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Quantitative Assessment of Differential Sensory Nerve Block after Lidocaine Spinal Anesthesia

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Background: **Recent technology allows for quantitative and selective measurement of Aβ, Aδ, and C fiber nerve transmission. To gain further insight into the physiology of differential block after lidocaine spinal anesthesia, the function of these different fibers was quantitatively measured over time, and these measurements were correlated with regression of anesthesia to pinprick, touch, cold, and tolerance of tetanic electrical current (equivalent to surgical incision).**

Methods: **Six volunteers received lidocaine spinal anesthesia with 50 mg lidocaine (5% in dextrose). Cutaneous current perception thresholds at 2,000, 250, and 5 Hz, which stimulate Aβ, Aδ, and C fibers, respectively, were determined at L2–L3 (medial aspect above knee) before and every 10 min after spinal anesthesia. Dermatomal levels to pinprick, touch, and cold were assessed every 5 min after spinal anesthesia. Tolerance to tetanic electrical stimulus was assessed at L2–L3 every 10 min after spinal anesthesia.**

Results: **Differential block was demonstrated by the sequential return of sensation to touch, pinprick, and cold at L2–L3. Recovery of function of Aβ, Aδ, and C fibers correlated with return of sensation to touch ($R^2 = 0.7$, $p = 0.03$), pinprick ($R^2 = 0.75$, $p = 0.02$), and cold ($R^2 = 0.67$, $p = 0.04$) respectively. Loss of tolerance of surgical anesthesia corresponded to return of Aβ current perception thresholds to baseline, whereas current perception thresholds for Aδ and C fibers were still increased to greater than baseline ($p = 0.025$).**

Conclusions: **Differential sensory block during spinal anesthesia is due to different recovery profiles of Aβ, Aδ, and C fibers. Return of Aβ current perception thresholds to baseline correlated with duration of surgical anesthesia as assessed with an electrical stimulation model. (Key words: Anesthetic techniques; spinal: current perception threshold; differential nerve block; quantitative sensory testing; tetanic stimulation.)**

SPINAL anesthesia results in temporal and spatial zones of differential sensory block.[1] Although differential block theoretically occurs because of varying degrees of block of Aβ, Aδ, and C fibers, little direct evidence exists to support this theory. Previous studies examining differential sensory blockade have used crude measurements, such as pinprick, touch, and cold, that do not directly measure function of different types of nerve fibers.[2,3] In addition, the relative importance of blockade of each class of fiber (Aβ, Aδ, C) for surgical anesthesia is unclear.

Recent technologic advances allow for direct, quantitative measurement of Aβ, Aδ, and C fibers *via* cutaneous current perception thresholds (CPTs).[4,‖] This technology has been validated by endocrinologists, neurologists, and neurosurgeons as a useful tool in the clinical evaluation and management of a variety of neurologic disorders.[4–6] To gain insight into mechanisms of differential sensory nerve blockade and surgical anesthesia, we designed this study to quantitatively measure blockade of Aβ, Aδ, and C fibers after lidocaine spinal anesthesia and compared these measurements with regression of pinprick, touch, cold, and simulated surgical incision.

Materials and Methods

After Institutional Review Board approval and informed consent were obtained, six unmedicated, ASA physical status 1 volunteers were studied. Spinal anesthesia was induced with the subject in the left lateral decubitus position with 50 mg 5% lidocaine in 7.5% dextrose at the L2–L3 interspace with a 25-G Whitacre needle. After injection of spinal solution, subjects immediately were turned supine and remained level for the duration of the study.

CPT testing (Neurometer CPT, Neurotron, Baltimore, MD) was measured at 5, 250, and 2,000 Hz. The CPT is the minimum amount of cutaneously applied current that an individual consciously perceives 50% of the time. The testing procedure determines the range of current for each frequency such that the upper end of the range is always felt and the lower end is never felt.

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‖ Dent MT, Ward JD, Ryder REJ: Testing for diabetic neuropathy: Part 1. Somatic nerve function current perception thresholds. Practical Diabetes 9:24–28, 1992.

Thus, at each frequency, the current is slowly increased (to a maximum of 9.99 mA) until the subject reports sensation. The current is then terminated, decreased by 0.08 mA, and reapplied. This sequence is repeated, with increasing and decreasing adjustments in current, until a bracket is found where the upper end of the current range is always felt and the lower current intensity is never felt. The middle of this 0.08 mA bracket is reported as the CPT. The 5-Hz CPT stimulus has been shown to correlate with C fiber function as measured with quantitative thermal threshold measures and pain sensation, and the 250 and 2,000 Hz CPT stimuli have been shown to correlate with Aδ and Aβ fiber function as measured with sensory nerve conduction and quantitative vibratory evaluations.[4,7] Approximately 5 min are needed to test all three frequencies at a given site; therefore, only the right L2–L3 dermatome (medial aspect above knee) was tested and only temporal differential sensory blockade was examined. The rapid and dynamic onset of sensory blockade during spinal anesthesia does not allow accurate measurement of CPTs during initiation of spinal anesthesia; thus, CPTs were measured before anesthesia (baseline), 30 min after initiation of anesthesia, and then every 10 min until CPTs returned to within 10% of baseline. CPTs were also measured before anesthesia and 30 min after initiation of anesthesia at a site beyond neural blockade (C2 dermatome) to verify the lack of a systemic effect from the spinal anesthetic.

