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Opinion Article

Heart Rate and Arterial Pressure in Fetus

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DESCRIPTION

The rationale for considering these two aspects of the fetal circulation together comes from the well-known interaction between arterial blood pressure and heart rate in adults. In fact, attempts to extrapolate knowledge of circulatory control in adults back to the fetus have contributed significantly to our understanding of the control of the fetal circulation determine fetal heart rate and arterial pressure and to discuss the interaction between the two variables and the underlying physiological processes that determine them newer methods for measuring these variables are discussed, with particular reference to the clinical importance of understanding disturbances in t hem under normal physiological conditions.

Determinants of heart rate

The fetal heart is affected by a variety of mechanisms, just like the adult heart, to keep its rate higher than the Sino atrial node's intrinsic rate in the late-gestational sheep fetus, neural variables directly affect the Sino atrial node through the vagus and sympathetic nerves, as well as indirectly through the release of catecholamine's from the adrenal medulla these brain effects, which can compose the various components of reflex heart rate control, seem to have only a little impact during mid-gestation bradycardia initially results from late gestation, hypoxia, or asphyxia (whether caused by maternal inhalation of a hypoxic gas, cord blockage, or limitation of uterine blood supply), followed by a slow increase in heart rate which over the next several minutes recovers to or exceeds its control value given that the early Brady cardiac is eradicated by bilaterally chopping off the carotid sinus nerves or by administering atropine, the early Brady cardiac in these conditions is chemo-reflex in origin.

Since it happens after transient uterine artery occlusions or when the rise in arterial pressure, this Brady cardiac is not a baro reftex the delayed increase in fetal heart rate is caused by an 8-adrenergic stimulation from the rise in plasma catecholamine's other humeral agents released in hypoxia or asphyxia may also be involved, but their exact role has not yet been determined prevented by a- adrenergic blockade or transection of the spinal cord at Ll-2 which interrupts descending sympathetic outflow Arginine Vasopressin (AVP), which is secreted, is the most prevalent of them huge quantities in hypoxia, academia, asphyxia, or other conditions, and has been shown to lower fetal heart rate.

Therefore, it is believed that the so-called "early decelerations" brought on by contractions during a typical childbirth are caused by a quickly acting reflex with the vagus as the efferent limb since atropine does not prevent them, the mechanisms causing "late decelerations" are not clearly reflex in nature such fetal heart rate slowdowns are most likely caused by the direct effects of hypoxia on the Sino atria J node or conducting tissue, or it's also possible that humeral chemicals that depress the heart are released by reflexive and non-reflexive causes, such as in cases of pyrexia brought on by fever or maternal exposure to high environmental temperatures, it is possible to cause protracted increases in the mean fetal heart rate however, hemorrhage also causes a rise in fetal heart rate; this does not automatically reflect hypoxia or academic status in both sheep and human fetuses, fetal heart rate slowly decreases as gestation progresses this is accompanied by an increase in stroke volume as the heart expands and an increase in mean arterial pressure since a vagotomy during late pregnancy does not result in an increase in vagal tone, the decrease in heart rate cannot be explained exclusively in terms of a baro reftex.

Heart rate and arterial blood pressure in the fetus interact

For the sheep fetus, the negative relationship between fetal arterial pressure and heart rate previously stated is well established unfortunately, there are no similar statistics for fetuses of humans or any other primates, despite the fact that the reduction in fetal heart rate during this time in humans is well documented the inverse relationship implies that a barostat is in use cause and effect cannot be determined only from a correlation of this nature, though the baro reflex has been demonstrated to function in the late-gestation sheep fetus as a transitory rise in arterial pressure brought on by an intravenous infusion of phenylephrine results in a decrease in fetal heart rate that is reversed by chemo denervation or atropine the fact that the baroreceptors become less sensitive during this stage of gestation complicates the developing process, to a reduction in baro reflex "gain" may occur as a result of the increase in mean

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arterial pressure, as occurs during postpartum hypertension, but if this is the case, it is remarkable that the relationship between arterial pressure and heart rate has not been studied however, assuming this is the case, it is unexpected that the relationship between arterial pressure and heart rate over late gestation is linear for example, during exercise in the adult, a balance is struck between the vasodilator mechanisms in the exercising muscles and the vaso constriction necessary to re distribute cardiac output away from non-essential tissues and to increase arterial pressure in order to provide improved perfusion to permit such a rise in pressure, the fetus would benefit from study on the interaction between peripheral resistance and heart rate.