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Early decelerations: Are they really benign?

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Abstract

Early deceleration has been traditionally considered as a benign feature for fetal well-being, as opposed to late and variable decelerations. Several investigators correlated the increased intracranial pressure during labor and delivery with early decelerations. Others argued that if this is the case, early decelerations would have been a consistent feature in all laboring women. Evidence from recent studies paves the way for a plausible explanation for this cardiotocographic pattern. It considers both increased intracranial pressure due to head compression above a specific threshold, and fetal health status. Early deceleration should be considered as a feature of compensated fetalhypoxia and should alert the obstetrician for more close montoring for his/her patient.

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Early decelerations are shallow, short-lasting decelerations, with normal beat to beat variability within the deceleration, and are coincident with contractions [1]. The traditional knowledge and teaching stressed that early decelerations, regardless of their depth, duration and frequency, do not reflect any underlying fetal hypoxia or acidosis [2]. Several investigators argued that fetal head compression during labor is the main reason for early decelerations [3][4][5][6]. It increases the intracranial pressure (ICP) with subsequent stimulation of the vagus nerve causing a drop in the fetal heart rate and early decelerations [7]. If this argument is true, early decelerations should have been a consistent feature in all labors and deliveries as head compression normally occurs with head engagement during normal labors and deliveries. Actually, this is not the case. The evidence is derived from different studies. The incidence of early decelerations in laboring women is not uniform and widely varies in different studies. Cibalis [8] reported that early decelerations are found in 12%, 19% and 27% of all labors. Aladjem and Miller [9] found an incidence of early deceleration patterns following membrane rupture of 6.25 % during the first 15 minutes and 1.78 % after 45 minutes. Steer et al reported [10] an increase

in the frequency and amplitude of early decelerations after elective rupture of membranes. To the contrary, Chung and Hon [11] conducted an experimental human study on six laboring women with normal CTG and a cervical dilatation of 8 cm or more. They applied pressure over the fetal head through the vagina either by fingers or a vaginal pessary. Fetal responses with early decelerations were documented in all women, accounting for an incidence of 100%. Other investigators reported similar results on non-viable hydrocephalic fetuses. Mocsary et al [12] reported fetal heart rate decelerations during the first stage of labor in response to artifically induced high intracranial pressure in 2 nonviable hydrocephalic fetuses. Similarly. Mooij and his group in 1992 [13] were able to reach the same conclusion in a hydrocepahic living fetus in labor. As for animal studies, Paul et al. [14] performed six head compressions on four full-term lambs and reported fetal heart decelerations to this compression in all cases while Harris et al [15] failed to show any decelerations in response to increased intracranial pressure.

These contradictory findings do not support the assumption that fetal head compression is the reason of early decelerations. Similarly, extra-cranial forces, namely the uterine contractions associated with true labor and maternal pushing efforts, cannot be held responsible for early decelerations. They have no effect on intracranial pressure. They are not transmitted to the fetal head, and consequently, they do not compromise cerebral blood flow [16].

An acceptable explanation for early decelerations may involve both the head compression and fetal health status. 1) A threshold may exist for the increased intracranial pressure and the duration of exposure to this high pressure, above which abnormal CTG patterns start to appear. Evidence comes from the study by Mocsáry et al [12] who found that a fall in the FHR ensued at an average intracranial pressure of 55 mm. Hg. They claimed that this fall was within physiologic limits until the intracranial pressure reached 100 mm. Hg, but was of a pathologic nature above this value. This is supported by the findings of a recent study [17]. Lear et al found that it is only at extremely high levels of intracranial pressure that the parasympathetic nervous system is activated, with a subsequent fall in heart rate. 2) When fetal hypoxia occurs, the fetus utilizes different mechanisms with supreme efficacy to maintain blood flow to the brain "brain sparing effect" and the myocardium on the expense of the less vital organs and systems such as the kidneys and gastrointestinal tract. First, the high affinity of fetal hemoglobin to oxygen allows it to bind to huge amounts of oxygen and release them slowly to ensure adequate oxygenation to the different tissues and organs [18]. Second, the fetal vascular shunts such as ductus venosus and ductus arteriosus provides extra-supply of oxygenated blood to tissues and organs at risk of damage such as heart, brain and kidneys, and lastly the huge amount of stored glycogen in the fetal myocardium acts as a high anaerobic reserve to support myocardial contractility [19][20]. These mechanisms do not operate fully with each fetus. Each fetus has its own reserve and capability to adapt depending on its health status. That is why a contraction may correspond to a fetal early deceleration in one fetus but not another. A healthy fetus can tolerate mild degree of hypoxia without decelerating its heart rate. Early deceleration occurring early in labor before engagement and head compression often reflect cord compression and may serve as a warning of impending fetal problem [21].

To conclude, early deceleration may be the earliest CTG pattern to indicate minimal degree of hypoxia, which may progress to late decelerations and other abnormal CTG patterns if hypoxia persists. It is the author's view that it is time to reconsider the assumption that early decelerations are completely benign and can be tolerated without any negative consequences. Early decelerations should ring a bell and alert the obstetrician for close monitoring of his/her patient and

to decide whether to expedite delivery with emergency Cesarean section or instrumental delivery if the circumstances allow.

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