Fetal Surveillance

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Preface

The educational material entitled “Fetal surveillance” has been developed to provide midwives and doctors with up-to-date knowledge relating to the ability of the fetus to utilize its defense against threatening oxygen deficiency. It is hoped that the user’s ability to interpret the physiological reactions of the fetus will improve, in combination with the new STAN® recorder. The format of the teaching and training package is based on experience acquired from years of clinical STAN® development work.

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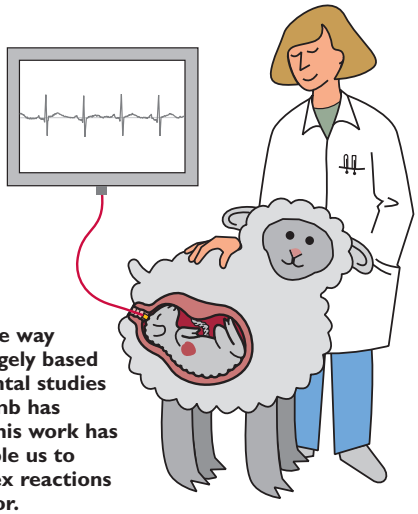
Basic Physiology

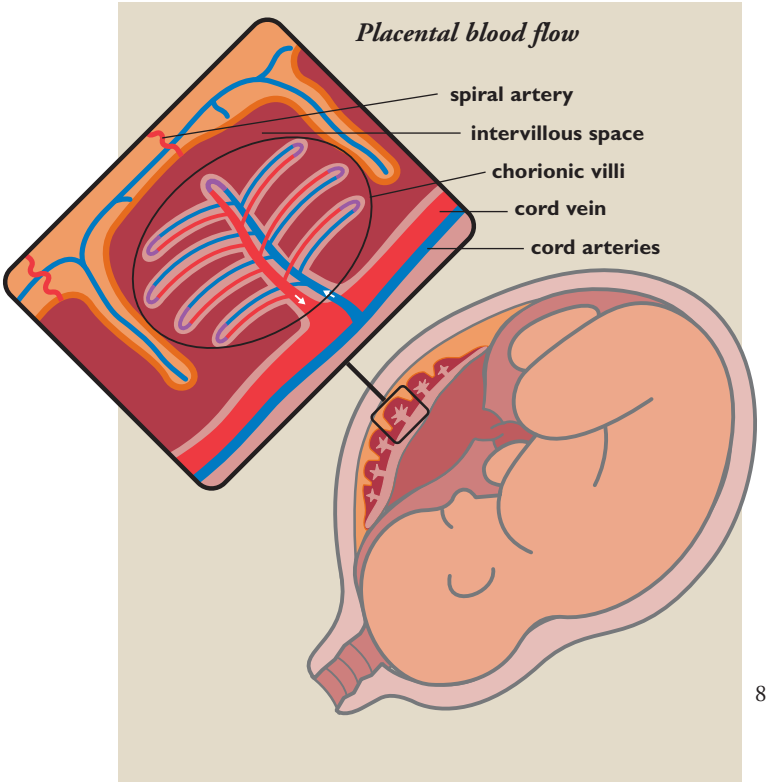


Our knowledge of the way the fetus reacts is largely based on animal experimental studies in which the fetal lamb has served as a model. This work has been needed to enable us to interpret the complex reactions that exist during labor.

Introduction

Being born is the greatest challenge in the life of an individual. Not only must the baby adapt to a completely new environment, but this transition is also associated with hypoxia and acidemia. The aim of being born is for the child to establish itself as an air-breathing individual with its own nutritional supply and pattern of reactions. These patterns of reaction have a meaning as the baby is bound to the mother to obtain her continuing support. To handle labor, the fetus is equipped with defense mechanisms, which enable it to manage even marked oxygen deficiency.

The experience acquired during the past 30 years has shown that a healthy fetus that is exposed to marked hypoxia during labor but which manages the newborn period adequately will develop normally. This makes intrapartum surveillance a prime task for obstetrics and we have learned more about the way the individual baby reacts to the stress of labor. This should enable us to intervene in the appropriate manner when the fetal defense has been activated but before there is an increased risk of long-term sequelae.



Improvements in fetal surveillance must be based on a more thorough understanding of the physiological mechanisms that are involved and the fetal reactions to the stress and strain of labor.

Placental blood flow

The key placental function is to enable exchange between the fetus and the mother. The placenta has both a fetal and a maternal component. The fetal vascular bed is composed of the main branches of the cord artery that divide into the fine arteries that penetrate the chorionic villi and end at the capillary bed, the capillary bed is situated on the surface of the villi, which protrude like fingers into the maternal pool of placental blood, the intervillous space. Thin veins take the blood back to the cord vein and the fetus.

The maternal blood comes from the aorta, via the iliac arteries to the uterine arteries. The spiral arteries bring the blood into the intervillous space situated between the chorionic villi.

A thin capillary membrane, which enables the efficient exchange of gases and substrates, separates the maternal and fetal blood. The maternal placental blood flow is normally high, about 500 ml a minute. This flow is markedly affected by the tone of the uterine muscle. With a contraction exceeding 30 mmHg, the maternal flow stops and the fetus has to rely on the stores that are available in the intervillous space.

The placental circulation is critical for the fetus but of little consequence to the mother. Sometimes the mother has to prioritize her own blood supply if she is in danger in any way. As a result the fetus may suffer, as it is dependent on a continuous supply of oxygen and nutrition from the maternal blood and of carbon dioxide transportation from the fetal tissues to the lungs of the mother.

Fetal circulation

The fetal blood circulation is characterized by a rapid flow of blood, facilitated by the low fetal blood pressure. The hemoglobin concentration is high and the fetal hemoglobin is better able to bind to oxygen. In spite of the oxygen tension (PO_2) being reduced by 70% as compared with that of the mother, the oxygen saturation (SaO_2) is decreased only by approximately 35%. The combination of moderately low oxygen saturation, high transport capacity (high hemoglobin concentration) and rapid blood circulation makes the oxygen supply to the growing fetal tissue more than adequate. This also holds true for most nutritional substances.

The oxygenated blood from the placenta is transported via the cord vein to the fetus. In the fetus, the blood enters the portal vein and is transported via the ductus venosus to the inferior vena cava. At this point, mixing occurs with deoxygenated blood coming from the lower part of the fetal body. If the rate of blood flow is normal, most of this well-oxygenated blood coming from the placenta will pass directly via the foramen ovale across to the left atrium. This separation of oxygenated blood is essential as oxygen-rich blood is transported from the left ventricle to the myocardium and the upper part of the fetal body, i.e. the brain. The blood with a low oxygen concentration is transported via the right atrium to the right ventricle and the pulmo-

nary artery via the ductus ateriosus to the aorta. From the abdominal aorta, blood is transported via the cord arteries to the placenta for reoxygenation.

Fetal membranes and amniotic fluid

A thin double layer of membranes, the chorion and amnion, surrounds the fetus. These membranes protect the fetus from micro-organisms and provide a container for the fetus and the amniotic fluid. The amniotic fluid is constantly produced and circulated throughout pregnancy. It is produced mainly in the fetal lungs, taken up by the fetus by swallowing and reabsorbed in the gastrointestinal tract. At the same time, the fetal kidneys produce urine that becomes part of the amniotic fluid. In early pregnancy, the color is clear, but with increasing gestation it starts to contain waste products from the fetal skin. The volume may vary from 500-2 000 ml. This volume enables the fetus to move and movements are important when it comes to the development of the muscle and skeletal structures. Furthermore, the amniotic fluid protects the fetus from external mechanical forces. As long as the fetal membranes are intact, the amniotic fluid prevents the cord from being compressed during contractions.

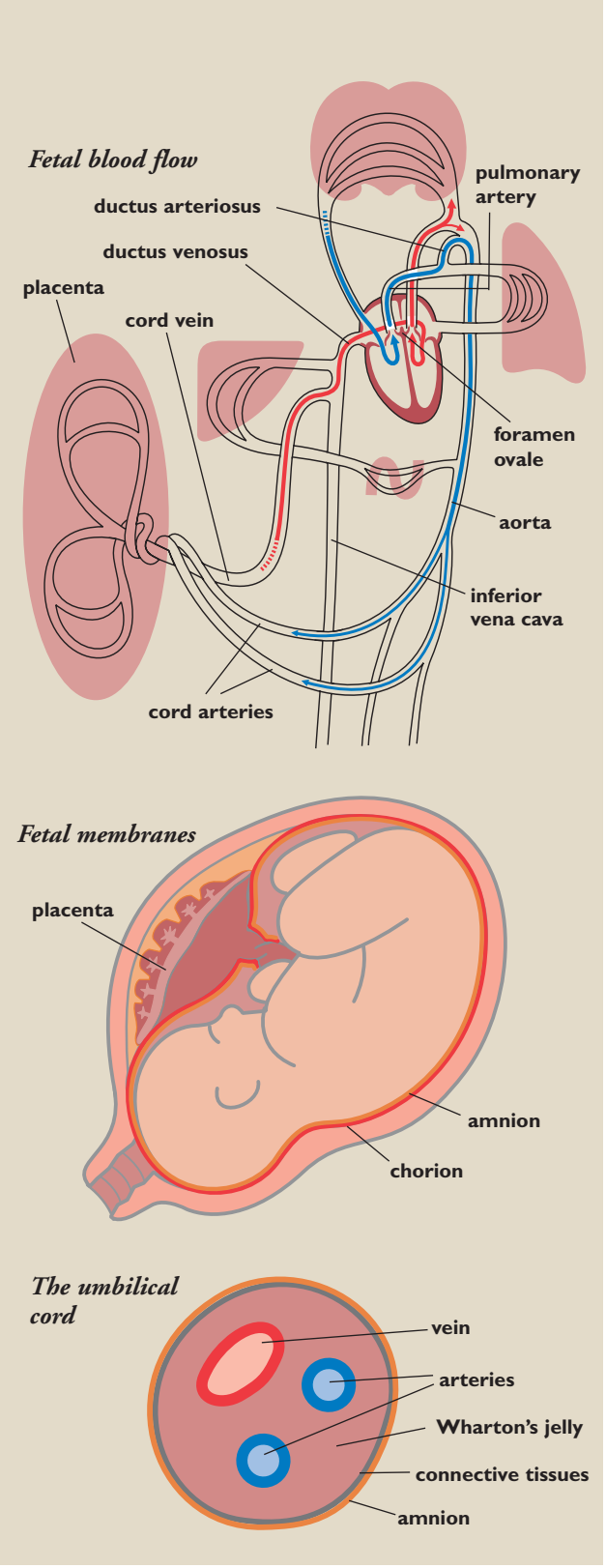
The umbilical cord

The umbilical cord links the fetus with the placenta. The two thin cord arteries transport deoxygenated blood from the fetus to the placenta. The thick cord vein transports the oxygenated blood from the placenta to the fetus. A soft jelly-like substance, called Wharton's jelly, surrounds these vessels. Amniotic membranes and a thick layer of connective tissues cover the cord vessels. This connective tissue is important, as it equalizes external pressure exerted on the cord during a contraction. This means that moderate contractions during the first stage of labor would not normally affect cord circulation, whereas during active pushing, the forces are often such that they block the umbilical vein blood flow in particular.

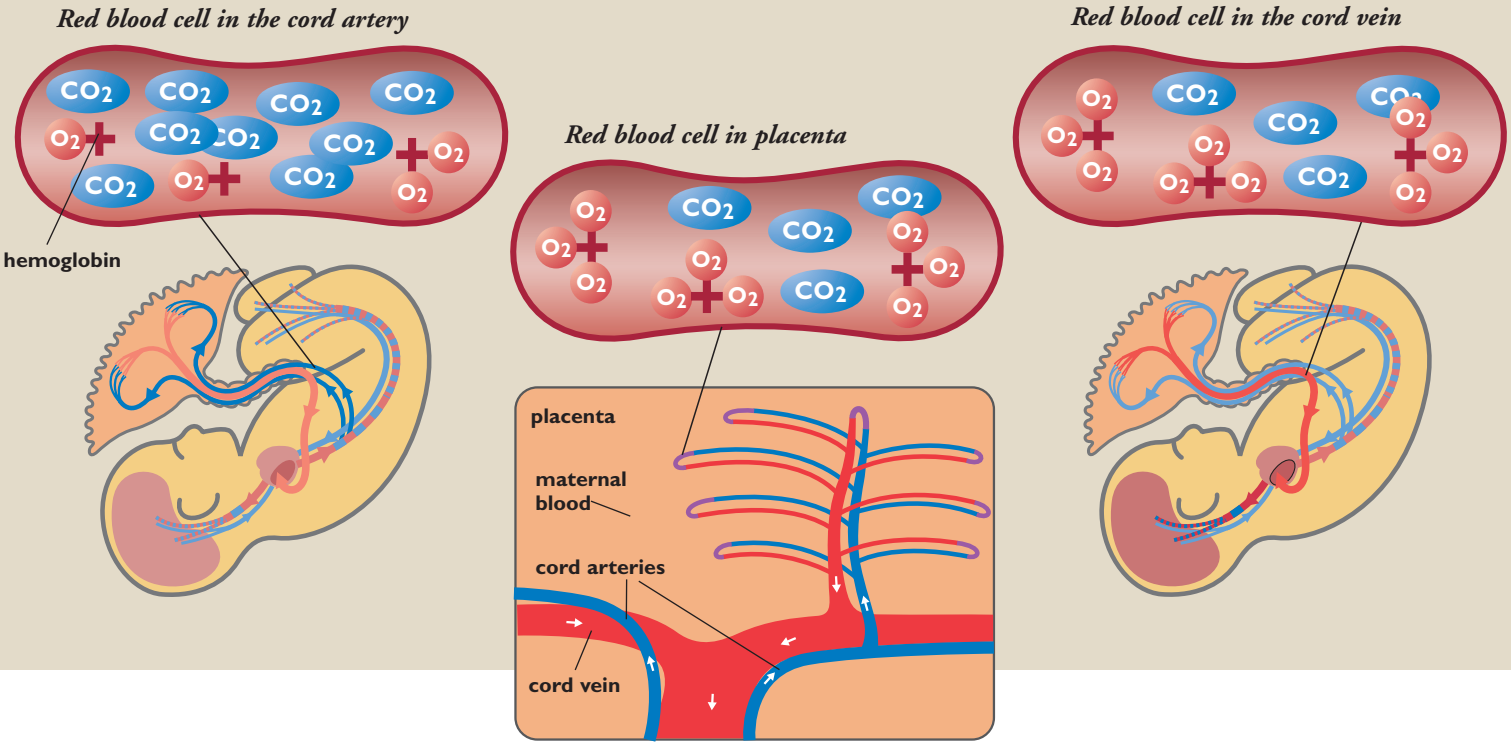
Placental gas exchange

Oxygen has to be transported to the tissues and the cells for energy production purposes. The energy is used for different activities and growth. At the same time, a large amount of carbon dioxide is produced and it has to be removed in order for the tissues to maintain their activities.

Blood from the fetus is transported through the cord arteries to the placenta. Approximately half the blood that leaves the fetal heart is transported to the placenta and this blood flow is regulated by the fetal blood pressure. The fetus attempts to increase its blood pressure in response to oxygen deficiency in order to maximize placental blood flow and thereby the exchange of gases and nutritional uptake. The cord artery blood has a low oxygen concentration and a high carbon dioxide concentration. Oxygen is transported, bound to hemoglobin. We can record how many of the four binding sites of the hemoglobin molecule are occupied by oxygen. We call this the oxygen saturation of the blood. The oxygen saturation of the cord artery blood is approximately 25%.



When the red blood cell reaches the placenta, oxygen is attached and, at the same time, carbon dioxide is removed from the fetal blood via the thin capillaries of the fetal placenta. The diffusion of gases is regulated by the difference in gas partial pressure between the fetus and the mother. Normally, the fetus has a much lower partial pressure for oxygen and a higher partial pressure for carbon dioxide. This means that it is the blood flow that regulates the amount of oxygen and carbon dioxide that can be transported between the fetus and placenta. The most important function of the placenta is to serve as the lung of the fetus and this is usually done in the most efficient manner. How-



ever, with increasing fetal growth, most of this capacity is utilized and no reserve capacity is left during labor.

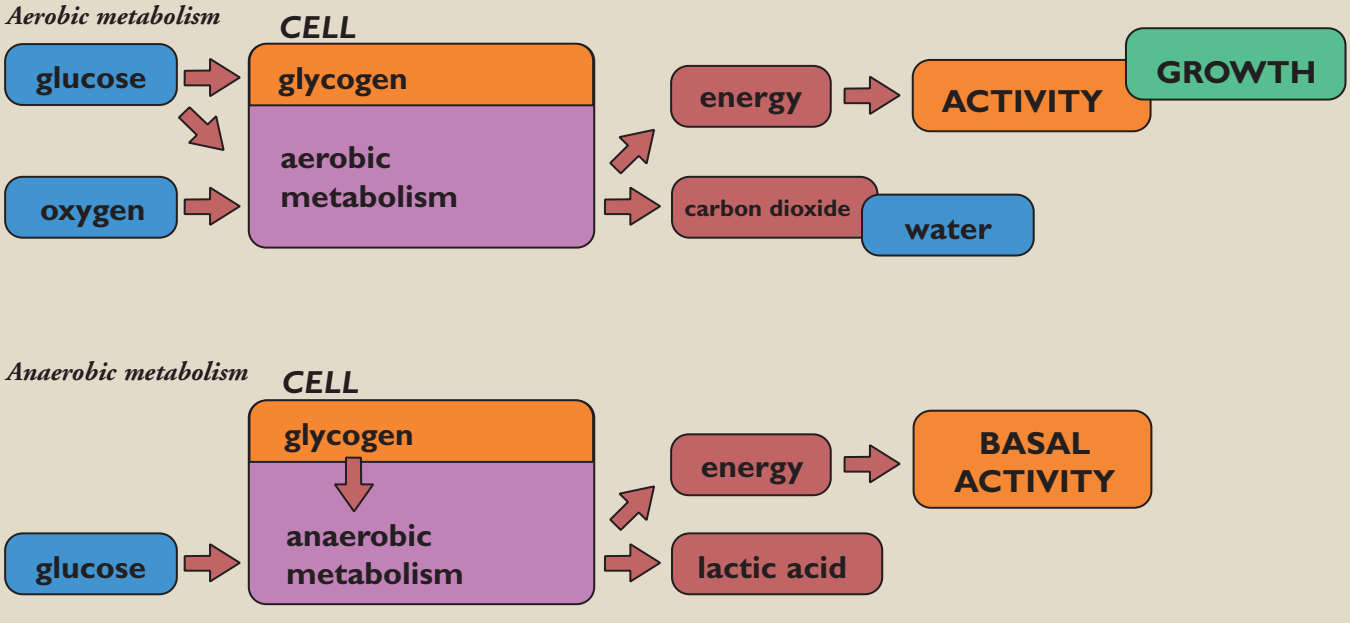
After the placental gas exchange, the blood is transported back to the fetus via the cord vein. The blood now has a high oxygen content and a low carbon dioxide content. The oxygen saturation is approximately 75%. This comparatively high oxygen saturation depends on the increased ability of the fetal hemoglobin, as compared with adult hemoglobin, to bind oxygen. Together with a high blood flow to the tissues and the extraordinarily fine ability of the fetal tissues to extract oxygen, this secures an adequate oxygen supply and even a reserve of oxygen.

The oxygenated blood passes across the fetal heart and, from the left heart ventricle, the most oxygenated blood is delivered to the heart muscle and the brain.

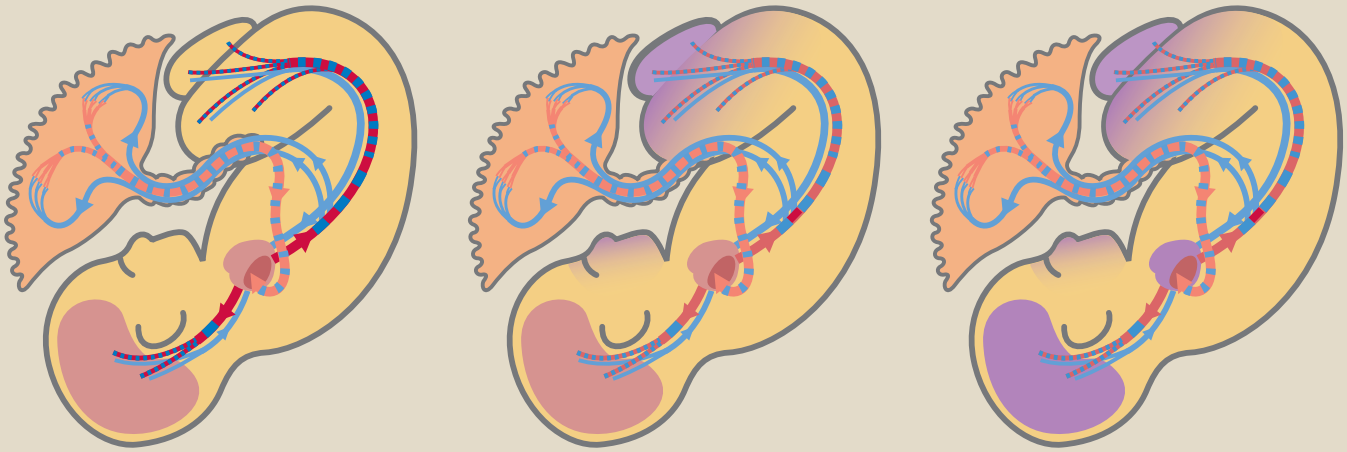
Cell metabolism

The normal cellular metabolism utilizes predominantly glucose and oxygen. This is labeled aerobic, oxygen-dependent metabolism. Some of the glucose taken up by the cell may be stored as glycogen. These stores are generated during the last trimester. A preterm fetus has less glycogen stored than a term fetus. During aerobic metabolism, the energy that is produced is utilized during activity and growth. It is important to note that carbon dioxide and water are the waste products that need to be removed from the cell by the blood flow.

During hypoxia, the fetus is capable of supporting the aerobic metabolism using the non-oxygen-dependent anaerobic metabolism. Blood glucose and stored glycogen are then utilized and energy is produced to cover the basal



Basic definitions



hypoxemia – affects the arterial blood **hypoxia – affects the peripheral tissues** **asphyxia – affects the central organs**

activity. The waste product produced by this process is lactic acid.

The amount of energy produced from glucose during anaerobic metabolism corresponds to 1/20 of the energy produced during normal oxygen-dependent metabolism.

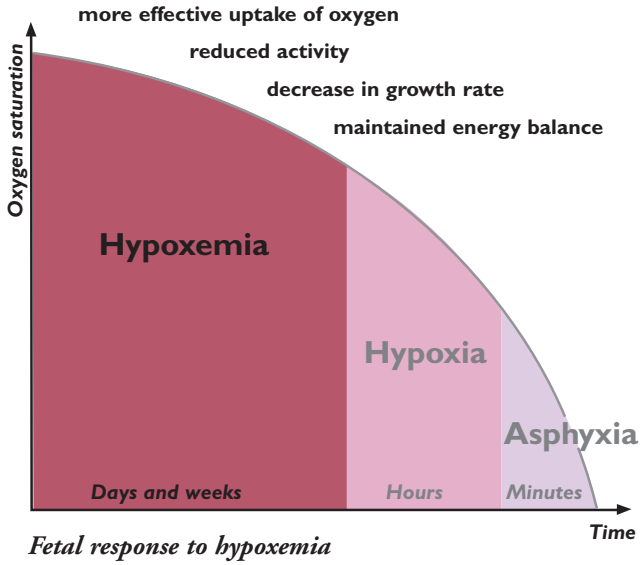
Basic definitions

When we discuss the oxygen deficiency of the fetus during labor, there are three terms that need to be distinguished.

