

# Forcep Deliveries: Are They Worth The Risk?

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## I. Background

The use of forceps has always been “controversial.”<sup>1</sup> Forceps were originally designed to be used “in desperation” and only as a “last resort.”<sup>2</sup> Given the risk of fetal injury, some obstetricians question whether forceps use is still acceptable.<sup>3</sup> The risk of fetal injury is a major reason for the decline in forceps use over the last few decades.<sup>4</sup> With fewer doctors teaching and performing forceps deliveries, it is possible that their use may be “obsolete” in the near future.<sup>5</sup> Also, because of fewer deliveries and less training, operators performing forceps deliveries have less experience than ever before. Unfortunately, many injuries result from inexperienced operators attempting forceps deliveries. Inexperience breeds errors in performance which can have devastating and lifelong effects on newborns and their families.

## II. Conditions for Forceps Use

Any obstetrician planning to use forceps must be able to use them safely.<sup>6</sup> The following are “minimum” criteria which must be present before forceps are used:

- “1. The fetal head is engaged at 0 station or lower.
2. The membranes are ruptured.
3. The cervix is fully dilated and effaced.
4. The exact position of the fetal head is known.
5. The patient’s pelvis has been assessed for adequacy and the maternal-fetal size available.
6. Appropriate maternal anesthesia is available.
7. Necessary personnel and support equipment are available.
8. The surgeon is knowledgeable about the instruments and technique and possesses the skills necessary to use them.
9. There is a willingness to abandon attempts if the forceps delivery does not proceed readily.

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<sup>1</sup>Hankins, et al, *Operative Obstetrics* (1995) (“Use of obstetrical forceps has been controversial since their invention.”) p. 130.

<sup>2</sup>Hankins, et al, *Operative Obstetrics* (1995) (“...Forceps were invented as an extricating tool to be used in desperation in the most difficult circumstances and usually as a last resort.”), p. 129.

<sup>3</sup>Apuzzio, et al, *Operative Obstetrics* (2006) (Modern obstetrical management, offering other alternatives, calls into serious question whether the benefits justify the risk of such an instrumental delivery.”), p. 281.

<sup>4</sup>Hankins, et al, *Operative Obstetrics* (1995) (“Concern for fetal injury is one of the major reasons for the decline in operative vaginal delivery. Fear of litigation certainly contributes; however, most obstetricians deny this as a large factor.”), p. 169.

<sup>5</sup>Apuzzio, et al, *Operative Obstetrics* (2006) (“In this climate, teaching the use of obstetrical forceps becomes increasingly difficult. This can only accelerate the end of this era in obstetrics. The profession may be in the twilight zone of the age of forceps delivery.”), p. 281.

<sup>6</sup>Apuzzio, et al, *Operative Obstetrics* (2006) (“It is the responsibility of the obstetrician who contemplates the use for forceps to ensure that the conditions for the safe use of these instruments have been fulfilled.” ), p. 286.

10. The patient's informed consent has been obtained orally or preferable in written form."<sup>7</sup>

If any of these criteria are not present, forceps should not be used.

Similarly, physicians considering forceps must consider all "contraindications" for forceps use. Examples of contraindications include: uncertain or unknown fetal position, "mentum posterior face presentation," brow presentation, unengaged fetal head, incomplete cervical dilatation, a contracted pelvis, fetal macrosomia, and "lack of experience of the operator!"<sup>8</sup> An experienced operator is one of the "most important prerequisites" for forceps delivery because "incorrect application increases the risk of injury."<sup>9</sup>

### III. Use and Placement of Forceps

In the event that forceps are used, proper placement and use is essential to a successful delivery. Moreover, proper placement and use of forceps alone should not cause intracranial bleeding, seizures, or permanent brain damage. Defendants and experts will agree that normal forceps use should not result in seizures, intracranial bleeding, and permanent brain damage.

When placing forceps, "the blades should lie evenly against the sides of the head, reaching to and beyond the malar eminences, symmetrically covering the space between the orbits and the ears."<sup>10</sup> Asymmetric application of forceps is a known cause of "asymmetric compression of the fetal head",<sup>11</sup> as well as intracranial bleeding.<sup>12</sup> The types of brain bleeds seen with improper forceps use include subdural and subarachnoid hemorrhages,

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<sup>7</sup>Hankins, et al, Operative Obstetrics (1995), p. 140.

<sup>8</sup>Apuzzio, et al, Operative Obstetrics (2006), p. 286.

