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# Fetal Head Compression: Its Possible Role in Neurologic Injury

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## Abstract

It is widely assumed that fetal ischemic brain injury during labor derives almost exclusively from severe, systemic hypoxemia with marked neonatal depression and acidemia. Severe asphyxia, however, is one of several causes of perinatal neurological injury and may not be the most common; most neonates diagnosed with hypoxic-ischemic encephalopathy do not have evidence of severe asphyxia. Sepsis, direct brain trauma, and drug or toxin exposure account for some cases, while mechanical forces of labor and delivery that increase fetal intracranial pressure sufficiently to impair brain perfusion may also contribute. Because of bony compliance and mobile suture lines, the fetal skull changes shape and redistributes cerebrospinal fluid during labor according to constraints imposed by contractions, and bony and soft tissue elements of the birth canal as the head descends. These accommodations, including the increase in intracranial pressure, are adaptive and necessary for efficient descent of the head while safeguarding cerebral blood flow. Autonomic reflexes mediated through central receptors normally provide ample protection of the brain from the considerable pressure exerted on the skull. On occasion, those forces, which are transmitted intracranially, may overcome the various adaptive anatomical, cardiovascular, metabolic, and neurological mechanisms that maintain cerebral perfusion and oxygen availability, resulting in ischemic brain injury. Accepting the notion of a potentially adverse impact of fetal head compression suggests that avoidance of excessive uterine activity and of relentless pushing without steady progress in descent may offer protection for the fetal brain during parturition. Excessive head compression should be considered in the differential diagnosis of ischemic encephalopathy.

## Keywords

Fetal Brain Injury, Fetal Head Compression, Ischemic Encephalopathy,

Neonatal Encephalopathy

## 1. Introduction

Enmeshed in the controversies surrounding the pathophysiology of intrapartum fetal brain injury is the debate over the role of the forces of labor on the fetal head in causing or contributing to such damage [1] [2]. The debate, going back centuries, has been constrained by the widespread contemporary assumption that fetal ischemic brain injury during labor derives almost exclusively from hypoxic stress that results in severe, systemic asphyxia with marked fetal acidemia and impaired neonatal adaptation [3] [4]. Several lines of evidence do not support such a monolithic perspective.

For example, as many as 40% of neonates with a radiological diagnosis of hypoxic-ischemic encephalopathy (HIE) do not have evidence of severe asphyxia at birth [5], casting doubt on use of the term "hypoxic" in this diagnostic rubric. Moreover, most newborns with severe acidosis have neither acute problems nor long-term neurologic dysfunction [5]. And infants with clinically mild neonatal encephalopathy (NE) have an incidence of neuroradiological markers of injury comparable to those with more severe NE, and subsequent handicap that might not be revealed until adolescence [6] [7]. Existing data permit the conclusion that neither the umbilical pH nor the severity or pattern of NE is a sufficiently sensitive clinical biomarker to define the etiology of NE reliably, to explain either the type or duration of a harmful exposure, or to identify term infants who would benefit from therapeutic hypothermia [7] [8] [9]. Thus, although hypoxia is certainly an important risk factor for neurological harm, it is neither a sensitive surrogate nor a gold standard for identifying the timing or the mechanism of intrapartum neurologic compromise.

Excessive mechanical forces acting on the fetal head during labor (**Table 1**) [10]-[15] would seem capable of causing serious brain damage. In this report we attempt to elucidate the impact of intracranial pressure (ICP) on cerebrospinal fluid (CSF) dynamics, fetal heart rate (FHR) patterns, and normal compensatory responses in protecting the brain exposed to increased pressures. In addition, we emphasize that such protection probably has limits, and that avoiding excessive fetal head compression during labor may have important salutary effects. Our goal is to summarize what is known about the potential effects of intrapartum increases in ICP and to stimulate further investigation of this important but relatively unexplored area.

## 2. Intracranial Pressure

ICP is the pressure within the skull, brain tissue and CSF. The ICP postnatally is normally 7 - 15 mm Hg. It is about 10 mm Hg higher in the fetus [16], the passive consequence of resting intrauterine pressure (IUP) imposed on the fetal body.

**Table 1.** Potential contributors to excessive head compression.

| Condition | Reference |
|---|---|
| Prolonged rupture of the membranes | [49] |
| Excessive uterine stimulation | [87] |
| Pushing without descent | [14] |
| Dysfunctional labor patterns | [15] |
| Malpositions (esp. OP, asynclitism) | [58] |
| Operative vaginal delivery (esp. from midcavity) | [1] [14] |
| Large head circumference | [12] |

The adult generally manifests a consistent relationship among ICP, intracranial volume and cerebral perfusion pressure. Referred to as the Monro-Kellie doctrine [17] [18] [19], it holds that the contents of the cranium are incompressible and that the volume inside the skull is fixed in a state of equilibrium such that any increase in volume of one of the constituents must be compensated for by a decrease in volume of another, lest the ICP rise. For example, cerebral edema from ischemia would increase brain volume, causing a rise in ICP, unless there was a compensatory fall in CSF or intracranial blood volume. This traditional view of pressure-volume relationships has not been studied in the human fetus directly, but may help explain some interesting recent observations.

Intrapartum and neonatal MRI imaging has demonstrated marked narrowing of the lateral ventricles in vaginally delivered babies [20] [21] [22]. The latter was unrelated to cerebral edema, and was presumably caused by a reduction in or redistribution of fetal CSF volume in response to increased ICP [20] [21] [22] [23]. Babies delivered by elective cesarean showed no such changes in the CSF [20]. These findings bolster the evidence for the transmission of extracranial forces into the cranial cavity.

With the fetus floating in the amniotic cavity, the intrauterine pressure (IUP) is applied symmetrically to all its parts, including the head. Once membranes are ruptured and the fetal head descends, the IUP is no longer imposed symmetrically (**Figure 1**). Then, pressure on the circumferential portion of the cranium juxtaposed to the maternal pelvis exceeds that on the remainder of the head and other body parts [24] [25]. It is increased further during contractions, expulsive efforts, and operative vaginal delivery, when it may exceed 250 mm Hg [26], far greater than any IUP generated by the contracting term uterus.

## Does External Pressure Applied to the Fetal Skull Increase the Fetal ICP?

Investigations performed on experimental animals, as well as clinical observations, provide inescapable evidence that extracranial pressures acting on the fetal skull from contractions and forceps are transmitted intracranially to increase the fetal ICP [16] [25] [27] [28] [29] [30] (**Figure 2**).

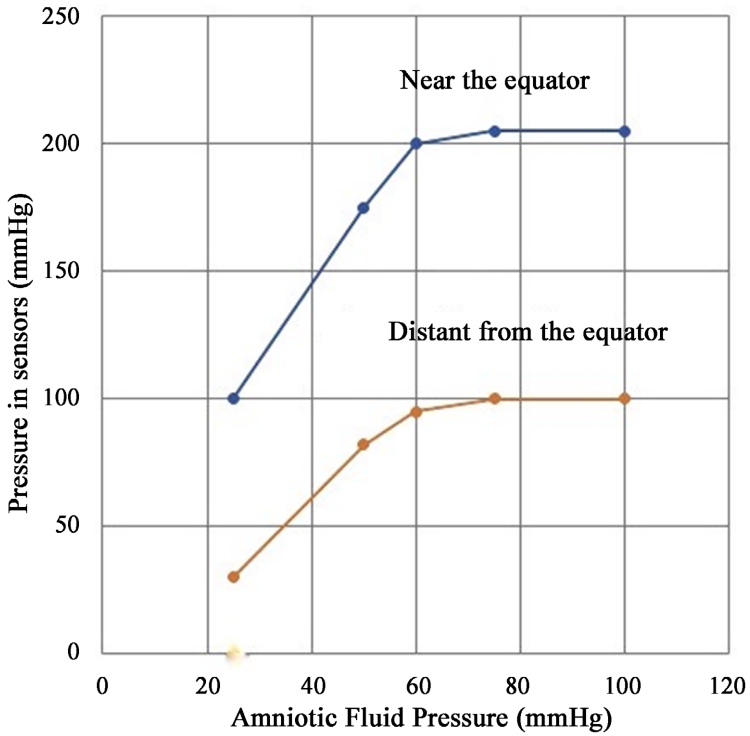


**Figure 1.** The pressure exerted by contractions on sensors placed between the fetal head and the uterine wall is asymmetric and higher than the pressure in the amniotic cavity after membranes rupture. The equator of the fetal head is exposed to the greatest force. Redrawn from Lindgren [24].

