

Birth Trauma

Position: Relationship of the presenting part to the internal pelvis. ROP: right occiput posterior; or LOA: Left occiput anterior.

Engagement: The fetus is engaged if the widest leading part (typically the widest circumference of the head) is negotiating the inlet.

Station: Relationship of the leading bony part of the fetus to the maternal ischial spines. If at the level of the spines, it is at “0” (zero) station; if it passed it by a 2 c.m., it is at “+2” station.

Pelvic Inlet: The line between the narrowest bony points formed by the sacral promontory and the inner pubic arch is termed the obstetrical conjugate.

Gynecoid: Ideal shape, with round to slight oval inlet. Best chances for a normal vaginal delivery. An abnormal pelvis is android, anthropoid and platypoid.

Lawyers investigating a CP claim will be confronted with the prevailing obstetrical view that asphyxia rarely causes CP. **Asphyxia does play a critical role in the development of the neonatal brain during labor and around the time of birth**—and that is preventable.

Asphyxia and hypoxic ischemic injury affect a substantial number of babies, and they are potentially preventable causes of CP. Many combinations of clinical markers can be used to prove that a baby with CP was exposed to damaging intrapartum asphyxia, including non-reassuring FHR patterns; signs of fetal distress; a neurological syndrome, such as HIE, evident at birth or during the first hours of life; early-onset seizures; abnormal blood gas studies (pH of less than 7.20) at birth or during the first hours of life; and neuroimaging findings confirming cerebral edema and other changes (basal ganglia pathology) consistent with a hypoxic or ischemic insult occurring during labor or around the time of birth.

Three features are important in considering that intrapartum insult is the likely cause of neonatal brain injury: (1) evidence of fetal distress (for example, fetal heart rate abnormalities, meconium-stained amniotic fluid); (2) depression at birth; and (3) an overt neonatal neurological syndrome in the first hours and days of life. The issue is whether asphyxia was the most likely cause of the child’s brain damage.

ACOG’s views about the minimal role of asphyxia in causing CP are self-serving and not generally accepted in the worldwide scientific community.

Partial, prolonged asphyxia is accompanied by a more predictable sequence of events, with the fetus capable of compensating to some extent before reaching the threshold for irreversible brain damage. Human fetuses have the same ability as primates to compensate during episodes of hypoxia by redistributing blood flow to the brain, heart, and adrenal glands. Until this compensatory mechanism fails, the human fetus will not begin to suffer neonatal cell death in the brain. In a study, the episode of hypoxia usually

lasted longer than an hour before the neuropathologic damage responsible for motor and cognitive deficits developed.

All babies, term and preterm, are at risk for developing irreversible brain damage when they are exposed to asphyxia during the antepartum, intrapartum and post-partum periods. However, both animal and human studies confirm that brain damage is not automatic when a fetus is exposed to asphyxia, asphyxia may be reversible, the risk of brain injury depends on the duration and/or severity of the asphyxia, and more brain damage occurs with the passage of time.

In most cases, the records need to be reviewed by an obstetrician, possibly a labor and delivery nurse with a strong background in fetal heart monitoring, a pediatrician or neonatologist on resuscitation and newborn medical issues and a pediatric neurologist for opinions on diagnosis and etiology/causation. It is usually a good idea to have the slides of the placenta reviewed by a placental pathologist and the CT scans and MRI scans reviewed by a neuro-radiologist. The placental pathologist will look at the placenta under a microscope for such things as clinically un-diagnosable maternal infection or microscopic meconium particles which might account for asphyxiation in utero or deterioration after birth, as well as a number of other possible explanations for the poor outcome. Neuro-radiologists can describe "patterns" of injury in the brain on CT and MRI scans.

An important publication to which experts often refer in support of their causation opinions is published by the American College of Obstetricians and Gynecologists (ACOG) on the subject of "Fetal and Neonatal Neurologic Injury", Committee Opinion #197 (February, 1998). **This publication sets forth criteria which ACOG claims must be present before a "plausible link [between perinatal asphyxia and neurological injury] can be made."**

When obstetrical malpractice cases are lost at trial, more often than not it is because the plaintiff did not adequately establish the causal relationship between the medical mismanagement and the cerebral palsy. This is the most fertile area for defense experts to theorize and speculate about other possible causes.

General Concepts: When the delivery of oxygen and nutrition to the brain is interrupted or severely impeded or decreased for a period of time, the brain becomes injured.

"Live Birth": Public Health Law section 4130 states: 1. Live birth is defined as the complete expulsion or extraction from its mother of a product of conception, irrespective of the duration of pregnancy, which, after such separation, ***breathes or shows any other evidence of life such as beating of the heart, pulsation of the umbilical cord, or definite movement of voluntary muscles***, whether or not the umbilical cord has been cut or the placenta is attached; each product of such a birth is considered live born.

2. The birth of each child born alive in this state shall be registered within five days after the date of birth by filing with the registrar of the district in which the birth occurred a certificate of such birth, which certificate shall be upon the form prescribed therefore by the commissioner.

Public Health Law section 4103 provides: 3. A certified copy of the record of a birth or death, a certification of birth or death...shall be prima facie evidence in all courts and places of the facts therein stated.

Metabolic Acidosis: Sodium bicarbonate may be helpful to correct metabolic acidosis that has resulted from a buildup of lactic acid. **Lactic acid is formed when tissues have had insufficient oxygen.** Severe acidosis will cause the myocardium to contract poorly and will cause the blood vessels of the lungs to constrict, thus decreasing pulmonary blood flow and preventing the lungs from adequately oxygenating the blood.

When sodium bicarbonate mixes with acid, carbon dioxide is formed. The lungs must be adequately ventilated to remove the carbon dioxide.

Although it is not unusual for metabolic acidosis to be found in a severely compromised baby, the condition will usually self-resolve with restoration of adequate circulating volume and adequate oxygenation.

Sodium bicarbonate is very caustic and hypertonic and therefore, must be given in a large vein, from which there is good blood return. Sodium bicarbonate corrects metabolic acidosis by producing carbon dioxide and water.

Narcotics given to the mother to relieve pain associated with labor commonly inhibit respiratory drive and activity in the newborn.

Pattern of Neurologic Injury: The pattern of neurologic injury following an acute catastrophic hypoxic-ischemic insult can involve the thalami and basal ganglia predominantly and may be different from the common pattern following chronic insult in the term newborn that involves predominantly the cerebral cortex and the subcortical white matter.

Multi-Organ Dysfunction: The shunting of blood flow from other organs to the brain and from the cerebral hemispheres to the thalamus and brainstem may explain the involvement of the cerebral hemispheres and multi-organ dysfunction seen with the more prolonged type of fetal hypoxia associated with uteroplacental ischemia.

Possible Causes of Cerebral Palsy during the pregnancy: Specific types of infection occurring for the first time during pregnancy; placental abnormalities including placental insufficiency or premature aging of the placenta during the pregnancy, premature or sudden rupture of the placenta from the wall of the uterus (placental abruption), intrauterine growth retardation (IUGR) of the fetus; severe malnutrition of the mother, frequent use of certain types of prescription, non-prescription or illegal drugs, frequent use of alcohol by mother during pregnancy, exposure to certain types of toxic chemicals or other harmful environmental hazards; mother's untreated high blood pressure, preeclampsia, eclampsia, toxemia, diabetes, problems with her thyroid or other conditions.

Placental Abruption: Premature separation of the normally located placenta from the uterus after the 20th week of gestation and prior to birth. Patients with placental abruption typically present with bleeding, uterine contractions and fetal distress. Symptoms may include vaginal bleeding, contractions, abdominal tenderness and decreased fetal movement.

Placental abruption must be entertained as a diagnosis whenever third trimester bleeding is encountered. The overall fetal mortality rate for placental abruption is 20%-

40%, depending on the extent of the abruption. This rate is significantly higher in patients with a significant smoking history.

Fetal morbidity is caused by the abruption itself and by issues related to prematurity when early delivery is required to alleviate maternal or fetal distress. If significant placental abruption is present, the fetal heart rate tracing typically shows evidence of fetal decelerations and even persistent fetal bradycardia.

