Spinal cord pathways mediating somatosensory evoked potentials

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Using a CO₂ laser, discrete thoracic spinal cord lesions were made in cats anesthetized with ketamine and xylazine (Rompun). Differences in cortical somatosensory evoked potentials (SEP's) produced with high-intensity stimulation (20 times the motor threshold) of each posterior tibial nerve were determined for nine different combinations of unilateral spinal cord lesions.

The results of these studies show that nerve fibers in the ipsilateral dorsal column, the ipsilateral dorsal spinocerebellar tract, and the contralateral ventrolateral tracts with respect to the side of leg stimulation, contribute to cortical SEP's. A lesion of the dorsal spinocerebellar tract affected only the early waves (< 30 msec) of the SEP from leg stimulation ipsilateral to the side of the lesion, whereas a solitary lesion of the ventrolateral tract caused changes primarily in the amplitude of later waves (> 30 msec) of the SEP produced by contralateral leg stimulation. Lesions involving one-half of the dorsal column caused changes in the amplitude of both the early and late waves produced by stimulation ipsilateral to the side of the lesion. The effects of various combinations of lesions on the cortical SEP's were not additive, which indicates significant interaction between afferent pathways.

These findings suggest that high-intensity peripheral nerve stimulation, which activates both C and A fibers, could be used intraoperatively to assess spinal cord function with more accuracy than the current practice of using a stimulus strength of twice the motor threshold. The importance of using anesthetic agents that do not depress cortical activity (which may affect the later components of the SEP) is also emphasized.

KEY WORDS somatosensory evoked potentials dorsal spinocerebellar tract dorsal column system ventrolateral tract spinal cord injury carbon dioxide laser

Despite a great deal of experimental and clinical research, a controversy exists concerning which spinal pathways mediate cortical somatosensory evoked potentials (SEP's). Several investigators have concluded that the dorsal and dorsolateral columns contain the pathways that mediate the cortical SEP's. Early clinical investigations into SEP's in patients with dissociated sensory loss have shown that the integrity of the dorsal columns must be maintained for transmission of the cortical SEP's after stimulation of peripheral nerves. However, other researchers have demonstrated that impulses transmitted along the ventrolateral fiber tracts (spinothalamic projections) contribute to cortical SEP's.

Discrepancies between the results of different experimental studies have been caused by barbiturates (which eliminate the later components of the SEP) that were used for anesthesia; by the use of either low-intensity peripheral nerve stimulation, which only activates large-diameter sensory fibers (Group I and Group II fibers), or high-intensity stimulation, which activates all nerve fibers; and by the use of techniques that produce imprecisely placed and diffuse lesions. We have addressed each of these problems in the study reported here.

To insure evaluation of the later wave components of the cortical potential, cats used in this study were anesthetized with ketamine and xylazine (Rompun). Only high-intensity peripheral nerve stimulation was used, which allowed evaluation of input from all afferent fibers. A microscopic laser was used with a
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“no touch” technique to produce discrete spinal cord lesions.

Materials and Methods

Fourteen adult mongrel cats were anesthetized with 10 mg/kg of ketamine HCl and 0.5 mg/kg of xylazine given subcutaneously. Femoral arterial and venous catheters were inserted. Cats were intubated, paralyzed with gallamine (Flaxedil), and ventilated. The mean arterial pressure was maintained above 100 mm Hg by intravenous injection of normal saline as needed. Body temperature, measured with a rectal core probe,* was kept between 36° and 37° C with an external heat lamp. The tidal volume and respiratory rate were adjusted to maintain the arterial pO2 greater than 90 mm Hg, the pCO2 between 35 and 45 mm Hg, and the pH between 7.30 and 7.45 on room air.

Operative Technique

With the aid of an operating microscope,† a wide four-level laminectomy was made from T-6 to T-9 using microrongeurs and a dental drill. The pedicle(s) were removed with the drill from the respective vertebra(e), at which levels ventrolateral fiber tract lesions were to be made. Great care was taken to preserve the integrity of the epidural veins, because injury of these veins would require that the epidural space be packed with thrombogenic material that would deform the dural sac and produce changes in the SEP’s. The SEP recordings were made before and after laminectomy; if SEP’s changed after laminectomy, the cats involved were excluded from the study.

