A Systematic Review of Intrapartum Fetal Head Compression: What Is the Impact on the Fetal Brain?

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Abstract

Objective During labor the fetal head is subjected to pressure related to uterine contractions and maternal pushing. Here we systematically review what is known about fetal head compression and its effects on fetal intracranial pressure, oxygenation, blood flow and cerebral function, and the plausibility that it might cause isolated fetal brain injury.

Study Design Systematic review of intrapartum fetal head compression and fetal brain injury in accordance with the MOOSE methodology. The PubMed database was searched using a combination of the terms "fetal," "head," "cranial," "extracranial," "pressure," and "compression." Additional references were obtained using multiple strategies. Results were evaluated, and relevant studies encompassing animal and human data using several approaches are summarized in this review.

Results Studies support a significant increase in fetal extracranial pressure with contractions and pushing. However, available data do not support a concomitant significant relative increase in intracranial pressure, a reduction in cerebral circulation or oxygenation, or an impact on cerebral function.

Conclusion A review of the literature indicates that fetal intracranial pressure is well protected from extracranial forces. Available data do not support intrapartum fetal extracranial pressure as a cause of fetal brain injury.

Precis The fetal brain is relatively unaffected by intrapartum fetal head compression.

Keywords

- mechanical forces of labor
- fetal head compression
- fetal brain injury
- fetal intracranial pressure

During human parturition, the fetal head is subjected to external forces resulting from uterine contractions and maternal pushing efforts. The impact of these extracranial forces on intracranial pressure (ICP) and the fetal brain is difficult to study, and it has received relatively little attention in the literature. Though generally regarded as a normal component of labor and delivery, historically some obstetrical providers have opined that these extracranial forces can directly injure the fetal brain.[1–3] Even though many of these speculations antedated our current detailed understanding of intrapartum fetal neurologic injury, such speculation calls attention to the need to better understand the potential impact of extracranial forces.

A more recent version of extracranial pressure as a cause of brain injury has been hypothesized, speculating that uterine contractions or maternal pushing efforts alone are capable of causing permanent fetal brain injury in the absence of generalized fetal acidemia and its typical correlates (multisystem organ damage, low Apgar scores, and other indicators of an intrapartum event).[4] This hypothetical

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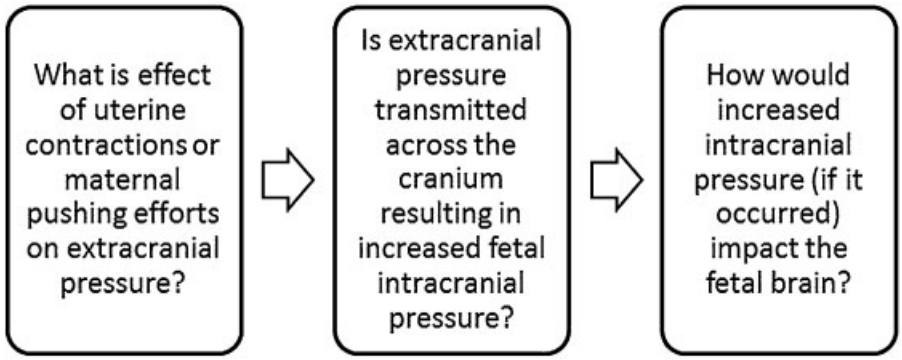

Fig. 1 Hypothetical three-step mechanism of intrapartum fetal brain injury.

injury mechanism depends on three steps: First, uterine contractions or maternal pushing efforts lead to increased pressure on the fetal cranium; second, this extracranial pressure is transmitted across the cranium resulting in increased fetal ICP; third, this increased ICP results in decreased cerebral perfusion and brain injury.

This three-stage framework shown in ►**Fig. 1** provides a useful approach to review what is known about the effects of extracranial pressure. This systematic review aims to tabulate available data regarding extracranial forces and their impact on ICP as well as oxygenation, blood flow, and cerebral function.

