Briefly Legal: Fetal Heart Rate Patterns and the Timing of Fetal Neurological Injury

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More than 5 decades after its introduction, the benefits and even the classification of FHR patterns continue to be debated. Contradictory research appears about the relationship between FHR patterns, fetal acidemia, and/or low Apgar scores, and the risks of subsequent neurological injury. With regularity, articles continue to appear on the role (adverse to some) of EFM tracings in pursuing claims against physicians and hospitals alleging obstetrical negligence in the causation of subsequent injury. In these judicial deliberations, the interpretation of fetal heart rate patterns is often raised, and Daubert's challenges (asserting that EFM is nothing more than junk science) are not uncommon. Based on a recent case decided by the Italian Supreme Court of Cassations' several Italian authors have joined American authors in the crusade to abolish or leverage the use of EFM (CTG) in the courtroom. (1 2,3 4 5) While most of these challenges fail (deservedly so), some may succeed even in the same state.

Our purpose here is not to argue these issues in depth or even gainsay the widespread notion among obstetricians that EFM tracings are "litogens" – instruments that promote lawsuits. Instead, we discuss an EFM tracing that was of inestimable help to defend a malpractice case, essentially vitiating an allegation of obstetrical malpractice during the discovery period.

Case Report:

At the beginning of her second pregnancy, conceived with in-vitro fertilization, the mother was 39 years of age. Her first pregnancy, four years previously, delivered spontaneously at 38+ weeks' gestation of a 3200-gram baby who has done well.

This pregnancy was followed closely with frequent prenatal visits, several ultrasound examinations early in the pregnancy, and NST testing over the last several weeks. Three Non-stress tests (NSTs) were invariably reassuring.

At about 38.3 weeks gestation and 6 days after the last NST, she appeared at the hospital at 0900 in early labor. She was examined and found to be 3 cm dilated, unchanged from an earlier examination in the obstetrician's office. She was sent home to return in about 12 hours for admission. There was no attempt to monitor the fetal heart rate (FHR) then. Other than occasional mild contractions, there were no complaints of vaginal bleeding, abdominal pain, rupture of the membranes, or decreased fetal movements.

With no change in symptoms, she returned to the hospital at about 22:00. She was examined, and the cervix was again found to be 3 cm dilated. She was placed on the fetal monitor. In contrast to the previous NST, the tracing showed a stable baseline FHR of 160 bpm, diminished baseline variability, and recurrent decelerations with contractions – now coming every 5-6 minutes. Rupture of the membranes revealed a small amount of meconium-stained fluid but failed to promote progress in cervical dilatation over the next 4 hours. With the administration of misoprostol (Cytotec), the progress in labor became fairly brisk, and within an hour, she was fully dilated, at which time the fetal monitor tracing was discontinued. An epidural was administered, which did not appear to be effective. In the delivery room, spinal anesthesia was administered. The doctor did not arrive until 20 minutes later; 15 minutes thereafter, the baby delivered spontaneously. The mother's postpartum course was uneventful.

"The baby weighed 2660 grams at birth, about 600 grams smaller than the mother's first child. The Apgar scores were 2 (for heart rate) and 6 at 1 and 5 minutes, respectively. The body and head were stained with meconium. Initially, the baby was not breathing, crying, or moving; its skin color was blue/purple. Because of breathing difficulties (felt to be due to meconium aspiration syndrome), the baby was intubated and admitted to the NICU."

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difficulties (felt to be due to meconium aspiration syndrome), the baby was intubated and admitted to the NICU. Umbilical blood gases do not appear in the record, although the nurse's notes record them as being obtained. Arterial blood gas obtained at about 45 minutes of age revealed a pH of 7.20 with a base deficit (BD) of 8. Calculating backward using the formula of Shah (6) suggests a BD of about 13 at delivery. Based on this blood gas result, the decision was made to forego therapeutic hypothermia (cooling). The placenta was unavailable for examination.