Ethical considerations obviously preclude catheterizing the normal fetal skull during labor to obtain direct measurements of ICP. Such investigations, performed on experimental animals and in unique clinical circumstances where the human fetus had a lethal anomaly or was dead [16] [25] [27] [28] [31] provide considerable support for the notion that fetal extracranial pressure is transmitted intracranially to cause increased ICP, decelerations in the FHR, and decreases in fetal CBF and cerebral $O_2$ consumption, potentially to the point of ischemic injury [16] [25] [27] [28] [29] [30] (**Figure 2**).

In the experiments of Mann, *et al.*, when head compression was applied to fetal lambs to mimic the effect of uterine contractions, the ICP increased, the electroencephalogram (EEG) became isoelectric, and brain blood flow diminished along with cerebral oxygen consumption [28]. Also in fetal sheep, increased ICP that was less than the systolic blood pressure triggered intense autonomic activity that elicited hypertension, cerebral vasodilation, and peripheral vasoconstriction, but not bradycardia [31]. When the ICP was made to exceed the mean blood pressure in a neonatal rabbit model, a deceleration in heart rate occurred [32]. Other studies in intact fetuses confirmed that external pressure on the skull caused alterations in FHR, CBF and cerebral oxygenation [24] [28] [29] [30] [33] as well as impairment of venous flow in the superior sagittal sinus, depending upon fetal head position and molding [34] [35].

In the normal human fetus, studies using carotid artery Doppler flow, near

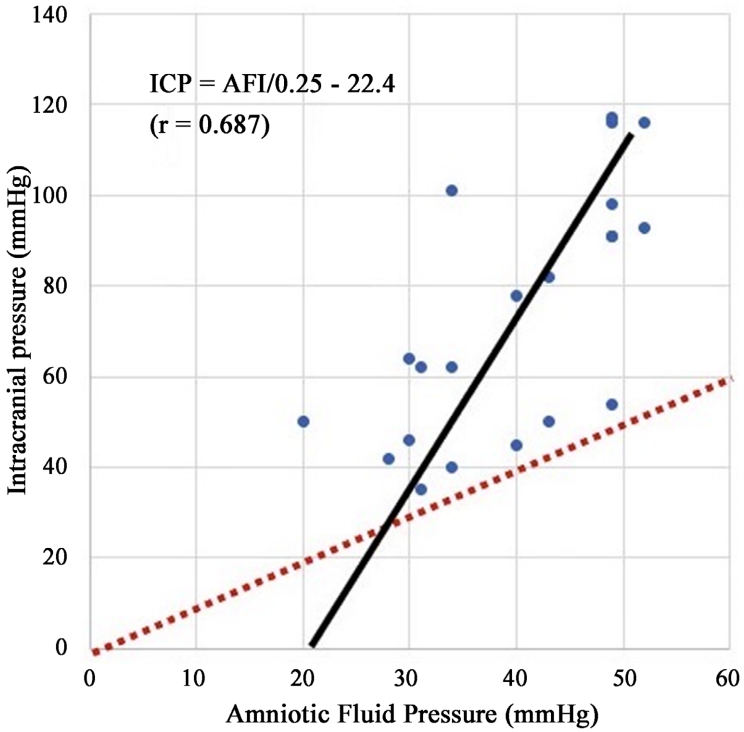


**Figure 2.** The relationship of fetal intracranial pressure (ICP) and amniotic fluid pressure (AFP) obtained with an intracranial catheter in a hydrocephalic fetus. Increases in AFP during uterine contractions were accompanied by concomitant rises in ICP, demonstrating that external pressure on the skull is transmitted intracranially. For every measurement, the ICP exceeded the IUP. Redrawn from Sugarman, *et al.* [16].

infrared spectroscopy and fetal EEG during the 2nd stage of labor, showed changes in cerebral blood flow and EEG activity unrelated to fetal hypoxia [36] [37] [38]. Kelly, *et al.* showed that IUP from contractions with pushing may easily equal the pressure applied by forceps and is frequently accompanied by FHR decelerations [1] [2] [39]. Compression of the fetal head accompanied by increases in intracranial pressure is as foreseeable a development as is the reduction in uterine blood flow associated with myometrial contractions.

The increase in ICP attending the rise in IUP during a uterine contraction may be considered protective (up to a point), in that the higher the ICP, the greater is the resistance to further head compression. This benefit is potentially offset by the potential compromise of cerebral blood flow. To deal with increases in ICP that might diminish brain perfusion, the term fetus and newborn are endowed with robust, brain-centered, compensatory adaptations that are unrelated to the presence of systemic hypoxemia or acidemia. The best known is the Cushing response, which is clearly present in the fetus and the newborn [40] [41] [42].

## 3. Compensatory Responses to Hypoxia and Ischemia

Depending on its severity, systemic hypoxemia induced by uterine contractions is detected by peripheral chemoreceptors and provokes a series of adaptive neu-

rohumoral responses that serve to slow the heart rate (late decelerations), attempt to maintain cardiac output and direct it away from the peripheral circulation toward the priority organs (placenta, heart, brain and adrenals). Additional fetal adaptations to hypoxemia include a non-injurious reduction in brain oxygen consumption, and EEG changes. In this hypoxemic model of injury, when the fetus is sufficiently hypoxic and acidotic to impair cardiac output and CBF the potential for central nervous system injury from the related cerebral ischemia develops [3] [43] [44]. This is reflected in a severely compromised newborn with very low pH, persistently low Apgar scores, systemic hypotension, and multisystem organ involvement, likely to meet the criteria for moderate-severe encephalopathy and be a candidate for therapeutic hypothermia.

In addition to these well described adaptations to hypoxemia, the fetus also possesses adaptive responses to help it adjust to diminished brain perfusion, even without accompanying hypoxia. These reflexes derive from receptors centered in the fetal face and brain that are sensitive to stimulation by alterations in brain perfusion [38] [45] [46] [47].

To maintain CBF during a contraction, the fetal BP normally rises passively with the increasing IUP. This may be accompanied by increased sympathoadrenal tone as reflected by fetal activity and FHR accelerations that commonly occur with or in anticipation of contractions early in labor. When this proportional elevation of fetal BP is inadequate to maintain perfusion, it will be bolstered by robust adaptive reflexes to protect the fetal brain from direct ischemia, unrelated to impaired cardiac output.

In adults an increased ICP, with a reduction in CBF, induces the Cushing response, which raises the BP to support cerebral perfusion, but also affects breathing and heart rate [40] [41]. At least in experimental animals, cerebral ischemia (diminished cerebral perfusion) decreases the cerebral surface pH, but at least initially, not the systemic arterial pH [42]. A secondary fall in systemic pH may ensue over time as the response to cerebral hypoperfusion contributes to peripheral hypoperfusion by increased systemic resistance as the body optimizes flow to the ischemic brain [42].

