TITLE: CEREBRAL HEMODYNAMIC RESPONSE TO PROGESTERONE

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Recent events in the news have demonstrated the absence of information concerning the response of the cerebral circulation to pregnancy. With permission of our Animal Institute, this study was designed to determine the hemodynamic effects of progesterone on the microcirculation of the brain.

A total of 19 white Wistar rats were used: 7 pregnant, 6 nonpregnant females and 6 males. Anesthesia was induced with pentobarbital. Femoral arterial blood pressure was recorded and pH and CO2 were maintained by controlled ventilation. A left parietal craniotomy with an encapsulated cranial window was prepared for biomicroscopy. One mg of a 0.1% solution of progesterone was injected intraperitoneally. Changes in the internal diameter of cerebral arterioles were measured by image shearing. Red blood cell velocity (Vrbc) was measured by dual-slit photo metric method and correlation technique.[1] Volumetric blood flow was calculated from the diameter and the Vrbc. The procedure was then repeated for 2 mg progesterone, as well as 5 mg and 10 mg of an estradiol preparation. Percent changes from baseline were calculated and data were analyzed by ANOVA.

Progesterone 1 mg intraperitoneal injection resulted in a vasodilation of cerebral arterioles in the nonpregnant female rats (mean increase 10.3% ± SD 3.4%). This represented a significant difference ($P<0.005$) over the response of the pregnant rats (0.6% ± 4.8%). A minor response was noted in the males (1.2% ± 1.9%) which did not differ significantly ($P>0.5$) from the changes noted in the pregnant animals. Vrbc remained essentially unchanged, yielding a mean increase in volumetric flow rate of 21.8% ± 7.3% ($P<0.01$). The results with 2 mg progesterone were similar. Estradiol produced no noticeable effect in any group.

We have found that progesterone produces a vasodilation in the cerebral arterioles of nonpregnant female rats. Since it is unlikely that progesterone creates an "uncoupling" effect on cerebral metabolic utilization of oxygen, the increase in volumetric flow rate could imply an increase in Cmr O_2. Pregnant rats already have high circulating levels of progesterone acting upon the cerebral vessels. It is therefore not surprising that they show little reaction to small doses of exogenous hormone. The lack of response among the male rats probably implies either diminished receptor activity as compared with females or an antagonistic effect from male hormones.

Reference

1.Microvasc Res 15:93-101, 1978

TITLE: ECHOCARDIOGRAPHIC EVALUATION OF MATERNAL MYOCARDIAL FUNCTION DURING CESAREAN DELIVERY UNDER REGIONAL ANESTHESIA

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A recent study by Palmer, et al.,[1] documented a high incidence of electrocardiographic changes in parturients during cesarean delivery under regional anesthesia; these changes were often characteristic of myocardial ischemia. The current study has been undertaken to determine if these ECG changes are associated with systolic wall motion abnormalities on 2-D echocardiography or diastolic dysfunction by Doppler evaluation of mitral inflow.

Seven ASA I & II term parturients for elective cesarean delivery under regional anesthesia gave written consent and participated in this IRB-approved study. Baseline ECG, trans-thoracic echocardiogram (ECHO), and pulsed wave mitral flow (DOPP) were obtained prior to the procedure. ECHO (parasternal short and long axis, 2 and 4 chamber views) was used to assess myocardial wall motion as a measure of systolic function; DOPP was used to assess early mitral inflow velocity (E), mitral inflow velocity during atrial systole (A), and E/A ratio as a measure of diastolic function. Seven leads of the maternal ECG were recorded at intervals during the procedure. ECHO and DOPP were recorded continuously following induction until 16 to 20 minutes after delivery. ECG recordings obtained intraoperatively were compared to baseline by a blinded reviewer. ECHO and DOPP obtained intraoperatively were compared in blinded fashion to baseline to ascertain the occurrence of changes in wall motion or flow velocity pattern. Finally, findings from ECG and ECHO/DOPP studies were temporally correlated to determine any relationship.

Baseline ECG was within normal limits in all patients. Preoperative analysis of ECHO and DOPP revealed normal global cardiac function in all patients. Intraoperatively, non-diagnostic QRS, ST-Segment, and/or T-Wave changes were noted in 3 of 7 patients. Intraoperatively, all 7 patients demonstrated hyperdynamic function, which was most pronounced in the immediate post-delivery period; this was characterized by increased ejection fraction in all, with no segmental wall motion abnormalities. No abnormalities of diastolic function (E,A, or E/A ratio) were noted during the procedure, however, tachycardia was common and caused superimposition of E and A.

Palmer et al.[1] found a 47.3% incidence of ECG changes during cesarean delivery under regional anesthesia; this study confirms a comparable incidence of ECG change (42.8%). Further, time course of the ECG changes followed the same pattern. Palmer et al. speculated that myocardial ischemia could be the cause of the ECG changes. The present study demonstrates enhanced ventricular (systolic) function during cesarean delivery under regional anesthesia, and no evidence of diastolic dysfunction. Diastolic dysfunction is a very sensitive measure of ischemia, and wall-motion abnormalities are a more specific indicator of ischemia than ECG changes. Thus, the non-diagnostic ECG changes observed in this study are not likely due to ischemia; rather, medication, transient metabolic changes, catecholomines or changes in cardiac function due to delivery are possible causes of these ECG changes. Further study is necessary to determine if the ECG changes of a more pronounced degree observed by Palmer et al. are associated with abnormalities of myocardial function detectable by ECHO and/or DOPP.

REFERENCE

1. Anesth Analg 70:36-43, 1990.