"The baby remained intubated for 8 hours after birth and remained in the NICU for 8 days, with an additional night in the hospital prior to discharge. Cranial ultrasounds were performed on days 2 and 9, and a Brain MRI/MRA was performed on day 14. The results of these examinations are as follows:"

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Cranial ultrasound (day 2)

The left lateral and third ventricles are asymmetric, showing narrowing compared to the right. No dilatation is detected in the right ventricular system.

- No intraventricular mass lesion or hemorrhage is observed.
- No midline shift is present.
- Bilateral symmetric increases in echogenicity are seen in the periventricular areas. No significant hemorrhagic focus is detected.
- The gray/white matter areas of the cerebral and cerebellar hemispheres are normal, as are the basal ganglia and the caudothalamic groove. The brainstem is of normal appearance with an intact corpus callosum. Follow-up ultrasound and clinical correlations are recommended to differentiate between periventricular flaring and leukomalacia.

Cranial ultrasound (day 11)

Both cerebral hemispheres, especially in the parieto-occipital and periventricular areas, reveal widespread heterogeneous hyperechoic changes. The right lateral and third ventricles are of small caliber, with a cystic enlargement located in the superior part of the right lateral ventricle, related to the ventricle.

- The left lateral ventricle has a normal width.
- The extra-axial CSF space is enlarged around the left cerebral hemisphere. Within this enlarged extra-axial space, occasional thin septations form a suspicious cystic appearance.
- No hemorrhagic findings were noted at the level of the bilateral basal ganglia or caudothalamic notch. However, at the level of the thalamus, the echo structure is heterogeneous.
- The posterior fossa structures cannot be optimally evaluated due to dense parenchymal heterogeneity.

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Conclusion:

- The case should be evaluated in conjunction with MRI to investigate hypoxic-ischemic encephalopathy and other potential pathologies.

MRI – 15 days of age:

The fourth ventricle is midline, with normal width.

- Mild sequelae with areas of increased T2 signal are observed in the lateral portions of the right cerebellar hemisphere. Apart from these areas, the posterior fossa structures and brainstem are normal.
- In the supratentorial region, widespread end-stage encephalomalacia is observed in both cerebral hemispheres. A large peri-cerebral hygroma is noted on the left.
- In the territory of the right middle cerebral artery, a large area showing diffusion restriction, compatible with subacute ischemia, is observed, involving the deep gray matter and cerebral cortex.
- In the MR angiography, the distal part of the right internal carotid artery in the cavernous segment is occluded. No flow is detected in the middle cerebral artery. Both anterior cerebral arteries and the left middle cerebral artery are filled from the left carotid system. No pathological findings were observed in the posterior circulation. · No significant mass effect or hematoma is detected.

Interpretation and Recommendations:

- Findings were consistent with widespread hypoxic brain injury and encephalomalacia in both cerebral hemispheres.
- Diffusion restriction in the territory of the right middle cerebral artery is compatible with subacute ischemia.
- Occlusion of the right internal carotid artery.

Cranial MRI and MR Angiography: at 2 months of age - compared to earlier examination:

In the supratentorial region, widespread hypoxic damage is noted

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- The previous examination observed a large diffusion restriction area in the right cerebral hemisphere within the middle cerebral artery territory. In the current examination, this area has regressed. The development of encephalomalacia in these regions is noted.
- The cerebellar hemispheres, medulla, and brainstem are normal in the infratentorial region. No additional findings were noted.

MR Angiography:

- Antegrade flow is present in both internal carotid arteries.
- The right middle cerebral artery appears occluded.
- The left anterior cerebral A1 segment is not visualized. A2 and distal branches are filled from the right carotid system. The left middle cerebral artery is open.
- Both internal carotid arteries are visualized with low calibers.
- In the posterior circulation, the distal segments of the vertebral arteries, basilar artery, and its branches are open.

Interpretation and Recommendations:

- Widespread encephalomalacia areas, nearly covering both cerebral hemispheres, are consistent with diffuse hypoxic damage in the supratentorial neural parenchyma.

