normal.

It is debatable for how long the oxytocin infusion should be stopped once the FHR abnormality is detected. It is usual to wait until the abnormal features disappear and the reactive trace is seen; however, it is known that, although the trace is then normal, the fetal blood biochemistry reflected on scalp blood testing may still show a low pH, high PCO_2 and low PO_2. Additional time is required for the blood biochemistry to become normal, which takes place rapidly once the FHR is normal. Noting the time necessary for the FHR to become normal after the oxytocin infusion is stopped, and allowing an equal

length of time to elapse before restarting the infusion, would allow the biochemistry to become normal. Doubling the time period in this way before restarting oxytocin should cause little or no FHR changes compared with restarting oxytocin immediately after the FHR returns to normal. It is also advisable to resume the infusion at half the previous dose rate to reduce the chances of hyperstimulation or abnormal FHR changes. As the sensitivity of the uterus to oxytocin increases with the progress of labour,[6] such careful titration is likely to produce fewer problems of abnormal FHR changes or uterine hyperstimulation. Increased uterine activity in the late first stage and second stage of labour may be due to reflex release of oxytocin due to distension of the cervix and the upper vagina (i.e., the Ferguson reflex).[7]

Figure 10-5A shows abnormal FHR changes produced by oxytocic hyperstimulation. Even with immediate cessation of oxytocin infusion it takes about 45 min for the FHR to return to normal (Fig. 10-5B) and hence sufficient time should be given for recovery. Although it is advisable to stop the oxytocin infusion as soon as abnormal FHR patterns, such as decelerations or bradycardia, are observed, it may be adequate to reduce the oxytocin dose by half or less when the FHR is normal but there is abnormal uterine activity.

Figure 10-6A shows a reactive FHR at the beginning, but decelerations and tachycardia subsequently develop owing to increased frequency of contractions. In Figure 10-6B the FHR becomes tachycardic; towards the latter part of the trace, the dose of oxytocin was reduced to half and the tocographic transducer was adjusted. In Figure 10-6C the contractions have become less frequent, the FHR has settled to a normal baseline rate and is followed by a reactive pattern.

In cases of failure to progress in labour, oxytocin is commenced to augment uterine contractions. This may bring about FHR changes when the dose is increased to achieve the optimal target frequency of contractions. If the dose is reduced, the FHR pattern returns to normal but the uterine activity drops to suboptimal levels with no progress in labour. When FHR changes are encountered in such a situation, they may be transient and it may be worth stopping and restarting oxytocin or reducing the dose. However, if pathological FHR changes appear when oxytocin is recommenced despite these efforts, it may be better to deliver abdominally. In selected cases, further time may be given to see whether the labour will progress without the use of oxytocin. An alternative would be to stop oxytocin and perform a fetal blood sample 20–30 min later and, if the pH is normal, to restart the oxytocin infusion and observe for any rise in the baseline rate and/or reduction of baseline variability. In the absence of these changes, or in the absence of increase of the width or depth of the decelerations, a cervical assessment can be made to assess progress after 1–2 h.

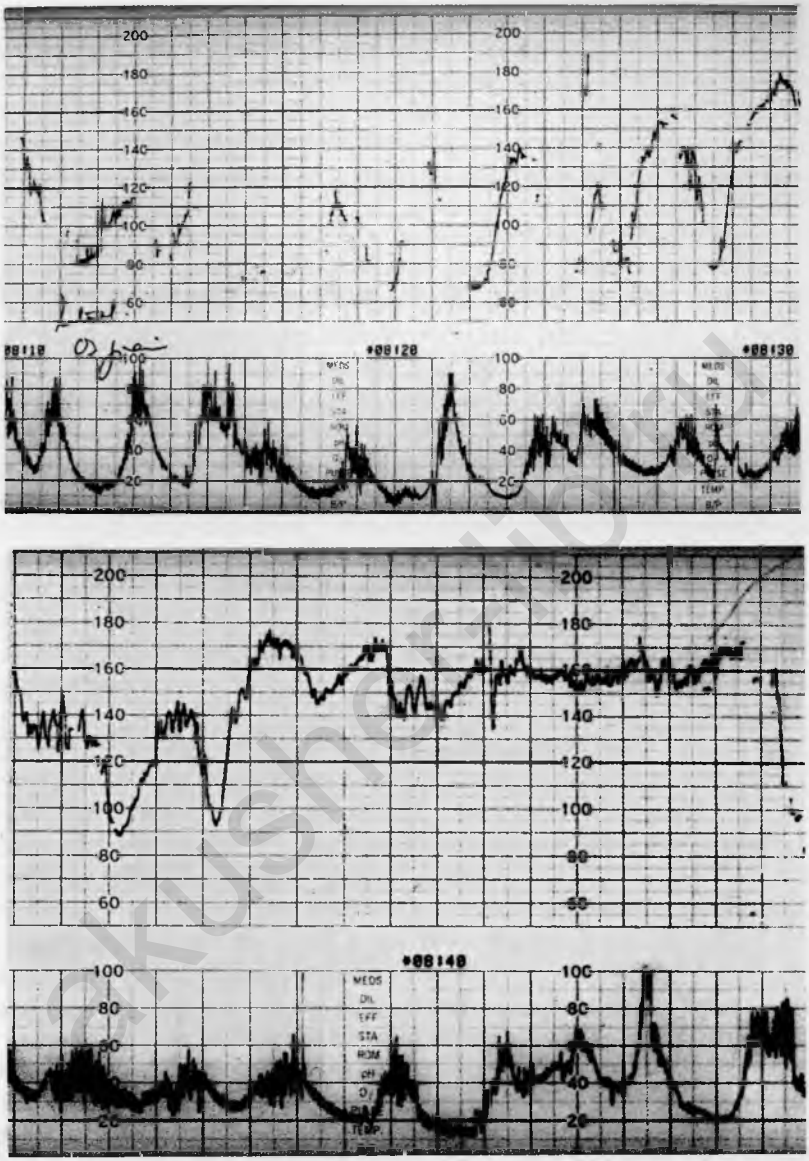

Figure 10-5. (A) Hyperstimulation and abnormal trace, followed by (B) correction of trace after cessation of oxytocin.

If there is no progress then caesarean section may be appropriate. If there is adequate progress a repeat pH can be performed. The rate of decline of pH related to the rate of progress of cervical dilatation can be deduced and a decision made to allow progress if the pH is unlikely

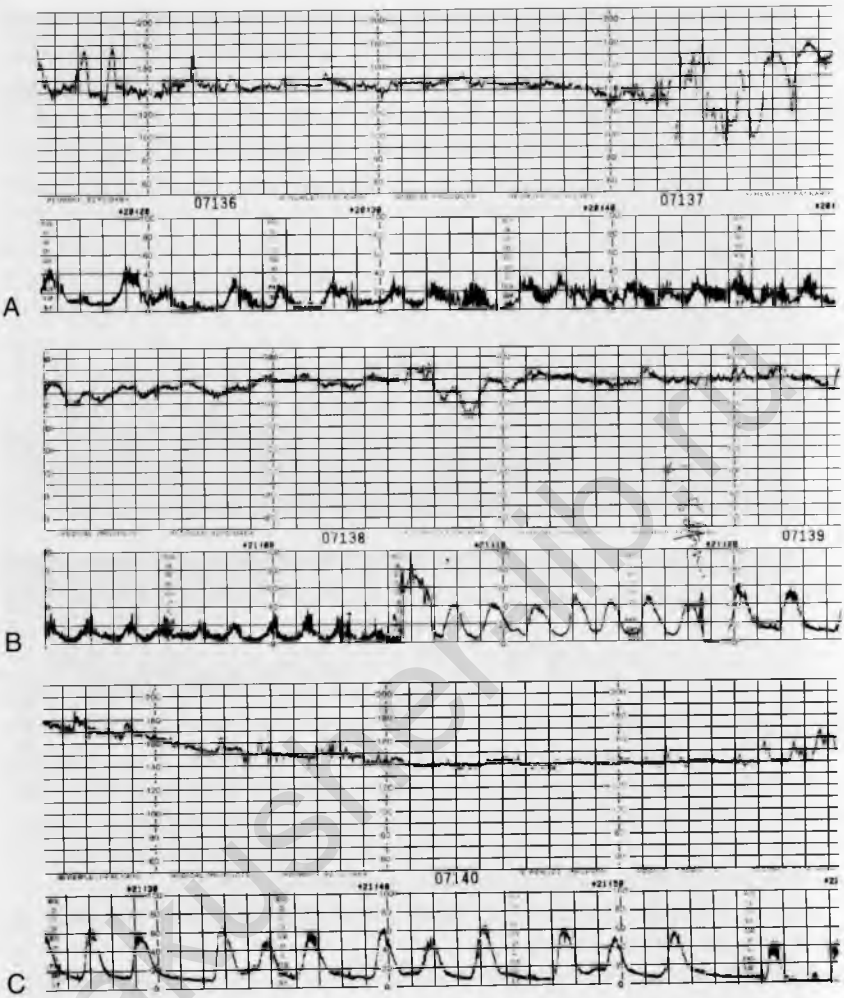

Figure 10-6. (A) Increased frequency of contractions: changes in trace; (B) sustained tachycardia; (C) reversion to normal after reduction of oxytocin.

to be acidotic by the time of anticipated delivery. Obviously the plans need to be changed if there is a worsening FHR pattern.

With experience and confidence in interpretation of CTG, performance of FBS may not be necessary. One should note the cervical dilatation at the time of commencement of oxytocin. Continuous CTG monitoring helps to observe FHR changes. If there are decelerations and tachycardia and if there is reduction in baseline variability, or if the decelerations are lasting longer than the duration of the FHR at the baseline in between decelerations, then oxytocin

infusion should be stopped and cervical dilatation assessed. If there is acceptable progress, one could wait for the FHR pattern to return to near normal (back to the preoxytocin baseline rate) with more time at the baseline in between decelerations, then the oxytocin infusion could be restarted and progress assessed in a couple of hours or if the CTG becomes abnormal. If there is no or very slow progress at the time abnormal CTG was observed, one should consider stopping the oxytocin infusion and progressing to a caesarean section or resorting to FBS if one still wants to continue with oxytocin infusion. The stopping and starting of oxytocin may cause the labour to be a little longer but the baby should be born in good condition.

In induced labour, in the absence of disproportion, the uterus has to perform a certain amount of uterine activity depending on the parity and cervical score to achieve vaginal delivery. Considering this, it may be possible to achieve optimal uterine activity that does not cause FHR changes but is adequate to bring about slow but progressive cervical dilatation.[8] The labour may be a little longer, during which time adequate contractions are generated to achieve vaginal delivery. However, such management needs intrauterine catheters and equipment to compute uterine activity, and it may not be possible to achieve optimal uterine activity without FHR changes and achieve vaginal delivery.

MEDICO-LEGAL CONSIDERATIONS

Oláh and Steer have recently reviewed the use and abuse of oxytocin.[2] They highlight that, although recognizing the appropriate use of oxytocin may be helpful, its abuse is implicated in many cases with adverse outcome and medico-legal sequelae.

The major concerns with oxytocin and medico-legal issues relate to the following:

- inadequate uterine contraction monitoring
- poor technical quality of the FHR trace
- cessation of monitoring the FHR or uterine contractions much earlier than the time of delivery
- commencement of oxytocin when there are major risk factors, e.g., thick meconium-stained scanty fluid, evidence of chorioamnionitis and a suspicious or abnormal FHR trace
- failure to recognize that the uterus is contracting >5 in 10 min despite no increase in oxytocin infusion and failure to reduce or stop the oxytocin infusion, thereby causing a pathological FHR pattern such as prolonged decelerations and fetal compromise
- failure to use tocolytics in some cases to alleviate the problem early, as time is needed for oxytocin-induced contractions to reduce/abate

- failure to recognize that prolonged decelerations following a normal FHR trace may recover, but the trace may not recover despite stopping oxytocin if the FHR prior to the decelerations was suspicious or abnormal
- when prolonged decelerations occur the fetal monitor may record the 'maternal heart rate', which may not be recognized by the caregiver
- the fetus may be affected with hypoxia despite prompt action (e.g., delivery), but the caregiver may be liable if the prolonged FHR decelerations were caused by uterine hyperstimulation
- oxytocin should be used with caution when there are FHR changes as it may make things worse; careful consideration should be given to deliver rather than to augment or induce labour
- in the presence of thick meconium and scanty fluid, meconium aspiration syndrome is a possibility with late or atypical decelerations suggestive of hypoxia even without acidosis
- decelerations in early labour, or prolonged decelerations with the use of oxytocin may imply impending scar rupture with oxytocin in a woman with a previous scar
- if the FHR shows what appears to be accelerations, they may be decelerations if the baseline rate does not settle and show an 'active and quiet sleep cyclicity' pattern with continuation of the recording for 2 hours.

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MECONIUM, INFECTION, ANAEMIA AND BLEEDING

Leonie Penna

INCREASING FETAL RISK

This chapter will review some specific clinical circumstances that increase the risk that the fetus will develop intrauterine hypoxia with a risk of adverse neonatal outcome. This association means that extra vigilance is needed in observing any abnormalities seen on the cardiotoccograph (CTG) and that a change in threshold for recommending intervention is sometimes needed.

MECONIUM

The passage of meconium is common resulting in meconium-stained amniotic fluid (MSAF) in about 10% (range 7–22%) of term deliveries.[1] Most meconium is physiological and is due to spontaneous peristaltic contraction of the bowel, which may occur at fetal maturity without an adverse event as part of normal fetal behaviour. The incidence of MSAF at 42 weeks is about 30% and this falls as gestational age and thus physiological maturity reduces.[1] In preterm gestations, meconium is uncommon (<5% at 34 weeks).[2] There is also an increased incidence of MSAF reported in pregnancies complicated by obstetric cholestasis, gastroschisis and fetal bowel pathologies.[3]

Why is Meconium Important to Neonatal Outcome?

- Meconium is an independent risk factor for poor neonatal outcome, with an increased risk of cerebral palsy in neonates where meconium was observed during labour.[4]
- Meconium passage can occur as a result of hypoxic stress; reduction in oxygen results in an adrenergic response with redistribution of blood to essential organs and reduction in blood supply to non-essential organs including the bowel. Peristaltic bowel contractions and relaxation of the anal sphincter are adrenergic responses that can result in the passage of meconium.
- Meconium is acidic and if it enters the fetal lungs it causes a chemical pneumonitis and respiratory distress in a condition called meconium aspiration syndrome (MAS). Pulmonary hypertension can occur as a complication of this condition.[5]

- Meconium can enter the fetal lungs if the fetus makes reflex gasping movements, which are known to occur in response to sudden acute hypoxia or as an end stage in slow-developing subacute hypoxia.[6]
- Overall about about 5% of infants born following a labour complicated by MSAF will develop MAS. Physiological meconium can result in MAS if a hypoxic event occurs causing the fetus to gasp, but the risk is greater with thicker meconium. MAS is a serious condition causing significant morbidity and mortality (accounting for about 2% of all perinatal mortality in the UK in 2007).[7]
- Thick meconium is known to increase the risk of cord spasm in utero with resultant increased risk of hypoxaemia and hypoxia.[8]
- Meconium inhibits the phagocytic activity of macrophages in amniotic fluid and may enhance bacterial growth, increasing the risk that intrauterine infection will occur. It has been suggested that the fetal systemic inflammation that occurs in chorioamnionitis may be an important factor in why fetuses develop MAS.[9]

Meconium and Fetal Monitoring

Although most meconium is physiological, its association with adverse neonatal outcome means that it must be considered as a potential complication of labour, with a review of risk factors in the pregnancy and labour. The relationship of meconium to fetal infection is important and often overlooked and this should be considered if the fetal heart pattern becomes abnormal.

Historically meconium has been graded:

- grade 1 – light meconium with good volumes of amniotic fluid (AF)
- grade 2 – heavier meconium staining but still with a good volume of AF
- grade 3– very heavy meconium with reduced AF.

As these descriptions are highly subjective, more recent classifications suggest the use of just two categories:[10]

LIGHT = meconium contamination where there is a large volumes of AF.

- Light meconium will usually have occurred some time prior to rupture of membranes and be present at the time of spontaneous rupture of membranes or of amniotomy. Light meconium is likely to be physiological but the possibility of hypoxaemic stress and of infection should still be considered in all cases.
- If the woman is low risk for the development of infection or hypoxia, then intermittent auscultation (IA) can be recommended. In women planning birth outside an obstetric unit

(home birth or midwifery-led unit), this plan should be carefully scrutinized with consideration of transfer to the obstetric unit.[10]

- In a woman with risk factors or where the clinic circumstances suggest it is less likely to be physiological (gestations below 38 weeks), CTG monitoring should be recommended.[10]
- If IA suggests any possible fetal heart rate abnormality, including a rising baseline rate (even if within normal limits), then conversion to CTG monitoring should be recommended.[10]

HEAVY = meconium contamination where there is reduced AF resulting in the meconium being much more concentrated.

- Although heavy meconium may be present at the time of rupture of membranes, it more commonly develops during labour. Heavy meconium, in small volumes of fluid, is much less likely to be physiological and has a much higher association with MAS,[11] and thus careful review is needed of all maternal risk factors.
- CTG monitoring should be recommended in all cases.[7]

Other Management Decisions

In cases of prelabour rupture of membranes (>34 weeks), meconium is an indication for recommending immediate induction of labour. This is as much because of the increased risk of infection associated with meconium as the more commonly cited concerns about fetal wellbeing.[12]

If the CTG trace is normal (see Ch. 6) then no additional action needs to be taken even in the presence of very thick meconium. Although meconium is a cofactor for the development of infection, there is no good evidence that the administration of antibiotic to otherwise-asymptomatic women improves fetal outcome and so is not recommended[13] – although the most recent Cochrane review on the subject concluded that antibiotics may reduce chorioamnionitis and recommended further research in this area.[14] Likewise studies of amnioinfusion in labours complicated by thick meconium have not shown any benefits for the neonate and so this is not recommended.[15]

If the CTG trace becomes suspicious then the possibility of hypoxaemic stress must be considered. The decisions about management must be individualized but will depend on parity, stage and progress in labour and the wishes of the parents according to their view of the risks involved. As MSAF is associated with a 1 in 20 risk of MAS, it is essential that women and their partners are included in decision making where options for management exist. The normal intrauterine resuscitation measures of maternal repositioning, intravenous (IV) fluids, reduction in oxytocin dose and IV antibiotics (if infection is suspected) should be undertaken without delay.

If the CTG trace becomes pathological, the risk of fetal gasping and MAS increases.[11] This is particularly so if prolonged decelerations occur. The combination of thick meconium, clinical signs of infection and a pathological CTG is particularly ominous and immediate delivery is indicated.

There is a common perception that fetal blood sampling (FBS) should not be performed in the presence of heavy meconium and a pathological trace because there is a high risk that fetal hypoxia is developing and thus a fear of MAS. The threshold for FBS is definitely altered, with delivery preferable in many cases. However, not to do FBS in all circumstances incurs maternal risk related to emergency caesarean section and so decisions should be individualized. FBS for a pathological trace with meconium prior to potentially difficult instrumental delivery and in women with a high expectation of vaginal delivery in the near future are examples of situations where FBS may be the appropriate management.

Not all fetuses with subacute hypoxia will pass meconium, and fetuses that experience a sudden severe hypoxic event such as uterine rupture may also not pass meconium. Also, even if present, new meconium may not be seen with a deeply engaged head as occurs in the second stage of labour. Therefore, the absence of MSL in the presence of an abnormal fetal heart rate pattern should not be considered as reassuring.

Clear amniotic fluid is reassuring. Thick, fresh meconium in a situation of high risk is of great concern.

An attempt should be made in all cases with a fetal heart trace not classified as normal to release amniotic fluid from above the presenting part if necessary; this is done by pushing the presenting part gently upwards. If no fluid appears then the possibility of oligohydramnios and potential fetal compromise must be considered.

INFECTION

Maternal infection is common during labour, with 1–4% of labours complicated by chorioamnionitis.[16] Infection is an important cofactor in the development of hypoxia as evidence shows that infection increases the risk of poor neonatal outcome.

Why is Infection Important to Neonatal Outcome?

- Clinical (and subclinical based on histology of the placenta) chorioamnionitis during labour is an independent risk factor for poor neonatal outcomes including cerebral palsy (CP).[17] Microbial toxins or cytokines released during maternal infection can cause 'fetal inflammatory response syndrome' (FIRS) with fetal cytokine production. FIRS has been implicated as a cause of cystic periventricular leucomalacia and CP without evidence of direct infection in the fetus.[18]

- Clinical chorioamnionitis can result in a neonatal infection with pneumonia, meningitis or generalized sepsis. Fetal infection in utero may cause fetal tachycardia (the mother having a normal pulse), with a resultant increase in basal metabolic rate (BMR) with increased oxygen/energy requirements for normal functions and the risk of a more rapid decompensation than would occur in the non-infected fetus if hypoxia developed.
- Maternal infection causes a shift of the oxygen dissociation curve to the right due to hyperthermia reducing oxygen delivery to the fetus, which results in greater hypoxaemia and a higher risk of developing hypoxia.[19]

Maternal pyrexia from any cause will increase the fetal temperature owing to a reduction in passive heat loss. Maternal pyrexia can cause concomitant fetal tachycardia. Even in the absence of fetal infection, pyrexia and tachycardia will increase both the fetal BMR and the fetal energy requirements (in adults an increase in BMR of up to 13% per degree rise in temperature[20]). This increases the chances that the hypoxaemia occurring from intermittent cord compression is more likely to result in the development of hypoxia. This can occur in the healthy fetus, but there is an even greater risk for a fetus already compensating for a stress such as placental insufficiency.

Infection and Fetal Monitoring

The fact that fetal neurological injury may occur secondary to infection, and that infection per se may reduce the threshold for hypoxia and for hypoxic brain injury, means that CTG monitoring could avoid superimposing intrapartum hypoxia on intrauterine infection. For these reasons, continuous electronic fetal monitoring is recommended in any labour where there is a significant risk of infection.[10,21]

Although this is a standard recommendation, the effect of chorioamnionitis on fetal heart rate patterns is uncertain as no pattern specific to chorioamnionitis alone has ever been identified. The most common finding is fetal tachycardia due to fetal sepsis or as a response to pyrogens crossing the placenta from the mother. Reduced variability and variable-type decelerations have been reported in a number of small case series but have not been proven to have any specific association with infection.[22]

Term fetuses with both intrauterine infection and non-reassuring FHR patterns are at higher risk of developing CP than are fetuses with only one risk factor,[23] suggesting that if evidence of hypoxia develops in the presence of infection then swift intervention is required.

There is often concern that the use of FBS in the presence of infection carries a risk of inoculating the fetus with infection, that capillary stasis from sepsis could give erroneous results and that the association of

infection with cerebral palsy means that any suspicion of hypoxia requires immediate delivery, which are cited as reasons for not doing it. However, there is no evidence to confirm these concerns, which remain theoretical.

Adopting a 'no FBS' policy in suspected chorioamnionitis will result in unnecessary caesareans, both in women who do not actually have infection and in those with infection where the CTG has been falsely suggestive of hypoxia, and there is no evidence that caesarean section will alter the outcome for the neonate but it may be associated with increased infective morbidity in the mother.[24]

Fetuses with infection who develop hypoxaemia will potentially deteriorate more rapidly, developing severe hypoxia and asphyxia; therefore in deciding whether to undertake FBS the presence of other risk factors for the development of hypoxia, slow progress or the development of fetal heart abnormalities in early labour (especially in a primigravida) mitigate against performing this test. If there are no other risk factors for hypoxia and labour is established and progressing well then prompt FBS can be considered. Evidence suggests that FBS is not a quick procedure[25] and in the presence of infection it should be undertaken by the most experience clinician available and abandoned (with recourse to caesarean) if a sample is not obtained in a timely manner. If the CTG abnormality persists after a normal FBS result, an early repeat test (30 min or less) should be undertaken owing to the risk of more rapid deterioration in chorioamnionitis, and caesarean section is recommended if there has been any significant deterioration in the pH or base excess.

Other Management Decisions

Other infections can cause direct effects on the fetal heart with resultant changes in the CTG. These include infections by organisms such as cytomeglovirus (CMV) and *Listeria*;[26] no specific pattern has been described but, for an abnormal CTG with no other obvious explanation, these diagnoses should be considered especially if a history of recent non-specific febrile illness or other risk factors for infection is elicited. Apparently unprovoked reduced variability, decelerations and a tachycardia without other explanation may be seen on fetal monitoring.

In severe maternal sepsis, a maternal metabolic acidosis may develop as part of the disease process. Even in the absence of fetal infection or maternal hypotension, an uncorrected maternal acidosis will result in a slowly developing fetal acidosis due to the inability of the placenta to clear hydrogen ions and lactate.[27] The trace may show reduction in variability and unprovoked decelerations without the development of tachycardia (Fig. 11-1). Correction of the maternal condition may reverse the fetal condition, but this needs to be done as quickly as possible to avoid the risk of long-term neurological damage due to lactic acidosis. At viable gestations where the maternal condition is considered sufficiently stable, delivery by emergency caesarean section should be considered (Fig. 11-2).

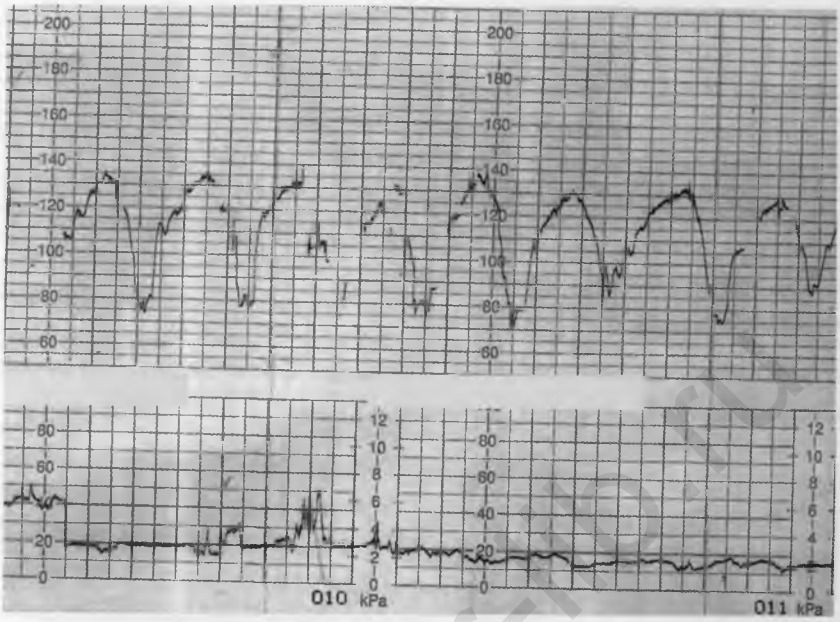

Figure 11-1. 37 weeks: severe maternal metabolic acidosis secondary to peritonitis from a ruptured appendix. Prompt emergency CS (and appendicectomy) with good maternal and neonatal outcome.

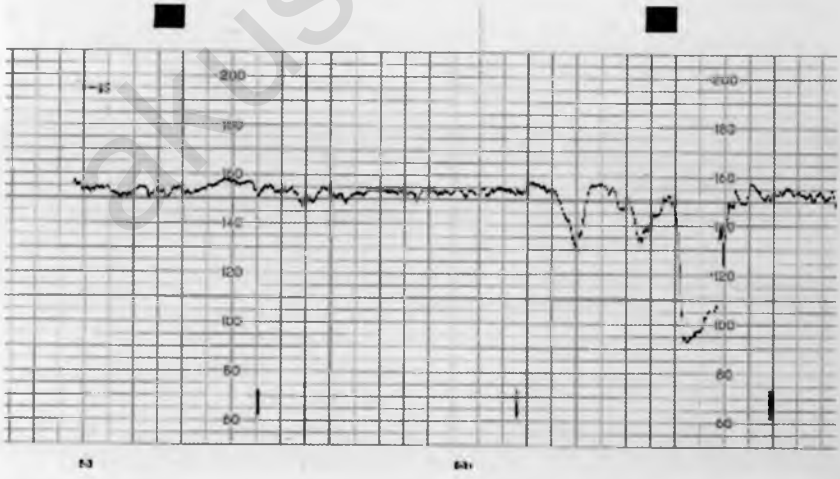

Figure 11-2. 34 weeks: recent travel to the USA presented with reduced fetal movements. Ultrasound showed mild ascites. Delivery was by CS with poor condition at birth. Investigations confirmed fetal/neonatal *Listeria* infection. There was normal neurodevelopment at age 5 years.