Dermatomal levels to pinprick (18-G needle), touch (self-assessed), and cold (ice-cold test tube) were assessed every 10 min after injection of the spinal anesthetic. A tetanic electrical stimulus (60 mA, 50 Hz for 5 s) was administered to the right L2–L3 dermatome every 10 min after injection of spinal anesthetic. Testing began with 10 mA, and the current was increased by 10-mA increments to a maximum of 60 mA for 5 s. The maximal stimulus has been demonstrated to be equivalent to surgical incision.[8]

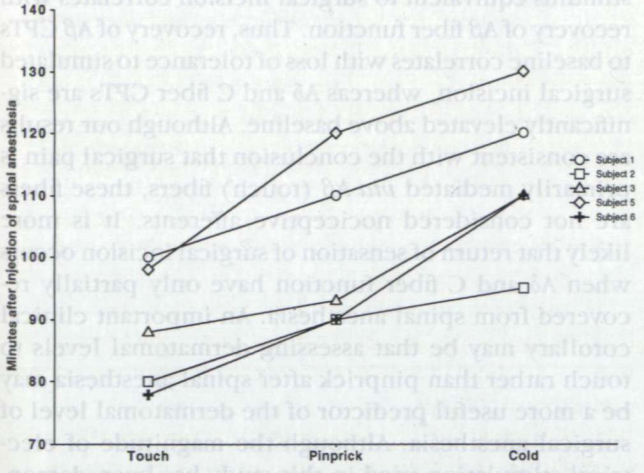

Fig. 1. Time until return of sensation of touch, pinprick, and cold at the L2–L3 dermatome after lidocaine spinal anesthesia.

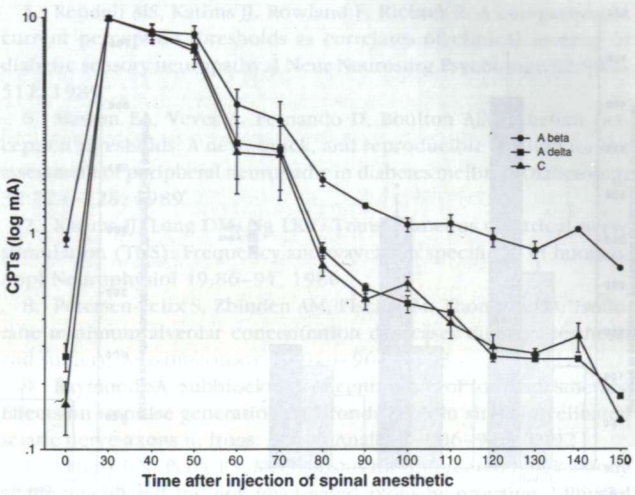

Fig. 2. Regression of current perception thresholds (CPTs) at the L2–L3 dermatome after lidocaine spinal anesthesia. Mean and standard deviation displayed.

Data Analysis

Differential sensory nerve block was analyzed with paired *t* test and correlated with regression of sensation of touch, pinprick, and cold. Fisher's r-to-z transformation was used to determine significant correlations. A $P < 0.05$ was defined as significant.

Results

Spinal lidocaine consistently produced temporal differential sensory nerve blockade, as evidenced by the initial return of sensation to touch, then pinprick, and finally cold in all subjects (fig. 1). Function of Aβ, Aδ, and C fibers was markedly blocked by lidocaine spinal anesthesia, and recovery of function of each fiber type paralleled the others (fig. 2). Return to baseline of CPT for Aβ fibers correlated with return of sensation of touch ($R^2 = 0.7$, $P = 0.03$), whereas Aδ and C fiber CPTs were significantly increased above baseline ($P < 0.05$, fig. 3A). Return of CPT for Aδ fibers correlated with return of sensation of pinprick ($R^2 = 0.75$, $P = 0.02$),