Follow up

At 2 years of age, her severe brain injury carried a diagnosis of Spastic Quadriplegic Cerebral Palsy - GMFCS Level 5. Additional diagnoses included Microcephaly, Severe cortical visual impairment, Non-verbal, Mild dysphagia, Precocious puberty (first period at age 2, enlarged breast tissue), Hip dysplasia (anticipating surgical intervention), scoliosis, severe cognitive impairment, but seizure-free. She requires assistance 24/7.

A lawsuit was filed, and discovery was pursued. An expert report, in support of the plaintiff's allegations of obstetrical negligence, found fault in the failure to perform FHR testing on the morning admission as well as the subsequent management of the labor upon her return to the hospital at 10 pm that evening, including the need for cesarean section upon admission. This report stated that the FHR pattern, Category III, demanded immediate intervention shortly after admission with the expectation that the child would be normal or the labor would be foreshortened by some 6 hours. A provisional defense argument alleged that the injury had preceded the delivery by several weeks.

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A secondary review of the medical records included a more detailed and nuanced evaluation of the NST tracings at 31-, 36-, and 37 weeks gestation, bringing a different perspective to the allegations. Comparing the last NST, performed just 6 days earlier, with the initial tracing on admission to the hospital provided critical information impacting the allegations of negligent care and the timing of the fetal neurological injury. The last NST tracing betrayed a stable baseline FHR in the normal range (130 bpm) with obvious cyclic accelerations with fetal movements, absent decelerations in the face of the few obvious uterine contractions, and normal beat-to-beat variability in the FHR. Reasonably, this NST tracing represented conclusive evidence of a normally responsive, active fetus with no apparent risk of hypoxia (decreased oxygen lack) or mechanical challenges, for example, from decreased amniotic fluid volume or compromised umbilical blood flow (cord compression). It offers no insight into the presence of any growth restriction. It is a neurological/behavioral assessment with implications for satisfactory cerebral perfusion, even during intermittent contractions. There is simply no published example of a hypoxic, injured fetus with a normal FHR pattern during labor. If, for whatever reason, the baby had been delivered at that point, it would have been reasonable to conclude that the baby would have been born healthy without neurological consequences. There would appear to be no basis for fetal injury prior to that point.

However, the tracing on admission to the hospital at 2200 **represents a dramatic change in the FHR pattern from the outset** compared to the previous NST. The baseline rate is now much higher (160 bpm) (outside the normal range at this gestational age), there are no accelerations, the variation in the rate from beat to beat is diminished, and obvious decelerations consistently accompany frequent contractions. These changes in the FHR pattern reflect a dramatic change in the vulnerability of

the fetus to the point, highly likely, of neurologic injury. Despite increases in uterine contractility and periods when the monitor has been removed, there is no change in the pattern during the time of labor.

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Thus, the second expert report concluded that the baby had suffered a neurologic injury well prior to the time of the assessment on her return to the hospital. Further, this expert concluded that an immediate cesarean section at the time of admission would not likely have changed or avoided the unfortunate outcome. While there existed the potential for additional insults with the additional labor or with the duration of spinal anesthesia prior to delivery, as well as from the efforts to extract the child at the time of the cesarean section, these did not mitigate the notion that the baby had already suffered a neurological injury, one that interfered with the neurological control over the FHR. The decelerations on admission preceded the onset of monitoring, and the persistently abnormal pattern was quite stable over time, not one evolving from a recent injury. It was also reasonable to conclude that the pattern while anticipating subsequent disability and problems of neonatal adaptation, was not life-threatening (no bradycardia, no prolongation of decelerations, or failure to maintain a stable baseline FHR).

Given the ability to observe the evolution of the tracings over time, there seemed to be few alternatives to this explanation of the baby's circumstances at birth and the subsequent neurological disability. The explanation would seem to be supported by thick meconium, the small fetus size with a likely diminution in the amniotic fluid volume. The mechanism of injury seemed straightforward. Placental insufficiency decreased fetal growth (the baby was about 600 grams smaller than the first baby at the same gestational age). In addition, there was decreased amniotic fluid volume (thick meconium), potentially interfering with blood flow through the umbilical cord (decelerations) and oxygenation of the fetus. During one or more of these episodes, which antedated the onset of monitoring, the fetus, no longer capable of dealing with these compromises to the perfusion of its brain (with or without significant hypoxemia), suffered an injury revealed by the details of the tracing elaborated above. The ability of the baby to maintain a stable FHR, though elevated, in the face of repetitive cord compressions (presumably) made it unlikely that there was severe acidosis or that the child would be moribund at delivery.