<sup>9</sup>Hankins, et al, Operative Obstetrics (1995), p. 141.

<sup>10</sup>Hankins, et al, Operative Obstetrics (1995), p. 147.

<sup>11</sup>Hankins, et al, Operative Obstetrics (1995), p.147.

<sup>12</sup>"When forceps blades are applied symmetrically, the cephalic curve of the forceps matches the cranial curve and makes it possible to apply the force on the largest surface possible. Conversely, **if the forceps blades are not applied symmetrically to the sagittal plane, the cephalic curve of the blade does not match the fetal skull; the tensile strain increases, and a deformation (i.e., a DSF) can occur.**" Dupuis, O. et al., "Comparison of 'instrument-associated' and 'spontaneous' obstetric depressed skull fractures in a cohort of 68 neonates", Am Journal of Obstetrics & Gynecology, Vol. 192, Issue 1 (January 2005). "A mechanical traction can be subdivided into a normal stress and a shearing stress. Symmetric application of forceps blades leads to a compressive force without shearing stress, whereas **asymmetric application creates shearing forces.**" Dupuis, O. et al., "Comparison of 'instrument-associated' and 'spontaneous' obstetric depressed skull fractures in a cohort of 68 neonates", Am Journal of Obstetrics & Gynecology, Vol. 192, Issue 1 (January 2005). "When applied to the neonate skull, **shearing forces might tear the bridging cerebral veins and lead to intracranial hemorrhage. This mechanical theory may explain the significant difference in the rate of intracranial-associated lesions between the 2 groups.** This theory advocates the symmetric application of forceps blades." Dupuis, O. et al., "Comparison of 'instrument-associated' and 'spontaneous' obstetric depressed skull fractures in a cohort of 68 neonates", Am Journal of Obstetrics & Gynecology, Vol. 192, Issue 1 (January 2005). "Asymmetry can result either from the application of the forceps blades on an unengaged head (misdiagnosis of large caput succedaneum, such as the 2 dramatic cases reported here), from the misdiagnosis of the head location (this occurs in < or = 76% of cases), or from incorrectly positioned blades (specific skills are required to locate the anterior blade correctly when the head is in an oblique position). Dupuis, O. et al., "Comparison of 'instrument-associated' and 'spontaneous' obstetric depressed skull fractures in a cohort of 68 neonates", Am Journal of Obstetrics & Gynecology, Vol. 192, Issue 1 (January 2005).

intraparenchymal and intraventricular bleeding.<sup>13</sup> Overriding occipital sutures, is also a complication of overly traumatic forceps use.<sup>14</sup> When forceps are used incorrectly, cerebral swelling and resulting ischemic brain damage have been reported in the literature.<sup>15</sup> Traumatic