Only a few risk factors have been linked to this condition: cigarette smoking/tobacco abuse, maternal HTN (most common cause of abruption), alcohol consumption, cocaine use, trauma and advanced maternal age.

Placenta Previa: Placental previa literally means afterbirth first and defines a condition wherein the placenta implants over the cervical opening. The significance of a placental previa is sudden unexpected life-threatening vaginal bleeding. It may cause serious morbidity and mortality to both fetus and mother.

The exact etiology of placenta previa is unknown. The classic presentation of placental previa is painless vaginal bleeding.

Fetal Monitoring: The focus is on early detection of fetal distress and immediate appropriate action. The monitor checks the baby's heart rate and measures the intensity and frequency of the uterine contractions. It is changes in the heart rate—speeding up or slowing down beyond the normal range just before, during and immediately after the contractions that determines whether there is any fetal distress.

The monitor attaches to the mother in one of two ways—externally or internally. The former requires a belt around the abdomen and the latter necessitates inserting an electrode into the birth canal. Both get attached to the same electronic device with the internal electrode usually producing more reliable readings with fewer artifacts.

The ongoing observation of the readings is the nurse's responsibility. The obstetrician relies on the nurse to report any signs or suspicions of distress.

A sudden increase in the strength and frequency of contractions could precipitate a dangerous fetal event resulting in the passing of meconium (fetal bowel movement in the uterus). This causes irreparable harm to the lungs because the baby is actually breathing in a liquid environment within the amniotic sac. If the meconium reaches the lungs it causes pneumonia and increases the likelihood of irreversible brain damage.

Apgar: A rapid evaluation is mandatory during the first few seconds after birth as the cord is being clamped. A screening assessment recorded at 1 and 5 minutes after birth. This scoring system provides points between 0 and 2 for each of 5 categories, including color, tone, respiratory effort, reflex activity and heart rate. The best Apgar score is 10 and the lowest score is 0. Equal or greater than 7 is considered normal, 4-6 is compromised, and 0-3 is a medical emergency. The scores may be recorded every 5 minutes until a score of 7 or above is reached.

The Apgar scores are not a good measure of asphyxia or of long-term outcome.

Apgar scores have become a standard clinical tool to assess newborns and determine whether they need resuscitation. In 1953, Virginia Apgar published a paper proposing a scoring system to evaluate newborns at one minute of life.

In 1992, ACOG decided to elevate the Apgar score from a tool to determine the need for newborn resuscitation to one used to defend obstetrical malpractice claims. ACOG issued a technical bulletin that called a low Apgar score (0-3 for longer than five minutes) an essential criterion for establishing whether a child's cerebral palsy was caused by asphyxia during labor. ACOG Technical Bulletin No. 163, Fetal and Neonatal Neurologic Injury (1992).

In January, 2003, without explanation, ACOG departed from its 1992 and 1996 guidelines, demoting the Apgar score and recommending that a score of 0-3 for longer than five minutes be treated as a non-essential criterion.

The NEACP indicated that a low Apgar score could not clearly establish damaging intrapartum asphyxia, but it could help establish whether the asphyxial event occurred within 48 hours of labor and delivery.

ACOG's use of Apgar scores as a litigation tool is based on a scientifically invalid hypothesis—that all newborns who suffer damaging intrapartum hypoxia (or asphyxia) must have low Apgar scores (0-3) at 1, 5 and 10 minutes after birth.

In a 2004 committee opinion, ACOG retreated from its 2003 NEACP statement and restored the importance of the Apgar score, stating that evidence of profound acidemia (cord-blood pH of less than 7), with low Apgar scores (0-3) beyond five minutes are still essential criterion.

Asphyxia: Asphyxia refers to a sequence of events that begins when blood flow to the brain is reduced (ischemia), or the oxygen content of the blood perfusing the brain falls below normal levels (hypoxia). As the hypoxia or ischemia persists, harmful acids capable of causing brain damage can accumulate in the blood leading to metabolic acidosis.

Asphyxia is a high level of carbon dioxide and a low level of oxygen. It is worse than anoxia. Hypercarbia produces asphyxia.

Arterial Cord pH: The arterial cord pH is another indicator of status at birth. Normally, arterial cord pH is greater or equal to 7.21. **A pH less than 7.00 indicates significant acidosis.**

The term pH describes how acidic or alkaline a substance is. A pH below 7 is an acid; the lower the number, the stronger the acid. Blood pH is slightly alkaline, with a normal range of 7.36-7.44. pH is a measurement of the acidity of a solution based on the amount of hydrogen ions available.

Metabolic acidosis means that two poisonous acids increase in your bloodstream, lactic acid and pyruvic acid.

External Fetal Monitoring: Most external fetal monitors use a Doppler ultrasound transducer applied to the mother's abdomen. The transducer detects fetal heart activity and triggers the rate-computing circuitry. **A disadvantage of this method is imprecision in detection of beat-to-beat variability.**

A fixed baseline with absent variability has been associated with preexisting neurologic impairment. A non-reactive tracing with an invariable baseline suggests preexisting neurologic damage, fixing the insult to the antepartum period. There can be intrapartum injury superimposed on preexisting antepartum injury.

Decelerations: Periodic declines in heart rate (from the baseline), usually in response to uterine contractions. With early decelerations, the onset of the deceleration occurs with the onset of the contraction, the nadir of the heart rate occurs with the apex of the contraction, and the heart rate increases back to baseline as the contraction ebbs.

Early deceleration is caused by compression of the fetal head and is much more common with ruptured membranes. It is a benign finding. However, if early deceleration patterns are severe, prolonged, persistent or accompanied by thick meconium staining, the problem may be serious.

Late Decelerations: The pattern occurs later than the contraction; that is, the onset of the FHR deceleration occurs after the contraction onset, **the nadir of the FHR occurs after the apex of the contraction, and the FHR deceleration continues after the contraction has abated.**

Late deceleration is commonly associated with utero-placental insufficiency and is a consequence of hypoxia and metabolic disorders. Thus, this is the most ominous fetal heart rate pattern.

With severely affected fetuses, the FHR takes longer to return to baseline from the deceleration.

Variable Decelerations: Caused by umbilical cord compression. Decelerations are classified as severe if they last more than 60 seconds or lead to a FHR of less than 90 bpm.

The most common combined deceleration pattern is that of variable and late deceleration; this is a most ominous pattern-nearly all fetuses with this pattern are severely compromised and immediate delivery should be considered.

Tachycardia: Occurs when the baseline FHR is greater than 160 beats per minute (“bpm”) lasting for more than 10 minutes. It is further quantified as moderate (160-180 bpm) or severe (greater than 180 bpm). **Tachycardia may result from fetal distress.**

Variation: The time interval between heart beats varies slightly.

Baseline: Average FHR prevailing apart from beat-to-beat variability and periodic changes.

Bradycardia: Fetal Heart Rate, “FHR”, less than 120 beats per minute; **moderate bradycardia (100-119 bpm) may be associated with severe fetal distress, whereas severe bradycardia (FHR less than 100 bpm) is likely to be agonal or due to heart block.**

Fetal Distress: The infant's critical response to stress, most usually deprivation. **The most immediate fetal needs are the acquisition of oxygen and the elimination of carbon dioxide.**

Placenta Previa: Placenta previa occurs when the placenta develops low, within the zone of dilation-effacement of the lower uterine segment. Thus, the placenta precedes the fetus

and can block vaginal delivery. Placenta previa complicates 1:200 - 1:250 pregnancies that continue beyond the 28th week.

The types of placenta previa are complete, in which the placenta totally covers the internal os, partial, where a portion of the internal os is overlaid by the placenta and low-lying, in which the placenta is just above the os but situated where it may deflect or obstruct the presenting part.

Painless vaginal bleeding is the presenting complaint in placenta previa. Breech or an abnormal position is common because the forelying placenta alters the usual intrauterine space available. Even a small vertex may not engage.

No laboratory tests will aid in the diagnosis of placenta previa. However, blood studies should be obtained periodically because of blood loss and the threat of anemia.

Ultrasonography is the modality of choice for the diagnosis of placenta previa.

Preterm delivery occurs in 60% of infants of mothers with placenta previa and is the prime cause of neonatal complications. In addition to early or chronic blood loss, acute fetal bleeding may occur during caesarian section when an anterior placenta previa is torn.

