Concussive head injury producing suppression of sensory transmission within the lumbar spinal cord in cats

YOICHI KATAYAMA, M.D., D.M.Sc., JAMES D. GLISSON, B.S., DONALD P. BECKER, M.D., AND RONALD L. HAYES, PH.D.
Division of Neurosurgery, Department of Surgery, and Richard Roland Reynolds Neurosurgical Research Laboratories, Medical College of Virginia, Virginia Commonwealth University, Richmond, Virginia

This study examines the effects of concussive levels of a fluid-percussion head injury on sensory transmission within the lumbar spinal cord of the cat. Primary afferent depolarization (PAD) was suppressed for 2 to 5 minutes following injury, as assessed by dorsal root potentials and augmentation of antidromic dorsal root potentials, both evoked by stimulation of adjacent dorsal roots. Polysynaptic reflex discharges in ventral root potentials evoked by dorsal root stimulation were also profoundly suppressed during this same period, even when spontaneous and monosynaptic reflex discharges were facilitated. Changes in PAD produced by injury were abolished by spinal cord transection, but were not affected by midpontine transection. These findings suggest that concussive head injury can produce suppression of segmental sensory transmission by neurally mediated processes involving the bulbar brain stem. Recordings of dorsal root resting potentials, antidromic dorsal root potentials, and reductions of antidromic dorsal root potentials induced by tetanic root stimulation indicated that depressed segmental sensory function produced by injury was due to suppression of postsynaptic interneuronal transmission rather than to excitability changes in primary afferent fibers. Somatosensory cortical potentials evoked by dorsal root stimulation were profoundly depressed at the same time as segmental sensory transmission was suppressed, suggesting that suppressed segmental sensory transmission may also contribute to suppression of ascending sensory transmission. It is hypothesized that transmission failure of interneuronal systems in the initial period following insult may be a general response occurring in wide areas of the central nervous system, and not restricted to areas to which mechanical stress is directly applied. This response pattern may result from indiscriminate activation of interconnected excitatory and inhibitory elements of interneuronal systems.

KEY WORDS - concussion • evoked potentials • head injury • reflex suppression • sensory transmission • spinal cord • cat

In the course of experiments examining changes in spinal cord functions following concussive head injury, we have found that primary afferent depolarization (PAD) evoked by stimulation of adjacent dorsal roots is profoundly depressed in the initial several minutes following insult. Extensive research has established that PAD evoked by afferent stimulation reflects phasic presynaptic inhibition of primary afferent terminals generated by interneurons which receive sensory inputs. Thus, it appeared to us likely that concussion could reduce the transmission of sensory input at primary afferent terminals and/or at postsynaptic interneurons within the spinal cord during the initial several minutes following concussive head injury.

Earlier studies of functional changes of sensory systems following concussive head injury have devoted much attention to the conductivity of neural pathways within supraspinal structures. However, it is important to note that the brain has descending systems that can strongly influence sensory transmission even at the level of the spinal cord. Furthermore, a number of studies have shown that mechanically induced changes in activity in supraspinal motor systems can influence motor functions at the level of the spinal cord. Thus, it is possible that concussive head injury can produce similar changes in sensory systems as well.

In the present study, we have attempted to extend our initial observations by further examining the possible mechanisms responsible for PAD depression and the relationships of these changes to alterations in segmental as well as suprasegmental sensory transmis-
tion. In this research we used a fluid-percussion head injury model since previous research has established that this type of injury can produce a momentary mechanical stress that is transmitted to the brain stem and can be biomechanically well controlled. A property shared with acceleration models of concussion. In addition, acute physiological responses to fluid-percussion head injury are indistinguishable from those obtained after acceleration concussion. Thus, fluid-percussion injury is a model appropriate for study of at least the initial responses following concussive head injury. Movements of the body at the moment of experimental head injury usually make detailed electrophysiological recordings impractical; however, in contrast to acceleration models, fluid-percussion injury minimizes movement artifacts for electrophysiological recordings from spinal cord roots.

Materials and Methods

Surgical Procedures

Twenty-one adult cats, each weighing from 2.3 to 3.5 kg, were used. Fluid-percussion head injury was administered by procedures identical to those described in previous reports from this laboratory. Briefly, animals were subjected to head injury by a pressure pulse transmitted into the epidural space through a hollow metal injury screw (11 mm in diameter, with the center located at the midline and 5.5 mm anterior to the interaural line). Animals were paralyzed with a single intravenous dose of pancuronium bromide (0.08 mg/kg), mechanically ventilated, and anesthetized with a 3% lidocaine hydrochloride. Stainless steel screws were affixed to the skull bilaterally above the parietal and occipital regions for recording of electroencephalograms (EEG’s), with one screw placed in the frontal sinus region serving as a reference electrode. The intensity of the fluid-pulse was adjusted to an amplitude of 2.1 to 2.5 atm, for a duration of 21 to 24 msec. Previous research has shown that these parameters produce a concussive syndrome characterized by initial generalized areflexia and a subsequent comatose state without fatal apnea, profound circulatory collapse, or overt intraparenchymal hemorrhages.

