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15

Risk Management in Obstetrics and Neonatal-Perinatal Medicine

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1. Introduction

The professional liability crisis remains a common problem for obstetricians. Approximately 90% of American College of Obstetricians and Gynecologists fellows have been sued at least once and 25% have been sued four or more times. Approximately 15% of obstetricians have ceased obstetric practice because of exorbitant premiums and the prevalence of nonmeritorious claims in this field of practice. The average age at which an obstetrician/gynecologist stops providing obstetrical care is currently 48 years of age; the age at which most physicians approach the peak of judgment and experience.

This current liability crisis is very relevant to all practitioners who care for newborns. Neonatologists, pediatricians, hospitalists, and nurse practitioners all provide critical care to sick newborns in different venues. These newborns are younger, more fragile, often extremely small and the risk of life long chronic disease, pain and disability are significant for these patients. Parents often experience emotional and economic distress when their newborn is in the NICU. These factors have contributed to an increased number of allegations against practitioners of neonatal/perinatal medicine.

Juries tend to have a natural sympathy for disabled children even when allegations are nonmeritorious. In addition, many states exempt minors from the statute of limitations for medical liability which can lead to a physician defending claims 10-20 years after the alleged incident. Capping noneconomic damages in children is difficult. The increase in litigation cases is mirrored by an increase in the awards received by the plaintiff. Today the average jury award for poor obstetric and neonatal outcome exceeds $3,000,000. Obstetricians pay some of the highest insurance premiums, up to $300,000 per year in some states. Efforts at tort reform, award caps and the policing of junk science have not been uniformly successful.

The purpose of this Chapter is to identify the etiology, pathology and prevention of common allegations of professional liability for the obstetrician and practitioner of neonatal-perinatal medicine. The author has reviewed 100 closed cases of alleged professional liability against obstetricians for causation of poor neonatal outcome and 100 closed cases of alleged professional liability involving practitioners of neonatal perinatal medicine as an expert. These cases were reviewed over a 25 year period (1985-2010). Approximately 75% of

the cases were reviewed for the defense and 25% for the plaintiff. Of these, 75% of the cases were settled, 19% were dismissed and 6% went to trial with a favorable jury verdict for the defense in 75% of the trial cases. Based on our experience, we developed an evidence-based work-up that can confirm or refute allegations of acute intrapartum asphyxia sufficient to cause cerebral palsy.

2. Common allegations of obstetrical professional liability

Table 1 lists the eight major categories that resulted in allegations of obstetric professional liability. The most common obstetrical allegation was failure to perform a timely C-section. The inability to recognize and react to nonreassuring fetal heart tones was the dominant allegation. Poor communication between the nurse, obstetrician and anesthesiologist in making the decision and provisions to perform an emergency C-section was common. The ability to perform an emergency C-section as a rescue procedure for the patient and/or fetus is a necessary part of the practice of moderate obstetrics. Although only accounting for 3% of all cesarean sections, the timeliness of cesarean sections is a frequent source of litigation. Even today it is unclear if this 30 minute rule from decision to incision is valid and more studies need to be performed. In fact, a recent study showed that approximately one-third of primary C-section deliveries were performed for emergency indications and were commenced more than 30 minutes after the decision to operate, mainly for nonreassuring fetal heart rate tracings. In this study, adverse neonatal outcomes were not increased. Unfortunately despite limited data, the 30 minute response time has become a medical/legal benchmark for adequacy of obstetrical care when a cesarean section is indicated.

Failure to triage a mother appropriately was the next most common allegation of professional liability against practitioners of obstetrical care. It is essential that all emergency rooms have specific protocols in the evaluation and management of the pregnant patient even when the primary complaint may not be obstetrically related. Misdiagnosis of preeclampsia/HELLP syndrome can be fatal to the mother and newborn. More common in group practices, the problems that result from a failure to follow-up on specific tests ordered in the prenatal period. The failure to follow-up on fetal ultrasounds that demonstrated twin to twin transfusion is a specific example. “If you do not document it, you did not do it” is a common cause of speculation

Complicated deliveries can result in catastrophic neonatal outcomes. Many high-risk situations, such as delivering a poorly controlled diabetic, VBAC, forceps, and vacuum require that the obstetrician initiate pediatric/neonatal presence in the delivery room. Infants born under these situations can appear stable and decompensate 12-48 hours after the initial event. The pediatrician needs to be alert for signs and symptoms of anemia, seizures and any altered neurologic status. A twin pregnancy is high risk and should command the presence of appropriate personnel for the delivery.