With proper management, the maternal prognosis in placenta previa is excellent.

The perinatal mortality rate associated with placenta previa is less than 10% with current management.

Uterine contractions can be monitored externally, either manually or electronically, and internally by means of an electronic pressure gauge. The contraction pattern consists of three elements, frequency, duration and intensity.

The critical responsibility is to recognize deviant patterns; the normal uterine pattern is a regular rhythm of contractions, gradually increasing in force and frequency, yet with adequate resting time between contractions to allow resumption of uteroplacental perfusion and thus fetal oxygenation.

The normal baseline fetal heart rate ranges between 120 and 160 beats per minute (BPM); a baseline greater than 160 BPM is considered tachycardia. The opposite of tachycardia is bradycardia - this is defined as a baseline fetal heart rate of below 120 beats per minute.

Oxytocin is a potent natural hormone that causes the uterine muscle to contract.

The umbilical cord should have two arteries and one vein.

Premature rupture of the membranes is defined as spontaneous rupture at any time before the onset of labor.

Premature: born before 38 weeks.

Postmature: born after 42 weeks.

Fetus at term: infant born after at least 38 weeks of gestation from mother's last known menstrual period.

Nausea and vomiting are common complaints from the second through fifth months of pregnancy. They are usually worse in the morning, but may persist throughout the day.

Cerebral Palsy: One to two newborns in every 1,000 births develop cerebral palsy as a result of brain damage. **Various criteria identify infants who were brain damaged as a result of decreased oxygen during labor and delivery: (1) severe acidosis; (2) a five minute or longer APGAR score of three or less; (3) evidence of neurologic injury -**

seizures - within the first 24 hours of life; and (4) damage to other organs - such as the kidneys - that is consistent with decreased oxygen.

Since respiratory acidosis does not necessarily lead to brain damage, **strong evidence of metabolic acidosis is needed**. CT or MRI scans are important in ruling out other causal events.

The birth asphyxia case should not include any evidence of decreased fetal movement or trauma during the pregnancy. Any fetal ultrasound examination should be normal both for anatomy and for fetal size to gestational age.

There must be clear evidence of fetal distress, which is generally diagnosed from electronic fetal heart monitor. **A significant indicator of distress is a heart rate outside the normal range of 120 to 160 beats per minute.** Other common indicators include late decelerations and decreased short or long term beat to beat variability.

A late deceleration is a decrease in the fetal heart rate after a uterine contraction. **Since the heart rate is controlled by the brain, any changes that produce anoxia or ischemia will cause a decrease in variability.**

A perinatologist, an expert in high risk pregnancies, can evaluate the management of labor. A developmental pediatrician can explain the profound nature of the brain damage.

Placenta: The placenta transfers oxygen and nutrients from the maternal blood to the fetus. At the same time, carbon dioxide and other waste products pass across the placenta from the fetus to the mother. The placenta is essential for the transfer of nutrients and gases from the mother to the fetus and for the removal of fetal waste products.

The goal of the fetal heart rate monitoring is to detect fetal hypoxia at its earliest stage and prevent asphyxia from resulting. Detection is possible because **the effects of hypoxia and ischemia on the central nervous system can produce abnormal heart patterns associated with fetal distress.** The sooner the clinician intervenes, the higher the probability of avoiding irreversible brain damage or fetal death.

Meconium: The presence of meconium in the amniotic fluid is often used by attorneys as evidence that the baby's oxygen supply was compromised. Meconium is used by some experts as a marker of fetal distress, when it is present in the amniotic fluid.

Meconium passage occurs by three distinct mechanisms: (1) as a physiologic maturational event; (2) as a response to acute hypoxic events; (3) as a response to chronic intrauterine hypoxia. Meconium passage might merely be a marker of chronic intrauterine hypoxia or can predispose to aspiration of meconium

Meconium is the green viscous fluid that consists of fetal gastrointestinal secretions, cellular debris, mucus, blood and vernix. **Passage of meconium in utero with staining of the amniotic fluid occurs in 12% to 16% of all deliveries and often is not associated with fetal distress or neonatal death or disability.** Meconium passage occurs in up to 20% of full-term gestations.

Meconium aspiration is defined as the presence of meconium below the vocal cords. This finding occurs in 20% to 30% of all infants with meconium-stained amniotic fluid. MAS is one of the most common causes of neonatal respiratory distress. **Infants born through meconium-stained amniotic fluid are about 100 times more likely to develop respiratory distress than those born through clear liquid.** Death occurs in

about 12% of infants with MAS, and MAS is associated with about 5% of all perinatal deaths.

Some believe that meconium passage is related in large part of maturation events only and not to intrauterine stress or hypoxia. **8% to 20% of all deliveries have meconium staining of amniotic fluid.**

Meconium passage in utero has been attributed to a fetal response to intrauterine stress and is often associated with fetal hypoxia, asphyxia and acidosis. **Hypoxia causes increased gastrointestinal peristalsis and relaxed anal sphincter tone.**

Meconium in the amniotic fluid can also simply represent the maturation of fetal intestinal function. Meconium passage is rare before 34 weeks of gestation. After 37 weeks gestation, its incidence increases steadily with increasing gestational age.

Several investigators have suggested that most cases of meconium aspiration occur in utero when fetal gasping is initiated before delivery. Is meconium harmless itself and merely a marker of fetal maturation or of chronic fetal hypoxia? Some found that MAS was primarily associated with acute hypoxic events late in labor.

Generally, the consistency of meconium is divided into two categories: thin meconium, and thick or particulate meconium. Thin meconium is yellow to light green and is watery. Thick or particulate meconium is pasty or granular and has a variety of colors including dark brown or black.

Thin meconium occurs in 10% to 40% of the cases of meconium passage. **There is a relation between the consistency and timing of meconium passage.** The risk of perinatal death is increased five to seven times when thick meconium is present at the onset of labor. Infants with thin meconium are more likely to have passed meconium as a physiologic maturation process and are more likely to be healthy at birth.

The finding of either thick or thin meconium at the onset of labor reflects events that occurred before labor. Meconium that is detected during labor after clear fluid has passed indicates an acute event. **All labors with meconium-stained amniotic fluid should be continuously monitored.**

Most meconium passage in post-term pregnancies is due to normal fetal maturation and infrequently leads to fetal compromise.

Infants with acute hypoxia events, near and after the onset of labor, are more likely to pass thick meconium and to suffer meconium aspiration. Interventions to clear meconium are more likely to be beneficial for these infants than for infants born through thin meconium

Meconium aspiration might be merely a marker of chronic intrauterine hypoxia. Interventions for the prevention of MAS, includes methods to remove meconium from the respiratory tract.

The findings of meconium passage in utero should prompt a thorough evaluation of the patient for general high risk factors in pregnancy and the institution of continuous monitoring for fetal well-being. The most effective interventions for prevention of MAS include various methods to remove meconium from the pharynx, trachea and stomach during and immediately after delivery.

Clear the airway of material, particularly if blood or meconium is present. This has special importance in the newborn because of the narrow airway, which creates high resistance to gas flow. Clearing the airway will also provide additional respiratory stimulation. Clearing of secretions should be accomplished with a suction device.

Hypoxia causes increased gastrointestinal peristalsis and relaxed anal sphincter tone. Meconium in the amniotic fluid can also simply represent the maturation of fetal intestinal function.

The consistency of meconium is divided into two categories: thin meconium, and thick or particulate meconium. Thin meconium is yellow to light green and is watery. Thick or particulate meconium is pasty or granular and has a variety of colors including dark brown and black.

The risk of perinatal death is increased five to seven times when thick meconium is present at the onset of labor. Thick meconium early in labor generally reflects low amniotic fluid volume, a risk factor for neonatal morbidity and mortality itself. Infants with thin meconium are more likely to have passed meconium as a physiologic maturation process and are more likely to be healthy at birth.

The finding of either thick or thin meconium at the onset of labor reflects events that occurred before labor. Meconium that is detected during labor after clear fluid has passed indicates an acute event.

All labors with meconium-stained amniotic fluid should be continuously monitored.

