Briefly Legal: Neonatal Neurological Injury Associated with Vacuum-Assisted Delivery

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Vacuum-assisted deliveries (VAD) account for about 5% of all deliveries. (1) They are considered safe and effective when maternal, fetal, or obstetrical indications (Table) mandate an expedited delivery and prerequisites are met. Serious fetal injuries are uncommon, and long-term population studies comparing children delivered by VAD with those delivered by normal spontaneous vaginal delivery demonstrate no difference in neurocognitive outcomes. (2). Notwithstanding the infrequent association of neonatal injury with VAD, they are commonplace in medico-legal allegations of fetal injury during labor.

Case History

Past Medical History

At the time of her delivery, the patient was a 31-year-old primigravida. Her past medical history included a LEEP procedure on her cervix. During her pregnancy, she developed hyperemesis gravidarum with an 18 lb. weight loss, treated with intravenous hydration. At 20 weeks gestation, she developed palpitations, tachycardia, and tremors, and from that time forward, her care was provided by high-risk specialists. She had two glucose challenge tests: the first was normal, and the second was elevated at 8.1 mmol/L. A follow-up GTT was normal. Her prenatal antenatal lab work was normal. At 29 weeks; gestation, a Cardiology consultation found bouts of premature atrial contractions but no pathologic source for her cardiac symptoms and tremors. The fetus demonstrated normal activity, growth, and amniotic fluid volume despite these issues.

At 40.5 weeks gestation, she appeared at the hospital complaining of contractions. She was assessed as being in "false labor" with a reassuring NST. She was scheduled for induction of labor for postdates the next day only to begin labor spontaneously with leakage of clear fluid early the next morning. On admission to the hospital, her cervix was 3 cm. dilated, 80% effaced, and the head was at -3 station (3/80/-3). The initial tracing showed moderate variability, accelerations with fetal movement, and absent decelerations with intermittently prolonged and coupled contractions. After 3 hours, she was 5 /100/-2. After 9 hours with the cervix unchanged, the patient requested analgesia, and shortly after that, she requested a cesarean section rather than continue with a trial of labor. The nurse encouraged her to think it over, and the trial of labor was continued.

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At 10 hours, the patient was given Demerol for pain; after 15 hours, the cervix was unchanged over the past 9+ hours. At 17 hours of labor, oxytocin augmentation was initiated, and at 19 hours, an epidural was placed. At 24 hours, the cervix was only 7 cm dilated; at 28 hours, the cervix was 8 / 100% / -1; and by 30 hours, the cervix was only 9 cm dilated. Despite the excessive uterine activity, oxytocin was increased following each of these examinations. Finally, at 32 hours, the cervix became fully dilated, with the presenting part at 0 station. The FHR tracing showed fetal tachycardia (165 bpm) with moderate variability and accelerations; decelerations were absent. There had been numerous runs of excessive uterine activity in which four contractions occurred within 6 minutes. Over time, the baseline heart rate rose to 180 bpm with decreased to absent variability and prolonged accelerations with intermittent, brief variable decelerations.

With the onset of pushing at 33+ hours, the baseline rose to 190 bpm, accompanied by late decelerations with frequent and coupled contractions. Almost 90 minutes later, at 35 hours, she stopped pushing because she was tired. When pushing ceased, the patient received a top-off dose of anesthesia. In response, the fetus showed further deterioration in the tracing with more obvious late and prolonged decelerations with decreased /absent variability and baseline FHR of about 180 bpm - a "conversion pattern." The fetus had likely suffered an injury – prior to applying the vacuum. (3)

Table I

Indications for VAD

Maternal	Maternal exhaustion or inability to push.
	Cardiac, pulmonary, vascular, neurological, and ophthalmological conditions in which (lengthy) pushing is contraindicated
Fetal -	Fetal compromise that is not relieved by conservative measures.
Obstetrical -	Prolonged second stage.