Hypoxemia, which means a decrease in the oxygen content of the arterial blood alone.

Hypoxia, which means a decrease in the oxygen content that affects the peripheral tissues.

Asphyxia, which means a general oxygen deficiency that affects the high priority organs as well.



Fetal response to hypoxemia

Fetal response to hypoxemia

Hypoxemia is the initial phase of oxygen deficiency. During hypoxemia, the oxygen saturation decreases and affects the arterial blood, but cell and organ functions remain intact. What we notice is a decrease in oxygen saturation with intact organ function.

The fetal response depends on the activation of chemoreceptors, located in the major vessels. These receptors are activated by a decrease in the oxygen saturation of the arterial blood, and the response depends on the level of oxygenation. In adults, we may see a similar situation when they are exposed to a high altitude. The body reacts with increasing breathing, increasing blood flow through the lungs and an increase in the number of red blood cells.

The initial fetal defense against hypoxemia is the more effective uptake of oxygen. Reduced activity, in other words a decrease in fetal movements and fetal breathing, may serve as another defense mechanism. Eventually, a decrease in growth rate may become part of the defense against long-lasting hypoxemia. All these reactions reduce the need for oxygen as the energy requirement decreases and, as a result, a maintained energy balance is seen. The fetus can handle a situation of controlled hypoxemia for days and weeks. However, the development of organ systems may be affected and we should expect a fetus exposed to long-term stress to have less ability to handle acute hypoxia during labor.

Fetal response to hypoxia

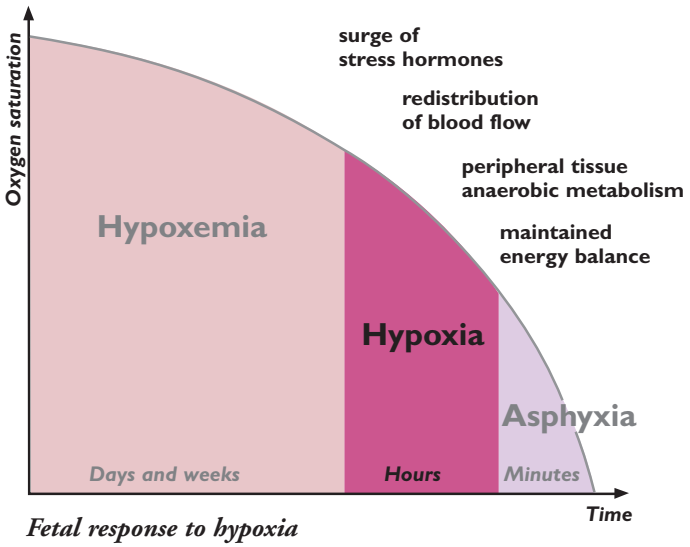
If oxygen saturation decreases still further, the defense used by the fetus during the initial hypoxemia phase may not be sufficient to maintain an energy balance and the fetus may then enter the hypoxia phase. This means that the oxygen deficiency now starts to affect the peripheral tissues in particular. The fetus has to utilize forceful defense mechanisms to handle the situation. The prime reaction to hypoxia is a fetal alarm reaction with a surge of stress hormones and a reduction in peripheral blood flow. This causes the redistribution of blood flow to favor the central organs, the heart and the brain. Peripheral tissue anaerobic metabolism

occurs. These alterations secure and maintain the energy balance in the central organs and the fetus can manage this situation for several hours.

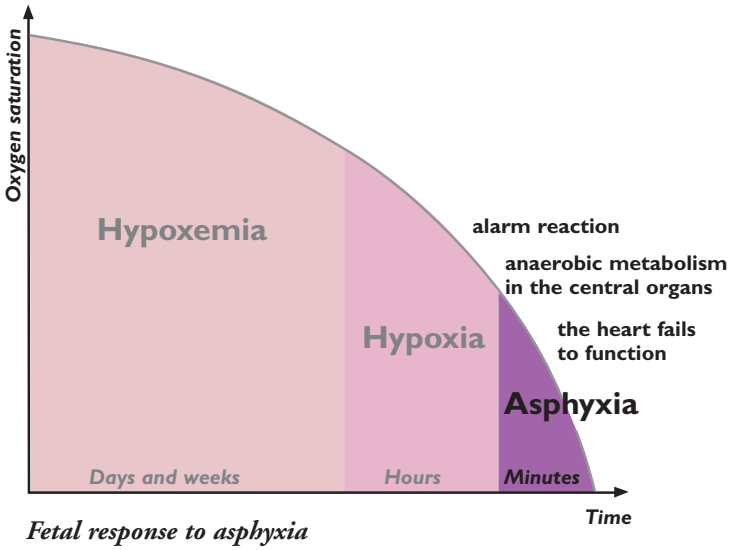
A comparison could be made with the adult body during heavy physical work when the muscle cells have to work so hard that the blood flow no longer provides enough oxygen. The ability of the cells to generate work is directly related to the ability to create extra energy by non-oxygen-dependent metabolism.

Fetal hypoxia causes a forceful alarm reaction with a marked surge of the stress hormones adrenalin (epinephrine) and noradrenalin (norepinephrine) from the adrenals and the sympathetic nervous system. The blood flow to peripheral tissues is reduced and blood is shifted towards the central organs, the heart, the brain and the adrenals. Blood flow may increase two to five times, securing an adequate supply of oxygen and maintained activity. The surge of adrenalin activates beta-receptors located on the cell surface, causing activated cyclic AMP to upgrade cellular activities, including the activation of the enzyme phosphorylase. This enzyme converts stored sugar (glycogen) into free glucose (glycogenolysis). As a result anaerobic metabolism begins. Obviously, this happens in the peripheral tissues initially, due to the reduced blood flow and concomitant hypoxia.

If hypoxia is limited to the peripheral tissues alone, no fetal damage occurs. In this situation, the central, high-priority organs secure their supply of blood, glucose and oxygen and, as a result, when the fetus is born, the baby is capable of adapting normally. As long as the central organ energy balance is maintained, the fetus is capable of handling oxygen deficiency adequately. The fetus can handle this degree of hypoxia for several hours.



Fetal response to hypoxia



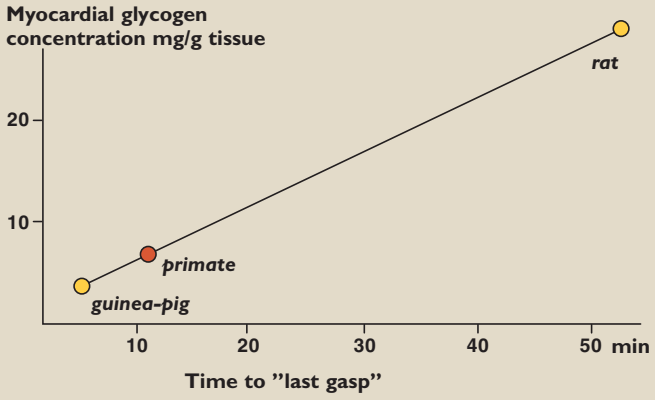
Fetal response to asphyxia

Fetal response to asphyxia

There is an increased risk of organ failure in connection with asphyxia. The cellular energy production is no longer sufficient to meet the demands. The oxygen saturation has now become very low and there is a risk of central organ function failure. The fetus now reacts with a very marked alarm reaction with maximum activation of the sympathetic nervous system and the release of stress hormones. There is anaerobic metabolism in the central high-priority organs and the fetus has to utilize its glycogen reserves in the liver and the heart muscle. In the brain, very little glycogen is stored and therefore the brain is dependent on glucose supplied from the liver. The fetus tries to keep the cardiovascular system operating for as long as possible and the redistribution of blood is even more pronounced. This marked adaptation requires a regulatory system of different reflexes and hormones securing optimal organ function. When the fetal defense reaches its final stage, the system collapses very rapidly with brain and heart failure. If asphyxia is discovered in parallel with the final bradycardia, the baby has to be delivered within minutes.

What is the most important fetal defense against hypoxia? Almost 50 years ago, Professor Geoffrey Dawes and coworkers studied the ability of fetuses of different species to tolerate a total lack of oxygen and related this ability to the concentration of myocardial glycogen. The guinea-pig fetus that was the most neurologically mature had the least ability to handle asphyxia. The rat fetus had the greatest ability, directly related to the concentration of myocardial glycogen.

The relationship between myocardial glycogen stores and the ability of fetuses of different species to withstand asphyxia measured as time to "last gasp".



Fetal defense mechanisms

- Increased tissue oxygen extraction
- Decreased non-essential activity
- Increased sympathetic activity
- Redistribution of blood flow
- Anaerobic metabolism

Intact	Reduced	Lacking
• Healthy fetus responding to acute hypoxia during labor	• Previously healthy fetus exposed to repeated episodes of hypoxemia with progressively diminishing reserves. The post-term fetus.	• Antenatal problems with chronic distress. Potential defence utilised or not available. Growth-restricted fetus.
• Optimum reaction to hypoxia • Full compensation	• Blunted reaction to hypoxia • Reduced compensation	• Minimal or no reaction to hypoxia • Decompensation
• Characteristic signs of fetal distress • Low risk of asphyxial damage	• Variable signs of fetal distress • Risk of asphyxial damage	• Uncharacteristic signs of distress • High risk of asphyxial damage

Fetal defense mechanisms

We have previously discussed the way different defense mechanisms can support the ability of the fetus to handle oxygen deficiency. These mechanisms can be summarised as follows:

- Increased tissue oxygen extraction
- Reduced non-essential activity
- Increased sympathetic activity
- Redistribution of blood flow
- Anaerobic metabolism with the metabolism of blood sugar-glucolysis, and glycogen-glycogenolysis.

Intact defense

If these defense mechanisms are intact, an optimum reaction to hypoxia with full compensation is seen. This is a healthy fetus facing acute hypoxia during labor with a low risk of asphyxial damage. We would expect characteristic EFM and ECG signs of fetal distress, as everything is in place and the fetus is capable of responding fully.

Reduced defense

The situation becomes worse when the defense mechanisms are reduced, causing a blunted reaction to hypoxia with reduced compensation. One example of this kind is the previously healthy fetus exposed to repeated episodes of hypoxia with a progressively diminishing reserve. A clinical example of this situation is the post term fetus. The blunted reaction causes an increased risk of damage and we would also expect variable signs of fetal distress.

Lacking defense

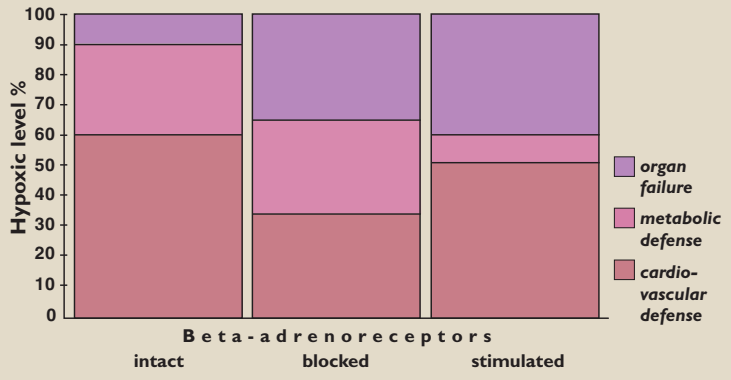
When fetal defense is lacking, a minimal reaction to hypoxia is seen, as most defense mechanisms have already been utilized or have not had the chance to develop. Clinically, we would expect a situation like this when there are antenatal problems with chronic distress, as in the severely growth-retarded fetus. There is a high risk of asphyxial damage and uncharacteristic signs of fetal distress should be expected.

The most characteristic fetal defense against hypoxia is the marked activation of the sympatho-adrenergic system. If it is blocked by giving the mother beta-blocking agents, the fetal defense is curtailed and the ability to handle hypoxia is reduced. Extensive beta-adrenoreceptor activation causes an overreaction and the glycogen and glucose that are available rapidly disappear.

Hypoxic episodes caused by uterine contractions are repetitive by nature. It is important for the fetus to rapidly redistribute the oxygen, which returns when a contraction eases off. If the ability to react is hampered, as is the case if the beta-adrenoreceptors are blocked, the fetal brain suffers, whereas the heart may be protected.

Beta-adrenoreceptors and hypoxia

The graph indicates the relationship between the degree of oxygen deficiency, activation of fetal defense systems and the impact of beta-adrenoreceptor activation and blockade. The sensitivity of these receptors will increase with hypoxia and externally given beta-mimetic drugs, such as terbutaline may cause a metabolic overreaction with rapid utilization of glycogen stores with decreased ability to handle hypoxia.



EFM Physiology

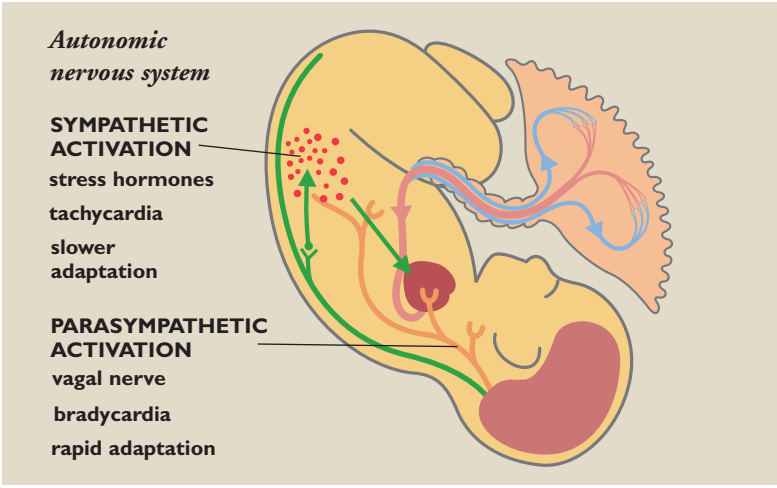
Introduction

Fetal heart sounds have been used for more than 100 years to distinguish between a live and a dead fetus. Pinard's stethoscope is still a useful instrument for this purpose.

It appeared natural to continue and develop these observations still further when the new electronic fetal monitoring technique was introduced during the 1960s. The ability to monitor fetal reactions continuously, using more detailed heart frequency analysis, was thought to provide a unique opportunity to identify hypoxia and prevent brain damage. Interest focused initially on episodes of bradycardia, but, as the fetal monitors improved, heart rate variability, i.e. beat-to-beat variation, became a more important parameter.

EFM technology has become very robust and technically easy to operate. However, very little new information about the physiology behind fetal heart rate changes has emerged. The main problem has been the identification of specific hypoxia-related patterns and, as a result, numerous deliveries have unnecessary intervention in an attempt to prevent intrapartum hypoxia. Today, we have to accept that the electronic fetal monitor cannot provide all the information that is required, and scientists have been working for the past 25 years to develop new technologies for continuous intrapartum fetal monitoring. In this process, it is important to build on areas of strength. Undoubtedly, the fetal monitor tracing contains important information, and there are two situations in which the fetal monitor provides valuable insight into the condition of the fetus. A normal, reactive fetal monitoring tracing identifies a fetus untroubled by the events of labor and a non-reactive, non-reassuring tracing with complete loss of reactivity and variability identifies a fetus that is unable to respond.

So, for the foreseeable future, fetal heart rate analysis will maintain its role as a basic function in fetal monitoring. Computing capacity should improve the presentation of the information contained in the heart rate and also provide new means of teaching and training.



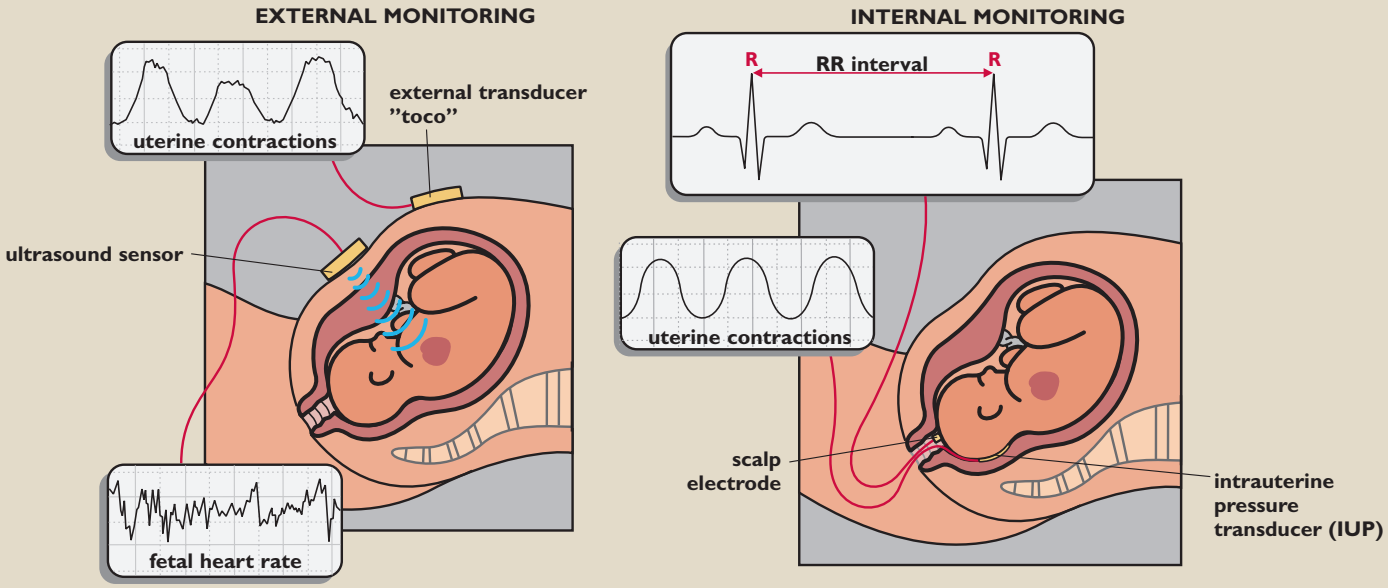
What are we recording?

Before the rupture of membranes, external fetal monitoring using external methods could be applied. An external transducer called toco records uterine contractions. The fetal heart rate is detected from an ultrasound sensor including both a transmitter and a receiver located on the abdomen of the mother. This external fetal heart rate recorder has certain limitations, and to obtain accurate fetal heart rate variability recordings and to enable accurate heart rate recordings during marked bradycardia, internal monitoring is required. This enables the accurate detection of each heart beat by utilizing the R-R interval of the fetal ECG from a scalp electrode. Alterations in intrauterine pressure could be recorded from an intrauterine pressure transducer (IUP).

Autonomic nervous system

The fetal heart rate is regulated through changes in the autonomic nervous system. This is an independent part of the central nervous system that guides basal reactions and dominates during fetal life. The main components are the parasympathetic and sympathetic limbs.

Recording of fetal heart rate and uterine activity

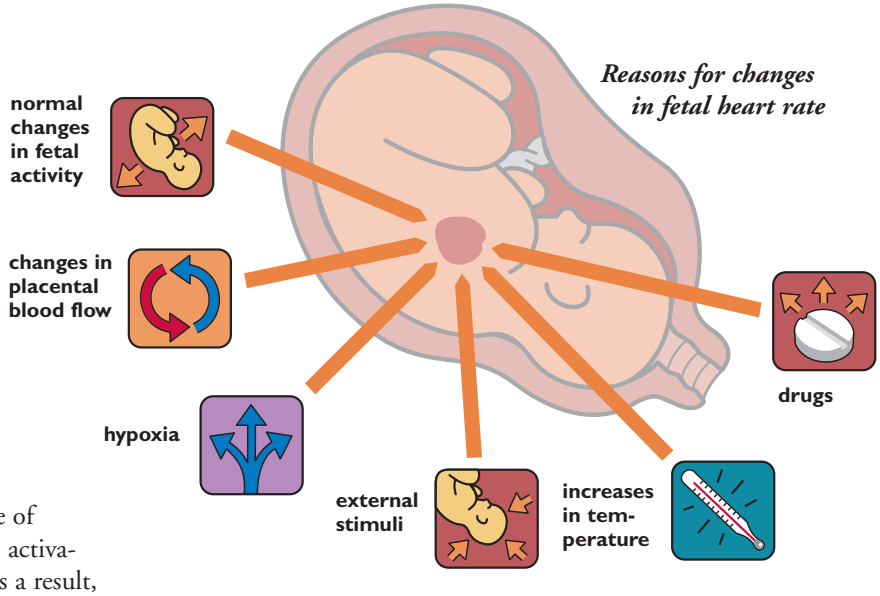


The parasympathetic activation operates mainly via the vagus nerve. The main aim of parasympathetic activation is the rapid adaptation of the cardiovascular system to a changing internal and external environment. An example of this is the marked response seen when increased pressure is applied to the eye bulb. Parasympathetic activation causes a reduction in fetal heart rate, otherwise known as bradycardia.

Sympathetic activation causes the release of stress hormones from the adrenals and the activation of the sympathetic nervous system. As a result, the fetal heart rate may increase and we may see tachycardia. Sympathetic responses result in slower adaptation than that seen when the parasympathetic limb is activated.

The most important factor is the ability of the catecholamines to counteract the depressant effect of hypoxia per se on the fetal heart and brain function. Even a normal vaginal delivery causes very marked activation of the sympathetic system to support the function of the lungs and neonatal metabolism, as well as general arousal and alertness.

In the event of asphyxia, the fetus depends on sympathetic activation to maintain cardiovascular activity with the redistribution of blood flow and the utilization of glycogen stores in the liver and myocardium.



Changes in fetal heart rate

There are many different reasons for changes in fetal heart rate. Most of them have nothing to do with oxygen deficiency but are due to normal adaptation by the fetus to changes in its environment. The fetus regulates its cardiac output by changing heart rate and there are numerous reasons for a change in cardiac output.

One example is the changes that occur due to normal alterations in fetal activity. Other reasons for alterations in fetal heart rate include changes in placental blood flow, hypoxia, external stimuli, increases in temperature and drugs.

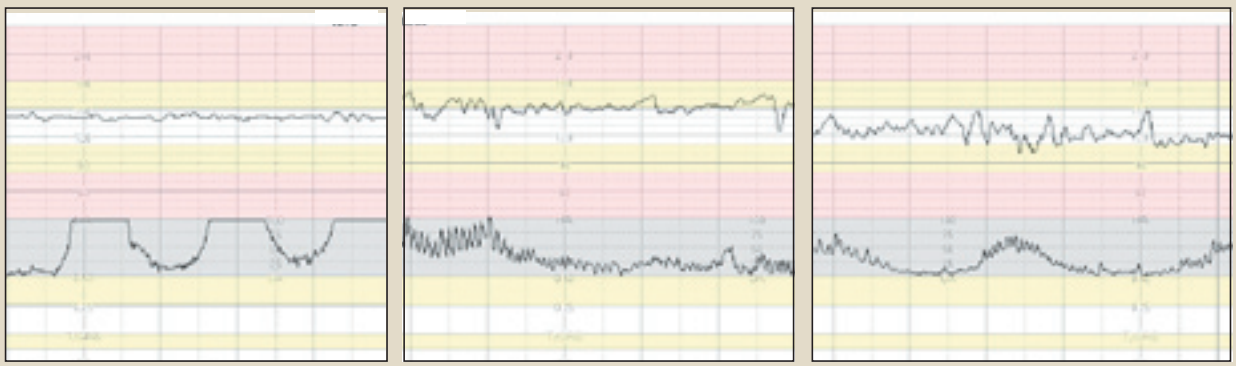
Normal changes in fetal activity

During the most restful, quiet sleep, the fetus displays little movement and the nervous system shows reduced sensitivity to stimuli. Less demand is imposed on circulatory regulatory mechanisms and fetal heart rate variability is reduced. It may be very difficult at this stage to make a fetus respond to attempts to awaken it.