This explanation of the preventability of injury, notwithstanding, as a practical matter, this second expert also opinioned that to comply with reasonable standards of care, closer attention had to be paid to the fetus, and intervention had to occur much earlier, especially with the appearance of the thick meconium-stained fluid. The long intervals where no monitoring was performed should not have occurred. The expert reaffirmed, however, that even had a cesarean section been performed immediately upon admission, the baby would likely be injured. Similarly, it is improbable that cooling would have been beneficial as it must be undertaken within 6 hours of injury.

Thus, despite the deviations in the standard of care by the care providers, that deficiency appeared to have had little to do with the ultimate injury to the baby, which had reasonably preceded that time. That is, there was no connection between the failure of the standard of care and the injury (causation). Without the link between deviations from the standard of care and the causation of the injury, the legal case could not succeed.

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It is important to emphasize that specific details of the prenatal course were sought to attempt to satisfy the clinical features expected in this circumstance, narrow the range of the estimate of the time of injury, and simultaneously change the direction and weight of the plaintiff's allegations. Irrespective of the notations in the medical record, direct inquiries of the mother repeatedly assured that there were: "no complications detected throughout the pregnancy and in the period leading up to the six-hour timeframe of the labor." This lack of complaints made refining the timing of injury insurmountable. The mechanism and the window of the timing of the injury/injuries seemed readily discernible, but without (the expected) complaints of maternal weight loss, lack of growth of the abdomen, rupture of the membranes, change in the pattern of fetal movement, or frequent contractions, or change in the final tracing, there was no obvious clinical clue to the timing of the injury beyond the notion that it happened in the 6-day interval between the last NST and significantly before the later onset of monitoring.

An assessment of the timing of the injury based on the cranial ultrasound and MRI images adds to the speculation but does not resolve the issue of the timing or preventability of injury, except perhaps to bring the estimated timing closer to the date

of admission. It also invites the speculation that there were two separate injuries, one related to the stroke and the other, a more diffuse hypoxic-ischemic injury, perhaps related to the events of labor and delivery – or vice versa.

MRI changes with HIE are dynamic. Imaging looks at a single point in time and may not show the complete picture. The ventricular asymmetry seen on day 2 can be a common finding after a vaginal delivery. The bilateral symmetrical increases in echogenicity are concerning for white matter injury, specifically PVL. The cranial ultrasound on day 11 shows the evolution of the injury. Together, they suggest that the injury was closer to the day of delivery than a week earlier.

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The MRI changes are consistent with vascular occlusion normally a finding which prompts a search for cardiac disease and such acute problems as dehydration and clotting disorders. Cerebellar injury due to HIE is increasingly understood to be a vulnerable region when cerebral blood flow is impaired.

There is no description of dissection of the carotid artery or trauma. The descriptions speak to occlusion secondary to thrombosis, although no thrombus was identified. The placenta, a potential source of embolus, was not saved or examined in detail. HIE can lead to endothelial damage, altered blood flow, and a pro-thrombotic state, all of which can contribute to further neurologic injury and worsen the clinical course.

The meconium staining of the skin speaks to exposure for several hours and diminished amniotic fluid volume but does not inform the question of the timing of the injury. Acute meconium and meconium aspiration syndrome can be related to anoxia, where the sphincter is relaxed, as well as decreasing the tone in the upper airway, making the newborn more vulnerable.

There is no basis to support the notion that this severe injury preceded the previously normal NST 6 days earlier. Nor did clinical evidence permit the conclusion that the injury (more than a stroke) occurred a few weeks before birth. The tracing, while anticipating subsequent disability, was reasonably reassuring that the fetus was not threatening to die.