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<sup>13</sup>“**Subdural, subarachnoid, intraparenchymal, and intraventricular hemorrhages have been identified in symptomatic full-term neonates. Several factors have been reported to increase the risk of symptomatic ICH in full-term newborns, and these factors include assisted vaginal delivery (forceps or vacuum extraction) . . .**” Looney, C., et al., “Intracranial hemorrhage in asymptomatic neonates: Prevalence on MR Images and relationship to obstetric and neonatal risk factors.” *Radiology*, Vol. 242 (2006). “**Trauma** seems to play some role in the genesis of primary **subarachnoid hemorrhage (SAH)** in neonates, given the increased frequency with which it is documented in instrumented deliveries, **especially those in which forceps and vacuum are used.**” Noetzel, M. et al., “Perinatal Trauma and Cerebral Palsy.” *ClinPerinatol*, Vol. 33, Issue 2 (June 2006). “The prevalence of symptomatic **subarachnoid hemorrhage** ranges from 1.3 per 10,000 live births in spontaneous vaginal deliveries to **2 to 3 per 10,000 live births in vacuum and forceps deliveries.** Increased prevalence occurs with prematurity and asphyxia. . . .” Uhing, M., “Management of Birth Injuries.” *Clinics in Perinatology*, Vol. 32, No. 1 (March 2005). “Several factors have been reported to **increase the risk of symptomatic ICH in full-term newborns, and these factors include assisted vaginal delivery (forceps or vacuum extraction), maternal parity, fetal weight, and prolonged duration of labor.**” Looney, C. et al., “Intracranial Hemorrhage in Asymptomatic Neonates: Prevalence on MR Images and Relationship to Obstetric and Neonatal Risk Factors”, *Radiology*, Vol. 242, No. 2 (Feb 2007). “**Infants delivered by vacuum extraction or other operative techniques may be more likely to sustain major injuries** than those delivered spontaneously, but the extent of risk is unknown.” Towner, D. et al., “Effect of Mode of Delivery in Nulliparous Women on Neonatal Intracranial Injury.” *New England Journal of Medicine*, Vol. 341, No. 23 (Dec 2, 1999). “**. . . and the use of forceps is more likely to be associated with subarachnoid and subdural hemorrhage.**” Jhavar, B. et al., “Risk Factors for Intracranial Hemorrhage among full-term infants: a case-control study.” *Neurosurgery*, Vol. 52 (March 2003). “**A retrospective case-control study in 66 term infants imaged within 7 days after birth showed an increased risk of intracranial hemorrhage with forceps-assisted delivery.**” Gupta, S. “Intracranial Hemorrhage in Term Newborns: Management and Outcomes” *Pediatric Neurology*, Vol. 40, No. 1 (2009). “**Intracranial hemorrhage may occur in the subdural, subarachnoid, intraparenchymal, and intraventricular spaces. It occurs in approximately 5 to 6 live births per 10,000 and can be potentially fatal or cause lifelong disability. Forceps and vacuum delivery, precipitous delivery, prolonged second stage of labor, and macrosomia are recognized risk factors.**” Doumouchtsis, S. et al., “Head Trauma after Instrumental Births”, *Clinics in Perinatology*, Vol. 35, Issue 1 (March 2008). “**Jhavar and colleagues found that among full term infants forceps delivery was 6 times more likely to be complicated by subarachnoid or subdural hemorrhage than spontaneous vaginal delivery.**” Doumouchtsis, S. et al., “Head Trauma after Instrumental Births”, *Clinics in Perinatology*, Vol. 35, Issue 1 (March 2008). “**The types of intracranial hemorrhage associated with instrumental deliveries are subdural and subarachnoid hemorrhages** rather than intraventricular.” Doumouchtsis, S. et al., “Head Trauma after Instrumental Births”, *Clinics in Perinatology*, Vol. 35, Issue 1 (March 2008). “**The degree of compression must be minimized so that the balance will be in favor of the amount of stress resulting from forceps use, as compared to the forces at work during spontaneous expulsion.**” Mines, J., “Application of the Obstetric Forceps”, *Obstetrics & Gynecology*, Vol. 36, NO. 5 (November 1970). “**Intracranial hemorrhage** occurred in 1 of 860 infants delivered by vacuum extraction, **1 of 664 delivered with the use of forceps**, 1 of 907 delivered by cesarean section during labor, 1 of 2750 delivered by cesarean section with no labor and 1 of 1900 delivered spontaneously.” Towner, D. et al., “Effect of Mode of Delivery in Nulliparous Women on Neonatal Intracranial Injury”, *The New England Journal of Medicine*, Vol. 341, No. 23 (Dec 1999). “In addition, incorrect application of the obstetric forceps is often held responsible for the small, ping-pong ball depression.” Menkes, *Child Neurology*, 7<sup>th</sup> edition, pp. 368 (2006).

<sup>14</sup>According to Volpe, *Neurology of the Newborn*, “occipital diastasis” (overriding occipital sutures) involves the “forward movement of the lower portion of the squamous bone” which “distorts the occipital sinus and its tributaries with a similar effect on venous pressure.” “**These bony distortions** would be expected with breech extractions and **difficult forceps extractions.**” p. 496.

<sup>15</sup>“Additionally, **ischemic cortical involvement can be related to direct trauma or a stretch injury of an intracranial vessel.**” Noetzel, M. et al., “Perinatal Trauma and Cerebral Palsy.” *ClinPerinatol*, Vol. 33, Issue 2 (June 2006). “**Diffuse cerebral swelling following pediatric TBI is an important contributor to intracranial hypertension, which can result in ischemia and herniation.**” Huh, J. et al., “New Concepts in Treatment of Pediatric Traumatic Brain Injury”, *Anesthesiology Clinics*, Vol. 27, Issue 2 (June 2009). “**Cerebral swelling**

brain injury can also result in secondary damage, including ischemia, cerebral swelling,