Materials and Methods

The author performed a systematic review of the published literature regarding the impact of intrapartum fetal head compression on the fetal brain using the MOOSE (Meta-Analysis of Observational Studies in Epidemiology) methodology.[5] Specifically, this review addresses the hypothesis that intrapartum fetal head compression may result in isolated cerebral ischemia and brain injury in the absence of systemic hypoxemia and acidemia.

The NCBI PubMed portal was used to conduct searches of "fetal head compression," "fetal head pressure," "fetal cranial pressure," "fetal cranial compression," and "fetal extracranial pressure." The searches included all publication dates and languages included in PubMed. Resulting articles included an abstract, animal studies, case series, cohort studies, and case-control studies.

Next, all obtained articles were placed into one of five categories: Articles presenting original data regarding extracranial forces (fetal extracranial forces); articles presenting original data investigating how fetal extracranial forces affect the fetal ICP, oxygenation, blood flow, or cerebral function (fetal extra- and intracranial pressure); and articles presenting original data on how an increase in ICP impacts fetal cerebral blood flow, metabolism, and function (fetal ICP and cranial blood flow, metabolism, and function). General reviews of fetal brain injury hypothesize how fetal head compression might result in fetal brain injury, but do not present original data. Miscellaneous articles discuss a myriad of topics regarding fetal physiology, brain injury, etc., but they do not present original data relevant to[1–3] mentioned as above.

Next, to further ensure a complete review, bibliographies from references categorized as above were manually searched. Utilizing the "Cited by" feature of PubMed, references of articles categorized as above were manually searched. References from a recent monograph chapter of fetal head compression and brain injury were manually searched for additional relevant references.[4] Last, the bibliographies of two unpublished articles complied by plaintiffs' experts who support this hypothesis were also included in this systematic review.

Finally, articles obtained from these secondary search strategies were added to categories as above. All articles categorized in the first three categories as above were maintained for this review. All subjects from these studies were included in this systematic review. References categorized in the last two categories as above[4,5] are summarized in Supplementary File and were not considered further.

Results

►**Tables 1–3** summarize the articles from the aforementioned categorization maintained for further review, and they are categorized as: fetal extracranial forces, fetal extra- and intracranial pressure, or fetal intracranial pressure and cranial blood flow, respectively.

Fetal Extracranial Forces

The studies in ►**Table 1** report on pressures applied to the fetal head during labor and pushing, using mechanical sensors placed alongside the fetal head.[6–12]

These studies report that the peak head-to-pelvis pressure varies from patient to patient and during labor in the same patient. Six studies indicate that the peak pressure between the head and pelvis may be as high 120 to 300 mm Hg during normal labor.[6–11] The study by Antonucci et al[12] reports pressures from 235 to 514 mm Hg. The highest pressures reported by all studies occur at the cranial "equator" (largest diameter of the skull) and are lower elsewhere.

Collectively, these studies indicate that the portion of the fetal skull opposed to the boney pelvis may be subjected to pressures as high as 120 to 500+ mm Hg intrapartum.

Table 1 Fetal extracranial forces

Topic	Study	N	Comments
Head-to-pelvis forces, all studies of human subjects in labor with mechanical sensors adjacent to fetal head	Antonucci et al[12]	6	Peak head to cervix pressure 235–514 mm Hg.
	Lindgren[6]	Not stated	All remaining studies report peak head-to-pelvis pressures of 120–300 mm Hg. No evidence provided linking higher pressures with poor neonatal outcomes.
	Rempen and Kraus[7]	42	
	Schwarcz et al[9]	18	
	Schwarcz[8]	Same cohort as Schwarcz et al	
	Svenningsen and Jensen[10]	18	
	Svenningsen et al[11]	46	

Fetal Extra- and Intracranial Pressure

Conceptually, how and to what extent extracranial forces might be transmitted to the intracranial space is not immediately obvious. Different parts of the skull are subjected to different forces (intra-amniotic, intravaginal, cervical, head-to-pelvis). The cranium, with its unfused sutures, is able to change shape to accommodate this pressure. Is the highest pressure transmitted? The lowest? An average? It is also important to consider precisely what pressures and what pressure changes might be important. It seems appropriate to evaluate ICP in relation to intra-amniotic pressure, as this is the ambient pressure to which the rest of the fetus is exposed. An increase in intra-amniotic pressure per se would not be expected to redistribute flow to the vital organs (brain, heart, kidneys, etc.), as pressure on the heart, vasculature, organs, etc. would all increase in the same amount. It would only be with a *relative* increase in ICP out of proportion to the intra-amniotic pressure that potential compromise might occur. This perspective was shared by the investigators authoring the papers in ►**Table 1**, who uniformly used intra-amniotic pressure as the reference baseline. The primary concern would occur with a rise in ICP *out of proportion to* the intra-amniotic pressure.

