Title: THE EFFECT OF SPINAL ANESTHESIA ON LOCAL SPINAL BLOOD FLOW AND GLUCOSE UTILIZATION

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INTRODUCTION: Despite extensive clinical experience with spinal anesthesia (SA), understanding of its spinal cord circulatory and metabolic effects is remarkably limited. Therefore, the objective of the present study was to determine whether SA alters local spinal blood flow (LSCBF) or glucose utilization (LSGU).

METHODS: SA was produced with preservative-free 0.75% bupivicaine in conscious male Sprague-Dawley rats and LSCBF or LSGU measured 10 min later with, respectively, the iodo-[^{14}C]antipyrine (IAP)[1] or 2-[^{14}C]deoxyglucose (2-DG)[2] methods. Briefly, 24-72 h prior to experiments a length of PE 10 tubing was inserted through a slit in the cisternal membrane and advanced 8.5 cm into the lumbar subarachnoid space.[3] Animals were observed for neurologic deficits, such as paralysis, spasticity, or abnormal gait, and only normal animals were used subsequently. On the day of experiments, spinally catheterized animals were anesthetized briefly with 1% halothane:70% N_2O for femoral artery and vein cannulation and were then partially restrained with a pelvic plaster cast and allowed at least 3 h to recover. The dose of bupivicaine necessary to produce anesthesia, as assessed by tail-flick latency and tail and hindlimb immobility, was determined in a separate group of animals to be 15 µl (112 µg). SA was induced, therefore, by the spinal administration of 15 µl of 0.75% bupivicaine followed by 10 µl of saline flush. In order to maintain a constant level of anesthesia during the longer 2-DG experiments an additional 10 µl of drug and 10 µl of flush were administered 25 min after the first dose. Control animals received an equal volume of saline intrathecally. Rectal temperature, MABP, arterial blood gases, and pH were monitored. LSGU and LSCBF were measured in separate groups of 6 control and 7 anesthetized rats. The measurement of glucose utilization began with the bolus intravenous injection of 2-DG, 125 µCi/kg, and was continued for 45 min, during which period timed arterial blood samples were taken for plasma glucose and 2-DG determinations. During the 45-sec LSCBF experiments 50-60 µCi of IAP was infused intravenously at an increasing rate and timed arterial blood samples were collected on pre-weighed filter paper discs. Each blood sample was weighed and the concentration of IAP determined by scintillation counting. At the end of flow and metabolism experiments, animals were killed and the lumbar spinal cord removed rapidly (2-3 min) and processed for autoradiography as described previously.[1,2] Optical density measurements of the autoradiographs generated by these procedures were made with the aid of a computerized image-processing system. LSGU and LSCBF were calculated according to the respective operational equations of the methods[1,2] and data were analyzed with a grouped t test.

RESULTS: Differences between physiologic variables were confined to mild hypotension (89±7 vs. 115±5 mmHg in controls, $P < 0.01$) and slight hypocarbia (30±1 vs. 33±1 mmHg in controls, $P < 0.01$) during LSCBF measurement. SA produced a statistically significant 27-34% reduction in the LSCBF of most spinal regions measured (Table). Except in lamina I-III and dorsal white matter, LSGU was reduced 11-21% but the changes reached or approached statistical significance, respectively, only in ventral and lateral white matter and laminae VII ($P = 0.09$) and VIII ($P = 0.06$) (Table).

TABLE

	LSGU ($\mu mol \cdot 100g^{-1} \cdot min^{-1}$)		LSCBF ($ml \cdot 100g^{-1} \cdot min^{-1}$)	
	Control (6)	SA (7)	Control (6)	SA (7)
GRAY MATTER				
Lamina(e) I-III	37 ± 3	36 ± 2	67 ± 4	44 ± 3†
IV-VI	49 ± 3	44 ± 1	101 ± 4	68 ± 5†
VII	57 ± 4	48 ± 2	121 ± 5	86 ± 7†
VIII	57 ± 4	48 ± 2	124 ± 6	83 ± 6†
IX	55 ± 5	46 ± 2	109 ± 5	74 ± 5†
WHITE MATTER				
Dorsal	18 ± 2	17 ± 1	26 ± 1	18 ± 1†
Lateral	31 ± 2	26 ± 1*	37 ± 2	27 ± 3†
Ventral	29 ± 2	23 ± 1*	33 ± 2	24 ± 2†

* $P < 0.05$, † $P < 0.01$. Data represent Mean ± SEM for the number of animals in parentheses.

DISCUSSION: The present study is the first to demonstrate a decrease in spinal metabolism and blood flow during SA. SA produces a profound functional depression of the spinal cord and, considering the well-known relationship in the CNS between functional activity, metabolic rate, and blood flow,[2] it is only surprising that spinal metabolism is not reduced further by SA. Although the reduction in LSCBF during SA probably represents the physiologically appropriate response to reduced metabolic demand, reduced metabolism does not account completely for the decrease in LSCBF. When corrected for differences in arterial PCO_2, however, assuming a 4% change in blood flow per mmHg change in PCO_2,[4] the percent reductions in LSCBF during SA approximate more closely those in LSGU. Regional differences in the LSGU and LSCBF effects persist in laminae I-III and dorsal white matter, however, suggesting the possibility that SA alters regionally the CO_2 sensitivity and/or autoregulatory capability of the spinal circulation. Nevertheless, the excellent safety record of SA indicates that any changes in these homeostatic mechanisms must be small and well-tolerated.

References

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(This study was supported by NIH Grant GM 30502.)