TITLE: CONTINUOUS NON-INVASIVE BLOOD PRESSURE TO DETECT HYPOTENSION AFTER REGIONAL ANESTHESIA IN OBSTETRICS

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Introduction. It is well known that epidural analgesia for labor can produce hypotension through sympathetic blockade. It is common practice in many institutions to measure the parturient's blood pressure manually or with an automatic non-invasive blood pressure device (NIBP) every five minutes after injection of local anesthetic through an epidural catheter. Blood pressure determination at five-minute intervals could theoretically permit hypotension to persist for up to five minutes before detection. One-minute cycling of the NIBP shortens the period until detection of hypotension. Even the five-minute inflation of the NIBP is often disturbing to the parturient in what should be a non-obtrusive, private atmosphere on the labor suite.

This study was undertaken to decide if use of a continuous non-invasive blood pressure device, the Finapres [1], would allow faster detection and treatment of hypotension with less annoyance to the patient. The first portion of the study was to directly compare the Finapres with a NIBP in the obstetric population. The Finapres has not been studied in this population previously.

Methods and materials. This study was approved by the Institutional Review Board. Patients were term parturients who had elected to received epidural analgesia for labor. All patients gave informed consent. Patients were connected to a Dinamap 1846SX (Critikon, Tampa, FL) and to a Finapres (Ohmeda, Boulder, CO). The devices were placed on opposite arms. During the first 20 minutes after injection of local anesthetic, the Dinamap was cycled at one minute intervals and the readings were recorded. The Finapres provided continuous blood pressure readings, which were recorded every 15 seconds. Matching systolic, mean, and diastolic pressures were thus recorded from 50 patients.

For comparison purposes, the standard technique of the Association for the Advancement of Medical Instrumentation (AAMI) was adapted for this study [2]. The error of measurement between the Dinamap and the Finapres was considered 0 if the Dinamap reading was equal to or between the highest and the lowest Finapres readings during its inflation. (The four manual recordings from the Finapres for the minute were used to determine the highest and lowest points.) If the Dinamap reading was above the highest reading of the Finapres, the error was the difference between the Dinamap reading and the highest Finapres reading, and similarly if it was below the lowest Finapres reading. (The rationale for this correction is that the Dinamap might read any pressure that occurred during its inflation.)

The systolic pressure from the Finapres (adjusted by applying the formula above) was plotted against the systolic from the Dinamap.

Results. The paired Dinamap and Finapres systolic pressures for the 50 patients were plotted. The regression line is:

Finapres = (1.5 x Dinamap) - 45

and the 95% confidence limits of the coefficients are 1.3-1.7 and 17-73 respectively.

Conclusion. The Finapres and the Dinamap showed substantial disagreement in the measured systolic pressure. The result was far from the y = 1x + 0 regression line that one would have wished. Our suspicion is that the vascular dynamics in these patients are causing the discrepancy. Future studies will determine if the two monitors follow trend changes satisfactorily, if the Finapres can be used to provide an earlier indicator of hypotension, and if it is less annoying to the patient.

References:
1. J Clin Monit 1:17-29, 1985.
2. J Hypertens 11:913-918, 1988.

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DOES MAGNESIUM SULFATE ($MgSO_4$) ALTER THE MATERNAL CARDIOVASCULAR RESPONSE TO VASOPRESSOR AGENTS IN GRAVID EWES? S Sipes, M.D., D Chestnut, M.D., R Vincent, M.D., C Weiner, M.D., Depts of Anesthesia and Ob-Gyn, University of Iowa, Iowa City, IA, 52242

$MgSO_4$ attenuates the compensatory response to hemorrhage in gravid ewes[1] and decreases the hypertensive response to angiotensin II and norepinephrine in gravid rabbits.[2] Some clinicians believe it facilitates the control of preeclampsia-associated hypertension. One possible mechanism of these effects is that magnesium decreases cardiovascular response to endogenous vasopressors. The purpose of this study was to determine whether $MgSO_4$ decreases the response to endogenous and exogenous vasopressors in gravid ewes. The Animal Care Committee approved the protocol. Eight chronically instrumented ewes between 0.8 and 0.9 of gestation received 4 randomized doses of each active vasopressor (phenylephrine, angiotensin II, or vasopressin) and one dose of normal saline (NS)-control in the presence of either a 4 g/hr $MgSO_4$ or NS infusion. Statistical analysis was by repeated measures ANOVA with Scheffe's posthoc test. $P < 0.05$ was significant. $MgSO_4$ infusion increased the mean ± SEM serum Mg concentration to 5.2 ± 0.1 mg/dl. Maternal MAP (MMAP) measurements (mean ± SEM mmHg) for each vasopressor are shown. $MgSO_4$ significantly attenuated the increase in MMAP with each vasopressor.

	Angiotensin II (ug/kg/min)				
	Control	.01	.02	.04	.08
Control MMAP	96(±10)	102(±9)	105(±12)	107(±13)	114(±17)
$MgSO_4$ MMAP	91(±10)	95(±14)	103(±11)	102(±16)	106(±15)
	Phenylephrine (ug/kg/min)				
	Control	1	2	4	8
Control MMAP	94(±8)	100(±8)	102(±9)	110(±14)	119(±21)
$MgSO_4$ MMAP	94(±8)	94(±11)	100(±12)	106(±15)	111(±20)
	Vasopressin (ug/kg/min)				
	Control	.002	.004	.008	.016
Control MMAP	93(±7)	97(±8)	100(±10)	100(±8)	100(±7)
$MgSO_4$ MMAP	86(±8)	89(±9)	94(±8)	95(±8)	97(±9)

MMAP, MHR, CO, uterine blood flow, SVR, and UVR were significantly affected by both vasopressor dose and group assignment ($MgSO_4$ vs. control). FHR was consistently affected by dose and variably affected by group. Conclusion. $MgSO_4$ significantly attenuated the increases in MMAP and SVR, and the decreases in MHR, with each vasopressor. These findings confirm a decreased response to vasopressors in the presence of $MgSO_4$ infusion in the ewe and may support the use of $MgSO_4$ in preeclampsia.

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References. 1. Am J Obstet Gynecol 159:1467, 1988. 2. Am J Obstet Gynecol 149:705, 1984.