Regionalization continues to have a role and is in the best interest of the mother and newborn. The state and perinatal centers oversee the rules and regulations that dictate the level of care of the high risk mother and newborn provided at specific hospitals. Triplets and higher order pregnancies, newborns with known congenital anomalies and extremely low birthweight newborns are best delivered and cared for in a tertiary center. The best ambulance is the uterus. Ego can cloud good judgment and compromise the care and outcomes of the mother and fetus.

Cause for Obstetric Allegations	Case Examples		N = 100
1. Failure to perform a timely C-section	Non-reassuring fetal heart tones Poor communication between OB's and Anesthesiologists Obstetrical nurse failure to interpret ominous fetal strip Nurse spent too long trying to obtain FHT when none were present Inadequate fetal monitoring for prolonged periods of time	Postponing aggressive treatment for the next shift Inadequate physician sign out at change of shifts Failure of midwife to recognize ominous fetal heart tracings Failure of midwife to have appropriate resuscitation equipment and personnel for home delivery	40%
2. Failure to triage mother appropriately	Failure to follow-up test results Failure to give antenatal steroids Emergency room triage errors (misdiagnosis of Mirror Syndrome) Misdiagnose pre-eclampsia as gallbladder disease in ER Mother sent home at 39 weeks in active labor Failure to detect rupture of membranes	Failure to rule out abruption Failure to diagnose HELLP syndrome Diagnostic difficulties due to maternal obesity Failure of triage nurse and/or house staff to present an accurate picture of the case to the attending	21%
3. Complicated delivery	Shoulder dystocia Uterine rupture with VBAC Double footling breech delivered vaginally	Neonate born with fractured ribs, skull fracture, fractured clavicle Twin pregnancy in which in utero demise of viable twin was due to nonviable twin death	17%
4. Failure to transport mother to tertiary case center in appropriate timing	Expected difficult delivery with complicated neonate was not preemptively transferred to a tertiary care center Delivery of triplet or higher order pregnancy in a level 2 center	Congenital anomalies Complicated twin pregnancies, triplets, quadruplets (twin to twin transfusion, significant discordancy) 24 weeker	11%
5. Pharmacologic error	Dosing errors with Pitocin Using Pitocin instead of Magnesium Failure to follow Pitocin protocol	Failure to discontinue Pitocin with non-reassuring fetal heart tones and/or hyperstimulation	5%
6. Failure to diagnose maternal infection	Failure to diagnose chorioamnionitis Failure to obtain and document GBS status Failure to recognize fetal tachycardia as a sign of chorioamnionitis		3%
7. Inappropriate use of labor induction	Maternal request Physician convenience	Late preterm newborns	2%
8. Failure to educate patient	Patient not instructed exactly when she should go to the hospital for labor		1%

Table 1. Common Allegations of Obstetrical Professional Liability

Another common allegation of professional liability with poor neonatal outcome involves the use of Pitocin. In our experience, many obstetricians and obstetrical nursing personnel were not familiar with their hospital specific protocol for the use of Pitocin. Failure to discontinue Pitocin with nonreassuring fetal heart tones and the inability to recognize hyperstimulation generates arguments for poor neonatal outcome. Since 1994 the use of antenatal steroids to enhance fetal pulmonary and brain maturation has become the standard of care. Failure to give antenatal steroids between 24 and 34 weeks gestation with evidence of imminent delivery can result in poor newborn outcomes.

Neonatal sepsis can have significant morbidity and mortality. Failure to obtain and document Group B Streptococcus (GBS) status was common. Failure to recognize fetal tachycardia as a fetal response to chorioamnionitis was noted. Chorioamnionitis is one of the most common causes for newborn depression often requiring significant resuscitation in the delivery room. The presence of maternal chorioamnionitis which can include a fever, elevated white count, left shift, fetal tachycardia and foul-smelling amniotic fluid should mandate the presence of pediatrics/neonatology for the delivery.

In the last decade a significant awareness on the dangers of induction for convenience and/or maternal request has evolved. Numerous studies have shown that the late preterm newborn has significant morbidity and mortality compared to their term counterparts. One should never assume that a late preterm newborn at 34-36 weeks will have an uneventful nursery course. In our experience and supported by numerous studies, the male infant is at least one week behind in maturation compared to their female counterparts. Some of the most severe cases of hypoxic respiratory failure can occur in these late preterm newborns.