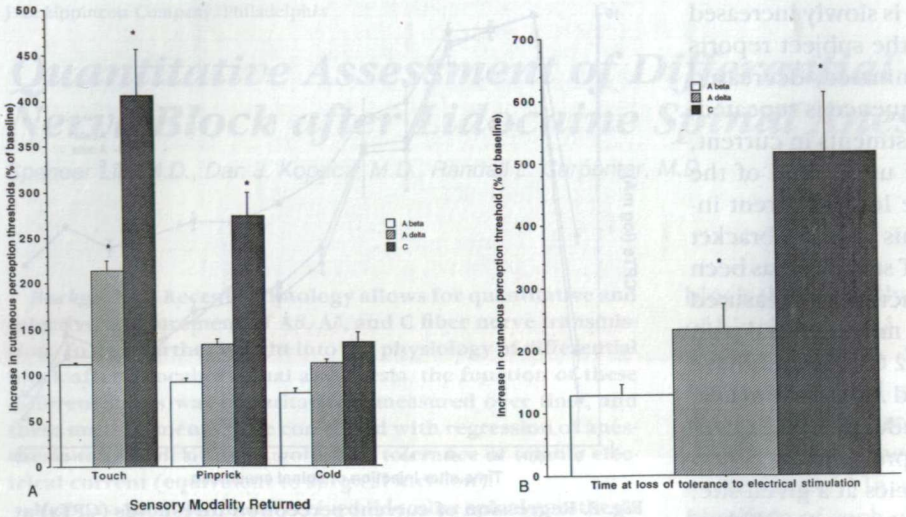

Fig. 3. (*A*) Sequential return of nerve function by class of fiber correlates with sequential return of sensation at the L2–L3 dermatome after lidocaine spinal anesthesia. Mean and standard deviation displayed. *Different from baseline ($P < 0.05$). (*B*) Relative function of each nerve fiber type at time of loss of tolerance to electrical stimulus. Mean and standard deviation displayed. *Different from baseline ($P = 0.025$).

whereas C fiber CPTs were significantly increased above baseline ($P < 0.05$, fig. 3A). Return of CPT for C fibers correlated with return of sensation of cold ($R^2 = 0.67$, $P < 0.05$) and were the last fiber type to have CPTs return to baseline (fig. 3A). Loss of tolerance to electrical stimulation corresponded to return of Aβ CPTs to baseline, whereas CPTs for Aδ and C fibers were increased to greater than baseline ($P = 0.03$; fig. 3B). CPTs measured at C2 were unchanged between baseline and 30 min after initiation of anesthesia ($P > 0.3$).

Discussion

Temporal differential sensory blockade frequently is observed during spinal anesthesia but is not easily quantitated. Our results (fig. 2) agree with previous studies demonstrating sequential return of sensation of touch, pinprick, and cold after spinal anesthesia.[2] However, we add new information by directly demonstrating through quantitative measurement that recovery of function of Aβ, Aδ, and C fibers correlate with return of sensations of touch, pinprick, and cold, respectively (fig. 3A). This finding is also consistent with previous studies suggesting that Aβ fibers conduct touch whereas Aδ and C fibers conduct pinprick and cold.[1] Thus, our data confirm the hypothesis that temporal differential block is due to distinct recovery profiles of the different fiber types from spinal anesthesia.

Our results do not allow further insight into the mechanisms of different recovery profiles of Aβ, Aδ, and C fibers, which may be due to physical and electrophysiologic differences between fiber types. Axon diameters and degree of myelination vary considerably between fibers (Aβ > Aδ > C), yet neither diameter nor myelination appear to be important factors determining differential block.[1,9] On the other hand, conduction safety[10] and conduction velocity also differ (Aβ > Aδ > C), and these factors may be important for differential neural blockade.[9,11] In addition, differences in state-dependent sodium channel blockade[12] and frequency-dependent blockade[13] between fiber types may explain the mechanism of differential sensory blockade.

We also attempted to determine the relative importance of blockade of each class of fiber to provision of surgical anesthesia *via* an electrical stimulation model. Our study indicates that loss of tolerance to an electrical stimulus equivalent to surgical incision correlates with recovery of Aβ fiber function. Thus, recovery of Aβ CPTs to baseline correlates with loss of tolerance to simulated surgical incision, whereas Aδ and C fiber CPTs are significantly elevated above baseline. Although our results are consistent with the conclusion that surgical pain is primarily mediated *via* Aβ (touch) fibers, these fibers are not considered nociceptive afferents. It is more likely that return of sensation of surgical incision occurs when Aδ and C fiber function have only partially recovered from spinal anesthesia. An important clinical corollary may be that assessing dermatomal levels to touch rather than pinprick after spinal anesthesia may be a more useful predictor of the dermatomal level of surgical anesthesia. Although the magnitude of electrical stimulation used in this study has been demonstrated to be equivalent to surgical incision in studies

of volatile anesthetics, this has not been verified in studies examining spinal anesthesia. Further clinical studies may be warranted to examine whether sensation of touch is a useful predictor of surgical anesthesia during spinal anesthesia.

In conclusion, this is the first study to use CPT technology to assess differential sensory nerve block. Our data demonstrate that temporal differential sensory block after spinal anesthesia occurs because of differential recovery of $A\beta$, $A\delta$, and C fibers and also suggest that duration of surgical anesthesia may correlate with duration until recovery of sensation of touch.

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