When the fetus shifts into active sleep, which is also called REM sleep, there is fetal breathing and an increase in episodic movements. During REM sleep, there are rapid shifts in autonomic nervous system activity and, as a result, accelerations and increases in fetal heart rate variability are seen. A fetus that is awake shows arousal reactions when the sympathetic nervous system is activated. This fetus shows maximum reactions to given stimuli. One example is a reaction caused by fetal kicking with rapid accelerations and increased fetal heart rate reactivity.

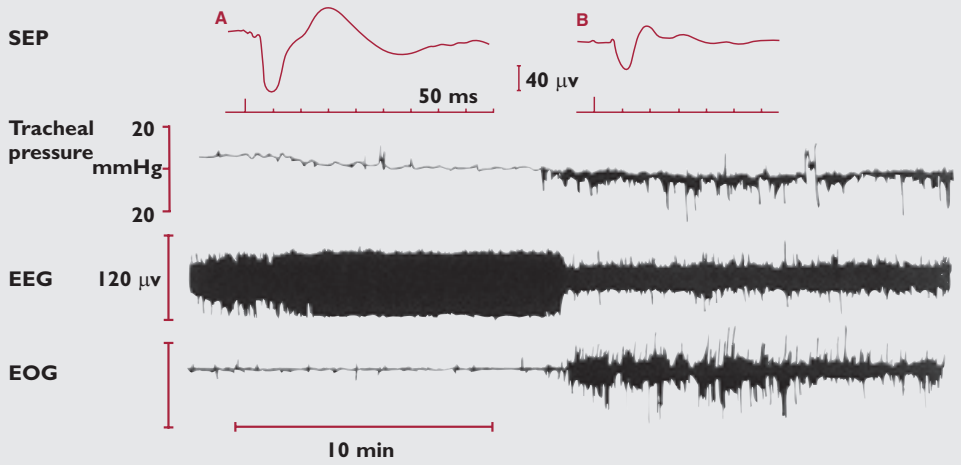
A healthy fetus shifts between different sleep states. Sometimes the fetus sleeps for long periods of time and then lacks signs of a reactive tracing. It may be difficult to judge the status of these fetuses on the basis of the fetal monitor tracing alone.

Normal changes in fetal activity



quiet sleep | active sleep | fetus is awake

Quiet and active sleep in a lamb fetus



A recording obtained from a mature lamb fetus illustrating the differences in fetal activity in relation to quiet (A) and active REM sleep (B). Active sleep is characterized by fetal breathing (episodes of negative tracheal pressure) and rapid eye movements (EOG). The somato sensory evoked potentials (SEP) were recorded as electrical potentials obtained from the surface of the fetal cortex in response to a tactile stimulus of the nostril. Note the decrease in SEP with REM as a sign of alterations in central nervous system behaviour.

Changes in placental blood flow

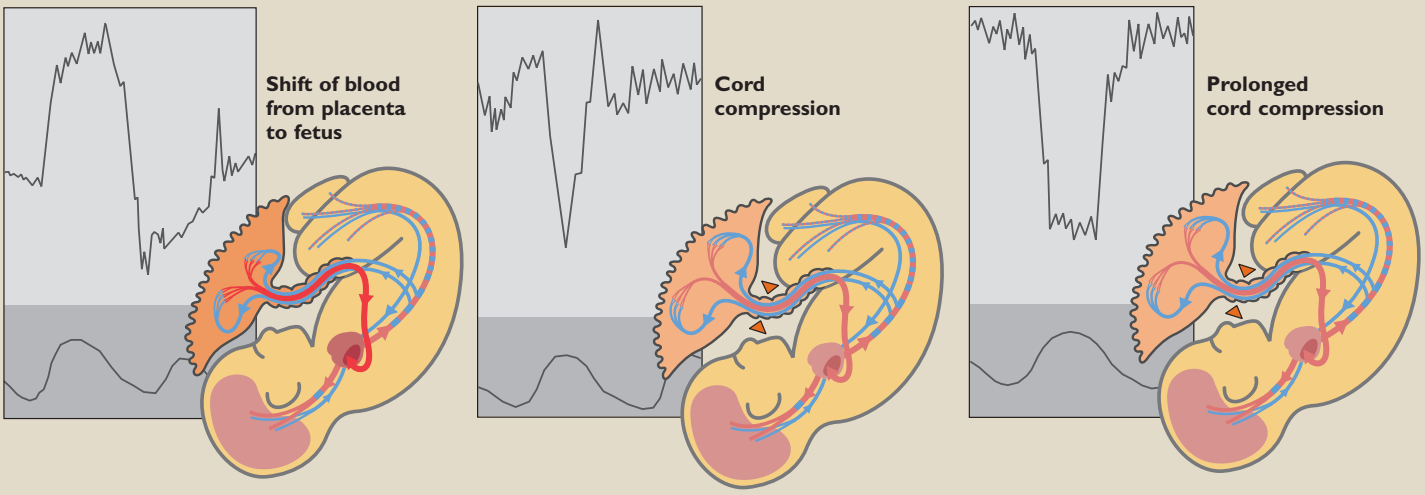
Labor could be regarded as a stress test in which the performance of the cardiovascular system is continuously tested. The main factor is obviously the strain and stress caused by the uterine contractions. Cord compression reduces blood flow to the fetus and different pressure-sensitive receptors in the heart and the main vessels respond, enabling the fetus to adapt immediately to these changes. Furthermore, the placenta contains approximately 250 ml of blood, some of which could rapidly be shifted across to the fetus during the initial phase of a contraction. All this makes labor a true stress test, and we shall now discuss
the mechanisms involved in changing the fetal heart rate during contractions.

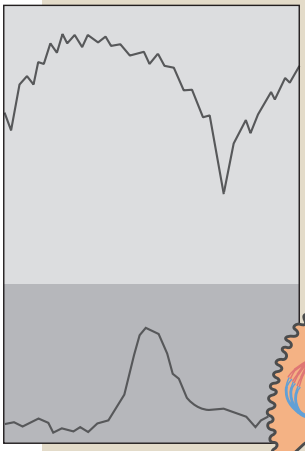
At the start of a contraction compression of the cord may cause blood to be shifted across to the fetus via the large cord vein. This causes an increase in heart rate, as the heart needs to pump this extra volume. The increase in blood volume causes an increase in blood pressure, thereby activating pressure-sensitive baro-receptors and causing a drop in fetal heart rate. So, in these circumstances, there is a shift in blood from the placenta to the fetus that causes a delayed deceleration.

As human beings walk on two legs, we are equipped with a narrow long birth canal. As a result, there are episodic reductions in cord blood flow as the baby and cord are compressed during labor. Changes related to a situation of this kind can be illustrated as follows.

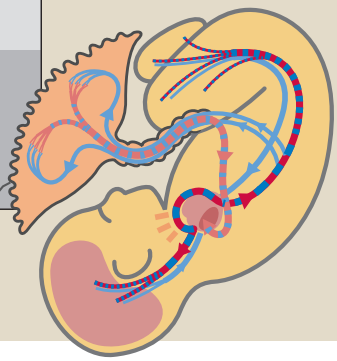
When a contraction starts, blood is pushed from the placenta towards the fetus. The heart rate increases, as the heart needs to pump more blood. As the uterine pressure increases still further, the cord vein is compressed. This stops the blood flow from the placenta to the fetus, causing a reduction in the blood volume returning to the heart. With less blood to be pumped, the heart needs to adapt rapidly with a sharp drop in heart rate. In this situation, fetal blood is trapped in the placenta as cord artery flow will continue for a short period of time. When the uterine pressure drops, the

Changes in placental blood flow

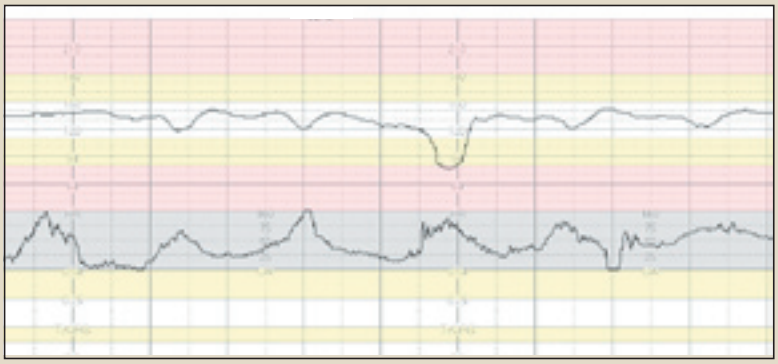




Decrease in placental blood flow during a contraction



Severe asphyxia



When hypoxia has been long-lasting and severe, the central nervous system may no longer be able to regulate and fine-tune the cardiovascular system. The EFM will then display a preterminal pattern with loss of heart rate reactivity and variability. This is a most abnormal finding and should cause immediate clinical intervention.

cord flow is rapidly restored and an acceleration takes place, as some blood may once again be shifted from the placenta to the fetus. An extension of this process is when the uterine contraction is prolonged. As before, the increase in uterine pressure causes the initial compression of the cord vein. The corresponding decrease in blood flow to the fetus causes a rapid drop in heart rate. In a moment, the placenta will no longer be able to handle the blood pushed from the fetus and the cord artery flow will stop. The fetal blood pressure increases with the activation of so-called baro-receptors. Their task is to keep the blood pressure constant in connection with changes in heart rate and heart pumping adjustments. Baro-receptor activation causes a vagally-mediated wide variable deceleration. With the decrease in uterine activity, blood flow and heart rate rapidly return to normal.

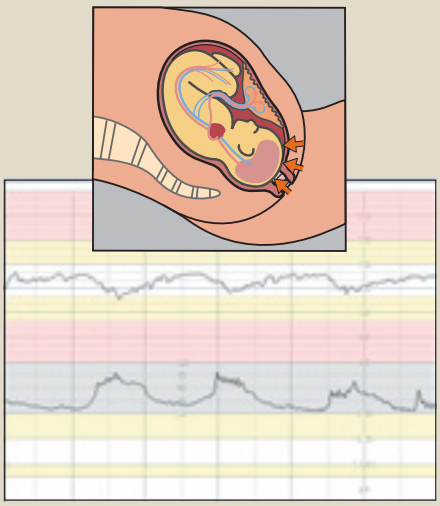
Adaptation to hypoxia

When a fetus is suffering from acute hypoxia, receptors sensitive to a decrease in oxygen partial pressure are activated. These receptors are called chemo-receptors. Chemo-receptor activation stimulates both sympathetic and parasympathetic activity and the result is an initial reduction in fetal heart rate. However, the change in heart rate also varies with the type of hypoxia. Acute hypoxia causes bradycardia, while a gradually developing or evenly maintained hypoxia causes an increase in fetal heart rate. It is important to know that the fetal heart rate pattern may differ with progressive hypoxia as the fetus adapts.

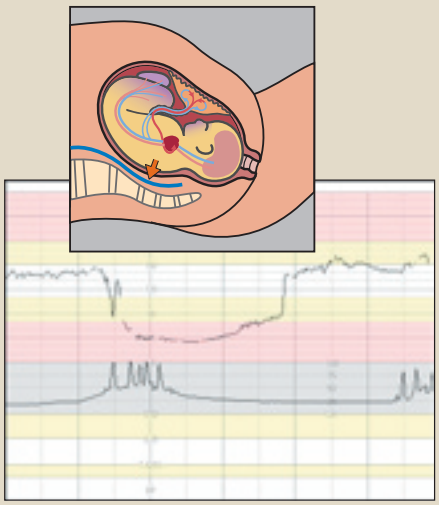
A reduction in placental blood flow during a contraction may cause a reduction in oxygen supply with the activation of chemo-receptors. Repeated decelerations, which start after the contraction has reached its peak may be seen. A pattern of this kind may also be induced by the increase in blood pressure as part of the cardiovascular adaptation to hypoxia. These decelerations are called late decelerations. With the return of blood flow and oxygenation, the sympathetic activation is maintained, causing tachycardia.

Bradycardia due to the direct depressant effect of hypox-

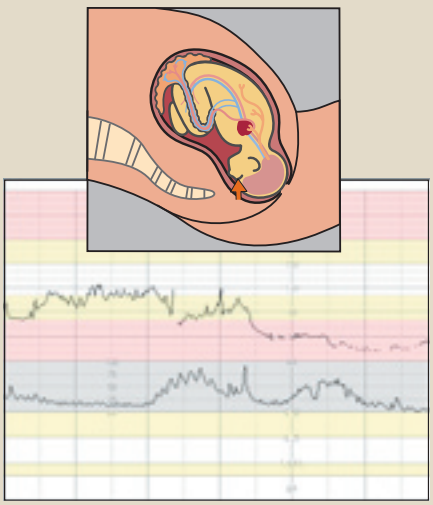
External stimuli



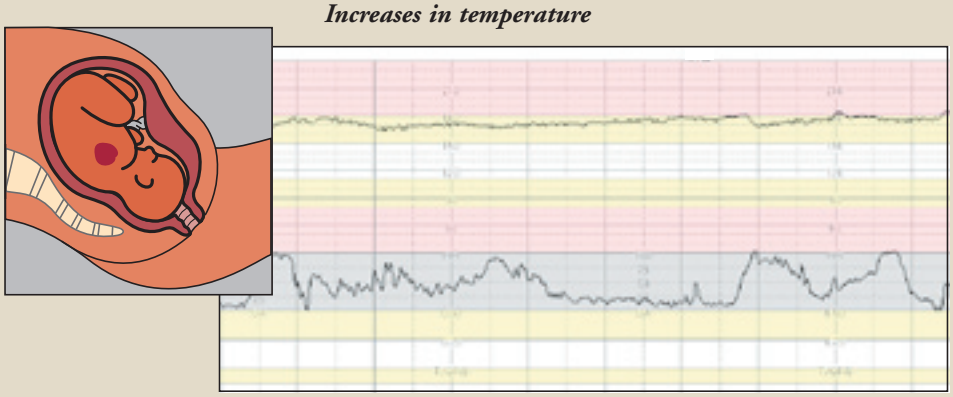
Uterine contraction and increased cranial pressure

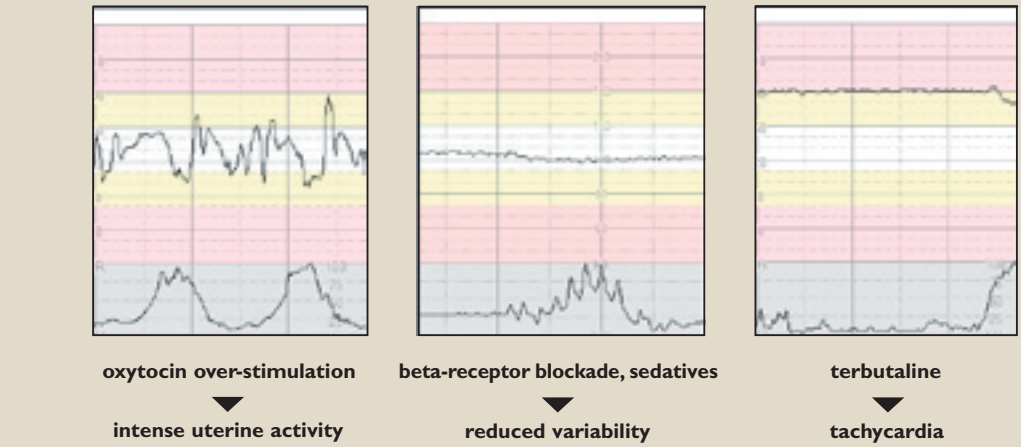


Compression of the vena cava



Increased eye bulb pressure





ia on myocardial function is most rare. Experimental data indicates that it takes approximately 90 seconds of a complete stop in maternal placental oxygenation before the hypoxia starts to affect myocardial performance.

External stimuli

The fetus has the ability to sense and react to changes in its external and internal environment.

During a contraction, the fetus is pushed down the birth canal and an episodic increase in head pressure is seen. The fetal heart rate tracing now displays early decelerations where the drop in heart rate matches the uterine activity curve. Another example is the arousal reaction caused by the squeezing and squashing of labor causing tachycardia.

With the mother on her back, there is a risk that the uterus will compress the abdominal veins. This reduces maternal placental blood flow and may cause fetal hypoxia resulting in a prolonged deceleration. The remedy is to turn the mother on her side to improve the maternal uterine blood flow.

During the last phase of labor, a marked increase in eye bulb pressure is not uncommon, causing a marked vagally induced bradycardia.

Increases in temperature

Labor is a physical stress for the mother. As with all physical exercise, water may be lost, which leads to a fluid deficit. This causes the mother to decrease her peripheral circulation because the blood volume is reduced. As a result, she reduces her ability to relieve herself of the extra heat generated by the exercise and fever may occur. The rise in temperature causes an increase in fetal metabolic rate and an increase in oxygen consumption and blood flow through the tissues. This may result in fetal tachycardia. The margins are reduced and the fetal ability to handle oxygen deficiency decreases. Appropriate treatment of maternal fever by increasing fluid intake and giving acetaminophen should make the tachycardia disappear. In the event of an ongoing infection, the ability of the fetus to handle asphyxia is markedly reduced.

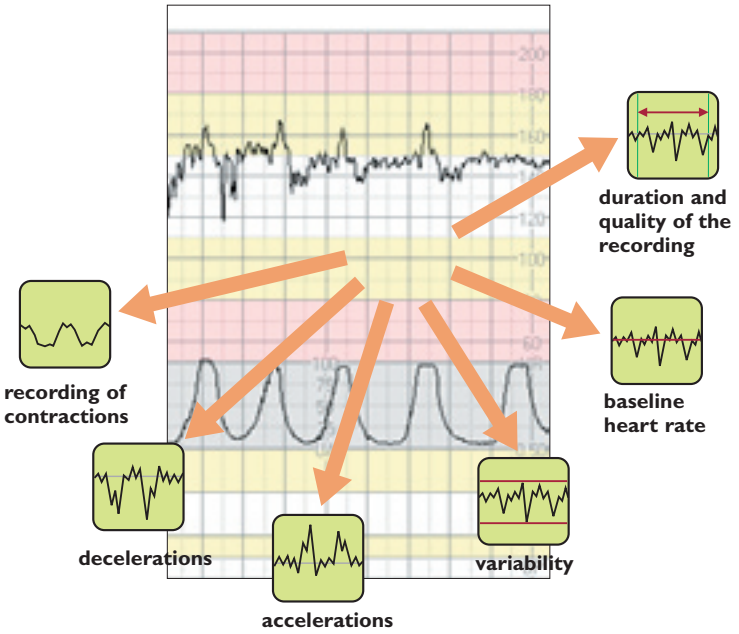
The effect of drugs

As previously discussed, different drugs may not only affect the ability of the fetus to handle hypoxia, but may also make the fetal heart rate tracing interpretation more difficult. There are numerous ways in which drugs could affect the heart rate and the ability of the fetus to handle oxygen deficiency.

Over-stimulation with oxytocin, for example, may cause hypoxia due to intense uterine activity. Beta-receptor blockade and sedatives may cause a curtailed fetal response and reduced variability. Beta-receptor-activating drugs such as terbutaline may cause tachycardia. Local anesthetics may be transferred to the fetus and cause fetal bradycardia as a result of a direct effect on the myocardium. An epidural may cause a lowering of the maternal blood pressure with reduced maternal blood flow and fetal hypoxia. If the mother is given any sedation, the drug will be transferred to the fetus, and reduce its activity and the fetal tracing reactivity. Furthermore, drugs may be accumulated in the fetus and the potential effect of any drug would have to be considered when administered in conjunction with labor.

EFM interpretation

To enable an accurate EFM analysis, the teminology has to be known and used appropriately.



Duration and quality of the recording

A minimum duration of 20 minutes is required for fetal heart rate tracing recording to be properly interpreted because of changes in sleep state and uterine activity. The speed of the recording is usually 3 cm a minute and there are 10 minutes between the printouts of the labeling of the scaling. The fetal heart rate can be plotted between 30 and 240 bpm. Uterine activity is depicted in a range of 0-100 relative units when using a tocometer and 0-100 mm of mercury when applying an intrauterine pressure sensor. Time is indicated every 10 minutes and the date is printed every 30 minutes. Information relating to the transducers that are in use is indicated every 30 minutes and whenever there is a change. If an internal recording is used, FECG is printed and when an external recording by ultrasound is used, US is printed. If an external uterine sensor is used, TOCO is printed.

Good signal quality is absolutely essential to enable accurate interpretation. If there is poor signal quality, it is better to spend time improving the signal by replacing the scalp electrode or the toco sensor rather than trying to interpret erroneous data.

Baseline heart rate

The baseline fetal heart rate is defined as the mean fetal heart rate rounded to the nearest 5 beat per minute increment over a 10-minute segment, with a stable duration of greater than 2 minutes, excluding periodic or episodic changes and periods of marked variability that differ by greater than 25 beats per minute. The normal ranges from 110 to 150 bpm with bradycardia <110 bpm and tachycardia >150 bpm.