This vagal response, unrelated to systemic hypoxia, is well developed in the term fetus via receptors within the brain [36] [41] [45]. In addition to the Cushing response, human newborns demonstrate various well-developed, innate responses to stimulation of the face and head, (trigeminal, diving reflexes) that raise blood pressure and induce vasoconstriction in the peripheral circulation. Like the Cushing response, these reflexes are maximal at birth and diminish progressively thereafter, as if they evolved specifically for the events of labor and delivery [46] [47].

## 4. Fetal Head Molding

The bones of the fetal calvarium are usually relatively thin, not fully calcified, and unfused. These features increase compliance and make them vulnerable to

changes in shape and position during labor according to constraints imposed by contractions, maternal pushing, and the bony and soft tissue elements of the birth canal [23] [48]. Normal molding is another means by which the fetus adapts to external pressures on its head. Extreme molding, especially in the context of ruptured membranes, however, is a risk factor for neurological injury, emphasizing the limited protection provided by this commonplace, adaptive mechanism [49].

A failure to mold, as well as large head size, has been associated with labor abnormalities, traumatic and ischemic intracranial injury and subsequent handicap including autism [12] [49]-[54].

## 5. Head Compression and Fetal Heart Rate Patterns

As noted, contractions (especially with pushing) elevate ICP and induce hemodynamic changes even in the normally oxygenated fetus [37] [55]. They may also diminish fetal $pO_2$ which, if sufficient, will trigger the peripheral chemoreceptors and cause FHR decelerations. Generally, even with mild hypoxemia, the rise in fetal arterial pressure is initially sufficient to sustain cerebral perfusion [55].

Adding maternal pushing efforts causes a more rapid onset, a greater peak pressure and a longer duration of maximum intrauterine and intracranial pressures. While facilitating molding and descent, these forces may impede CBF by further increasing the resistance to flow into the head. Distortion of brain vasculature can also reduce perfusion, contributing to the high frequencies of variable and early FHR decelerations in the 2$^{nd}$ stage [56].

Early decelerations are widely considered to be caused by fetal head compression [56] [57]. They are more common in occiput posterior presentations [58], and appear only after an IUP threshold has been exceeded [59]. They can sometimes be produced by manual compression of the fetal head through the uterine wall, by pressure on the anterior fontanel, or during external version [57] [60] [61] [62]. Importantly, in fetal sheep, decelerations are not observed despite increased ICP as long as systemic hypertension and CBF are maintained [31].

Early FHR decelerations appearing with contractions are associated with an increased resistance index and with absent or reversed diastolic blood flow in the fetal middle cerebral artery [33]. These decelerations in normal fetuses are most likely caused by a vagally mediated reflex response to a fall in cerebral perfusion, the result of cranial compression [33] [60] [63] [64]. The abrogation of the decelerations by the pre-administration of atropine confirms the reflex vagal nature of the response [65]. Thus, decelerations in response to head compression appear to represent an early, preemptive response to any threat to CBF and not some response to tactile stimulation of the scalp or long-standing or severely compromised CBF [29].

Classical teaching holds that fetal head compression causes early decelerations, while umbilical cord compression results in variable decelerations [56]. These distinctions are probably moot because the morphology of decelerations

and the severity of fetal head compression and the dynamics of fetal ICP change over time, especially during the 2nd stage of labor [37] when both variable and early decelerations most often result from head compression, not cord compression [30] [62] [63] [64].

Sometimes there may be delayed return of the deceleration to the baseline, or the secondary appearance of late decelerations after the variable deceleration has recovered. Walker *et al.* reported that pressure over the inferior aspect of the uterus often produced decelerations with slow resolution [57]. Similarly, Moccsary, *et al.* found delayed recovery of the deceleration, when ICP was experimentally increased above that produced by normal contractions [27]. In the sheep fetus manual head compression is associated with a significant reduction in carotid blood flow that continues beyond the deceleration, accompanied by profound suppression of fetal EEG activity and reduced brain metabolism [27] [28] [29] [30].

These findings suggest it may not be the pressure on the head per se, but the impact on CBF that produces the deceleration. They further suggest that despite rapid return to normal IUP after a contraction, more time may be necessary to restore normal fetal CBF and FHR, and that abnormal recovery of the FHR may reflect a persistently elevated ICP or more severe cerebral ischemia separate from or in addition to any hypoxemia [27] [63].

Excessive uterine contractility would be expected to compromise fetal oxygen availability as well as recovery from the effects of head compression. Indeed, Bakker, *et al.* showed that excessive uterine activity was more often accompanied by variable than late decelerations [66]. The fetus is generally tolerant of substantial amounts of uterine activity for many hours, especially in the first stage of labor [67]. When contractions are excessive, especially when combined with maternal pushing and limited fetal descent, the deleterious effects of hypoxia and of head compression may not permit sufficient recovery from the previous deceleration before the next contraction starts. Such deterioration is expressed on the FHR tracing in the form of recurrent variable decelerations with erratic changes during recovery of the deceleration, often with significant overshoot and a rising baseline. The variable decelerations may be followed immediately by hypoxemia-induced late decelerations [68] [69] [70]. Consistent with the notion of combined effects (head compression and hypoxia), an associated progressive reduction in fetal pH has been demonstrated. Such patterns have been described as *subacute hypoxia* [71] [72] with the recommendation that they be treated by the immediate reduction of pushing and uterine activity to allow the fetus to recover.

The understanding that intrapartum HI injury may occur without systemic asphyxia and neonatal depression has significant therapeutic implications for the neonate and the use of therapeutic hypothermia [73]. Most clinical trials of hypothermia involve infants with moderate to severe NE in whom there is both severe acidosis and neonatal depression. Although there have been clinical trials

of hypothermia in infants with mild encephalopathy, there is yet no consistent recommendation on its use in those circumstances. It is relevant to point out that the experimental model of neurological injury used to test the value of cooling involved not systemic asphyxia, but a single, mechanical interruption of the cerebral circulation, where the exact time of the occlusion was known [74].

## 6. Fetal Head Compression and Neurological Injury

The cause-effect relationship of mechanical factors to fetal neurological injury pervades almost two centuries of literature, especially in association with difficult or instrumental delivery (**Table 2**) [1] [2] [75] [76] [77] [78] [79]. Modern reviews of intrapartum fetal neurological injury or the effects of pushing, however, do not generally mention head compression as a possible etiology of injury [80] [81]. The finding of MRI evidence of recent ischemia in the face of near-normal umbilical pH cannot reasonably be explained by severe hypoxic acidemia during labor [72] [82] [83] while normal growth, normal fetal activity, normal amniotic fluid volume and a normal FHR tracing on admission would seem to preclude an earlier injury [4]. The diagnosis of "presumed perinatal stroke" refers to the discovery of cerebral ischemic lesion(s) in the perinatal period, but which may have become symptomatic only weeks or months later [84] [85]. Excessive uterine activity, prolonged pushing in the 2$^{nd}$ stage and instrumental delivery (especially in the nullipara) are considered risk factors for HIE and subsequent neurological handicap including CP and stroke [15] [85] [86] [87] [88]. As expected, given the slow evolution of these changes, most of the cerebral lesions involve the cortex and white matter, (a prolonged, partial, HIE) and less commonly the basal ganglia (an acute, near-total HIE).