Several organizations have proposed expert guidelines for the management of infants exposed to meconium-stained amniotic fluid. In 1992, the Committee on Neonatal Ventilation and Meconium of the American Heart Association recommended that all infants exposed to meconium-stained amniotic fluid have obstetric pharyngeal suctioning. They further recommended that tracheal suctioning be performed if (1) there is evidence of fetal distress....

Infants with acute hypoxic events, near and after the onset of labor, are more likely to pass thick meconium and to suffer meconium aspiration. Interventions to clear meconium are more likely to be beneficial for these infants than for infants born through thin meconium. Aspiration of meconium with the first breaths after birth is more likely, and the infants are at higher risk for the obstructive and local inflammatory effects of meconium.

The finding of meconium passage in utero should prompt a thorough evaluation of the patient for general high-risk factors in pregnancy and the institution of continuous monitoring for fetal well-being.

Meconium Aspiration: Meconium aspiration is defined as the presence of meconium below the vocal cords. This finding occurs in 20% to 30% of all infants with meconium-stained amniotic fluid.

Aspiration is the removal by suction of a gas or liquid from a body cavity.

The most effective interventions for prevention of MAS include various methods to remove meconium from the pharynx, trachea, and stomach during and immediately after the delivery. Pharyngeal suctioning performed by the delivering attendant before the delivery of the shoulders has become almost universally accepted.

Tracheal suctioning, on the other hand, is a matter of great controversy. Some have recommended suctioning to empty the infant's stomach of meconium after initial stabilization. This maneuver is done to remove meconium that later could be regurgitated and aspirated.

Umbilical cord: It is not necessary to cut the umbilical cord before resuscitation of the newborn. The umbilical cord can be cut after the baby is spontaneously breathing and the cord has stopped pulsating.

Asphyxia: hypoxia and respiratory acidosis.

Labor: Labor is involuntary and unpredictable. The only thing doctors have had any success with controlling in labor is pain and length of labor.

In active management, the woman adjusts to the hospital by giving up any semblance of control of her body and her birth.

Often too many cesarian sections are performed because doctors misdiagnose the indications for them.

Umbilical Cord: An umbilical cord typically has a median length of 57.5 centimeters and has three blood vessels (two arteries and one vein). **A short cord (30 cm or less) or a long one (110 cm or more) can be associated with neonatal problems.**

Ischemia: Diminished amount of blood perfusing the brain.

Hypoxia and ischemia, alone or in combination, can lead to blood gas and pH abnormalities, which may be referred to as acidosis, either respiratory or metabolic.

Baseline Rate: Baseline rate is the fetal heart rate that persists over a given period of time.

Periodic Changes: Periodic changes are accelerations or decelerations in the fetal heart rate that occur with contractions.

Variability: Variability refers to the beat-to-beat changes in the fetal heart rate.

Early Decelerations: Early decelerations refer to a depression of the fetal heart rate, usually on the order of 10 to 20 beats per minute, which coincides with a uterine contraction. The onset occurs with the beginning of a contraction, continues through the height of a contraction and returns to the baseline as the contraction subsides.

Variable Decelerations: Variable decelerations are associated with umbilical cord decompression. Compression causes an increase in the fetal blood pressure, reduces oxygen supply to the fetus, and activates responses in the central nervous system that result in a decrease in the fetal heart rate and the development of variable decelerations.

Electronic Fetal Monitor: EFM is used both as an acute and chronic marker of fetal well-being; EFM records the fetal heart rate and measures the uterine activity on a strip that enables the clinician to assess the response of the fetal heart rate to the stresses of labor.

ACOG Clinical Markers: ACOG's list of markers that must be present before a causal link between cerebral palsy and asphyxia can be considered. These markers are: (1) pH less

than 7.0; (2) APGAR scores of 0-3 for more than 5 minutes; (3) neonatal neurological sequelae, such as seizures; and (4) multi-organ system dysfunction.

Glascow Coma Scale: The GCS may underestimate the severity of head injury because it does not detect minor alterations of mental status or abnormal focal neurological signs.

Eye opening:

Spontaneously: 4

To verbal command: 3

To pain: 2

None: 1

Verbal response:

oriented-converses: 5

Disoriented-converses: 4

Inappropriate words: 3

Incomprehensible: 2

No response: 1

Motor response

Obey verbal commands: 6

Localizes to painful stimuli: 5

Flexion withdrawal: 4

Abnormal flexion: 3

Abnormal extension: 2

No response: 1

Edema: swelling of the brain, or edema, is a result of excessive water retained in the brain's tissue subsequent to acute insult. Brain edema may be isolated or diffuse. A major cause of secondary brain injury occurs due to increased intracranial pressure caused by cerebral edema.

Ventilation: Ventilation is a general term for the movement of air into and out of the lungs. Air reaches the alveoli and takes part in gas exchange. The gas exchange involves the transfer of oxygen and carbon dioxide.

Anemia: Anemia is a condition where the red blood cell count in the blood is lower than normal. Red blood cells carry oxygen to vital organs.

The most common treatment of anemia is blood transfusion, which is the administration of blood or a blood component (red blood cells) into the blood stream.

PCO₂: Clinically important information can be obtained from measuring the partial pressure of carbon dioxide in the arterial blood (PCO₂). Carbon dioxide is a byproduct of food metabolism. Carbon dioxide is literally a waste product that must be eliminated for the body to function normally.

Decreased respiratory exchange with retention of carbon dioxide results in a high PCO₂ which then causes retention of bicarbonate.

Carbon dioxide is transported in the blood by three forms: as bicarbonate (the greatest amount), combined with hemoglobin, and other proteins. Carbon dioxide is accomplished by bringing fresh air into the lungs. There is no other way to excrete the body's CO₂ production.

Without alveolar ventilation, carbon dioxide will build up in the blood since there is no other way to eliminate it. As a result, severe acidity and death will quickly follow.

PO₂: Arterial oxygen pressures.

Fetomaternal Hemorrhage: In most FMH cases, the etiology is obscure, the gestation is uneventful until decreased fetal movements are noted and the placenta, fetal movements and umbilical cord show no pathologic findings on either gross or microscopic examination. Most FMH occur silently in the mother and without a known predisposing cause. Often FMH was unsuspected despite obvious anemia in the infant at delivery.

FMH is uncommon. Its diagnosis is poor and usually made postpartum. **A sinusoidal heart rate pattern and decrease in fetal movement are considered important signs of FMH.** SHR and decrease in fetal movement may be late signs of FMH. The non-stress test and ultrasound appeared to be useless for early detection of FMH except in unusual cases.

The diagnosis of FMH is made by demonstrating fetal erythrocytes in the maternal circulation. A 5% fetal hemoglobin level in the maternal circulation may be associated with fetal death.

The most common presenting symptoms of FMH were anemia at birth, decreased or absent fetal body movement. Decreased or absent fetal body movement, SHR pattern, or hydrops fetalis are late sign of FMH.

FMH was diagnosed in utero by the maternal reporting of decreased or absent fetal body movement, the detection of sinusoidal heart rate pattern, or the demonstration sonographically of hydrops fetalis. Unexpected anemia at birth is often the presenting manifestation.

It is known that the fetus can tolerate an acute blood loss of 40 percent of the blood volume; larger volumes result in fetal death. The large FMH in many of the cases reviewed here, occurred over a period of time allowing cardiovascular adaptation. High erythroblasts and reticulocyte blood counts suggest an interval of time sufficient to stimulate the bone marrow. It is not known, however, how long after FMH those indices increase.

During the last few years, large FMH have been diagnosed more frequently. During this period, **intrauterine transfusions have been mostly used to treat fetal anemia.** IUT may be lifesaving when FMH is diagnosed before term.

Non-lethal, massive FMH is likely to occur over prolonged periods of time. It is possible that intermittent bleeding episodes may be superimposed over a chronic leakage of fetal blood into the maternal circulation.

In acute anemia redistribution of blood flow to the brain and heart while blood flow and oxygen delivery to the kidneys, intestines, muscles and skin decreases. In contrast,

prolonged fetal anemia is associated with decreased vascular resistance to all tissues (except the placenta).

In severe anemia, despite successful cardiac adaptation, an increase in hydrostatic pressure leads to tissue edema and hydrops fetalis.