FETAL ANAEMIA

There are many reasons why a fetus can develop anaemia in pregnancy (Table 11-1) but all are rare.

Occasionally the risk can be anticipated from the maternal history, but in the majority anaemia occurs in an unheralded fashion either because risk factors have gone undetected or because the anaemia occurs from an unpredictable event. As untreated fetal anaemia can result in fetal death or survival with neurological damage it is essential that all clinicians are familiar with fetal heart rate patterns that may indicate anaemia.

Why is Anaemia Important to Neonatal Outcome?

- Anaemia reduces the oxygen-carrying capacity of the fetus blood, making hypoxaemia more likely.
- Haemoglobin and plasma bicarbonate are the major buffers utilized by the fetus to neutralize hydrogen irons and maintain extracellular pH within a critical range, avoiding effects in the CNS and cardiovascular system. Any reduction in haemoglobin will reduce the fetal ability to withstand even short periods of anaerobic metabolism and this worsens with the degree of anaemia.
- A fetus with chronic anaemia compensates for the low haemoglobin by a hyperdynamic circulation, but as the anaemia progresses this will result in cardiac failure and fetal hydrops.[28]
- A fetus with sudden blood loss is in 'double jeopardy': firstly of becoming hypovolaemic due to loss of circulating blood volume and secondly of losing buffering ability that allows the fetus to withstand minor hypoxic events.

Table 11-1 Causes of Fetal Anaemia

ACUTE ANAEMIA (HYPOVOLAEMIC)	CHRONIC ANAEMIA (NORMOVOLAEMIC)
Any acute fetomaternal haemorrhage, e.g., placental abruption, abdominal trauma	Fetal infections, e.g., CMV, parvovirus
Bleeding from vasa praevia	Alloimmune haemolytic anaemia, e.g., rhesus or other red cell antibodies
Transplacental delivery (CS) for placenta praevia	Genetic syndromes, e.g., Blackfan–Diamond anaemia, aneuploidy
Acute TTTS in monochorionic twins	Chronic TTTS in monochorionic twins

CMV = cytomegalovirus; CS = caesarean section; TTTS = twin-to-twin transfusion syndrome.

- Fetal heart rate changes occur only when severe anaemia is present and thus prompt action is required following a suspicion of anaemia to ensure a good outcome.

Anaemia and Fetal Monitoring

A sinusoidal pattern of the fetal heart in severe fetal anaemia was first described in 1972 and is now accepted as pathognomonic of fetal anaemia[29] if a persistent feature on monitoring. The pathophysiology underlying the pattern remains enigmatic, but two factors are changes in the autonomic nervous activity secondary to hypoxia from the reduced oxygen-carrying capacity and baroreceptor-mediated changes due to hypovolaemia.

The sinusoidal pattern is not seen in fetuses with mild anaemia and occurs only when the haemoglobin is below 100 g/l.[30]

There are two distinct types of sinusoidal pattern; both exhibit reduced variability:

- *Typical sinusoidal* is the pattern associated with chronic anaemia (e.g., rhesus disease) where there is no reduction in the circulating blood volume but a low haemoglobin. In the absence of any additional hypoxic stress, the fetal heart rate will be in the normal range and will show frequent low-amplitude (5–10 bpm) oscillations at a frequency of 3–5 per minute (Fig. 11-3).
- *Atypical sinusoidal* is the pattern seen in an acute anaemia where the fetus is hypovolaemic and anaemic owing to loss of circulating

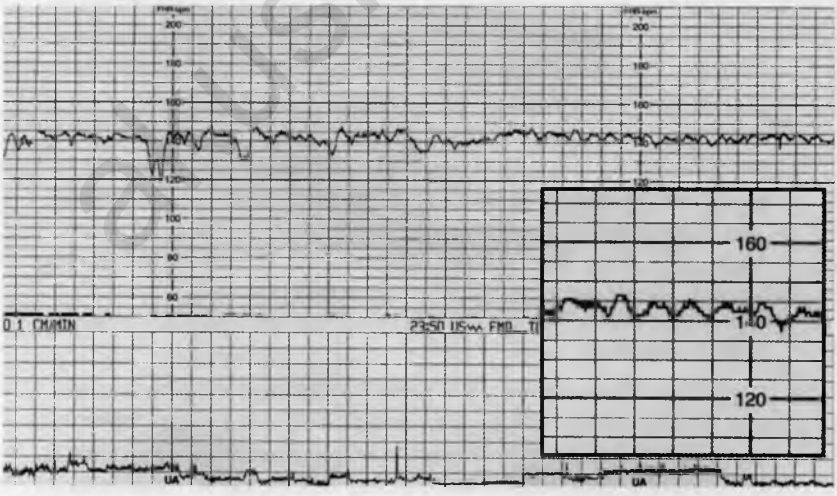

Figure 11-3. 32 weeks: admitted with reduced fetal movements. The trace shows a typical sinusoidal pattern in a fetus with parvovirus infection; note the enlarged portion of trace, which demonstrates the profound reduced variability with 'castle-wall' effect. Intrauterine transfusion was performed with good neonatal outcome.

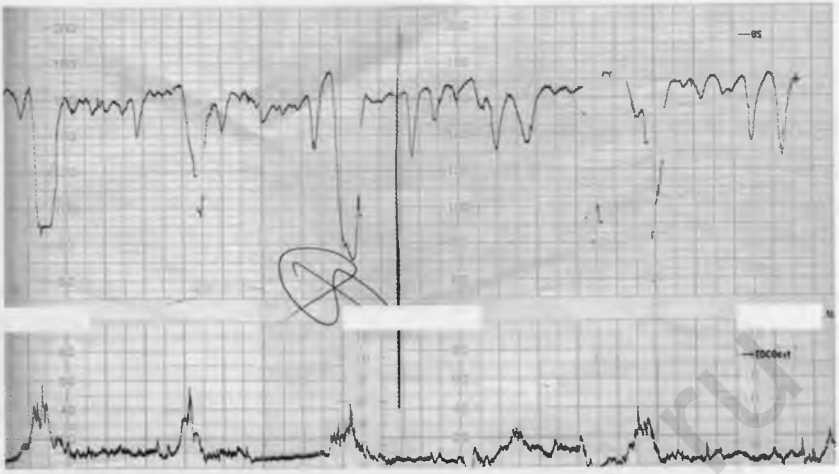

Figure 11-4. 39 weeks: admitted with contractions and light vaginal bleeding after spontaneous rupture of membranes. The trace shows an atypical sinusoidal pattern (note the raised baseline). The trace appearances were mistaken for infection until terminal decompensation. There was emergency CS delivery of the neonate in very poor condition and haemoglobin 30 g/l, and early neonatal death.

blood volume. The fetal heart rate will be tachycardic and show more frequent high-amplitude oscillations (15–20 bpm) with some similarity to saltatory pattern variability (Fig. 11-4).

Many CTG traces will show short periods of sinusoidal-type pattern but, as a true sinusoidal pattern doesn't self-correct because fetal anaemia is never transient (it takes time to recover even if the cause of the problem has resolved), these short periods interspersed with a normal trace are not significant and therefore not a cause for concern or intervention.

A pseudosinusoidal pattern describes a pattern that may be seen in non-anaemic fetuses; the pattern is not persistent and has been attributed to fetal thumb sucking and is a finding in the premature fetus. Maternal opiate use can also cause CTG changes that can be mistaken for a sinusoidal pattern.

Other Management Decisions

A fetus with significant anaemia is already coping with stress due to a reduced ability to carry oxygen and any additional stress reducing fetal gas transfer, such as normal contractions, may result in decompensation and rapid development of hypoxia. Therefore, the treatment of a persistent sinusoidal trace in labour is immediate delivery by caesarean section with availability of facilities for advanced neonatal resuscitation. Vasa praevia is a rare condition

Figure 11-5. Placenta from the fetus in Figure 11-4. Membranous cord insertion and bleeding fetal vessel in an undiagnosed vasa praevia were clearly visible on examination.

where unprotected fetal blood vessels are present in the membranes overlying the cervix, with the risk of disruption and haemorrhage when the membranes rupture.[31] Neonatal mortality rates of up to 60% are described, but prompt recognition of the significance of the atypical sinusoidal pattern associated with relatively small amounts of bleeding followed by urgent delivery will improve the outcome (Fig. 11-5).

Sinusoidal traces (usually typical pattern) may also be seen in women presenting with reduced fetal movements who are not in labour. In the absence of a history of bleeding an urgent fetal medicine opinion should be requested. Measurement of the middle cerebral artery (MCA) Doppler in the fetus will allow confirmation of fetal anaemia as the peak systolic velocity (PSV) will be elevated.[28] Steroids should be recommended in preterm gestations and a Kleihauer–Betke test performed to look for fetomaternal haemorrhage. Treatment should be individualized depending on the suspected cause.

MATERNAL BLEEDING

Vaginal bleeding during pregnancy is common (3–5% of pregnancies) and may occur antenatally or as an intrapartum complication.[32]

Why is Bleeding Important to Neonatal Outcome?

- Any antepartum haemorrhage (APH) is a risk to the wellbeing of the fetus as placental abruption is the cause of the bleeding in a significant number of cases.
- If abruption occurs there is separation of some part of the placental mass from the uterine wall; this may be small marginal-type abruption with no immediate fetal effect, or separation of a large area of the placenta resulting in severe fetal compromise. Separation of any part of the placenta reduces the placental area available for transfer of oxygen and nutrients to the fetus. Although the fetus is able to withstand reduction of placental area on a

chronic basis (as in fetal growth restriction due to infarction) without immediate effect, a loss of a similar volume very suddenly is likely to cause changes in the fetal heart rate pattern as the fetus tries to adapt to the new situation. The addition of a stress such as contractions (intrapartum abruption) may result in decompensation unless the fetal reserve is unusually large. A sudden large separation of an area of the placenta will result in rapid and profound hypoxia and fetal decompensation regardless of whether other stresses on fetal wellbeing are present.

- In the absence of other causes, even very small APHs must be considered as possible abruption and at the point of presentation should be considered as potentially unstable; this could be the beginning of an evolving large abruption or a process of recurrent smaller haemorrhages, each reducing the available placental reserve for the fetus.
- Abruption is more common in pregnancies complicated by poor placentation[33] and therefore is more likely to occur in a fetus with a degree of compensated chronic placental insufficiency. Decompensation may occur even though the maternal symptoms are seemingly trivial (light bleeding and no pain).
- The possibility that bleeding could be of fetal origin should be considered, especially if the onset of even a relatively small amount of bleeding is associated with the onset of fetal tachycardia. In abruption there is a risk that fetomaternal haemorrhage will occur (further increasing fetal risk).
  A Kleihauer–Betke test should be requested in all cases of suspected abruption[31] and should be undertaken as soon as possible after the acute presentation. The laboratory often needs to be reminded that the test is required for clinical reasons and even in Rhesus-positive women.

Bleeding and Fetal Monitoring

Continuous CTG and intravenous access should be recommended in all cases of significant intrapartum bleeding. In women presenting with APH antenatally, fetal monitoring should be commenced as soon as possible so as to confirm fetal wellbeing, as seemingly trivial revealed bleeding may be only part of a larger concealed bleed.

There are no specific fetal heart rate patterns that are pathognomonic of abruption. An acute massive separation of the placenta is one of the causes of a sudden-onset non-recovering fetal bradycardia – usually developing features of terminal pattern with loss of variability or a deep swinging saltatory variability. This requires urgent caesarean section and is a situation where the aim must be for delivery by 20 minutes to ensure a good fetal outcome;

however, as this situation carries significant maternal risk, the need for rapid delivery must be balanced against the need for effective maternal resuscitation before surgery.

An uncomplicated tachycardia may occur as a sign of fetal stress and should be treated with maternal fluid resuscitation (essential if there is concomitant maternal tachycardia or heavy vaginal bleeding).

Late decelerations may be precipitated by intrapartum abruption as this can reduce the available placental reserve.

Persistent reduced variability in the trace in the fetus of a mother with APH should be considered as indicating impending decompensation.

If CTG abnormalities occur, then fluid resuscitation of the mother should be commenced and consideration given to delivery by urgent caesarean section (at viable gestations).

The combination of a developing fetal anaemia and compromise due to reduction in placental reserve can give rise to unusual patterns in the CTG, and so it is important that the clinician is very cautious about any CTG that is not classifiable by standard guidelines (Fig. 11-6).

Evaluation of uterine activity as shown on by the toccograph can reveal evidence of uterine irritability, which may indicate a more significant 'concealed' abruption with tracking of blood into the myometrium causing recurrent low-amplitude contractions and an increase in resting uterine tone (Fig. 11-7). These may not be palpable

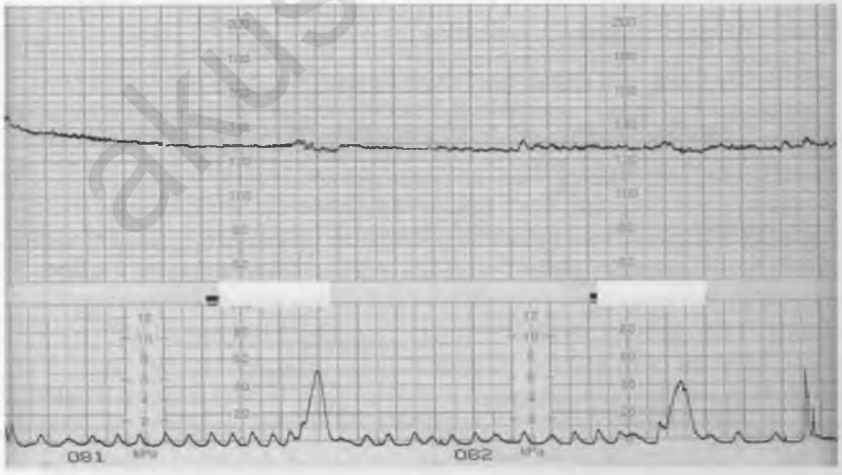

Figure 11-6. 36 weeks: admitted with significant APH in a pregnancy being monitored for pre eclampsia. Note the reduction in variability and the contraction pattern. The cervix was very unfavourable and a decision for emergency CS was made. A large retroplacental clot was present at delivery. There was a good neonatal outcome.

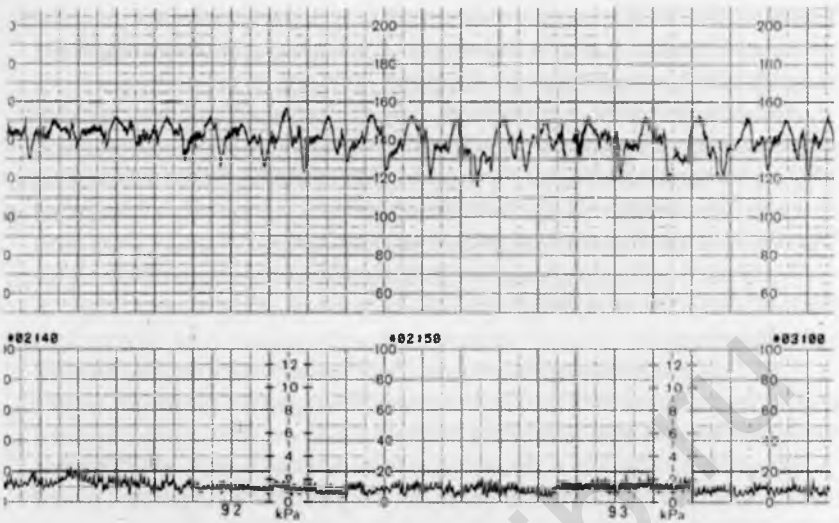

Figure 11-7. 38 weeks: admitted with a small vaginal bleed, contractions and abdominal pain. Cervix was 3 cm dilated. Note the activity on the tocograph and the CTG pattern that is difficult to classify by any normal guideline; 60 minutes later a sudden bradycardia commenced, immediate CS was performed with delivery of a fresh stillborn infant. A large concealed abruption was present.

and the woman may not describe contractions, being more likely to describe constant pain with exacerbations. In such cases there is a risk of sudden fetal decompensation; the management plan for care should encompass this.

Other Management Decisions

Delivery by caesarean section should be considered in all cases of non-reassuring fetal heart rates in the presence of significant bleeding that could be due to abruption. As the rate of deterioration of the fetal condition can be rapid, FBS should not be considered.

Usually the bleeding from placenta praevia occurs following minor placental separation and therefore fetal heart rate abnormality in small bleeds is uncommon. However, in the event of a large haemorrhage, acute fetal hypoxia can occur as a result of significant separation and/or maternal hypotension. Prompt maternal fluid resuscitation is essential for both mother and fetus prior to urgent delivery by caesarean section as reversal of maternal hypotension will improve the fetal condition.[34]

Non-uterine causes of APH should not be associated with fetal compromise unless maternal hypotension occurs and thus assessment of the fetal heart is an essential clinical sign that should be recorded in relation to all vaginal bleeding.

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CARDIOTOCOGRAPHIC INTERPRETATION – ADDITIONAL CLINICAL SCENARIOS

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CHAPTER 12

TWIN PREGNANCY

Perinatal mortality in multiple pregnancy is considerably higher than in singleton pregnancy, and particular risks are present during labour and delivery. It is now known that this mortality is considerably increased for monochorionic twins compared with their dichorionic counterparts. The rare monoamniotic twins should be delivered by caesarean section because of the risk of cord accidents, particularly after the delivery of the first twin. There is an increasing tendency to deliver monochorionic, diamniotic twins by caesarean section because of the risk of acute fetomaternal transfusion. If this is not done then very careful electronic monitoring must be undertaken. Twins are generally smaller than singletons, with more pathological growth restriction. The second twin may be at greater risk of this and the ability to electronically monitor both twins continuously is therefore important. The latest generation of fetal monitors has been specially designed to perform this function. One twin can be monitored on direct electrode with the other on ultrasound, or both can be monitored using external ultrasound.

To have only one machine at a woman's bedside is a considerable advantage that should be fully exploited. The Huntleigh Sonicaid prints its own paper and therefore has the novel feature of a three-channel trace (Fig. 12-1). The Hewlett-Packard and Corometrics models have a technique of printing out both traces in the same channel but in different shades (Fig. 12-2). It is critical to follow the second twin with the ultrasound transducer; however, this may prove difficult, especially in an obese mother. Assisted delivery is performed for the same indications as in a singleton pregnancy. A senior resident doctor must supervise the delivery of the second twin and ensure continuous electronic fetal monitoring during the interval between deliveries. Such an approach permits a more measured, less anxious delivery process; however, this should not be used as a justification for undue prolongation of the interval.

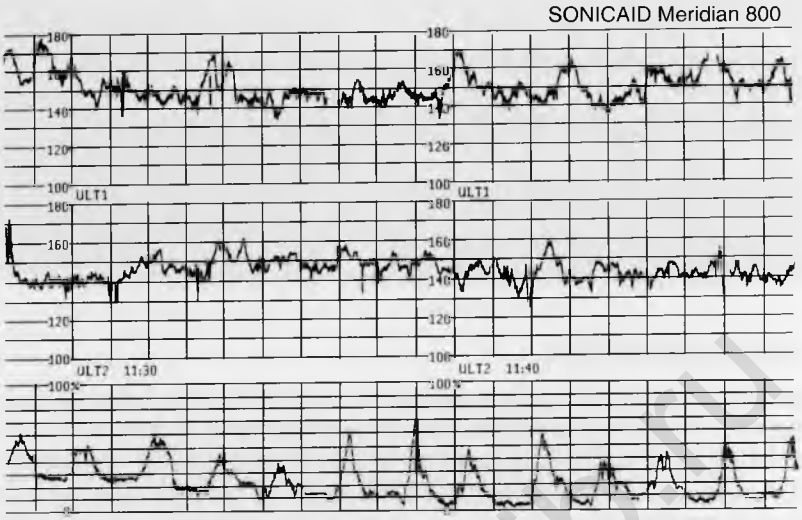

Figure 12-1. Monitoring twins – three-channel trace (Oxford Sonicaid Meridian). *(Courtesy of Huntleigh Healthcare Ltd.)*

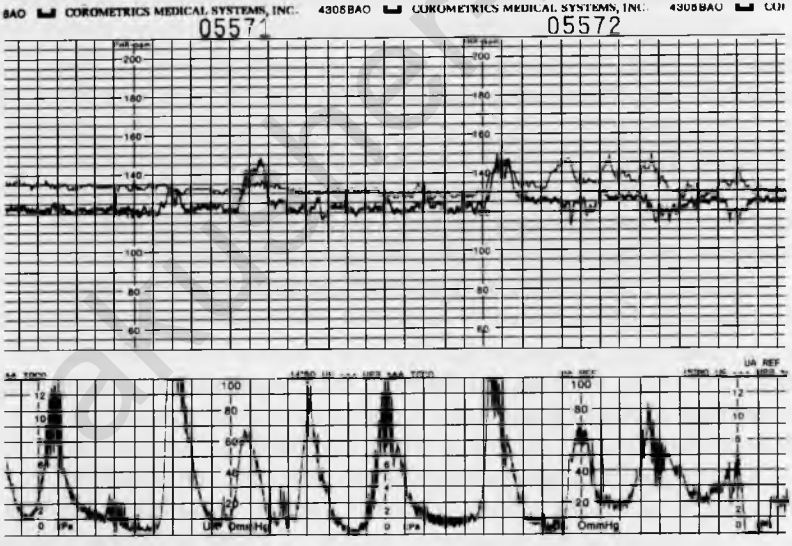

Figure 12-2. Monitoring twins – two-channel trace (Corometrics 116). *(Courtesy of GE Healthcare.)*

BREECH PRESENTATION

Babies presenting by the breech are acknowledged to be exposed to more risks than those presenting by the head. The Term Breech Trial Collaborative Group's study has resulted in most breech babies being

delivered by caesarean section.[1] This is unfortunate as women who like to deliver vaginally are now being denied the opportunity of vaginal breech birth and doctors in training no longer have the opportunity to acquire this skill, which will be necessary in an emergency delivery.

There are several risks, but intrauterine growth restriction (IUGR) and umbilical cord compression have particular implications for fetal monitoring. The footling or flexed breech has a greater chance of cord prolapse and compression of the umbilical cord in labour. This is a classical scenario for variable decelerations due to cord compression, as outlined in Chapter 5. This is one of the reasons why such cases usually have planned caesarean section. There is also evidence that compression of the skull above the orbits by the uterine fundus is a mechanism for variable decelerations. Figure 12-3 shows a typical pattern of cord compression in a breech. Should the misfortune of umbilical cord prolapse occur then the dramatic decelerative pattern shown in Figure 12-4 may be seen. The presence or absence of developing asphyxial features, such as changes in the baseline rate, baseline variability and magnitude of the decelerations related to the speed of the evolving labour process, will relate to the outcome. Breech presentation presents special risks, and in view of these there is little or no place for fetal blood sampling in a breech labour. The blood is more difficult to obtain from the tissues of the breech and it may be different from that obtained from scalp skin. Having understood the normal mechanisms of cardiotocograph (CTG) changes

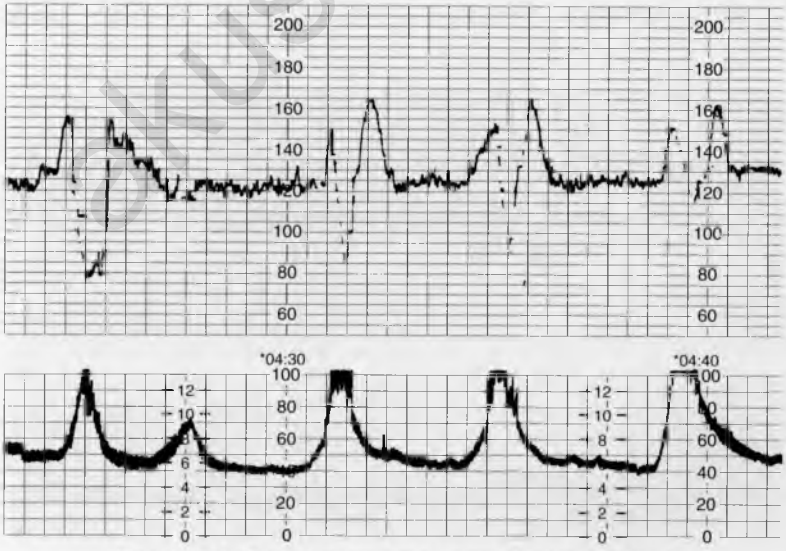

Figure 12-3. Breech presentation – variable decelerations.

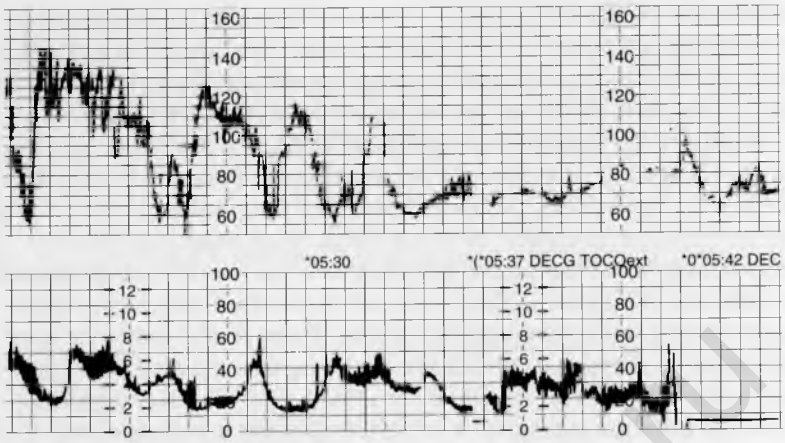

Figure 12-4. Breech cord prolapse.

in a breech, if there is a good indication for pH measurement then there is a good indication for caesarean section.

BROW PRESENTATION

Brow presentation in labour in late pregnancy is very unfavourable for vaginal delivery. The mentovertical diameter, which is usually about 13 cm, presents at the pelvic brim. This leads to head compression due to a mechanical misfit. Early and variable decelerations (Fig. 12-5) are associated with this.

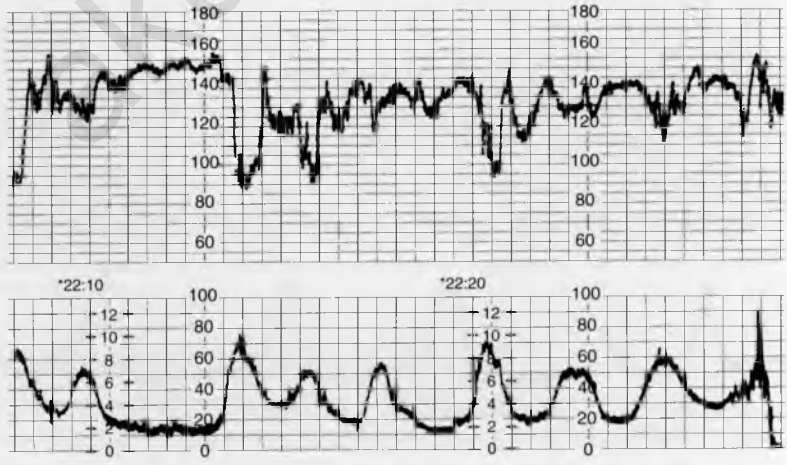

Figure 12-5. Brow presentation – decelerations.

There are no typical features associated with a face presentation. The placement of a fetal electrode should be avoided in a recognized face presentation.