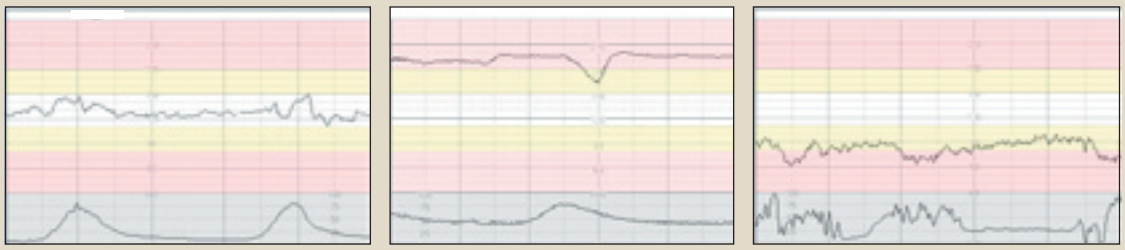
Variability

Variability describes the fluctuations in baseline fetal heart rate (peak to nadir amplitude range) that are irregular in amplitude and frequency and defined as:

Absent = undetectable fluctuations
Minimal = 5 beats per minute or less
Moderate = 6 to 25 beats per minute
Marked = 25 beats per minute or more

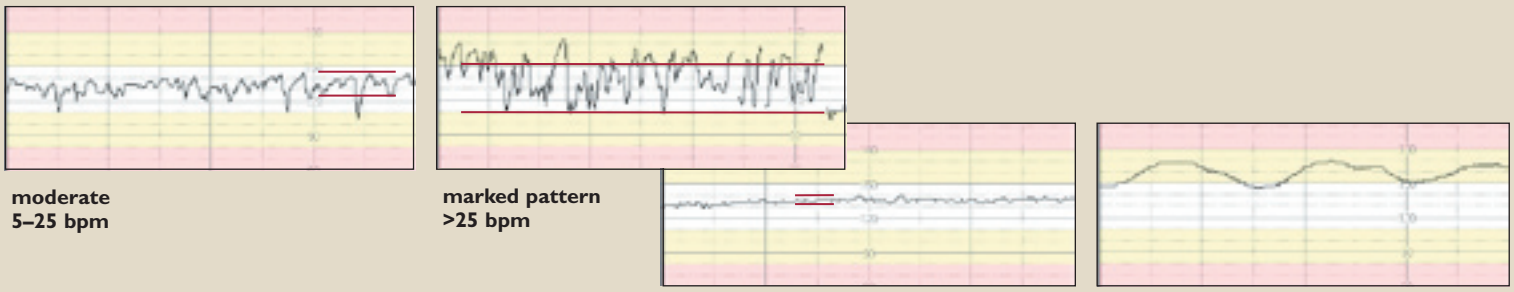
The fetal heart rate normally displays beat-to-beat variation, which are not accelerations or decelerations. This aspect of the fetal heart rate tracing provides information about the ability of the central nervous system to monitor and adjust the cardiovascular system. This so-called short-

Baseline heart rate



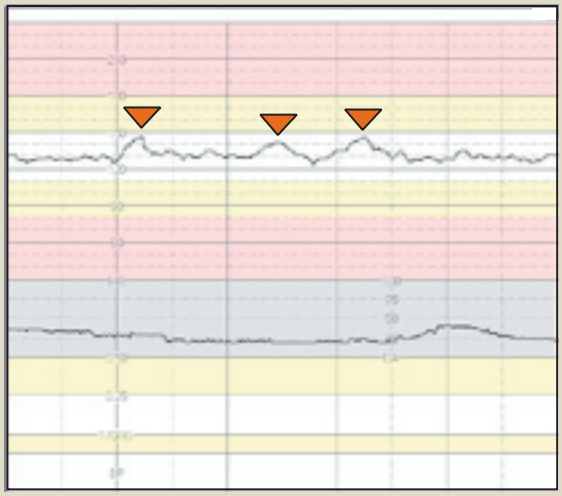
normal 110–150 bpm | tachycardia >150 bpm | bradycardia <110 bpm

Variability

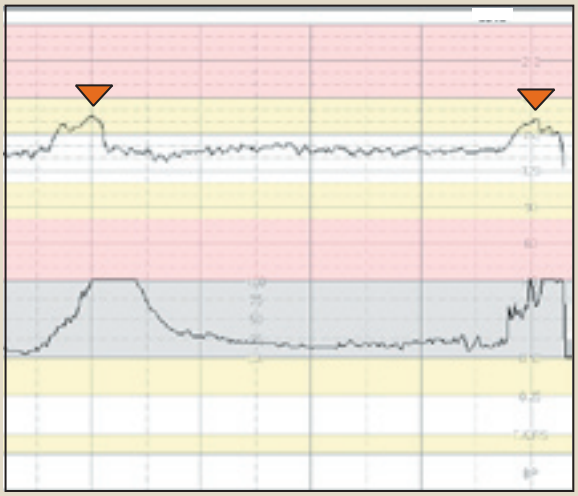


moderate 5–25 bpm | marked pattern >25 bpm | minimal <5 bpm | undetectable fluctuations

Accelerations



Periodic accelerations

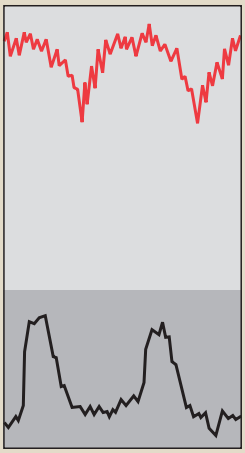


term variability may vary with time, depending on variations in sleep and activity. The same type of pattern, with a loss of heart rate variability, is one of the most important features when hypoxia is emerging. Reduced variability reflects an increase in sympathetic tone, but when there is a complete loss of beat-to-beat variation, this may also depend on the inability of the myocardium to respond.

Our ability to assess heart rate variability may be reduced when the fetal heart rate is recorded using the ultrasound technique. The reason is that accurate beat-to-beat variation requires the identification of each individual heartbeat. The ultra-sound technology is based on a methodology called auto-correlation, which means that three consecutive heartbeats are used for heart rate detection purposes. This may cause an artifactual reduction in the recorded heart rate variability. At the same time, the ultra-sound signal may change slightly due to a shift in the position of the fetal heart in relation to the sensor, causing other components of the heart wall movements to be identified. A fetal ECG recording enables the system to trigger on each heartbeat to secure accurate assessments of short-term heart rate variability.

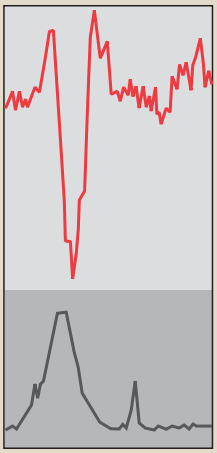
In the event of severe fetal anemia, due to immunization or fetal bleeding, the heart rate pattern may be sinusoidal. A pattern of this kind may also be noted with asphyxia. The sinusoidal heart rate pattern is defined as periodic shifts in heart rate with no beat-to-beat variation and no accelerations.

Uniform and variable decelerations



UNIFORM

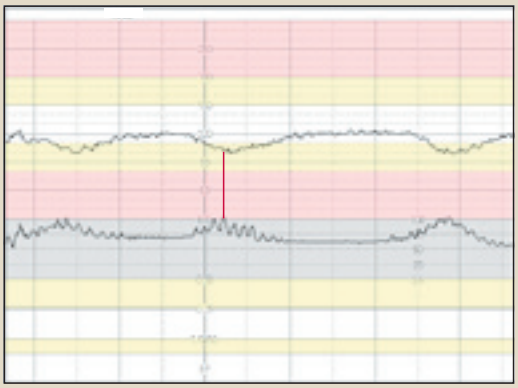
- **rounded pattern**
- **shape is similar**
- **uncommon to cause a marked loss of beats**



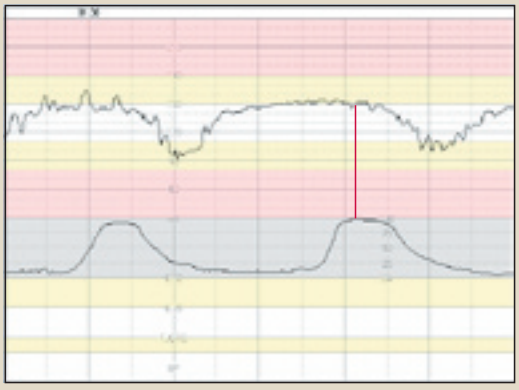
VARIABLE

- **rapid loss of beats**
- **pattern may vary**
- **marked loss of beats**

Early and late decelerations

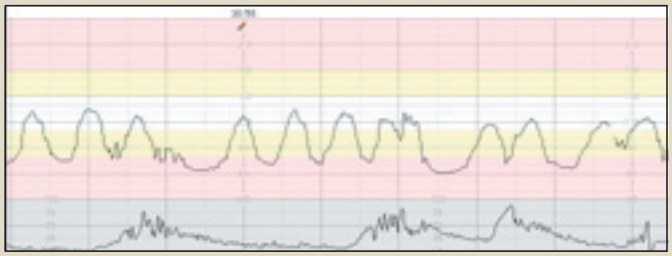


early



late

Sinusoidal tracing



A pattern like this may signify fetuses that have already suffered brain damage.

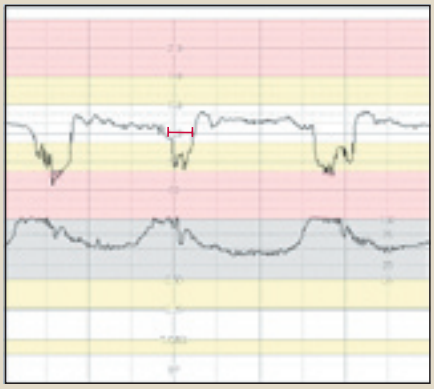
Accelerations

Accelerations are abrupt (peaks in less than 30 seconds) increases in fetal heart rate above the baseline greater than 15 beats per minute lasting from 15 seconds to less than 2 minutes. From 2 to 10 minutes is termed a prolonged acceleration and greater than 10 minutes equals a baseline change.

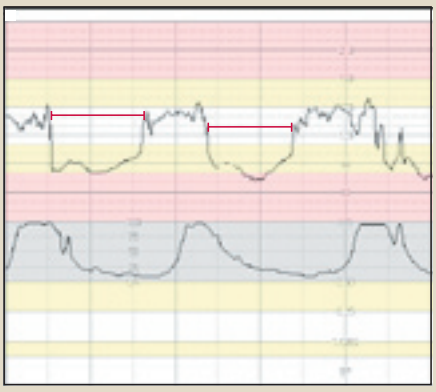
In the same way as a loss of variability may indicate hypoxia, the appearance of accelerations is an important sign of normal oxygenation. A reactive tracing should contain at least two accelerations during a period of 20 minutes.

Accelerations are signs of adequate oxygenation and verify the fact that the fetus has a capacity to respond whereas as a complete loss of heart rate variability identifies a fetus that is incapable of responding.

Periodic accelerations are repetitive episodes of marked accelerations in conjunction with contractions. They may occur as a sign of shift in blood from placenta to fetus and a change towards variable decelerations may often be seen as labor progress.



Variable decelerations (<60 sec)



Variable decelerations (>60 sec)

Decelerations

Decelerations are defined as a recurring drop in heart rate of more than 15 beats, lasting for more than 15 seconds. Decelerations may be significant findings, as they are related to contractions and thereby the development of hypoxia. However, the majority of decelerations have no relation to hypoxia but is caused by changes in the fetal environment.

There are 3 types of decelerations, early, late and variable decelerations, depending on their relation to the contractions.

Early decelerations are uniform in shape. An early deceleration is a reflex-generated drop in heart rate that matches the contraction curve. It starts before the contraction reaches its peak. The reason is usually the mechanical forces acting on the fetus after ruptured membranes and with active pushing. An early deceleration is usually handled well by the fetus and is not related to hypoxia.

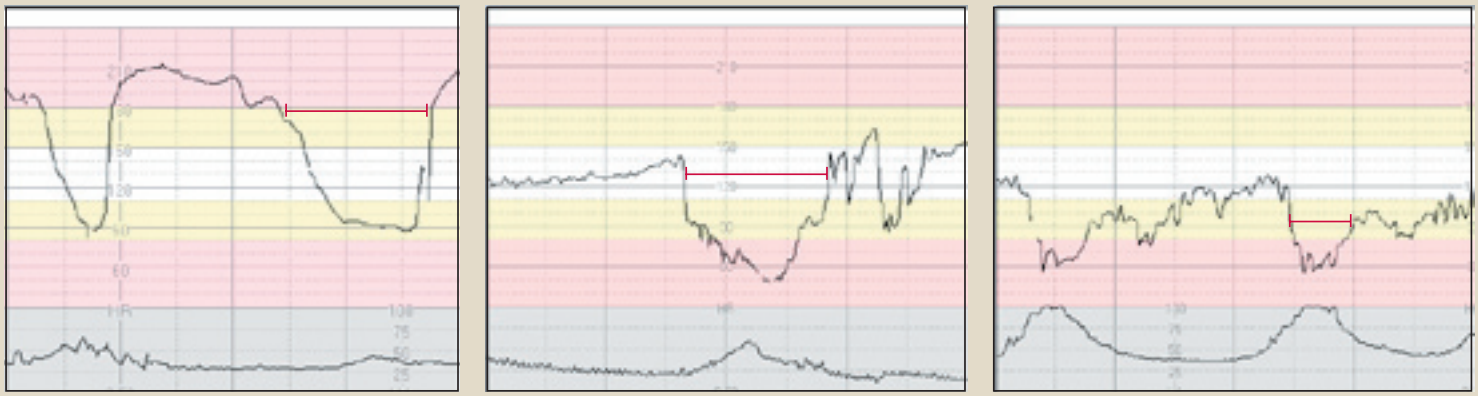
Late decelerations are characterized by a uniform pattern. There is a time lag between the onset and peak of the contraction and the onset and peak of the deceleration. There may be a relation to intermittent hypoxia caused by reductions in placental blood flow. It is unusual for late decelerations to have a marked loss of beats, but as the contractions increase in intensity, there may be an increase in the loss of beats. Late decelerations are frequently associated with an increase in baseline heart rate. They may also be related to short-lasting hypoxia related to a reduction in placental blood flow. They are often associated with abnormal uterine activity with an increasing frequency of contractions and may be seen in relation to placental insufficiency as a consequence of pre-eclampsia and growth retardation.

Variable decelerations are an abrupt (less than 30 seconds to nadir) decline in fetal heart rate greater than 15 beats per minute lasting longer than 15 seconds and less than 2 minutes from onset to return to baseline and associated to uterine contractions. The depth and duration of the variable decelerations may vary with successive uterine contractions. Variable decelerations are the most commonly occurring decelerations and account for approximately 80% of all decelerations.

The most important parameter to assess in connection with variable decelerations is the duration. Variable decelerations become ominous when they last 60 seconds or more.

The rapid loss of beats is a sign of a reduction in cord blood flow and would normally occur after ruptured membranes and during the second stage. These decelerations are signs of active adjustments. When the cord vein is compressed, approximately 50% of the blood, which normally returns to the fetal heart, is prevented from doing so. The amount of blood to be pumped by the fetal heart is thus reduced by 50%, as is the

Different patterns of variable decelerations (>60 s)



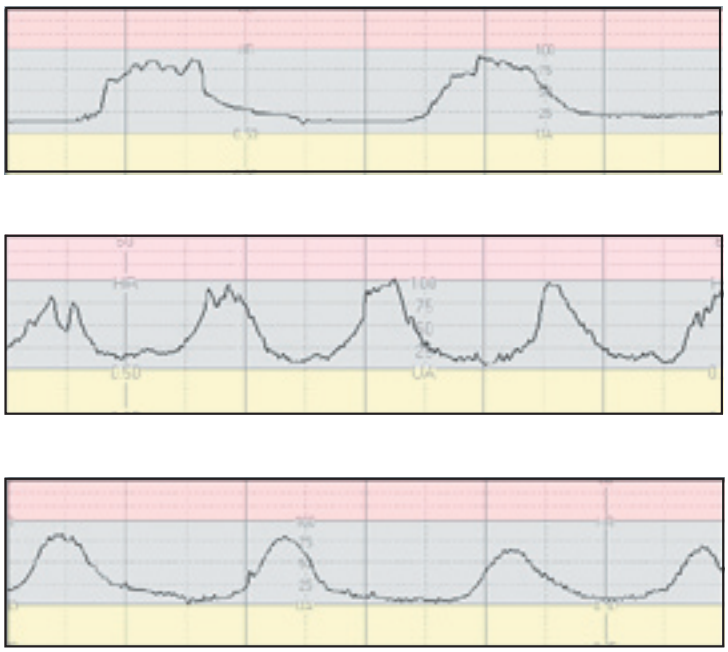
heart frequency. A variable deceleration with duration <60s is often associated with accelerations occurring before or after the deceleration. A pattern of this kind is associated with the shift of blood volume between placenta and fetus.

The fetus is very capable of handling variable decelerations, even for long periods of time if their duration is short (<60 s). The reason why they do not cause hypoxia is that they are of short duration and the oxygen supply is not significantly reduced.

A variable deceleration with duration >60 s means that there is an increased risk of the fetus experiencing hypoxia. A variable deceleration is regarded as complicated when the duration exceeds 60 seconds.

The ability of the fetus to restore blood flow becomes most essential when it is exposed to uterine contractions affecting cord blood flow. The oxygenated blood should be rapidly distributed within the fetus for the hypoxic process to be blocked. A variable deceleration with duration >60 s induces a risk of more long-lasting disturbances in cord blood flow and thereby the development of hypoxia. The duration of the contraction also affects the time allowed to recover before the next contraction starts. The risk of hypoxia therefore increases if decelerations last for more than 60 seconds. In these circumstances, there is always an accumulation of carbon dioxide in the blood and the scalp pH decreases.

A prolonged deceleration is the decrease in the fetal heart rate greater than 15 beats per minute lasting longer than 2 minutes but less than 10 minutes (more than 10 minutes is a baseline change). Usually the fetus handles the situation well. A vagal reflex caused by vaginal examination or fetal blood sampling is a common cause. Prolonged decelerations are also associated with the mother on her back, on a bedpan or vomiting.



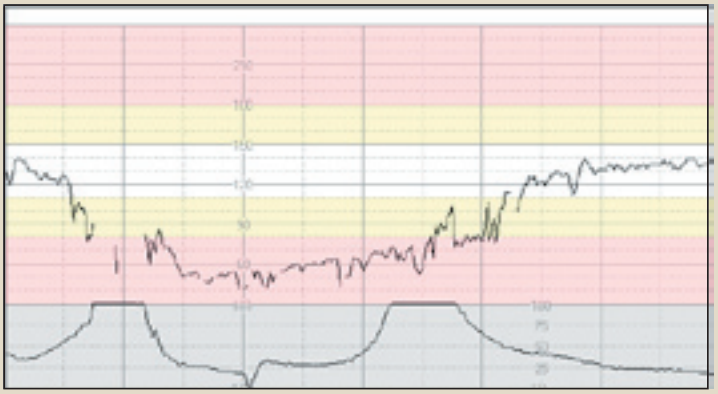
Recording of contractions

Recording of contractions

It is as important to assess uterine activity, as it is to assess heart rate. Normally, uterine activity is recorded from an external Toco sensor. Uterine activity should be validated against the frequency of contractions. The frequency should be two to three contractions every 10 minutes during the initial phase of the first stage, usually increasing to four to five contractions every 10 minutes during the later phase of the first stage. Infrequent contractions may cause slow progress and prolonged labor, which in itself increases the risk to the fetus. More than five contractions every 10 minutes may jeopardize fetal oxygenation, as the ability of the fetus to reoxygenate itself between contractions may decrease.

The duration of the contraction is important when it comes to assessing its efficiency. During the first stage, the duration may vary between 30 and 60 seconds and then increase during the final stages of the first stage and during the second stage to as much as 90 seconds. The intensity can only be recorded using an internal uterine pressure sensor. However, some information can be obtained by manually assessing uterine tone. The same holds true for assessments of the basal tone of the uterus which is most important to assess during oxytocin infusion and when

Prolonged deceleration



placental abruption is suspected.
The exchange of gases between the fetus and the mother is stopped during a contraction when the intra-uterine pressure exceeds 30 mmHg, as this causes a temporary block in placental blood flow. The fetus needs 60-90 seconds between contractions to regain normal blood gases. The ability of the fetus to handle labor often equals its ability to handle changes occurring during contractions. The duration of labor and thereby the exposure to intermittent, potentially hypoxic periods is the single factor most closely related with intrapartum hypoxia. In particular, the duration of active pushing in the second stage should always be considered as a prime factor when assessing the risk of intrapartum hypoxia.

Classification of FHR patterns
FHR tracings may be interpreted as reassuring or non-reassuring. Reassuring tracings display a baseline within the normal range with evidence of beat-to-beat variability. There are also accelerations present. Reassuring FHR tracings correlate with a good fetal outcome.

Non-reassuring FHR patterns are separated into 2 grades. Grade 1 indicates the need for further fetal assessment by ST waveform analysis. Level 2 - preterminal FHR pattern - requires immediate delivery. The characteristics of these patterns are found below.

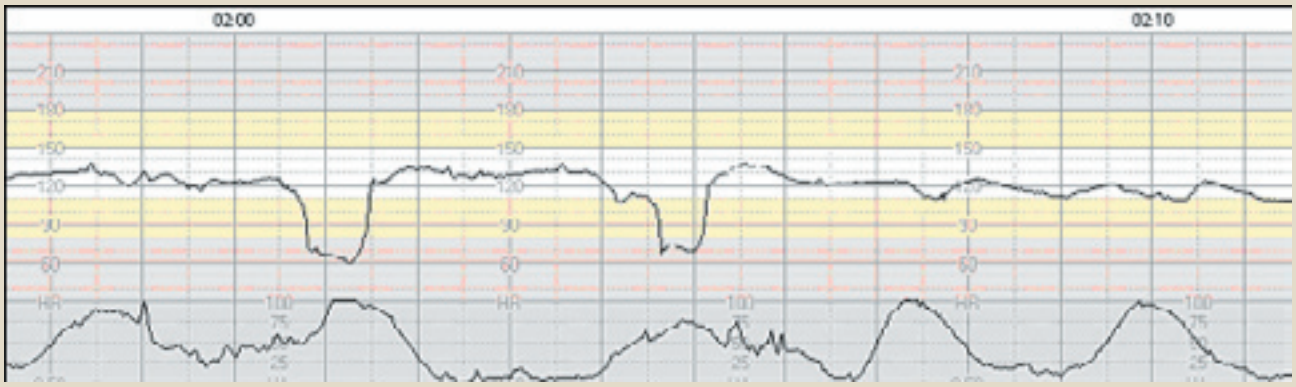
Classification of FHR patterns

FHR Classification	Baseline Heart Rate	Variability Reactivity	Decelerations
Reassuring	• 110–150 bpm	• 6–25 bpm • Accelerations present	• Early decelerations • Variable decelerations with a duration of <60 sec and depth <60 beats
Non Reassuring, Grade1	**Bradycardia:** • Rate <110 bpm (without accelerations) • Episode >2 minutes duration regardless of activities or variability **Tachycardia:** • Rate 150-170 bpm and minimal variability • Rate >170 bpm	• ≤5 bpm for >40 min • ≥25 bpm for >40 min • Accelerations absent	• Variable decelerations with a duration of >60 sec or depth >60 beats • Repetitive late decelerations
Non Reassuring, Grade2 - Preterminal	• Absent variability and reactivity regardless of other FHR patterns • Sinusoidal pattern		

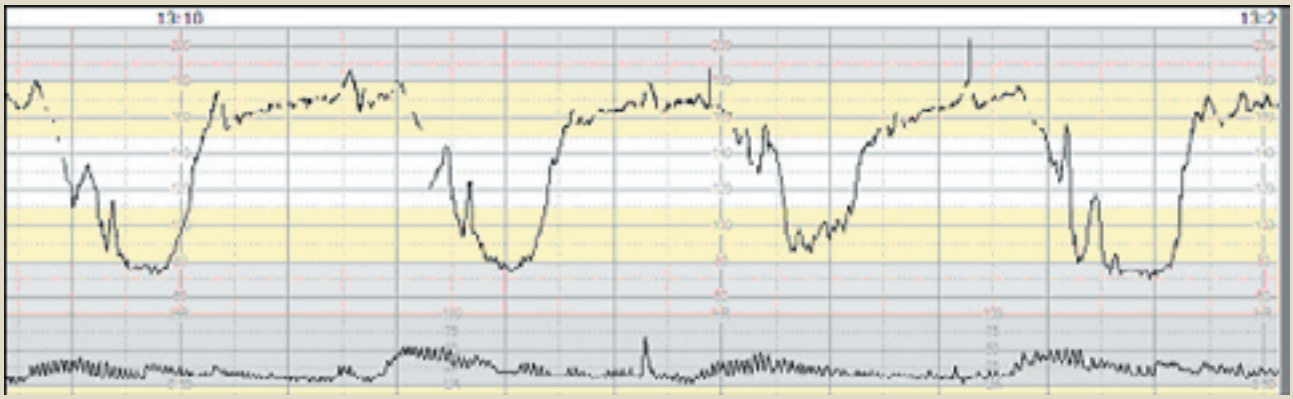
The intended use of this FHR classification system is to suggest clinical conditions in which adjunctive use of ST waveform changes may aid the interpretation of specific non-reassuring FHR patterns.