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**contributes to intracranial hypertension, leading to secondary ischemia**, and, if uncontrolled, to brain herniation after TBI.” Bayir, H. et al., “Traumatic brain injury in infants and children: Mechanisms of secondary damage and treatment in the intensive care unit”, *Crit Care Clin*, Vol. 19 (2003). “Early low CBF occurs during a time when cerebral metabolic demands are increased. After the initial 24 hours, when cerebral metabolic rate for oxygen is depressed, glucose use may be normal or increased, suggesting that in some cases low CBF may represent **ischemia**.” Bayir, H. et al., “Traumatic brain injury in infants and children: Mechanisms of secondary damage and treatment in the intensive care unit”, *Crit Care Clin*, Vol. 19 (2003). “Subdural, subarachnoid, **intraparenchymal**, and intraventricular **hemorrhages have been identified in symptomatic full-term neonates. Several factors have been reported to increase the risk of symptomatic ICH in full-term newborns**, and these factors include **assisted vaginal delivery (forceps or vacuum extraction)** . . .” Looney, C., et al., “Intracranial hemorrhage in asymptomatic neonates: Prevalence on MR Images and relationship to obstetric and neonatal risk factors.” *Radiology*, Vol. 242 (2006). “**Intracranial hemorrhage may occur in the** subdural, subarachnoid, **intraparenchymal**, and intraventricular spaces. It occurs in approximately 5 to 6 live births per 10,000 and can be potentially fatal or cause lifelong disability. **Forceps** and vacuum delivery, precipitous delivery, prolonged second stage of labor, and macrosomia are **recognized risk factors**.” Doumouchtsis, S. et al., “Head Trauma after Instrumental Births”, *Clinics in Perinatology*, Vol. 35, Issue 1 (March 2008). “These cases demonstrate that the forces generated on the fetal cranium by vacuum extraction are similar to those produced by forceps and result in **tentorial laceration, venous rupture, and subdural hemorrhage**. Because these hemorrhages may be associated with significant ischemic injury, serial radiologic evaluation is recommended for the detection of persistent structural abnormalities.” Hanigan, W., “Tentorial hemorrhage associated with vacuum extraction”, *Pediatrics*, Vol. 85 (April 1990). “**Intracerebral hemorrhage in a neonate rarely is an isolated phenomenon, if the injury stems primarily from mechanical factors**. More commonly it is associated with depressed skull fractures and subarachnoid hemorrhage, as well as epidural hematomas.” Noetzel, M. et al., “Perinatal Trauma and Cerebral Palsy.” *ClinPerinatol*, Vol. 33, Issue 2 (June 2006). “More typically, **traumatic tentorial laceration** and injury to the sinuses or the vein of Galen produce a posterior fossa hematoma that is associated with a cerebellar parenchymal hemorrhage in about 16% of the cases.” Noetzel, M. et al., “Perinatal Trauma and Cerebral Palsy.” *ClinPerinatol*, Vol. 33, Issue 2 (June 2006). “Intracranial hemorrhage in term neonates usually occurs in the vicinity of the falx and **tentorium cerebelli**, producing posterior fossa hemorrhage in the dural space, **or it may occur within brain parenchyma**.” Gupta, S. “Intracranial Hemorrhage in Term Newborns: Management and Outcomes” *Pediatric Neurology*, Vol. 40, No. 1 (2009). “In clinical practice, **hemorrhage involving multiple compartments is not unusual**.” Gupta, S. “Intracranial Hemorrhage in Term Newborns: Management and Outcomes” *Pediatric Neurology*, Vol. 40, No. 1 (2009). “**The pathogenesis of intracerebellar hemorrhage is undoubtedly multifactorial, but particular importance can be attributed to traumatic delivery** (breech or **forceps extractions** or both) and circulatory events related to prematurity. . . . **In the term infant the pathogenesis appears to relate principally to traumatic events**. The pathogenesis of intracerebellar hemorrhage is best considered in term so intravascular, vascular, and extravascular factors.” Volpe, *Neurology of the Newborn*, 4<sup>th</sup> edition, pp. 410 (2001). “In a major tear of the **tentorium** with rupture of the vein of Galen, straight sinus, or transverse sinus, the hemorrhage extends into the posterior fossa, compresses the cerebellum, obstructs the 4<sup>th</sup> ventricle, and rapidly resulting lethal brainstem compression.” Huang, C. et al., “Tentorial Subdural Hemorrhage in Term Newborns: Ultrasonographic Diagnosis and Clinical Correlates”, *Pediatric Neurology*, Vol. 7, No. 3 (1991). “Focal areas of cortical necrosis and hemorrhage result from direct compressive effects in the newborn. . . .**The tears of white matter are attributed to shearing forces within subcortical cerebral substance produced by rapid and extreme deformation of the brain**. The latter is made possible by the pliability of the newborn skull. An additional predisposing factor may relate to the relative lack of myelin in the developing cerebral white matter.” Volpe, *Neurology of the Newborn*, 4<sup>th</sup> edition, pp. 820 (2001). “**Intrapartum factors present in this cohort of infants with cerebellar injury included** abnormalities in fetal heart rate tracings and **instrument-assisted vaginal delivery in approximately one third of newborns**, and the need for emergent cesarean section in one out of every four deliveries.” Limperopoulos, C. et al., “Cerebellar Injury in Term Infants: Clinical Characteristics, Magnetic Resonance Imaging Findings, and Outcome”, *Pediatric Neurology*, Vol. 41, Issue 1 (July 2009). “Neonatal intracerebellar bleeds are very rare and often seen in association with forceps or vaginal breech deliveries. . . . These neonates are most often left with significant neurologic sequelae.” Rijhsinghani A., “Neonatal intracerebellar hemorrhage after forceps delivery. Report of a case without neurologic damage”, *J Reprod Med*, Vol. 42 (Feb 1997).