PREVIOUS CAESAREAN SECTION: TRIAL OF LABOUR WITH A SCAR

The stability of the placental circulation and uteroplacental perfusion is dependent on the integrity of the uterus and vasculature. With the dehiscence or rupture of the scar, the major uterine blood vessels may become stretched and torn, compromising the perfusion of the placenta (see Fig. 13-4A and B). There is also the possibility of the umbilical cord prolapsing through the dehisced scar, giving rise to a dramatic cord compression pattern (see Fig. 14-1A–F). It is, therefore, believed that changes in the fetal heart rate (FHR) as a result of this may be one of the first signs of scar dehiscence. The other signs of scar dehiscence, such as scar pain, tenderness, vaginal bleeding or alterations in maternal haemodynamics, are notoriously late and unreliable. Figure 12-6 shows a trace from a woman having a trial of scar where, at laparotomy shortly after the trace, the scar was found to have ruptured. Figure 12-7 shows another trial of labour where emergency caesarean section was undertaken for prolonged bradycardia with a suspicion of scar dehiscence. The baby was delivered by immediate caesarean section (less than 15 min from the decision to delivery), and had Apgar scores of 4 at 1 min improving to 7 at 5 min, making a good recovery. There were no signs of placental abruption, scar dehiscence or any other explanation for the abnormal tracing. Figure 12-8 illustrates another case where the fetus was

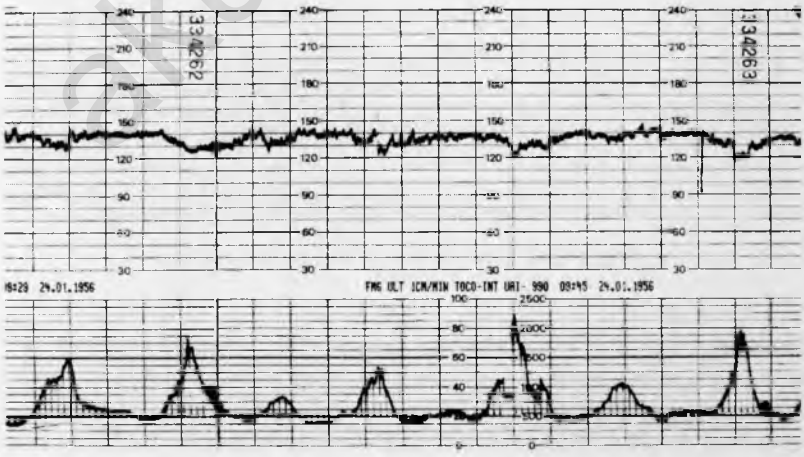

Figure 12-6. Scar rupture – trace with no alarming features.

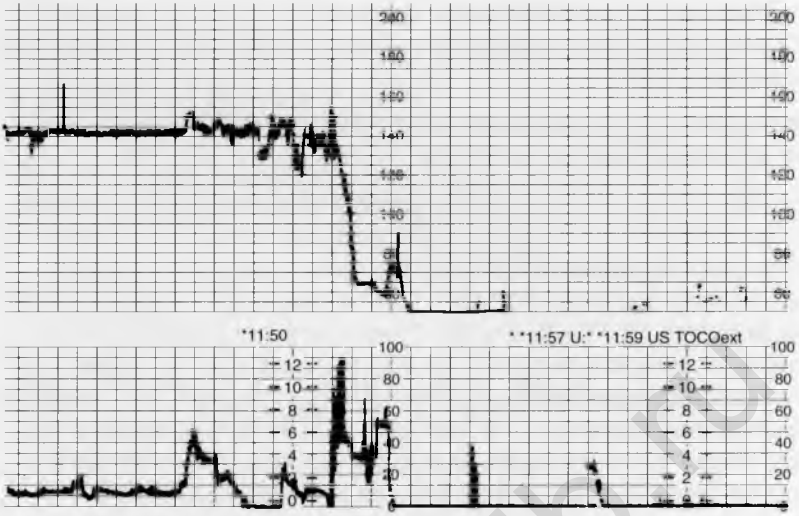

Figure 12-7. Prolonged bradycardia.

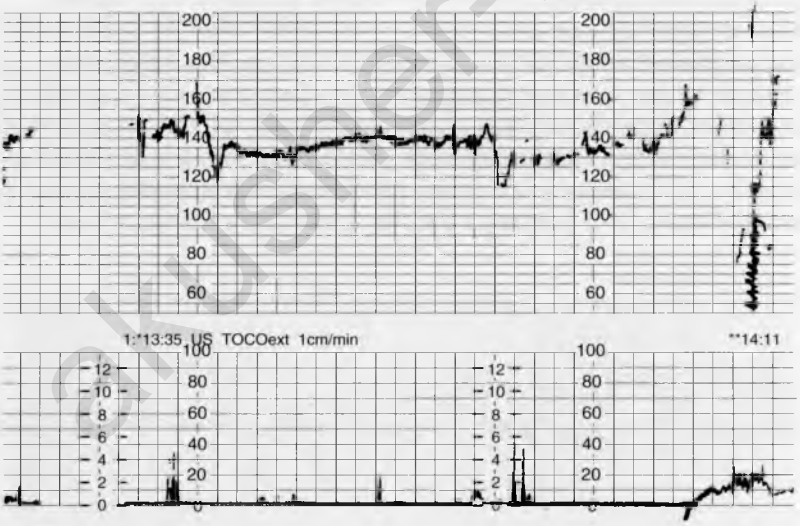

Figure 12-8. Scar rupture – relatively normal trace.

already passing into the peritoneal cavity with a relatively normal trace and subsequently good outcome. Presumably there was some maintenance of placental perfusion. Continuous electronic FHR monitoring in a trial labour with a scar may be helpful in the diagnosis of scar dehiscence, although this is variable.

SEVERE HYPERTENSION

Women suffering from severe hypertensive disease of pregnancy have at least two possible reasons for having an abnormal CTG. The first is the disease itself and its possible association with IUGR; the second is medication. Antihypertensive drugs, by their very nature, have effects on the maternal and fetal cardiovascular systems. Methyldopa leads to reduction in baseline variability and accelerations. Beta-blocking drugs result in reduced baseline variability and accelerations.[2] Figure 12-9 shows the trace of a fetus whose mother was being treated with labetalol for her hypertension; in spite of numerous fetal movements, accelerations are limited and baseline variability reduced. The picture is confounded by medication in these high-risk pregnancies, and complementary tests such as biophysical profile and Doppler studies are appropriate.

ECLAMPSIA

A convulsion represents a major stress to the fetus, which it may not survive. It is likely that such a fetus is already suffering from IUGR because of severe pre-eclampsia. Figure 12-10 shows a trace during an eclamptic fit. After any major acute stress it is important to check fetal condition by ultrasound scan or Doppler transducer of CTG before caesarean section.

The mother's condition must be stabilized before she faces the further challenge of caesarean delivery. If the fetal heart tracing is not of major concern after the convulsion then assessment and preparation for 1–2 h is reasonable. Undue haste may lead to maternal complications.

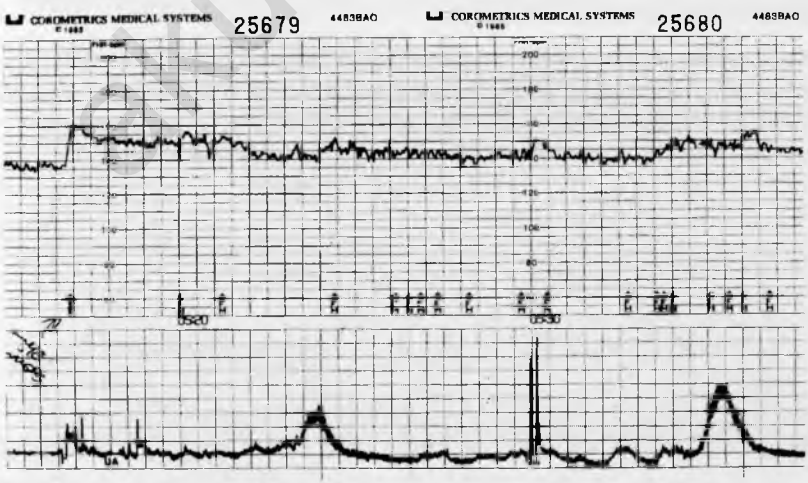

Figure 12-9. Hypertension treated with beta-blocker.

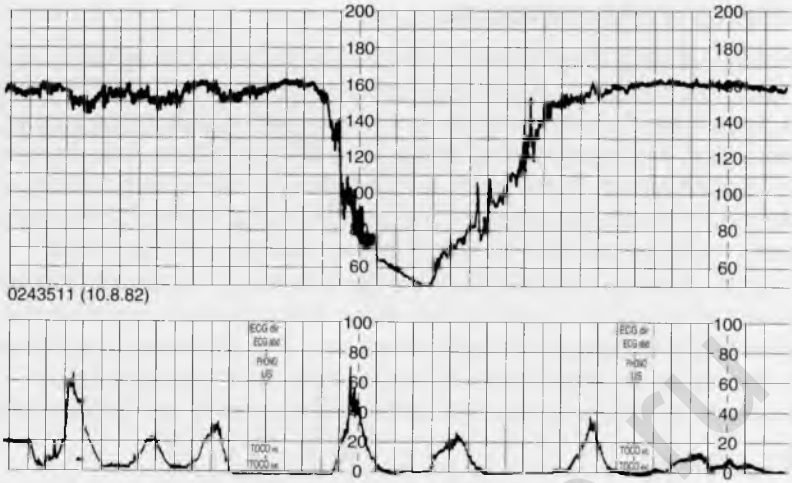

Figure 12-10. Deceleration – eclamptic fit.

MEDICATION

High-risk women may be on multiple drug therapy. Figure 12-11 shows a trace from a woman with a functioning transplanted kidney who had been prescribed azathioprine, ciclosporin, prednisolone, antibiotics and atenolol. The low baseline is remarkable. Other tests of fetal wellbeing were normal. The trace remained normal in induced labour and the baby was in excellent condition at birth.

A baseline rate below 100 beats per min (bpm) in a non-hypoxic fetus is exceptional.

EPIDURAL ANAESTHESIA

The insertion of an anaesthetic agent into the epidural space can be associated with a degree of instability of the maternal vascular system. Provided the preceding trace has been normal then this represents a stress that the fetus can withstand. After attention is paid to the

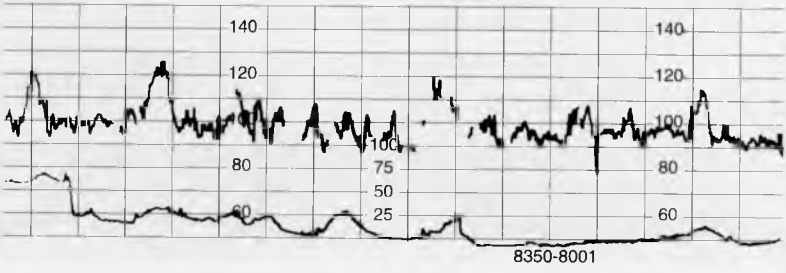

Figure 12-11. Unusual trace – multiple drug therapy.

circulating volume, and vascular stability returns, then the trace returns to normal. This is a form of stress test. However, if the preceding trace has not been normal then it is wise to apply a scalp electrode before the manipulation for insertion of the epidural to facilitate monitoring. If the preceding trace has been abnormal then a more ominous situation may develop. Figure 12-12A is a trace erroneously not recognized to be abnormal before the insertion of the epidural. The cervix was already 3 cm dilated and the trace should have prompted membrane rupture, which would have revealed thick meconium and facilitated the application of a scalp electrode. Unfortunately, the stress of epidural insertion resulted in serious asphyxial CTG changes (Fig. 12-12B) and the birth by immediate caesarean section of a compromised baby.

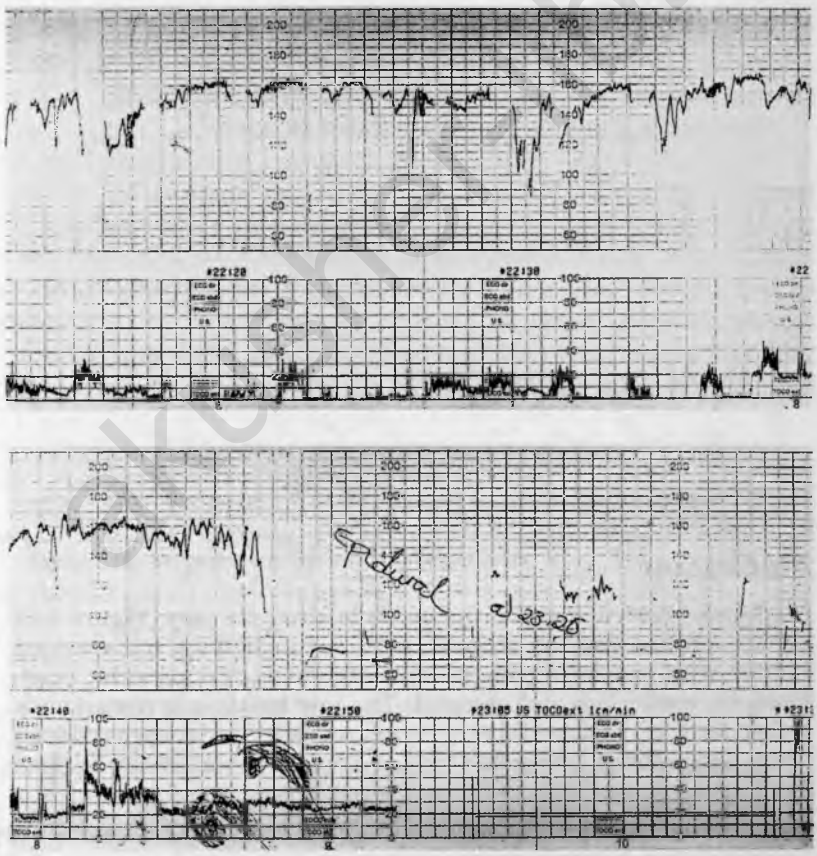

Figure 12-12. (A) Abnormal trace not recognized before insertion of epidural; (B) after epidural, grossly abnormal FHR pattern, leading to operative delivery.

SECOND STAGE OF LABOUR

The second stage is a time of very specific changes in the mechanical effects resulting from descent of the fetus. In a cephalic presentation the initial appearances result from head compression. It is commonly seen in a multiparous mother in good labour that the onset of progressive early decelerations is a sign of the second stage before it has been confirmed by vaginal examination or the appearance of the head at the perineum.

Decelerations are common in the second stage.

Early decelerations gradually becoming deeper and developing variable features are characteristic of the second stage of labour. Reassurance is provided by a good recovery from each deceleration and a return to normal rate and normal variability, however short, before the next contraction (Fig. 12-13). Under these circumstances, assisted delivery is not necessary except for other reasons relating to maternal condition. Signs of hypoxia are gradual tachycardia, reduced baseline variability in between and during decelerations (Fig. 12-14), additional late decelerations (Fig. 12-15) and failure of FHR to return to the baseline rate after decelerations (Figs 12-16 and 12-17).[3]

Prolonged bradycardia necessitates delivery.

Failure of the FHR to return to the baseline, and especially failure to recover to at least 100 bpm, is a serious sign and delivery should be undertaken. Figure 12-16 is an example where the doctor was called within 3 min of a bradycardia. At that point the fetal heart then recovered. There was a further bradycardia of 3 min, which did not then recover. At 6 min the mother was prepared, at 9 min the forceps were prepared and at 12 min the forceps delivery was performed with the baby born in good condition.

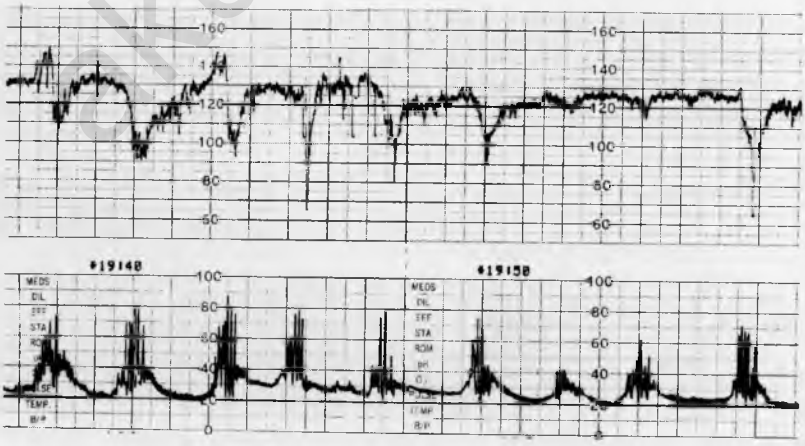

Figure 12-13. Normal second stage FHR trace – variable decelerations.

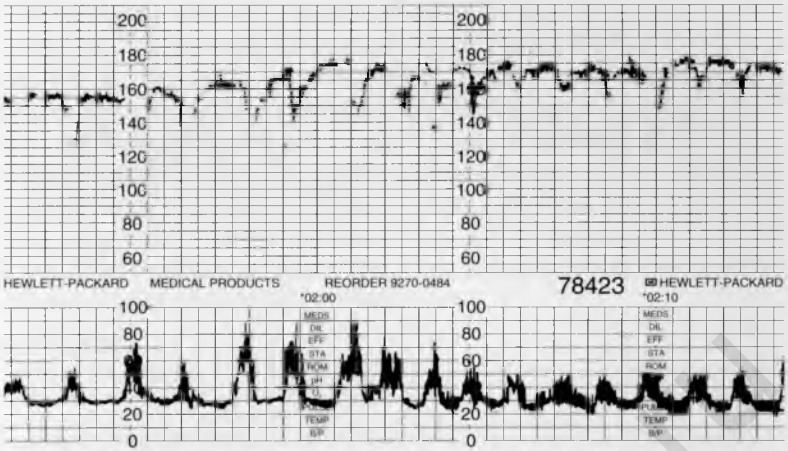

Figure 12-14. Abnormal second stage FHR trace – developing tachycardia.

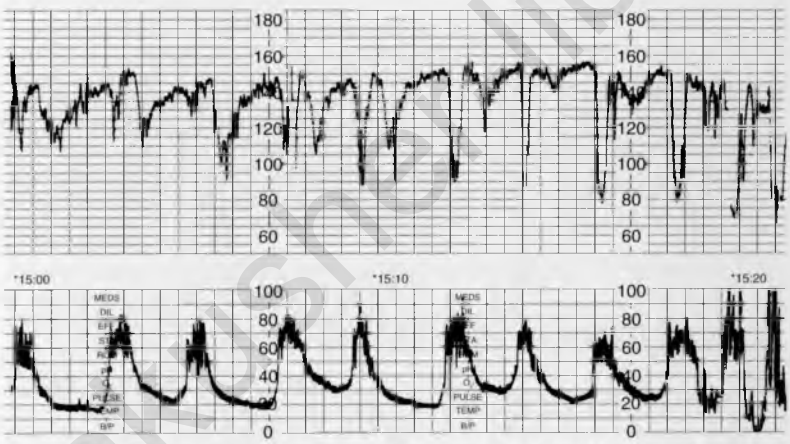

Figure 12-15. Abnormal second stage FHR trace – additional late decelerations.

THE 3, 6, 9 AND 12 MIN RULE

- 3 min: call the doctor
- 6 min: prepare the mother
- 9 min: prepare the forceps
- 12 min: deliver the baby

A delay of 20 min or more may result in an asphyxiated baby.

With a head on or near the perineum one should try to achieve an early delivery. There is no need for washing, gowning, draping and catheterization. A pair of gloves and an instrument such as a Kiwi cup are sufficient. Time is of the essence.

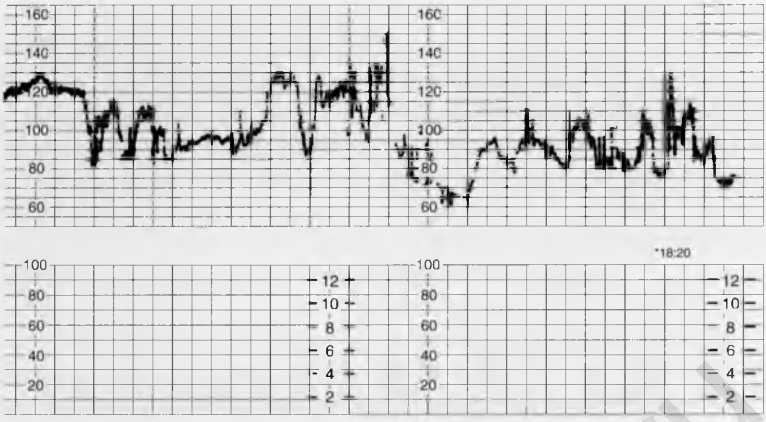

Figure 12-16. Abnormal second stage FHR trace – prolonged bradycardia.

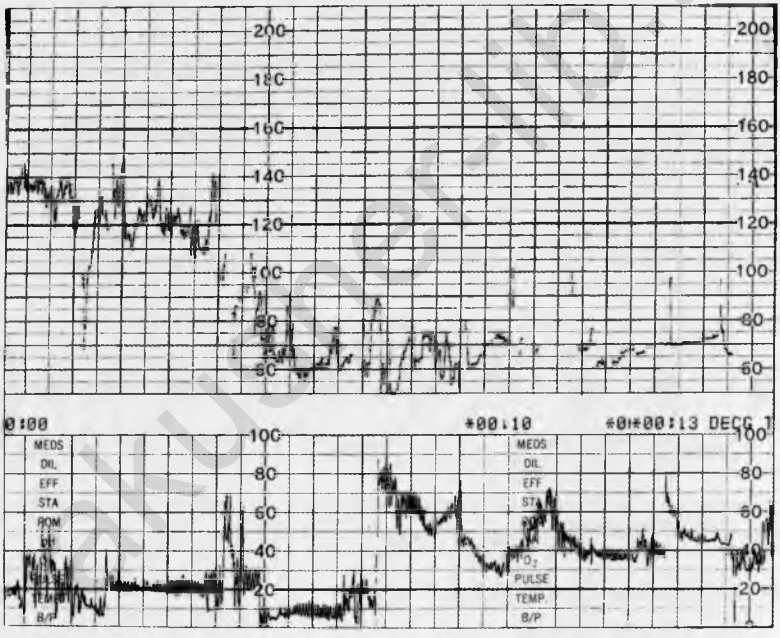

Figure 12-17. Abnormal second stage FHR trace – prolonged bradycardia.

PROLONGED DECELERATIONS IN THE FIRST STAGE OF LABOUR

Immediate delivery in this situation will necessitate a caesarean section. There are several publications in the literature that have given the audit findings of the decision to delivery interval, and onset of bradycardia to delivery interval.[4–7] These studies show that in a

reasonable proportion of cases delivery was possible by 20 min, and in a further considerable proportion of cases within 30 min. The discussion is related to the possibility of such timings in a busy set-up, especially if the registrar is busy attending to another case. The recommendations of the Royal College of Obstetricians and Gynaecologists to have consultant presence in the labour ward for longer periods, especially for 24 h in units delivering more than 6000 cases, may help in such situations.[8] The aim of the teaching of 3, 6, 9, 12 and 15 min guidance is to emphasize the urgency of the situation in the presence of prolonged decelerations.

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CARDIOTOCOGRAPHIC INTERPRETATION: MORE DIFFICULT PROBLEMS

Donald Gibb, Sabaratnam Arulkumaran

Situations that are of specific concern and interest include prolonged deceleration (bradycardia) and the dying fetus. In recent times, inadvertent recording of the maternal heart rate (MHR) mimicking the fetal heart rate (FHR) that has not been recognized by staff has led to adverse outcomes, and is also discussed here.

PROLONGED DECELERATION (BRADYCARDIA)

Prolonged FHR deceleration (bradycardia) (FHR <80 beats per min [bpm]) for less than 3 min is considered suspicious, and that for greater than 3 min is regarded as abnormal. A deceleration of greater than 3 min could be due to an acute event and may be a warning signal of acute hypoxia due to cord compression or prolapse, abruptio placentae, scar dehiscence, uterine hyperstimulation or another unknown cause. It does occur in healthy fetuses (possibly due to cord compression). Reversible causes for such an episode are epidural top-up, vaginal examination and uterine hyperstimulation. Simple measures such as adjusting the maternal position, stopping the oxytocin infusion, attending to hydration and giving oxygen by face mask may correct the condition. A patient who presents with continuous abdominal pain, vaginal bleeding, a tender, tense or irritable uterus and prolonged fetal bradycardia is likely to have suffered an abruption and warrants immediate delivery (see Ch.11). Those in whom scar dehiscence or rupture is suspected, and those with cord prolapse, may present with prolonged bradycardia and need immediate delivery.

Most cases of prolonged bradycardia with no major pathology will show signs of recovery towards the baseline rate within 6 min.

If the clinical picture does not suggest abruption, scar dehiscence or cord prolapse, and if the fetus is appropriately grown at term with clear amniotic fluid and a reactive FHR pattern prior to the episode of bradycardia, return back to the baseline FHR pattern within 9 min is to be expected. The recovery towards the normal baseline within 6 min with good baseline variability at the time of the bradycardia and during recovery are reassuring signs, and one should wait with confidence

that the FHR will revert to the normal baseline with a normal pattern. The staff should hold their nerve with confidence. If there are no signs of recovery towards the baseline rate by 6 min, action should be taken to determine the cause, to determine cervical dilatation and to consider delivery. If the cervix is fully dilated and the head is low, a forceps or ventouse delivery should be carried out, but a caesarean section may be preferred if the cervix is not fully dilated or the head is high. This caesarean section is considered category 1 or grade 1 in terms of the classification for the urgency with which it should be done. Category 1 should have a specific code or term assigned, such as 'grade 1', 'code red' or 'immediate', 'emergency' or 'crash' caesarean section, in order to mobilize all the staff (obstetricians, anaesthetists, additional midwifery staff, theatre staff, operating department assistants and paediatricians) needed to accomplish delivering the baby within 30 min of the decision being made. Obviously the decision should be made as early as possible but without overreaction. The best policy may be for the midwife and the doctor in that room to push the bed to the theatre while the midwife on duty calls for the anaesthetist, paediatric and theatre staff. Early entry into the theatre offers the opportunity for more people to help with the various tasks of setting up an IV line, sending blood for Hb and 'group and save', catheterization, and explaining to the couple the need for caesarean section and reassuring them.

A 45-year-old multiparous woman was well known to the medical staff and midwives. A diagnosis of term labour was made at 22.00 h when the cervix was 5 cm dilated and the initial cardiotocograph (CTG) was normal (Fig. 13-1A). Shortly before midnight a prolonged bradycardia became manifest after an otherwise-normal trace (Fig. 13-1B). The midwife correctly annotated 'FHR' at the end of this strip of trace. Figure 13-1C shows the heart rate improving with good variability; however, the inexperienced obstetric registrar decided to perform a caesarean section and consequently the trace shows 'discontinued for theatre'. Not surprisingly the Apgar scores were 9 at 1 min and 10 at 5 min. If the trace had not been disconnected it would have reverted to normal; a premature decision led to an unnecessary caesarean section in a multiparous woman in whom labour was probably progressing rapidly. A longer contraction duration or transient cord compression might account for the deceleration. The diagnosis was 'obstetric registrar's distress'!