The dura was opened, and selective lesions were made in the cord using a CO2 laser‡ attached to the operating microscope. The diameter of the laser beam on the tissue surface was 450 μ (0.45 mm). A power output of between 6 and 12 watts was used for making lesions with short repetitive “bursts” of the laser onto the selected area of the spinal cord. To minimize thermal injury to the cord, the area surrounding the lesion site was irrigated with normal saline.

Lesions

Three of the 14 cats had solitary unilateral lesions created at T-8: one on the dorsal column system (DCS), one on the dorsal spinocerebellar tract (DSce), and one on the ventrolateral fiber tracts (VL). In the 11 other cats, sequential lesions were created at T-8 and T-6 that yielded various combinations of lesions involving the DCS, DSce, and VL of each half of the spinal cord. Nine different combinations of lesions were evaluated with SEP’s (Fig. 1).

Dorsal Column System. In three cats, one-half of the DCS was ablated at T-8. In two of these, second lesions that involved the contralateral ventrolateral fiber tract (cVL) in one cat and the ipsilateral dorsal spinocerebellar tract (iDSce) in the other cat at the T-6 level were created at a second procedure.

Dorsal Spinocerebellar Tract. Lesions of the DSce at T-8 were created in three cats. Second lesions at T-6 were created at either the iDCS or the cVL in two cats, respectively.

Dorsal Column and Ipsilateral Spinocerebellar Tract. Three cats were evaluated for lesions created on one side of the DCS and the DSce. Two of these cats had undergone sequential lesioning of the DCS and DSce, as mentioned above. The third cat had both fiber tracts sectioned, with the initial lesion at T-8. A second lesion was then made at T-6 that interrupted fibers in the cVL.

Ventrolateral Fiber Tracts. In seven cats, unilateral lesions of the fibers in the VL at T-8 were created first, after which six cats had a second lesion created at T-6: cDCS in one, iDCS and iDSce in one, cVL in two, and cDCS and cDSce in two.

Combinations of Lesions. The creation of lesions described above produced the following combinations that were evaluated: DCS and cVL in two cats; DSce and cVL in one; DCS, iDSce, and cVL in one; DCS, iDSce, and iVL (hemisection) in one; and bilateral VL in two cats.

Recording of Somatosensory Evoked Potentials

The SEP’s were obtained by stimulating each posterior tibial nerve. Platinum pin electrodes§ were placed into the leg percutaneously. The stimulating cathode for each leg was inserted into the popliteal fossa, and the anode was placed into the calf 2.0 cm distal to the cathode. Each leg was grounded proximally. A square-wave stimulus pulse of 300-μsec duration and 20-μsec delay was delivered at a rate of 7

* Rectal core probe manufactured by Omega Engineering, Inc., 1 Omega Drive, Stamford, Connecticut.
† Opmi-1 operative microscope manufactured by Carl Zeiss, Inc., Carson Optical Instruments, Oakland, California.
§ Electrodes manufactured by Grass Instruments, Inc., 101 Old Colony Avenue, Quincy, Massachusetts.
Spinal Cord Lesion | #Cats Evaluated | Spinal Cord Lesion | #Cats Evaluated
--- | --- | --- | ---
DCS | 3 | DSCe and cVL | 1
DSCe | 3 | DCS, iDSCe and iVL | 1
DCS and iDSCe | 3 | DCS, iDSCe and cVL | 3
VL | 7 | VL and cVL | 2
DCS and cVL | 2

Fig. 1. Combinations of spinal cord lesions studied with SEP's from posterior tibial nerve stimulation. See Definitions of Abbreviations table.

pulses/sec by a Grass S88 stimulator with a constant voltage isolation unit. The voltage was adjusted to 20 times that required to produce a muscle twitch in the foot before paralysis (range 35 to 40 volts); the SEP response is maximal at this level of stimulation (Fig. 2).

The SEP's were recorded from the scalp and upper lumbar area before and after laminectomy, after durotomy, and after lesioning. To monitor the consistency of the amplitude and the latency of the incoming SEP's, recordings were made between L-1 and L-2. For scalp recordings, an active electrode was placed percutaneously in contact with the midsagittal skull 2 mm behind the coronal suture, and a reference electrode was laced subdermally in the nasal skin. Potentials were amplified by 10,000 through a bandpass filter of 30 to 3.0 Hz.† Electrode impedances of less than 3 kilo-ohms were maintained. A high-speed signal averager‡ was used to automatically average 1024 signal sweeps, and the results were displayed on an oscilloscope; the sweep time was 123 msec. To verify the reproducibility of the waveforms, SEP's were recorded three times. The summed potentials were then plotted on graph paper with a Hewlett-Packard x-y plotter.‡ Wave latencies and amplitudes were calculated using cursors incorporated into the signal averager.