The ischemic effects of pressure on the fetal head will likely be augmented when superimposed on an already asphyxiated fetus. If, during systemic asphyxia,

**Table 2.** Potential consequences of excessive fetal head compression.

| Condition | Reference |
|---|---|
| Extreme molding | [23] |
| Skull fractures | [98] |
| Distortion of brain | [23] |
| Subdural hemorrhage | [99] |
| Retinal hemorrhage | [100] [101] |
| Tentorial tears | [101] |
| Venous hemorrhage | [103] |
| FHR decelerations | [103] [104] |
| Impaired cerebral blood flow | [26] [30] |
| Encephalopathy | [8] [20] [86] |
| Contusions | [103] |

the fetal BP is struggling or failing to provide sufficient cerebral perfusion pressure, then any factor that increases ICP (such as excessive uterine activity, maternal pushing) would aggravate the already impaired CBF. Instituting forceful pushing to effect delivery in the face of fetal bradycardia or decelerations with severe tachycardia, for example, may be counterproductive; reducing contractions and curtailing pushing may be far more beneficial. As mentioned above, prevention would be a better strategy. Thus, assessing the feasibility of safe vaginal delivery, maintaining a controlled rhythm and frequency of uterine contractions, not to exceed 4 contractions in 10 minutes [89], observing the response of the FHR pattern to the mother's pushing efforts, and discouraging pushing in those situations in which the FHR pattern does not return to its previously normal rate and variability will avoid both urgent intervention and fetal injury [89] [90] [91] [92].

### 6.1. Fetal Heart Rate Patterns of Injury

Considerable data attest to the association of various mechanical factors (**Table 1**) with adverse FHR features and with subsequent neurological impairment in the newborn and older child [1] [3] [10] [78] [90]. Schifrin and Ater identified a unique FHR pattern in previously normal fetuses that reliably anticipated fetal neurological injury irrespective of acid-base status at delivery [92] [93]. This "conversion pattern" was commonly seen as part of the evolution of the subacute hypoxia pattern described above, with frequent contractions and compulsive maternal pushing. Acidemia was present in less than 30% of these cases.

### 6.2. The Head Compression Controversy

There is considerable controversy about the role of head compression as a cause of FHR decelerations and of adverse perinatal outcome, as one would expect when any new approach is introduced that challenges embedded dogma or recalls neglected science. Some doubt its existence [94]; others reject its pertinence to clinical outcomes, except perhaps in the most extreme circumstances [38]; others focus on its medico-legal implications [95] [96], and some advise caution before rejecting the notion [97].

In an on-going debate over the role of mechanical forces acting on the fetal head, Lear *et al.*, based upon animal experimentation, suggested that head compression in the human fetus has been mistakenly overemphasized as a common mediator of intrapartum decelerations in late 1st and 2nd stage labor [38]. Further, they opined, such attention distracts caregivers from focusing on the depth, duration, and frequency of decelerations that represent primarily hypoxemic debt, not one related primarily to ischemia. They agree with the assertions made above that the fetus is capable of robust adaptations to deal with increased ICP, that the isolated periods of head compression as may occur during a normal, spontaneous delivery are highly unlikely to lead to significant injury, but that

fetal head compression is, "not intrinsically benign." In their view, its only potential role in significant injury is when the fetus is already severely compromised by preceding severe hypoxemia.

Some skepticism is understandable, because there are obvious limitations of extrapolating findings in laboratory animals to the human fetus in labor, and experimental studies in the human, understandably, are few. That said, it is abundantly clear that not all acute NE results from severe fetal hypoxia and acidosis or results in a severely depressed newborn. Further, there seems little doubt that excessive cranial compression from forceps, obstructed labor, or presumably from prolonged pushing in the 2nd stage of labor without fetal descent, for example, can lead to intrapartum fetal brain damage [15] [36] [37]. But to employ that knowledge gainfully in the context of clinical decision-making urgently requires that we better understand fetal intrapartum ICP dynamics and their clinical concomitants.

## 7. Implications

We must further our understanding of how the factors affecting head compression, including excessive uterine activity, dysfunctional labor progress, fetal head molding and various obstetric procedures, including pushing strategy, influence outcomes. There is no evidence that FHR patterns will fail to reflect impaired CBF or hypoxia. To better appreciate the impact of contractions on cerebral blood flow, FHR patterns must be used as an instrument of preventive care rather than to rescue a fetus who may already be compromised. Thus, early and variable decelerations should not be considered innocuous when they are unassociated with fetal acidemia. They are likely tolerable as long as there is prompt return to the previously normal baseline rate and variability. Abnormalities of deceleration recovery (prolonged, overshoot, rising heart rate, altered variability) are often markers of deterioration. Excessive uterine activity should be avoided, irrespective of the FHR pattern. Moderating expulsive efforts, even intermittently, may assist in the recovery of the fetus to its previously normal pattern and should be included in any list of techniques of intrauterine resuscitation. In this respect, current NICE guidelines from England recommend a limit of four contractions in ten minutes [89].

## 8. Conclusion: Fetal Neurological Injury—A Revised Perspective

Prevention of injury must begin with understanding its mechanisms and clinical correlates. The traditional view that intrapartum brain injury is exclusively the consequence of severe global asphyxia and acidosis and severe neonatal depression informs current protocols for interpreting FHR patterns and guiding intrapartum care, but is not consistent with the available clinical or experimental data. Such a narrow perspective of adverse outcomes underestimates both the true incidence of immediate and long-term perinatal harm, its timing, mechanism

and preventability, as well as the potential role of enlightened obstetrical and neonatal care in preventing injury [98]-[103]. Recommendations that focus on protecting the fetal brain from hypoxia and excessive compression (minimizing excessive uterine activity, moderating maternal pushing as a strategy of intrauterine resuscitation, using the fetal response to decelerations to moderate the administration of oxytocin and pushing) should prevail whether the primary mechanism is ischemia from head compression or hypoxia from impaired utero-placental exchange. There is compelling evidence that the fetal head is exposed sometimes to extremely high external pressures during labor and delivery. It is also clear that these forces are transmitted into the cranial cavity, and that under normal circumstances there are physiologic countermeasures that protect brain perfusion. Like any such protective mechanisms, these can be overcome if compression of the head is well beyond normal, leading to the potential for impeding brain blood flow through intracranial pressures that exceed brain perfusion pressures or distortion of intracranial vasculature. Although the data linking excessive head compression to brain injury are strongly inferential at this time, cranial compression should be considered in the differential diagnosis of causes of ischemic encephalopathy. Further research in this dimension is needed desperately.

## Author contributions

All authors have accepted responsibility for the entire content of this manuscript and approved its submission.

## Ethical Approval

As a review article that discloses no patient information this project was exempt from review by the local Institutional Review Board.

## Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

## References

[1] Kelly, J.V. (1963) Compression of the Fetal Brain. *American Journal of Obstetrics & Gynecology*, **85**, 687-694. https://doi.org/10.1016/0002-9378(63)90317-X

[2] Schifrin, B.S., Deymier, P. and Cohen, W.R. (2014) Cranial Compression Ischemic Encephalopathy: Fetal Neurological Injury Related to the Mechanical Forces of Labor and Delivery. In: Zhang, L. and Longo, L., Eds., *Stress and Developmental Programming of Health and Disease*: *Beyond Phenomenology*, Nova Science Publishers, Inc., New York, 651-688.