If the FHR does not show signs of recovery by 9 min the likelihood of acidosis is increased, and one should take action to deliver the fetus as soon as possible.[1] The clinical picture has to be considered while anxiously awaiting the FHR to return to normal. Fetuses who are post term, growth restricted, have no amniotic fluid or have thick meconium-stained fluid at rupture of membranes are at a greater risk

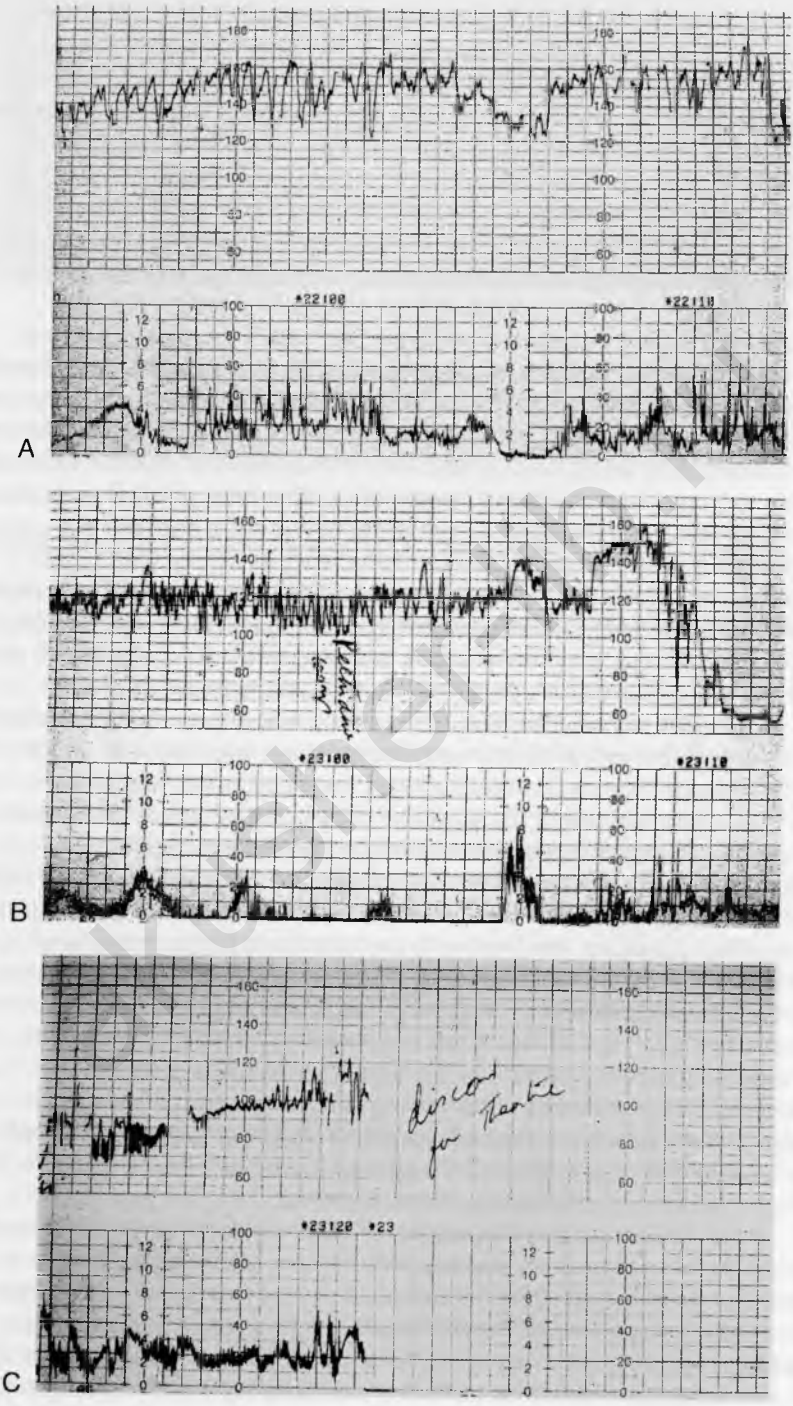


Figure 13-1. (A) Cardiotocograph: normal reactive pattern; (B) prolonged bradycardia; (C) improvement in heart rate.

of developing hypoxia. Those with an abnormal or suspicious FHR trace prior to the episode of bradycardia are also at a greater risk of hypoxia developing within a short time. In these situations it may be better to take action early if the FHR fails to return to normal. If uterine hyperstimulation due to oxytocics is the cause, oxytocin infusion should be stopped. Inhibition of uterine contractions with a bolus intravenous dose of a betamimetic drug may be of value in some situations. Fetal scalp blood sampling (FBS) at the time of persistent prolonged deceleration, or soon after, may delay urgently needed action and is contraindicated.[2] Figure 13-2 shows the trace in a case without obvious risk factors. FBS, which can prolong the deceleration due to pressure on the fetal head, delayed delivery. Caesarean section was eventually performed. The baby had very poor Apgar scores and died on the third day of life after a period of neonatal convulsions.

Fetal acidosis observed soon after a prolonged deceleration (Fig. 13-3A) will recover when the trace returns to normal (Fig. 13-3B). However, if the fetal heart rate does not return to normal then delivery should be undertaken. During a prolonged deceleration the fetus reduces its cardiac output. Carbon dioxide and other metabolites cannot be cleared by the respiratory function of the placenta. The initial pH at the end of a prolonged deceleration is low with a high PCO_2 showing a respiratory acidosis. Once the FHR returns to normal the carbon dioxide and metabolites are cleared and the pH and blood gases return to normal in 30–40 min. If the episode of prolonged deceleration continues then the fetus switches to anaerobic metabolism, resulting in metabolic acidosis, which is harmful to the fetus. Hence excessively prolonged deceleration results in a poor outcome.

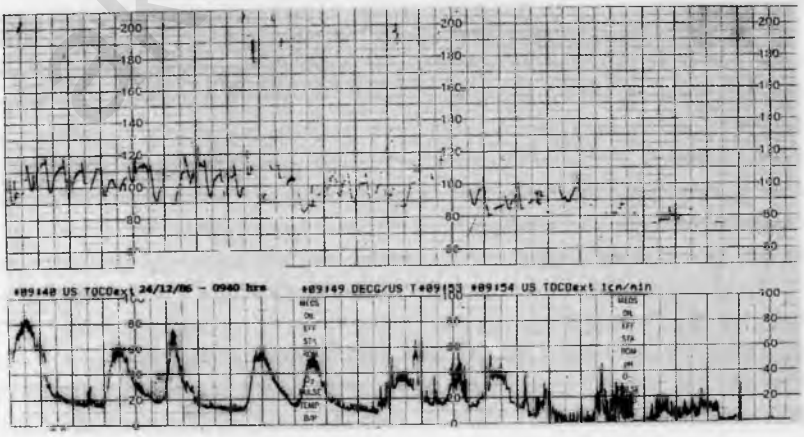


Figure 13-2. Fetal scalp blood sampling delays delivery: poor outcome.

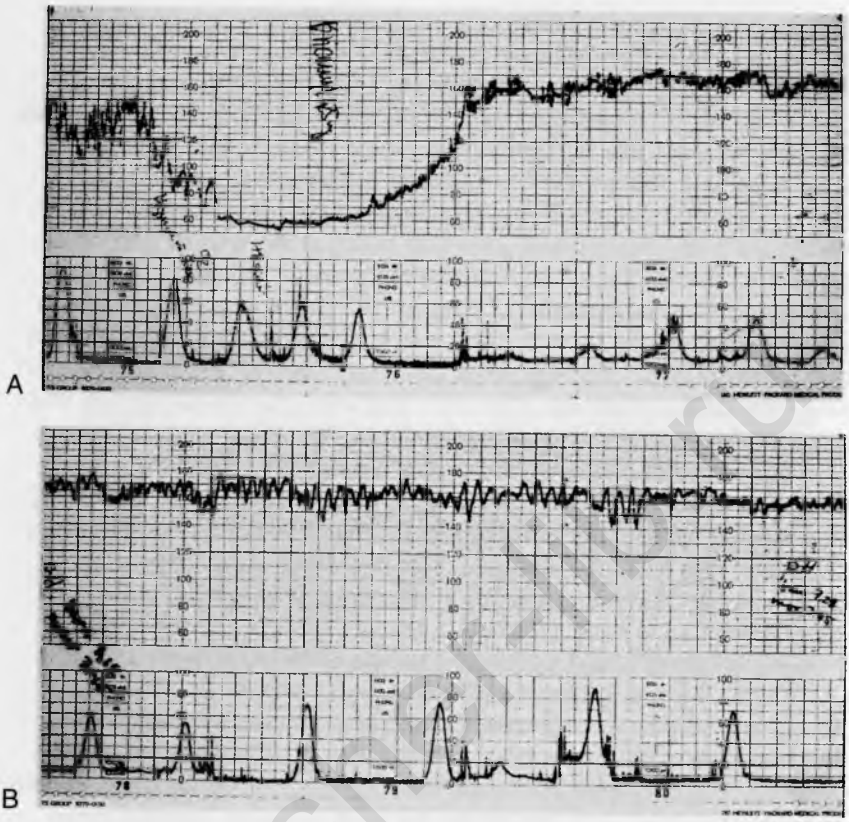


Figure 13-3. (A) Acidosis at time of bradycardia; (B) pH recovers after trace returns to normal.

Scalp pH measurement should not be performed for prolonged deceleration.

Scar rupture or dehiscence may not show the classical symptoms and signs of scar pain, tenderness, vaginal bleeding or alteration in maternal pulse or blood pressure. Changes in FHR or uterine activity may be an earlier manifestation of loss of integrity of the scar, and prompt action should avoid fetal or maternal morbidity or mortality. In these cases a prolonged deceleration may be an ominous sign and may indicate scar rupture. Figure 13-4A shows a prolonged deceleration in a case of labour with a previous caesarean section. Delivery was delayed (Fig. 13-4B), resulting in a baby with poor Apgar scores and neonatal asphyxial death on the second day. Whenever an operative delivery is planned the fetal heart should be checked prior to delivery as the baby may be already dead if there has been delay, but if scar rupture is suspected then caesarean section needs to be done to repair the scar.

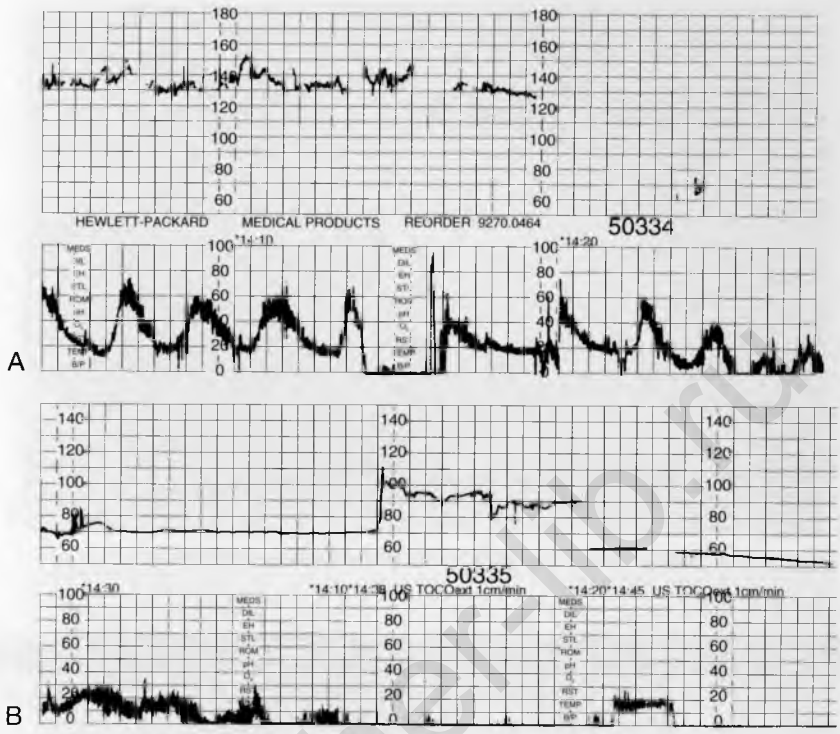


Figure 13-4. (A) Labour with previous caesarean section: prolonged bradycardia; (B) delay in delivery leading to poor outcome.

A prolonged deceleration following an eclamptic fit is shown in Figure 13-5. The convulsions were controlled and the baby was delivered in 30 min – a reasonable delay to stabilize the maternal condition. The fetal heart was not verified just before delivery and the baby was a fresh stillbirth.[3] In cases of placental abruption it may not be possible to listen to the fetal heart with a stethoscope or an electronic monitor. An ultrasound scan is therefore useful.

The procedure in the case of prolonged deceleration is shown in Table 13-1. Each hospital should have facilities to perform an immediate caesarean section and deliver the baby within 15–20 min of taking the decision, especially in the case of high-risk labours (such as those of previous caesarean section). This is referred to as delivery from a 'hot start'. Delivery by caesarean section from a 'cold start' may be permitted with a decision-to-delivery interval of 30 min. Audit and review of this in any unit is important.

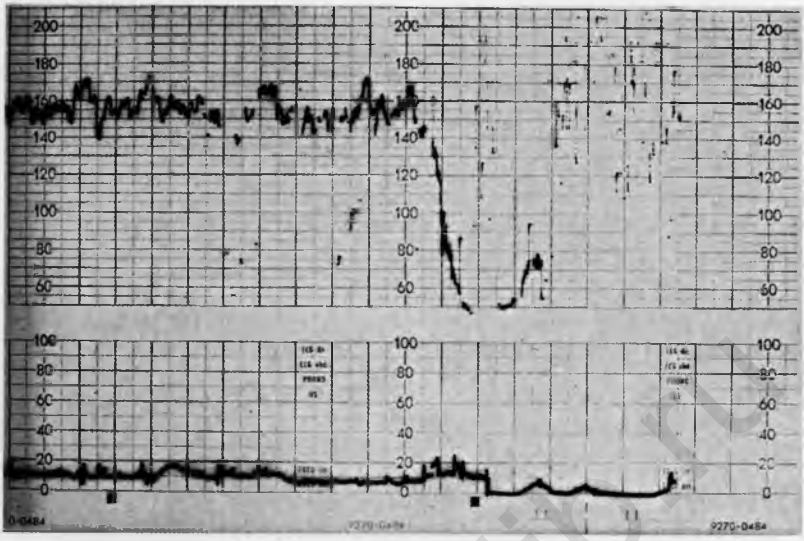


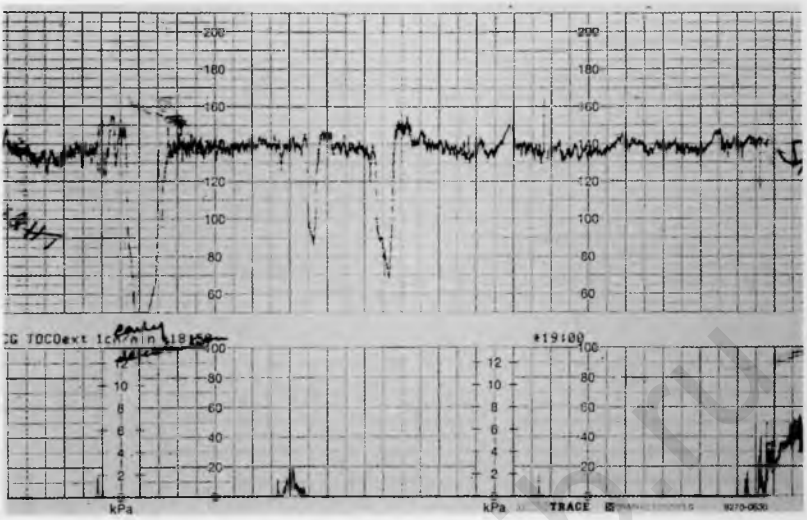
Figure 13-5. Prolonged bradycardia following eclamptic fit.

Table 13-1 Procedure for Prolonged Bradycardia	
3 min	Draw attention and review clinical picture and prior FHR trace
6 min	Expect recovery of FHR towards the baseline
9 min	If no recovery, prepare for operative delivery
12 min	Operative procedure should have started
15 min	Baby is delivered

THE DYING FETUS

Fetal death is always preceded by a terminal bradycardia. The trace preceding this may show a variety of features, most commonly a tachycardia.

Figure 13-6A–J shows 10 sequential hourly traces in a mismanaged case of a high-risk mother suffering from sickle cell disease. This case occurred many years ago. The baby was known to be small with oligohydramnios. For reasons difficult to comprehend the medical staff failed to act and at delivery this baby was in serious trouble. Severe variable decelerations are seen with a classical progression to tachycardia, absence of accelerations, reduced variability and terminal bradycardia. The baby was a fresh stillbirth. Knowing that the patient was a high-risk nulliparous woman, all who have read this book would have delivered the baby by the time of the third strip of tracing, when



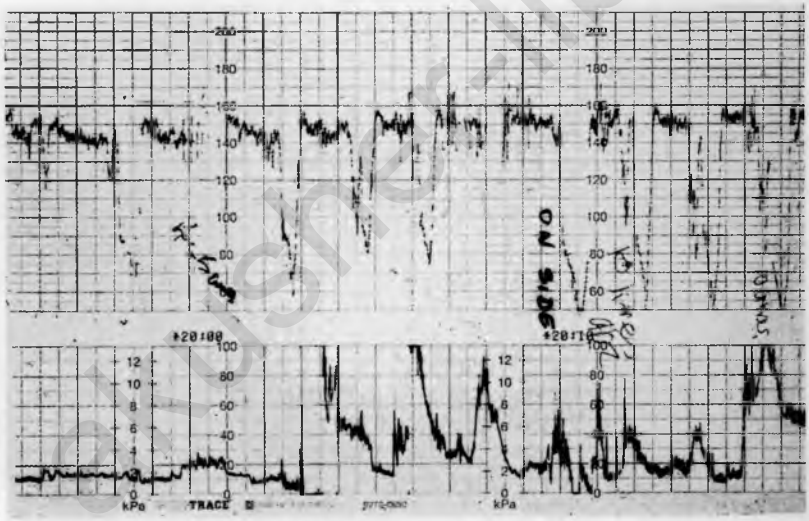


Figure 13-6. Dying fetus: (A) 1 h; (B) 2 h;

(Continued)

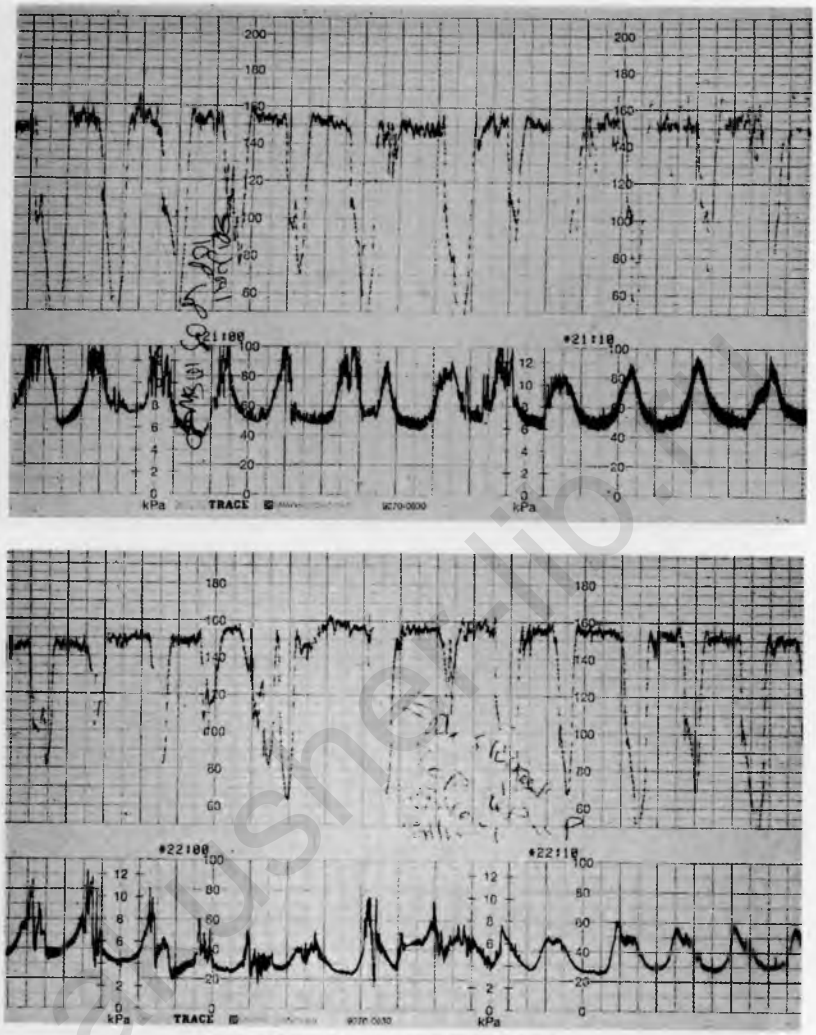
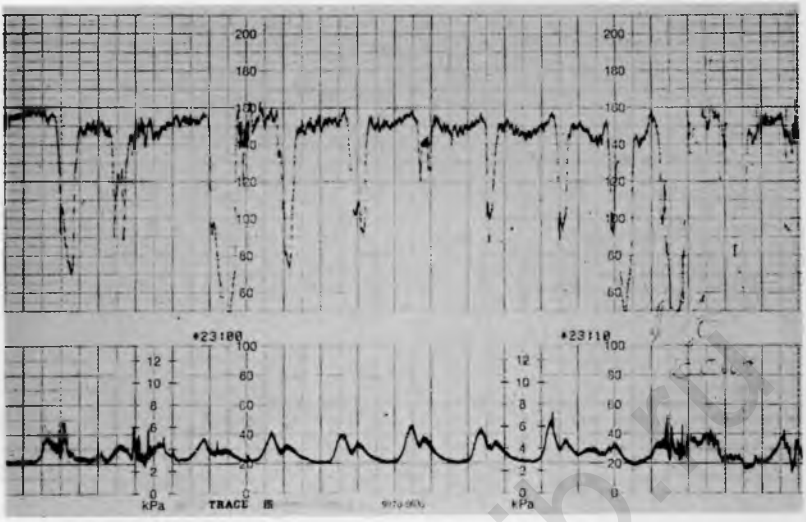


Figure 13-6. (*Continued*) (C) 3 h; (D) 4 h;

E



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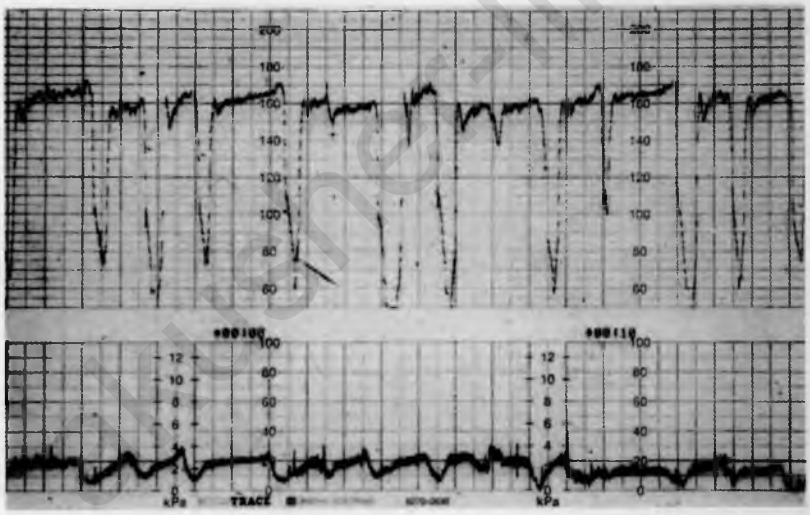

Figure 13-6. (*Continued*) (E) 5 h; (F) 6 h;

(Continued)

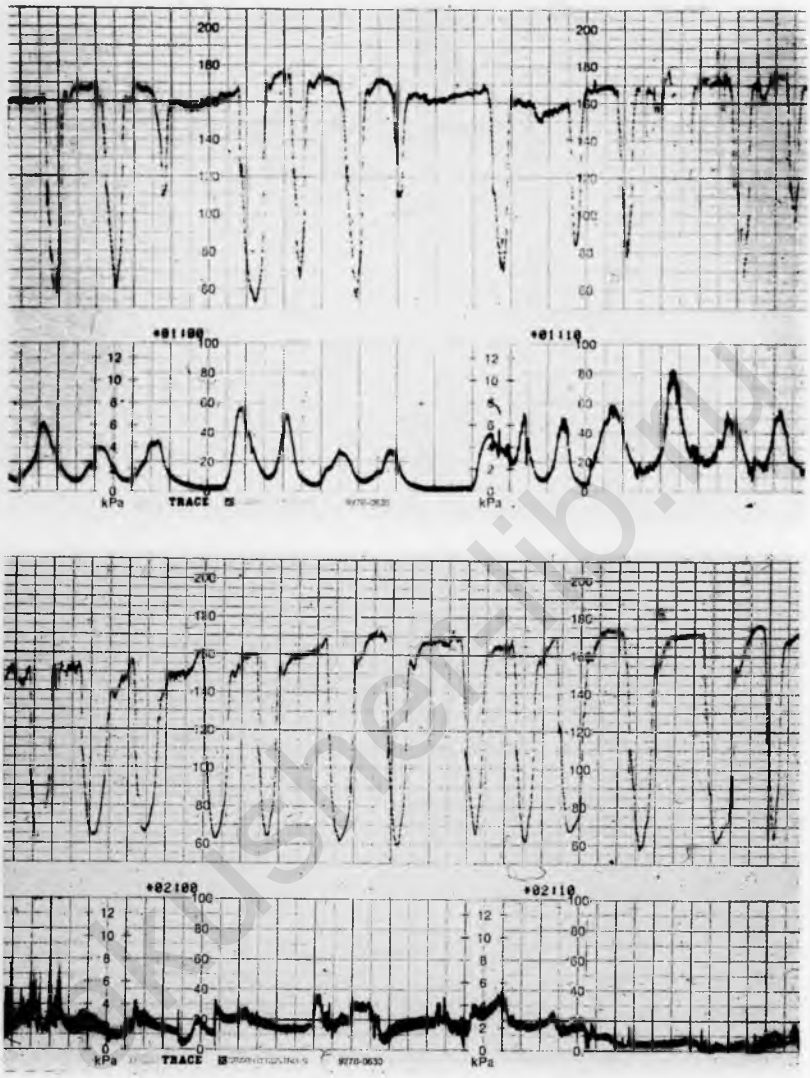

Figure 13-6. (*Continued*) (G) 7 h; (H) 8 h;

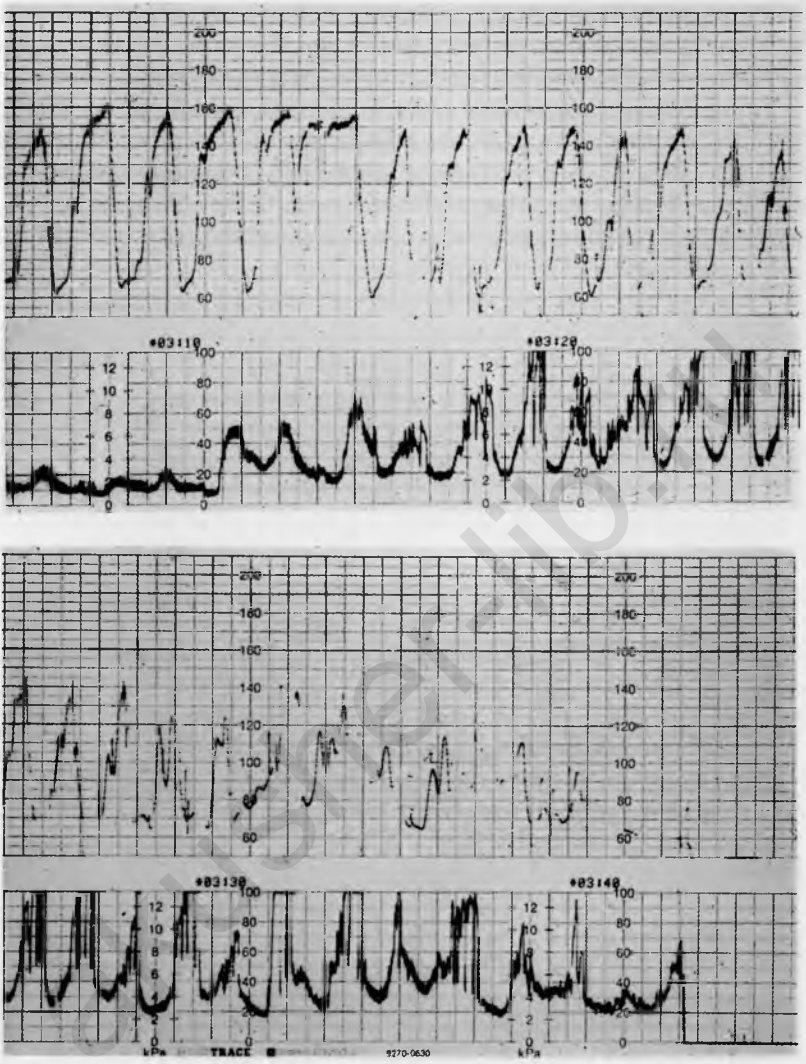

Figure 13-6. (*Continued*) (I) 9 h; (J) 10 h.

the baby would have been in a reasonable condition. This high-risk woman had everything modern technology could offer, with the notable exception of basic common sense on the part of the staff.