A common pathway leading to litigation from the previous eight categories of the obstetrical allegations discussed is whether with a reasonable degree of medical certainty a deviation in the standard of care caused morbidity and/or mortality in the newborn. The proportion of cerebral palsy associated with intrapartum hypoxia-ischemia is 8-14.5%. Despite this fact, the use of junk science, unethical expert witness testimony, and speculation in childbirth litigation persist.

3. Proposed work-up to confirm or refute allegations of acute intrapartum asphyxia

The next section summarizes our work in developing a workup for the newborn to confirm or refute the 4 essential and 5 suggestive criteria proposed in defining an acute intrapartum event sufficient to cause cerebral palsy as defined in the 2003 ACOG and AAP Task Force publication on Neonatal Encephalopathy and Cerebral Palsy. (Table 2)

Each case of alleged intrapartum asphyxia is unique and no single test can time an alleged event. The College criteria have been criticized for being too restrictive and potentially not being able to identify many cases of intrapartum asphyxia. Many consider a sentinel event to be a critical and essential first step in linking intrapartum asphyxia to neonatal encephalopathy. Aside from a sentinel event during labor, the College criteria are postdelivery assessments. Despite this controversy, we feel this proposed workup will provide significantly more objective evidence-based data in the medical record to support or refute allegations of intrapartum asphyxia. Table 3 outlines an evidence-based work-up to be considered in term and near term newborns with unexplained depression at birth with evidence of encephalopathy including seizures.

ESSENTIAL CRITERIA (Must meet all four)	Clinical work-up
Evidence of a metabolic acidosis in fetal umbilical cord arterial blood obtained at delivery (pH < 7.0 and base deficit ≥ 12 mmol/L)	Arterial Cord Gas
Early onset of severe or moderate neonatal encephalopathy in infants born at 34 or more weeks of gestation	EEG
Cerebral palsy of the spastic quadriplegic or dyskinetic type	MRI Head
Exclusion of other identifiable etiologies such as trauma, coagulation disorders, infectious conditions, or genetic disorders	Newborn Weight, Length and Head Circumference Placental Pathology CBC with Differential, blood cultures U/S Head MRI Head
Criteria that suggest an intrapartum timing	**Clinical Work-Up**
A sentinel (signal) hypoxic event occurring immediately before or during labor	Electronic Fetal Heart Rate Interpretation CBC with Differential, Platelets, NRBCs
A sudden and sustained fetal bradycardia or the absence of fetal heart rate variability in the presence of persistent, late, or variable decelerations, usually after a hypoxic sentinel event when the pattern was previously normal	Electronic Fetal Heart Rate Interpretation CBC with Differential, Platelets, NRBCs
Apgar scores of 0-3 beyond 5 minutes	Apgar Score 10 and 15 min
Onset of multisystem involvement within 72 hours of birth	PT, PTT, Fibrinogen, LFTs, Creatinine, Electrolytes, Glucose, Calcium, ECHO
Early imaging study showing evidence of acute nonfocal cerebral abnormality	Ultrasonography of the head MRI of the head

EEG: electroencephalogram; MRI: magnetic resonance imaging; NRBC: nucleated red blood cell; PT: prothrombin time; PTT: partial thromboplastin time; LFT: liver function tests; ECHO: echocardiogram

Table 2. Criteria to define an acute intrapartum event sufficient to cause cerebral palsy

Umbilical cord blood gas assessments are the most objective determinants of the fetal metabolic condition at the moment of birth. Umbilical arterial blood reflects fetal status more directly and umbilical venous blood more closely reflects whether the oxygen exchange of the uteroplacental unit is optimal. Westgate et al recommend obtaining cord blood from the artery and vein. However, in clinical practice this is not practical and an umbilical cord arterial gas is most often obtained. Fetal scalp blood sampling has been virtually eliminated in clinical practice without an increase in adverse newborn outcomes. An ongoing dilemma with the College criteria is the requirement of metabolic acidemia to determine whether an insult occurred intrapartum. Many term newborns who are delivered in the presence of fetal acidemia are not recognized by intrapartum events and are triaged to the regular nursery with an uneventful hospital course. Studies have demonstrated when the umbilical artery pH was less than 7.0 at birth, 67% had a metabolic component in their acidemia compared with 14% for those with pH of 7.0 to 7.2. One study showed with an

