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Title : EPINEPHRINE, NOREPINEPHRINE AND HEMODYNAMIC CHANGE DURING SUBARACHNOID BLOCK

Authors : K. Balasaraswathi, M.D., P. Kumar, M.D. & S.N. Glisson, Ph.D.

Affiliation: Department of Anesthesiology, Loyola University Stritch School of Medicine
2160 South First Avenue, Maywood, Illinois 60153

Introduction. Subarachnoid analgesia has survived the assault of modern general anesthetics and muscle relaxants. The special advantage of subarachnoid analgesia for surgical operations is an awake patient with apparent hemodynamic stability. Subarachnoid analgesia has been advocated for surgical procedures of the lower extremity especially in patients with cardiovascular and respiratory diseases.

This study was undertaken to determine to what extent hemodynamic and circulating epinephrine and norepinephrine change accompanies subarachnoid analgesia.

Methods. Eight adult male Beagle dogs were prepared with a femoral artery cannula, thermal dilution Swan-Ganz catheter and a femoral vein catheter cephalad to the adrenal gland outflow. The spinal tap was made between L_3-L_4 and a spinal catheter inserted to the L_1 level. Using the paw-withdrawal response to toe clamping, it was determined in each dog that the sensory level achieved following 0.80 cc of 5% lidocaine in 7½% dextrose was T_8-T_{10}. To eliminate erratic hemodynamic values resulting from body movement we found it necessary to maintain the dogs on enflurane anesthesia during the experiment. A complete hemodynamic profile, and epinephrine and norepinephrine levels were determined during a control period, after a spinal injection of saline and 15, 30, 60 and 120 minutes after the spinal injection of lidocaine.

Results. As shown in the TABLE significant hemodynamic changes occur in conjunction with subarachnoid block. Cardiac output, heart rate and mean arterial blood pressure are all significantly decreased from control values following the lidocaine injection with the greatest changes occurring during the period of maximum analgesia - the first 30 minutes. The level of circulating norepinephrine was found to be significantly lowered during this 30-minute period although the epinephrine levels remained constant. One hour after the lidocaine injection heart rate and mean blood pressure increased, as well as the levels of epinephrine and norepinephrine. Cardiac output remained below the control output. At 120 minutes hemodynamic and catecholamine values returned to baseline.

Discussion. These findings demonstrate peripheral vascular pooling below the level of the spinal block leading to decreased venous return and activation of the Bainbridge reflex - bradycardia, diminished cardiac output and hypotension - as shown by other investigators. This vascular pooling was suggested by Greene to have a distinct relationship with the sympathetic blockade accompanying spinal block. The loss of sympathetic tone with the accompanying peripheral pooling of blood is reflected in the reduced level of circulating norepinephrine observed in this study. Of importance is the finding that the return of heart rate, arterial pressure and subsequently cardiac output to baseline during subarachnoid block coincides with a marked release of catecholamines from the adrenal medulla as indicated by the significant rise in plasma epinephrine and norepinephrine.

Thus, these results offer evidence for the often suggested role of catecholamines on the hemodynamic changes that occur during subarachnoid block.

TABLE

		Lidocaine				
	Saline	15	30	60	120 min post	Mean Values
CI	4.4	3.7*	3.8*	3.8*	4.2	L/min/M^2
SV	15.2	14.5	14.7	13.1*	14.9	mls/beat
HR	135	120*	123*	135	134	b/min
$\bar{x}$BP	80.3	64.5*	69.7*	85.2	82.3	torr
NE	1.05	.67*	.68*	1.26	1.10	ng/ml Plasma
EPI	1.23	1.66	1.98	2.81*	1.28	ng/ml Plasma

* = Statistically different from "sham" saline spinal value $p<.05$ (Paired t)