Arginine vasopressin mediates cardiovascular responses to hypoxemia in fetal sheep

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Acute hypoxemia results in hypertension, bradycardia, and cardiac output redistribution in fetal sheep. The blood flow redistribution is produced by differential changes in vascular resistance of various fetal organs. ?-Adrenergic activity is one of the few vasoconstrictor mechanisms thus far identified in the hypoxemic fetal sheep. Arginine vasopressin (AVP) is a potent vasoconstrictor in adults. Since AVP administration to the normoxic fetus mimics some of the fetal cardiovascular responses to hypoxemia and fetal plasma AVP levels increase with hypoxemia, we examined the hypothesis that AVP modifies the fetal cardiovascular response to hypoxemia by changing the vascular resistance of some fetal vascular beds. To test this we determined fetal systemic arterial pressure and fetal cardiac output and its distribution during hypoxemia with and without the V1 AVP antagonist d(CH2)5-Tyr(Me)AVP. Fourteen fetal sheep (0.79-0.90 of gestation) were chronically catheterized. Five days after surg