Studies that directly measured fetal extra- and intracranial pressure are summarized in ►**Table 2**. Placement of intracerebral and intraamniotic catheters allowed continuous simultaneous measurements in these fetuses judged to have lethal hydrocephalus. The detailed findings of these studies are included in ►**Table 2**.

The findings are quite consistent. All report a resting intracranial baseline pressure of approximately 22 to 30 mm Hg and a resting intraamniotic pressure of approximately 10 mm Hg, resulting in a resting gradient of 12 to 20 mm Hg. With contractions, both pressures increase, resulting in a net change in the gradient of −12 to +12 mm Hg. Thus, while high external pressures may be applied to the cranium, the ICP remains quite stable.

The applicability of these data to fetuses with normal cranial anatomy is unknown. Given these limited direct ICP data, it is critical to examine any indirect evidence of increased ICP due to labor or pushing. Important information of this nature comes in three forms as summarized in ►**Table 3**.

Four studies monitored fetal cerebral oxygenation during uterine contractions or with maternal pushing using near-infrared spectroscopy (NIRS).[13–16] All studies revealed a

Table 2 Fetal extra- and intracranial pressure—direct measurement

Study	Comments	Resting gradient IAP-ICP (mm Hg)	Peak gradient IAP-ICP (mm Hg)	Change in gradient with contractions (mm Hg)
Schwarcz et al[9]	Fetal demise, IAP not measured. ICP increased 30–50 mm Hg with contractions	NA	NA	NA
Mocsáry et al[21]	2 hydrocephalic fetuses measured early labor through 8–9 cm dilation	20	15–20	−5 to 0
Mooij et al[32]	Hydrocephalic fetus, severe hydrocephalus	20	15–20	−5 to 0
McCrann and Schifrin[33]	2 hydrocephalic fetuses	12[a]	0–25	−12 to 12

Abbreviations: IAP, intraamniotic pressure; ICP, intracranial pressure; NA, not available.
[a]Negative resting ICP suggests monitor not zeroed in one of two fetuses. Data from this fetus not included.

Table 3 Fetal extra- and intracranial pressure—indirect studies

Topic	Study	Species	N	Comments
Effect of uterine contractions on cerebral oxygenation as measured by NIRS in human patients in labor	Aldrich et al[14]	Human	10	Cerebral blood volume increases with contractions
	Aldrich et al[13]		30	Reduced cerebral oxygenation during labor
	Aldrich et al[15]		41	Cerebral and systemic oxygenation similar with labor and delivery—no isolated cerebral hypoxia
	Peebles et al[16]		10	Reduced cerebral oxygenation during labor
Effect of uterine contractions on fetal EEG in human patients in labor	Wilson et al[17]	Human	25	Fetal EEG not impacted with abnormal labor, even with severe head molding; fetal EEG is impacted by systemic acidosis
	Rosen et al[18]		300+	Fetal EEG not impacted by normal labor
Effect of extracranial pressure on cerebral blood flow in lambs	O'Brien et al[19]	Sheep	4	200 mm Hg × 120s applied to lamb cranium caused decrease in cerebral blood flow
	Mann et al[20]		30	Pediatric rib spreader used to apply pressure to lamb cranium resulting in decreased cerebral oxygen consumption

Abbreviations: EEG, electroencephalogram; NIRS, near-infrared spectroscopy.

reduction in cerebral oxygenation during labor. Though superficially these studies might seem to indicate an adverse effect of extracranial pressure, this is in fact not the case, because the changes in cerebral oxygenation merely mirror changes in systemic oxygenation. This was demonstrated by comparing intrapartum cerebral oxygenation late in labor with newborn cord blood gasses and showing that they are concordant.[15] Thus these data show that cerebral oxygenation tracks systemic oxygenation with labor and delivery with no incremental decrease in cerebral oxygenation related to fetal head compression.