Samples of EFM tracings

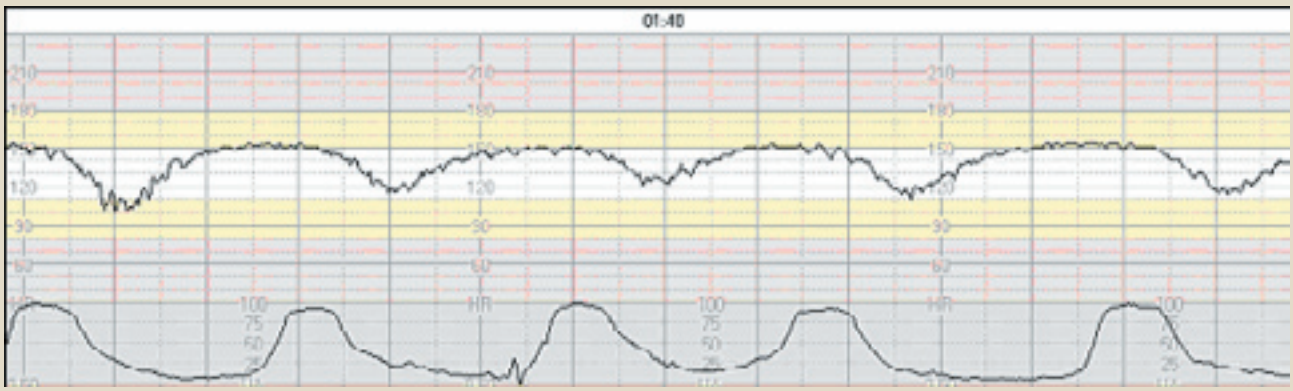
Sample of Reassuring Fetal Heart Rate
Early decelerations



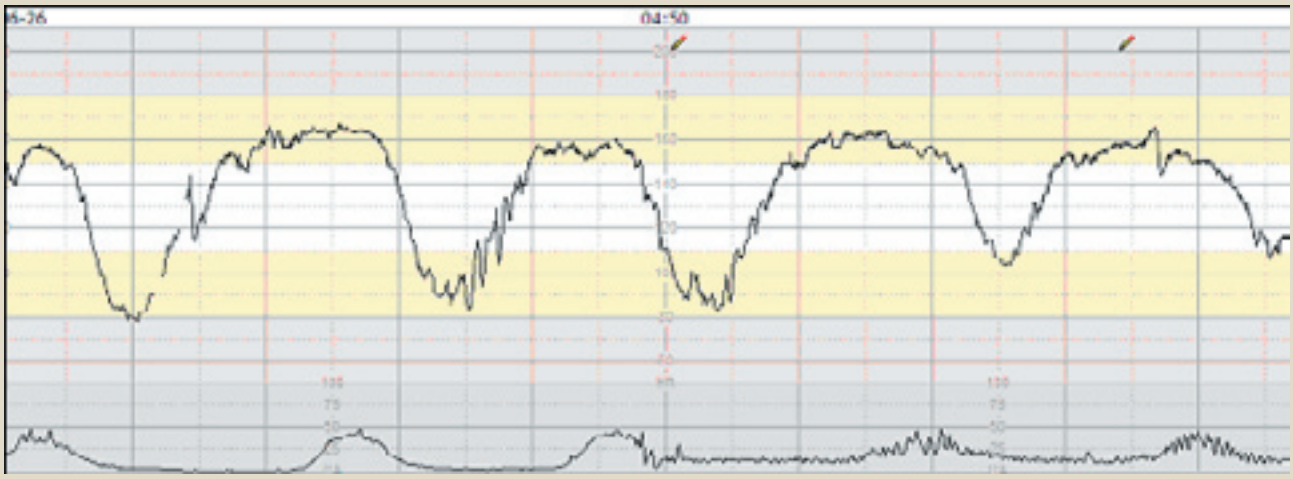
Sample of Non-Reassuring Fetal Heart Rate Grade I
Complicated variable decelerations



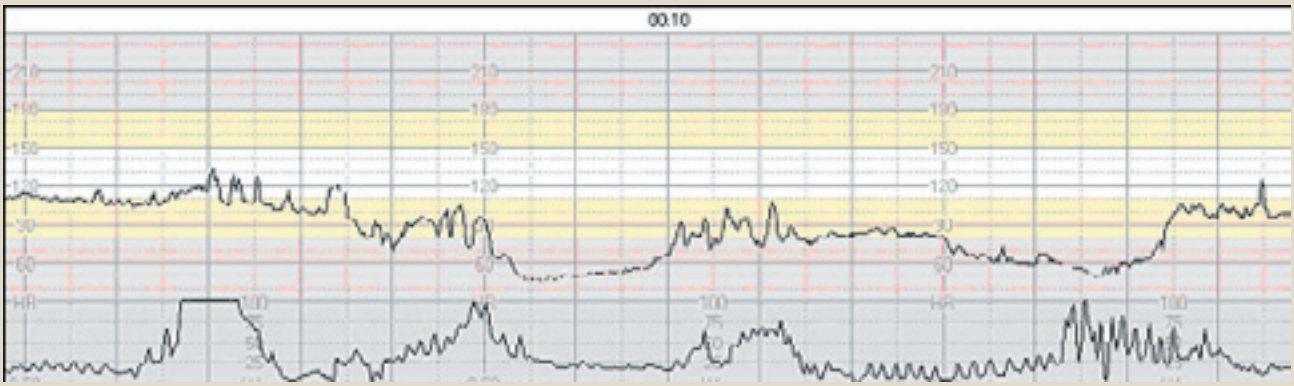
Late decelerations



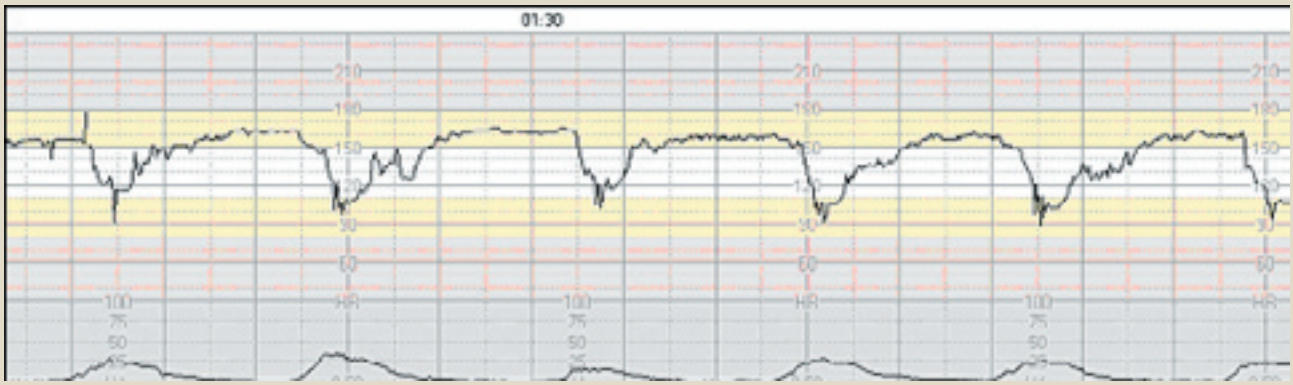
Late decelerations



Prolonged decelerations



Sample of Non-Reassuring Fetal Heart Rate Grade 2



Fetal ECG Physiology

Introduction

The task of obstetric care is safe delivery for mother and child. When the EFM was introduced 30 years ago, it was assumed that electronic fetal monitoring would identify fetus-es affected by intrapartum asphyxia, resulting in early intervention and a reduction in cerebral palsy. Unfortunately, this has not been the case, as a large number of fetuses show changes in fetal heart rate without being asphyxiated. This has caused an increased rate of intervention and uncertainty about the clinical value of EFM. This uncertainty about data interpretation has caused abnormal EFM patterns to be missed and babies to suffer from intrapartum asphyxia. It is clear, however, that the EFM is very good at identifying the normal healthy fetus but is unable to provide diagnostic information on the degree of hypoxic stress.

Oxygen deficiency is a known cause of neurological damage. What opportunity is there to monitor the fetus and to intervene appropriately to avoid hypoxic damage? How should we assess the events of labor to distinguish between a normal and an abnormal situation?

The aim should be to intervene when required and not just "in the event of". It is important that emergency clinical action is undertaken based on strict guidelines, as uncertainty about data interpretation creates problems with the management of emergency situations. As a result, we may see an increase in interventions which may be overly hasty and in themselves risk damage.

Fortunately, intrapartum asphyxia with neurological damage or perinatal death is unusual and we have to monitor many healthy fetuses to find those that are suffering. However, the consequences of a damaged child are such that, for humanitarian, social and economic reasons, we should continue to develop our ability to identify the baby that is suffering from intrapartum asphyxia.

The STAN® concept is based on the unique ability of the ST interval to reflect the function of the fetal heart muscle (myocardium) during stress tests. In adult cardiology, ST analysis is performed to assess and diagnoze myocardial insufficiency. During labor, we can assess the condition of the fetus from the only routinely available fetal signal, the electrocardiogram. It is important to recognize that the fetal heart and brain are equally sensitive or insensitive to oxygen deficiency and, as a result, the information relating to myocardial function provides an indirect measurement of the condition of the fetal brain during labor.

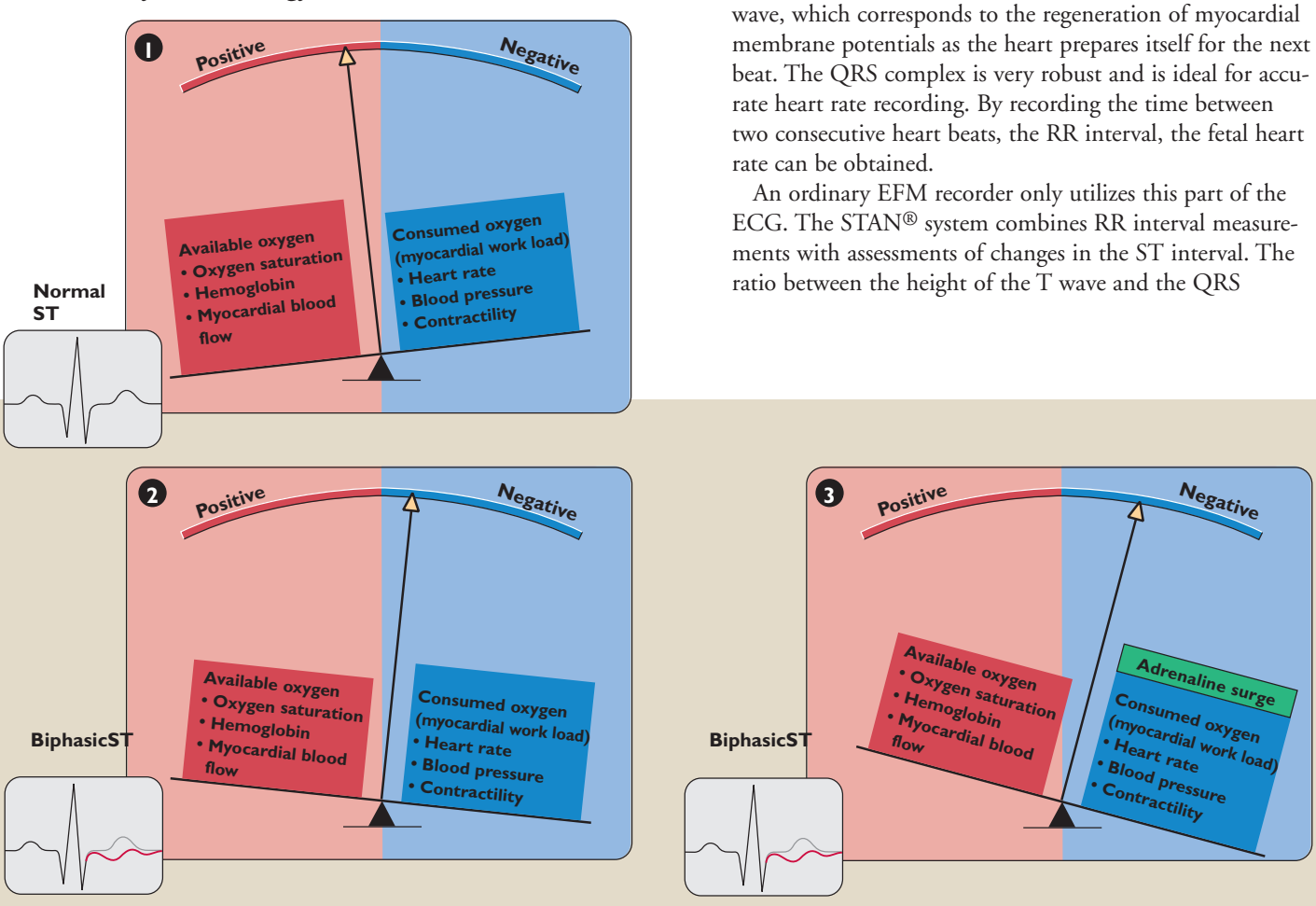
Intervention according to ST wave form analysis has been found appropriate and it results in a significant reduction in the number of acidotic babies. At the same time, unnecessary interventions are avoided.

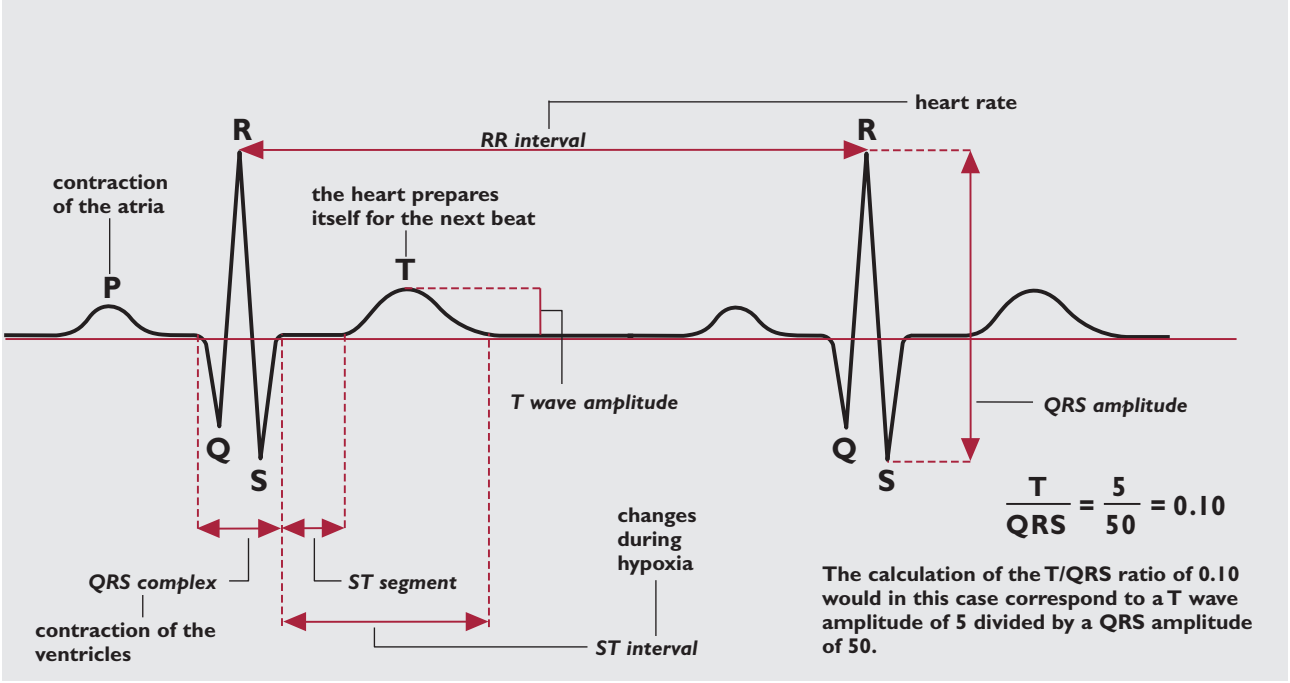
ECG complex

A scalp electrode is required to obtain the fetal ECG. The ECG is a reflection of the electrical currents generated by the heart muscle, the myocardium. The first waveform, the P wave, corresponds to the contraction of the atria. The next phase is the contraction of the ventricles, which corresponds to the QRS complex. The final component is the T wave, which corresponds to the regeneration of myocardial membrane potentials as the heart prepares itself for the next beat. The QRS complex is very robust and is ideal for accurate heart rate recording. By recording the time between two consecutive heart beats, the RR interval, the fetal heart rate can be obtained.

An ordinary EFM recorder only utilizes this part of the ECG. The STAN® system combines RR interval measurements with assessments of changes in the ST interval. The ratio between the height of the T wave and the QRS

Myocardial energy balance





amplitude provides us with the T/QRS ratio, which serves as an accurate measurement of changes in T wave height.

Myocardial energy balance

The ability of the fetal heart to pump blood is dependent on a balance between energy-yielding and energy-consuming processes. This energy balance could be illustrated as a set of scales. In one of the scales, we have the amount of energy available and in the other the amount of work performed. In normal circumstances, the amount of available oxygen is always greater than the amount consumed. The fetal heart is then utilizing oxygen-dependent, aerobic metabolism, the energy balance is positive and the ECG shows a normal ST waveform.

The amount of available oxygen is dependent on oxygen saturation, the concentration of hemoglobin in the blood and myocardial blood flow. Oxygen consumption is regulated by myocardial work load. The work load is affected by heart frequency, the blood pressure against which the heart pumps blood and the contractility, i.e. the force of heart muscle contractions.

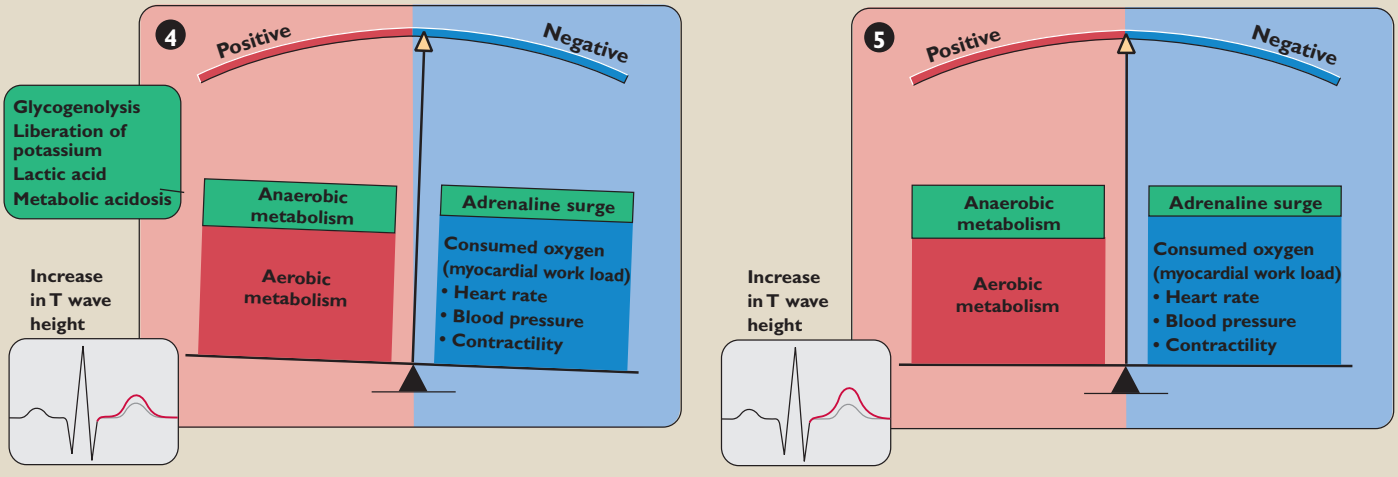
In the event of hypoxia, the amount of available oxygen decreases at the same time as the work load is maintained. This causes a negative energy balance. The ECG now changes because of myocardial hypoxia and we see a biphasic ST. A biphasic ST is identified from a downward slope of the ST segment.

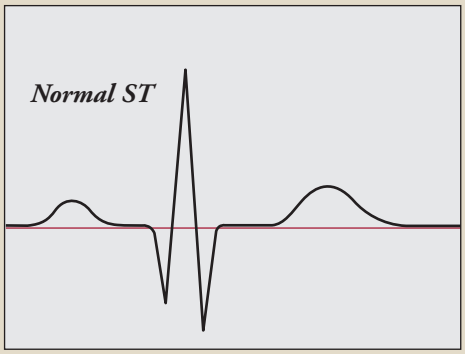
The fetus now normally reacts with a surge of adrenalin, which activates the myocardium still further. As a result, the energy balance threatens to become even more negative and additional energy is required.

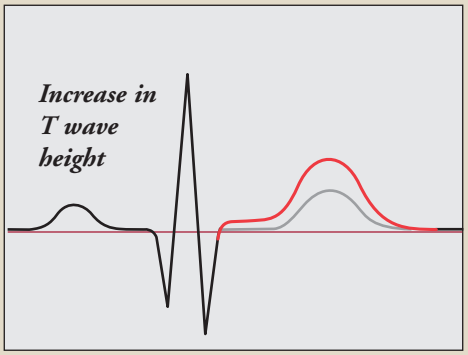
Adrenalin activates beta-receptors, which in turn activate cyclic AMP, the phosphorylase enzyme is activated, and stored sugar is released. The utilization of stored glucose (glycogen) is called glycogenolysis. With the release of glycogen, potassium ions are set free and, as a result, the amplitude of the T wave increases. At the same time, some lactate is produced and contributes to the development of metabolic acidosis.

The energy balance now regains its equilibrium as the aerobic metabolism is supported by anaerobic metabolism. As the rate of glycogenolysis increases, there is an increase in T wave amplitude.

Biphasic ST segments could be noted during the initial phase of hypoxia, when the fetal heart has not yet had time to respond to an acute hypoxic event, or it may appear if the fetus is not capable of responding to hypoxia for different reasons. The







increase in T wave amplitude requires active adaptation to hypoxia, while biphasic ST serves as an indicator of the direct depressant effect of hypoxia on myocardial function.

ST waveforms

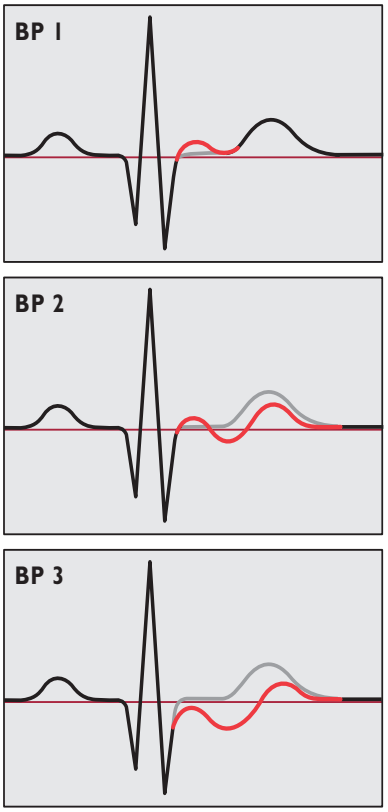
A horizontal or upward leaning, positive ST segment and a T wave height that is stable and does not increase define a normal ST. This indicates a positive energy balance with aerobic myocardial metabolism. As long as there is a positive energy balance in the central organs, the fetus is capable of handling the stress of labor.