umbilical arterial pH less than 7.0 at birth, neurologic damage was found in 23%, with the remaining 77% being neurologically normal at the time of neonatal discharge. The pH is a direct measurement, whereas the base deficit is a calculated value obtained by the Siggard-Andersen alignment nomogram. This nomogram can confirm the biochemical authenticity of arterial cord blood gases. Umbilical arterial pH decreases and the base deficit increases during the course of normal labor, because a buffer base is depleted before the pH declines. The pH decreases approximately 0.07 units for every 10-mm Hg increment increase in PCO2. The respiratory component of acidosis cannot damage the newborn, and when present, the onset of hypoxia can be established because this component cannot last more than 20 to 30 minutes. In our experience, the absence of a cord arterial blood gas leads to more speculation between the plaintiff and defense experts than any other laboratory value and should be drawn in all deliveries and sent for analysis when clinically indicated.

Clinical Work-Up		Days of Life			
		1	2	3	7
1	Arterial Cord Gas	X			
2	Apgar Scores at 10′ and 15′ (if 5 minutes ≤ 6)	X			
3	Physical Exam: Newborn weight, length and head circumference	X			
4	Placental Pathology	X			
5	CBC with differential, Platelets, blood cultures	X			
6	U/S Head	X	X		
7	NRBCs	X	X	X	
8	PT, PTT, Fibrinogen, LFTs, Creatinine, Electrolytes, Glucose, Calcium, ECHO		X		
9	EEG		X		
10	MRI Head				X

Table 3. Proposed Clinical Work-Up of Newborns > 34 weeks GA with Alleged Perinatal Asphyxia in the First Week of Life

Neonatal encephalopathy is a clinically defined syndrome of disturbed neurological function in the earliest days of life, manifested by depression of tone and reflexes, subnormal levels of consciousness, and often times, seizures. After intrapartum asphyxia, hypotonia is the norm and, in general, early hypertonia or absence of hypotonia (normal tone) point to other neurological abnormalities. The grading of neonatal encephalopathy as mild, moderate, or severe was originally described by Sarnat and Sarnat. The presence of seizures is required to meet the Sarnat criteria for moderate to severe encephalopathy. Neonatal seizures can be subtle, often presenting with oxygen desaturations and focal motor abnormalities such as eye deviation, smacking of lips, and staring. Also, the presence of atypical apnea with desaturations frequently was not identified as seizures and delayed appropriate therapy. An electroencephalogram can be used to confirm the presence of seizures. Seizures soon after birth (1–6 hours or more than 24 hours of life are not consistent with acute intrapartum asphyxia). When seizures occurred within 24 hours, 48% of newborns were significantly negatively affected compared with when the seizures occurred after 24 hours.

Cerebral palsy (CP) is most often not diagnosed until well after the first year of life. White matter lesions such as cystic periventricular leukomalacia is a common lesion of prematurity (less than 34 weeks of gestation), often results in spastic diplegia, and is usually not associated with intrapartum asphyxia in the term infant. However, focal noncystic white matter injury is increasingly recognized in term newborns with neonatal encephalopathy. In term newborns, the gray matter is the most metabolically active and therefore most vulnerable to an acute intrapartum event. Although spastic quadriplegia with a dyskinetic, chorioathetoid component is the most common subtype of CP associated with an acute profound hypoxic intrapartum event, it is not specific to intrapartum hypoxia.

The majority of cases involving neonatal encephalopathy and CP are associated with maternal and antenatal factors such as intrauterine infection, maternal/fetal coagulation problems, antenatal hemorrhage, abnormal presentation, preterm birth, and developmental/chromosomal abnormalities. Plotting out weight, length, and head circumference is a vital component of the initial newborn assessment. The presence of microcephaly at birth can be consistent with an earlier pregnancy insult and usually results in a poor neurological outcome. The presence of intrauterine growth restriction and status of small for gestational age at birth can be associated with poor neurodevelopmental outcomes.