hypoxemia, hypotension, intracranial hypertension, hypercarbia, hyper- or hypoglycemia, electrolyte abnormalities, enlarging hematomas, coagulopathy, seizures, and hyperthermia.<sup>16</sup> Often times, intracranial bleeds may not be immediately symptomatic.<sup>17</sup> Apgars and cord blood

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<sup>16</sup>**“Immediate or primary brain injury results from the initial forces generated following trauma. ...The first form of secondary brain injury, such as hypoxemia, hypotension, intracranial hypertension, hypercarbia, hyper- or hypoglycemia, electrolyte abnormalities, enlarging hematomas, coagulopathy, seizures, and hyperthermia are potentially avoidable or treatable.”** Huh, J. et al., “New Concepts in Treatment of Pediatric Traumatic Brain Injury”, *Anesthesiology Clinics*, Vol. 27, Issue 2 (June 2009). “The other form of secondary brain injury involves an endogenous cascade of cellular and biochemical events in the brain that occurs within minutes and continues for months after the primary brain injury, leading to ongoing or “secondary” traumatic axonal injury (TAI) and neuronal cell damage (delayed brain injury), and ultimately, neuronal cell death.” Huh, J. et al., “New Concepts in Treatment of Pediatric Traumatic Brain Injury”, *Anesthesiology Clinics*, Vol. 27, Issue 2 (June 2009). **“Diffuse cerebral swelling following pediatric TBI is an important contributor to intracranial hypertension, which can result in ischemia and herniation.”** Huh, J. et al., “New Concepts in Treatment of Pediatric Traumatic Brain Injury”, *Anesthesiology Clinics*, Vol. 27, Issue 2 (June 2009). “Other data suggest that **posttraumatic hypoperfusion** was more common and a global decreased CBF (<20 mL/100 g/min) in the initial first day following TBI in infants and children was associated with poor outcome.” Huh, J. et al., “New Concepts in Treatment of Pediatric Traumatic Brain Injury”, *Anesthesiology Clinics*, Vol. 27, Issue 2 (June 2009). “One of the most important consequences of secondary brain injury is the development of **intracranial hypertension.**” Huh, J. et al., “New Concepts in Treatment of Pediatric Traumatic Brain Injury”, *Anesthesiology Clinics*, Vol. 27, Issue 2 (June 2009). “After traumatic brain injury (TBI), **irreparable brain damage, termed primary injury resulting from direct disruption of brain parenchyma.** Potentially reversible secondary injury also takes place as the result of altered hemodynamic and respiratory function and cellular derangement. TBI triggers a common pathway of neuronal death involving posttraumatic ischemia, energy failure, excitotoxicity, mitochondrial failure, oxidative stress, and secondary cerebral swelling.” Bayir, H. et al., “Traumatic brain injury in infants and children Mechanisms of secondary damage and treatment in the intensive care unit.” *Crit Care Clin*, Vol. 19 (2003). “In the largest report of CBF [cerebral blood flow] after severe TBI in infants and young children, **hypoperfusion** was common during the first 24 hours, and ischemia (global CBF < 20mL) was associated with poor outcome.” Bayir, H. et al., “Traumatic brain injury in infants and children Mechanisms of secondary damage and treatment in the intensive care unit.” *Crit Care Clin*, Vol. 19 (2003). “Early low CBF occurs during a time when cerebral metabolic demands are increased. After the initial 24 hours, when cerebral metabolic rate for oxygen is depressed, glucose use may be normal or increased, suggesting that in some cases low CBF may represent ischemia.” Bayir, H. et al., “Traumatic brain injury in infants and children Mechanisms of secondary damage and treatment in the intensive care unit.” *Crit Care Clin*, Vol. 19 (2003). **“Cerebral swelling contributes to intracranial hypertension, leading to secondary ischemia,** and, if uncontrolled, to brain herniation after TBI.” Bayir, H. et al., “Traumatic brain injury in infants and children Mechanisms of secondary damage and treatment in the intensive care unit.” *Crit Care Clin*, Vol. 19 (2003). “The blood-brain barrier is more vulnerable to disruption in the immature brain than in adults. Experimental studies suggest **that blood-brain barrier disruption is maximal during the first few hours after injury, and that secondary insults such as hypoxemia and hypotension worsen blood-brain barrier damage.**” Bayir, H. et al., “Traumatic brain injury in infants and children Mechanisms of secondary damage and treatment in the intensive care unit.” *Crit Care Clin*, Vol. 19 (2003).