Monitoring of Systemic Physiological Parameters

Arterial blood pressure, intracranial pressure, heart rate, and end-tidal CO₂ were monitored as in previous studies, and detailed procedures are described elsewhere. These variables were continuously recorded on a Beckman polygraph and by EEG. Arterial blood gases and pH were frequently analyzed and maintained within physiological ranges (PaCO₂ 30 to 40 mm Hg, PaO₂ ≥ 100 mm Hg, and pH 7.4). End-tidal CO₂ was maintained between 3.8% and 4.0%. Core temperature was also monitored and maintained at 37.5° to 38.5°C.

Monitoring of Spinal Cord Functions

A laminectomy of the L4–S1 segments was performed. The dorsal roots L-6 and L-7, and the ventral root L-7 were sectioned. The dorsal root L-7 was placed on a bipolar Ag/AgCl wire electrode for single pulse stimulation at supramaximal intensities (frequency 0.2 to 0.9 Hz, pulse duration 0.01 msec). For recording of dorsal root potentials evoked in adjacent dorsal roots by dorsal root stimulation, the L-6 dorsal root was mounted on a bipolar Ag/AgCl wire electrode with one pole close to but not touching the cord, and the other on the cut peripheral end (Fig. 1A). In five cats, a glass-insulated tungsten microelectrode was inserted into the interneuronal cell group of L-6 in the middle of the dorsal horn for stimulation at submaximal intensities (frequency 0.5 to 1.5 Hz, pulse duration 0.01 msec). Antidromic action potentials in response to stimulation of this site, which reflect the excitability of primary afferent terminals, were recorded from the L-6 dorsal root (Fig. 1B). Another bipolar Ag/AgCl wire electrode (not shown in Fig. 1B) was placed for tetanic stimulation of the L-6 dorsal root (frequency 100 Hz, pulse duration 0.01 msec, train pulse for 10 sec). Stimulation intensities were adjusted to submaximal ranges for post-tetanic potentiation of antidromic L-6 dorsal root potentials.

To record ventral root potentials evoked by L-7 dorsal root stimulation, the ventral root of L-7 was placed on the bipolar Ag/AgCl wire electrode (Fig. 1C). Somatosensory cortical potentials evoked by stimulation of the L-7 dorsal root were monitored simultaneously (Fig. 1D), with one stainless steel screw placed on the skull over the medial somatosensory cortex, and the other screw placed within the frontal sinus region. A deep skin pouch was formed at the site of the dissection so that the exposed neural tissue was bathed by a warm (37°C) pool of mineral oil. A drainage tube with a 6-mm diameter opening was placed at the bottom of the oil pool through the abdominal wall.

Signals were amplified by a Grass AC amplifier and/or Tektronix differential amplifier, displayed and photographed on a storage oscilloscope, averaged by a signal processor, and stored on magnetic tape for subsequent analysis. Averaged potentials were recorded by an ink-
Head injury suppression of spinal sensory transmission

![Diagrams](image)

**Fig. 1.** Schematic diagrams illustrating procedures for monitoring spinal cord functions. A: Dorsal root potentials evoked by stimulation of the adjacent dorsal root. Stimulation (S) of a dorsal root excites the dorsal horn cells which in turn produce depolarization of primary afferent terminals (PAD). This depolarization can be detected as dorsal root potentials recorded from the adjacent dorsal root (R). B: Augmentation of antidromic dorsal root potentials evoked by stimulation of the adjacent dorsal root. Stimulation of the terminal field of primary afferent fibers (S₁₃) evokes antidromic potentials in the dorsal roots (R). This antidromic potential could be augmented by conditioning stimulation of the adjacent dorsal roots (S₃). TS = test stimulus; CS = conditioning stimulus. C: Polysynaptic ventral root potentials evoked by dorsal root stimulation (S). This stimulation excites the dorsal horn cells, which in turn evoke polysynaptic reflex discharges in the ventral roots (R). D: Somatosensory near-field and far-field cortical potentials evoked by dorsal root stimulation. Somatosensory cortical potentials are generated predominantly by the dorsal column-medial lemniscal system. However, impulses within the extralemniscal systems (the dorsal column-post-synaptic tract (DCPT), the dorsal column tract (DCT), the spinocervical tract (SCT), the spinoreticular tract (SRT), and the spinothalamic tract (STT)), which are mediated by dorsal horn cells, significantly contribute to responses evoked by supramaximal stimulation of peripheral nerves (S) in the cat.⁶