FMH is a poorly understood condition. Although specific causes have been identified, the etiopathogenesis of most of the cases remains unknown. **Decreased or absent fetal body movement, detection of a sinusoidal heart rate pattern, or hydrops fetalis are late signs. An acute FMH constitutes an unexpected medical emergency.** Future efforts should concentrate on the early recognition of subacute and chronic FMH.

The volume of blood transfused should correct the anemia without a volume overload.

Massive FMH has been defined as bleeding in which more than 150 ml of fetal blood is found in the maternal circulation.

FMH is an important cause of neonatal anemia. Probably many deaths during pregnancies are due to chronic FMH without any clinical sign.

If bleeding is acute, a dead fetus is often found, while chronic loss allows cardiovascular adaptation. The amount of blood for lethal consequences has been established from hemoglobin values. The risk of tissue oxygenation correlates with counts of hemoglobin below 8 mg.

The real cause of such large amounts of bleeding in severe fetomaternal hemorrhage is unknown. Probably many deaths during pregnancy are due to hemorrhage without any clinical sign. The silent nature of that process and the lack of clinical parameters however hinders the diagnosis.

At present, the absence of fetal movements, the sinusoidal pattern in the fetal heart rate, even atrial fibrillation, intrauterine growth delay, hydrops fetalis, severe anemia or unexpected death are defined as clinical signs of FMH, which neurological dysfunction, respiratory distress syndrome, persistent fetal circulation or the different degrees of multiorgan disturbances, are considered fetal complications coming from the bleeding. Neurological troubles overshadow prognosis.

FMH has an important role as a cause of hypoxic-ischemic encephalopathy.

Bleeding problems that have started after the 20th week of gestation, when aerobic neuronal metabolism has already been established, seems essential for a long-term prognosis. At that time, neurons would be extremely sensitive to anoxia and especially to the results of anemia.

The main problem remains silent clinical data.

Massive FMH has been variously defined as a bleed of 50 to 150 ml. It is an important cause of fetal morbidity and mortality. FMH is a rare but important cause of severe anemia at birth. When there is a severe anemia with moderate-to-severe heart failure, an exchange transfusion leads promptly to the restoration of the full oxygen-carrying capacity.

FMH has been treated successfully with serial fetal intravascular transfusions.

Anemia commonly causes fetal hydrops.

Many cases of acute and chronic FMH are of unknown cause. Third trimester uterine activity might cause microscopic areas of placental capillary damage. Women with

FMH usually present with complaints of decreased fetal movement and fetal heart tracing abnormalities.

A rare but ominous baseline fetal heart rate which has been reported previously is the undulating or sinusoidal baseline fetal heart rate pattern.

Despite massive transplacental hemorrhage, anemia does not occur in all newborn infants, and it is believed that **the clinical features of transplacental hemorrhage are related not only to the size of the hemorrhage but also to the time at which it occurs.** If the bleeding is limited and occurs several weeks before delivery, the fetus may correct the anemia by compensatory erythropoiesis before delivery. **The finding of massive numbers of nucleated red blood cells at birth suggests an interval of sufficient duration to stimulate the fetal bone marrow.**

The physiologic mechanisms that produce a sinusoidal FHR pattern strongly point toward severe fetal jeopardy. This pattern may represent virtual absence of central nervous system control over the heart rate. Most cases of severe fetal asphyxia do not show sinusoidal heart rate patterns, and the most commonly reported finding in fetuses with sinusoidal patterns is fetal anemia and apparently high output heart failure.

Whatever pathophysiologic mechanisms are involved in producing a sinusoidal FHR pattern, its presence strongly suggests impending fetal death, and delivery should be considered if the gestational age is consistent with neonatal viability.

Anemia of the newborn can be due to acute or chronic loss of fetal cells into the maternal circulation. Massive FMH has been defined as bleeding in which more than 150 ml of fetal blood is found in the maternal circulation. There can be remarkable fetal compensation in cases involving massive chronic FMH resulting in severe anemia.

The cause of FMH in most reported cases has been unexplained. Most cases represent acute rather than chronic FMH, generally resulting in stillbirth. **The occasional infant that survives an acute hemorrhage is hemodynamically compromised and acidotic.** That the infant was hemodynamically stable at birth, manifesting only pallor and not requiring fluid resuscitation or blood transfusion, is further indication that the blood loss was chronic. The uncorrected reticulocyte count of 39.9% was markedly elevated above the normal newborn value of 3-7%, indicating a chronic process: Two to three days is required for a reticulocyte response to be observed, with a peak response requiring 10 to 14 days.

The FHR pattern classically associated with fetal anemia is a sinusoidal pattern. Late decelerations have also been reported in acute fetal blood loss with the development of fetal acidosis.

Increase in uterine activity during the third trimester could result in microscopic areas of capillary damage in the placenta and therefore allow transplacental passage of fetal erythrocytes into the maternal circulation.

A sinusoidal heart rate pattern is defined as a rate of 120 to 160 beats per minute, no reactivity, fixed or flat short-term variability, and oscillation of the sinusoidal wave form above and below the baseline.

A fetus in the early part of the third trimester who has evidence of chronic, massive FMH (severe anemia, normal results of blood gas studies, sinusoidal heart tracing, and often hydrops) can sometimes be managed with serial fetal intravascular transfusions.

Of importance, **serial Kleihauer-Betke tests would be useless after the initial transfusion because most cells transferred from the fetus to the maternal compartment would be adult cells.**

Serial Kleihauer-Betke tests should be coupled with serial ultrasound examinations to assess fetal growth or to detect fetal hydrops.

The fetal transfusion alone cannot be expected to stop the flow of fetal blood into the maternal compartment. Pregnant women must be aware of fetal movements during the third trimester. Maternal perception of decreased fetal movement prompted fetal monitoring and recognition of a possible sinusoidal heart tracing.

The volume of fetal blood present in the maternal circulation is usually very small. The cause of FMH in many cases is obscure. **Although the American College of Obstetrics has cited high-risk circumstances for FMH of more than 30 ml, other authors have reached the conclusion that in a significant percentage such hemorrhage cannot be predicted by obstetric events.**

Acute fetal blood loss of 20% is associated with a very high mortality rate, but remarkably, fetal compensation can occur in cases of chronic fetal blood loss.

The fetal sinusoidal heart rate pattern is well documented to occur in association with severe fetal anemia.

Massive FMH is defined as a loss of more than 150 cc or approximately 50% of the fetal blood volume and occurs in one out of 1,000 deliveries.

The Kleihauer-Betke technique of acid elution is the simplest and the most commonly used test. Adult hemoglobin dissolves out of the cells, whereas fetal hemoglobin, which is acid resistant, remains intra-cellular and is stained and enumerated by microscopic examination. The results are given by the ratio of fetal red blood cells to maternal red blood cells. The results are most reliable when the test is done soon after birth. When performed later, false-negative results may occur because of progressive elimination of fetal red blood cells from the maternal circulation.

In acute hemorrhages the red cells appear normochromic and normocytic, whereas in chronic hemorrhage the cells are generally hypochromic and microcytic. *Increased nucleated red blood cells may be seen in acute and chronic hemorrhage.*

Nucleated red blood cells appear in the peripheral blood when the marrow is subjected to intense stimulation, as in response to acute hemorrhage. The presence of these cells in the peripheral blood is an important indication of serious disease except in asplenic individuals. **The high percentage of NRBC is a witness to the extent, and probably duration, of the FMH. Evidence for chronicity of the FMH is also demonstrated by the sinusoidal fetal heart rate pattern, the organomegaly and the microcytic, hypochromic anemia at birth.**

Nucleated Red Blood Cells: Nucleated red blood cells are commonly found in the circulation of the normal newborn. Fetal cells have been identified in the maternal circulation in the first trimester. About one-half of women have fetal red cells in their circulation in the first trimester. About half of women have fetal red blood cells in their circulation that are demonstrable by acid elution test. Both the frequency of finding fetal cells and the volume of those cells increase as pregnancy increases. This is most likely due to the increasing size and gradual deterioration in the placental blood barrier in the

later weeks of gestation. Therefore, one might expect large FMH to occur more often in post-term pregnancies.