The placenta can be an excellent source of information to confirm alternate etiologies such as metabolic disorders, adverse growth events, and infections. Intraamniotic infection is the most common antecedent to birth depression, low Apgar scores, and neonatal encephalopathy in term newborns. The presence of chorioamnionitis and funisitis are significant risk factors for CP in term/near-term newborns. Fetal inflammatory response syndrome caused by cytokine expression in the fetus after exposure to maternal infection can also result in neonatal encephalopathy, often with negative cultures, cord arterial pH more than 7.0, and Apgar scores more than 3 to 5 at 5 minutes. Infection, inflammation, thrombosis, and coagulopathy are recognized as being associated with white matter-mediated damage caused by the elevated fetal cytokines and are ultimately associated with periventricular leukomalacia and encephalopathy. A newborn with neutropenia (absolute neutrophil count less than 2,000) and a band-to-segmented neutrophils ratio of more than 0.2 on a complete blood count more probably than not has clinical sepsis despite negative cultures. Newborn blood cultures should be obtained any time sepsis is suspected. A genetic work-up may be helpful to direct postnatal testing. Newborn thrombophilias also can be a congenital cause of abnormal neonatal outcome and may present as a hemorrhagic or thrombotic lesion. Many known thrombophilias, such as antithrombin III deficiency, protein C or S deficiency, prothrombin genetic deficiencies, hyperhomocystinemia, and factor V Leiden mutation, can all lead to strokes in the newborn, which can cause neonatal encephalopathy with CP and mental retardation and/or fetal/neonatal death. Meconium stained amniotic fluid is often erroneously associated with intrapartum fetal distress. In reality, 15% of the 4,000,000 annual births in the United States have meconium-stained amniotic fluid.

Vaginal bleeding during labor can signal trauma, such as a ruptured uterus, abruptio placenta/placenta previa, or fetal bleeding from a vasoprevia. When bleeding leads to fetal damage, it is usually associated with a significantly abnormal electronic fetal heart rate

tracing such as bradycardia (usually less than100 beats per minute for more than 10 minutes) and/or repetitive late decelerations with absent fetal heart rate variability. Bleeding can also be concealed and such fetal heart rate tracings may be the only suggestion of fetal compromise.

The presence of anemia in the newborn at birth also can point to nonpreventable etiologies such as maternal–fetal transfusion as well as chronic abruption. Unexplained anemia in the newborn should prompt the pediatrician/neonatologist to request a maternal Kleihauer-Betke test. In the newborn, a complete blood count with differential and a platelet count at birth as well as nucleated red blood cell often can be helpful in differentiating the patient with intrapartum asphyxia from other causes of encephalopathy.

The presence of thrombocytopenia (less than 150,000) as well as an elevated hemoglobin (greater than 18 g/dL) and hematocrit (greater than 55%) in the newborn can be consistent with chronic hypoxia in utero. Serial nucleated red blood cell counts in the first 3 days of life can provide helpful information because an elevated nucleated red blood cell count at birth with delayed clearance (greater than 72 hours) does not support a diagnosis of acute intrapartum asphyxia. The proportion of CP associated with intrapartum hypoxia-ischemia is 8% to 14.5%.Certain preexisting conditions such as perinatal ischemic stroke, neuromuscular disorders, and certain in-born errors of metabolism can present at birth with a clinical picture not unlike intrapartum asphyxia. Likewise, elevated lymphocyte counts in the fetus may be predictive of earlier hypoxia that antedates labor. Finally, a detailed note by the obstetrician after delivery that summarizes the intrapartum course may be helpful in ruling out asphyxia in labor as the cause of newborn depression.

Nonspecific criteria collectively suggestive of intrapartum timing include sentinel hypoxic intrapartum event. Cord prolapse, ruptured uterus, maternal shock amniotic fluid embolus, and acute bleeding can result in catastrophic intrapartum asphyxia.

The National Institute of Child Health in Human Development's Research Planning Workshop on electronic fetal heart rate monitoring offers standardized definitions for such tracings. The participants agreed that tracings with a normal fetal heart rate pattern including baseline heart rate within the normal range and normal fetal heart rate variability with the presence of accelerations and absent of decelerations (type I) confers an extremely high likelihood of a normally oxygenated fetus. At the other end of the spectrum, when there is bradycardia or repetitive (greater than 50% of contractions) late or significant variable decelerations, each with absent fetal heart rate variability (type III), there is a substantial risk of impending damaging asphyxia. However, the false positive rates of these patterns (type III) are very high, and the majority of nonreassuring fetal tracings during labor are associated with normal outcomes. Thus, none of these patterns can be used to predict CP and mental retardation as an outcome ascribed to intrapartum asphyxia. However, if accelerations occur above a normal baseline and variability of any degree is present, then it frequently rules out intrapartum acidosis or asphyxia as a cause of neonatal encephalopathy and CP. An in-depth review of the fetal heart rate tracing is helpful in confirming or refuting asphyxia as the cause of newborn depression.