When asphyxia becomes severe and long lasting, the ST waveform returns towards normal, in parallel with a markedly reduced ability by the fetus to respond. This also means that the same type of change in the ST interval should not be expected as asphyxia progresses, simply because the capacity of the fetus to utilize its defenses diminishes.

An increase in T wave amplitude is the classical reaction by a fetus responding to hypoxia. A fetus that responds with an adrenalin surge and myocardial anaerobic metabolism characterizes this reaction. This pattern signifies that the fetal metabolic defense is intact and the fetus thereby has the ability to handle hypoxia. The rate of increase in T wave amplitude depends on the amount of glycogen the fetus needs to utilize to maintain its myocardial energy balance.

A biphasic ST is defined as a downward-leaning ST segment. This pattern occurs in two situations. The first is when the fetal heart is exposed to hypoxia and has not had the opportunity to respond. The second is when the fetal heart has a reduced capacity to respond because it has been exposed to previous stress situations and resources are lacking or have already been utilized. Biphasic ST changes could also be noted with disturbances in heart muscle function, such as with infections or malformations. It appears that the premature myocardium may display more frequent biphasic ST events. The ability to respond to hypoxia with an adrenaline surge is reduced, as is the ability to utilize stored glycogen. Biphasic STs are divided into three categories; Grade 1 is a downward leaning ST segment with the entire segment above the baseline. Grade 2 means that an ST segment component now crosses the baseline and Grade 3 occurs when the whole of the ST segment is below the baseline.

A significant biphasic event occurs when there are more than two consecutive biphasic ECG complexes. With the progression of disturbance in myocardial function, a shift from biphasic Grade 1 to Grade 2 and 3 may be seen.



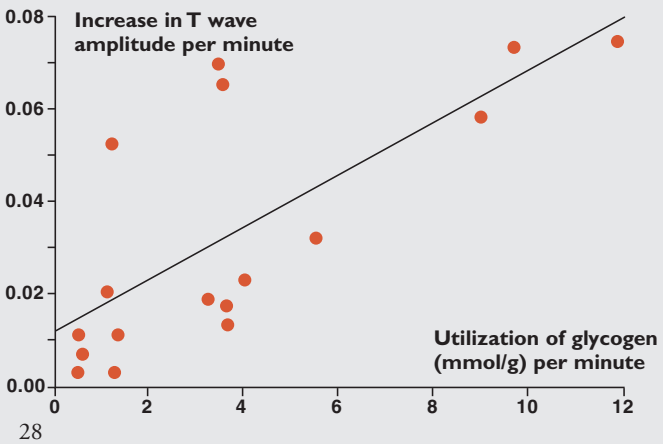
Biphasic ST grade 1

grade 2

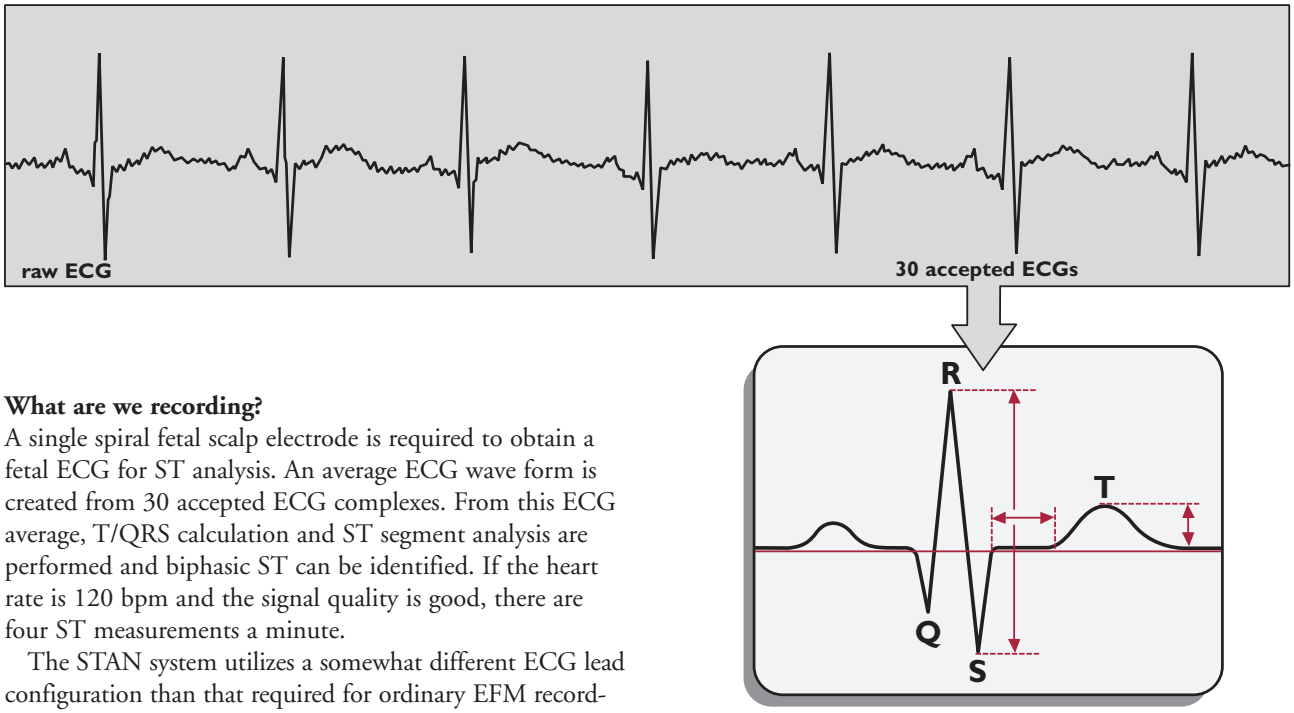
grade 3

The relationship between the rate of myocardial glycogenolysis and the rate of increase in T/QRS ratio during hypoxia

The graph illustrates the close relationship between myocardial workload and the T/QRS ratio during acute hypoxia. This is the key relationship behind the T wave rise. Obviously, some fetuses may display a rise in myocardial workload in response to an extra catecholamine surge due to the general stress of labor without being hypoxic. We may then record some rise in T/QRS with a normal reactive EFM tracing.



Fetal ECG Interpretation



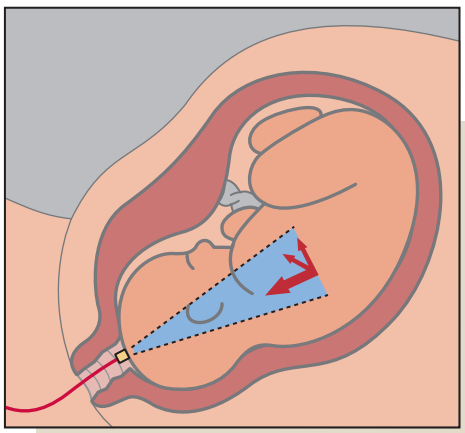
ECG average

What are we recording?
A single spiral fetal scalp electrode is required to obtain a fetal ECG for ST analysis. An average ECG wave form is created from 30 accepted ECG complexes. From this ECG average, T/QRS calculation and ST segment analysis are performed and biphasic ST can be identified. If the heart rate is 120 bpm and the signal quality is good, there are four ST measurements a minute.

The STAN system utilizes a somewhat different ECG lead configuration than that required for ordinary EFM recordings. All the experimental data have been based on the ECG recorded from the chest of the fetus and it could not be taken for granted that the scalp electrode would identify ST events. Recordings from the fetal lamb showed that our ability to identify ST changes depended upon where the exploring ECG electrodes were positioned. The crucial question of securing a consistent ECG signal that was sensitive to ST changes was solved by utilising a unipolar scalp ECG lead that has formed the basis of the STAN® system.

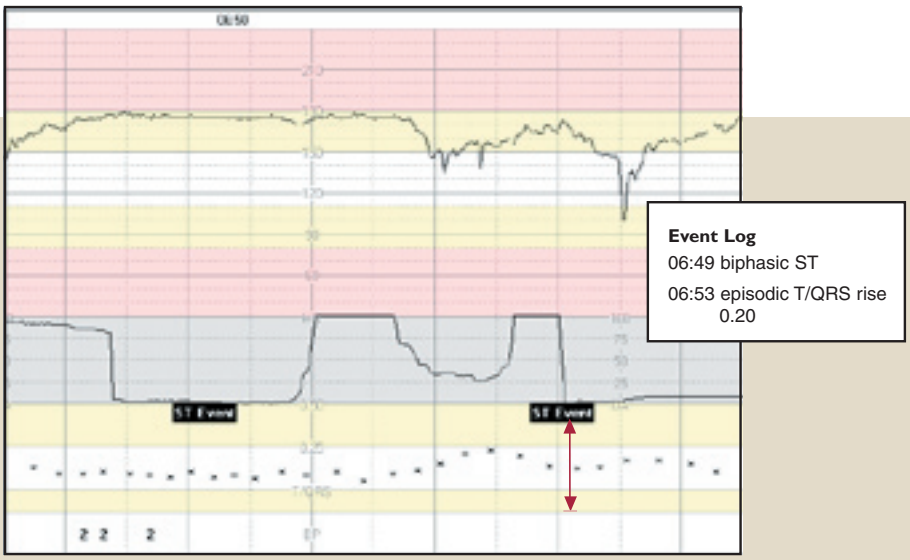
ST changes
The fetus usually displays a fairly stable T/QRS ratio throughout labor. There should normally be no marked ST elevations and no biphasic ST. In these circumstances, the Event Log will not display any messages about ST events. The lack of significant ST events indicates that the fetus is

ECG vectors



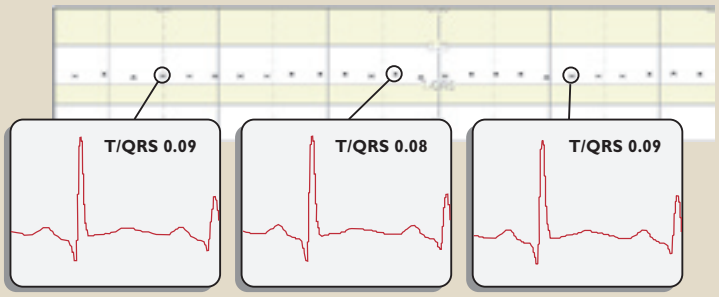
The unipolar ECG lead provides an opportunity to record changes in the ST waveform during labor. It identifies ECG changes occurring in the crown-rump axis of the fetus, the Y-lead, and enables the monitoring of ST events both in crown and breech presentations (data from K. Lindecrantz et al).

ST presentation



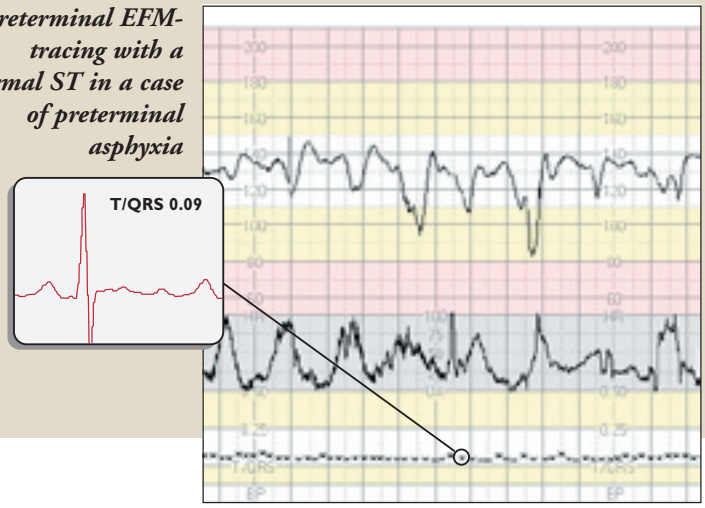
The T/QRS ratio is printed on a scale ranging from –0.125 to 0.50. The T/QRS ratio is plotted as a cross. Corresponding to each T/QRS, there is also the identification of biphasic ST. It is printed as the digits 1, 2 or 3, depending on the level of abnormality.

The STAN® system contains a log function that automatically identifies significant ST changes. The information is provided by the message, "ST Event", printed on screen. To obtain more information about the type and degree of abnormality, you have to enter the "Event Log" function. Here you will find the time of occurrence and the type of ST event being identified.



Normal ST

A preterminal EFM-tracing with a normal ST in a case of preterminal asphyxia



well in control of the situation and there is a positive energy balance within the myocardium.

Furthermore, ST analysis is based on our ability to record a situation in which the fetus is defending itself from hypoxia. It is, however, possible that a recording may start late in a hypoxic process when resources have already been utilized. This is a situation in which the T/QRS ratio might be constant. In these circumstances, the EFM pattern is always consistently abnormal with a complete lack of reactivity and variability, what we call a non-reassuring grade 2 (preterminal) tracing.

Episodic T/QRS rise

An episodic rise means that the T/QRS ratio rises and returns within 10 minutes. The degree of change in T/QRS reflects the fetal stress. If the increase exceeds 0.10, we regard it as a significant event and it is registered as an ST event. The Event log then reads the time and the extent of the increase. Please note that it is the change and not the actual T/QRS peak value that is relevant.

The clinical impact of a T/QRS increase depends on the EFM pattern. When the EFM tracing is reassuring, an increase in T/QRS can be accepted. This is not the case when there is a non-reassuring tracing. Physiologically speaking, an episodic T/QRS increase corresponds to short-lasting hypoxia in which the fetus is forced to utilize anaerobic metabolism to support heart function. It signals a situation of distress, although short-lasting but still providing significant information of a reduced placental reserve.

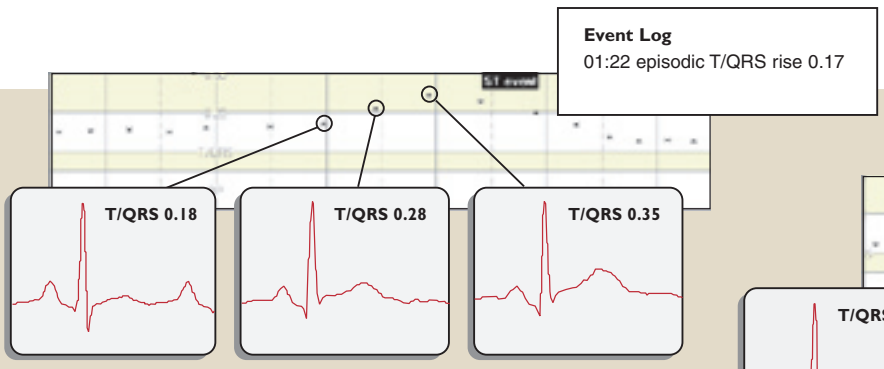
Baseline T/QRS rise

A baseline rise means that the increase in T/QRS ratio lasts for more than 10 minutes. A baseline T/QRS rise of more than 0.05 is regarded as significant and is indicated as an ST event. The Event log provides information about the change and time of occurrence.

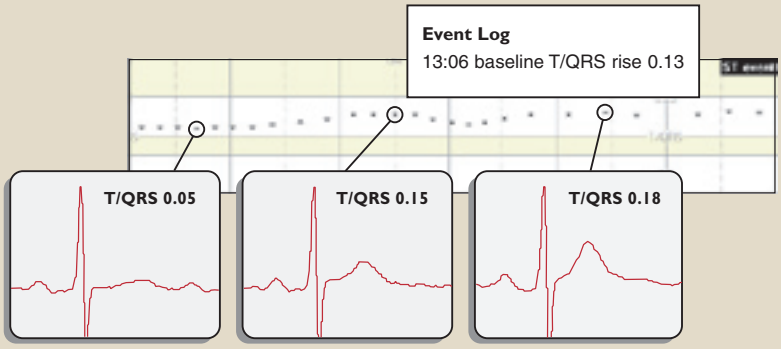
The baseline T/QRS rise appears in a situation in which the fetus has to respond to hypoxia with anaerobic metabolism. There is persistent stress and no opportunity for recovery. We may see a baseline T/QRS rise that may progress for hours with a very slow increase in T/QRS. However, it is more common to note an increase that occurs during the space of a couple of minutes but then becomes persistent. Some healthy fetuses responding to the stress and strain of labor display an increase in baseline T/QRS with a normal, reactive EFM tracing.

Biphasic ST

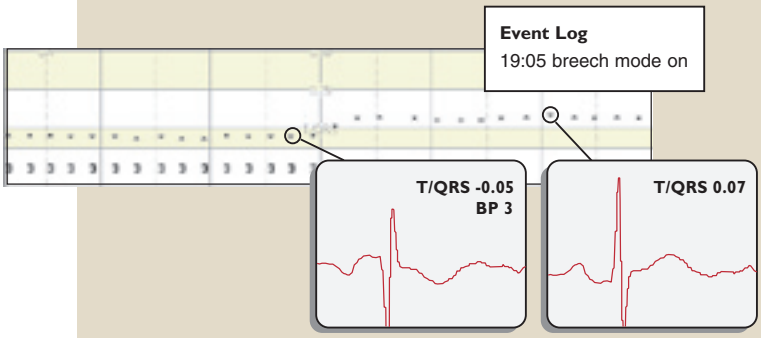
STAN® automatically identifies an abnormal ST segment. These abnormalities are called biphasic STs and they are divided into three grades, depending on how much the ST segment differs from the norm. Grade 1 is a downward-leaning ST segment above the baseline, Grade 2 is an ST segment that cuts across the baseline and Grade 3



Episodic T/QRS rise

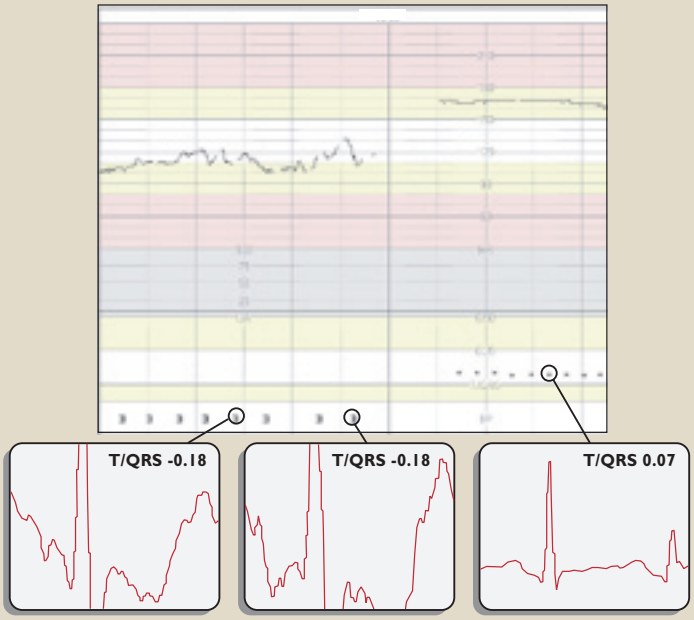


Baseline T/QRS rise



A breech recording

A case of 5 minutes of erroneous data as the breech mode was not activated in a breech delivery.



Maternal ECG

There is a risk of the maternal ECG to be recorded if the scalp electrode is positioned on the cervix. This situation could easily be identified as the averaged maternal ECG complex will not display a P wave.

corresponds to a situation in which the entire ST segment is below baseline. Grade 2 and 3 signify an abnormality, which becomes significant if repeated. The Event log displays the time of occurrence and the text "biphasic ST" is printed. There is no need for interventions in the event of biphasic ST Grade 1, whereas repeated episodes of biphasic ST Grades 2 and 3 should be regarded as a sign of abnormality.

They indicate a situation in which the fetal heart has not had time to react or respond to hypoxia or does not have the capacity to react. They may also be seen with infections and cardiac malformations. The immature fetus appears to display biphasic ST more regularly during labor.

Special ECGs

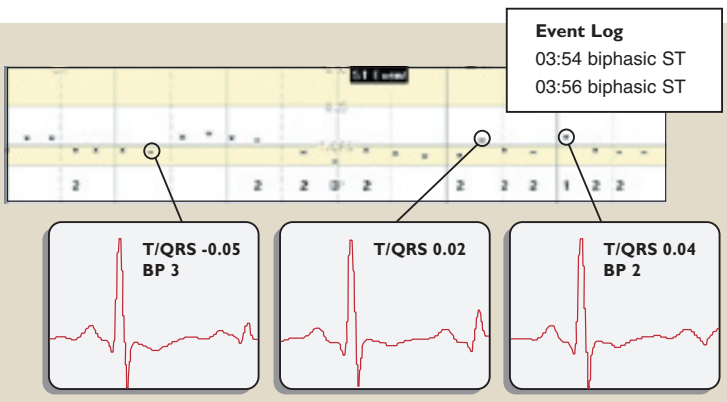
The average ECG waveform should always be examined at the start of the recording. If a scalp electrode has been placed on a breech, the ECG will be recorded upside down and an ECG waveform with a negative P wave and negative ST will be seen. The STAN® system includes a special feature whereby the ECG is turned upside down during a breech labor, thereby permitting standard ST analysis.

If the scalp electrode is attached to the cervix or to a dead fetus, there is a risk that the maternal ECG will be recorded. This ECG waveform will have a different appearance. It will not display a P wave, the QRS complex will be wider and it will coincide with the maternal pulse.

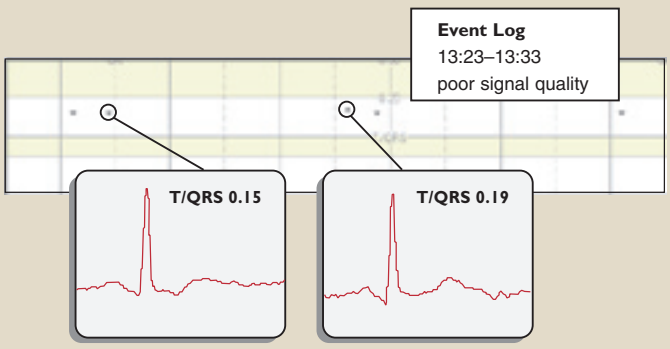
Poor signal quality

Good signal quality is required for ST analysis. If the scalp electrode is applied through the fetal membranes, or if the electrode is touching the cervix or the vaginal wall, there may be signal disturbances. If there is poor signal quality, ST events will not be detected. The system automatically identifies poor signal quality and informs the user. The time at which the signal becomes poor and at which the signal quality is recovered is shown in the Event log.

Automatic ST waveform assessment is not performed if there is a delay between subsequent T/QRS plots. However, the T/QRS ratios presented on screen and paper are accurate and may provide additional information. It makes sense to improve the situation by replacing a scalp or skin electrode rather than spending time and energy trying to interpret poor signal quality data.



Biphasic ST



Poor signal quality

STAN® simplified clinical guidelines

The aim of the STAN® system is to provide continuousinformation about the ability of the fetus to respond to the stress and strain of labor. The specific ST information should be used together with EFM tracing. In principle, a normal reactive fetal heart rate pattern tells us that the fetus is well in control of the situation. When there are changes in the fetal heart tracing, ST waveform analysis provides additional information about the severity of the stress and the clinical guidelines provide recommendations for clinical action. These guidelines are only valid for a term fetus, that is, a pregnancy of more than 36 weeks gestation.

When the guidelines indicate an adverse situation, it is necessary to intervene. In most cases, operative intervention is recommended. However, if a good reason for fetal stress can be identified, such as over-stimulation or maternal hypotension, these causes should obviously be dealt with first. During the second stage of labor, intervention always means immediate operative delivery.

