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Title : BRADYCARDIA FOLLOWING PCB* IN THE FETAL BABOON

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Introduction. Fetal bradycardia is a not infrequent complication following paracervical block anesthesia (PCB).* Until recently the direct toxic effect of the anesthetic on the fetal heart has been the most commonly supported hypothesis for the mechanism of post-PCB bradycardia.[1] A significant relationship between bradycardia and high blood levels of mepivacaine in the human fetus (as high as 6.3 μg/ml) accompanied by a decreased pHa has been reported.[2] However, we have demonstrated previously[3] that the blood concentration of lidocaine in healthy fetus required to produce cardiovascular toxicity is much higher than has been reported.

Methods. In order to further explore the causal relationship between the use of local anesthetics and fetal bradycardia, 52 PCB's were administered to 27 baboons. Twenty-seven PCB's were performed with lidocaine and 25 with 2-chloroprocaine; the dosages used were comparable to those used clinically in pregnant patients on the basis of body weight. Maternal and fetal arterial pressure, heart rate, intravascular fetal PaO_2, uterine blood flow and intraamniotic pressure were monitored continuously. Arterial blood samples were obtained simultaneously from mother and fetus for determination of pH, PCO_2, PO_2 and lidocaine concentration.

Results. Forty PCB's were performed with non-asphyxiated (normal) fetuses with the mean (± SE) pHa of 7.35 ± 0.023; $PaCO_2$, 42 ± 3.4 torr; and SaO_2, 62 ± 3.2%. The remaining 12 PCB's were given to asphyxiated fetuses with pHa of 7.10 ± 0.063; $PaCO_2$, 58 ± 4.0 torr; and SaO_2, 22 ± 2.3% prior to the PCB. Thirty percent of the normal fetuses developed post-PCB bradycardia, while bradycardia occurred in all asphyxiated ones. In the normal group, the heart rate decreased from the pre-PCB value of 184 ± 4.8 to 168 ± 6.0 beats/min ($P<0.05$), and in the asphyxiated fetuses from 186 ± 6.5 to 157 ± 12.5 beats/min ($P<0.05$). The bradycardia was associated with a fall in fetal oxygenation in all asphyxiated fetuses, while 50% of the bradycardia was accompanied by a decrease in SaO_2 values in the normal group. A marked decrease in uterine blood flow and an increase in uterine activity occurred during the first ten minutes after PCB. All changes were self-limiting in nature; they returned to the pre-block values during the next 20 - 30 min. However, recovery of SaO_2 in the asphyxiated group was slower (45 - 60 min). Peak lidocaine concentrations in maternal and fetal blood were found at the 8-min sampling, the average value being 2.25 and 0.78 μg/ml, respectively. There was no apparent difference in the type of anesthesia used on the uterine activity, blood flow, the incidence or the severity of post-PCB bradycardia. No maternal complication due to PCB was observed. Maternal arterial pressure, heart rate, and acid-base state remained essentially unchanged throughout.

Discussion. These observations indicate that post-PCB bradycardia produced in the baboon fetus is related to decreased fetal oxygenation secondary to an increase in uterine activity and a reduction in utero-placental perfusion.[4] These phenomena can occur at drug concentrations in both the maternal and fetal blood much lower than necessary to produce toxic manifestation. It should be emphasized that the use of PCB is not recommended in cases where the utero-placental circulation is imparied and/or the fetus is already acidotic and hypoxemic. Compared to healthy fetuses, all initially asphyxiated fetuses manifested hypoxemic bradycardia and slow recovery in our baboon study.

References

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