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catheter passage into the subarachnoid space and possibly may provide more cranially directed catheters.* Yurino *et al.* described a preformed, coiled-tipped microcatheter that could easily be placed in the subarachnoid space within 2 min and provide adequate blocks in 94% of patients, suggesting that these catheters remained at the level of the puncture site or took a cranial direction.⁴ In one study we investigated the impact of different needle designs, directional and nondirectional, on the intrathecal catheter tip position.⁵ The use of a nondirectional Quincke needle was associated with a 35% incidence of caudad catheter tip positions, and the directional Sprotte needle provided no caudally positioned tips of the 28-gauge catheters.

The results of the three studies suggest that technical modifications can help to increase the percentage of spinal catheters with the intended cranial tip position.

Second, we would like to stress the different injection speeds of micro- and macrocatheters. The fact that maldistribution was not significantly related to the baricity of the applied LA in Biboulet's study may give the impression that hyperbaric LAs can be used in combination with microspinal catheters in the same way.

Rigler and Drasner clearly demonstrated in 1991 that a low injection speed produces inadequate distribution of dye-colored hyperbaric lidocaine.⁶ The mean injection time of 1.0 ml is 50 s for 32-gauge and 28 s for 28-gauge microcatheters compared with 3 s for 20-gauge, large-bore catheters.

Therefore, the reduced injection speed through microcatheters appears to be an additional factor, besides the orientation of the catheter tip, which may significantly enhance the risk for maldistribution of hyperbaric LA. The combination of the reduced injection speed through microcatheters, high doses of hyperbaric LA, and caudally oriented catheter tips have produced significant maldistribution in models of the spinal canal and probably have caused the well-known cauda equina syndrome as a result of toxic concentrations of the hyperbaric LA at the dorsal sacral spinal nerves.⁷⁻⁹

Although the results of the spinal canal models cannot completely be transferred on patients in whom lower concentrations of hyperbaric LA may occur because of diffusion and vascular uptake, and although neurologic sequelae have also been reported after macrocatheter CSA, the conclusion in Biboulet's article that "hyperbaric solutions do not appear to be a clinical factor in the development of limited block" should be taken with caution. In our opinion, this conclusion is only justified for the use of large-bore catheters and must not be transferred to microcatheter CSA.

* Ata S, Shulman MS: Causes for the difficulty with placement of continuous subarachnoid catheters. *ANESTHESIOLOGY* 1991; 75:A1092

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In Reply:—Dr. Standl is right in highlighting the fact that several clinical studies have described techniques destined to decrease the incidence of caudally directed spinal catheters. However, it is important to note that these studies were performed when the role of the catheter's sacral direction in the occurrence of maldistribution was but an experimental hypothesis.^{1,2} The point of our work was to objectively identify the clinical causes of maldistribution. Using 19-gauge, end-holed catheters, the study showed that the caudal orientation of

As a consequence we would like to recommend techniques that facilitate cranial catheter tip placement and advise against the combination of microcatheters and hyperbaric LA for CSA. In light of these aspects we agree with the statement of the authors that we should not discourage the use of microcatheters for CSA.

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the catheter tip is a factor of maldistribution rather than the caudal direction of the catheter. As such, the sacral flow of local anesthetics seems to be the most important factor of maldistribution; a cranially directed catheter can have a distally oriented catheter tip if a loop is created during catheter insertion, leading to a distal flow of local anesthetics.

Second, the role of injection speed, lower when local anesthetics are administered *via* microcatheters and experimentally evoked as being

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associated with the maldistribution of hyperbaric solutions,¹ has already been debated in the literature.³⁻⁵ According to Wendell and Cianci³ and Erian,⁴ neither the catheter diameter nor the baricity of the injected solution was a factor of maldistribution. However, once again, these results were derived from experimental models. Using 19-gauge catheters, we demonstrated that maldistribution did not occur more often with either isobaric or hyperbaric bupivacaine. Nevertheless, the comparison has not been clinically studied using microcatheters. As such, I find it difficult to advise against the use of hyperbaric solutions *via* microcatheters before clinical evaluation. In one study, although retrospective, the required doses of hyperbaric lidocaine, 5%, administered *via* microcatheters were not greater than those using macrocatheters.⁶ Finally, Horlocker *et al.* reported, also in a retrospective study, that the incidence of inadequate anesthesia was no greater when using microcatheters rather than macrocatheters.⁷ As such, in light of these experimental^{3,4} and clinical results,^{6,7} we cannot conclude that microcatheters and hyperbaric solutions are factors of maldistribution. The only current, clinically demonstrated factor of maldistribution is the caudal orientation of the catheter tip.⁸

It is important to note, however, as highlighted in our manuscript, that the danger of maldistribution does not lie in its occurrence but rather in its not being diagnosed, leading to the administration of high doses of potentially neurotoxic local anesthetics. The diagnosis and early management of maldistribution, as well as abandoning the administration of high doses of local anesthetics (lidocaine, 5%), should limit the occurrence of cauda equina syndrome after continuous spinal anesthesia.

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Intrathecal Sufentanil Produces Sensory Changes without Hypotension in Male Volunteers

To the Editor:—The article by Riley *et al.* regarding sensory changes after intrathecal sufentanil was well written, detailed, and informative. The authors stated that the basis for the neuroselectivity of the different stimulus frequencies used in the CPT evaluation performed by the Neurometer® CPT device (Neurotron, Inc., Baltimore, MD) was "theoretical and unsubstantiated." Unfortunately, the authors must have been unaware of the significant number of peer-reviewed studies that have been published during the past 10 years, establishing the neuroselectivity of the CPT stimuli.^{1,2} These studies include, but are not limited to, comparison with other neurodiagnostic tests,³ peripheral nerve demonstrations of neuroselectivity,⁴ and spinal cord demonstrations of neuroselectivity.⁵ In fact, there have been more than 190 articles published in peer-reviewed journals using and validating the clinical use, reproducibility, and sensitivity of the CPT evaluation.

Apparently the only statistically significant change detected in

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CPTs before and after intrathecal administration of sufentanil was at 250 Hz at the knee. I agree with their point in the discussion section that there should have been a greater effect at 5 Hz. The reason for this discrepancy could be the way the data were analyzed. CPT values before and after intervention should always be expressed as a percent change as opposed to change in intensity (mA) because the amount of charge delivered is different for a 5-Hz *versus* 2,000-Hz sine wave stimulus. For instance, a 1-mA, 5-Hz sine wave stimulus delivers approximately $\times 400$ the charge (coulombs) as a 1-mA, 2,000-Hz sine wave stimulus. Therefore, a 10-CPT unit (100 μ A) change at 5 Hz results in approximately $\times 400$ greater difference in charge delivery than a 10-CPT unit change at 2,000 Hz. Perhaps looking at the data as a percent change before and after sufentanil administration would reveal a significant effect at 5 Hz.