writing recorder.⁶ In all cases, the amplitude of PAD (recorded as the dorsal root potential), the mono- and polysynaptic reflex discharges of the ventral root potential, and each wave of the early near-field cortical potentials were measured as the averaged amplitude (peak to baseline) of each recorded potential taken either from photographs of storage oscilloscope tracings (four sweeps superimposed) or potentials averaged by the signal processor (four to eight sweeps averaged for dorsal and ventral root potentials and 36 to 90 sweeps for cortical potentials), and displayed by the ink-writing recorder.

Identical experiments were also performed in chronic spinal cord-transected preparations and acute midpontine-transected preparations. Three days prior to the experiment, the cords of two cats were completely sectioned at the T-4 segment. The spinal section, performed using osteoplastic procedures, was made through a small incision in the dura. The incised dura was then tightly closed by sutures and bonding. Postoperatively, antibiotic agents were given and the animals’ bladders were emptied manually twice a day. Midpontine transections were performed in five cats by radiofrequency lesions (300 mA for 10 seconds) delivered through a four-pronged lesion rake approximately 3 hours before each experiment. Additional lesioning procedures are reported elsewhere.⁶ Transsection was made at a 45° angle from the coronal plane at the caudal aspect of the trigeminal motor nucleus (the transection passed through Horsley-Clarke coordinates at P5,0, H5,0). Therefore, the greater part of the pontomedullary reticular formation, including the nucleus raphe magnus, nucleus reticularis gigantocellularis, and magnocellularis, maintained its continuity with the spinal cord.

**Histological Examination**

At the conclusion of the experiment, each animal was administered an overdose of pentobarbital and then perfused transauricularly with 10% buffered formalin. We discarded data obtained from animals in which intraparenchymal petechial hemorrhages were found in the brain stem. Complete transections of the spinal cord and brain stem were verified.

**Statistical Analyses**

Student’s unpaired and paired t-tests were employed for analysis of the data. When significant differences in variances of two populations occurred, the Aspin-Welch test was used.⁶ Variability is always expressed as the standard error of the mean.

**Results**

**Dorsal Root-Evoked Dorsal Root Potentials**

The primary afferent depolarization (PAD), as detected by dorsal root potentials in response to stimulation of the adjacent dorsal root, was depressed in all five animals (p < 0.01) immediately following injury (latency to maximal suppression, 1 to 3 seconds, Fig. 2A to C) and continued for several minutes (2 to 5 minutes, Fig. 2I). This PAD suppression was observed even in the five midpontine-transected preparations (p < 0.01, Fig. 2I). In contrast, PAD suppression was abolished in the two spinal cord-transected preparations (Fig. 2I).
Antidromic Dorsal Root Potentials

The excitability of primary afferent fibers, as tested by antidromic dorsal root potentials in response to stimulation within terminal fields of the dorsal horn, was augmented by stimulation of the adjacent dorsal root (Fig. 2D). The time course of this augmentation coincided well with the time course of PAD recorded as dorsal root-evoked dorsal root potentials, and thus most probably reflects depolarization of primary afferent terminals.

Prior to injury, tetanic stimulation of the same dorsal roots reduced antidromic dorsal root potentials in three animals (31.7% ± 7.2%), indicating the occurrence of post-tetanic hyperpolarization of the primary afferent terminals. This reduction of antidromic dorsal root potential elicited by tetanic stimulation of the same dorsal root was not significantly affected following injury (±26.7% ± 8.3%). Resting levels of antidromic dorsal root potentials showed a transient decrease (Fig. 2G) followed sometimes by an increase. However, such changes in resting levels of antidromic dorsal root potentials subsided within a minute, and no noticeable changes correlated to depressed afferent-induced PAD following injury were detected.

Dorsal Root Resting Potentials

While resting potentials of dorsal roots commonly showed spontaneous fluctuations in intact animals before injury, these potentials stabilized following injury. No noticeable changes in dorsal root resting potentials were observed (Fig. 2H).