Some fetuses become so compromised in a more chronic way that they are unable to generate decelerations. This type of trace (Figs 13-7 and 13-8) is often misunderstood. There may be little in the way of a tachycardia but there is a complete absence of accelerations, a silent pattern of baseline variability and subtle,

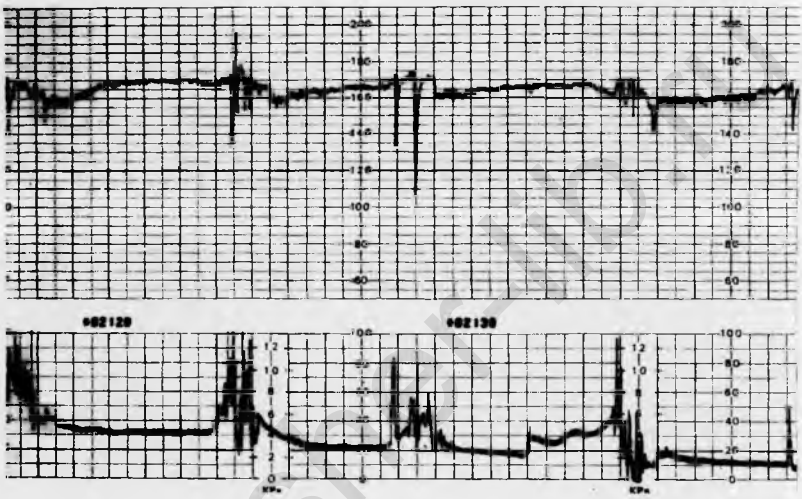

Figure 13-7. Ominous trace.

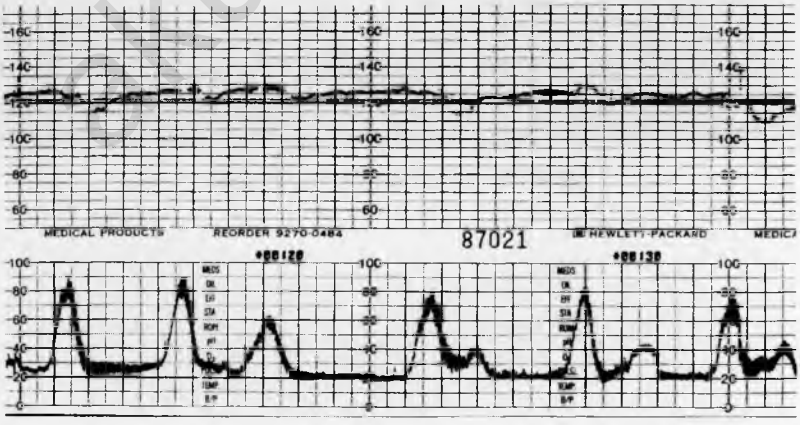

Figure 13-8. Ominous trace.

shallow late decelerations. This is an ominous picture and the baby must be delivered. These babies tend to have other clinical symptoms or signs such as absent fetal movements, intrauterine growth restriction, intrauterine infection, bleeding, post-term pregnancy or scanty fluid with thick meconium.

An ominous tracing demands delivery.

Birth asphyxia is often associated with prelabour asphyxia. This highlights the value of the admission test whenever there is a suspicion of fetal compromise or in an unbooked case. Should all babies with ominous traces be delivered with the expectation of a living, undamaged child? We are obliged to deliver all such babies, but some features may indicate a poor prognosis.

A good trace within a reasonable period of the deterioration with an acute event such as an abruption suggests rapid intervention will be productive, assuming a reasonable gestational age. Intervening when the main feature is tachycardia suggests some ability of the fetus to survive. Once the terminal bradycardia develops after the tachycardia the situation may be irretrievable (Fig. 13-9), especially when there are features of a random, uncontrolled undulatory pattern with no baseline variability (Fig. 13-10). This pattern suggests the possibility of central nervous system damage due to hypoxia. The challenge is to intervene in such pregnancies before this situation is reached; however, it should be kept in mind that central nervous system malformations can give rise to such patterns (see Ch. 7). We are now in such a state of knowledge that the parents may be informed of the likelihood of a poor outcome in spite of intervention. For the moment, delivery remains mandatory.

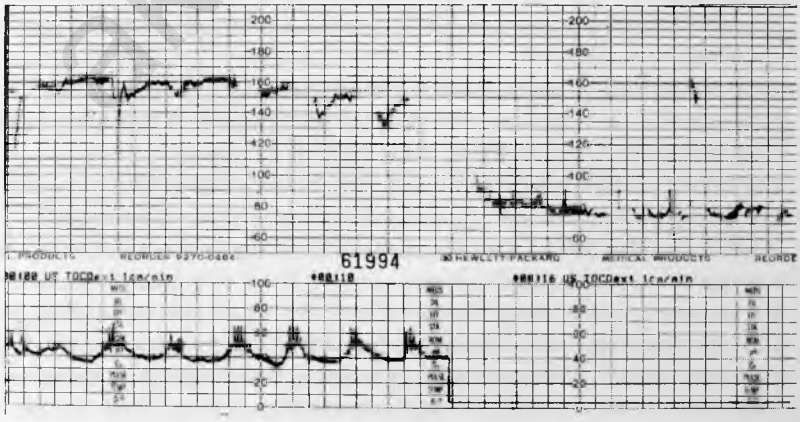

Figure 13-9. Terminal bradycardia.

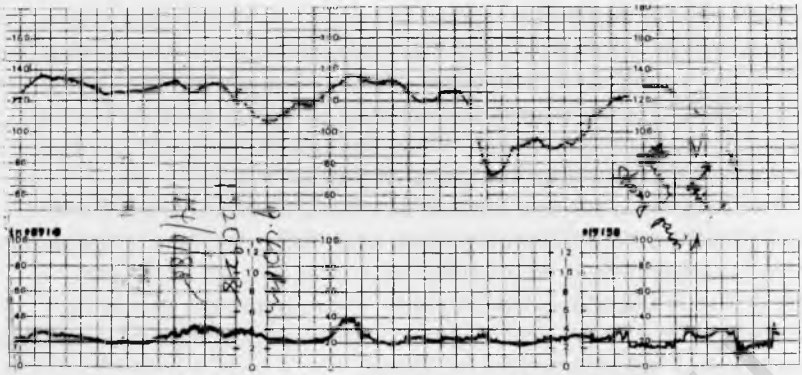

Figure 13-10. Terminal hypoxic central nervous system damage.

RECORDING OF THE MATERNAL HEART RATE THAT CAN MIMIC THE FETAL HEART RATE

Maternal heart rate (MHR) recording can mimic the FHR recording. This can arise in many situations and the steps to avoid this are:

1. Follow the current recommendation of the Medical Devices Agency. At the onset of the electronic fetal monitoring auscultate the FHR and apply the transducer, rather than cross-checking with the maternal pulse. The reason for this is that the maternal pulse can be picked up by the ultrasound transducer and can be doubled (an increase of 100%), or it could be increased by 50%. It would be difficult to state whether the recording seen is that of the mother or the fetus.
2. It is also not uncommon for the machine to switch from fetal to maternal heart rate halfway through the recording. Any sudden shift in the baseline rate or a double baseline rate should indicate the possibility of recording the MHR and should warrant auscultation of the FHR.
3. Should there be a technically unsatisfactory recording with an ultrasound it is important that a scalp electrode is applied to obtain continuous FHR recording unless there is a contraindication to the use of a fetal scalp electrode. This occurs more commonly in the late first and second stage of labour when the head moves down, or when the mother is restless, or there are too-frequent contractions with decelerations.
4. Be wary of a clear step change in the fetal heart pattern during the late first stage and the second stage of labour as the fetal head descends. This may not necessarily be a change in baseline rate, but rather a change in appearance (Fig. 13-11). The overall features of baseline variability and reactivity seen on a trace are

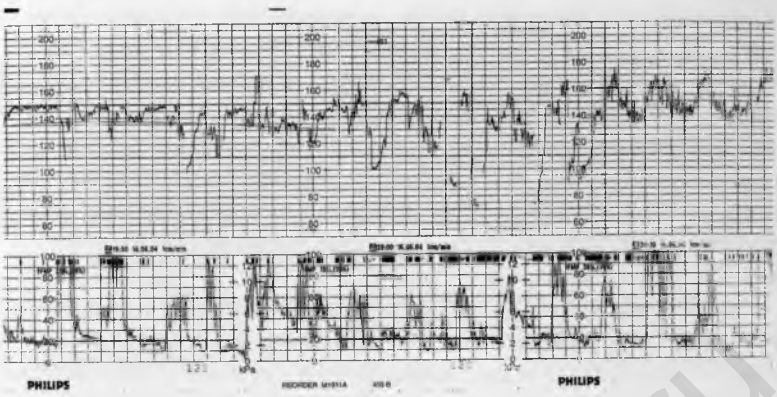

Figure 13-11. Observe the change in the fetal heart rate decelerative pattern with each contraction suddenly shifting to an accelerative pattern of maternal heart rate. The rise starts with the onset of the contractions and returns to its baseline rate with the offset of the contractions.

consistent throughout labour allowing for the normal variation of fetal sleep/wake cycles. At this stage of labour the baseline heart rates of mother and baby may be rather similar. The reappearance of accelerations is not reassuring after their preceding absence. It may be acceleration of the mother's heart (see below). Auscultation may help, but application of a fetal scalp electrode will clarify the picture.

The following gives an explanation of how these incidents occur. The characteristics of the MHR recording are different from those of the FHR recording in the second stage of labour. The FHR decelerates with head compression while the MHR often increases with the uterine contractions. This should be identified and the FHR should be auscultated if there is any doubt. This knowledge should be disseminated widely to the maternity service practitioners (doctors and midwives). It would also be useful for those who are working in the community and midwifery birthing centres.

The Appearance of the Maternal Heart Rate in Labour

The traces shown in Figure 4-28 are simultaneous recordings of the FHR (upper trace) and MHR (middle trace). The MHR is recorded by a precordial electrocardiograph (ECG) lead on the anterior aspect of the mother's chest and is indicated automatically by the machine as 'MECG' in between the contraction (lower trace) and the FHR chart channels. The MHR recording shows features of accelerations and increased baseline variability.

Unless closely observed for the accelerations corresponding to the contractions it is similar to the FHR. The MHR pattern in labour has been studied.[4] Following such studies the unintentional recording of MHR in labour has been increasingly reported[4,5] and it has been shown that increase of the MHR with contractions is present in most cases. *This rise in the baseline heart rate may be a response to the increased blood flowing into the maternal heart during the uterine contractions.* A typical example is shown in Figure 13-11.

The CTG shown in Figure 2-6 was that of a dead fetus and the signals were recorded with the use of a scalp electrode, which shows the accelerations corresponding to the uterine contractions. In situations of fetal death the ultrasound transducer may pick up one of the maternal vessel pulsations and present it on the recorder, which gives a false impression of the FHR. If the characteristics of the MHR are not recognized one may continue to record, thinking that it is the FHR, only to find that the baby is stillborn or in a poor condition.

One has to think why the heart rate is accelerating with contractions in the late first and second stage of labour instead of having early or variable decelerations compatible with compression of the fetal head. Unfortunately this is not common knowledge to clinicians, nurses or midwives and many interpret this as an FHR trace with accelerations in the second stage of labour. Alternatively they mistake the peak of the increase of the MHR to be the baseline FHR (if the MHR remains high for a longer duration) and the return of the MHR to its baseline rate as FHR decelerations.

Figure 13-12 illustrates how the fetal heart rate is recorded for continuous fetal heart rate monitoring. Mostly it is done using an ultrasound transducer or a scalp electrode. If the baby is dead there is a possibility that the maternal ECG could be transmitted via the electrode and recorded on the chart, and for observers to believe that it may be the FHR. Similarly the ultrasound transducer can pick up any pulsating maternal vessels, calculate the rate and record this on the chart, mimicking an FHR, especially when there is no FHR or a very low FHR.

Figure 13-13 illustrates how the ultrasound transducer can slip from its original position where it was picking up the fetal heart, and then pick up a maternal pulsation, giving a trace of the MHR that may appear like the FHR, unless someone recognizes it and readjusts the transducer to get the FHR. If it were not recognized, the MHR would have been recorded without knowledge of the FHR until the end of labour. Here the sudden shift is obvious, but in exceptional cases it may be very subtle and difficult to pick up

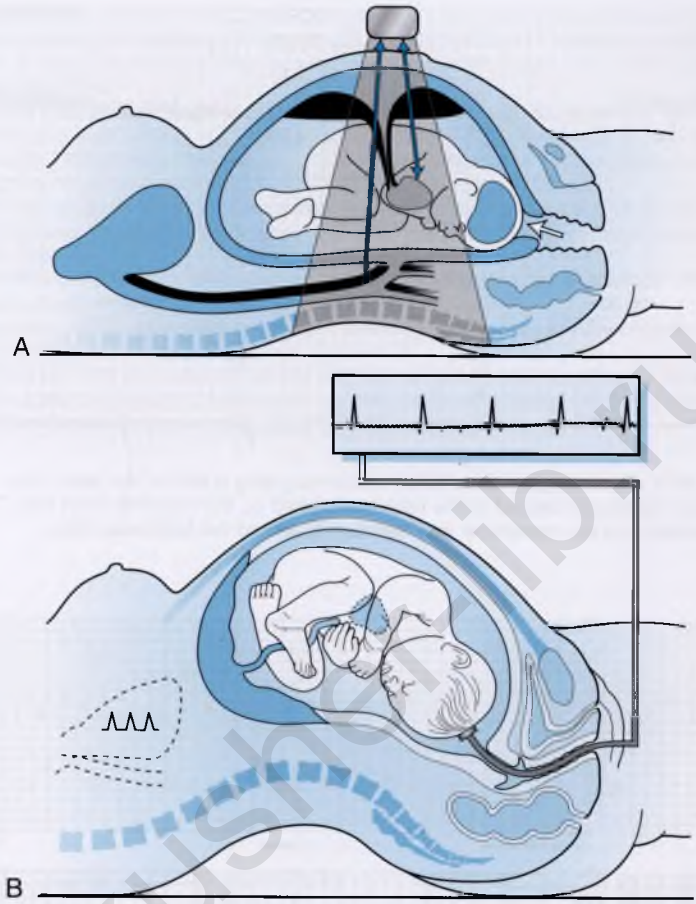

Figure 13-12. Recording fetal heart rate by (A) ultrasound transducer; (B) scalp electrode.

unless there is close scrutiny to observe the sudden changes in the baseline rate or the characteristics of the heart rate pattern.

It is known that the ultrasound transducer may pick up the maternal signal if the target signal moves away, such as after delivery of one twin or when there is sudden death of a fetus or acute fetal bradycardia.

Figure 13-14 shows how the machine doubles the FHR with bradycardia. In this case the two rates are seen, the lower line showing the true baseline FHR and the upper one the heart rate due to doubling. It is important to auscultate the FHR when two rates are recorded so as to identify whether it could be fetal or maternal.

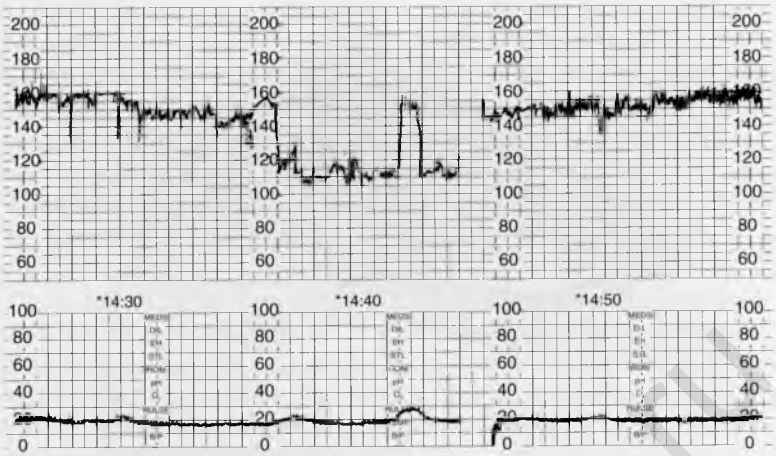

Figure 13-13. Recording by ultrasound – initial recording is that of the fetus. The ultrasound transducer slipped to the flank and picked up the maternal heart rate. This was identified and the transducer was replaced to record the fetal heart rate.

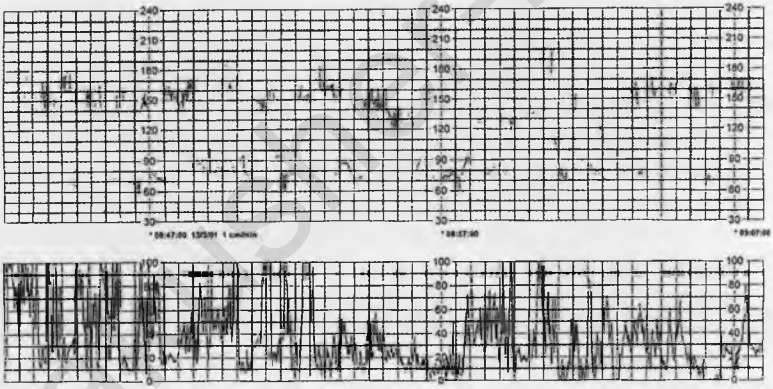

Figure 13-14. Trace showing fetal bradycardia of 70–75 bpm and doubling of the rate to 140–150 bpm thus giving two heart rates.

At times the machine can record the MHR (perhaps doubled) with occasional glimpses of the FHR at a lower rate.[6] One should observe the heart rate in relation to the contractions with the bearing-down efforts in the second stage of labour. If the heart rate increases when the mother has painful contractions and returns to the baseline after the contraction returns to the baseline, it is most likely to be the MHR because the FHR should decelerate with contractions due to head compression.

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FETAL SCALP BLOOD SAMPLING: pH AND LACTATE

Donald Gibb, Sabaratnam Arulkumaran

The availability of fetal scalp blood sampling (FBS) for the assessment of fetal scalp capillary blood pH and its application in practice vary enormously. The NICE guidelines have suggested the use of scalp pH in situations with a suspicious and/or pathological cardiotocograph (CTG) after due consideration to the clinical situation.[1] At times the clinical situation may demand early delivery rather than a scalp pH. In reality, junior doctors use scalp pH more when they have less experience of labour ward responsibilities. This is understandable as they have a greater anxiety with a less-developed degree of understanding. As they gain experience and use pH as a guide, they then understand better the associations of an abnormal and a normal pH and will need this reassurance less often. The process of scalp blood sampling is undignified and uncomfortable for the woman. This is not to say it should not be done if properly indicated. However, its value in modern practice has been challenged on the grounds that scalp capillary pH may not reflect the arterial pH, may be false if contaminated with amniotic fluid, may occasionally give rise to massive fetal haemorrhage, may inadvertently lead to leak of cerebrospinal fluid and may delay the urgently needed intervention.[2]

When the fetal heart rate (FHR) is reactive and normal, the chance of fetal acidosis is extremely low.[3–5] Nevertheless, suspicious and abnormal FHR changes are not always associated with acidosis.[4–7] Such observations form the basis of the perceived need to measure fetal scalp pH for further investigation.

Changes in the CTG cause anxiety to the person not familiar with CTG interpretation. An inexperienced person in a centre with FBS facilities might perform FBS more frequently. When properly interpreted, assessment of FHR changes in most cases proves of equal value to pH in predicting fetal outcome.[8] FBS is a useful adjunct because, even with the worst pattern of tachycardia, reduced baseline variability and decelerations, only 50–60% of the fetuses are acidotic.[4] A wall chart correlating different FHR patterns to the percentage who are likely to be acidotic is available in most labour wards. It is clear from that chart and other studies that when the FHR pattern exhibited accelerations the chance of fetal acidosis was zero, emphasizing

accelerations as the hallmark of fetal health.[4] One problem of these charts is that all fetuses do not conveniently provide a fetal heart tracing that easily falls into one category. There is the added perspective of the need for a time continuum, which is so important in trace analysis. The physiological reserve of some fetuses will show more decline in pH with a given CTG trace than others (e.g., an appropriately grown fetus compared with one with intrauterine growth restriction [IUGR]).

Baseline variability is another good indicator of fetal health. When normal baseline variability is observed in the last 20 min prior to delivery, the babies are in good condition at birth regardless of other features of the trace.[9] Fetal acidosis is more common when there is a loss of baseline variability with tachycardia or late decelerations.[4,10] The preservation of normal baseline variability indicates that the autonomic nervous system is responsive and the fetus is trying to compensate despite other abnormal features in the trace. The reason that, with a given FHR pattern, there are different percentages of fetuses showing acidosis depends on the duration for which the suspicious or abnormal FHR pattern was present before the time of FBS.[11] The approximate duration after which acidosis develops in an appropriately grown term fetus with a given FHR pattern has been discussed previously. It is also known that, in fetuses with less 'placental reserve' such as those with IUGR, thick, scanty meconium-stained fluid,[12] in the presence of bleeding and in post-term infants, the rate of decline of pH is steep compared with term infants appropriately grown with abundant, clear amniotic fluid.

RESPIRATORY AND METABOLIC ACIDOSIS

Assessment of pH alone does not suffice to identify the fetus at risk, and more comprehensive blood gas analysis may be necessary for clinical management. The placenta is the respiratory organ of the fetus. Reduction of perfusion of the placenta from the fetal circulation is manifest as variable decelerations due to cord compression, and reduction of perfusion from the maternal circulation is manifest as late decelerations. During the early stage of such threats the transfer of carbon dioxide from the fetal to maternal side is reduced, leading to its accumulation. This results in respiratory acidosis manifested by a low pH and a high PCO_2. Respiratory acidosis is transitory, particularly when corrective measures are taken and can be managed conservatively provided the FHR pattern improves. With a further reduction of perfusion from the maternal or fetal side the oxygen transfer becomes affected, leading to anaerobic metabolism and metabolic acidosis in the fetus. This is manifested by a low pH, low PO_2 and high base excess. Such metabolic acidosis is damaging to the

tissues. Transitory low pH values of respiratory type are not uncommon in low-risk labours. Acidotic pH values in cord arterial blood in babies born with good Apgar scores are due to this phenomenon: 73% of babies with cord pH below 7.00 had a 1 min Apgar score of more than 7, and 86% had a 5 min Apgar score greater than 7.[13] These findings are probably due to respiratory acidosis, which does not correlate well with the fetal or neonatal condition. In this situation a comprehensive blood gas analysis, including PCO_2, base excess and preferably lactic acid, is desirable and more predictive. Caution should be exercised in using equipment that measures only pH. It is possible to determine the degree of metabolic acidosis by measuring the lactic acid level by the bedside with 5 µl of blood using the lactate card.[14] Intrauterine infection with a high metabolic rate presents a greater oxygen demand to the fetus, and metabolic acidosis might develop with minimal interruption of placental perfusion.

WHEN TO DO FETAL BLOOD SAMPLING

Gradually Developing Hypoxia

The fetus becomes hypoxic and acidotic in labour in association with compromise of perfusion to the fetal or maternal side of the placental circulation. With the exception of situations of acute hypoxia due to cord prolapse, scar dehiscence, abruption and prolonged bradycardia, it is unusual for a fetus who has shown accelerations and good baseline variability to become hypoxic without developing decelerations in labour. The decelerations indicate the presence of stress to the fetus, whether from the challenge of poor perfusion or from mechanical pressure. Provided that the baseline FHR has not started to rise and there is no reduction in the baseline variability to less than 5 beats, there is little to be gained by performing FBS, as the pH is likely to be normal unless the decelerations are prolonged and last for a duration two to three times greater than the duration of baseline FHR between the decelerations. If the baseline FHR has risen by 20–30 beats and is not showing any further rise, with a reduction in variability to less than 5 beats, then hypoxia is probable. Despite the fetus having increased its cardiac output to a possible maximum by increasing the FHR, the functioning of the autonomic nervous system controlling the baseline variability is compromised by hypoxia. The time course of this process may be referred to as the *stress-to-distress period*. This period varies from fetus to fetus depending on the physiological reserve. This reserve is low in high-risk situations of postmaturity, IUGR and intrauterine infection and in those with thick meconium and scanty amniotic fluid.

When the FHR shows hypoxic features suggestive of distress it is important to perform FBS for pH and blood gases as the fetus may

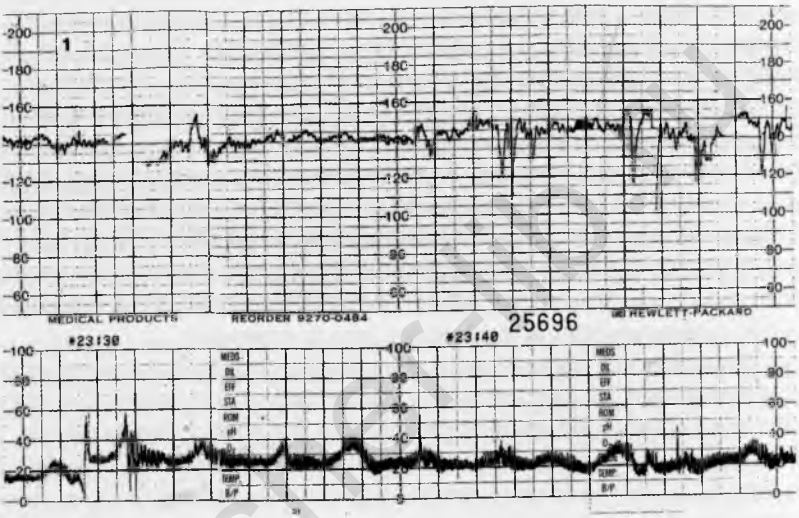
be, or become, acidotic. Initially this will be a respiratory, followed by a metabolic, acidosis. Once the FHR shows a distress pattern (markedly reduced baseline variability with late or atypical variable decelerations), the time taken for metabolic acidosis to develop is unpredictable. This pattern is referred to as a *preterminal pattern* by some authors. After a certain duration of the distress pattern (the *distress period*) the FHR starts to decline in a rapid stepwise pattern, culminating in terminal bradycardia and death (the *distress-to-death period*). The stress-to-distress interval (20.00–00.00 h, i.e. 4 h), the distress period (00.00–03.00 h, i.e. 3 h) and the distress-to-death period (03.00–03.40 h, i.e. 40 min) are illustrated in Figure 13-6A–J. Another example where the stress-to-distress period, the distress period and the distress-to-death period are much shorter is shown in Figure 8-4 A–F. Clinical interpretation of the FHR pattern will identify the onset of stress, distress and the stress-to-distress period. It will also identify the fetus in the distress period. An accurate prediction of the distress period cannot be made based on the FHR pattern, as illustrated by these two examples. During the final decline phase (distress-to-death period), when the fetal heart rate drops irretrievably within a short period, it is often too late to intervene.

The value of FBS may be at the onset of the distress period and again repeated 30–40 min later or earlier, depending on the first pH and base excess, baseline variability and the type of decelerations. Adherence to the recommendation of immediate delivery when the pH is less than 7.20 (acidosis), and a repeat sample after 30 min or less when the pH was 7.20–7.25 (preacidosis) is good practice. Previous recommendations were that when the pH was greater than 7.25 the repeat sampling was not required unless the FHR deteriorated. This approach may generate a false sense of security when the trace does not deteriorate, although the pH is declining. Repeat measurement in appropriate time, based on the first pH and increasing abnormality of the trace (further rise in baseline rate, deepening and widening of the decelerations and reduction of the duration of the FHR at the baseline rate and reduction in baseline variability) even when the first pH is in the normal range, helps to identify the rate of decline.[15] A decision for delivery can be made considering the rate of decline of the pH, the clinical risk factors (IUGR, thick meconium), parity, current cervical dilatation and rate of progress of labour.