Histopathological Studies

After the completion of each study, cats were sacrificed with a bolus of intravenous KCl. The segment of spinal cord from T-6 to T-9 was removed, fixed in 10% formalin, and embedded in paraffin. Serial sections (8 μ thick) were taken from the respective lesions and stained with hematoxylin and eosin. Transverse dimensions of each lesion were evaluated by projecting transparencies of the lesional section onto a photograph of a normal section of spinal cord at the same level. Drawings of the lesions were made by summing the projected areas of involvement. The SEP's were then correlated with the fiber tracts traversing the areas of injury.

Results

With posterior tibial nerve stimulation, the cortical SEP typically has four or five negative waves within 100 msec of the onset of stimulation (Fig. 3). The early components of the SEP, which probably represent transmission of the potential through the brain stem and thalamic projections, are composed of a

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§ Model S88 stimulator and Model SIU 4678 isolation unit manufactured by Grass Instruments, Inc., 101 Old Colony Avenue, Quincy, Massachusetts.

* Model HGA-200A amplifier and Model 501A amplifier/filter manufactured by Nicolet Instruments Corp., 5225 Verona Road, Madison, Wisconsin.

† Signal averager, Model 1170, manufactured by Nicolet Instruments Corp., 5225 Verona Road, Madison, Wisconsin.

‡ X-y plotter, Model 7010B, manufactured by Hewlett-Packard Corp., 1820 Embarcadero Road, Palo Alto, California.
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FIG. 2. Effect of stimulus intensity on cortical SEP (posterior tibial nerve stimulated).

small negative wave (N₁) at 12 msec, a large positive wave (P₁) at 18 msec, and a large negative wave (N₂) at 25 msec. The later components of the SEP, N₃ to N₅, are cortical potentials and are suppressed by barbiturates (Fig. 4).

Because we evaluated the cortical SEP’s from stimulation of each posterior tibial nerve, the results are discussed in terms of wave amplitude and latency changes for the side of the leg that was stimulated with respect to the side of the dorsal spinal cord lesion. The following terminology is used: iP₁N₂ means the P₁N₂ wave complex of the cortical SEP from stimulation of the posterior tibial nerve that is ipsilateral to the side of the dorsal spinal cord lesion(s), cP₁N₂ means the P₁N₂ wave complex of the cortical SEP from stimulation of the posterior tibial nerve contralateral to the side of the dorsal lesion(s), and iN₄ and cN₄ indicate the N₄ wave from stimulation ipsilateral and contralateral to the side of the dorsal spinal cord lesion(s), respectively. Values for the components of the SEP’s recorded for all nine combinations of lesions are listed in Table 1, and Figs. 5 to 11 are drawings that show locations of and representative recordings for the lesions.

Dorsal Column System Lesions

Three cats were evaluated for unilateral DCS lesions. Prominent changes were seen in the early components of the cortical SEP. The normal N₁P₁N₂

<table>
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<tr>
<th>Location of Lesion</th>
<th>iP₁N₂</th>
<th>cP₁N₂</th>
<th>iN₄</th>
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<td>DCS</td>
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<td>—</td>
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<td>DSce</td>
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<td>VL</td>
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<td>severe</td>
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<td>DCS and cVL</td>
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<td>severe</td>
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<td>severe</td>
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<td>VL and cVL</td>
<td>severe</td>
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* Severe = greater than 40% decrease; moderate = 16% to 40% decrease; mild = less than 15% decrease; — = no change. See Definition of Abbreviations table.
complex of the cortical SEP from stimulation of the posterior tibial nerve ipsilateral to the lesion was replaced by a single broad negative wave with a peak at 19.6 msec ± 3.7 msec (standard deviation). The amplitude of this negative wave was 26.6 ± 6.5% of the height of the original P1 to N2 (P1,N2) interpeak amplitude. Changes in amplitude of later waves, particularly N3 and N4, occurred only in the cortical SEP produced by stimulation of the ipsilateral posterior tibial nerve. The N3 wave amplitude was decreased by 54.7 ± 11.1%, and the N4 wave amplitude was decreased by 51.9 ± 6.7%. The latency of the N3 wave did not change. However, the latency of the N4 wave peak was prolonged bilaterally by 3.0 ± 0.5 msec.