A completely normal fetal heart rate tracing means that the fetus has the situation under control and we can accept ST changes. A healthy fetus in particular may react with arousal reactions in which an elevation of the T/QRS ratio lasting some 20 to 30 minutes would be seen. This is a sign of health and shows that the fetus has the capacity to react and respond.

If there is a non-reassuring tracing grade 1 and an episodic T/QRS rise of more than 0.10, intervention is indicated. In the case of a more prolonged increase in T/QRS ratio, a baseline T/QRS rise, in conjunction with a non- reassuring tracing, less of an ST change is required as more persistent stress now can be observed. The cut-off is 0.05 for a baseline T/QRS rise. A change in baseline T/QRS of this kind during the second stage of labor with active pushing should always result in immediate delivery. An intervention is also required when biphasic ST changes appear in association with a non-reassuring tracing. These biphasic ST waveform changes become significant when they have lasted for more than two minutes continuously or if there are repeated episodes of grouped biphasic ST Grade 2 or 3.

A preterminal tracing should always result in intervention, regardless of the ST.

ST waveform analysis is based on our ability to record changes in the fetal electrocardiogram, such as a rise in T/QRS ratio or the appearance of biphasic ST. It is therefore important that the recording starts before the fetus has utilized all its resources. During the second stage of labor hypoxia may occur very rapidly. So, if you decide to monitor only the second stage, it is recommended that the STAN® recording should start during the end of the first stage of labor.

Furthermore, when the STAN® system is started, the event log requires time, the length of which depends on signal quality, before it is able to identify the baseline from which subsequent changes may be identified.

A non-reassuring fetal heart rate tracing grade 1 should only be allowed to exist for a maximum of 90 minutes during the second stage of labor. After this time period, the fetal acid-base buffers may have been utilized to such an extent that acute hypoxia may not be handled accurately.

Fetal defense

The ability of the fetus to handle hypoxia depends on several different factors. This ability is optimal if the fetus has not been exposed to previous stress. The response also depends on the severity of hypoxia, the rate at which it appears and the duration. A healthy fetus exposed to oxygen deficiency reacts forcefully and a marked episodic T/QRS rise may be seen during the initial phase.

A fetus exposed to more long-lasting stress may not respond with the same forceful reaction. Biphasic ST may be the initial response with or without an increase in baseline T/QRS. The latter may also occur by itself. A fetus suffering from long-term distress may only display biphasic ST changes and even a slight T/QRS increase with continuous and progressive

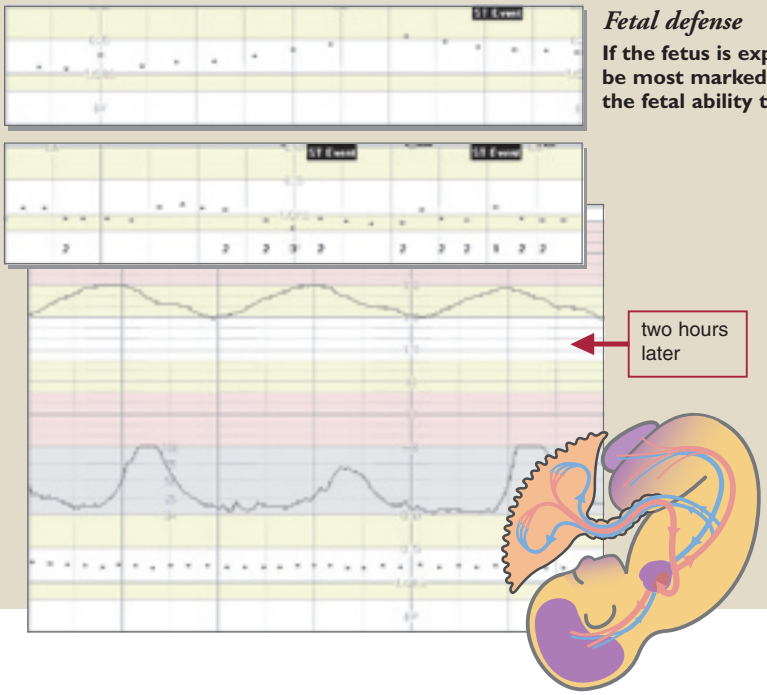
STAN Simplified Clinical Guidelines

<table>
<tr><th></th><th>Reassuring FHR</th><th>Non-reassuring FHR, grade I</th><th>Non-reassuring FHR, grade 2 Preterminal FHR</th></tr>
<tr><td>No ST change</td><td>“Routine Management”
Continued observation</td><td>Expectant management in first stage of labor.
Delivery within 90 minutes during 2nd stage of labour</td><td rowspan="4">Resuscitation and delivery should be undertaken as soon as possible regardless of any ST changes</td></tr>
<tr><td>Episodic T/QRS rise
(>0.10* and duration < 10 min)</td><td>“Routine Management”
Continued observation</td><td rowspan="3">Delivery should occur within 30 minutes in 1st stage of labor
Delivery should occur as soon as possible during 2nd stage of labor</td></tr>
<tr><td>Baseline T/QRS rise
(>0.05* and duration < 10 min)</td><td>“Routine Management”
Continued observation</td></tr>
<tr><td>Biphasic ST
If 2 biphasic log messages**</td><td>Closer observation</td></tr>
</table>

*Compared to baseline T/QRS **BP's grade 2 and 3 are regarded as significant

Recommendations for intervention using FHR patterns and ST waveform changes

Non-reassuring, Grade 2 (preterminal) would prompt an expedited delivery without the need to consider ST data.
Non-reassuring, Grade 1 with episodic T/QRS rise, baseline rise, or repeated biphasic ST pattern would mandate delivery within 30 minutes or less.
At start-up and when there is a decrease in signal quality with discontinous T/QRS ratios, manual data analysis is required.

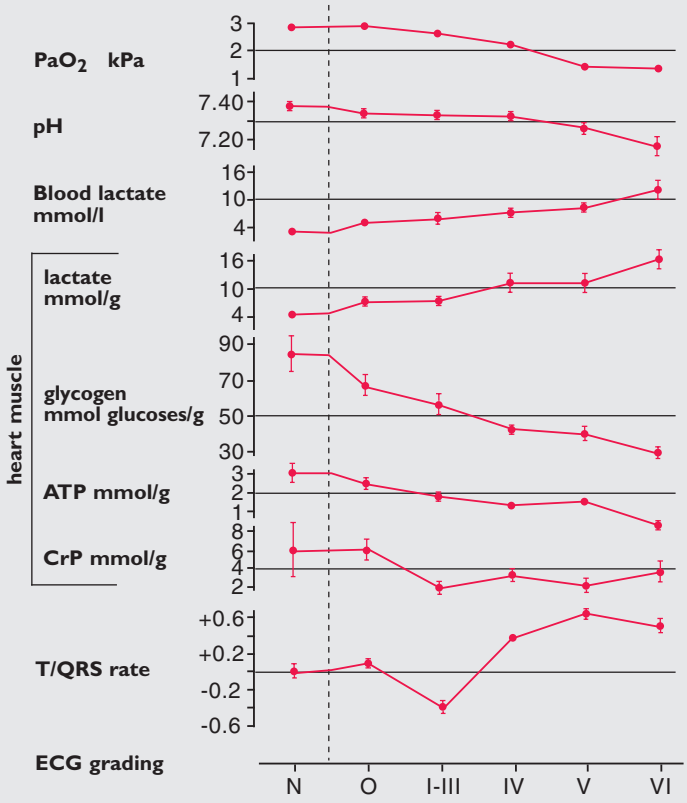


Fetal defense

If the fetus is exposed to persistent hypoxia, the ST changes will be most marked initially and we would expect some decrease as the fetal ability to maintain its responses is reduced with time.

Changes in myocardial energy-rich substrates during hypoxia in the term lamb fetus

The figure illustrates the observations made when serial biopsies from working fetal sheep hearts were analysed for their content of energy-rich substrates. The fetal ECG was scored using both the T/QRS ratio and a scoring system where grade I-III identified biphasic/negative ST changes, grade IV-V; a progressive increase in T amplitude and grade VI; a decrease in T amplitude. In the latter situation, the stores of myocardial glycogen and energy-rich phosphates were depleted. Please note that myocardial lactate is accumulated more rapidly then plasma lactate.



fetal heart rate tracing abnormalities is a sign of significant hypoxia. If a fetus is exposed to hypoxia and reacts with ST interval changes, the reaction is usually more pronounced initially and the reaction could be less marked later on if hypoxia progresses and the fetus becomes more affected. The appearance of less pronounced ST changes or even the disappearance of ST changes should not be interpreted as a sign of recovery by the fetus. The purpose of the STAN® recording and these guidelines is to identify a fetus that is not responding normally to the stress of labor.

We may also have a situation in which a fetus exposed to long-term stress decides to "quit" and hibernate. The reduced oxygen and nutritional supply causes the fetus to reduce its metabolic demands as far as possible, which means that even the fetal heart reduces its activity. In a situation of this kind, it is not sure that ST changes will emerge, but fetal heart rate variability and reactivity have disappeared and a preterminal fetal heart rate tracing would be seen.

Fetal scalp sampling

Fetal scalp pH is regarded as a valuable tool for assessing the condition of the fetus during labor in parallel with the fetal heart rate tracing. The technique of obtaining a fetal scalp sample requires the rupture of fetal membranes and a cervical dilatation of at least two centimetres. An amnioscope is introduced, allowing undisturbed access to the presenting part of the fetus, and a small incision is made in the skin and a drop of blood is allowed to fill a capillary tube. A fetal pH machine can determine the pH of the blood. Modern blood gas machines permit full acid-base assessments of a capillary sample.

What are the advantages and disadvantages of fetal scalp sampling? There is always a risk of the sample being contaminated with amniotic fluid or maternal blood and the contact between the drop of blood and air immediately causes a lowering of carbon dioxide and prevents the calculation of metabolic acidosis.

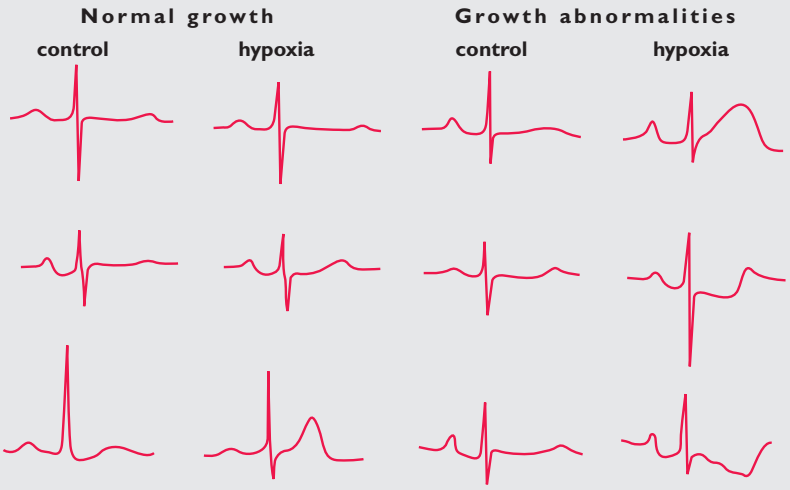
However, scalp pH is recommended when there are doubts about the fetal condition and there is difficulty in interpreting the fetal heart rate tracing. The development of STAN® provides us with new and continuous information about the condition of the baby.

Apart from only providing instantaneous information there are other limitations to the information obtained by a fetal scalp sample. The sample is obtained from blood which originates from the peripheral tissues. This makes the sample more difficult to interpret because of the rapid accumulation of carbon dioxide that occurs with a reduction in not only fetal placental blood flow but also as a result of reduced peripheral blood flow. A reduction of this kind is linked with all vagally mediated decelerations and a locally generated accumulation of carbon dioxide and respiratory acidemia takes place. Not until later can we expect respiratory acidemia to affect the fetal blood in general.

The advantage of the scalp pH is that this is objective information that could be used clinically. However, when using such information, one should be aware that scalp pH only

provides instantaneous information from a low-priority tissue. Furthermore, there is a risk that, in the event of a normal pH, we would regard the fetal situation as being under control in spite of fetal heart rate and ST changes. A blood pH on its own is always dominated by the respiratory component. Metabolic acidosis develops in the tissues and time is required for the free hydrogen ions to be transported from the tissues into the blood compartment. In the early stages of metabolic acidosis, we should expect a scalp pH to be within the normal range. If the STAN® guidelines indicate the need for intervention, no additional information is obtained from fetal scalp sampling. Under these circumstances, fetal scalp sampling may delay clinical action and during the second stage in particular urgent action is required.

ST changes during hypoxia in the normal and growth retarded guinea-pig fetus



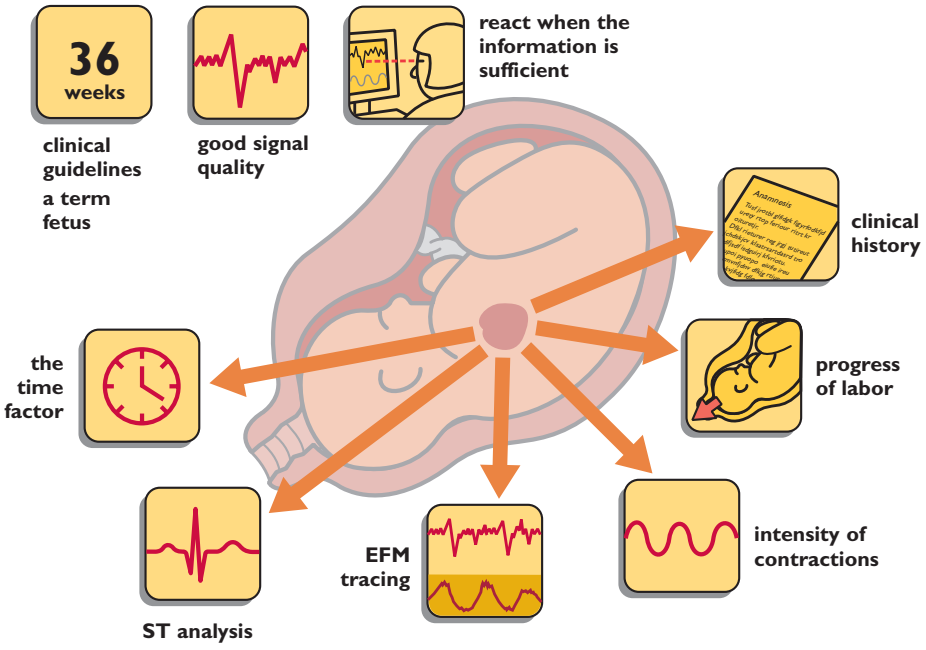
ECG patterns recorded in normal and growth retarded guinea-pig fetuses before and during hypoxia (data from C. Widmark).

Surveillance

The basic rules for fetal surveillance with STAN® are as follows.

- The clinical guidelines should only be used when monitoring a term fetus; that is, a pregnancy lasting more than 36 completed weeks. Gestational age may affect ST changes. For instance, the immature fetus is less able to utilize its glycogen stores due to the lack of a myocardial enzyme.
- Good signal quality is required to make an accurate assessment of the condition of the fetus and poor signal quality warrants appropriate measures.
- It is important to recognize that intervention should take place when the information from the fetus is sufficient. A preterminal tracing with a total lack of variability and reactivity is very abnormal and no further information is required for clinical intervention.

Basic rules for fetal surveillance



In stage 2 with active pushing, if STAN-recording (FHR+ST) indicates that the baby is being exposed to significant hypoxia, delivery should be performed immediately (within 20 minutes) to avoid metabolic acidosis.

When the decision to undertake an operative delivery is taken, it is recommended to retain the scalp electrode and continue monitoring. Fetal monitoring should not be done in isolation. The data that are obtained should be linked to other factors such as:

- clinical history
- progress of labor
- intensity of contractions
- fetal heart tracing
- the appearance or lack of ST changes obtained by ST analysis
- the time factor

All of these parameters should be observed when assessing the fetal situation. The issue of when to monitor a fetus during labor is under debate. Continuous recording throughout labor is only necessary in a small number of babies. In the event of abnormalities such as meconium or slow progress, further information is required and a scalp electrode is recommended. If any drugs are administered there is also a need for further information. By far the greatest risk arises during active pushing in the second stage. The fetus is exposed to intense forces as the contractions increase in strength and frequency. The second stage of labor should always be regarded as a high-risk situation requiring continuous surveillance. A STAN® recording should start no later than the end of the first stage and should continue throughout the second stage of labor.

Assessment of the child

What do we want to know?
Fetal monitoring during labor is used to identify fetal hypoxia. When the child is born, we need to know the extent to which the baby has suffered. At the same time, we need to know if additional intervention is required during the neonatal period, such as further surveillance or specific treatment.
The main issues when it comes to hypoxia are as follows.
- How marked was it?
- How long did it last?
- Are there any reasons to believe that the baby requires additional help to support neonatal adaptation?

Assessment methods
The methods we use to assess the condition of the child are Apgar scores, cord acid-base analysis and the occurrence of neonatal complications. The combination of these parameters enables us to assess the condition of the child and take the appropriate action.

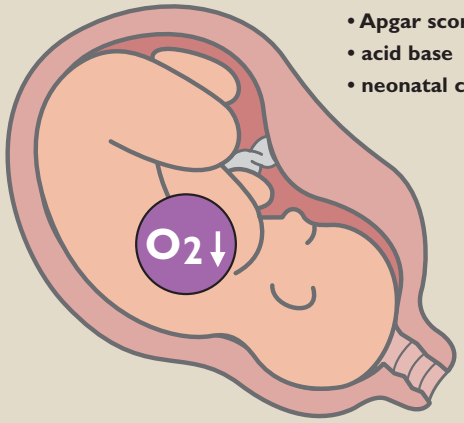
Apgar scores
Virginia Apgar created the Apgar scoring system in 1953. The initial aim was to assess how different anesthetic drugs given to the mother would affect the condition of the child at birth. The aim was not to use the scores to assess the degree of asphyxia. The scoring system is based on five parameters: heart rate, breathing, skin color, muscular tone and reflex irritability. Each parameter can be given a score of 0 to 2 and the maximum score is 10. The child should be given a score at the age of 1, 5 and 10 minutes.

There is an association between asphyxia and low Apgar scores, but most babies born with low Apgar scores do not suffer from asphyxia. There are many different reasons for low Apgar scores apart from asphyxia, such as immaturity, labor trauma, drugs, infections, the activation of reflexes through manipulation of the upper airways, meconium aspiration or carbon dioxide narcosis.

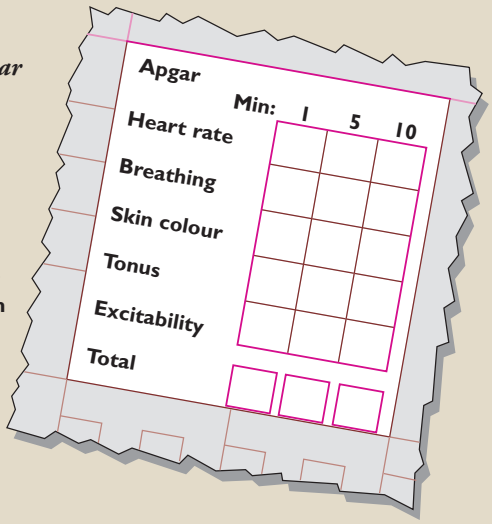
Acid base
The physiology of acid base
The appearance of metabolic acidosis or respiratory acidemia is the result of a decrease in placental blood flow with a reduction in gas exchange. Respiratory acidemia is caused by a decrease in the transport of carbon dioxide from the fetus to the mother. Carbon dioxide is produced in large amounts in the cellular energy-yielding metabolic processes and a continuous placental blood flow is required to avoid carbon dioxide accumulation. If this occurs, carbon dioxide forms hydrogen ions, some of which are freed and cause respiratory acidemia with a rapid decrease in pH.

A reduction in oxygen saturation, which is the other result of a decrease in placental gas exchange, has completely different consequences to those of carbon dioxide accumulation. A reduction in fetal oxygenation with hypoxia means that the fetus is reacting with anaerobic metabolism. This takes place in the tissues and lactic acid is produced. It is split into lactate and hydrogen ions, some of which are free and cause metabolic acidosis with a decrease in pH.

Methods to assess the condition of the baby



Reasons for low Apgar
- asphyxia
- immaturity
- labor trauma
- drugs
- infections
- activation of reflexes
- meconium aspiration
- carbon dioxide narcosis

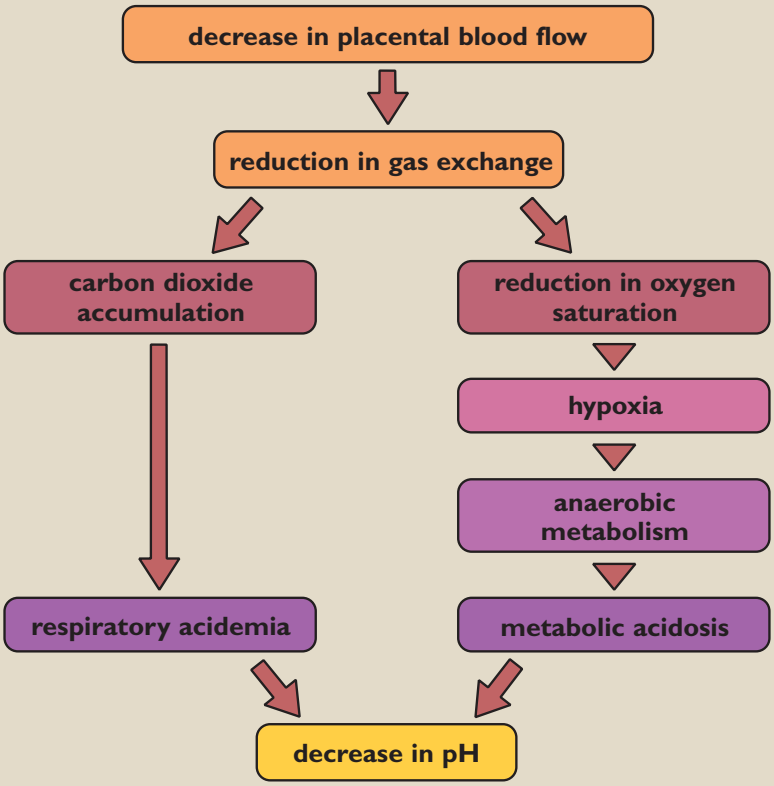


Respiratory acidemia and metabolic acidosis have different origins and mean different things to the fetus. Respiratory acidemia is part of normal delivery; it emerges rapidly and disappears rapidly with the first breath of air. Very high carbon dioxide concentrations may delay the first breath of air. A cry from the child is all that is needed and the carbon dioxide level rapidly decreases as carbon dioxide leaves with the child's first breath.

Metabolic acidosis carries with it a risk that the tissues will be affected. Metabolic acidosis requires time to develop and it remains for longer periods of time. There is an additional effect, which means that repeated episodes might be added to one another, thereby causing a reduction in the safety margins with a decrease in buffering capacity.