NRBC are not useful in timing of the injury leading to the IVH detected in the first week of life. **There is too much overlap between the normal neonatal values and the values of impaired infants with injuries for the test to be useful in timing a neurologic insult or attributing it to intrapartum events. Abnormal values have no value in determining the timing of a neurologic insult.**

Most hypoxic neonatal brain injuries cannot be pinned down to an exact moment. Hypovolemia compensation may occur before delivery.

Preeclampsia: Toxemia of pregnancy which can result in the death of both mother and fetus is characterized by high blood pressure, fluid retention, and excessive protein in the urine. Preeclampsia is a disorder of pregnancy that can strike without warning, causing high blood pressure and protein in the urine. In turn, preeclampsia may progress to eclampsia—hypertension and generalized convulsions—which may be fatal.

The only curative treatment for the overall condition is immediate delivery. **The only real cure is to deliver the baby.** Sometimes the baby has to be delivered prematurely. The cause of preeclampsia remains unknown. The fetal mortality rate is 13-30%.

Abuptio placentae can complicate severe preeclampsia.

Magnesium sulfate has anti-convulsant properties and can be used for seizure prophylaxis.

Pregnancy-Induced Hypertension: Pregnancy-induced hypertension is defined as systolic blood pressure of at least 140 mm Hg or a diastolic pressure of at least 90 mm Hg on at least two occasions taken several hours apart. Preeclampsia is defined as the presence of hypertension with proteinuria or pathologic edema, or both, occurring after 20 weeks of gestation. The etiology of PIH and preeclampsia is unknown.

The disease process may be associated with adverse fetal effects. Maternal-placental perfusion is decreased with women with PIH and is believed to account for increased perinatal morbidity and mortality.

Magnesium sulfate improves cerebral blood flow and oxygen consumption in women with PIH. Magnesium sulfate therapy is believed to dilate human umbilical vessels and possibly, increase fetoplacental blood flow.

Causation of Cerebral Palsy (ACOG Technical Bulletin, Number 163, January, 1992):

Cerebral is defined as a chronic neuromuscular disability characterized by aberrant control of movement or posture appearing early in life and not the result of recognized progressive disease. It may be accompanied by a seizure disorder or mental retardation or both. Epilepsy or mental retardation is seldom associated with perinatal asphyxia in the absence of cerebral palsy.

Gestational age of less than 32 months is strongly predicative of cerebral palsy. The causes of most cases of cerebral palsy are unknown.

Asphyxia around the time of labor and delivery can be a cause of cerebral palsy, but the asphyxia must be nearly lethal to be considered a possible cause. It has been estimated that 10% of cerebral palsy in term infants is associated with perinatal asphyxia. About 90% of children with cerebral palsy were not asphyxiated at birth.

Although there is relatively widespread opinion that persistent late decelerations, severe variable decelerations (variously defined), and prolonged bradycardia constitute possible fetal distress, there is no agreement on how quickly the nonreassuring pattern must be terminated or how other abnormalities of the EFM tracing affect the decision.

EFM patterns that have been thought to reflect fetal distress may reflect intrinsic fetal abnormality and not necessarily acidosis.

In assessing a possible relationship between perinatal asphyxia and neurologic deficit in a patient, all of the following criteria must be present before a plausible link can be made: (1) profound umbilical artery metabolic or mixed acidemia (pH less than 7.0); (2) persistence of an Apgar score of 0-3 for longer than five minutes; (3) neonatal neurologic sequelae, i.e., seizures, coma, hypotonia; (4) multiorgan system dysfunction, i.e., cardiovascular, gastrointestinal, hematologic, pulmonary or renal.

There is no clear-cut way to determine when intrapartum asphyxia is severe enough to cause cerebral palsy. A very low (0-3) Apgar at 10 minutes was found to be a powerful predictor of cerebral palsy.

Fetal asphyxia causes redistribution of blood from nonvital organs (the kidney) to vital organs (the brain, heart and adrenal gland). The severely asphyxiated neonate will therefore demonstrate evidence of asphyxial effects in other organs, such as the kidneys, lungs, gut, and heart. Without such evidence, it is unreasonable to postulate an asphyxial effect on the brain. Neuroimaging studies in such infants commonly show cerebral edema.

About 25-30% of cases of cerebral palsy are identified in children who were delivered preterm.

Causation of Fetal Brain Damage: In trying to date fetal brain damage, the most revealing information can be gleaned from imaging studies, EEG's and pathologic examination. Distinct pathologic changes occur in the brain in response to hypoxic-ischemic events.

Genetic Abnormalities—Blood Testing: The defendants are “hoping to find something that could lead to a poor outcome”. There are two types of tests for genetic abnormalities: (1) a metabolic screen; and (2) a chromosome screen.

The metabolic screen is a test to detect the “inborn errors of metabolism”. The “inborn errors of metabolism” can cause seizure disorders, neurological disorders, mental retardation and the child does not reach developmental milestones. A metabolic screen “looks at enzymes the cells use” and added that “enzyme deficiencies can cause” the various problems listed above.

“Metabolic disease does not present with low Apgars and low pH” and there is “nothing in the chart that would lead you down that path [of metabolic disease]”. Each state has a different metabolic screening test in terms of “what they test for”. A metabolic screen “doesn't look for everything in every baby”. Even though a baby's metabolic screening was normal, the defendants may be asking for an “extended test” that goes beyond the tests conducted in the initial newborn screening.

You must ask the defendants what tests they intend to conduct as part of the metabolic screening because they may be repeating the same tests that have already been done.

The second type of genetic testing is chromosomal testing and there are two types of chromosomal testing: (1) Karyotype; and (2) Fish. Chromosomal tests can show that the baby had “extra or missing chromosomal material” which can cause symptoms.

Infants with genetic abnormalities are “usually born stillborn” or die within the first year of life. If children with genetic abnormalities are born alive, they have an “abnormal physical appearance”, such as dysmorphic features. Children with genetic abnormalities have a “whole different physical appearance”.

You must find out from defendants’ counsel what blood tests that they will be conducting, i.e., “what are you actually ordering?” If they are ordering a metabolic screen, “why are they ordering another one?”

A baby can have both a chromosome abnormality and birth asphyxia and the defense may be trying to blame a genetic abnormality on the baby’s condition.

Placental Inspection—Placental Pathologist: The placenta weighed 520 grams which is “normal for a term placenta”. The weight of the placenta was within a normal range and she would expect that there was a “normal baby” attached to the placenta. The expert will want to know the birth weight of the baby.

The placental pathologist will check for any “evidence of chronic deprivation”, or any abnormality, with regard to the placenta, and whether there is any “meconium discoloration” on the placenta.

The placental pathologist will examine the length of the umbilical cord; a cord that is evaluated to be 21 centimeters is “abnormally short” in length. 30 centimeters to 35 centimeters is the “cut-off for a short cord” and the range for the length of an umbilical cord is “greater than 30 to 35 centimeters” and “less than 75 centimeters”. The umbilical cord may be abnormally short because the cord is often cut at birth and the placental pathologist does not have the umbilical cord in its entirety.

An abnormally short umbilical cord can be caused by “neurodevelopmental syndromes”. “Fetal movements increase the length of the umbilical cord” and “if the umbilical cord is not long, there is a presumption that the baby didn’t move”.

Group B Strep: Group B Strep, sometimes called GBS, is a type of bacteria that is often found in the vagina and rectum of healthy women. This type of bacteria is very common to all types of women and can be passed on to your baby during childbirth. In the United States, about one in four women carry this type of bacteria.

Being a carrier for this bacteria does not mean you have an infection. It only means that you have Group B strep bacteria in your body, usually living in the rectum or vagina. These bacteria are usually not harmful to you—only to your baby during labor.

The medicine to stop GBS from spreading to your baby is an antibiotic given during labor. The antibiotic is only given during labor—you do not need to worry about getting it for yourself before labor. The antibiotics work best if you get them at least four hours before you deliver. The antibiotic (usually penicillin) is given to you through an IV during childbirth. At the time of labor or rupture of membranes, intrapartum chemoprophylaxis should be given to all women identified as GBS carriers.

