TITLE: VASOPRESSIN CONSTRICTS PERIPHERAL, BUT DILATES CEREBRAL ARTERIES

AUTHORS: C. Tong, M.D., J.C. Eisenach, M.D.
AFFILIATION: Section of Obstetric Anesthesia, Wake Forest University Medical Center, Winston-Salem, NC 27103

INTRODUCTION: During fetal asphyxia distribution of cardiac output changes, with blood flow increasing to the brain and decreasing to the skeletal musculature. Preliminary work in adult dogs suggests that vasopressin (AVP), secreted during stress, is instrumental in this blood flow distribution.[1] As a first step in determining the role of AVP in this protective reflex in the fetus, we examined the effects of AVP on femoral and cerebral arteries from adult ewes in vitro.

METHODS: The protocol was approved by the Animal Care and Use Committee. Basilar (N=7) and femoral (N=8) arteries were obtained from adult ewes, adhesive connective tissue removed and arteries cut into 4 mm long rings. Endothelial cells were removed from some rings by gently rubbing with a stainless steel wire. Rings were placed in oxygenated Krebs solution, stretched to their optimum length tensions relationship, then exposed to increasing concentrations of AVP. To exclude actions of prostaglandins, indomethacin (10-5 M) was included in solutions in some experiments. Effects of AVP on vessel tension were compared to maximal contraction to norepinephrine and maximal relaxation to papaverine.

RESULTS: AVP produced dose-dependent contractions in femoral artery rings with an without endothelium (Fig. 1). In contrast, AVP produced dose-dependent relaxation in basilar artery rings, and this response was endothelial-dependent (Figure 1). Responses were not affected by indomethacin.

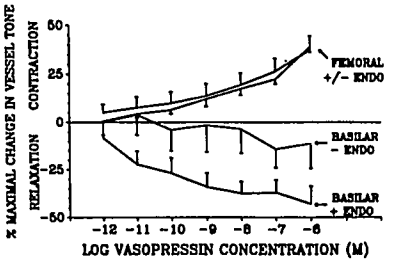

FIG. 1: Effect of AVP on Vessel Tone

DISCUSSION: These data in sheep agree with observations in dogs[1] that AVP, secreted during periods of hypotensive or hypoxemic stress, may redistribute blood flow by producing peripheral vasoconstriction and cerebral vasodilation. Future studies will examine the effects of AVP on fetal vessels, as well as the effects of local anesthetics (known to alter endothelial-dependent vascular relaxation) on these responses.

REFERENCES:

1. J Hypertension 2:421-422, 1984

A552

Title: THE EFFECTS OF DESFLURANE (D) AND ISOFLURANE (I) ON CARDIOVASCULAR (CV) AND CORONARY DYNAMICS (CoD) IN THE CHRONICALLY INSTRUMENTED DOG

Authors: R.G. Merin, M.D.; J-M. Bernard, M.D.; M-F. Doursout, Ph.D.; J.E. Chelly, M.D., Ph.D.
Affiliation: University of Texas Medical School, 6431 Fannin, MSB 5020, Houston, TX 77030

The CV effects of the new inhalation anesthetic D are similar to those of I in chronically instrumented swine.[1] There are no reports of the effect of D on CoD. We have studied such effects using D and I on the same chronically instrumented dogs on different days.

Seven dogs were instrumented for measurement of mean arterial pressure (MAP), left ventricular dP/dt, cardiac output (CO), coronary blood flow (CoF), and wall thickening fraction (WT). At least 10 days following recovery, each dog was administered both D and I on different days at multiple MAC concentrations with ventilation and F_iO_2 adjusted to awake values. Statistical analysis utilized linear and non-linear regressions and ANOVA.

There were no significant differences between mean values of CV and CoD awake or during any of the anesthetic concentrations between D and I (Table). CO was slightly decreased by high concentrations of I but not by D. CoF was increased by both anesthetics but the major effect was seen at 1.2 MAC with little further change during D and slight increase during I. Co vascular resistance (R) was also decreased by both D and I. As in swine, CV effects of D were essentially the same as those of I. Both anesthetics appear to be coronary vasodilators. It seems likely if I produces "coronary steal"[2], that the same results will be also observed for D.

References:
1. Anesthesiology 69:303-309, 1988.
2. Anesthesiology 66:389-292, 1987.

		Awake	1.2	1.75	2 MAC
MAP (mmHg)					
D	7	93±8	73±12*	67±12*	62±16*
I	7	97±19	79±11	67±8*	64±17*
HR (bts/min)					
D	7	79±14	140±13*	143±16*	139±18*
I	7	82±14	119±30*	127±23*	127±23*
CO (l/min)					
D	6	2.25±0.4	2.53±0.5	2.35±0.4	1.98±0.3
I	6	2.10±0.1	2.03±0.2	1.88±0.2*	1.78±0.3*
SVR (mmHg/L/min)					
D	6	43.1±10.5	30.5±11.4*	29.4±11.9*	30.7±11.9*
I	6	47.1±11.3	39.3±5.3	34.7±3.2*	35.7±8.6*
dP/dt (mmHg/sec)					
D	5	2856±340	1755±490*	1508±283*	1326±378*
I	5	2820±458	1807±475*	1504±421*	1384±445*
WT (%)					
D	5	25.8±6	17.5±2*	12.6±3*	12.2±3*
I	5	25.8±10	18.9±5*	16.3±9*	15.6±7*
CoF (ml/min)					
D	7	42±8	55±7*	58±14*	58±21*
I	7	41±12	56±22*	51±25*	66±22*
CoR (mmHg/ml/min)					
D	7	2.29±0.4	1.35±0.2*	1.21±0.3*	1.18±0.4*
I	7	2.48±0.6	1.54±0.5*	1.29±0.6*	1.06±0.4*

* = $p < 0.05$ vs Awake Mean ± SD