Let us take a look at the development of respiratory acidemia. The common cause is a decrease in fetal placental blood flow. This is most commonly caused by the compression of the cord vein. Initially, there is always enough oxygen and glucose to be used with normal metabolism, in other words aerobic metabolism. Apart from energy, carbon dioxide and water are produced. Due to the reduction in blood flow, these waste products accumulate in the blood. Carbon dioxide and water are very rapidly turned

Development of a respiratory acidemia and metabolic acidosis



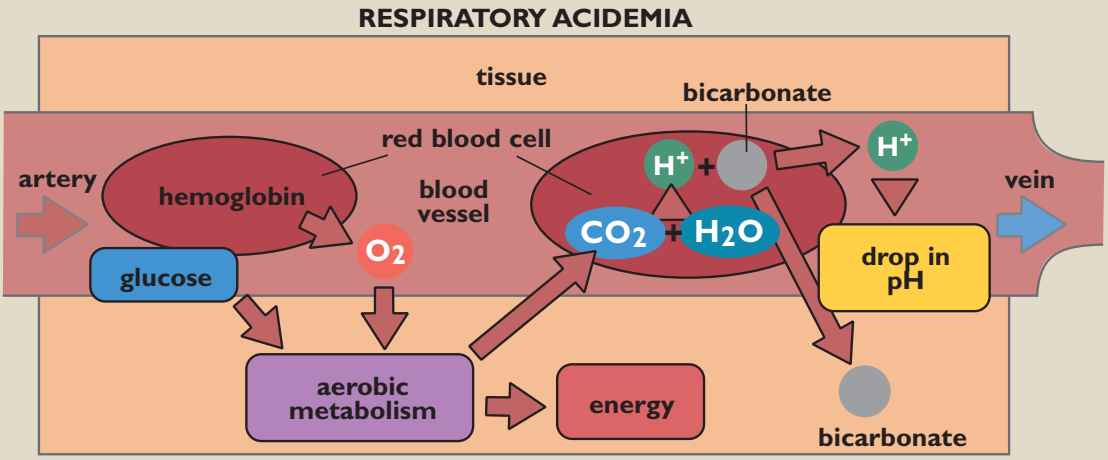
RESPIRATORY ACIDEMIA

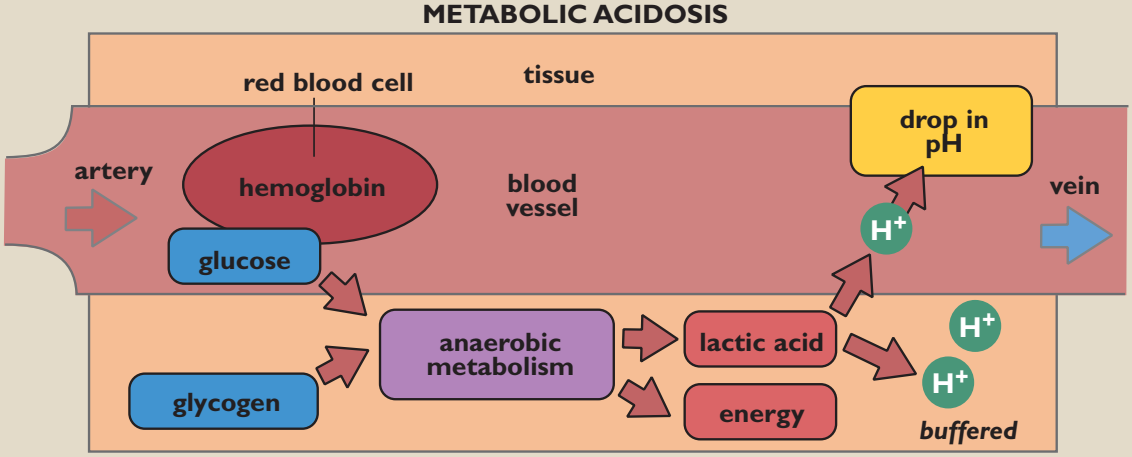
- is part of normal delivery
- emerges rapidly
- disappears rapidly
- may delay the first breath

METABOLIC ACIDOSIS

- risk that the tissues will be affected
- requires time to develop
- remains
- additive effect

Mechanisms behind respiratory acidemia and metabolic acidosis





into hydrogen and bicarbonate ions. The hydrogen ions are bound by hemoglobin. Normally, there is sufficient binding capacity, but, due to the slow blood flow, there is a lack of hemoglobin-buffering capacity and free hydrogen ions enter the plasma, causing a drop in pH. Bicarbonate ions are produced at the same time. They shift from the blood to the tissue where they serve as an additional buffer and protect the fetus from metabolic acidosis.

Metabolic acidosis occurs when insufficient oxygen is available for the tissues. The cells now react with anaerobic metabolism whereby glucose and glycogen are utilized. At the same time, energy is produced and lactic acid is generated as a waste product. Lactic acid is dissociated into hydrogen ions and lactate. Most of the hydrogen ions are buffered in the tissues, but some pass on to the blood stream and may cause a drop in pH. Obviously, metabolic acidosis is generated in the tissues and most free hydrogen ions exist outside the blood stream in the tissues where they are produced.

Metabolic acidosis means that the fetus has been utilizing some of its resources and there is a potential risk that the energy-yielding processes within the cell will be disturbed. Metabolic acidosis therefore constitutes a more relevant threat than respiratory acidemia. Much more is required from the child to handle metabolic acidosis and the neonatal adaptation process may be affected.

Peripheral and central metabolic acidosis

Hypoxia causes a redistribution of blood flow from peripheral to central organs. As a result of the marked reduction in peripheral low-priority organ blood flow, these tissues have to utilize anaerobic metabolism. Initial peripheral metabolic acidosis then occurs. A response of this kind is common during normal labor and a moderate increase in base deficit is seen.

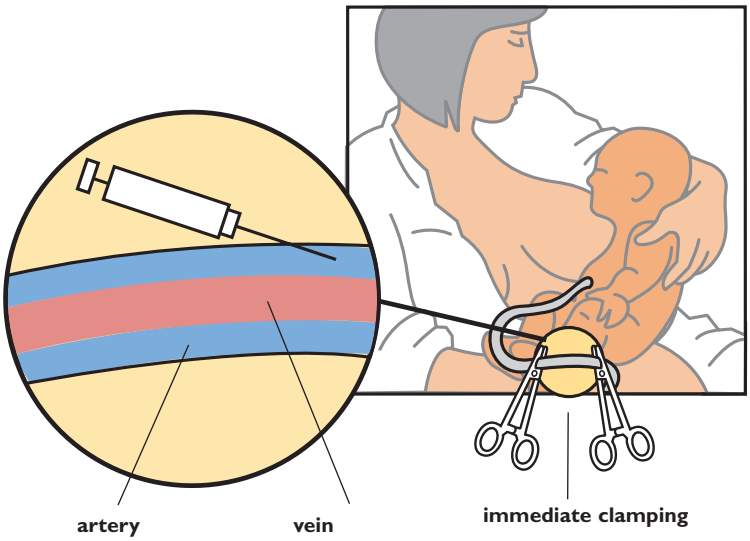
If hypoxia becomes more severe and prolonged, the central high-priority organs, such as the heart, the brain and the adrenals, may be affected. In these circumstances of central metabolic acidosis, the fetus is at risk of hypoxic damage.

Blood samples from the umbilical cord

Cord blood gas analysis requires accurate sampling techniques. The immediate clamping of the cord is most important. When the child takes its first breath, the lungs rapidly take over the placental function and the carbon dioxide concentration in the blood of the baby rapidly decreases. If this happens, there is no chance to calculate the degree of metabolic acidosis.

To what extent would early clamping affect the condition of the term neonate? Basically, the baby's blood is the baby's and the placental blood belongs to the placenta. It may not be an advantage for the baby to have an extra supply of blood, rather the opposite. An extra blood volume affects the neonatal adaptation in a negative way and the key symptoms related to late clamping are as follows.

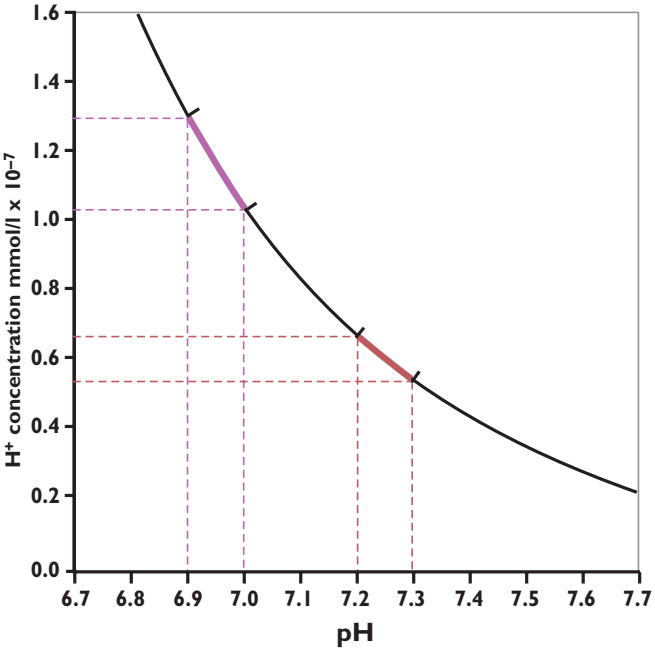
- Grunting during the first two hours.
- A risk for the central nervous system to be affected with delayed pulmonary adaptation and a risk for heart failure when the venous hematocrit exceeds >65%.



- Hyperbilirubinemia.
- Delay in oxygenation and carbon dioxide retention in the hypoxic child at birth.

Furthermore, the newborn baby is already suffering from a volume overload, as reflected by the rapid weight loss, which occurs during the first neonatal days.

So, there are no medical reasons for not clamping the umbilical cord at the time of delivery in the term infant. The clamping is performed and at least 10 cm of the cord is then sealed and set aside for subsequent sampling and



The pH indicates the concentration of free hydrogen ions in the blood. This graph shows the relationship between the pH and the free hydrogen ion concentration. This is a logarithmic relationship, which means that, when there is a drop in pH at a low range, for instance, between 7.0 and 6.9, there are twice as many free hydrogen ions generated as compared with a pH drop from 7.30 to 7.20.

blood gas analysis. The cord could be kept at room temperature for a short period of time but immediate sampling and analysis are recommended. Samples should be taken from both the artery and the vein and the needle should be introduced in an oblique fashion to enable blood to be withdrawn from the vessels.

BDecf (base deficit)

The degree of metabolic acidosis, as calculated by the BDecf, provides an estimate of the extent to which the baby has been exposed to intrapartum hypoxia.

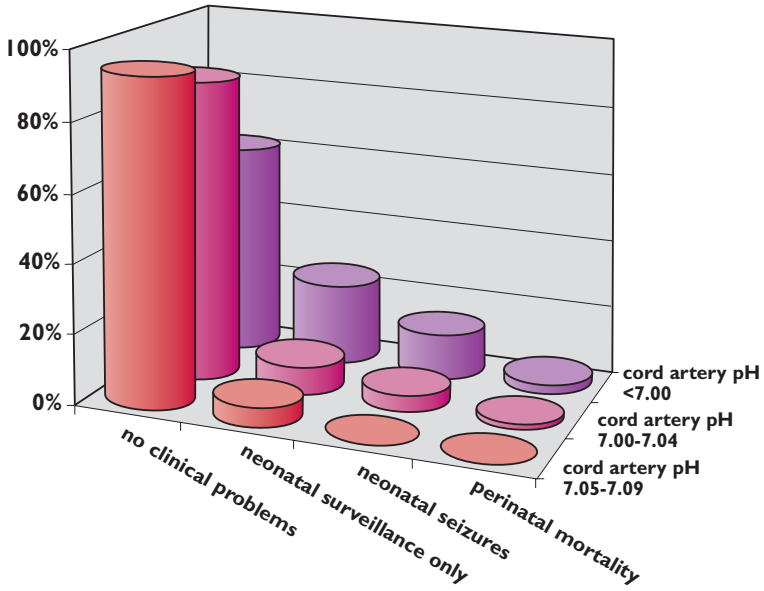
Free hydrogen ions are potentially damaging to the cell and the fetus tries to reduce the number of free hydrogen ions as much as possible. The most efficient buffers are the hemoglobin in the blood, proteins and bicarbonate ions located in the tissues and blood. Metabolic acidosis is defined as a situation in which these buffers have been utilized and metabolic acidosis is quantified by calculating the base deficit in the extra-cellular fluid. The base deficit is always calculated from measurements of pH and carbon dioxide. The base deficit in the extra-cellular fluid is abbreviated BDecf and indicates the amount of buffers in both blood and tissues that have been utilized due to the need to buffer hydrogen ions.

Unfortunately, the algorithms used in different blood gas machines may differ considerably and it may be difficult to decide if the base deficit data have been correctly calculated. In case of any doubt you may contact Neoventa Medical. If the wrong algorithms are used, more metabolic acidosis will be indicated.

Normal values

It is important to know the normal acid base values that can be recorded from the cord at the time of delivery. A normal pH in the cord artery is between 7.05 and 7.38.

When is there a risk of damage?

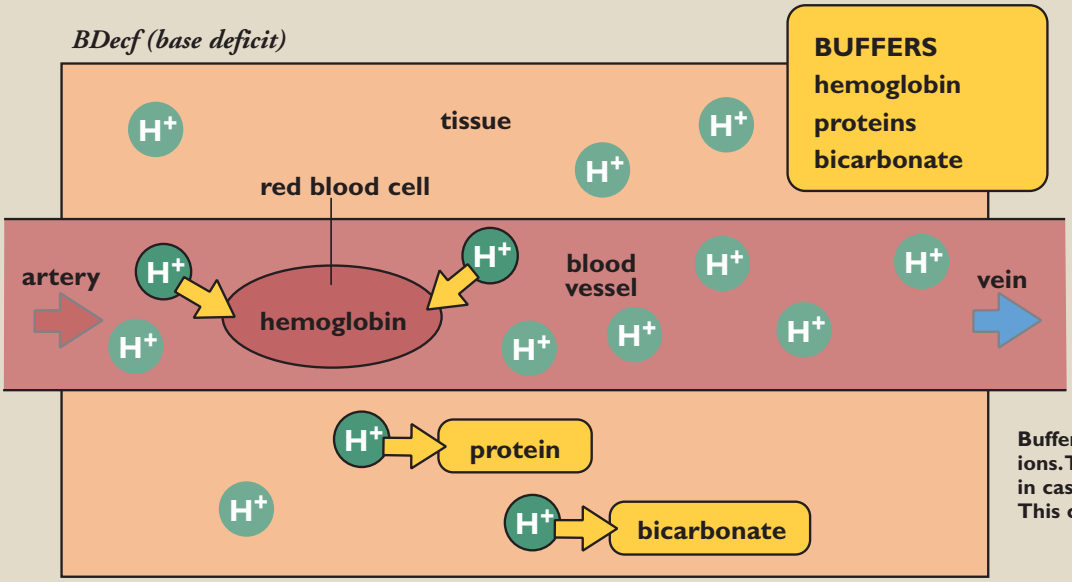


There are several studies that demonstrate that the cord artery pH has to drop to 7.05 and even below 7.00 before significant risks emerge. Even at such low readings, more than 60% of babies experience no problems in the neonatal period (Goldaber et al). In case a baby has been exposed to intrapartum hypoxia with marked metabolic acidosis, the risk of long-term sequele is very small provided the baby manages the neonatal period well.

The PCO_2 in the cord artery is normally between 4.9 and 10.7 kPa, but it may be much higher, and the base deficit in the cord artery should be between –2.5 and 10.0 mmol/l.

Values from the cord vein show a higher pH than in the artery, normally between 7.17 and 7.48, and the PCO_2 should be lower, between 3.5 and 7.9 kPa, but the base deficit is roughly the same, between –1.2 and 9.0 mmol/l.

So, we would normally expect marked differences in cord artery and vein pH and PCO_2. However, the BDecf should



Buffers capture free hydrogen ions. These buffers will be utilized in case of metabolic acidosis. This can be recorded as BDecf.

Erroneous cord acid base data

Samples from the same vessel!		
	artery	vein
pH	7.01	7.02
PCO_2	8.82	8.65
BDecf	12.8	12.5

Cord acid base data indicating a short-lasting hypoxic event

Large difference – short-lasting hypoxia		
	artery	vein
pH	7.01	7.27
PCO_2	8.82	5.14
BDecf	12.8	8.0

Cord acid base data indicating a long-lasting hypoxic event

Small difference – long-lasting hypoxia		
	artery	vein
pH	7.01	7.12
PCO_2	8.82	6.65
BDecf	12.8	11.5

Cord acid base data; normal values

	artery	vein
pH	7.05–7.38	7.17–7.48
PCO_2 (kPa)	4.9–10.7	3.5–7.9
BDecf (mmol/l)	-2.5–10.0	-1.2–9.0

be the same. An artery pH of <7.05 and a BDecf of >10 mmol/l is recorded in 2.5% of the population.

Accurate acid-base analysis

Immediate cord clamping is required for accurate cord acid-base analysis.

Samples should be taken from both the cord artery and the vein. There are several reasons for this; firstly, to determine that one sample is arterial and the other is from the vein. Furthermore, by comparing the arterial and venous samples, we can see whether the hypoxia has been acute or more long lasting.

How do we know if the samples are correct and contain data from both the artery and the vein? This is made possible by looking at the difference between pH and PCO_2. The pH should be at least 0.03 units lower in the artery and the PCO_2 should be at least 1.0 kPa higher in the artery.

By studying the base deficit in samples from the cord artery and vein, information is obtained about the duration of hypoxia. A high base deficit in the artery and a normal base deficit in the vein indicate short-lasting hypoxia.

If there is a high base deficit in both artery and vein, the hypoxic episode has lasted for a longer period of time and the risk of hypoxic damage increases.

What is asphyxia?

Until recently, an international document identifying the requirements for the diagnosis of intrapartum asphyxia has been lacking.

The following essential criteria for the diagnosis of acute intrapartum hypoxia causing persistent brain damage have been identified.

1. Evidence of metabolic acidosis in the umbilical arterial cord or early neonatal blood samples (pH<7.00 and base deficit ≥12 mmol/l).
2. Early onset of severe or moderate neonatal encephalopathy in term infants.
3. Cerebral palsy of the spastic quadriplegic or dyskinetic type.

Other criteria that together suggest intrapartum timing but by themselves are non-specific are as follows.

4. An observation of a hypoxic event occurring immediately before or during labor.
5. A sudden, rapid and sustained deterioration in the fetal heart rate pattern, usually after the hypoxic observation where the EFM pattern was previously normal.
6. Apgar scores of 0-6 for longer than five minutes.
7. Early evidence of multi-system involvement.
8. Early imaging evidence of acute cerebral abnormality.

What is asphyxia?

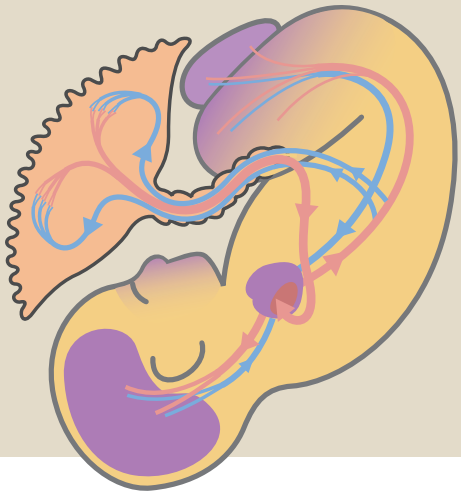
event during labor

the child should be affected

need for resuscitation

metabolic acidosis

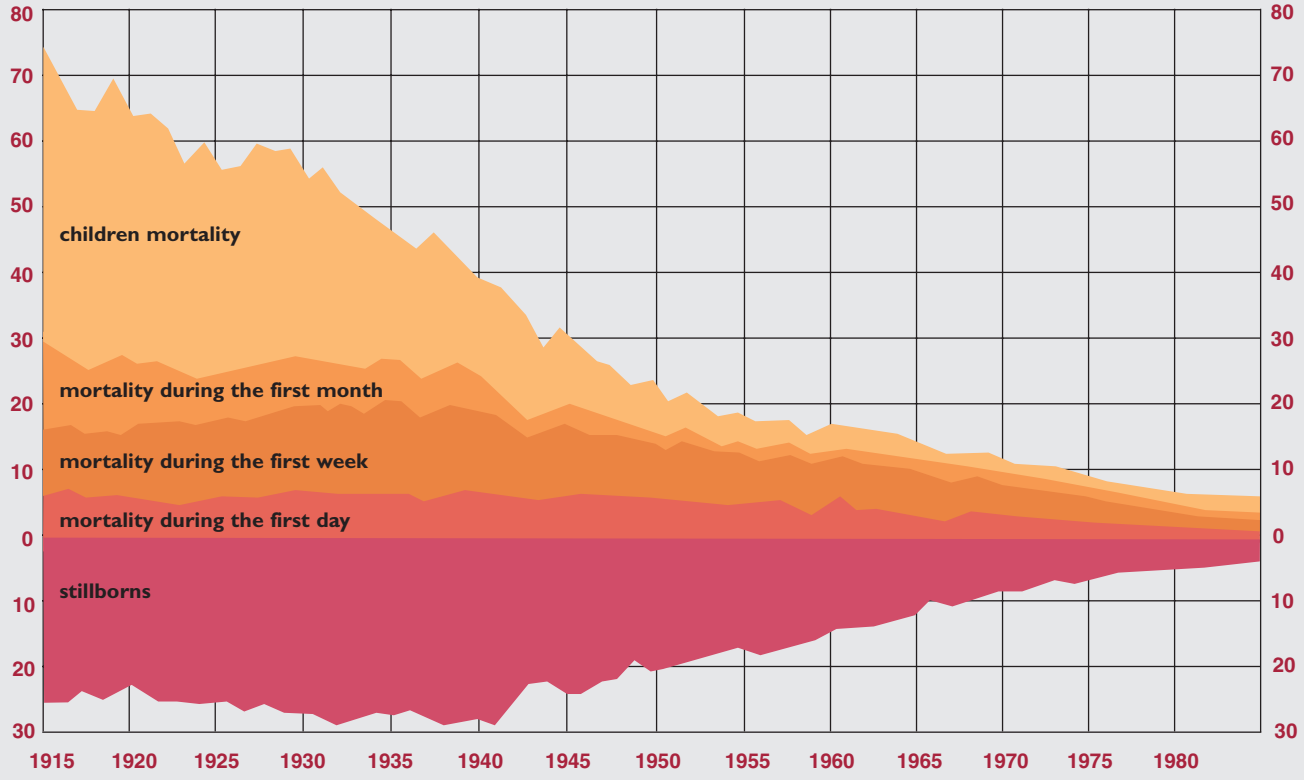
neonatal symptoms



All three essential criteria should exist for an intrapartum event to be regarded as a cause of a cerebral paresis. Only the degree of metabolic acidosis is regarded as having the specificity required to identify an intrapartum event. To ensure that the hypoxic process started in connection with labor, all of criteria four to eight should be fulfilled. Their relation to hypoxia is not very strong by themselves and, in the event of a normal five-minute Apgar, the likelihood of an hypoxic damage during labor is markedly diminished.

Summary
We have seen an extraordinary reduction in mortality among infants in relationship to pregnancy and birth during the last century. The challenge today is to maintain and develop this trend still further. Much can be learned by improving our understanding of how the fetus reacts to the stress of labor. Through this learning process, the risk of a child being injured will be significantly reduced and at the same time, the number of unnecessary operative deliveries for a non-reassuring fetal state will decrease.

Swedish infant mortality (per 1000 births) from 1900



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