Apgar scores can be subjective. Numerous factors can affect the Apgar scores, including intrapartum maternal sedation or anesthesia, congenital malformations, the individual assigning the score, resuscitative efforts, and the presence of an infection. This can result in

speculation on the quality and response to resuscitation. Although low Apgars are poor predictors of long-term neurologic outcome, there is a good correlation with extremely low Apgars (0, 1, and 2) at 15 to 20 minutes and subsequent neurologic dysfunction. For example, Apgar score of less than 3 at 15 minutes was associated with a 53% neonatal mortality rate and a 36% CP incidence. Conversely, it is also true that 75% of children with CP have normal Apgar scores. The fine details of resuscitation require documentation or they could be used erroneously to support intrapartum asphyxia. Inability to achieve an adequate airway in a depressed newborn or failure of a previously damaged fetus to transition to extrauterine life are common etiologies of low Apgar scores and can erroneously lead to the assumption that this depression is attributable to the obstetric care. This is also important because the 30-minute decision–incision guideline may impact Apgar scores, as well as umbilical and neonatal blood gas sampling. It is paradoxical to note, however, that in 50% to 65% of cases, the decision–incision interval exceeds 30 minutes, but the lower Apgar scores and blood gases are usually found in those who have an interval of less than 30 minutes and often less than 15 minutes.

Multisystem organ dysfunction is physiologically related to the diving reflex. In the majority of cases, intrapartum asphyxia deprives all other organs of oxygenated blood before the flow of oxygen to the brain is diminished. Studies have demonstrated that a cord pH 6.92 or less is the threshold linked with neonatal organ dysfunction at 72 hours of birth. Many expert witnesses erroneously consider a transient decrease in urine output (less than 2 $mL/kg^{-1}/h^{-1}$) or a slight elevation in liver enzymes to be signs of

multiorgan failure. The presence of pulmonary hypertension, tricuspid insufficiency, hypocalcemia, hypoglycemia, abnormal cardiac enzymes, and coagulopathy may be more supportive of multiorgan failure after a significant intrapartum event if other causes cannot be ruled out.

Several patterns of brain injury may result from hypoxic-ischemic episodes in the fetus and depend on the severity of cerebral hypotension, the maturity of the brain at the time of injury, and the duration or recurrence of the event. Cerebral edema usually appears approximately 24 hours after a significant asphyxial episode and resolves in 3 to 5 days. The presence of cerebral edema on an ultrasonogram on the first day of life would not be consistent with an acute intrapartum asphyxial event. The evolution of cystic periventricular leukomalacia in preterm newborns takes 2 to 3 weeks after an insult to be visualized using conventional imaging studies such as computed tomography and ultrasonography scans. Magnetic resonance imaging has emerged as a valuable tool for determining the timing and etiology of neonatal brain injury. Hypoxia-ischemia in term newborns typically results in one of two characteristic patterns of brain injury: 1) a basal ganglia distribution pattern involving deep gray nuclei, hippocampus, and perirolandic cortex with additional cortical involvement when severe, and 2) a watershed distribution pattern involving intervascular boundary-zone white matter plus cortical gray matter when severe. Acute total asphyxia mainly involves the brain stem nuclei, thalami, and basal ganglia and is associated with dystonic CP and brain stem deficits. Prolonged partial asphyxia involves mainly the cerebral cortex, especially parasagittal regions, and is associated with spastic quadriplegia and microcephaly. In term newborns, basal ganglia and thalamic lesions evolve through a neurotoxic cascade during the first week after the insult. Imaging studies obtained too early

after birth may appear normal even when there has been severe injury to the brain. It is important to consider not only which imaging studies to obtain but also when to schedule them to optimize the results in attempting to determine the timing of the alleged insult. Neuroimaging can be helpful in approximating a window of time when the injury might have occurred.