[3] Turner, J.M., Mitchell, M.D. and Kumar, S. (2020) The Physiology of Intrapartum Fetal Compromise at Term. *American Journal of Obstetrics & Gynecology*, **222**, 17-26. https://doi.org/10.1016/j.ajog.2019.07.032

[4] Clark, S.L. (2022) Category II Intrapartum Fetal Heart Rate Patterns Unassociated with Recognized Sentinel Events. *Obstetrics & Gynecology*, **139**, 1003-1008.

https://doi.org/10.1097/AOG.0000000000004801

[5] Jonsson, M., Agren, J., Norden-Lindeberg, S., Ohlin, A. and Hanson, U. (2014) Neonatal Encephalopathy and the Association to Asphyxia in Labor. *American Journal of Obstetrics & Gynecology*, **211**, 667.e1-8. https://doi.org/10.1016/j.ajog.2014.06.027

[6] van Handel, M., de Sonneville, L., de Vries, L.S., Jongmans, M.J. and Swaab, H. (2012) Specific Memory Impairment Following Neonatal Encephalopathy in Term-Born Children. *Developmental Neuropsychology*, **37**, 30-50. https://doi.org/10.1080/87565641.2011.581320

[7] Chalak, L.F. (2018) Best Practice Guidelines on Management of Mild Neonatal Encephalopathy: Is It Really Mild? *Early Human Development*, **120**, 74. https://doi.org/10.1016/j.earlhumdev.2018.02.005

[8] Cahill, A.G., Mathur, A.M., Smyser, C.D., McKinstry, R.C., Roehl, K.A., Lopez, J.D., *et al.* (2017) Neurologic Injury in Acidemic Term Infants. *American Journal of Perinatology*, **34**, 668-675. https://doi.org/10.1055/s-0036-1597135

[9] Chalak, L., Ferriero, D.M., Gressens, P., Molloy, E. and Bearer, C. (2019) A 20 Years Conundrum of Neonatal Encephalopathy and Hypoxic Ischemic Encephalopathy: Are We Closer to a Consensus Guideline? *Pediatric Research*, **86**, 548-549. https://doi.org/10.1038/s41390-019-0547-9

[10] Chaturvedi, A., Chaturvedi, A., Stanescu, A.L., Blickman, J.G. and Meyers, S.P. (2018) Mechanical Birth-Related Trauma to the Neonate: An Imaging Perspective. *Insights Imaging*, **9**, 103-118. https://doi.org/10.1007/s13244-017-0586-x

[11] Martinez-Biarge, M., Diez-Sebastian, J., Wusthoff, C.J., Mercuri, E. and Cowan, F.M. (2013) Antepartum and Intrapartum Factors Preceding Neonatal Hypoxic-Ischemic Encephalopathy. *Pediatrics*, **132**, e952-e959. https://doi.org/10.1542/peds.2013-0511

[12] Jensen, A. and Holmer, B. (2018) White Matter Damage in 4,725 Term-Born Infants Is Determined by Head Circumference at Birth: The Missing Link. *Obstetrics and Gynecology International*, **2018**, Article ID: 2120835. https://doi.org/10.1155/2018/2120835

[13] Lee, C.C., Lin, J.J., Lin, K.L., Lim, W.H., Hsu, K.H., Hsu, J.F., *et al.* (2017) Clinical Manifestations, Outcomes, and Etiologies of Perinatal Stroke in Taiwan Region: Comparisons between Ischemic, and Hemorrhagic Stroke Based on 10-Year Experience in a Single Institute. *Pediatrics & Neonatology*, **58**, 270-277. https://doi.org/10.1016/j.pedneo.2016.07.005

[14] Friedman, E.A. and Cohen, W.R. (2023) Dysfunctional Labor and Delivery: Adverse Effects on Offspring. *American Journal of Obstetrics & Gynecology*, **228**, S1104-S1109. https://doi.org/10.1016/j.ajog.2022.10.011

[15] Grobman, W.A., Bailit, J., Lai, Y., Reddy, U.M., Wapner, R.J., Varner, M.W., *et al.* (2016) Association of the Duration of Active Pushing with Obstetric Outcomes. *Obstetrics & Gynecology*, **127**, 667-673. https://doi.org/10.1097/AOG.0000000000001354

[16] Sugarman, R.G., Hadjiev, A. and Schifrin, B.S. (1976) Intracranial Pressure and Fetal Heart Rate Patterns in a Case of Hydrocephaly. *The European Journal of Obstetrics & Gynecology and Reproductive Biology*, **6**, 249-255. https://doi.org/10.1016/0028-2243(76)90067-8

[17] Kim, M.O., Adji, A., O'Rourke, M.F., Avolio, A.P., Smielewski, P., Pickard, J.D., *et al.* (2015) Principles of Cerebral Hemodynamics When Intracranial Pressure Is Raised: Lessons from the Peripheral Circulation. *Journal of Hypertension*, **33**, 1233-1241.

https://doi.org/10.1097/HJH.0000000000000539

[18] Mokri, B. (2001) The Monro-Kellie Hypothesis: Applications in CSF Volume Depletion. *Neurology*, **56**, 1746-1748. https://doi.org/10.1212/WNL.56.12.1746

[19] Wilson, M.H. (2016) Monro-Kellie 2.0: The Dynamic Vascular and Venous Pathophysiological Components of Intracranial Pressure. *Journal of Cerebral Blood Flow & Metabolism*, **36**, 1338-1350. https://doi.org/10.1177/0271678X16648711

[20] Nelson, M.D., Tavare, C.J., Petrus, L., Kim, P. and Gilles, F.H. (2003) Changes in the Size of the Lateral Ventricles in the Normal-Term Newborn Following Vaginal Delivery. *Pediatric Radiology*, **33**, 831-835. https://doi.org/10.1007/s00247-003-0967-9

[21] Brouwer, M.J., de Vries, L.S., Groenendaal, F., Koopman, C., Pistorius, L.R., Mulder, E.J., *et al.* (2012) New Reference Values for the Neonatal Cerebral Ventricles. *Radiology*, **262**, 224-233. https://doi.org/10.1148/radiol.11110334

[22] Winchester, P., Brill, P.W., Cooper, R., Krauss, A.N. and Peterson, H.D. (1986) Prevalence of "Compressed" and Asymmetric Lateral Ventricles in Healthy Full-Term Neonates: Sonographic Study. *AJR American Journal of Roentgenology*, **146**, 471-475. https://doi.org/10.2214/ajr.146.3.471

[23] Ami, O., Maran, J.C., Gabor, P., Whitacre, E.B., Musset, D., Dubray, C., *et al.* (2019) Three-Dimensional Magnetic Resonance Imaging of Fetal Head Molding and Brain Shape Changes during the Second Stage of Labor. *PLOS ONE*, **14**, e0215721. https://doi.org/10.1371/journal.pone.0215721

[24] Lindgren, L. (1977) The Influence of Pressure upon the Fetal Head during Labour. *Acta Obstetricia et Gynecologica Scandinavica*, **56**, 303-309. https://doi.org/10.3109/00016347709154983

[25] Schwarcz, R., Strada-Saenz, G., Althabe, O., Fernandez-Funes, J. and Caldeyro-Barcia, R. (1969) Pressure Exerted by Uterine Contractions on the Head of the Human Fetus during Labor. In: *Perinatal Factors Affecting Human Development*, Pan American Health Organization, Washington DC, 115-126.

[26] Dupuis, O. and Simon, A. (2008) Fetal Monitoring during the Active Second Stage of Labor. *Journal de gynécologie, obstétrique et biologie de la reproduction* (*Paris*), **37**, S93-S100. (In French)

[27] Mocsary, P., Gaal, J., Komaromy, B., Mihaly, G., Pohanka, O. and Suranyi, S. (1970) Relationship between Fetal Intracranial Pressure and Fetal Heart Rate during Labor. *American Journal of Obstetrics & Gynecology*, **106**, 407-411. https://doi.org/10.1016/0002-9378(70)90367-4

[28] Mann, L.I., Carmichael, A. and Duchin, S. (1972) The Effect of Head Compression on FHR, Brain Metabolism and Function. *Obstetrics & Gynecology*, **39**, 721-726.