<sup>17</sup>“...between birth and the 3<sup>rd</sup> day of life...” Nelson, *Pediatrics*, 16<sup>th</sup> ed., Birth Injury, p. 489 (2000). “A smaller tear of the tentorium or rupture of the bridging veins from the superior cerebellum, which may be more common, can result in subacute symptoms; a lag period of a few hours to a few days may appear between birth and the time neurologic symptoms appear.” Huang, C. et al., “Tentorial subdural hemorrhage in term newborns: ultrasonographic diagnosis and clinical correlates”, *Pediatric Neurology*, Vol. 7, No. 3 (1991). “...up to 3 days after its occurrence.” McKinstry RC, *Neurology*, 24-SEP-2002; 59(6): 824-33. The connection between VE and cerebral bleeds was noted by Christine Pope, M.D. at <http://www.emedicine.com/med/topic3389.htm>. “... within 48 hours of birth.” Uhing, *Clinical Perinatology*, 01 Mar, 2005; 32(1): 19-38 “...up to a few days after delivery,” Duchon MA, *Ob Gyn*, 01 Feb, 1988; 71(2): 155-8). “. . . five peri/intraventricular hemorrhages (cases 8, 9, 14, 17 & 18) occurred at 5 or more days following birth, which confirmed previous reports that these hemorrhages [in full-term infants] occurred at a later age than usually seen in preterm infants.”<sup>Ref 1</sup> Siu SLY, Kwong NS, So, KT. A 10-year Review of Intracranial Haemorrhage in Term Neonates, *HK J Paediatr (New Series)* 2006; 11:140-146. “In some infants bleeding can be a **slow process** rather than a sudden event... Term infants with intraventricular hemorrhage

gases are often normal. Neonates with intracranial bleeds are at increased risk for seizures<sup>18</sup> and status epilepticus later in life.<sup>19</sup>

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tend to become symptomatic at a later age, often not until the fourth week of life.” Menkes JH, Sarnat HB, Maria BL., *Child Neurology*, 7<sup>th</sup> ed., Intracranial Hemorrhage, pp. 390-391 (2006). “It is important to recognize that, although uncommon, full-term neonates may present with apnea as the initial manifestation of either right or left temporal lobe intracranial hemorrhage.” Sirsi, D. et al., “Apneic Seizures: A Sign of Temporal Lobe Hemorrhage in Full-Term Neonates”, *Pediatric Neurology*, Vol. 37, No. 5 (2007). “Clinical manifestations include seizures, irritability, recurrent apnea, depressed level of consciousness and focal neurologic signs. The diagnosis is made by cranial CT.” Doumouchsis, S. et al., “Head Trauma after Instrumental Births”, *Clinics in Perinatology*, Vol. 35, Issue 1 (March 2008).