Dorsal Spinocerebellar Tract Lesions

Three cats were evaluated for unilateral DSCe lesions. Again, predominant changes were produced in the early components of the SEP. The N2 wave of the SEP from ipsilateral nerve stimulation was lost completely. The P1 wave was broadened and the wave-peak latency was increased by 3.6 ± 0.3 msec. The later components of the SEP were not affected, except in one cat that had a second lesion of the ipsilateral DCS. In this cat the amplitude of the N4 wave in the SEP from unilateral stimulation was decreased by 11.5%. However, the first lesion extended into the lateral DCS unilaterally, which might account for these wave changes.

Ventrolateral Fiber Tract Lesions

Seven cats were evaluated for unilateral lesions of the fibers in the VL. There was a 42.1 ± 22.7% decrease in the iP1,N2 amplitude. This percentage is higher than expected because the lesions extended into the dorsolateral quadrant of the spinal cord in three of the cats, and the lateral portion of the ipsilateral DSCe was included in the area of injury. These cats had greater decrements in their iP1,N2 amplitude than the four other cats; these ranged between 54.4% and 71.9%, and correlated with the amount of iDSCe involved. If these three cats are excluded from analysis, the decrement in the iP1,N2 is 28.1 ± 14.8%. The iP1,N2 was decreased by 26.8 ± 11.9% in amplitude after the lesion was created. The waveform and latency in the early components of the SEP from stimulation of either side were not affected.

The most dramatic change in the SEP was in the amplitude of the cN4 wave, which was decreased 43.0 ± 21.0% compared to a decrease of 5.9 ± 5.2% in the iP4 wave. The latency of the N4 wave was prolonged by 3.0 ± 2.3 msec for stimulation of the posterior tibial nerve ipsilateral and by 6.4 ± 4.9 msec contralateral to the side of the lesion. There was a decrease in the amplitude of the cN4 wave, which was decreased 43.0 ± 21.0% compared to a decrease of 5.9 ± 5.2% in the iP4 wave. The latency of the N4 wave was prolonged by 3.0 ± 2.3 msec for stimulation of the posterior tibial nerve ipsilateral and by 6.4 ± 4.9 msec contralateral to the side of the lesion. There was a decrease in the amplitude of the cN4 wave, which was decreased 43.0 ± 21.0% compared to a decrease of 5.9 ± 5.2% in the iP4 wave. The latency of the N4 wave was prolonged by 3.0 ± 2.3 msec for stimulation of the posterior tibial nerve ipsilateral and by 6.4 ± 4.9 msec contralateral to the side of the lesion.

FIG. 5. Comparison of the cortical SEP's from stimulation of the posterior tibial nerve on the right and on the left before (upper tracings) and after (lower tracings) lesioning of the left dorsal spinocerebellar tract (black area, right). Major amplitude changes are noted in the early (P1,N2) waves of the SEP from posterior tibial nerve stimulation ipsilateral to the lesion. The amplitude of the later waves from ipsilateral stimulation and the amplitude of all waves from opposite leg stimulation are not affected.

FIG. 6. Comparisons of the cortical SEP's from stimulation of the posterior tibial nerve on the right and on the left before (upper tracings) and after (lower tracings) lesioning of the left dorsal spinocerebellar tract (black area, right). Major amplitude changes are noted in the early (P1,N2) waves of the SEP from posterior tibial nerve stimulation ipsilateral to the lesion. The amplitude of the later waves from ipsilateral stimulation and the amplitude of all waves from opposite leg stimulation are not affected.
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in the amplitude of the iN3 wave but not of the cN3 wave. The change in the amplitude of the iN3 wave varied tremendously (range 0 to 77.8%), with a mean decrease of 37%. Stimulation of each leg caused only a minor increase in the N3 wave latency (3.6 ± 3.0 msec ipsilateral, 3.3 ± 2.6 msec contralateral).