It does not work to take antibiotics for GBS before labor. The bacteria can grow back so fast that taking the medicine before you begin labor does not prevent the bacteria from spreading to your baby during childbirth.

Ask your doctor for a GBS test when you are 35 to 37 weeks pregnant. All pregnant women should be screened at 35-37 weeks gestation for vaginal and rectal GBS colonization.

Each time you are pregnant, you need to be tested for GBS. It doesn't matter if you did or did not have this type of bacteria before—each pregnancy is different.

Erb's Palsy/Shoulder Dystocia

Shoulder dystocia is not always preventable. Most injuries (80 percent) occur in macrosomic infants; thus, an obstetrician can be well prepared. Identification of multiple risk factors should allow a physician to be well-prepared.

Macrosomia is frequent in pregnancies of obese, diabetic women because the principal substrate for fetal growth is glucose. The risk of macrosomia will increase when more than one risk factor is present, from 15 to 30 percent.

Shoulder dystocia will increase tenfold at 4,000 grams and will increase tenfold with a flat pelvis.

ADOPE: "A": maternal age greater than 30 years; "D": diabetes; "O": obesity; "P": post-datism (41 weeks); "E": excessive maternal weight gain (35 plus pounds). Older mothers are at risk for increased fetal weight.

There are inherent errors in the accuracy of ultrasound methods. In good hands, the estimated fetal weight by ultrasound is within ten percent of the actual weight.

Ultrasonography is not an accurate predictor of macrosomia.

The preferred definition of macrosomia in the ACOG Technical Bulletin is 4,500 grams.

Prolonged second stage of labor is a well-recognized marker for shoulder dystocia.

Cephalopelvic distortion, arrested or protracted descent, or a prolonged second stage, are conducive to subsequent entrapment of the shoulders at delivery. All antenatal risk factors for macrosomia must be considered at the beginning of labor.

The head is usually delivered gradually and with difficulty; then immediately after its emergence, the head is pulled back tightly against the perineum. Prolongation of the second stage of labor might predict, in the case of a large infant, an impending shoulder dystocia.

The best approach to shoulder dystocia is prevention. The bony pelvis can be evaluated in the first prenatal visit with clinical pelvimetry. An abnormal pelvis, especially the flat type, is an important predisposing factor as well as a small pelvis. **Clinical pelvimetry should be performed on all patients antepartum.** Shoulder dystocia occurs ten times more frequently with a flat or platypoid pelvis than in women with a gynecoid pelvis. The platypoid pelvis is easy to identify on clinical examination. Once recognized, it should alert the clinician to the possibility of a problem with the shoulders.

Each subsequent fetus tends to increase in weight.

The McRobert's maneuver is the most popular and effective current technique. If shoulder dystocia is an anticipated possibility, and a vaginal delivery is planned, an obstetrician experienced with the problem should be present. The patient should be placed in the McRobert's position.

The performance of the McRobert's maneuver is a reasonable initial maneuver. This involves hyperflexion and abduction of the hips causing cephalad rotation of the symphysis pubis and flattening of the lumbar lordosis that frees the impacted shoulder. Suprapubic pressure may be used at the same time to assist in dislodging the impacted shoulder. In contrast, fundal pressure may further worsen the impaction of the shoulder and also may result in uterine rupture.

There is no evidence that any one maneuver is superior to another in releasing an impacted shoulder or reducing the chance of injury.

Further pulling or traction will increase the impaction and may cause injury to the child. Fundal pressure will do the same. Pivoting or twisting the neck adds nothing good and may lead to neurological damage. Vaginal lubricants are not useful.

The first thing that most operators do is apply more downward pressure to the head synchronously with the next uterine contraction, in an effort to dislodge the anterior shoulder from where it is impacted under the pubic symphysis.

When shoulder dystocia is confirmed, one should not touch the baby's head again until after the shoulder impaction is corrected. Pulling on the head causes brachial plexus injury.

Once the head has emerged, and it is evident that shoulder dystocia exists, several courses of action are available. As the first step, it is essential that any possible interference from the soft tissues of the lower birth canal be eliminated by an adequate episiotomy. A deep mediolateral episiotomy or a deliberate perinectomy, which divides the anal sphincter and extends well up to the rectovaginal septum, furnishes the needed space. Failure to perform a large episiotomy or episoproctomy can be a major error and a breach of the standard of care.

It is essential to perform the McRobert's maneuver—it is easy, safe and can be performed rapidly. It consists of removing the patient's legs from the stirrups and sharply flexing them against the abdomen. This maneuver does change the dimensions of the pelvis. The superior rotation of the symphysis tends to free the impacted shoulder. As part of the maneuver, if the shoulder is not released immediately, one can combine it with other techniques (i.e., suprapubic pressure), followed by attempts at rotation and/or grasping of the posterior arm.

No child with signs persisting at the age of 13 months recovered completely, and no further improvement was seen after the age of two years. Fewer than 10% of all cases of shoulder dystocia result in a permanent brachial plexus injury.

The availability of anesthesia is important, as is the presence of a pediatrician and adequate nursing staff.

If possible, the distinction between a bilateral versus unilateral shoulder dystocia should be made.

Advanced maternal age and multiparity are also risk factors.

Shoulder dystocia is caused by the impaction of the anterior fetal shoulder behind the maternal pubis symphysis. It can also occur from impaction of the posterior fetal shoulder on the sacral promontory.

Macrosomia describes a newborn with an excessive birth weight (a birth weight greater than 4,000 grams). Macrosomia affects 1-10% of all pregnancies. Macrosomia is reportedly associated with neonatal morbidity, neonatal injury, maternal injury and cesarean section. 4,000 grams equals 8 lbs., 13 ounces.

Infants with a birth weight of 4,000 grams or more comprise up to 10% of infants born in the U.S. Higher fetal mortality rates are associated with a birth weight of greater than 4,250 grams in non-diabetic mothers.

Macrosomic neonates are at risk for shoulder dystocia and birth trauma. The risk of brachial plexus injury is approximately 20 times higher when the birth weight is greater than 4,500 grams.

Gestational age is associated with macrosomia. Birth weight increases as gestational age increases. Ultrasounds of the fetus and its size can be useful for identifying macrosomic infants. The physician must be familiar with procedures that release a shoulder dystocia at delivery.

Pitfalls: Failure to diagnose macrosomia and birth injury at delivery; use of incorrect maneuvers for releasing shoulder dystocia in a macrosomic newborn.

The maternal pelvis is composed of a series of bones forming a circle protecting the pelvic organs. The front-most bone is the symphysis pubis; it is on this structure that a baby's anterior shoulder gets caught during a delivery complicated by shoulder dystocia.

The bone at the back of the maternal pelvis is the sacrum; because of its shape, the sacrum generally serves as a slide over which a baby's posterior shoulder can descend freely during labor and delivery. The side walls of the maternal pelvis do not contribute to shoulder dystocia.

The axis of the fetal shoulders must descend into the maternal pelvis at an angle oblique to the pelvis's anterior-posterior dimension; this position affords the shoulders the most room for their passage.

The back of the mother's pubic bone may form a shelf on which the baby's anterior shoulder can get caught. If this happens, the shoulders cannot deliver and a shoulder dystocia occurs.

Usually it is the fetal head that has the largest dimensions. Thus, if it can pass through the maternal pelvis without difficulty, the rest of the baby usually follows easily.

However, when the dimensions of the fetal shoulders or chest rival those of its head, the chances of a shoulder dystocia occurring are much increased. Such situations occur more frequently both in large babies and in babies of diabetic mothers.

Once the fetal head and shoulders have been delivered, the remainder of the fetal trunk and legs slide out easily. Large baby is greater than 4,000 grams.

The brachial plexus consists of the nerve roots of spinal cord segments C5, C6, C7, C8 and T1. These nerve roots form three trunks which divide into anterior and posterior divisions.

Erb's palsy involves the upper trunk of the brachial plexus (nerve roots C5 through C7). This palsy affects the muscles of the upper arm and causes abnormal positioning of the scapula called "winging".

Patients with upper lesions—Erb's palsy—have a better prognosis than those with a lower brachial plexus injuries—Klumpke palsy. Upwards of 90 to 95% of all Erb's palsies totally resolve.