[29] Paul, W.M., Quilligan, E.J. and Maclachlan, T. (1964) Cardiovascular Phenomenon Associated with Fetal Head Compression. *American Journal of Obstetrics & Gynecology*, **90**, 824-826. https://doi.org/10.1016/0002-9378(64)90949-4

[30] O'Brien, W.F., Davis, S.E., Grissom, M.P., Eng, R.R. and Golden, S.M. (1984) Effect of Cephalic Pressure on Fetal Cerebral Blood Flow. *American Journal of Perinatology*, **1**, 223-226. https://doi.org/10.1055/s-2007-1000009

[31] Harris, A.P., Koehler, R.C., Gleason, C.A., Jones, M.D. and Traystman, R.J. (1989) Cerebral and Peripheral Circulatory Responses to Intracranial Hypertension in Fetal Sheep. *Circulation Research*, **64**, 991-1000. https://doi.org/10.1161/01.RES.64.5.991

[32] Ogilvy, C.S. and DuBois, A.B. (1987) Effect of Increased Intracranial Pressure on Blood Pressure, Heart Rate, Respiration and Catecholamine Levels in Neonatal and Adult Rabbits. *Biology of the Neonate*, **52**, 327-336.

https://doi.org/10.1159/000242728

[33] Ueno, N. (1992) Studies on Fetal Middle Cerebral Artery Blood Flow Velocity Waveforms in the Intrapartum Period. *Nihon Sanka Fujinka Gakkai Zasshi*, **44**, 97-104. (In Japanese)

[34] Newton, T.H. and Gooding, C.A. (1975) Compression of Superior Sagittal Sinus by Neonatal Calvarial Molding. *Radiology*, **115**, 635-640. https://doi.org/10.1148/15.3.635

[35] Cowan, F., Rutherford, M., Groenendaal, F., Eken, P., Mercuri, E., Bydder, G.M., *et al.* (2003) Origin and Timing of Brain Lesions in Term Infants with Neonatal Encephalopathy. *The Lancet*, **36**, 736-742. https://doi.org/10.1016/S0140-6736(03)12658-X

[36] Wilson, P.C., Philpott, R.H., Spies, S., Ahmed, Y. and Kadichza, M. (1979) The Effect of Fetal Head Compression and Fetal Acidaemia during Labour on Human Fetal Cerebral Function as Measured by the Fetal Electroencephalogram. *British Journal of Obstetrics and Gynaecology*, **86**, 269-277. https://doi.org/10.1111/j.1471-0528.1979.tb11254.x

[37] Aldrich, C.J., D'Antona, D., Spencer, J.A., Wyatt, J.S., Peebles, D.M., Delpy, D.T., *et al.* (1995) The Effect of Maternal Pushing on Fetal Cerebral Oxygenation and Blood Volume during The Second Stage of Labour. *British Journal of Obstetrics and Gynaecology*, **102**, 448-453. https://doi.org/10.1111/j.1471-0528.1995.tb11316.x

[38] Lear, C.A., Westgate, J.A., Bennet, L., Ugwumadu, A., Stone, P.R., Tournier, A., *et al.* (2023) Fetal Defenses against Intrapartum Head Compression—Implications for Intrapartum Decelerations and Hypoxic-Ischemic Injury. *American Journal of Obstetrics & Gynecology*, **228**, S1117-1128. https://doi.org/10.1016/j.ajog.2021.11.1352

[39] Turner, J.M., Mitchell, M.D. and Kumar, S.S. (2020) The Physiology of Intrapartum Fetal Compromise at Term. *American Journal of Obstetrics & Gynecology*, **222**, 17-26. https://doi.org/10.1016/j.ajog.2019.07.032

[40] Giussani, D.A. (2016) The Fetal Brain Sparing Response to Hypoxia: Physiological Mechanisms. *The Journal of Physiology*, **594**, 1215-1230. https://doi.org/10.1113/JP271099

[41] Fodstad, H., Kelly, P.J. and Buchfelder, M. (2006) History of the Cushing Reflex. *Neurosurgery*, **59**, 1132-1137. https://doi.org/10.1227/01.NEU.0000245582.08532.7C

[42] Berman, I.R. and Rogers, L.A. (1970) Cerebral Acidosis Following Increased Intracranial Pressure. *Surgery, Gynecology and Obstetrics*, **130**, 483-486.

[43] Perlman, J.M. (1997) Intrapartum Hypoxic-Ischemic Cerebral Injury and Subsequent Cerebral Palsy: Medicolegal Issues. *Pediatrics*, **99**, 851-859. https://doi.org/10.1542/peds.99.6.851

[44] Sarnat, H.B. and Sarnat, M.S. (1976) Neonatal Encephalopathy Following Fetal Distress. A Clinical and Electroencephalographic Study. *Archives of Neurology*, **33**, 696-705. https://doi.org/10.1001/archneur.1976.00500100030012

[45] Marina, N., Christie, I.N., Korsak, A., Doronin, M., Brazhe, A., Hosford, P.S., *et al.* (2020) Astrocytes Monitor Cerebral Perfusion and Control Systemic Circulation to Maintain Brain Blood Flow. *Nature Communications*, **11**, 131-140. https://doi.org/10.1038/s41467-019-13956-y

[46] Panneton, W.M. (2013) The Mammalian Diving Response: An Enigmatic Reflex to Preserve Life? *Physiology* (*Bethesda*), **28**, 284-297. https://doi.org/10.1152/physiol.00020.2013

[47] Pedroso, F.S., Riesgo, R.S., Gatiboni, T. and Rotta, N.T. (2012) The Diving Reflex in

Healthy Infants in the First Year of Life. *Journal of Child Neurology*, **27**, 168-171. https://doi.org/10.1177/0883073811415269

[48] Churchill, J.A., Willerman, L., Grisell, J. and Ayers, M.A. (1968) Effect of Head Position at Birth on WISC Verbal and Performance IQ. *Psychological Reports*, **23**, 495-498. https://doi.org/10.2466/pr0.1968.23.2.495

[49] Caldeyro-Barcia, R. (1974) Adverse Perinatal Effects of Early Amniotomy during Labor. In: Gluck, L., Ed., *Modern Perinatal Medicine*, Year Book Medical Publishers, Chicago, 431-449.

[50] Carlan, S.J., Wyble, L., Lense, J., Mastrogiannis, D.S. and Parsons, M.T. (1991) Fetal Head Molding. Diagnosis by Ultrasound and a Review of the Literature. *Journal of Perinatology*, **11**, 105-111.

[51] de la Fuente, A.A. (1991) Locking and Reverse Molding of the Fetal Skull. *Pediatric Pathology*, **11**, 271-280. https://doi.org/10.3109/15513819109064764

[52] Passerini, K., Kurmanavicius, J., Burkhardt, T. and Balsyte, D. (2020) Influence of Newborn Head Circumference and Birth Weight on the Delivery Mode of Primipara: What Is More Important? *Journal of Perinatal Medicine*, **48**, 681-686. https://doi.org/10.1515/jpm-2019-0410

[53] Lipschuetz, M., Cohen, S.M., Ein-Mor, E., Sapir, H., Hochner-Celnikier, D., Porat, S., *et al.* (2015) A Large Head Circumference Is More Strongly Associated with Unplanned Cesarean or Instrumental Delivery and Neonatal Complications than High Birthweight. *American Journal of Obstetrics & Gynecology*, **213**, 833e1-e12. https://doi.org/10.1016/j.ajog.2015.07.045

[54] Elvander, C., Hogberg, U. and Ekeus, C. (2012) The Influence of Fetal Head Circumference on Labor Outcome: A Population-Based Register Study. *Acta Obstetricia et Gynecologica Scandinavica*, **91**, 470-475. https://doi.org/10.1111/j.1600-0412.2012.01358.x

[55] Llanos, A.J., Court, D.J., Block, B.S., Germain, A.M. and Parer, J.T. (1986) Fetal Cardiorespiratory Changes during Spontaneous Prelabor Uterine Contractions in Sheep. *American Journal of Obstetrics & Gynecology*, **155**, 893-897. https://doi.org/10.1016/S0002-9378(86)80046-1

[56] Hon, E.H. and Quilligan, E.J. (1967) The Classification of Fetal Heart Rate. II. A Revised Working Classification. *Connecticut Medicine*, **31**, 779-784.