Combined Lesions of the DCS and iDSCe

Three cats were evaluated for lesions of one entire dorsolateral quadrant. The iN1P1N2 complex was lost and replaced with a single broad negative wave at 26.3 ± 9.4 msec, the amplitude of which was 39.3 ± 19.0% of the height of the original iP1N2 wave complex. The amplitude of the iN4 wave was greatly diminished or the wave was lost altogether after ipsilateral stimulation. The N4 wave amplitude was decreased by 54.7 ± 10.4% with ipsilateral stimulation and by 12.6 ± 4.3% with contralateral stimulation. The N4 wave peak latency was increased by 4.3 ± 2.1 msec and 2.0 ± 1.7 msec for ipsilateral and contralateral stimulation, respectively.

Combined Lesions of the DCS and cVL

Two cats were evaluated for lesions involving one half of the DCS and the contralateral VL. The greatest change in the SEP was produced by stimulation ipsilateral to the side of the DCS lesion. The iP1N2 amplitude was decreased by 80.4 ± 3.2%, the waveforms were flattened in shape, and the latency of the early waveforms was prolonged. The iP1 latency was increased between 4.2 and 4.8 msec, and the iN2 latency was prolonged to a lesser extent (0.8 to 1.7 msec). The cP1N2 amplitude was reduced by 34.1 ± 6.4%.

The amplitude of the iN4 wave was reduced by 76.3 ± 19.4% compared to a reduction of 6.1 ± 2.3% for the cN4 wave. Separate SEP's produced by bilateral stimulation had a 4- to 5-msec increase in the N4 wave latency. With ipsilateral stimulation the amplitude of the N3 wave was decreased by 7.62 ± 13.4%; with contralateral stimulation it was decreased by 62.1 ± 2.2%. The N3 wave latency varied, but generally increased from 2.9 to 7.4 msec.

FIG. 8. Cortical SEP's from stimulation of the posterior tibial nerve ipsilateral to complete transection of all afferent pathways on the same side of the spinal cord (black area, right). An evoked response is still present, indicating transmission of impulses is occurring through the contralateral cord.

FIG. 7. Recordings of the cortical SEP's from stimulation of the left posterior tibial nerve. There is progressive loss of the early waves (N1P1N2) with sequential ablation of the ipsilateral dorsal column (DCS, center tracing) and of the dorsal spinocerebellar tract (lower tracing). The later-occurring N4 wave is decreased in amplitude after the dorsal column lesion, but the amplitude does not decrease further following ablation of the ipsilateral dorsal spinocerebellar tract. Lesioned areas are shown (black, right).

FIG. 9. Cortical SEP's from stimulation of the right posterior tibial nerve before (upper) and after ipsilateral ablation of the right dorsal column system (DCS) and dorsal spinocerebellar tract (center), and after a second lesion of the left ventrolateral fiber tract (lower). This combination of lesions (black area, right) results in complete absence of the SEP.
were greatly diminished in height (88.9% and 87.8%, respectively). However, the latencies of these waves were increased (13.2 msec for iN₃ and 6.7 msec for iN₄). With stimulation contralateral to the DSCe lesion, there was an average decrease in amplitude by 57% in the later waves (cN₃ and cN₄) and a prolongation in latency of 10.8 msec for cN₃ and 2.2 msec for cN₄. There was a minor decrease in the cP₁N₂ amplitude of 19%, but the latency of these early waves did not change.

**Combined Lesions of the DCS, iDSCe, and iVL**

Except for the most medially located ventral fibers, one cat had all of the white matter fiber tracts ablated on one side of the spinal cord (hemisection). Posterior tibial nerve stimulation from each side with respect to
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the site of the lesion produced changes in the SEP's, the most striking of which were caused by stimulation of the leg ipsilateral to the lesion. The iP1N2 was replaced with a single negative wave with an amplitude equal to 17.7% of the original iP1N2 amplitude, and had a latency of 18.5 msec. The amplitudes of the iN3 and iN4 waves were decreased by 84.8% and 50.8%, respectively, and the latencies increased by 1.7 msec and 6.9 msec, respectively.

Stimulation of the leg contralateral to the lesion produced minor changes in the early components of the SEP. The cP1N2 wave complex was decreased by 40%, but waveforms and latencies were unaffected. However, the later components, particularly wave N4, were more severely affected. The amplitude was decreased by 59.3% for cN4 and by 41.2% for cN3. The cN4 wave had a biphasic appearance, and the latency was prolonged by 7.9 msec. The cN3 latency was unchanged.