4. Allegations of professional liability in neonatal-perinatal medicine

We next identified the most common events in the care of sick newborns leading to litigation against practitioners. Multiple allegations were common due to the prolonged care of the newborn. Table 4 lists the top ten allegations of professional liability against practitioners of neonatal perinatal medicine. The ten most frequent allegations brought against practitioners who care for newborns included: inadequate airway/intubation (21%), failure to recognize air leak (18%), delayed transfer to Level III facility (14%), inadequate treatment of seizures (11%), delayed attendance at delivery (10%), cardiac tamponade (malpositioned central line) (6%), failure to perform eye exam (6%), medication error (6%), midgut volvulus (5%), and hyperbilirubinemia (kernicterus) (3%). Meritorious allegations against practitioners in newborn care are frequently preventable events. Substandard neonatal resuscitation in the delivery room can also propagate non-meritorious allegations against obstetricians.

Allegation	**N = 100**
Inadequate airway/intubation	21%
Failure to recognize air leak (pneumothorax)	18%
Delayed transfer to Level III facility	14%
Inadequate treatment of seizures	11%
Delayed attendance in the NICU/delivery room	10%
Cardiac tamponade (central line)	6%
Failure to do eye exam (blindness)	6%
Medication error (overdose)	6%
Midgut volvulus	5%
Hyperbilirubinemia (kernicterus)	3%

Table 4. Top ten allegations against practitioners of newborn medicine

We found that the most common allegations were a result of difficulties in the management of airways and air leaks in newborns. The procedural skills, including proficiency in intubation and thoracentesis, require a significant amount of clinical experience. Evolving technology over the last two decades with steroids, surfactants and ventilation have reduced the acuity of neonatal lung disease with concomitant reduction in intubations and chest tube placement. The recent restrictions on the time that pediatric residents are allowed to spend in intensive care units, set by the Accreditation Council for Graduate Medical Education, has contributed even more to their reduced experience with these procedures. More than half of all intubation attempts by pediatric residents are unsuccessful leading to multiple attempts by caregivers to properly place the endotracheal tube. Also, general pediatricians are often the primary caregiver when resuscitation of newborns in the delivery room is required. Their residency programs must ensure they become proficient in the resuscitation and care of the newborn.

Procedural skills teaching based on observing the skill, performing the skill, then teaching the skill is not adequate for proper training. Improving opportunities for clinical experience with intubation and thoracentesis may reduce legal actions against practitioners. Simulation-based training can have a role to provide a realistic medical situation in which learners can gain exposure to clinical tasks and anatomical regions. Approximately 10% of newborns require some form of resuscitation at birth, and a skilled resuscitator is necessary for all deliveries even when they are considered low risk. In our clinical experience, the most common etiology of decompensation in a newborn is airway related, with chest compressions rarely indicated when an adequate airway is effectively established. When an adequate airway is achieved, but newborns do not respond to resuscitation, one needs to expediently consider a pneumothorax in the differential diagnosis. Failure to recognize an air leak was the second most common allegation found in this study. An unrecognized air leak is the most common etiology for sudden unexplained death in unsuccessful newborn resuscitation. A tension pneumothorax is an acute life threatening event that may not allow the time for x-ray confirmation. Prompt recognition and needle aspiration of the pleural space should result in rapid clinical improvement for these newborns. It has been our experience that poor newborn outcomes as a result of improper delivery room resuscitation often are erroneously attributed to the delivering obstetrician. A depressed newborn requiring vigorous resuscitation with poor Apgars more often than not creates the mindset that it must be the obstetrician's fault.

Due to the critical state of newborns in the NICU, numerous protocols have been instituted to reduce iatrogenic events. When set protocols were not followed rigorously, we found that cardiac tamponade and blindness resulted in allegations of malpractice. Central lines are frequently used in the treatment of newborns for both medication and nutrition infusion, but their use carries significant risks. The possible malposition and migration of a central catheter can result in perforation of the myocardium or pericardial effusion which can be fatal. It is recommended that the central line be optimally placed outside of the right atrium to reduce these risks. Newborns with central lines must be carefully monitored with serial radiographs to confirm the position of the central line throughout the course of their treatment. Another protocol set forth in the care of newborns is an eye exam for all preterm newborns less than 33 weeks gestational age at 4 to 6 weeks chronological age. A newborn is almost never too sick for an eye exam, although nurses may feel that their patient is too unstable to be dilated and examined.