[57] Walker, D., Grimwade, J. and Wood, C. (1973) The Effects of Pressure on Fetal Heart Rate. *Obstetrics & Gynecology*, **41**, 351-354.

[58] Ingemarsson, E., Ingemarsson, I., Solum, T. and Westgren, M. (1980) Influence of Occiput Posterior Position on the Fetal Heart Rate Pattern. *Obstetrics & Gynecology*, **55**, 301-304. https://doi.org/10.1097/00006250-198003000-00006

[59] Chung, F. and Hon, E.H. (1959) The Electronic Evaluation of Fetal Heart Rate. I. With Pressure on the Fetal Skull. *Obstetrics & Gynecology*, **13**, 633-640.

[60] Lear, C.A., Wassink, G., Westgate, J.A., Nijhuis, J.G., Ugwumadu, A., Galinsky, R., *et al.* (2018) The Peripheral Chemoreflex: Indefatigable Guardian of Fetal Physiological Adaptation to Labour. *The Journal of Physiology*, **596**, 5611-5623. https://doi.org/10.1113/JP274937

[61] Lear, C.A., Galinsky, R., Wassink, G., Yamaguchi, K., Davidson, J.O., Westgate, J.A., *et al.* (2016) The Myths and Physiology Surrounding Intrapartum Decelerations: The Critical Role of the Peripheral Chemoreflex. *The Journal of Physiology*, **594**, 4711-4725. https://doi.org/10.1113/JP271205

[62] Leung, T.Y., Sahota, D.S., Fok, W.Y., Chan, L.W. and Lau, T.K. (2004) External Cephalic Version Induced Fetal Cerebral and Umbilical Blood Flow Changes Are

Related to the Amount of Pressure Exerted. *BJOG*, **111**, 430-435. https://doi.org/10.1111/j.1471-0528.2004.00127.x

[63] Sholapurkar, S.L. (2017) Critical Imperative for the Reform of British Interpretation of Fetal Heart Rate Decelerations: Analysis of FIGO and NICE Guidelines, Post-Truth Foundations, Cognitive Fallacies, Myths and Occam's Razor. *Journal of Clinical Medicine Research*, **9**, 2532-2565. https://doi.org/10.14740/jocmr2877e

[64] Ball, R.H. and Parer, J.T. (1992) The Physiologic Mechanisms of Variable Decelerations. *American Journal of Obstetrics & Gynecology*, **166**, 1683-1689. https://doi.org/10.1016/0002-9378(92)91557-Q

[65] Mendez-Bauer, C., Poseiro, J.J., Arellano-Hernandez, G., Zambrana, M.A. and Caldeyro-Barcia, R. (1963) Effects of Atropine on the Heart Rate of the Human Fetus during Labor. *American Journal of Obstetrics & Gynecology*, **85**, 1033-1053. https://doi.org/10.1016/S0002-9378(16)35639-3

[66] Bakker, P.C. and van Geijn, H.P. (2008) Uterine Activity: Implications for the Condition of the Fetus. *Journal of Perinatal Medicine*, **36**, 30-37. https://doi.org/10.1515/JPM.2008.003

[67] McNamara, H. and Johnson, N. (1995) The Effect of Uterine Contractions on Fetal Oxygen Saturation. *British Journal of Obstetrics and Gynaecology*, **102**, 644-647. https://doi.org/10.1111/j.1471-0528.1995.tb11403.x

[68] Stewart, R.D., Bleich, A.T., Lo, J.Y., Alexander, J.M., McIntire, D.D. and Leveno, K.J. (2012) Defining Uterine Tachysystole: How Much Is Too Much? *American Journal of Obstetrics & Gynecology*, **207**, 290e1-6. https://doi.org/10.1016/j.ajog.2012.07.032

[69] Krebs, H.B., Petres, R.E. and Dunn, L.J. (1983) Intrapartum Fetal Heart Rate Monitoring. VIII. Atypical Variable Decelerations. *American Journal of Obstetrics & Gynecology*, **145**, 297-305. https://doi.org/10.1016/0002-9378(83)90714-7

[70] Ingemarsson, E., Ingemarsson, I. and Westgren, M. (1981) Combined Decelerations—Clinical Significance and Relation to Uterine Activity. *Obstetrics & Gynecology*, **58**, 35-39.

[71] Jia, Y.-J., Ghi, T., Pereira, S., Perez-Bonfils, A.G. and Chandraharan, E. (2023) Pathophysiological Interpretation of Fetal Heart Rate Tracings in Clinical Practice. *American Journal of Obstetrics & Gynecology*, **228**, 622-644. https://doi.org/10.1016/j.ajog.2022.05.023

[72] Pinas, A. and Chandraharan, E. (2016) Continuous Cardiotocography during Labour: Analysis, Classification and Management. *Best Practice & Research Clinical Obstetrics & Gynaecology*, **30**, 33-47. https://doi.org/10.1016/j.bpobgyn.2015.03.022

[73] Hermansen, M.C. (2003) The Acidosis Paradox: Asphyxial Brain Injury without Coincident Acidemia. *Developmental Medicine & Child Neurology*, **45**, 353-356. https://doi.org/10.1111/j.1469-8749.2003.tb00408.x

[74] Gunn, A.J., Battin, M., Gluckman, P.D., Gunn, T.R. and Bennet, L. (2005) Therapeutic Hypothermia: From Lab to NICU. *Journal of Perinatal Medicine*, **33**, 340-346. https://doi.org/10.1515/JPM.2005.061

[75] Amiel-Tison, C., Sureau, C. and Shnider, S.M. (1988) Cerebral Handicap in Full-Term Neonates Related to the Mechanical Forces of Labour. *Baillière's Clinical Obstetrics and Gynaecology*, **2**, 145-165. https://doi.org/10.1016/S0950-3552(88)80069-5

[76] Schwarcz, R., Strada-Saenz, G., Althabe, O., Fernandes-Funes, J., Alvares, L. and Caldyro-Barcia, R. (1970) Compression Received by the Head of the Human Fetus

during Labor. *Proceedings of a Conference on the Etiology of Mental Retardation*, Omaha, 13 October 1968, 133-143.

[77] Friedman, E.A., Sachtleben, M.R. and Bresky, P.A. (1977) Dysfunctional Labor XII. Long-Term Effects on Infant. *American Journal of Obstetrics & Gynecology*, **127**, 779-783. https://doi.org/10.1016/0002-9378(77)90257-5

[78] Vlasyuk, V. (2019) Birth Trauma and Perinatal Brain Damage. Springer International Publishing AG, Cham, 271-283. https://doi.org/10.1007/978-3-319-93441-9

[79] Little, W.J. (1966) On the Influence of Abnormal Parturition, Difficult Labours, Premature Birth, and Asphyxia Neonatorum, on the Mental and Physical Condition of the Child, Especially in Relation to Deformities. *Clinical Orthopaedics and Related Research*, **46**, 7-22. https://doi.org/10.1097/00003086-196600460-00002

[80] Rennie, J.M., Hagmann, C.F. and Robertson, N.J. (2007) Outcome after Intrapartum Hypoxic Ischaemia at Term. *Seminars in Fetal and Neonatal Medicine*, **12**, 398-407. https://doi.org/10.1016/j.siny.2007.07.006

[81] Jackson, M., Holmgren, C.M., Esplin, M.S., Henry, E. and Varner, M.W. (2011) Frequency of Fetal Heart Rate Categories and Short-Term Neonatal Outcome. *Obstetrics & Gynecology*, **118**, 803-808. https://doi.org/10.1097/AOG.0b013e31822f1b50