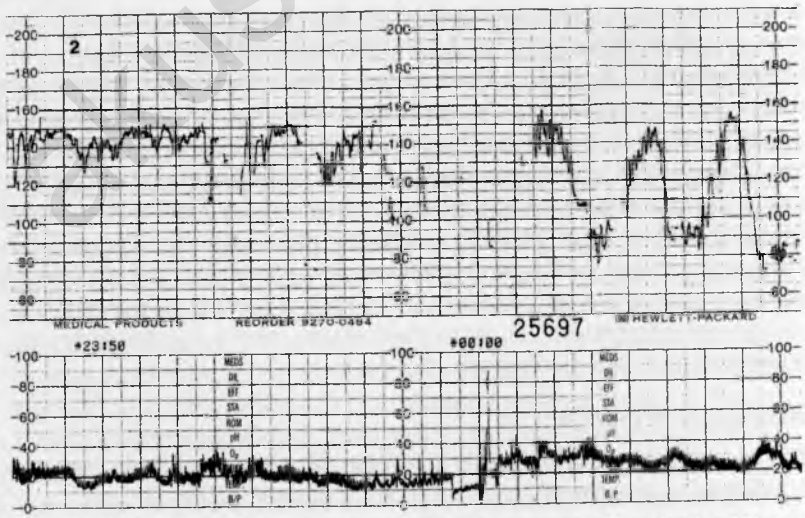
Subacute Hypoxia

The pH may deteriorate rapidly in a fetus who had previously had a reactive trace without an increase in the baseline FHR, if the decelerations are pronounced with large dip areas (drop of more than 60 beats per min [bpm] for over 90 s) with the FHR recovering to the baseline for only short periods of time (less than 60 s). Examples of such traces are

shown in Figure 14-1A–F. In these situations a drop in pH can be by as much as 0.01 every 3–4 min. This decline in pH will be even steeper if the preceding trace was suspicious or abnormal, or the clinical picture was one of high risk (IUGR, thick meconium with scanty fluid, or intrauterine infection). Further insults at this time, such as oxytocin infusion or a difficult instrumental delivery, may make the situation worse. With such traces, attempts at FBS will delay much-needed urgent delivery.

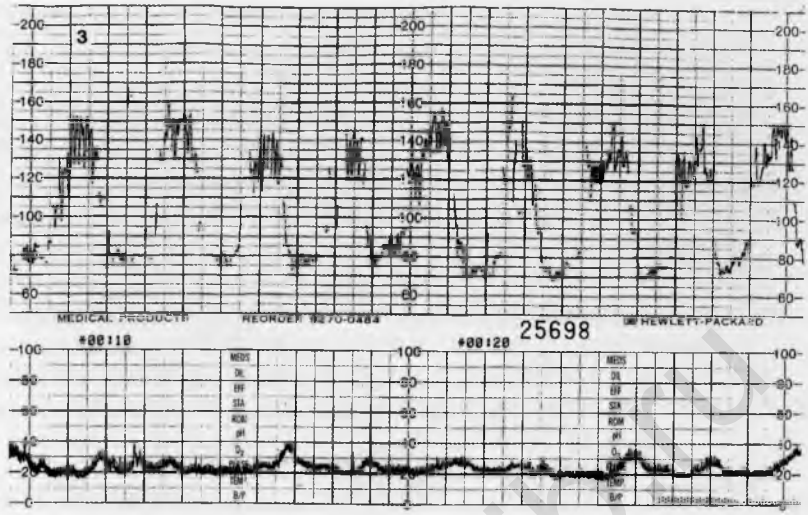


A



B

Figure 14-1. (A–F) Subacute hypoxia – prolonged decelerations (>90 s, depth >60 bpm) with short intervals of recovery (<60 s) to baseline rate.



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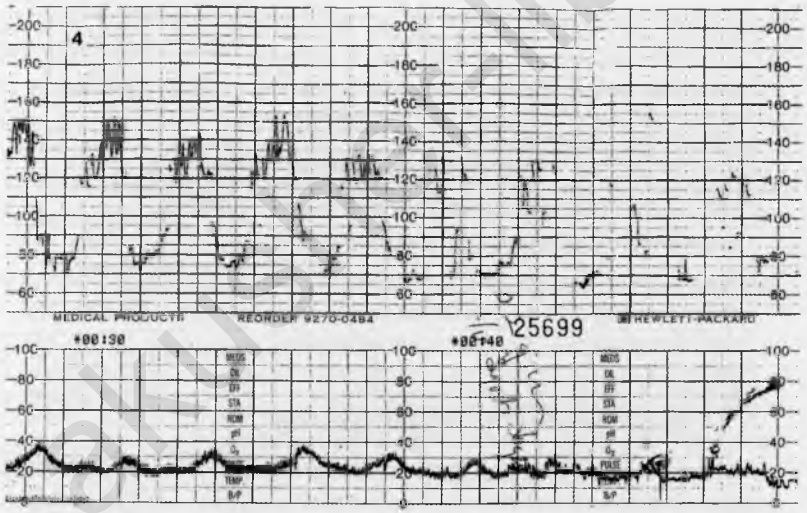
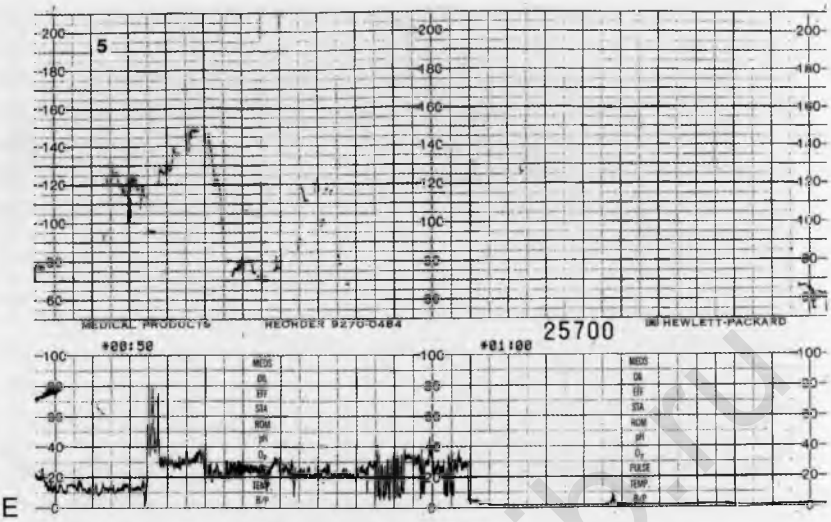


D

Figure 14-1. (*Continued*)



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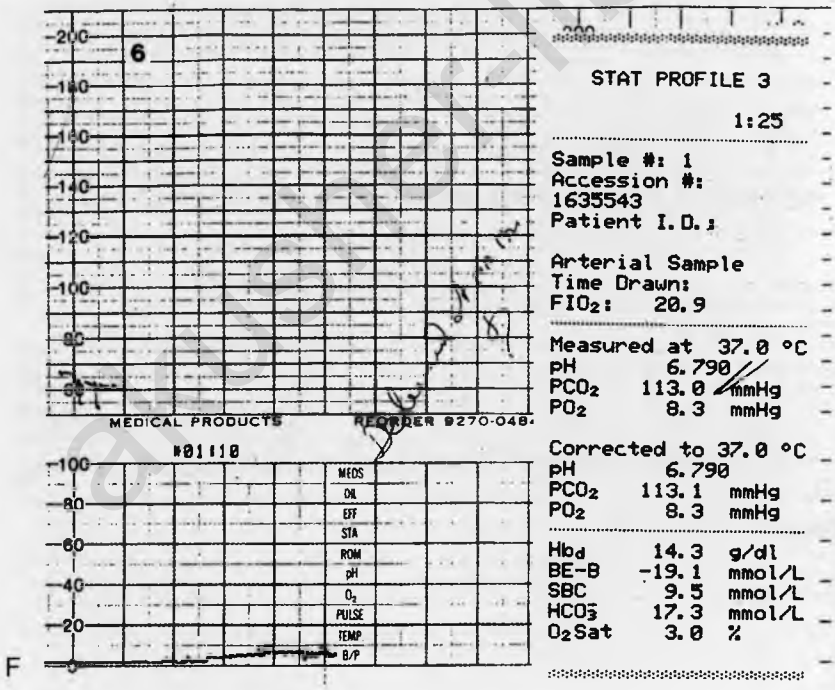

F

Figure 14-1. (*Continued*)

Chronic ('Long-Standing') Hypoxia

A non-reactive FHR pattern showing a baseline variability less than 5 beats with shallow decelerations (less than 15 beats for 15 s), even with a normal baseline rate, indicates severe compromise and delivery should be expedited without delay to avoid fetal death (see Fig. 8-6A–D). A non-reactive trace with a baseline variability of less than 5 beats but without decelerations lasting more than 90 min indicates the possibility of already existing hypoxic compromise or damage due to other reasons (e.g., cerebral haemorrhage). This needs further evaluation if the pH is normal. In these circumstances, fetal death may occur suddenly without further warning of a rise in baseline FHR or decelerations (see Fig. 8-5A–J). Hence, a non-reactive trace for greater than 90 min is abnormal and is an indication for further evaluation to rule out hypoxia.

Acute Hypoxia

Abruption, cord prolapse, scar dehiscence and uterine hyperstimulation may give rise to acute hypoxia. This may manifest as prolonged bradycardia; at other times prolonged bradycardia occurs without obvious reason and in all circumstances is associated with rapidly progressive acidosis. With a bradycardia of less than 80 bpm the pH is likely to decline at the rate of approximately 0.01 per min.[16] The decline may be steeper in the presence of an abnormal trace prior to the bradycardia.

With FHR patterns suggestive of acute or subacute hypoxia, performing a FBS might delay intevention, resulting in poor outcome. In FHR patterns with poor variability lasting for more than 90 min, but with no decelerations, investigations should be performed to identify the cause. The principle can be established that the FHR pattern identifies the onset of stress (decelerations) and of distress (maximal elevation of baseline FHR with baseline variability less than 5 beats). Although the onset of stress and distress can be identified, the duration of the distress period before the fetus becomes hypoxic and acidotic cannot be predicted. A decision is required to deliver or to perform FBS, bearing in mind the clinical picture, if the prospect of early delivery is poor.

WHEN NOT TO DO FETAL BLOOD SAMPLING

Frequently the FHR changes observed might be due to factors other than hypoxia. Dehydration, ketosis, maternal pyrexia and anxiety can give rise to fetal tachycardia but do not usually present with decelerations. Occipitoposterior position is known to be associated with more variable decelerations without hypoxic features, as

evidenced by normal baseline rate and variability.[17] Oxytocin can cause hyperstimulation resulting in FHR changes of various forms, which have been discussed in Chapter 10. Prolonged bradycardia can be due to postural hypotension following epidural analgesia. FHR changes should be correlated with the clinical picture before action is taken. In many instances remedial action such as hydration, repositioning of the mother or stopping the oxytocin infusion will relieve the FHR changes and no further action is necessary. When the FHR changes persist despite such actions, a FBS or one of the stimulation tests is warranted. At times FBS may not be necessary because the trace is reassuring with accelerations and normal baseline variability despite some decelerations (see Fig. 14-4), or it may show a low result transiently and later a good result; the pH may be low transiently owing to respiratory acidosis. Above all, when the trace is ominous or the clinical picture is poor it is better to deliver the baby rather than wasting time with FBS. At times a false reassurance leads to an unsatisfactory outcome.

Scalp FBS is often not appropriate under the following circumstances:

1. When the clinical picture demands early delivery (Fig. 14-2): 42 weeks' gestation, cervix 3 cm dilated, thick meconium with scanty fluid
2. When an ominous trace prompts immediate delivery (Fig. 14-3)
3. When the FHR trace is reassuring (Fig. 14-4)
4. When the changes are due to oxytocic overstimulation (see Fig. 10-5)
5. When there is associated persistent failure to progress in labour (Fig. 14-5)

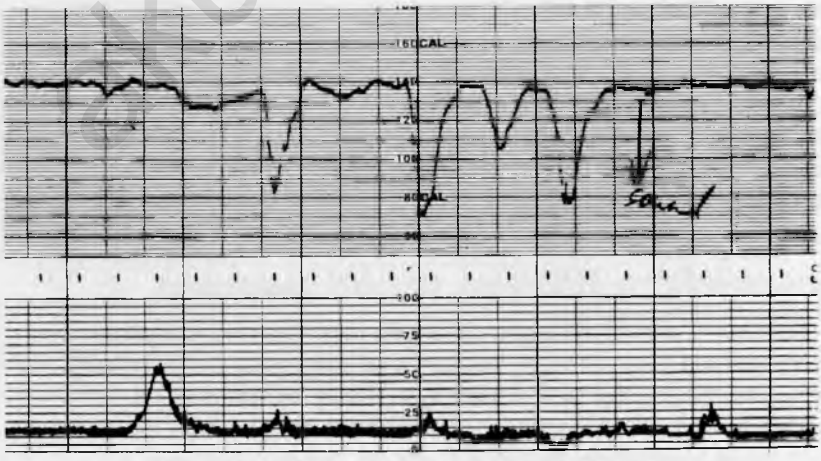

Figure 14-2. Clinical picture demands early delivery.

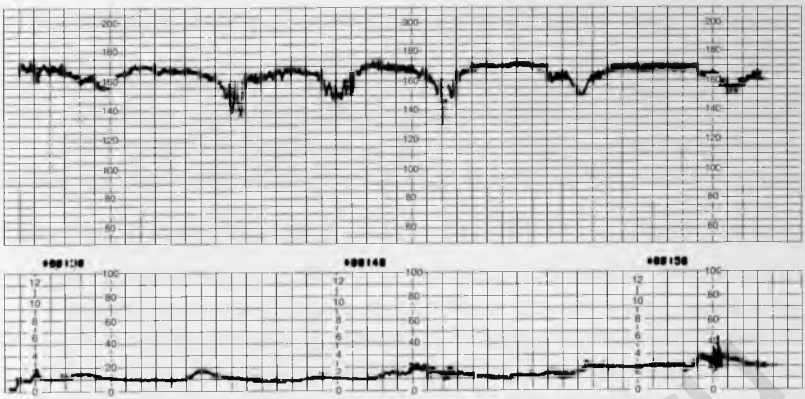

Figure 14-3. The FHR trace is ominous prompting immediate delivery.

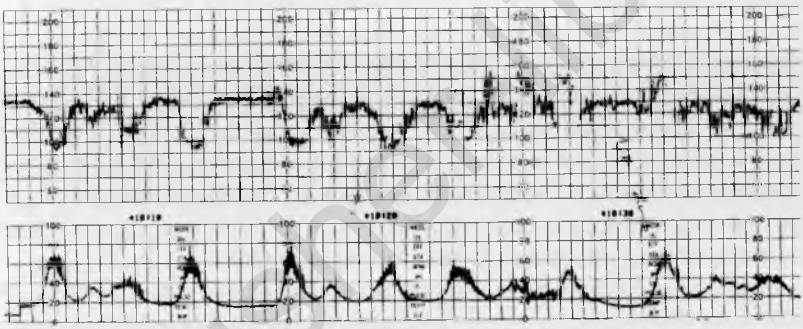

Figure 14-4. FHR in the second stage – reassuring.

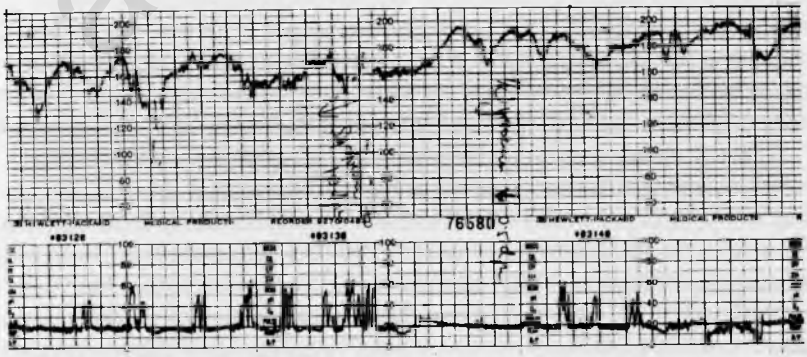

Figure 14-5. Changes in FHR – failure to progress in labour.

6. During, or soon after, an episode of prolonged bradycardia (see Fig. 10-4)
7. If spontaneous vaginal delivery is imminent or easy instrumental vaginal delivery is possible (see Fig. 12-14).

Following these principles will help to avoid unnecessary FBS, operative deliveries and fetal morbidity from undue delay in delivery.

ALTERNATIVES TO FETAL BLOOD SAMPLING FOR pH

Measurement of lactate in scalp blood is becoming more popular especially in Scandinavian countries because of the small blood samples required for analysis (5 µl instead of 35 µl for pH and BE). This leads to a failure rate for sampling of 2–4% compared with 15–18% with scalp sampling for pH. It also has an impact on the time needed for sampling. Caput formation does not alter the correlation between scalp and circulatory values. Lactate levels increase in the mother and the fetus in the second stage of labour throughout the period of bearing-down efforts – the lactate levels increase by 1 mmol/l every 30 min. Studies have shown a good correlation between the scalp and cord lactate when the sampling intervals have been close. The normal values for lactate vary based on the machine used (Lactate Pro or Accuport). Using the Lactate Pro machine, a value of <4.2 mmol/l is considered normal and a repeat performance needed only if the CTG abnormalities persist or get worse; a value of 4.2–4.8 as intermediate needing a repeat estimation within 30 minutes; and >4.8 is considered abnormal and an indication for immediate delivery.

In practice, FBS for pH or lactate may not be performed because the facilities or the expertise are not available, or because it is technically difficult. Alternative indirect methods are useful in this situation. A retrospective observation and correlation of the scalp blood pH to the presence or absence of accelerations at the time of FBS (Fig. 14-6) led to the *scalp stimulation test*.[18] When the scalp was stimulated by pinching with a tissue forceps, if an acceleration was present it was unlikely that the scalp blood pH was below 7.20.[19,20] In contrast, if there were no accelerations to such a stimulus then only about 50% had acidotic pH values (<7.20), whereas a significant proportion had preacidotic values (7.20–7.25) and others had normal values (Table 14-1).

Therefore, this test was useful in identifying those who are not at risk, although it was not good in predicting those who are likely to be acidotic. In centres where facilities do not exist for scalp FBS, such a test would be a useful adjunct in reducing the number of unnecessary caesarean sections for 'fetal distress', and in centres where facilities are available for FBS it will reduce the number of samples taken. Where there is a failure to obtain a sample during the FBS procedure, observation of an acceleration is very reassuring and the procedure can be discontinued.

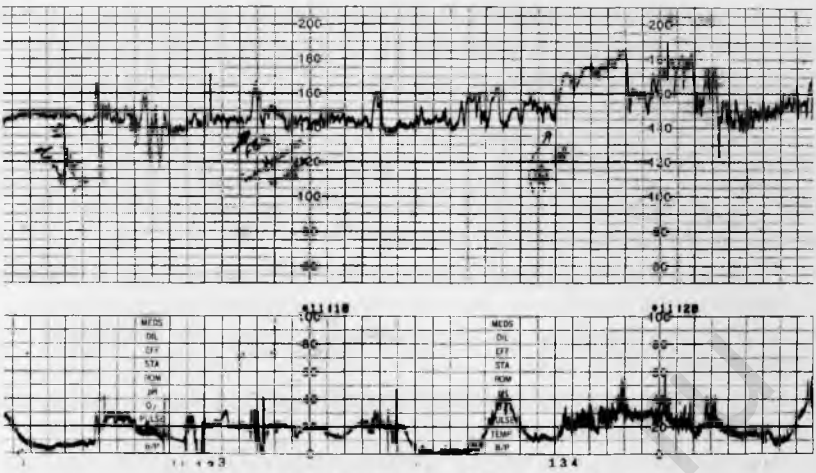

Figure 14-6. Acceleration at FBS – normal pH.

Table 14-1 Results of Scalp Stimulation Tests in Relation to Scalp Blood pH Values[20]

	Fetal Scalp Blood pH Values			
RESPONSE TO SCALP STIMULATION	**<7.20 (*n* = 82)**	**7.20–7.25 (*n* = 156)**	**>7.25 (*n* = 462)**	**TOTAL (*n* = 700)**
Positive response	1 (0.4%)	33 (12.7%)	226 (86.9%)	260
Negative response	40 (44.4%)	45 (50.0%)	5 (5.6%)	90
Total	41 (11.7%)	78 (22.3%)	231 (66%)	350

In the study described above, the case that recorded a positive response with an acidotic pH (see Table 14-1) showed respiratory acidosis, which is due to accumulation of CO_2, is not harmful to the fetus and is known to reverse itself once the FHR returns to normal. Careful observation of the characteristics of the FHR resulted in a fetus born with good Apgar scores. The latest NICE guideline also endorses the view that if FBS fails but digital stimulation provokes an acceleration then the situation needs to be reviewed.[1]

The Royal College of Obstetricians and Gynaecologists Study Group[21] and NICE have recommended that FBS facilities should be available in any hospital where electronic fetal monitoring is performed. However, clinicians who understand the clinical situation and the FHR pattern may make a decision without resorting to FBS and without an increase in caesarean section rate for fetal distress.[22] In many situations it may be wiser to proceed to delivery without wasting precious time. It has been shown that if the decision-to-delivery interval in situations of fetal distress is 35 min as opposed to 15 min then the admission rate to

the neonatal intensive care unit is doubled.[23] FBS is not always possible because facilities may not be available, or it may be difficult to perform owing to an undilated cervix or high head.[24,25] In these situations, decisions based on the CTG and the clinical situation remain critical.

POINTS TO PONDER

Although pH is a useful adjunct, the following points should be considered in clinical decision making:[26]

- Accelerations and normal baseline variability are hallmarks of fetal health.
- Accelerations without baseline variability should be considered suspicious.
- Periods of decreased baseline variability without decelerations may represent quiet fetal sleep.
- Hypoxic fetuses may have a normal baseline FHR of 110–160 bpm with no accelerations and baseline variability <5 bpm for >40 min (in the absence of adverse clinical parameters, observation for >90 min may be needed to recognize the abnormality).
- In the presence of baseline variability of <5 bpm, even shallow decelerations of <15 bpm are ominous in a non-reactive trace.
- Abruption, cord prolapse and scar rupture can cause acute hypoxia and should be suspected clinically (may give rise to prolonged decelerations/bradycardia).
- Fetal hypoxia and acidosis may develop faster with an abnormal trace when there is scanty, thick meconium, IUGR, intrauterine infection with pyrexia and/or pre- or post-term labour.
- In preterm fetuses (especially <34 weeks), hypoxia and acidosis can increase the likelihood of respiratory distress syndrome and may contribute to intraventricular haemorrhage, warranting early intervention in the presence of an abnormal trace.
- Hypoxia can be made worse by injudicious use of oxytocin, epidural analgesia and difficult operative deliveries.
- During labour, if decelerations are absent, asphyxia is unlikely although it cannot be completely excluded.
- Abnormal patterns may represent the effects of drugs, fetal anomaly, fetal injury or infection – not only hypoxia.

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FETAL ECG WAVEFORM ANALYSIS

Savvas Argyridis, Sabaratnam Arulkumaran

Intrapartum stillbirths are most distressing to the parents, staff and the institution. Despite decades of the use of CTG with FBS for pH or lactate measurement as an adjunct for intrapartum fetal surveillance, the incidence of intrapartum morbidity and mortality has remained relatively static in the UK. Based on this concern and static stillbirth rates, the Royal College of Obstetricians and Gynaecologists launched a programme 'Each baby counts' in 2015 to look at why we encounter term stillbirths especially in the intrapartum period and to learn lessons that can help us to avoid such events.[1] In 2016 NHS England launched a care bundle approach to reduce stillbirths and one of the components is training and assessment of intrapartum fetal surveillance.[2] This concern is not only in the UK but worldwide. The introduction of magnetic resonance imaging (MRI) that looks at specific lesions related to the types of hypoxia at term gestation suggests that pure intrapartum hypoxia may contribute up to 10% and intrapartum hypoxia superimposed on antenatal risks may contribute to about 15% of babies suffering from cerebral palsy.[3] We need to improve our methods of surveillance and actions based on them if we are to avoid intrapartum-related morbidity or mortality. A complementary approach of using fetal ECG waveform analysis in labour has been pursued by many centres in Scandinavian countries to reduce morbidity and mortality, and this is now gaining popularity in UK obstetric units. This chapter reviews fetal surveillance by fetal ECG–ST waveform analysis.

COMBINED CTG AND ECG WAVEFORM ANALYSIS

The presence of accelerations on the CTG suggests integrity of the 'somatic' nervous system and normal baseline variability suggests that of the 'autonomic' nervous system. Decelerations relate to the mechanisms that may cause hypoxia. In other words, the CTG assesses the integrity of the fetal nervous system, whilst the ST changes of T-wave or ST segment elevation and/or distortion reflect the strain to the heart. Hence a combination of both parameters gives information on hypoxic stress to the heart and the brain.

When all four features of the cardiotocograph (CTG) trace are normal, the chance of fetal acidosis is small. When all the features are abnormal,

just over 50% are noted to be acidotic.[4] Depending on the physiological reserve of each fetus, one may respond differently from another to the hypoxic insult. It has been observed that for 50% of appropriately grown term fetuses with clear amniotic fluid to get acidotic it takes 115 min with repetitive late decelerations, 145 min with repetitive variable decelerations and 185 min with a 'flat trace' (i.e., with reduced baseline variability).[5] This implies that some fetuses may become acidotic within a shorter period. This duration may be even shorter when there is reduced physiological reserve, i.e., in cases with infection, bleeding, post-term, growth restriction and in those with scanty thick meconium-stained fluid. Therefore, it becomes necessary to determine the fetal condition when there are abnormal FHR changes by fetal scalp blood sampling (FBS) or another appropriate alternate technology. This will help to identify those in need of delivery and to avoid unnecessary operative intervention when the fetus is coping well. However, the facilities and expertise are not available to perform FBS in many centres,[6,7] and its value is increasingly questioned.[8] It is also known that FBS is done when it is not warranted and is not done when it is needed.[9] In addition, the intermittent nature of the FBS readings makes it difficult to identify the optimal time to intervene without compromising the fetus, without increasing operative interventions. These issues have prompted more units to use ECG–ST waveform analysis for fetal surveillance in labour.

FETAL ECG–ST WAVEFORM ANALYSIS

The concept of electrocardiograph (ECG) waveform analysis relies on changes in the ST segment of the fetal ECG. These are related to metabolic events in the fetal myocardium during hypoxia. On the basis of experimental data, together with developments in bioengineering, advances have been made in the computerized analysis of ST waveform. The STAN-ST analyser (Neoventa, Gotenborg, Sweden) is a CTG machine that gives a CTG trace, and when the CTG is obtained using a scalp electrode on the fetal scalp and a skin reference electrode on the maternal thigh it provides ST waveform analysis.