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At 36 hours, the patient was positioned for a vacuum-assisted delivery (VAD). A Kiwi vacuum device was placed (at a "+2 station"), and traction was applied in concert with the patient pushing. The vacuum popped off and was then reapplied. After multiple tractions with pushing over the next 35 minutes, the infant's head was delivered, but the development of shoulder dystocia stalled the delivery of the body. Using various maneuvers (McRobert's, suprapubic pressure, and Woods), the shoulder was released after about 3 minutes. During these efforts after the pop-off of the vacuum, the FHR tracing revealed a stable heart rate of about 140 bpm, with moderate variability and accelerations coincident with the mother's expulsive efforts. Decelerations were absent despite the persistent attempts at VAD. This sequence represents the maternal heart rate (MHR) insertion, not the FHR, into the recording – a well-known pitfall of external fetal monitoring. (4)

The labor pattern represents a markedly abnormal labor curve – a significant protraction, if not arrest, of the active phase of labor. Having achieved full dilatation, it will be almost five hours before a complicated and prolonged operative vaginal delivery ultimately delivers the patient. The mother sustained a 3rd-degree laceration of the perineum. The procedure details are not adequately described in terms of the station, molding, caput, flexion, number of pulls, or effort. In his deposition, implausibly, the physician maintained that there was no caput, and the fetal head descended with each traction.

The placental weight plotted out at the 3rd percentile with an F/P ratio of 8.1. Microscopic examination revealed "acute chorioamnionitis" with Stage 2, Grade 1 maternal and fetal inflammatory responses. There was also a "mild to moderate" lymphohistiocytic villitis involving about 2% of the placental tissue.

The Newborn

The 3500-gram male infant received Apgar scores of 4, 6, and 8 at 1, 5, and 10 minutes respectively. His head circumference was 34.5 cm, and his length was 37.2 cm, yielding a ponderal index of 2.4. There is no immediate description of the baby's head following the prolonged VAD. Umbilical blood gases show mild metabolic acidemia. At birth, the baby's heart rate was >100 bpm, with poor muscle tone and weak respirations requiring CPAP with 100% oxygen. The umbilical venous pH was 7.22, pCO_2 39, pO_2 18, HCO3- 16 mmol/L, and base deficit (BD) 10. His arterial cord pH was 7.16, pCO_2 51, pO_2 20, HCO3- 18 mmol/L, and BD 11. A capillary blood gas collected at about 70 minutes of age revealed a pH of 7.17, pCO_2 49, pO_2 38, HCO3-18 mmol/L, and BD 10.9 mmol.

On admission to the nursery, the baby was described as "alert" and moving appropriately with intact primitive reflexes. He was maintained on CPAP. An IV was started, and antibiotics were administered. At about 1.5 hours of age, the baby's hemoglobin was 16.8 g/L. His WBC count was 40,850 x 10^6/L, the neutrophil count 25,570, the nucleated red blood cell count was mildly

Table II

Prerequisites for VAD

1. Gestational age at least 35 weeks gestation
2. Knowledge of previous deliveries and pelvic adequacy
3. Absent fetal disorders
   a. bleeding disorder (hemophilia, von Willebrand's, alloimmune thrombocytopenia)
   b. Osteogenesis imperfecta, ?IUGR
4. Properly obtained informed consent, including:
   a. the recommendation, indication, benefits, risks, and alternatives to VAD.
   b. The understanding that the procedure may be unsuccessful and that cesarean section may be necessary. (Risk >15%)
   c. Reassurance that the patient will be updated during labor about factors influencing the likelihood of operative delivery.
   d. A willingness to abandon the procedure if difficulties are encountered.
5. Immediate availability of anesthesia providers should a cesarean section be necessary. (especially if an indication is "fetal distress."
6. Empty maternal bladder
7. Adequate anesthesia
8, No previous attempt at operative vaginal delivery (e.g., forceps)

Table III'

The feasibility of safe delivery and the success of VAD

The previous obstetrical history

The course of labor (patterns of dilatation of the cervix and the descent of the fetal head)

The estimated fetal weight

The amount of caput, molding, asynclitism

The station, position of the fetal head

The fetal head engagement is not the same as 0 station – (May need an abdominal exam to verify.

elevated, and his platelet count was mildly depressed at 130,000 x 10^6/L.