[82] Looney, C.B., Smith, J.K., Merck, L.H., Wolfe, H.M., Chescheir, N.C., Hamer, R.M., *et al.* (2007) Intracranial Hemorrhage in Asymptomatic Neonates: Prevalence on MR Images and Relationship to Obstetric and Neonatal Risk Factors. *Radiology*, **242**, 535-541. https://doi.org/10.1148/radiol.2422060133

[83] Rooks, V.J., Eaton, J.P., Ruess, L., Petermann, G.W., Keck-Wherley, J. and Pedersen, R.C. (2008) Prevalence and Evolution of Intracranial Hemorrhage in Asymptomatic Term Infants. *AJNR American Journal of Neuroradiology*, **29**, 1082-1089. https://doi.org/10.3174/ajnr.A1004

[84] Kirton, A., Deveber, G., Pontigon, A.M., Macgregor, D. and Shroff, M. (2008) Presumed Perinatal Ischemic Stroke: Vascular Classification Predicts Outcomes. *Annals of Neurology*, **63**, 436-443. https://doi.org/10.1002/ana.21334

[85] Martinez-Biarge, M., Cheong, J.L., Diez-Sebastian, J., Mercuri, E., Dubowitz, L.M. and Cowan, F.M. (2016) Risk Factors for Neonatal Arterial Ischemic Stroke: The Importance of the Intrapartum Period. *The Journal of Pediatrics*, **173**, 62-68. https://doi.org/10.1016/j.jpeds.2016.02.064

[86] Hayes, B.C., McGarvey, C., Mulvany, S., Kennedy, J., Geary, M.P., Matthews, T.G., *et al.* (2013) A Case-Control Study of Hypoxic-Ischemic Encephalopathy in Newborn Infants at > 36 Weeks Gestation. *American Journal of Obstetrics & Gynecology*, **209**, 29.e1-29. https://doi.org/10.1016/j.ajog.2013.03.023

[87] Reynolds, A.J., Murray, M.L., Geary, M.P., Ater, S.B. and Hayes, B.C. (2022) Uterine Activity in Labor and the Risk of Neonatal Encephalopathy: A Case Control Study. *The European Journal of Obstetrics & Gynecology and Reproductive Biology*, **274**, 73-79. https://doi.org/10.1016/j.ejogrb.2022.05.011

[88] Thorngren-Jerneck, K. and Herbst, A. (2006) Perinatal Factors Associated with Cerebral Palsy and Children Born in Sweden. *Obstetrics & Gynecology*, **108**, 1499-1505. https://doi.org/10.1097/01.AOG.0000247174.27979.6b

[89] O'Heney, J., McAllister, S., Maresh, M. and Blott, M. (2022) Fetal Monitoring in Labour: Summary and Update of NICE Guidance. *BMJ*, **379**, o2854. https://doi.org/10.1136/bmj.o2854

[90] Wassink, G., Bennet, L., Davidson, J.O., Westgate, J.A. and Gunn, A.J. (2013)

Pre-Existing Hypoxia Is Associated with Greater EEG Suppression and Early Onset of Evolving Seizure Activity during Brief Repeated Asphyxia in Near-Term Fetal Sheep. *PLOS ONE*, **8**, e73895. https://doi.org/10.1371/journal.pone.0073895

[91] Le Ray, C., Rozenberg, P., Kayem, G., Harvey, T., Sibiude, J., Doret, M., *et al.* (2022) Alternative to Intensive Management of the Active Phase of the Second Stage of Labor: A Multicenter Randomized Trial (Phase Active du Second STade Trial) among Nulliparous Women with an Epidural. *American Journal of Obstetrics & Gynecology*, **227**, 639e1-e15. https://doi.org/10.1016/j.ajog.2022.07.025

[92] Schifrin, B.S. and Ater, S. (2006) Fetal Hypoxic and Ischemic Injuries. *Current Opinion in Obstetrics and Gynecology*, **18**, 112-122. https://doi.org/10.1097/01.gco.0000192984.15095.7c

[93] Evans, M.I., Eden, R.D., Britt, D.W., Evans, S.M. and Schifrin, B.S. (2019) Re-Engineering the Interpretation of Electronic Fetal Monitoring to Identify Reversible Risk for Cerebral Palsy: A Case Control Series. *The Journal of Maternal-Fetal & Neonatal Medicine*, **32**, 2561-2569. https://doi.org/10.1080/14767058.2018.1441283

[94] Heyborne, K.D. (2017) A Systematic Review of Intrapartum Fetal Head Compression: What Is the Impact on the Fetal Brain? *AJP Reports*, **7**, e79-e85. https://doi.org/10.1055/s-0037-1602658

[95] Sims, M.E. (2019) Legal Briefs: Head Compression, Ischemic Encephalopathy, and Adverse Outcome. *Neoreviews*, **20**, e432-e436. https://doi.org/10.1542/neo.20-7-e432

[96] Miller, L.A. (2017) The Latest Headache for Clinicians: Head Compression and Brain Injury. *The Journal of Perinatal & Neonatal Nursing*, **31**, 297-298. https://doi.org/10.1097/JPN.0000000000000285

[97] Sholapurkar, S.L. (2022) Birth Attendants Should Ponder the Broad Conciliant Evidence of Paramount Importance and Their Own Experience Confirming That Fetal Head Compression in Labor Causes Benign Decelerations. *American Journal of Obstetrics & Gynecology*, **227**, 119-121. https://doi.org/10.1016/j.ajog.2022.01.037

[98] Dupuis, O., Silveira, R., Dupont, C., Mottolese, C., Kahn, P., Ditmar, A., *et al.* (2005) Comparison of "Instrument-Associated" and "Spontaneous" Obstetric Depressed Skull Fractures in a Cohort of 68 Neonates. *American Journal of Obstetrics & Gynecology*, **192**, 165-170. https://doi.org/10.1016/j.ajog.2004.06.035

[99] Huang, C.-C. and Shen, E.-Y. (1991) Tentorial Subdural Hemorrhage in Term Newborns: Ultrasonographic Diagnosis and Clinical Correlates. *Pediatric Neurology*, **7**, 171-177. https://doi.org/10.1016/0887-8994(91)90080-5

[100] Callaway, N.F., Ludwig, C.A., Blumenkranz, M.S., Jones, J.M., Fredrick, D.R. and Moshfeghi, D.M. (2016) Retinal and Optic Nerve Hemorrhages in the Newborn Infant: One-Year Results of the Newborn Eye Screen Test Study. *Ophthalmology*, **123**, 1043-1052. https://doi.org/10.1016/j.ophtha.2016.01.004

[101] Zhao, Q., Zhang, Y., Yang, Y., Li, Z., Lin, Y., Liu, R., *et al.* (2015) Birth-Related Retinal Hemorrhages in Healthy Full-Term Newborns and Their Relationship to Maternal, Obstetric, and Neonatal Risk Factors. *Albrecht von Graefes Archiv fur Klinische und Experimentelle Ophthalmologie*, **253**, 1021-1025. https://doi.org/10.1007/s00417-015-3052-9

[102] Hong, H.S. and Lee, J.Y. (2018) Intracranial Hemorrhage in Term Neonates. *Child's Nervous System*, **34**, 1135-1143. https://doi.org/10.1007/s00381-018-3788-8

[103] Yatham, S.S., Whelehan, V., Archer, A. and Chandraharan, E. (2020) Types of Intrapartum Hypoxia on the Cardiotocograph (CTG): Do They Have Any Rela-

tionship with the Type of Brain Injury in the MRI Scan in Term Babies? *Journal of Obstetrics and Gynaecology*, **40**, 688-693.
https://doi.org/10.1080/01443615.2019.1652576