Animal experimental data have demonstrated a catecholamine surge with hypoxic stress. This results in mobilization of stored myocardial glycogen. This important defence mechanism of adrenoceptor stimulation[10] brings about a shift of glucose and K^+ ions into the cells, which increases the T-wave amplitude (T/QRS ratio) (Fig. 15-1A and B). The detection of ST changes is computerized and the STAN equipment highlights any significant changes in the ST segment. The ST events detected may be (i) a *baseline rise* of the T/QRS ratio, (ii) an *episodic rise* of the T/QRS ratio, or (iii) a *biphasic ST* segment. Each fetus has a steady level of T/QRS ratio in early labour that could be identified from the initial recording. The rise in the T/QRS

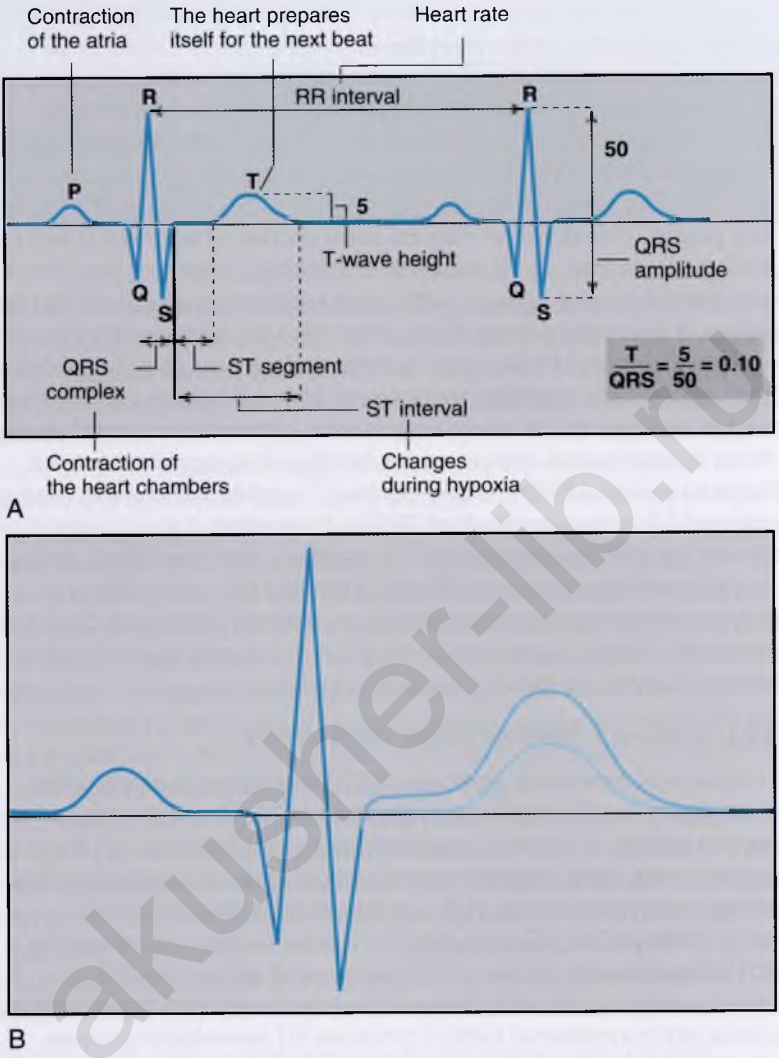

Figure 15-1. (A) Measurement of T/QRS ratio; (B) ST elevation and rise in the T wave.

ratio is calculated with reference to the lowest T/QRS ratio calculated over a period of 20 min in the previous 3 h period. This means that the equipment needs to be used for 20 min to calculate the baseline T/QRS ratio prior to major changes in heart rate or the ECG. In the immediate 20 min after start-up, and when there are poor discontinuous signals, manual data analysis is required. A *preterminal trace* that shows total lack of baseline variability and reactivity, with or without decelerations, and a prolonged deceleration warrants immediate delivery.

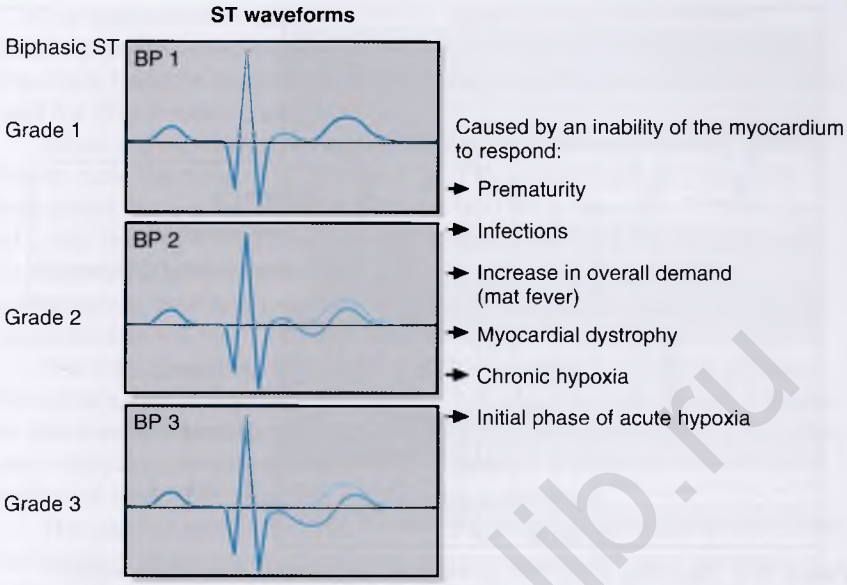

Figure 15-2. The different grades of biphasic events.

A steadily increasing T/QRS rise is termed a *baseline rise*, and if the ratio increases significantly and comes down within a brief period of a few minutes it is termed an *episodic rise*. The biphasic event refers to alteration of the ST segment where there is an initial rise and then a fall (Fig. 15-2). If the ST change is above the isoelectric line (the horizontal line constructed based on the resting level of the P wave), it is termed biphasic 1; if it cuts the isoelectric line it is called biphasic 2; and if it is below the isoelectric line it is called biphasic 3. Biphasic 2 and 3 are considered significant and are related to the electrical flow from the endocardium to the epicardium. Hence these changes may present themselves in the following situations: when the myocardium is thin (e.g., preterm fetuses), and when there is myocardial disease, infection and hypoxia.[11]

The FHR pattern, the ECG complex with T/QRS analysis, and uterine contractions are recorded onscreen on the same trace as shown in Figure 15-3.

The changes in the T/QRS ratio are highlighted as STAN events on the CTG trace if they are significant, and are recorded on a log event on the screen. The early studies in the 1980s showed promising results,[12,13] but inconsistent results from other studies[14] highlighted the need for computerization of ECG analysis and to take a rise in T/QRS from its own baseline levels instead of considering fixed values applicable to all fetuses. The ECG waveform analysis is used with

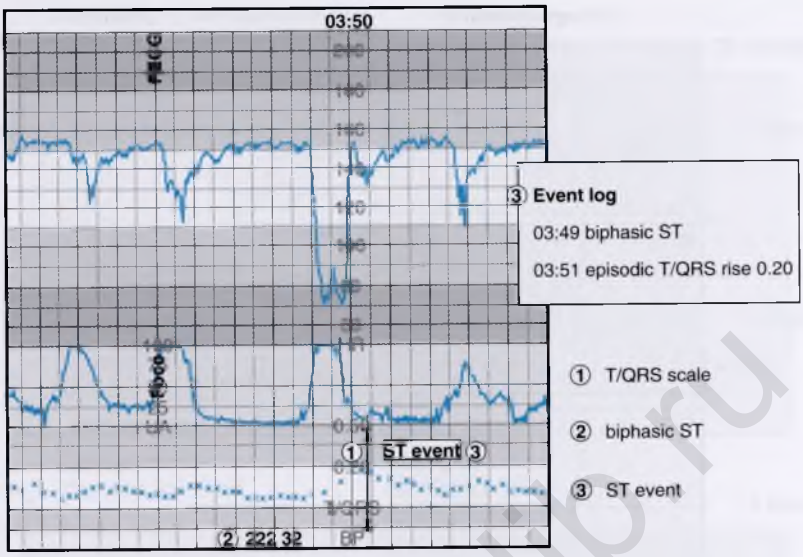

Figure 15-3. Recording of the FHR, contractions and computerized ECG waveform analysis of T/QRS plotted on the lower channel of the trace.

the CTG (Table 15-1), as STAN or ST events can present owing to mechanical stresses to the fetus.

The following guidelines apply for using the STAN technology.

This technology is applicable to pregnancies that are >36 weeks. Significant ST events, when judged along with the CTG, indicate the need for intervention. This could be delivery of the fetus or alleviation of a cause of abnormal FHR changes such as oxytocin overstimulation or maternal hypotension. If the ST event takes place in the active second stage of labour, immediate delivery is recommended. If the

Table 15-1. Decision-making Algorithm using Computerized Analysis of ECG Waveform with Visual Interpretation of the CTG

ST ANALYSIS	INTERMEDIARY CTG	ABNORMAL CTG	PRETERMINAL CTG
Episodic T/QRS rise	>0.15	>0.10	Immediate
Baseline T/QRS rise	>0.10	>0.05	Delivery
Biphasic ST	Continuous >5 min or >2 episodes of coupled biphasic 2 or biphasic 3	Continuous >2 min or >1 episode of coupled biphasic 2 or biphasic 3	

CTG is suspicious or abnormal in the second stage and if the ST analyser was started when the CTG was normal, or immediately after the trace became suspicious in the first stage of labour, then one could wait for 60 min before intervention.

When a STAN event is flagged up by the STAN equipment, one has to note the type of STAN event and the magnitude of change in that event (e.g., a baseline T/QRS rise of 0.06 or episodic T/QRS rise of 0.09). Having noted this, one has to interpret the CTG as abnormal or suspicious to decide on the action to be taken. If the CTG is pathological then action is warranted with a baseline rise of 0.06 or an episodic rise >0.10.

The CTG classification used for STAN analysis[15] is slightly different from that used in the NICE guidelines.[16] A baseline rate of 110–150 bpm is considered normal, early and simple variable decelerations <60 beats and <60 s are considered normal, and variable decelerations <60 s but with beat loss >60 beats are considered suspicious.[15]

The case shown in Figures 15-4–15-6 illustrates the use of the STAN technology. A primigravida needed augmentation for poor progress of labour. Variable decelerations are seen with oxytocin hyperstimulation. When oxytocin was stopped or reduced the contractions became less frequent, lasting for a shorter duration and no progress was being made. The ST analyser was used for continuous additional information. Even if the fetus showed FHR decelerations and an increase in baseline rate, the absence of STAN events (significant ECG changes) would give reassurance to continue with the oxytocin infusion with the aim of achieving a normal vaginal delivery.

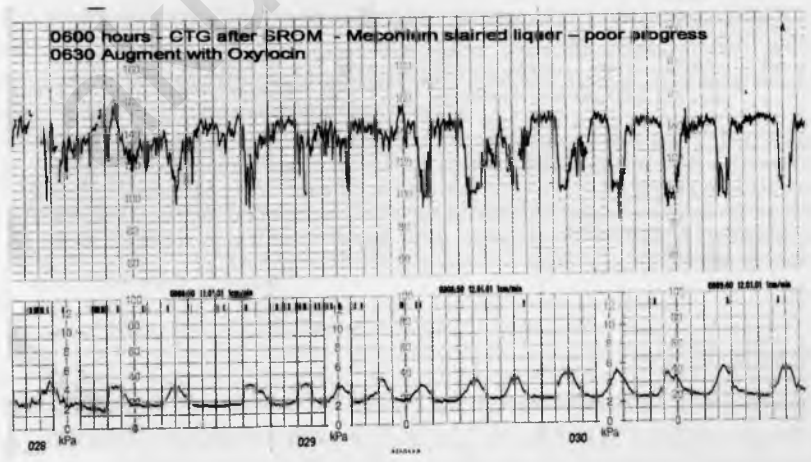

Figure 15-4. Cardiotocograph showing variable decelerations in early labour – related to hyperstimulation with oxytocin used for augmentation of labour.

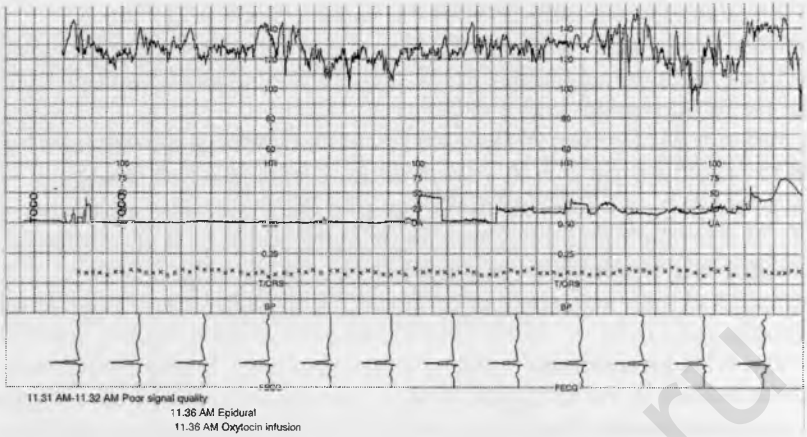

Figure 15-5. Oxytocin infusion was stopped. The cardiotocograph returned to normal and was reactive with no decelerations, but the contractions became less frequent. The ST analyser was connected and oxytocin infusion restarted.

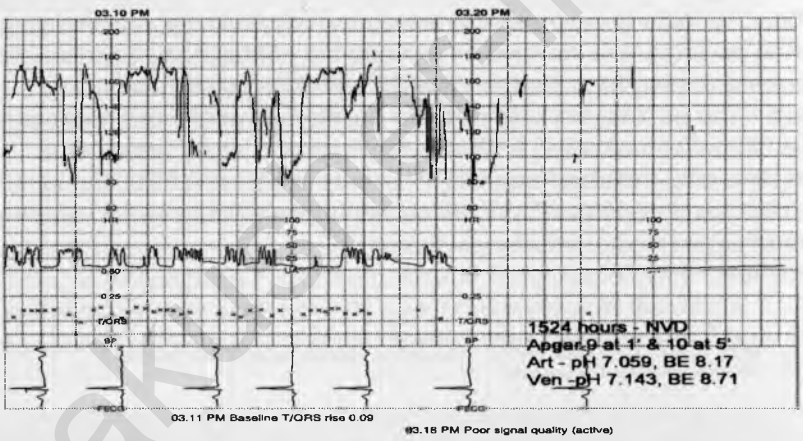

Figure 15-6. There was a gradual rise in the baseline rate following decelerations, followed by a reduction in baseline variability. No fetal blood sampling was done as there were no ST events. At 15.11 h there was an ST event of baseline rise indicating the need for delivery. A spontaneous delivery was imminent and the woman delivered spontaneously at 15.24 h. The baby had good Apgar scores and no evidence of metabolic acidosis.

CONCLUSION

Despite the enthusiasm shown for more and more units, there appears to be no convincing evidence that ECG waveform analysis delivers substantial benefits to the mother or baby. The latest Cochrane review on this subject included six randomized control trials (RCTs) including a

large American study and consisted of 26 446 women.[17] The meta-analysis did not show any reduction in caesarean delivery, low Apgar scores, incidence of metabolic acidosis or admission to special care baby unit. It did significantly reduce the FBS rates and instrumental vaginal delivery rates. The results may not be due to poor technology but rather to 'human factors'. In a large observational study of over 1500 cases there were 14 cases of hypoxic encephalopathy and 12 were due to violation of protocol.[18]

There was one case where there was progression from a normal CTG to a preterminal trace without ECG changes and hence the guidelines were modified, prompting intervention if the CTG shows repeated late or atypical variable decelerations for 1 hour in a trace with absent variability.[19] This decision was made based on existing literature, which shows a higher prevalence of fetal acidosis when there are late or atypical variable decelerations with absent variability in the CTG.[20]

The preliminary findings of the recently concluded 'INFANT study' that included 45 000 women compared computer interpretation with manual interpretation of CTG (conference papers). The 'human factor' of not recognizing the problem by incorporating the clinical situation and failure to take appropriate and timely action has been the key issue in why the computer-assisted CTG interpretation did not result in better outcome. This study, and the one previously mentioned on poor outcome with the use of ECG–ST waveform analysis, points to the need for constant ongoing education and training on how to interpret findings and to take appropriate and timely action. However, regular training by dedicated knowledgeable personnel alone is not adequate; this has to be backed up by regular assessment.[21] The training assessment has been included in the care bundle approach of NHS England[2] and we hope this bold step forwards would improve the situation. The need for such training and assessment and how it could be achieved are discussed in Chapter 17.

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MEDICO-LEGAL ISSUES WITH CTG AND CURRENT STRATEGIES TO REDUCE LITIGATION

Philip J. Steer

SHOULD WE BE USING CONTINUOUS ELECTRONIC MONITORING OF FETAL HEART RATE AND UTERINE CONTRACTIONS DURING LABOUR?

Electronic fetal monitoring (EFM), consisting of continuous recording of the fetal heart rate and uterine contractions (cardiotocograph, CTG), was first widely introduced in the 1960s. Already by 1970, an article in the *Nursing Mirror and Midwives Journal*[1] suggested that 'By means of electronic techniques it is now possible with a minimum of staff to obtain continuous and reliable information on the intrauterine pressure and the fetal heart rate during labour. We have reason to believe that these measures will greatly diminish the risk of hypoxic fetal brain damage during labour.' By the end of the 1970s, in the majority of maternity units in the United Kingdom, at least a third of labours were being monitored by CTG and in a few units it had already become universal.[2] However, when it became apparent that national rates of cerebral palsy were not falling, doubts began to creep in, and these were increased following the publication of the Dublin randomized controlled trial in 1985, which showed no clear evidence of benefit.[3] In 1986 in the *British Medical Journal*,[4] Prof Peter Howie wrote in relation to CTG that 'its use has been the subject of deep controversy ... regular intermittent auscultation may prove to be sufficient in low risk mothers'. The controversy deepened when the follow-up to the Dublin trial showed no reduction in cerebral palsy rates in the EFM group.[5] However, a case–control study by Gaffney et al of intrapartum care, cerebral palsy and perinatal death, published in 1994, reported that the fetal heart rate pattern was abnormal in 67% of labours ending in perinatal death, and in 23% of labours with subsequent cerebral palsy, compared with only 10% in controls.[6] This confirmed that there was a link between an abnormal fetal heart rate pattern and a poor outcome for the baby, both short term and long term. In 1995, two influential meta-analyses were published. The first, by Vintzileos et al,[7] of nine

trials reported that, although there was no overall reduction in perinatal mortality from the use of CTG monitoring, hypoxic deaths were reduced by almost 60%. The second, by Thacker et al,[8] of 12 trials reported a statistically significant 50% decrease in the incidence of neonatal seizures. These data have been sufficient to ensure that EFM continues to be the standard of care for intrapartum surveillance, and in developed countries it is considered mandatory in high-risk labours.[9]

IS THE ABNORMAL CTG ASSOCIATED WITH THE LATER DEVELOPMENT OF CEREBRAL PALSY?

The study by Gaffney et al[6] went further than simply analysing the proportion of abnormal fetal heart rate patterns in relation to outcome, and assessed the proportion of cases where there was a 'failure to respond to signs of fetal distress'. In cases of perinatal death this was 50%, and in cases of cerebral palsy 26%, compared with only 7% in cases with a normal outcome. Extrapolation of their regional data to the whole of the UK suggested that, in any given year, cerebral palsy associated with a failure to respond to evidence of fetal compromise would occur in 174 cases. Even if only half of such cases lead to successful litigation, the cost of monitoring failure becomes enormous because of the very large monetary value of individual settlements. Their findings amplified those of a 1990 publication of a review of 64 cases with a poor outcome from the records of the Medical Protection Society.[10] They found that, in 11 cases, CTG monitoring had not been carried out when it should have been, was technically unsatisfactory in a further six (27% overall), and the CTG trace was physically missing in 19 (30%). However CTG abnormalities were either not recognized or ignored in 14 cases (22%), and in only 14 cases (22%) was the abnormality noted and responded to appropriately. A further study by the same authors, of 41 cases from the records of the Association for the Victims of Medical Accidents (AVMA), concluded that 'inadequate fetal monitoring and insufficient supervision of junior doctors were implicated in a high proportion of accidents, some junior doctors and midwives cannot recognize abnormal CTG traces, and most receive inadequate training in CTG monitoring'.[11] Such was the concern about preventable injury to the fetus during labour that the UK government funded a national Confidential Enquiry into Stillbirths and Deaths in Infancy (CESDI). The sixth annual report, published in 1999,[12] detailed a study of 567 cases of poor outcome. Entirely appropriate care was found in only 28%. In a further 21%, substandard care was identified but proper care would not have changed the outcome. Worryingly, substandard care that could possibly have affected the outcome was identified in 28%, and was likely to have changed the outcome in a further 22%. Therefore, in fully half of cases, the outcome might have been changed by correct management. The authors of the report

commented that 'fetal surveillance problems were the commonest cause (of problems in labour), with CTG interpretation ... the most frequent criticism'.[12]

THE MEDICO-LEGAL CRISIS

Before the 1980s, litigation against doctors alleging malpractice was rare. When I first qualified in 1971, my annual subscription to the Medical Defence Union was £5 for a year's cover. However, this started to rise in the 1980s and by 1987 it had risen to over £300 per annum. Over the next 3 years, the steady rise of the previous 15 years changed to a dramatic acceleration, so that by 1990 my annual subscription was £1400. As a consequence of the resulting erosion of annual salaries, especially in high-risk specialties such as obstetrics, health authorities in 1990 took over financial responsibility for negligence attributable to medical and dental staff employed in the National Health Service (NHS). In 1995, the NHS Litigation Authority (NHSLA) was set up to administer a national scheme of indemnity for medical and associated staffs. They set up a system called the 'Clinical Negligence Scheme for Trusts' (CNST), which linked the cover premiums charged to individual care groups (such as Hospital Trusts) to the development of good clinical care guidelines within the Trust. Unfortunately it eventually became apparent that good-quality guidelines had no discernible impact on the rate of medical legal claims, and in 2014 the CNST system was abandoned in favour of premiums based on the historical rate of claims attributed to each individual Trust.

The NHSLA has published its annual accounts since 1998, and these show that, from an annual pay-out of £70 million in 1998, the amounts paid out have risen in a linear fashion (Fig. 16-1), so that in

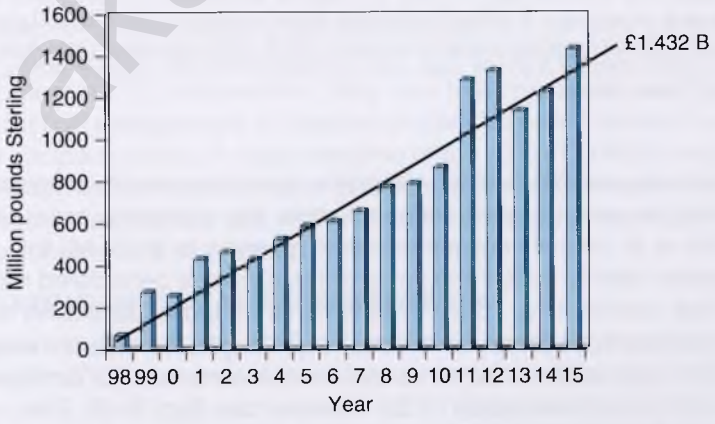

Figure 16-1. Total expenditure of the NHS Litigation Authority. *(The data have been extracted by the chapter author from the annual reports of the National Health Service Litigation Authority; the website containing these reports is http://www.nhsla.com/home.)*

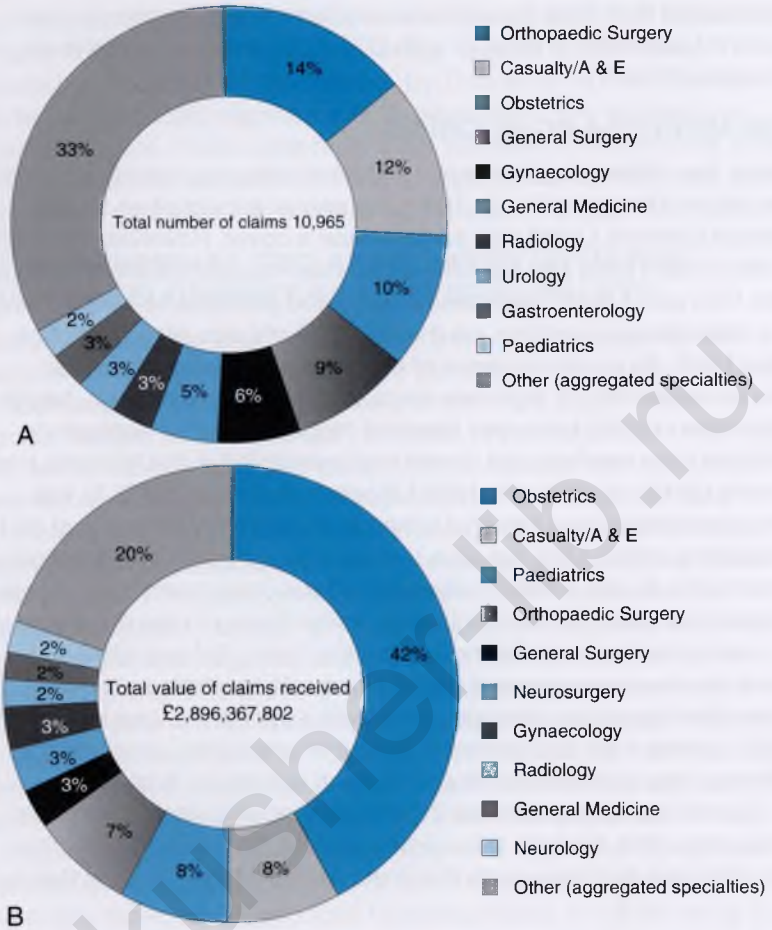

Figure 16-2. (A) Number of clinical negligence claims received in 2015–16 by specialty; (B) value of clinical negligence claims received in 2015–16 by specialty. *(From National Health Service Litigation Authority. Report and accounts 2015/2016. London: NHSLA; 2016:29,*[13] *with permission.)*

the financial year 2015–16 £1.432 billion was disbursed to claimants and their lawyers. Its latest accounts show that obstetrics accounts for 10% of all clinical negligence claims received in 2015–16; this proportion rises to 42% if the value of the claims is considered rather than their number (Fig. 16-2).[13] Settlements for individual claims have now reached more than £10 million.[14] In 2015–16, the annual value of obstetrics pay-outs to claimants and their lawyers was £578 million, equivalent to approximately £1.6 million per day (Fig. 16-3). This amounts to more than £800 for clinical negligence cover for every live birth in England, almost a fifth of all spending on maternity care.[15]

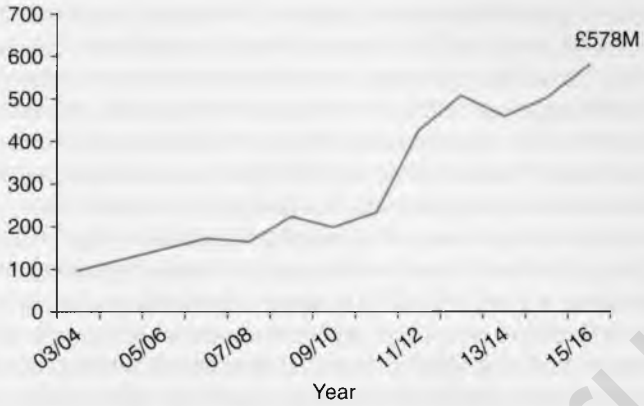

Figure 16-3. Annual value of NHSLA obstetrics pay-outs per year. *(The data have been extracted by the chapter author from the annual reports of the National Health Service Litigation Authority; the website containing these reports is http://www.nhsla.com/home.)*

Recent publications by the NHSLA confirmed that misinterpretation of CTG traces remains the most frequent example of negligence resulting in litigation and settlements. Its 2009 study of 100 stillbirth claims[16] reported that this occurred in 34 of these cases. Importantly, of the 39 clinicians involved in the 34 cases, 25 were midwives, eight were registrars or senior registrars (trainees) and only four were consultants (called in the USA 'attendings', short for attending physicians). In two cases, the identity of the clinician remained unknown! This emphasizes a situation in which the clinicians usually involved in the management of labour are the most junior of the team, especially at night and at weekends, when perinatal mortality is known to be higher.[17,18] This contrasts with most other branches of medicine, where the level of practitioner is closely matched to the complexity of the case. It leads to a paradox whereby once practitioners become experienced, and therefore presumably more competent in CTG interpretation, they tend to be promoted away from the practical management of intrapartum care into administration and other clinical activities. A major headline of the 2012–13 NHSLA report was 'a key finding from the report has demonstrated the need to focus on improving the detection and response to a deteriorating fetal heart rate through better fetal monitoring'.[19]

TRAINING IN CTG INTERPRETATION

Despite the acknowledged importance of correct CTG interpretation during labour, policies to improve performance remain in their infancy. In one important study published in 2001, Young et al[20] reported that a study of low Apgar scores at the North Staffs hospital in the UK revealed that 74% were associated with suboptimal care. They took

the important step of introducing regular low Apgar feedback meetings involving labour ward staff, and found that the incidence of suboptimal care dropped to 23%. However, with time the proportion of suboptimal care increased again to 32%, at which point they took the radical step of making CTG training compulsory for labour ward staff. The subsequent rate of suboptimal care fell to 9%. In most occupations, compulsory training for a key activity would not be seen as revolutionary; however, even in developed countries, most CTG training is based around a few formal lectures, self-directed learning and ad hoc mentoring. In the NHS, CTG courses are rarely run as part of the maternity unit official training programme, instead being provided by charities such as Baby Lifeline (http://babylifelinetraining.org.uk/home/courses/ctg-masterclass/). Web-based electronic systems for training are available, provided for example by K2 medical systems (https://training.k2ms.com/Secure/Logon.aspx?ReturnUrl=%2fdefault.aspx) and the RCOG (http://www.e-lfh.org.uk/programmes/electronic-fetal-monitoring). However, such systems are not based upon assessment of individual requirements, nor are the self-assessment components monitored by the supervising authorities in the NHS. It is a crucial weakness of the system that in most maternity units there is no systematic assessment of birth attendants' ability to interpret CTGs. This contrasts with the situation in, for example, aviation – where pilots' abilities are tested formally on a 6-monthly basis. Anyone who fails the assessment is taken out of service and retrained until they reach the appropriate standard (which is, for example, 100% success at taking off and landing without error or mishap). In one detailed study of the benefits of a computer-assisted teaching programme for intrapartum fetal monitoring, the success rate of medical staff in the interpretation of CTGs rose from about 70% before training to about 85% afterwards; 100% correct interpretation remained elusive, however. Worryingly, the success rate of midwives was originally only about 50%, and rose to only about 70% after training.[21]

THE MEDICO-LEGAL RISK ASSOCIATED WITH THE USE OF OXYTOCIN INFUSION TO AUGMENT LABOUR WHEN THE CTG IS NON-REASSURING OR ABNORMAL/PATHOLOGICAL

In the 1960s and 1970s, O'Driscoll, Master at a National Maternity Hospital in Dublin, promoted a concept that he dubbed 'the active management of labour',[22,23] which required the use of high-dose oxytocin infusion in more than 50% of nulliparous women in labour. He subsequently claimed that in his unit it was preventing the rise in caesarean section rates that have been seen widely throughout the developed world over the last 50 years.[24] Despite editorials in

the *British Medical Journal*[25,26] and *British Journal of Obstetrics and Gynaecology*[27] warning against the use of active management because of the dangers of uterine hyperstimulation, the policy has been widely adopted despite a Cochrane review of 14 trials including 8033 women, which found no significant effect on the caesarean section rate and a reduction in the duration of labour of only 1.3 hours.[28] Importantly, the use of oxytocin has been cited as a major component in disciplinary and legal actions in relation to intrapartum care. For example, Jonsson et al reported in 2007 that the 'injudicious use of oxytocin' occurred in more than two-thirds of 60 cases of intrapartum care resulting in disciplinary action, and was the primary reason for disciplinary action in one-third.[29] In a further study of 177 babies with severe birth asphyxia due to malpractice/poor supervision, the incautious use of oxytocin was implicated in 71%.[30] The latest recommendations of the National Institute for Health and Care Excellence in the United Kingdom[9] now state 'do not routinely offer the package known as active management of labour' (recommendation 1.12.10) and 'for all women with confirmed delay in the established first stage of labour: explain to her that using oxytocin after spontaneous or artificial rupture of the membranes will bring forward the time of birth but will not influence the mode of birth or other outcomes' (recommendation 1.12.18). They highlight that if the CTG is not entirely normal then any oxytocin infusion should be stopped immediately (not reduced, and not restarted if the CTG remains non-reassuring or pathological). Unfortunately, my personal experience of litigation cases shows that this instruction is widely ignored, and this can lead to major medico-legal settlements (see, for example, case two in the online NHSLA publication '10 years of maternity claims'[31]). A 2015 meta-analysis has reported that discontinuing oxytocin after the active phase of labour is established significantly decreases rates of caesarean section (OR 0.51, 95% CI 0.35, 0.74) as well as those of uterine hyperstimulation (OR 0.33, 95% CI 0.19, 0.58),[32] so there seems every reason to limit the use of oxytocin as far as possible, for both clinical and medico-legal reasons.