The most feared complication of shoulder dystocia is fetal asphyxia. The reason for the increasing acidosis and asphyxia that occurs during a shoulder dystocia delivery is that once the fetal head emerges from the mother, the baby's umbilical cord becomes tightly compressed between its body and that of the mother's birth canal. This significantly

decreases or totally cuts off blood flow between the mother and the infant. If the pressure on the cord is not rapidly relieved, the consequences of cessation of lack of umbilical flow—decreased delivery of oxygen to the fetus—will occur.

Given a severely injured infant, if it can be shown that a physician was negligent either in allowing a shoulder dystocia to occur or in his or her handling of the shoulder dystocia once it did occur, then the physician will be held liable.

The pelvic anatomy of a woman does not change in between pregnancies. Second and subsequent babies are likely to be larger than first or previous babies. ***Macrosomia is far and away the most significant risk factor for shoulder dystocia.***

Dystocia is defined as abnormal or difficult labor. Shoulder dystocia is said to occur when the fetal biacromial head diameter cannot negotiate the pelvic brim. Most will define shoulder dystocia as when the anterior shoulder cannot pass the pubic symphysis or when special maneuvers are applied to deliver fetal shoulders. Shoulder dystocia occurs when the anterior shoulder impacts behind the pubic symphysis. Shoulder dystocia is extremely unpredictable. General dystocia accounts for 30% of all primary cesarean sections. Regarding the management of shoulder dystocia, it is critical that the physician perform and clearly document the maneuvers used, i.e., McRoberts.

The most common adverse outcome of shoulder dystocia, brachial plexus nerve injury (Erb's Palsy), can often be prevented with properly performed maneuvers. However, even when the maneuvers are properly performed, witnessed and acceptably documented, the complication can still occur.

In years past, shoulder dystocia cases resulting in Erb's Palsy had a high rate of successful litigation.

The most common serious complication following a shoulder dystocia delivery is a brachial plexus injury. This is when the nerves in a baby's neck—the brachia plexus—are temporarily or permanently damaged. The nerves of the brachial plexus control the function of the arm and hand. Injury to the upper part of the brachial plexus is called Erb's Palsy while an injury to the lower nerves of the plexus is called Klumpke Palsy.

McRobert's Maneuver: When shoulder dystocia occurs, hyperflex and abduct the maternal pelvis while the patient is in the dorsolithotomy position. This causes cephalad rotation of the symphysis pubis, thus flattening the lumbar lordosis. As an assistant applies suprapubic pressure, dislodge the shoulder using the McRobert's Maneuver. Do not apply fundal pressure at any time in the management of shoulder dystocia.

When shoulder dystocia does occur, several well-known procedures are available to manage it, including episiotomy, suprapubic pressure, McRobert's maneuver, the Woods screw maneuver, delivery of the posterior shoulder, the Zavanelli maneuver, deliberate fracture of the clavicle, symphysiotomy and the all fours maneuver.

Shoulder dystocia means that one, less frequently both, shoulder(s) of the baby are not entering the pelvis during the birth as they should. Shoulder dystocia occurs in less than 1% of all births.

McRobert's Maneuver: Flex the mother's legs toward her shoulders as she lies on her back, thus expanding the pelvic outlet.

Suprapubic Pressure: The pressure is at the pubic bone, not at the top of the uterus. This might allow the shoulder enough room to move under the pubis symphysis.

Woods Maneuver: This is also known as the corkscrew, the attendant tries to turn the shoulder of the baby by placing fingers behind the shoulder and pushing in 180 degrees.

Zavanelli Maneuver: The final maneuver involves pushing the head back up into the vagina and delivering the baby by cesarean section.

Shoulder Dystocia: During the birth process, the baby's shoulder is caught behind the pelvic bone of the mother. When this occurs, there are increased risks for both the mother and the infant. The baby is unable to breathe in this position, so an immediate response is crucial.

Shoulder dystocia is the situation, when the baby's head emerges from the vagina, the shoulders fail to follow easily, either on their own with the mother's pushing, or with some gentle assistance from the midwife or doctor.

Dystocia: "not moving"; in other words, the shoulders are stuck.

Risk factors for shoulder dystocia include advanced maternal age (older than 35), maternal weight gain of 35 pounds or more, short maternal stature, and abnormal pelvic shape or size. An estimated birth weight of over nine pounds is also a risk factor. The use of labor-inducing drugs suggests an increased risk for shoulder dystocia.

During labor, the suspicion of possible shoulder dystocia is raised by a long first stage (dilation stage), long second stage (pushing stage) with slow descent of the baby through the birth canal.

When using an epidural, a mother loses the pushing sensation, which can result in the baby descending in an awkward position.

By the age of two, a child will make no further improvement or recovery with regard to a brachial plexus injury.

The brachial plexus is a network of nerves formed by fibers which are located between the shoulder and neck. 90% of brachial plexus injuries are caused by a traumatic stretching of the plexus during birth. **80% of these infants recover without surgical intervention.**

Surgery is generally recommended only if a child demonstrates no functional recovery by four months of age. Surgery should be performed between four and six months of age.

The true extent of the injury cannot be adequately discerned until surgery.

Most children with a brachial plexus injury have damage to multiple nerves and more than one procedure must be performed. Recovery of some function can be expected within four months of surgery, with younger children recovering with a faster rate. Injuries involving nerves below the elbow have a lower rate of return (approximately 50-60%) due to the location of these nerves and the distance needed for regeneration.

Erb's Palsy is a weakness of the arm that occurs in newborns. It is caused when the nerves that control the arm are injured during the birth process. Erb's Palsy is commonly associated with shoulder dystocia.

Shoulder dystocia occurs when an infant's shoulder gets caught and stretched behind the pelvic bone during delivery. The condition can be caused, albeit rarely, without being caused by shoulder dystocia.

Risk Factors: **Large babies are at a higher risk for deliveries that involve damage to their shoulder.** For the same reason, mothers who have a smaller pelvic opening will also have a greater risk for delivering an infant with Erb's Palsy. **Other risk factors include** maternal diabetes, prolonged labor, an infant that presents in the breech position or **induction of labor.**

Prognosis: 75 to 90% of infants with Erb's Palsy will recover completely after several months. Complications include permanent, partial or total loss of function of the affected nerves, causing arm weakness or paralysis.

The site and type of brachial plexus injury determines the prognosis.

The inability to move the arm is a symptom of an injury to the brachial plexus, a network of nerves that provides movement and sensation to the arm, hand and fingers. Most infants with Erb's Palsy will recover both movement and sensation in the affected arm without surgery.

Brachial plexus injuries in newborns usually occur during a difficult delivery, such as with a large baby, when the person assisting the delivery must exert some force to pull the baby from the birth canal. One side of the baby's neck is stretched, which can damage the nerves by stretching or tearing them. If the upper nerves are affected, the condition is called Erb's Palsy. Injuries that involve both the upper and lower nerves are more severe and resulting a condition called global palsy.

There are four types of injuries to the brachial plexus: (1) avulsion injuries—the nerve is torn from its attachment to the spinal cord. This is the most serious type of injury; (2) rupture injuries—the nerve is torn, but not at the spinal cord; (3) neuroma injuries—these injuries result from scar tissue that forms and puts pressure on the nerve; (4) stretch injuries—these injuries, known as neuropraxia, are the most common. The nerve is damaged but not torn. Normally, these injuries heal on their own, usually within three months.

The symptoms of a nerve injury are the same, regardless of the type of injury.

Diagnosis: A newborn with Erb's Palsy will have the arm straight down at the side and will not move it. The doctor may use an EMG or nerve conduction study to see if any nerve signals are present in the upper arm muscle. An EMG or CT myelogram are used to determine which nerves are affected.

If there is no change over the first three months, nerve surgery may be helpful. However, **nerve surgery will not restore normal function or help infants over one year old.**

Plaintiffs' Verdicts in Birth Trauma Cases: Cruz v. Mount Sinai Hospital, 191 A.D.2d 325, 594 N.Y.S.2d 776 (1st Dep't 1993): Plaintiff's jury verdict for \$15 million. Thomas Moore, Esq.

Administration of Pitocin: 8 N.Y.C.R.R. 405.21.