The baby's mean blood pressure at 4 hours of age was 37 mmHg but improved somewhat over time. At about 12 hours of age, the baby's capillary lactate level was elevated at 6.3 mmol/L. His blood glucose was 3.1 mmol/L, and his ionized calcium was low at 1.03 mmol/L. Because of an x-ray showing free air in the mediastinum, the infant was taken off CPAP. Subsequently, his oxygen saturation fell to about 40%, and he had an episode of "staring" associated with bradycardia to 90-95 bpm. Sepsis, intracranial pathology, seizures, and a metabolic abnormality were considered in the differential diagnosis. There ensued multiple episodes of apnea associated with oxygen desaturation and bradycardia. A head ultrasound revealed a right subependymal hemorrhage with some dilation of the third and fourth ventricles. At about 15 hours, the infant was described as "pale/pink, jittery, and hypertonic." He was noted to be less active than earlier in the day. His heart rate had slowed, and his urine output was "decreased." A spinal tap was negative, as was a CSF culture. At 18 hours, the infant displayed rhythmic, rightward movements of his eyes, right hand, and leg, for which lorazepam was administered, followed by phenobarbital and Acyclovir, for suspected seizure activity.

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About 25 hours after birth, the infant was transferred to a Children's Hospital. The admission note included a reference to "increased upper limb tone" and swelling over the occipital area of the head, attributed to the vacuum extraction. Blood work in the hours after the admission included a-mildly abnormal INR at 1.7 and 1.8; the serum creatinine was unremarkable

Although clinical seizure activity was absent, an aEEG revealed subclinical seizures for which pyridoxine and phenobarbital were administered. Subsequently, phenytoin was added to the regimen for "apnea" spells which required PPV. The aEEG continued to show a discontinuous voltage pattern and multifocal sharp waves but no seizures. There were no cortical responses on visual or somatosensory evoked potential testing.

The baby's urine output was low; at less than 0.5 ml/Kg/hour, his serum sodium had fallen to 122 mmol/L. His sodium level and urinary output were corrected with an infusion of concentrated sodium chloride. Various metabolic and genetic investigations collected on the first day of admission were negative. In the afternoon of DOL 2, a neurological examination was abnormal, with a lack of spontaneous movement, "quivering, shuddering, shivering" movements, and increased tone and deep tendon reflexes on his right side.

An ultrasound examination of the brain at 36 hours of life demonstrated minimally increased echogenicity in the basal ganglia, thalami, and slit ventricles. MRI examination on DOL 5 demonstrated increased signal (T1) restricted diffusion in the basal ganglia and thalami, perirolandic regions, parasagittal frontal lobes, and the corpus callosum splenium and in the posterior watershed regions, left greater than right with small posterior and posterior fossa subdural hematomas. T2 images reveal increased signals in the white matter and heterogeneous signals in the basal ganglia and thalami. Subarachnoid hemorrhage was also seen. The ventricles were normal in size, shape, and position. The MR angiogram and venogram were normal, but there was bilateral caput, mild bilateral cephalohematomas, and a mild subgaleal hemorrhage.

Follow-up MR performed at DOL 25 demonstrated a markedly increased signal in the BGT, with faint extension to the perirolandic regions. There was mild atrophy and prominence of the ventricles and sulci related to decreased brain volume. On T2, there was an increased signal in the white matter, especially in the perirolandic regions. The subdural hematomas had decreased in size but were still apparent.

These images were entirely consistent with a recent hypoxic-ischemic injury and obvious trauma. None of these findings

Table IV

Factors compromising the success of VAD

1. Maternal obesity, Large fetus
2. Protracted labor (dilatation or descent)
3. Malposition (OP, OT), Asyclitism
4. High presenting part
5. Pop-offs
6. Failure to obtain descent of the fetal head with each traction
7. The prolonged effort and need for "excessive force."
8. The improper direction of the traction

suggest any congenital abnormality or more remote injury. The basal ganglia and thalamus involvement are consistent with an acute near total episode, the parasagittal frontal lobes, and the posterior watershed regions, typical of (prolonged partial pattern) HI injury.