Combined Lesions of the DCS, iDSCe, and cVL

In three cats, one dorsal lateral quadrant and the cVL were destroyed. This combination of lesions caused complete obliteration of the cortical SEP from stimulation of the posterior tibial nerve ipsilateral to the dorsolateral quadrantectomy. The cP1N2 amplitude decreased by 38.0 ± 11.3%, but the latencies or waveforms of the early components did not change. The amplitudes of the later waves (cN3 and cN4) were decreased by 63.6 ± 1.7% and 59.3 ± 10.5%, respectively, and their latencies were increased by 5.4 ± 1.2 msec and 3.2 ± 1.3 msec, respectively.

Lesions of Both Ventrolateral Fiber Tracts

Two cats had both ventrolateral fiber tracts sectioned. Essentially the same changes were produced by stimulation of each posterior tibial nerve. The waveform and latencies of the early components (N1P1N2) were unchanged. The amplitude of each P1N2 wave complex was decreased by 56.5 ± 11.2%, which approximates the predicted value of adding two unilateral VL lesions (see Ventrolateral Fiber Tract Lesions, above). The amplitude of each N3 wave decreased by 72.1 ± 4.1%; the amplitude of each N4 wave decreased by 68.0 ± 4.2%. The latency of neither was prolonged, but the latency of N4 was prolonged by 8.8 ± 1.1 msec.

Discussion

Our results indicate that, in the cat, contributions to the cortical SEP from mixed nerve stimulation are made by afferent nerve fibers that ascend in the ipsilateral dorsal column, the ipsilateral dorsal spino-cerebellar tract, and the contralateral ventrolateral tract with respect to the side of the hindlimb stimulation. These results are in agreement with recent work by other investigators, who found that SEP impulses are transmitted in the dorsal columns and dorsolateral fasciculus predominantly by large-diameter, fast-conducting fibers that have a low threshold to peripheral stimulation, and by smaller-diameter nerve fibers in the ventrolateral tracts that have a high-stimulus threshold and conduct impulses more slowly. These findings do not agree with the results of many of the experimental studies in which animals were anesthetized with barbiturates, agents that significantly decrease the amplitude of the later components of the cortical SEP (see Fig. 4). Therefore, only the early waveforms of the cortical SEP could be examined.

Clinical and experimental studies have shown that either the DCS alone or in combination with the DSCe (Morin's spinocervical tract in the cat) exclusively transmitted impulses of the cortical SEP. These studies used low-intensity peripheral nerve stimulation that only activated large myelinated nerve fibers; because of this, the contribution of the smaller-diameter, slow-conducting fibers could not be evaluated. By using a stimulus intensity of 20 times the motor threshold, we observed the cortical SEP's with a maximum amplitude, which probably indicates that all sensory nerve fibers in the mixed nerve are activated at this level of stimulus intensity. Problems associated with barbiturates were eliminated in this study by using ketamine for anesthesia, and precisely located lesions were made with a CO2 laser directed with the aid of an operating microscope.

Sectioning of the DCS and DSCE, either separately or together, produced a major decrease in the amplitude of the early positive (P1) and negative (N2) SEP waves. Ablation of one side of the DCS decreased the interpeak amplitude of the ipsilateral P1N2 wave complex by 73.4 ± 6.5%, and ablation of a DSCe decreased the amplitude of the ipsilateral P1N2 wave complex by 74.5 ± 3.5%. The amplitude of the later waves of the SEP from posterior tibial nerve stimulation ipsilateral to the lesion side was decreased for lesions of the DCS but not of the DSCe. When both pathways were sectioned, there was obliteration of P1 that produced a low-amplitude broad negative wave with a latency 1 msec longer than that of the N2 wave in control animals. It is understandable why Cohen, et al., who studied the early positive component (P1) of the SEP, concluded that there was no contribution to the cortical potential from fibers in the anterolateral tracts. In this study, the later components of the SEP from ipsilateral stimulation were diminished by about 50% with interruption of the DCS alone or in conjunction with the ipsilateral DSCe. The DSCe, therefore, does not appear to contribute to the later waves (particularly N3 and N4) of the cortical SEP. The cortical potential waveforms seen after interruption of the iDSC and iDSCe must represent impulse activity from nerve fibers that ascend in the cVL, because the potentials are abolished by further interruption of this pathway.
If cortical potentials were composed of afferent volleys from independent fiber systems, it should be possible to calculate the relative percentage each pathway contributes to the amplitude of each wave of the cortical SEP; the ablation of a given sensory pathway would decrease the amplitude of a particular SEP wave by the amount it contributes to the amplitude. Uttal and Cook, 46 who studied cortical SEP's from median nerve stimulation in humans, proposed that different afferent pathways were responsible for the polyphasic evoked potentials recorded from the scalp. They suggested that the posterior columns were necessary for transmission of impulses that formed the "M"-wave (latency of 20 msec), that the spinthalamic tract was necessary for transmission of the "N"-wave (latency of 30 msec), and that the reticular pathways were responsible for the "O"-wave (latency of 50 msec). Our results suggest that this explanation is not correct. We found that the effects on SEP's of lesion in two or more of the afferent pathways were not additive except when both ventrolateral tracts were interrupted.