Other studies have assessed the impact of extracranial forces by determining the impact intracerebral blood flow. Because arterial pressures are higher than venous pressures, if compression of fetal cerebral vessels occurred, fetal cerebral blood volume would markedly decrease due to compression of both arteries and veins. On the contrary, some studies of fetal cerebral oxygenation have also measured fetal cerebral blood volume and found that blood volume actually *increases* with contractions.[14] This likely represents an increase in cerebral blood flow due to autoregulation in response to transient systemic hypoxemia, as the study's authors speculate, and shows directly that arterial and venous compression does not occur.

Third, the impact of contractions has also been assessed indirectly by studying the fetal electroencephalogram (EEG) in response to labor and delivery, head molding, and fetal acidosis in both normal and abnormal labor. The interesting study by Wilson et al argues quite strongly against an adverse effect of fetal head compression.[17] In a small study of 25 human high-risk labors with frequent administration of oxytocin, they demonstrated that the fetal EEG was not related to fetal head pressure as assessed by severe head molding but was related to systemic acidosis. A second study by Rosen et al also showed no effect on the fetal EEG with normal labor.[18] These findings thus confirm the traditional view that head pressure in both normal and abnormal labor has no effect on cerebral function, even with severe molding.

Two animal studies have attempted to study the impact of head compression on the fetal brain. O'Brien et al attempted to understand the effect of extracranial pressure on cerebral

Table 4 Experimental effect of increased fetal ICP on cranial blood flow and metabolism

Study	Species	Technique	Comments
Mocsáry et al[21]	Human	Human fetus with lethal hydrocephalus—artificial increase in ICP to 100–120 mm Hg required to cause fetal bradycardia	Supraphysiologic ICP far above observed pressures occurring in labor needed to cause bradycardia; potential effects on cerebral blood flow or oxygenation not studied
Harris et al[23]	Sheep	Fetal lamb model, infusion of fluid into intracerebral space —ICP increased by ± 50 mm Hg	Robust Cushing's reflex able to withstand wide range of supraphysiologic ICP increases
Harris et al[24]			
Harris et al[22]		Premature fetal lamb model, infusion of fluid into intracerebral space	Cushing's response less well developed in premature lamb model

Abbreviation: ICP, intracranial pressure.

blood flow, using a cuff to apply pressure to a fetal lamb skull.[19] An extremely high pressure (200 mm Hg) for 150 seconds was needed to produce fetal bradycardia with a resultant fall in cerebral perfusion. It is unclear whether the reduced perfusion results from the pressure per se or the bradycardia. Another study of mechanically applied extracranial pressure by Mann et al used a pediatric rib retractor to apply high forces to the fetal lamb skull.[20] The fetal lamb with its fused cranial sutures is likely a poor model for these studies. The relevance of these nonphysiologic animal models is unclear, but it is included for completeness.

In summary, directly measured ICPs and studies of fetal cerebral oxygenation, blood volume and EEG confirm that the fetal brain is well protected from extracranial forces that occur during labor.

Fetal Intracranial Pressure and Cranial Blood Flow and Metabolism

The studies in ►**Table 4** address the effects of a marked artificial increase in ICP in human and animal models. As noted, such an increase in pressure does not appear to actually occur, but these studies are included here for completeness.

The only human study is of a single fetus with lethal hydrocephalus from the previously mentioned paper by Mocsáry et al.[21] In an experimental phase of the study, fluid was infused into the cranium of a hydrocephalic fetus to cause an artificial increase in the ICP. Supraphysiologic pressures (> 100 mm Hg) were required to cause fetal bradycardia. Cerebral blood flow or oxygenation was not studied, so it is unknown whether the bradycardia occurred from hypoxia or a nonspecific vagal response.