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The allegations of the plaintiff

The care of the patient and her fetus during labor fell hopelessly below any reasonable standard of care. Throughout, there were failures to:

1. Appreciate the diminishing feasibility of safe vaginal delivery.
2. Appreciate and respond to the abnormalities of the FHR tracing, including the insertion of the MHR into the tracing.
3. Recognize and properly respond to abnormalities of the uterine contraction patterns.
4. Recognize and properly control the rate of infusion of oxytocin.
5. Failed to appreciate the improbability of safe vaginal delivery with a vacuum extractor and provide informed consent to the patient.
6. Improper use of the vacuum device
7. Failed to abandon attempts at VAD in a safe and timely manner.
8. Failed to make simultaneous preparations for double-setup should the procedure fail.
9. These failures led to permanent, hypoxic-ischemic, and traumatic injuries to the baby's brain during his mother's labor and delivery.
10. These injuries were preventable by adherence to reasonable standards of obstetrical care.
11. In addition, the nurses failed to properly inform the responsible physicians and substandard recording of the events of labor and delivery and a description of the head of the newborn.

The response of the defense:

1. While in utero, hypoxia-ischemia event was "a significant contributor" to the baby's neurological impairments, it was most likely due to an acute, severe event that likely took place between 12 hours and four days prior to birth.
2. The clinical signs manifested immediately after birth and resolving by two days of life were "not in keeping with having suffered [severe] hypoxia-ischemia in the final hours of labor."
3. Although the baby's birth weight and clear amniotic fluid volume were in the normal range, the small placenta raised concerns about long-term placental insufficiency leading to a hypoxic-ischemic insult.
4. While chorioamnionitis is commonly seen in normal births and babies with normal outcomes, the pathology report noted vasculitis of the umbilical cord blood vessels, a particularly strong risk factor for cerebral palsy. (5)
5. The infection is a contributing feature but was not the direct cause of the baby's encephalopathy.
6. The base deficit (BE) values of the cord (-11 mmol/L) and on follow-up at about 71 minutes (10.9) are seen very frequently in infants who go on to have a normal neonatal course and normal long-term outcome. Ultimately, these values failed to meet the "criteria for perinatal asphyxia" as vouchsafed by ACOG (6).

Table V

Neonatal complications (11-13)(12-14)

Hemorrhage – intracranial, Subgaleal, cephalohematoma

HIE – Consider injury in 2nd stage of labor preceding VAD.

Shoulder dystocia - brachial plexus injury.

Bruising, chignon, scalp trauma

"For the fetus to be injured on admission to the hospital, as believed by the defense experts, requires that the fetus suffer a significant hypoxic-ischemic event sufficient to cause discernible neurological injury in the two days between hospital visits without a change in fetal activity or FHR pattern. The experts do not comment on what that stealth event was or when it might have occurred to precipitate such an injury in this normally grown, apparently responsive fetus."

Comment

For the fetus to be injured on admission to the hospital, as believed by the defense experts, requires that the fetus suffer a significant hypoxic-ischemic event sufficient to cause discernible neurological injury in the two days between hospital visits without a change in fetal activity or FHR pattern. The experts do not comment on what that stealth event was or when it might have occurred to precipitate such an injury in this normally grown, apparently responsive fetus. The defense experts seem content to attempt to derive the timing and mechanism of injury from the clinical and laboratory circumstances accompanying the newborn. They paid no attention to the prolonged course of labor, the excessive uterine activity, fetal cardiac decelerations, tachycardia and the misadventures with the vacuum, and the potential for trauma associated with the operative delivery. There is also no appreciation that for over 20 minutes in the 2nd stage of labor, the MHR, not the FHR, is being monitored.

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Vacuum usage

The decision to proceed with VAD requires careful selection of patients, proper attention to technique, and continual progress in descent with each traction effort. The safety of a VAD is universally linked to the proper selection of patients based on maternal, fetal, and obstetrical indications and prerequisites (Tables I and II). (7) (8) The prerequisites for VAD include full dilatation of the cervix, an engaged presenting part, based on knowledge of the position and the "true" station of the presenting part (taking into account potentially confounding features such as molding, malposition, caput, and asynclitism). Prerequisites also include a reasonable expectation of success and a willingness to abandon the procedure when difficulties supervene (detachments or "pop-offs"), when descent is not made with each pull, or when the "excessive" effort is required. Vacca (9) pointed out that the maximum force applied to the vacuum handle can be generated with the operator's flexing fingers. In the appropriately selected patient, a vacuum-assisted vaginal delivery will be accomplished within 10-15 minutes using no more than five pulls in association with maternal pushing.

