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A New Look at Sympathetic Denervation during Spinal Anesthesia

CARDIOVASCULAR EFFECTS OF spinal anesthesia, by far the most prominent physiologic responses to this form of anesthesia, are simply and solely the result of preganglionic sympathetic blockade produced by the local anesthetic injected into the subarachnoid space. For almost 30 years it has been recognized that the level of sympathetic denervation lies above, cephalad to, the level of sensory blockade. Using loss of the ability to appreciate the sensation of cold as an indirect indication of the level of sympathetic denervation, the zone of differential sympathetic blockade averages two spinal segments during hyperbaric tetracaine spinal anesthesia, although, in some patients, it may be as great as six spinal segments.[1] The presence of this zone of differential sympathetic blockade has been ascribed to a combination of the fact that the concentration of local anesthetic decreases as a function of distance from the site of injection[2] and the fact that sympathetic preganglionic fibers are more sensitive to the effects of local anesthetics and, thus, are blocked by concentrations of local anesthetics too low to block somatic sensory fibers.

The concept that the zone of differential sympathetic blockade averages approximately two spinal segments has been accepted because the indirect measurement of the presence or absence of sympathetic activity based on loss of temperature discrimination agrees with what is known of concentration gradients of local anesthetics in cerebrospinal fluid during spinal anesthesia.[2–7] It has also been accepted because of clinical observation of the level of sensory anesthesia necessary for development of either Horner's syndrome or cutaneous vasodilation in upper extremities, or both, during high spinal anesthesia. More direct evidence of the level of sympathetic denervation of the trunk under clinical conditions during spinal anesthesia by using changes in cutaneous temperature, routinely and reliably measured in the skin of the head and the extremities, has long been thwarted by the extraordinary difficulty in the accurate measurement of cutaneous temperature on the trunk under clinical conditions as they exist in an operating room. On the other hand, ethical considerations make it essentially impossible to give nonoperative spinal anesthetics under environmentally rigidly controlled laboratory conditions to numbers of volunteers adequate to result in statistically meaningful data. The problem of accurate measurement of truncal skin temperature in normal patients under clinical conditions has, however, apparently been resolved, as reported by Chamberlain and Chamberlain in the present issue of this journal.[8]

Using thermographic imagery, a technique able to quantitate small changes in skin temperature, Chamberlain and Chamberlain confirm that a zone of differential sympathetic blockade exists during hyperbaric lidocaine or tetracaine spinal anesthesia as reflected by an increase in skin temperature associated with sympathetic denervation. They also show, however, that the zone of differential sympathetic blockade averages, not two spinal segments, but rather, six or seven spinal segments. This is a most remarkable finding. If confirmed, it will require radical alteration in our concepts of the causes and management of changes in cardiovascular function during spinal anesthesia.

Such an extensive zone of differential sympathetic denervation is a finding so unexpected as to be mind boggling. It is unexpected because it flies in the face of, as

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mentioned earlier, what we know about the concentrations of local anesthetics in cerebrospinal fluid during spinal anesthesia. Is it possible for a concentration of lidocaine in spinal fluid to exist at a level sufficiently high to impair sympathetic activity at T-1 when the concentration of lidocaine in spinal fluid is adequate to block sensory efferent fibers only to T-11 or T-10? As the authors point out, preganglionic sympahetic fibers ascend and descend in the paravertebral sympathetic chain after exiting through the dura to synapse with postganglionic sympathetic fibers above and below their segmental points of origin. Peripheral responses to preganglionic stimulation (or blockade) are thus diffuse, extending above and below the spinal segmental level of stimulation (or blockade). The evidence in humans, however, is that efferent impulses generated by stimulation of one preganglionic fiber ascend and descend only three spinal segments.[9] This would hardly be adequate to explain the Chamberlains' finding of sympathetic blockade at T-1, with a sensory level of T-10 or T-11, even given a two-segment zone of differential sympathetic blockade to T-8 or T-9. Along the same lines, the clinical observations mentioned earlier with regard to the level of sensory blockade present when Horner's syndrome or upper extremity cutaneous vasodilation occurs, and when changes in blood pressure and pulse rate occur, do not support the existence of zones of differential spinal anesthesia as extensive as those reported by Chamberlain and Chamberlain.

Also worrisome about the data in the Chamberlains' article is the fact that 18 of the 20 patients studied had impaired sympathetic activity to T-1, the level at which the highest preganglionic sympathetic fibers arise from the spiral cord. One of the remaining two patients had sympathetic denervation (temperature level) to T-2 (with a sensory level of T-10). Is clinical spinal anesthesia with sensory levels of T11–T5 really associated in clinical practice with T-1 or T-2 levels of sympathetic denervation 95% of the time? In only one of 20 patients in the Chamberlains' series was the level of sympathetic impairment below T-2. That one patient had a T-6 sensory level and a T-5 temperature (*i.e.*, sympathetic) level.

Certainly the thermographic imagery technique used by Chamberlain and Chamberlain accurately measures changes in cutaneous temperature, including on the trunk, in patients having spinal anesthesia. But has this study ruled out the possibility, however remote, that changes in temperature of the skin of the trunk were due to factors other than sympathetic denervation alone? What would the results have been if a double-blind study had been carried out using naive, unpremedicated patients (not informed volunteers) brought to the operating room and handled *exactly* as the patients in the present study, including: 1) positioning on the operating table under operating room lights; 2) performing lumbar puncture with exactly the same technique used in the patients in the present study but injecting a (double-blind) placebo instead of a local anesthetic solution; and then 3) exposing for 30 min these undraped control patients in the supine position, still under operating room lights, while the truncal cutaneous temperatures were measured? Would subjects not given a spinal anesthesia also have had increases in temperature similar to those who had spinal anesthesia? Similarly, what happens to cutaneous temperature on the trunk during saddle spinal anesthesia with a sensory level of L5? The Chamberlains' data seem to suggest that regardless of the sensory level of anesthesia, no matter how low, there might still be an increase in skin temperature to T-1 or T-2. If this is true with saddle block to L-5, what, then, do the data in the present report mean? Chamberlain and Chamberlain may well be correct in showing us that the extent of sympathetic denervation during spinal anesthesia is substantially greater than ever imagined. We owe them thanks for presenting us with such a new, different concept. We also owe them thanks for providing a potent impetus for further studies in which control patients are included.

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