The three animal studies investigated cerebral autoregulatory responses of fetal lambs in response to infusion of fluid into the intracranial space as a way to raise ICP.[22–24] In these studies the relative ICP was increased by 50 mm Hg, a supraphysiologic increase based on the above data. Importantly, they document a robust fetal Cushing's reflex protective against an increase in ICP were such an increase to occur.

It is important to note that these studies again clearly demonstrate that the relatively high extracranial pressures of 120 to 500+ mm Hg observed in normal labor are not directly transmitted to the intracranial space, as they would result in bradycardia with each contraction.

Conclusion

Fetal ICP, oxygenation, blood flow, and function appear well protected from the increased extracranial forces that occur during labor and pushing. Consistent findings using a wide array of techniques, including directly measured pressure, NIRS of both oxygenation and cranial blood volume, and EEG, support this conclusion.

It should be noted that the quality of studies reviewed here is mixed, and evaluating for bias is difficult. This is not a criticism of the studies per se but rather a reflection of the difficulty in studying this topic. Randomized controlled trials of "excessive" head compression (however, that might be defined) are of course not practicable. Invasive monitoring of normal fetuses with lethal anomalies is likewise not feasible. That said, it should be noted that proponents of this theory have taken no apparent measures to reliably model this hypothesis, as has been done for brachial plexus injuries resulting from should dystocia, for example.[25] Certainly biomechanical or animal studies could be conducted to investigate this hypothesis, although it would likely be important to use an animal model with unfused sutures, unlike the sheep studies.

It seems likely that molding of the fetal head allowed by the unfused sutures may be part of the mechanism by which these increased pressures are accommodated. An analogy may help demonstrate this point: Imagine a car raised on a lift and the tires inflated to 35 psi. When the car is lowered to the ground, the entire weight of the car is placed on the tires. The flexible tires (analogous to the fetal head) changes shape (molding) against the hard floor (bony pelvis) with the tires now supporting the entire 4,000 lb weight of the car (contractions or pushing), yet the tire pressure (ICP) does not

change. Similarly, despite significant extracranial pressure, the ICP changes minimally, and cerebral oxygenation, perfusion, and function are preserved.

Indeed the idea that extracranial forces might injure the fetal brain as hypothesized by some may strike many obstetrical attendants as improbable, given the known extracranial pressures and molding that occur with many vaginal births. It is important to emphasize that the findings summarized here arise from both normal and abnormal labor, including those augmented with oxytocin. In this regard, it must also be noted that proponents of this theory have failed to provide any specific criteria that might allow head compression as a cause of brain injury to be diagnosed or indeed avoided. Absent such criteria, it becomes a convenient wastebasket for brain injury not due to systemic hypoxic-ischemic injury without an alternative explanation.

If an increase in extracranial forces were indeed harmful, one might anticipate an increase in birth injury with an increased use of oxytocin or longer labor. In fact, the use of oxytocin has increased from zero prior to its synthesis in the 1950s to $>$ 50% in some modern cohorts[26] with no corresponding increase in the incidence of CP in term newborns.[27] A detailed secondary analysis of a recent study also failed to correlate the total number of contractions, the presence of frequent contractions ($>$ 20/h), or the use of oxytocin with the occurrence of encephalopathy.[28] A large cohort study did not find an increase in intracranial injury (as manifest by intracranial hemorrhage) in women with spontaneous vaginal delivery as compared with cesarean birth prior to labor.[29] A prolonged second stage of labor has been associated with some increase in adverse newborn outcome in some studies related to an increased incidence of fetal acidosis, but isolated brain injury in the absence of typical criteria for intrapartum injury has not been reported.[30] Indeed it seems likely in general that contractions putatively strong enough to cause brain injury would long before lead to impaired gas exchange at the placental level. Accordingly, the conclusions reached by Freeman et al appear apt: "Although (head compression as a cause of brain injury) has become a popular legal theory, there remains no scientific basis for the notion that cerebral ischemia caused by the pressures of labor and in the absence of fetal hypoxia, is a cause of cerebral palsy."[31]

Conflict of Interest
The author reports no conflict of interest.

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