The VAD is undertaken only with reasonable expectations of success. Numerous factors influence the likelihood of success after reaching full dilatation, and the prerequisites met. (Tables III and IV). The reported failure rate for vacuum extractions varies considerably from about 5% to about 25% and is significantly higher with the fetal head in the occiput posterior position. (10) It is widely understood. However, outcome statistics for vacuum deliveries are confounded by the fact that the indication for the vacuum, especially "fetal distress," is associated with adverse outcomes. In an analysis of 179 cases of neonatal injury associated with VAD that were the subject of medico-legal action, Schifrin et al. found a VAD failure rate of about 50%. In that study, the appearance of injury raised essential questions about the proper selection of patients, the technique, and especially in multiple applications of the device. Notably, hypoxic-ischemic injury following VAD was more likely to represent injury during the 2nd stage of labor before applying the vacuum. Abstract: Marinac-Dabic D, Schifrin BS, Bright R. Adverse Effects of Vacuum Assisted Delivery Devices. ACOG – Annual Clinical Meeting – April 2003, New Orleans, LA)

"Maternal complications occur in about 10% and consist mostly of perineal and vaginal lacerations. Fetal complications (Table V) are increased with prolonged or multiple applications or with the device's application at the high station, in nulliparas, or those with a history of cesarean section. VAD in a prior pregnancy is a risk factor for cesarean section in a subsequent delivery."

Maternal complications occur in about 10% and consist mostly of perineal and vaginal lacerations. Fetal complications (Table V) are increased with prolonged or multiple applications or with the device's application at the high station, in nulliparas, or those with a history of cesarean section. VAD in a prior pregnancy is a risk factor for cesarean section in a subsequent delivery. Sequential use of instrumental delivery carries significantly higher neonatal morbidity than when a single instrument is used. Undertaking a VAD is associated with an increased risk of shoulder dystocia and subsequent brachial plexus injury.

The known risks of VAD should prompt a directed evaluation of the neonate over the first several hours of life with prompt attention to the potential for clinically apparent SGH. During this observation period, a bonnet should not be placed on the baby's head.

Denouement

In the abstracted case, undertaking a vacuum under the circumstances was fraught, with few prospects of safe vaginal delivery, irrespective of the failure to recognize the insertion of the MHR into the tracing. Labor progress, especially in the 2nd stage, was poor. There was an apparent failure to descend, with a malposition (ROT). In this respect, given the duration of labor and pushing and the malposition of the fetal head, the physician's belief that there was no caput or molding of the fetal head seems implausible. These latter features are indeed present on the neonatal MRI examination. At the time of the VAD, the physician averred that the fetal head was in ROT position and at +2 station - presumably engaged. Because of the obvious caput, molding, and malposition (and the difficulty in effecting delivery), the fetal head was likely higher in the pelvis (and unengaged) despite the estimated +2 station. This interpretation would make the initial attempt at VAD contraindicated by reasonable standards of care. Further, it was only with extraordinary efforts of traction and maternal pushing that the fetus was delivered vaginally.

In this case, the appearance of a "conversion pattern" on the tracing permitted the conclusion that the fetus had indeed suffered a neurological injury before applying the vacuum. (3) This does not exclude the potential for additional ischemic or traumatic harm from the VAD or the manipulations associated with releasing the shoulder dystocia. It is also important to consider the potential synergistic effect of infection as evidenced by placental pathology showing chorioamnionitis on both the maternal and fetal sides of the placenta, along with sustained fetal tachycardia. Chorioamnionitis appears to reduce the fetal threshold for brain injury, especially in a fetus with abnormal FHR tracing and prolonged labor. (5)

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These features, including the normal tracing at the outset of labor, permit the conclusion that the most likely timing of the hypoxic-ischemic and traumatic injuries occurred during the intrapartum period and was preventable with the provision of reasonable standards of care and proper attention to the reasonable interpretation of the course of labor, uterine contractions, and the fetal responses thereto.

The case was settled prior to trial.

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