Dorsal Root-Evoked Ventral Root Potentials

Polysynaptic reflex discharges of ventral root potentials (P-VRP's) were profoundly depressed immediately following injury in five intact (p < 0.01) and five midpontine-transected animals (p < 0.01) (Fig. 3A to C). Depression of P-VRP's generally persisted longer (7 to 20 minutes) than did PAD depression in the five intact animals (Fig. 3D). Thus, during the period of PAD depression, P-VRP was always profoundly depressed. The period of P-VRP depression in the five midpontine-transected animals was significantly shorter (4 to 10 minutes, p < 0.05) than in intact animals (compare Fig. 3D and E). Thus, there was a more noticeable correlation between the magnitude of P-VRP and PAD depression (r = 0.32, p < 0.05; Fig. 3E).

In contrast, changes in monosynaptic reflex discharges of ventral root potentials (M-VRP) after injury followed a different time course from P-VRP as well as from PAD suppression seen in both intact and midpontine-transected animals. The M-VRP in intact animals was initially facilitated or unchanged and then moderately depressed. Initial facilitation was significantly enhanced in midpontine-transected preparations as compared to intact animals, and subsequent moderate depression was not reliably seen (Fig. 3A to C). Changes in P-VRP and M-VRP produced by injury were abolished in spinal cord-transected preparations.
Head injury suppression of spinal sensory transmission

Dorsal Root-Evoked Cortical Potentials

Somatosensory near-field cortical potentials (SSEP's) evoked by supramaximal stimulation of the L-6 dorsal root were similar to SSEP's evoked by sciatic nerve stimulation (Fig. 4A). Considering the peripheral nerve conduction time for sciatic nerve stimulation, the peak latencies for the initial positive wave (P1) and the subsequent large negative wave (N2) were in accordance with latencies reported for SSEP's evoked by sciatic nerve stimulation. In some instances, additional negative-positive waves (N4 and P2) were recorded between these two waves (Fig. 4A and D). Since recording of SSEP's requires the averaging of a large number of potentials, multiple recordings were not possible within the period in which profound changes in PAD and P-VRP's were observed. However, we were able to determine that all components of SSEP's were transiently abolished during the period in which PAD was also profoundly depressed. The amplitude of the initial positive early near-field potential (P1) of SSEP's recorded in three animals within 3 minutes after injury was significantly depressed (p < 0.01; Fig. 4A to C and E). The magnitude of depression of the P1 wave generally corresponded with the average value of the PAD depression recorded simultaneously following injury (r = 0.75, p < 0.01; Fig. 4F). The depression of late near-field potentials (N2) showed a similar time course (Fig. 4A to C). Although the limited number of averaged potentials precludes definitive statements regarding far-field SSEP's, four positive waves, which are presumably equivalent to waves IIa, IIb, III, and IV reported by others, were distinguished prior to P1 (Fig. 4D). All of these waves were depressed in recordings taken within the initial 3 minutes following injury (Fig. 4E).

Discussion

Data from the present study indicate that depression of segmental sensory transmission occurs following concussive head injury. This inference is based on the following observations. First, afferent-induced primary afferent depolarization (PAD) as detected by dorsal root-evoked dorsal root potentials, as well as by augmentation of antidromic dorsal root potentials, was depressed following injury. Since PAD is generated by the interneuronal circuits and is virtually independent of activity of motoneuronal cell groups, this observation indicates that segmental sensory transmission from primary afferent fibers to primary afferent terminals was depressed. Second, polysynaptic ventral root potentials (P-VRP's) were also profoundly depressed, even during the initial period associated with facilitated monosynaptic ventral root potentials (M-VRP's), indicating that P-VRP suppression, at least during the initial period, could be due to depressed sensory transmission from primary afferent fibers rather than a generalized decrease in excitability of motoneuronal soma. Third, at least the initial components of PAD and P-VRP suppression seem to be reliably associated, suggesting that certain aspects of PAD and P-VRP suppression may share common mechanisms related to depressed sensory transmission from primary afferent fibers. In intact animals, P-VRP depression generally outlasted the period of PAD suppression. This difference in the duration of suppression may be attributable to depression of motor components of P-VRP's as indicated by the later occurrence of M-VRP depression in intact animals. In contrast, a correlation in the time courses of depression of PAD and P-VRP's was more clearly seen in midpontine-transected preparations. This improved correlation could be attributable to the absence of depression of motor components of P-VRP's, a possibility suggested by the attenuation of later depression of M-VRP's in midpontine-transected preparations. Since PAD and P-VRP depression following concussive head injury was abolished in spinal cord-transected preparations, these changes appear to be neurally mediated phenomena originating in supraspinal structures rather than reflecting changes in systemic functions or humoral factors.