The potential for rapid decline in an unstable newborn requires that their caregivers not delay in proper treatment measures. A prolonged response time for physicians during an emergency situation, as well as delayed transfer of newborns to a proper level NICU were common allegations found in this study. All hospitals have contracts that require trained personnel to be at a high-risk scenario within a certain time frame. A delayed response to a page, and the lack of an alternative plan to notify a skilled resuscitator can result in catastrophic consequences for a compromised newborn. In addition, critical care of a newborn often requires advanced services that are not available in all NICUs. Level I, II, II+, and community Level III centers have set policies and regulations overseen by their regional perinatal center. The lack of experience of nursing and respiratory personnel in a low volume NICU can contribute to deviations in the standard of care. Regionalization continues to have a role, and is in the best interest of mothers and their newborns.

Common allegations in this study also resulted from a failure to recognize life-threatening conditions including seizures and intestinal mid-gut volvulus. Newborn seizures can be difficult to clinically diagnose due to subtle abnormal ocular and focal movements. Subtle motor abnormalities with concomitant desaturation and/or apnea often represent seizure activity. The first line of medication in the treatment of seizures in newborns is phenobarbital at a loading dose of 20 mg/kg to achieve therapeutic levels of 20-40 μg/mL. An adequate airway is essential if one desires to increase phenobarbital dosing. Persistent seizures may require the addition of phenytoin or ativan. Inadequately treated seizures can result in permanent neuronal cell damage due to enhanced metabolic activity. Malrotation of the intestine is usually observed in the neonatal period and presents with signs of acute intestinal obstruction and often bilious emesis. Mid-gut volvulus is a true surgical emergency, where delay can result in ischemic necrosis of the entire gut which is most often lethal. The upper GI series is the method of choice for diagnosing malrotation. Importantly, an acutely ill newborn with a history of bilious emesis needs immediate surgical consultation. Early diagnosis and treatment of these two conditions is essential in facilitating good outcomes.

Medication errors are preventable events that frequently occur in the NICU and are a common source of allegations. It has been previously reported that out of every five adverse drug events in pediatric patients, three of those events occurred in an NICU. Errors are particularly dangerous in the NICU due to the fragile state of newborns. The rapidly changing body weight, different rates of organ development affecting drug pharmacokinetics, and need for dilutions of medications contribute to the common occurrence of medication errors in the NICU. In this study, medication errors occurred as a result of incorrect dosing, documentation, or processing. Morphine, sodium supplementation, and aminoglycosides were the most frequent pharmacological agents administered inappropriately. With the advent of computerized order entry, a reduction in ordering errors is expected due to standardized templates for physicians and nurses. The computerized system also provides an additional way to intercept errors before they affect the newborn. Documentation, communication, and attention to detail can help to reduce preventable medication errors.

In the 21st century, kernicterus still occurs throughout the United States. The most common allegations in our experience were delayed contact and response of the blood bank as well as the inability to perform a timely exchange transfusion. An umbilical venous line is relatively easy to place, even in a newborn up to a week old. However, withdrawing blood is often problematic when using a 3.5 or 5.0 umbilical venous catheter due to the thin walled umbilical vein that collapses with minimal negative pressure. An exchange catheter in the exchange transfusion tray should be utilized whenever possible to expedite the procedure. Too often, subspecialty services are called to gain vascular access, which can greatly delay initiation of the exchange transfusion. A thorough physical exam documenting any signs or symptoms of kernicterus should be charted prior to, during, and after the exchange transfusion.

Although tort reform in some states has reduced non-meritorious legal suits, professional liability involving caregivers of mothers and newborns is significant. We have identified common areas in obstetrics and newborn medicine that resulted in malpractice claims. All

practitioners in our field need to examine these areas within their practice and address any deficiencies, implement new protocols, and improve communication and documentation in the medical record. Addressing the issues described can potentially have a favorable impact on the medical malpractice crisis, and more importantly avoid potentially preventable devastating outcomes. We cannot overemphasize the importance of honesty, humility, compassion and competency in all our interactions with our patients.

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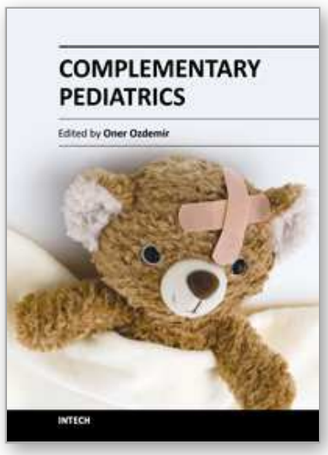

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