<sup>18</sup> “Intracranial hemorrhage (ICH) in full-term neonates commonly is associated with apnea, bradycardia, and seizures.” Looney, C. et al., “Intracranial Hemorrhage in Asymptomatic Neonates: Prevalence on MR Images and Relationship to Obstetric and Neonatal Risk Factors”, *Radiology*, Vol. 242, No. 2 (Feb 2007). **“Intracranial hemorrhage is a common cause of neonatal seizures in full-term infants.”** Sirsi, D. et al., “Apneic Seizures: A Sign of Temporal Lobe Hemorrhage in Full-Term Neonates”, *Pediatric Neurology*, Vol. 37, No. 5 (2007). “Intracranial hemorrhage is the cause of neonatal seizures in 17% of full-term infants, and is more commonly seen after vaginal delivery assisted with forceps or vacuum extraction.” Sirsi, D. et al., “Apneic Seizures: A Sign of Temporal Lobe Hemorrhage in Full-Term Neonates”, *Pediatric Neurology*, Vol. 37, No. 5 (2007). “Second, primary subarachnoid hemorrhage can result in seizures, especially in full-term infants. The seizures usually have their onset on the second postnatal day.” Volpe, *Neurology of the Newborn*, 4<sup>th</sup> edition, pp. 407 (2001). “If extensive, subarachnoid hemorrhage in the neonate may provide seizures.” Menkes, *Child Neurology*, 7<sup>th</sup> edition, pp. 368 (2006).

<sup>19</sup> “Repeated seizures in newborns may be accompanied by serious hypoventilation and apnea, which result in hypercapnia and hypoxemia. The latter is an important potential cause for brain injury, particularly in an infant whose brain already has been compromised by a serious insult.” Volpe, *Neurology of the Newborn*, 4<sup>th</sup> edition, pp. 182 (2001). **“Risk of epilepsy was increased after a mild brain injury, severe brain injury, and skull fracture. The risk was increased more than 10 years after mild brain injury, severe brain injury and skull fracture. ... The risk was slightly higher in women.”** Christensen, J. et al., “Long-term risk of epilepsy after traumatic brain injury in children and young adults: a population-based cohort study.” *Lancet* (Feb 23 2009). “The 27 survivors were followed for a mean of 31 months. PNE (postnatal epilepsy) developed in 56% (15 of 27) of the cohort. **The first seizure appeared at a mean-corrected age of 12.7 months and occurred despite ongoing antiepileptic medication in 60% (9 of 15) of the group.**” Clancy, R. et al., “Postnatal epilepsy after EEG-confirmed neonatal seizures.” *Epilepsia*, Vol. 32 (Jan 1991). “Neonatal seizures were associated with a high incidence of postnatal epilepsy in the cohort, including epileptic syndromes with catastrophic evolution.” Da Silva, L et al., “Risk Factors for Developing Epilepsy after Neonatal Seizures” *Pediatric Neurology*, Vol. 30, No. 4 (2004). **“neonatal seizures remain important predictors of future neurologic complications. ... Although mortality rates have been reduced, there remains a high morbidity rate, epilepsy being a frequent complication of neonatal seizures.”** Da Silva, L et al., “Risk Factors for Developing Epilepsy after Neonatal Seizures” *Pediatric Neurology*, Vol. 30, No. 4 (2004). **“The occurrence of epilepsy after neonatal seizures varies in frequency, as reported in previous studies, from 3.5% to 56% according to sample selection.”** Da Silva, L et al., “Risk Factors for Developing Epilepsy after Neonatal Seizures” *Pediatric Neurology*, Vol. 30, No. 4 (2004). **“A close association between seizures and the development of permanent deficits has been demonstrated in previous studies. Postnatal epilepsy is included among these deficits.”** Da Silva, L et al., “Risk Factors for Developing Epilepsy after Neonatal Seizures” *Pediatric Neurology*, Vol. 30, No. 4 (2004). “Early neonatal seizures seem to be associated with greater morbidity and mortality, directly related to the gravity of the encephalic injury.” Da Silva, L et al., “Risk Factors for Developing Epilepsy after Neonatal Seizures” *Pediatric Neurology*, Vol. 30, No. 4 (2004). “This hypothesis is in agreement with previous studies that additionally suggested that **the presence of multiple risk factors was associated with a higher probability of developing post-neonatal epilepsy, including epileptic syndromes with catastrophic evolution.** An abnormal neurologic examination on discharge was a good predictor of an unfavorable outcome, and abnormal polysomnographic recordings were moderate predictors.” Da Silva, L et al., “Risk Factors for Developing Epilepsy after Neonatal Seizures” *Pediatric Neurology*, Vol. 30, No. 4 (2004). “Generalized tonic-clonic status epilepticus may be recurrent in 17-25% of children with status epilepticus. **Recurrent status epilepticus primarily occurs in children with neurological abnormalities.**” Sotero de Menezes, M. et al., “Status Epilepticus.” <http://emedicine.medscape.com/article/908394>.