As a result of the second consultation, the lawsuit was not pursued.

Commentary:

This case raises many issues related to the timing of fetal neurological injury and the roles of FHR monitoring during labor, as well as the use of neuroradiological examinations in these medico-legal disputes. It highlights several pitfalls of the classification and clinical implementation of FHR patterns. Initially, there was no comparison of the antepartum and intrapartum tracings. Indeed, the former were unavailable to the team in labor and delivery and not part of the deliberations of the neonatologists deciding on the use of HT. The markedly abnormal pattern was monitored without intervention for over 6 hours with no warning to the physician or the undertaking of any remedial measures to moderate the decelerations or reduce the amount of uterine activity.

The first obstetrical consultant invoked the assumption that the consequences to the fetus from the markedly abnormal pattern on admission would be mitigated or prevented by early intervention. It is a pitfall of modern classifications that no prospectively identifies a pattern of preceding neurological injury. In this case, the injury diagnosis depended not only on the tracing after admission but also on the comparison with the earlier tracing which reveals the dramatic, now stable, change from demonstrably normal to a markedly abnormal FHR pattern. Many reports in the literature evaluating the relationship of outcome to FHR patterns use only the last 30-60 minutes before delivery without determining whether the tracing was normal on admission. (7) Assuming that the initial tracing was normal, using the last 1-2 hours of this tracing would have invited the notion that the injury occurred during labor. Indeed, this was the premise of the first expert retained by the plaintiff.

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Given the 6-day interval between normal and markedly abnormal tracings, with no signs or symptoms to go on, there is no clinical basis further to refine the estimate of the timing of the injury – only that it was unlikely to have arisen anew during the labor. Nor can one state, more probably than not, that the tracing was normal that morning when she was first seen at the hospital. Had the tracing at that time been normal, the estimate of the injury window would have been dramatically reduced, but the patient would have been sent home again, and the result would have been the same. Had the tracing been abnormal to the extent that decelerations promptly returned to a normal baseline FHR and moderate variability, it becomes reasonable to argue that continued surveillance and timely intervention would likely have made a difference in the outcome. Consider the difference in the merits (the weight of the evidence against the providers) and the

prosecution of the case if the mother had complained of decreased fetal movement and had called the hospital or physician, only to have the complaint dismissed and no testing undertaken.

The frequent Daubert challenges and the purported ramifications of a recent Italian court decision and ongoing rancor about EFM underscores the widespread misunderstanding of FHR patterns and the purpose of monitoring itself – for both detractors and supporters. Monitoring is a technique for which no real alternative exists, including intermittent auscultation (IA). For those claiming that the introduction of EFM preceded the necessary research to justify its widespread deployment, there is no such evidence for IA – a technique that is neither reliable nor predictive of outcome. IA cannot be reviewed, and while it cannot be used to disparage the care, it cannot be used to exculpate the caregivers.

Guidelines for its implementation of EFM took years, even decades, in some cases. Physiological underpinnings, classification of patterns, and revisions in the interpretation and clinical response to patterns continue to the present day. It is disappointing that even modern reviews of FHR patterns fail to mention any effect on FHR patterns of fetal head compression or direct impairment of cerebral blood flow (other than myocardial failure).

Detractors point to the high degree of inter-and intra-observer disagreement with examples of hindsight and outcome biases. These are functions of the unfortunate and unphysiological classification of patterns, the (mis)understanding of the timing and mechanisms and immediate manifestations of fetal neurological injury during labor and delivery, and the widespread but inappropriate use of umbilical cord blood gases as the gold standard of hypoxic-ischemic injury during labor. (8, 9-11)

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Those disparaging the use of EFM suggest that "*exclusive* acute intrapartum hypoxia/asphyxia plays a minimal role in causing neonatal cerebral damage." They fail to understand that irrespective of the contribution of various prenatal factors. Most hypoxic-ischemic injuries are recent – and potentially preventable. (12) Despite acknowledging such long-term disabilities as epilepsy, autism, and intellectual disability, they continue to believe that all harm during labor will be manifested immediately as neonatal encephalopathy (NE) – belying the notion that hypoxic-ischemic injury (HII) is the most common, but not the only cause of NE.