THE PRINCIPLES OF LEGAL PRACTICE AS APPLIED TO MEDICINE

In order for a medical practitioner to be found guilty of medical negligence, two aspects of each case have to be considered separately. Firstly, was there a breach of duty? Every doctor has a duty of care to their patient that requires them to treat the patient according to the accepted standards of the time. Did the doctor's practice meet those accepted standards? Secondly, causation needs to be established. In other words, was there a direct link between the

failure to practice according to accepted standards, and the outcome for which compensation is being claimed? Even if the doctor's practice fell well below an acceptable standard, compensation will not be paid if there is no clear link between the unacceptable care and an adverse outcome.

In English law, negligence was initially defined by reference to a hypothetical reasonable person, a concept first codified in Roman law as the 'bonus paterfamilias' (good family father). In Victorian times such a reasonable but average person was sometimes referred to as the 'man on the Clapham omnibus'. It is important to appreciate that the standards applied in clinical practice are those of the average clinician appropriate for the task, and not those of the super specialist. Acceptable practice is commonly a range of options rather than a single policy, which is why it is so hard to write general guidelines. The grounds by which breach of duty should be judged were particularly well defined in the 1957 case of *Bolam* v. *Friern Hospital Management Committee*.[33] This related to a patient suffering from a mental illness who sustained a fracture during the use of electroconvulsive therapy. There were, according to expert witnesses, two conflicting bodies of medical opinion, one of which favoured the routine use of muscle-relaxant drugs in order to prevent fracture, whereas the other felt that the risk from the relaxant itself was too high for routine use so that it should be used only when there were specific indications. The judge directed the jury as follows:

> A doctor is not negligent, if he is acting in accordance with a practice accepted as proper by a responsible body of medical men skilled in that particular art, merely because there is a body of such opinion that takes a contrary view.

In other words, as long as a reasonable number of doctors would do what you did in a particular circumstance, your actions are not negligent.

A particularly important obstetric case occurred in 1981, that of *Whitehouse* v. *Jordan*.[34] This related to the attempted delivery of a baby who had sustained 'brain damage'. Following a prolonged labour, the obstetrician conducted a trial of forceps delivery but after six pulls there was no movement and so he abandoned the procedure and performed a caesarean section. It was alleged that he was negligent because he had considered that forceps could be used to deliver the baby when in fact the outcome showed that this was not possible. Although the primary court held the obstetrician to be negligent, this decision was reversed by the Court of Appeal and the reversal was confirmed by the House of Lords (which has since become the UK Supreme Court). They concluded that 'an error of clinical judgment by a doctor is not the same thing as negligence. The test of an error of judgment in such a case is the standard of care of the ordinary skilled

man exercising and professing to have a particular skill. Accordingly an error of judgment might or might not have been negligent.' The obstetrician, Mr Joe Jordan, was found not guilty of negligence. What this means is that, although a course of action might be seen to have been unequivocally wrong in retrospect (in this case the baby would probably have survived intact if the caesarean had been done without a trial of forceps), it can often be an acceptable (and even the most appropriate) action when carried out without knowledge of the eventual outcome. Medical practitioners are not expected to be able to foresee the future, only to take reasonable action.

Another legal landmark occurred in 1997, with the case of *Bolitho* v. *City and Hackney Health Authority*.[35] This again was not an obstetric case but related to a 2-year-old boy with a past history of hospital treatment for croup. Following admission to hospital, he had difficulty in breathing and turned white on two occasions; unfortunately the two doctors who were called failed to attend (although they could have done if they had made it an appropriate priority). Following apparent recovery the boy later suffered total respiratory failure and cardiac arrest resulting in severe brain damage and he subsequently died. Although the lower court and the Court of Appeal decided that because a responsible body of professional opinion 'espoused by distinguished and truthful experts' held that even if they had attended, they would not necessarily have intubated the child and thereby prevented the cardiac arrest, the House of Lords held that a doctor could be held liable for negligence 'despite a body of professional opinion sanctioning his conduct where it had not been demonstrated to the judges' satisfaction that the body of opinion relied on was reasonable or responsible'. They went on to say that 'if it could be demonstrated that professional opinion was not capable of withstanding logical analysis, the judge was entitled to hold that the body of opinion was not reasonable or responsible'. However, in the case of Bolitho, the Court held that the body of opinion was in fact reasonable and therefore there was, in this particular case, no evidence of negligence. Nonetheless, the Court had established that not only must a course of action be that which would be supported by a reasonable body of medical opinion, but it must also stand up to logical analysis. You cannot escape liability for doing something unreasonable just because others would do the same.

The most important recent case is probably that of *Montgomery* v. *Lanarkshire Health Board (Scotland)*, 2015 SC 11.[36] This was again an obstetric case. Nadine Montgomery was a diabetic woman of short stature and her first baby was predicted by ultrasound to weigh more than 4 kg. She was not offered a caesarean section but instead labour was induced. Unfortunately she was not told about the potential risk of

shoulder dystocia, and when this occurred even more unfortunately the baby experienced severe asphyxia and later developed cerebral palsy. Although two lower courts rejected a claim of negligence, accepting that many obstetricians would also have encouraged vaginal birth, the UK Supreme Court upheld an appeal in favour of the claimant, affirming that women have a right to be told about any material risks in order to make an autonomous decision about how they wished to give birth. The level of risk to be disclosed is not judged on what the doctors think is important, but instead on the importance attached to it by the patient. Thus, obstetricians who do not advise their patients of any material risk are likely to be judged negligent. When there are options available, the patient should decide which they prefer, and not the doctor. Moreover, if a caesarean is a reasonable option, even though it may not be preferable, women are entitled to choose it.

A procedural point is that it is the courts that decide negligence, and obstetric experts who give evidence should avoid making such judgements. Instead, their evidence should indicate whether the care delivered to the patient was of a proper professional standard, and if the care fell below this standard then whether on the balance of probability (in civil cases) the deficiency in care led to the adverse outcome. 'On the balance of probability' means a likelihood of 51% or more. In recent years there has been increasing recourse to the use of the criminal law when the care has been particularly substandard. In such situations, obstetricians and midwives may be accused of manslaughter. This is quite difficult to define, but essentially means that the care given was reckless, or so grossly deficient that the adverse outcome ought to have been foreseeable (the final judgement is usually made by a jury). Examples might include doctors who refuse to attend despite an obvious duty to do so, or who are drunk (intoxicated by alcohol), are under the influence of intoxicating drugs, or act with a blatant disregard for the safety of the patient. In such cases, the criterion for causation is much higher, being the criminal standard of 'beyond any reasonable doubt'.

THE WAY FORWARD

The account above has highlighted the potential value of continuous intrapartum electronic fetal monitoring. However, it has proved difficult to realize its full value in practice. Rates of cerebral palsy associated with intrapartum asphyxia have not shown any significant decline, while litigation rates alleging malpractice continue to increase. The proportion of legal cases involving misinterpretation of CTG traces has remained stubbornly high over the last three decades. Part of the problem in this regard is the constant turnover of staff, with experienced clinicians being promoted away from the labour ward,

where they are regularly being replaced by less experienced trainees. In an attempt to deal with this problem, several groups have developed a computerized approach to CTG interpretation. The Plymouth group has been led by obstetrician Prof Keith Greene and bioengineer Robert Keith; they first reported their system combining the use of neural networks with rule-based algorithms in 1994[37–39] and by 1995 published a study showing that the system could work as well as the best of 17 UK experts at the detection of pathological fetal heart rate patterns.[40] They subsequently set up a prospective randomized trial of the technology,[41] which took a long time to fund and carry out because of the very large numbers of participants needed to have any expectation of showing an effect on significant outcomes such as perinatal mortality and hypoxic ischaemic encephalopathy. In the event, the trial (the INFANT trial) lasted for 5 years and included over 46 000 births in 24 different maternity units. Perhaps the most striking finding was that, in the study group (which was relatively high risk because it included only women with an indication for electronic fetal monitoring), there were only three intrapartum stillbirths (0.07 per thousand) and 10 neonatal deaths (0.2 per thousand) – an overall rate of only 0.27 per thousand. This was much lower than predicted. By comparison a study of intrapartum stillbirths and neonatal deaths in Finland in a low-risk population of 267 066 births published in March 2016 reported an intrapartum mortality of 2.7 per thousand newborns and an early neonatal mortality rate of 0.2 per thousand,[42] which is near what was predicted for the control group in the INFANT trial. The results of the comparison of the decision support group and the control group are as yet unpublished but in a presentation to the British Maternal and Fetal Medicine Society in April 2016 the lead researcher, Peter Brocklehurst, reported that computerised interpretation had no effect on outcome. However, in a sub-analysis of 71 cases of adverse outcome, it was found that 38% were associated with substandard care, mainly failure to appreciate the importance of additional risk factors such as fetal growth restriction, meconium staining of the amniotic fluid, maternal pyrexia, prolonged labour, and uterine hyperstimulation with oxytocin; and delays in responding to a recognised CTG abnormality. Perhaps what the INFANT study shows most strikingly is the protective power of being in a clinical trial, with all the heightened surveillance that this involves.

Another computerized approach has been developed by the Sis-Porto group in Portugal, by Ayres-de-Campos and colleagues.[43] As with the INFANT system, its complexity meant that it required 19 years to develop it. Called Ominview-Sis-Porto, an off-line analysis of its performance was comparable with that of three experienced clinicians, in terms of both classification of the fetal heart rate pattern[44] and

prediction of newborn umbilical artery blood pH.[45] The developers have recently performed a prospective randomized controlled trial similar to that of the INFANT system although with much smaller numbers[46] and the results although not yet published, have been reported at the European Congress on Intrapartum care in Porto May 2015 and computerised interpretation made no difference to the outcome.

It would appear that part of the problem with CTG interpretation has been the emphasis on pattern recognition, without sufficient emphasis on the interpretation of heart rate changes in the context of fetal pathophysiology. Intrapartum factors such as the duration of labour, maternal (and therefore fetal) fever, infection, meconium staining of the amniotic fluid, and mechanical forces/trauma play an equal (and sometimes more important) role in making a correct assessment of fetal condition and the need for expedited delivery. There needs to be an understanding that hypoxia/acidosis is the major causative factor in fewer than 50% of babies who are born in poor condition.[47] Training of intrapartum attendants in CTG interpretation and the management of labour is intermittent, fragmented and has no agreed curriculum, and effective assessment tools to determine the competence of individual practitioners have not yet been developed. A call to optimize and enforce training and introduce compulsory assessment of skills in the interpretation of intrapartum CTG traces has been published in 2016[48] – only time will tell whether the call will be heeded.

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COMPETENCY TESTING IN CTG INTERPRETATION PRIOR TO PRACTICE

Edwin Chandraharan

Cardiotocograph (CTG) interpretation requires 'pattern recognition' skills. Scientific studies have shown that such pattern recognition is fraught with significant inter- and intraobserver variability. Errors in CTG interpretation can lead to both maternal and fetal complications because 'over-classification' may lead to unnecessary intrapartum operative interventions (emergency caesarean sections, operative vaginal births and fetal scalp blood sampling) and their resultant complications. Conversely, under-classification (i.e., missing significant changes suggestive of intrapartum fetal hypoxia on the CTG trace) can lead to poor perinatal outcomes such as hypoxic ischaemic encephalopathy (HIE) and its long-term sequelae as well as intrapartum stillbirths or early neonatal deaths. Confidential enquiries into stillbirths have highlighted that substandard care, especially CTG misinterpretation, contributes to approximately 50% of intrapartum-related stillbirths.[1] The Chief Medical Officer's Report on '500 missed opportunities' in 2006 also highlighted the risks associated with CTG misinterpretation.[2] Moreover, the NHS Litigation Authority (NHSLA) report 'Study of stillbirth claims' in 2009 highlighted that errors in CTG interpretation were associated with 34% of all stillbirth claims.[3] The NHSLA report 'Ten years of maternity claims' has highlighted CTG misinterpretation as the key factor in cerebral palsy and stillbirth claims.[4]

Clinical tests that are based on pattern recognition (e.g., mammograms) usually involve some form of 'competency testing' of staff to reduce errors. Unfortunately, such a comprehensive, mandatory testing of CTG interpretation has not been enforced for CTG interpretation, despite its known pitfalls, which include a 60% false-positive rate.[5,6] St George's Hospital Maternity Unit in London has pioneered the development and introduction of the first mandatory competency testing of midwives and obstetricians on CTG interpretation in the UK with minimum criteria (85% pass mark) from 2005. This was part of a comprehensive 'Policy on achieving competency in intrapartum fetal monitoring', helping staff who fail to

achieve the required pass mark so that they are well supported whilst patient safety is ensured. This policy includes mandatory requirements for annual CTG updates as well as weekly attendance at CTG meetings to continuously update the knowledge and skills whilst providing mentorship for those who fail the test.

The St George's CTG test not only involves questions on 'pattern recognition' but also has sections on 'understanding fetal pathophysiology' as well as 'situational awareness' (see below). This is because failure to appreciate the wider clinical picture (e.g., presence of meconium staining of amniotic fluid, clinical chorioamnionitis or poor rate of progress of labour with injudicious use of oxytocin infusion) may lead to poor fetal outcomes, irrespective of patterns observed on the CTG trace.[6] This test has now been implemented in other hospitals as well.

This chapter aims to provide examples of intrapartum outcomes after implementation of intense physiology-based CTG training with mandatory testing of competency.

PHILOSOPHY

Intrapartum care should be safe, rewarding and provide a positive experience for women, babies and their families. Avoidable harm, including unnecessary operative interventions during labour and intrapartum hypoxic injury, should be eliminated in total. Midwives and obstetricians are caring professionals who aim to provide the best care for their patients at all times. Poor outcomes associated with CTG misinterpretation are predominantly due to system failures due to national guidelines[7] promoting pattern recognition without considering fetal response to hypoxic or mechanical stress, to labour ward curricula not incorporating physiology-based CTG interpretation, to the blind use of 'CTG stickers' with arbitrary cut-offs for baseline fetal heart rate during labour without considering the rise in the baseline heart rate of the individual fetus secondary to hypoxic stress, and to absence of a robust national mechanism for mandatory testing of competency of midwives and obstetricians prior to CTG interpretation in the labour ward.

The St George's CTG test has been based on the philosophy that intrapartum care should be provided by competent staff who have appropriate training and support to deliver safe care. Hence, the conduct of the CTG test should be viewed positively, to ensure that all staff should *pass* the test to demonstrate their competency and, therefore, the aim of the CTG test is to equip staff with knowledge and skills to avoid errors in CTG interpretation. Hence, all staff members have an intense training in fetal physiology, fetal response to intrapartum hypoxic stress, types of intrapartum hypoxia and the wider

clinical picture prior to attempting the test. All new personnel have an extra day of CTG training as part of their induction into the labour ward and their competency on CTG interpretation is tested prior to commencing their duties in the labour ward.

Questions on *pattern recognition* are aimed at helping rational application of national guidelines in clinical practice because the guidelines are based on recognition of certain patterns to enable classification of the CTG traces into different categories[7] (which show significant variation amongst various bodies such as ACOG, NICE and FIGO).

During the intense physiology-based CTG training, it is emphasized to staff that, although the arbitrary cut-offs recommended by guidelines[7] (e.g., atypical variable decelerations for 50% of uterine contractions for 30 minutes) are useful for classification of CTG traces into different categories, one needs to consider the fetal reserve (e.g., a fetus with intrauterine growth restriction may not tolerate 30 minutes of intense hypoxic stress secondary to umbilical cord compression), the type of intrapartum hypoxia, the fetal response to ongoing stress and the wider clinical picture (presence of meconium, evidence of clinical chorioamnionitis and the rate of progress of labour) prior to making management decisions. This is because pattern recognition alone has been shown to be ineffective in predicting cerebral palsy or neonatal outcomes.[8,9] In addition, increasing the caesarean section rate over a 30-year period has not been associated with any demonstrable reduction in cerebral palsy.[10]

Questions on *fetal pathophysiology* are aimed at testing clinicians' understanding of fetal physiology as well as the mechanisms (i.e., baroreceptor and chemoreceptor) underlying the decelerations[11] observed on the CTG trace and the function of the central nervous system. This is essential to highlight the fact that a fetus may be normal (i.e., stable baseline and reassuring variability) even if the CTG trace is pathological and, conversely, a fetus may be compromised even though the CTG trace is classified as normal (e.g., clinical chorioamnionitis with an increase in baseline fetal heart rate from 110 bpm to 155 bpm). In addition, questions on types of intrapartum hypoxia enable delegates to exclude pre-existing insult (e.g., chronic hypoxic pattern or loss of cycling) as well as to institute interventions based on urgency (e.g., subacute hypoxic pattern).

Questions on *situational awareness* are aimed at appreciating the 'wider clinical picture' such as the use of oxytocin-induced CTG changes, presence of meconium and other intrapartum risk factors for fetal hypoxic injury (e.g., diabetic ketoacidosis, uterine rupture) as well as erroneous monitoring of the maternal heart rate during labour.[12] Clinicians are made aware that the CTG trace is just *one* of

the key aspects of intrapartum care and it should be interpreted only in the context of the wider clinical picture. This includes avoidance of erroneous recording of the maternal heart rate as the fetal heart rate.[13] Alternative pathways of brain damage such as inflammation and non-hypoxic injuries may occur if labour is allowed to continue despite subtle CTG changes. The presence of intrauterine growth restriction may reduce the physiological reserves to withstand the same hypoxic stress compared with a term well-grown fetus and the magnitude of changes observed on the CTG trace may also be much smaller.

DOES THE INTENSE, PHYSIOLOGY-BASED CTG TRAINING HELP TRANSFER KNOWLEDGE?

Recent studies have suggested that CTG training based on understanding fetal physiology helps in improving knowledge and decision making amongst midwives and obstetricians.[14] A recent study that reported data on pre- and post-tests carried out before and after intense, physiology-based testing amongst 810 midwives and obstetricians in 15 centres in the UK reported a significant improvement in knowledge after training (Fig. 17-1).[15] Most notably, less than half of the delegates correctly answered basic questions on NICE guidelines during the pre-test, which illustrates the difficulties in pattern recognition promoted by national guidelines and resultant inter- and intraobserver variability in CTG interpretation.

DOES MANDATORY CTG TESTING HELP IMPROVE PERINATAL OUTCOMES?

St George's maternity data suggest that, after the introduction of an intense, physiology-based CTG training, the use of fetal ECG (STAN) and mandatory competency testing, the rate of emergency section had reduced by almost 50% with a concomitant reduction in instrumental vaginal births (Fig. 17-2). The current emergency caesarean section

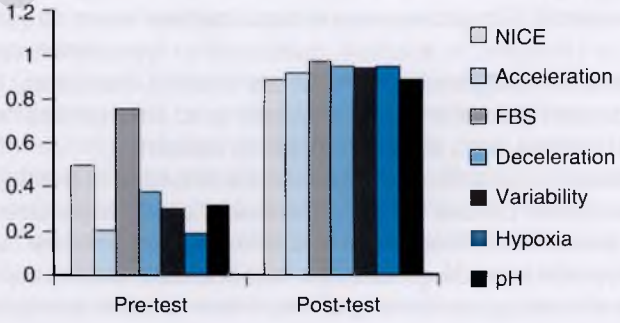

Figure 17-1. Improvement of knowledge after physiology-based training

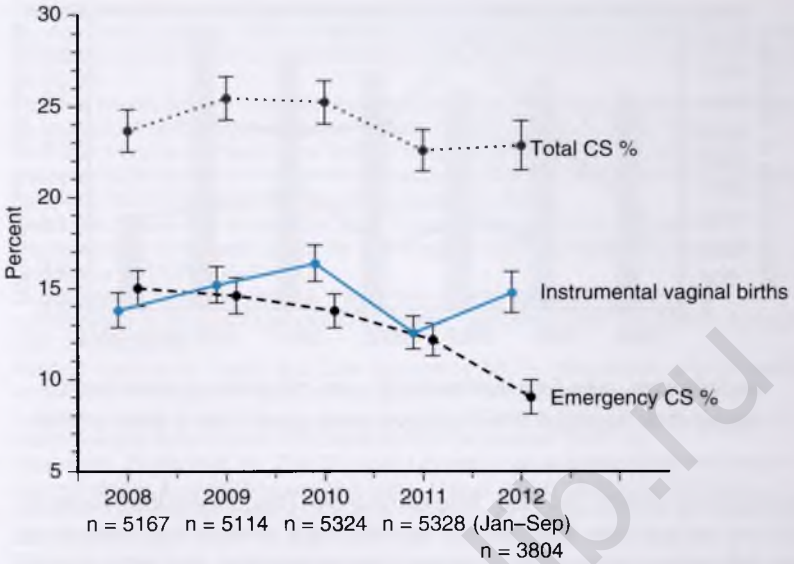

Figure 17-2. Reduction in the incidence of emergency caesarean section after the introduction of mandatory competency testing in 2009. (Error bars show 95% confidence interval.)

rate (7.8%) is approximately half the caesarean section rate of other teaching hospitals in London with similar complexity of cases. These data suggest that deeper understanding of the CTG and fetal pathophysiology combined with competency testing enables clinicians to avoid unnecessary operative interventions during labour and to institute intrauterine resuscitation to improve the environment so as to reduce ongoing hypoxic and mechanical stress. In addition, the rate of severe hypoxic ischaemic encephalopathy (HIE) is also half the nationally reported rate in the UK (Fig. 17-3). This indicates that these intrapartum interventions including mandatory testing enable midwives and obstetricians to differentiate a fetus exposed to intrapartum hypoxic stress and who is mounting an effective compensatory response from a fetus who is unable to compensate or has exhausted all physiological mechanisms to mount an effective response.

CTG TESTING: CURRENT DEVELOPMENTS

Several maternity units in the UK have implemented St George's CTG test. In addition, the national e-learning on CTG (a joint venture between the RCOG, Royal College of Midwives [RCM] and the Department of Health) are in the process of developing a training package with competency testing based on fetal physiology that will be made available for all midwives and obstetricians working in

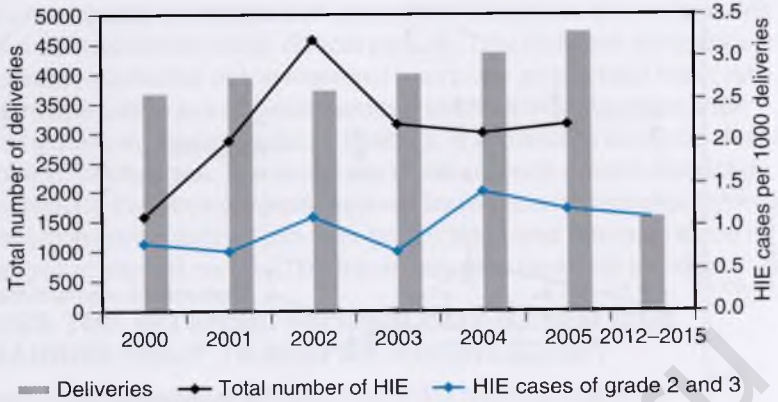

Figure 17-3. Reduction in the incidence of HIE grade 2 and 3 (2000 to 2015).

the National Health Service, free of charge. This is because there is growing recognition that, similar to passing a driving test, midwives and obstetricians need to undergo intense training and competency testing to eliminate avoidable harm during labour due to CTG misinterpretation.

CONCLUSION

CTG has a very high false-positive rate for intrapartum hypoxia because it relies on pattern recognition, which is fraught with errors of inter- and intraobserver variability. Mandatory competency testing is essential to reduce intrapartum hypoxic injuries as well as unnecessary operative interventions. Observational studies suggest that intrapartum interventions and perinatal outcomes are positively influenced by this approach. However, mandatory testing per se is unlikely to be effective and a combination of an intense physiology-based CTG training, use of a sensible additional test of fetal wellbeing such as the fetal ECG and ongoing support and education for staff in their daily clinical practice is essential to improve outcomes. Leadership and teamwork of a group of specialist midwives and obstetricians with a special interest in intrapartum fetal monitoring and support by senior management by appropriate resource allocation are equally important.

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Note: Page numbers followed by *b* indicates boxes, *f* indicates figures, and *t* indicates tables.