An example of such nonadditive interaction was found in two cats that underwent lesions of one DCS and the cVL. Independently, ablation of the DCS decreased the ip1N2 amplitude by 73.4 ± 6.5%, and ablation of the cVL decreased the ip1N2 amplitude by 26.8 ± 11.9%. The ip1N2 should be obliterated (100% decrease in amplitude) when both lesions are present simultaneously; however, the ip1N2 amplitude was only decreased by 80.4 ± 3.2%.

The interaction between the various afferent pathways that we observed in this study was complex and could not be explained simply in terms of inhibition or facilitation. Schieppati and Ducati 43 found an increase in the SEP's from triceps surae nerve stimulation after DCS section and an increase in the SEP's or facilitation. Schieppati and Ducati 43 found an increase in the SEP's from saphenous nerve stimulation after anterolateral tractotomy, suggesting that an inhibitory influence was interrupted. Independently, ablation of the DCS is not correct. We found that the effects on SEP's of interaction with ascending sensory impulses may also occur in either the reticular system of the brain stem 11,28,37 or in the thalamus. 3,31,32,40

Various components of cortical SEP's will be lost after the application of a conditioning stimulus to a sensory nerve other than that being tested. 3,31,32,37,39 Katz, et al., 31,32 found that cortical SEP's obtained by forelimb stimulation in the cat were altered after a conditioning stimulus of intensity that affected only A fibers or all fibers to either an ipsilateral hindlimb or a contralateral forelimb nerve, predominantly involved the loss of later SEP components (latencies greater than 40 msec). Thus, high-intensity stimulation of a peripheral nerve that activates nerve fibers with both large and small diameter may mask the SEP contributions of small-fiber systems secondary to inhibition. In order to assess the uninhibited effect of small-diameter fiber input on the cortical potential, we are beginning studies that will examine the effect of selective C fiber stimulation on cortical SEP's.

Our present results show that, in cats, fibers in the anterolateral tracts (mostly slow-conducting C fibers) contribute primarily to the later components of the cortical SEP (latency greater than 30 msec) produced by stimulation of the hindlimb contralateral to the lesion. Interestingly, Blair, et al., 9 found that patients in whom DCS stimulation produced appreciable pain relief had selective suppression of the later SEP (200 to 250 msec) components from tibial nerve stimulation. Likewise, Chen, et al., 12 demonstrated a strong linear relationship between subjective painfullness from dental stimulation and the amplitude of the later SEP wave(s) (between 175 and 260 msec). These observations tend to suggest that pain perception is represented by the later components of the cortical SEP. Therefore, it might be possible that SEP's could be used as an objective assessment of pain or to assess therapies for pain relief.

High-intensity peripheral nerve stimulation is required to produce full amplification of the later SEP waves; because of the discomfort this causes, it probably would not be suitable for use in routine patient diagnosis. However, in the anesthetized and often paralyzed neurosurgical patient undergoing either a spinal or a spinal cord procedure, it would be well tolerated. Both late (greater than 30 msec) and early (less than 30 msec) waves of the SEP's could be used intraoperatively for a precise evaluation of the segmental functional status of the spinal cord. Further studies on the effects on SEP's of various anesthetic agents, changes in blood pressure, and changes in arterial gases and pH will have to be performed.

Acknowledgments
We thank Beverly J. H. McGeehe for manuscript preparation, and Neil Buckley for editorial assistance.

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Manuscript received February 25, 1982.

This research was supported by the Veterans Administration.

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