#### **IV. Sequential Use of Forceps and Vacuum Extraction**

The sequential use of instruments significantly increases the risk of both maternal and neonatal morbidity.<sup>20</sup> Obstetricians should be trained in the appropriate use of instruments with the goal being safe delivery with one instrument.<sup>21</sup>

#### **V. Legal Considerations**

Attorneys pursuing forcep birth trauma cases should ask the following questions:

1. Were the forceps properly placed?
2. What was the fetal station at the time for the forcep use?
3. What was the level of forcep experience of the delivering doctor?
4. Did a resident perform the forcep delivery?
5. How much traction was used?
6. How many pulls were there?
7. Was it a failed forcep delivery?
8. Were forceps and a vacuum extractor sequentially used?
9. Was there bruising noted in the delivery and neonatal records?
10. Were there overriding sutures?
11. Did the head imaging show intracranial hemorrhages?
12. Were there neonatal seizures?
13. Did subsequent head imaging show atrophy and damage consistent with prior brain damage?
14. Did the child subsequently develop cerebral palsy, usually of a hemiparetic type?
15. Was adequate informed consent given?
16. Was the delivery rushed?

Many attorneys and experts will only get involved in the most obvious types of traumatic forceps cases involving the use of high or mid forceps, documented excessive traction, numerous pulls or sequential use of forceps and vacuum extraction. It may be a mistake to pass on what appears to be the more benign forceps delivery. For example, where the caregiver documents forceps use involving a single pull with gentle traction still malpractice may still exist. These more benign deliveries are sometimes still pursuable so long as there are neonatal findings consistent with forceps trauma.

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<sup>20</sup> Murphy, DJ, et al, "A cohort study of maternal and neonatal morbidity in relation to use of sequential instruments at operative vaginal delivery." *Eur J Obstet Gynecol Reprod Biol.* 2011 May;156(1):41-5;  
Gardella, C, et al, "The effect of sequential use of vacuum and forceps for assisted vaginal delivery on neonatal and maternal outcomes." *Am J Obstet Gynecol.* 2001 Oct;185(4):896-902.

<sup>21</sup> Murphy, DJ, et al, "A cohort study of maternal and neonatal morbidity in relation to use of sequential instruments at operative vaginal delivery." *Eur J Obstet Gynecol Reprod Biol.* 2011 May; 156(1):41-5.

## **Conclusion**

Though forceps deliveries are less and less frequent, the potential for catastrophic birth trauma still exists. Overall, any contraindications to performing a forceps delivery must be thoroughly explored prior to making a decision to perform a forceps delivery. When reviewing a potential case involving a forceps delivery, it is vital to consider the conduct of the physician applying the forceps, the application of the forceps, and the resulting injury. A detailed review of subsequent care records, including head imaging, may be beneficial as the initial injury may not always be symptomatic. Lastly, a number of questions should be considered prior to making a determination about whether or not to pursue the case. After a thorough review of the facts, even the cases that appear to be routine forceps deliveries may be persuuable.

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Jesse M. Reiter is Co-Chair of the Birth Trauma Litigation Group and has been practicing law for 24 years. He handles exclusively birth trauma cases, representing children and their parents. His firm, Reiter & Walsh, P.C., is listed in US News & World Report/Best Lawyers: Best Law Firms for 2010 and 2011-2012. Mr. Reiter's multi-million dollar birth trauma verdicts and settlements are routinely among the top reported in Michigan. He is admitted to practice in Michigan, Washington D.C., Ohio, and the United States Supreme Court. In 2006, Mr. Reiter was selected as a Michigan Lawyer of the Year. In 2012, he was chosen as a Leader in the Law in Michigan. He served as President of the Michigan Association for Justice from 2006-2007 and is a frequent speaker and moderator at seminars. He is a Fellow of the AAJ National College of Advocacy and a Fellow of the State Bar of Michigan Foundation. He served as Co-Editor of the Birth Trauma Litigation Group Newsletter from 2006-2011, and is secretary of AAJ's Professional Negligence Section. Jesse Reiter has been selected to Michigan Super Lawyers 2008 through 2012, as well as Best Lawyers in America since 2008, Crain's Detroit Business Top Lawyers, and DBusiness Best Lawyers in Metro Detroit. He is AV-Preeminent rated by Martindale-Hubbell.

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