Textbooks can be wrong - head compression is very unlikely to contribute to intrapartum decelerations

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Textbooks can be wrong - head compression is very unlikely to contribute to intrapartum decelerations

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Dear Editor,

We appreciate Mr Sholapurkar's interest in our systematic review of the head compression hypothesis, although it is disappointing to see a call to place more weight on 'wider observational evidence', that is to say anecdote.[1] He states the "dura-mater and falx, with rich vagal-innervation, are very stretch-sensitive triggering 'vagal bradycardia' (visceral non-baro/non-chemo reflex–Williams Obstetrics)". This hypothesis is not supported by the literature. The only relevant section in Williams Obstetrics is: "Head compression probably causes vagal nerve activation as a result of dural stimulation… (Paul and colleagues, 1964)." The animal study by Paul and colleagues does not mention dural stretching.[2] This study actually shows that decelerations were observed during head compression in association with a 50% reduction in carotid blood flow and suppressed electroencephalographic activity. Thus, neither Mr Sholapurkar nor Williams Obstetrics provide evidence for dural stretching contributing to fetal heart rate decelerations.

Although Mr Sholapurkar does not ascribe his hypothesis to any known reflex, the only potential candidate we can identify is the trigeminocardiac reflex. This reflex is predominantly stimulated by direct surgical manipulation of the dural mater or trigeminal nerve and triggers bradycardia, asystole and hypotension.[3] As far as we are aware, there is no evidence that it can be reliably stimulated without surgical exposure of the dura mater or other invasive procedures. This is fortunate since it does not seem to be benign.

Finally, the letter illustrates a misunderstanding of the integration of multiple reflexes. It is wrong to describe the peripheral chemoreflex as providing a limited or precarious defense.[4] The chemoreflex is rapidly triggered by falls in fetal oxygen tensions and offers a graded, consistent response. We propose that it is supported by the intracranial baroreflex, which is not observed only in chronic situations, but as demonstrated in adults, neonates and fetuses, is

activated over timeframes that are relevant to prevent or alleviate cerebral ischemia secondary to head compression.[1]

In conclusion, there is compelling evidence that uterine contractions impair utero-placental gas exchange, leading to brief, repeated fetal hypoxia.[1] Reassuringly, it is also clear that brief, shallow decelerations during contractions represent moderate hypoxia and that well-understood reflex responses help the fetus to fully compensate, essentially indefinitely.[4] It is not necessary to invent a completely new reflex to explain this exemplary fetal ability to adapt to transient moderate hypoxia. Fetal adaptation to deeper hypoxia, with correspondingly deeper variable decelerations, is a story for another day.

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