Critics point out that the risk of cerebral palsy (CP), for example, has not changed since the introduction of EFM, but do not comment on the fact that the dramatic increase in the survival of pre-term babies, for example, who are at risk of CP, has not been accompanied by a rise in the incidence of CP. A similar argument has been made for the stable rate of intraventricular hemorrhage (IVH) in the premature infant despite their increased survival. Nor do they consider the harm of using a "normal" FHR pattern to permit very prolonged durations of labor and pushing – with adverse consequences. (13)

At a minimum, the detractors seem mostly disturbed by the use of EFM to pursue claims of obstetrical negligence, and they have gone to considerable lengths to preclude the information from being brought to court.

"Despite overwhelming evidence that EFM is inefficient in preventing neonatal brain injury, this tool is still widely used in labor wards on both high- and low-risk pregnancies. Even worse, EFM is the cardinal driver of CP litigation as in the courts, judges and "experts" continue to convict caregivers for causing CP based on unreliable CTG's ex-post reinterpretation." "Allegations against the obstetrical care provider for negligently responding to the CTG pattern are, "biased and unscientific at best or fraudulent at worst." (4)

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Some authors have argued that there should be no re-analysis of the tracing from during labor and delivery. On this basis, the reliable fetal information contained in the tracing might even be discarded, with the expectation of a dramatic decrease in "brain-damaged baby" litigation. The impact on the intact survival of babies, the cesarean section rate, or the basis of progress in the specialty without reliable fetal information obtained from the EFM is rarely discussed. Further, the detractors argue that EFM has "done more harm than good." It has increased the cesarean section rate with the expected increases in repeated multiple cesarean sections, placenta previa, placenta accreta spectrum (PAS), and peripartum deaths. Whether there has been an adverse impact on neonates is debatable, as is the notion that the threat of litigation has caused young doctors to opt out of specializing in Obstetrics. Observing the impact of these trends will be interesting if EFM is eliminated.

There is much about the culture of fetal monitoring that would benefit from revision. The place to begin, perhaps, is the question of the purpose of monitoring. EFM is currently predicated on the search for hypoxia sufficient to warrant intervention (rescue) of the fetus but insufficient to cause the fetus harm. It contains the notion that all injuries received during labor will be immediately apparent, as manifested in NE. Keeping the fetus out of harm's way and protecting the fetus' defenses against hypoxia and ischemia in the first place seems eminently more appealing. (14)

The 3-tiered classification of patterns subscribes to no physiological construct. Tracings may or may not have abnormalities of the baseline (rate, stability, variability, accelerations), and they may

or may not have decelerations widely understood to represent impaired uterine, umbilical, or cerebral blood flow. Therefore, there is a minimum need for at least a 4-part classification dealing with the various combinations of baseline features and decelerations. The difficulties of teaching such a 3-tier classification belie the expectation that the interpretations and responses will be consistent and beneficial.

Using EFM constructively requires education in the proper conduct of labor, the proper evaluation of excessive uterine activity, and the evaluation of the FHR pattern. It requires an understanding of the types of injury that may occur during labor that are unrelated to systemic fetal hypoxemia that may not be immediately apparent in the adaptation of the newborn.

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The reasoned interpretation of EFM patterns, especially their evolution over time, permits the reliable interpretation of fetal neurological behavior, the normally robust adaptive responses to challenges from potentially correctable or avoidable problems of oxygen availability either systemically (impaired uterine or umbilical blood flow) or regionally (cerebral, visceral). (14) It should dramatically reduce not all cesarean sections but those emergency cesarean sections related to acute fetal distress during a trial of labor. On occasion, EFM tracings reasonably permit the prediction of the mechanism and the timing of neurological injury even if that injury is not manifest in the immediate neonatal period. EFM also invites the understanding that despite all these potential benefits optimally applied, not all fetal neurological injuries, even one that can be timed, are preventable. (15-18) There is more work to do and more about the fetus that needs understanding.

Conclusion:

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