Excitability changes of primary afferent fibers do not
appear to play a major part in depression of segmental sensory transmission since dorsal root resting potentials, antidromic dorsal root potentials, and reduction of antidromic dorsal root potentials following tetanic stimulation of the same dorsal root did not show noticeable changes correlated to depressed afferent-induced PAD after concussive head injury. Therefore, the depressed segmental sensory transmission demonstrated in the present study may be largely, if not exclusively, due to depression of interneuronal transmission.

In the present study, we also demonstrated the profound depression of all components of near-field SSEP’s during the period of PAD depression. Although near-field SSEP’s are mediated predominantly by the dorsal column-medial lemniscal system in which dorsal horn cells are not involved, other tracts originating from dorsal horn cells have been shown to contribute significantly to near-field SSEP’s evoked in the cat by supramaximal stimulation of peripheral nerves. The P wave has been reported to be generated by thalamocortical fibers of the lateral and medial thalamic nuclei and to be dependent upon afferent spinal input transmitted by both the dorsal columns and the spinothalamic and spinocervical tracts originating from dorsal horn cells. The N wave has been reported to be generated by association cortices and to be dependent on afferent input transmitted by both the dorsal columns and the spinothalamic tract. Therefore, profound depression of all components of near-field SSEP’s may indicate depression of ascending sensory transmission not only by dorsal columns but also by pathways originating in the dorsal horn. It appears possible that depressed segmental sensory transmission,
Head injury suppression of spinal sensory transmission

as detected by PAD and P-VRP depression, may partially contribute to depression of ascending sensory transmission, since polysynaptic segmental and ascending sensory transmission systems share a considerable number of elements within the spinal cord dorsal horn (about 37 elements). Data from the present study suggesting depression of far-field SSEP's within the first 3 minutes after injury further support this possibility, since possible components of far-field SSEP's include activities within the dorsal column system as well as those in the spinothalamic, spinocervical, and spinoreticular tracts originating from dorsal horn cells. Studies by Letcher, et al., 32 and Tsubokawa, et al. (unpublished data, 1982) have reported the abolition of entire components of SSEP's, except those components originating from peripheral nerves following acceleration concussive head injury. Although further detailed electrophysiological studies are clearly necessary to confirm these observations, it is important to recognize that the concussion-produced depression of ascending sensory transmission, even at the spinal level, could contribute to depressed sensory transmission recorded within supraspinal structures.

We have also shown that PAD as well as P-VRP depression in the initial period following concussive head injury is not abolished by midpontine transection. This observation, together with the results in spinal cord-transected animals, indicates that the bulbar brain stem is necessary for production of depressed sensory transmission within the spinal cord following concussive head injury. One may argue that a spinal shock-like situation would have been produced by acute depression of the bulbar brain-stem functions. The major difficulty in this argument is the fact that the period of depressed sensory transmission seen in the present study was also associated with facilitated M-VRP's. The above observation is not consistent with the concept of spinal shock, since this condition is a state associated with decreased spinal motoneuronal activity. Furthermore, it has been reported that acute transection or cold block of the spinal cord releases segmental sensory transmission at the caudal spinal cord from powerful tonic inhibitory impingements, 14,34,43 even though such changes cannot be neurologically seen because of depressed motoneuronal activity occurring at the same time. These inconsistencies between the present data and spinal shock itself suggest that separate inquiry into the physiological processes, rather than adoption of previously established concepts, is more useful for further elucidation of the mechanisms underlying our observations in the present study. A number of studies have demonstrated powerful modulation of segmental sensory transmission by the bulbar reticular formation. 5,20,26,27,33,34,36,48 It is generally accepted that such bulbar systems tonically inhibit segmental sensory transmission. 5,14,26,27,34,39 Thus, destruction of the bulbar brain stem results in hyperactivity of sensory transmission. 26,27,39 This implies that depressed sensory transmission following concussive head injury may not be due to simple removal of bulbar activity, since such depression would be expected to cause hyperactive segmental sensory transmission. Conversely, it is possible that increased activity of bulbar inhibitory systems may result in depressed segmental sensory transmission following concussive head injury. Alternatively, it is possible that depressed segmental sensory transmission results from processes occurring within the spinal cord initiated by indiscriminate excitatory as well as inhibitory inputs arising from the bulbar brain stem.

The period of depression of segmental sensory transmission inferred in the present study coincides well with the period of generalized areflexia observed by us in another study employing the same injury level (Y. Katayama, et al., in preparation, 1985). It is interesting to note that, in that motoneurons are rather excited as indicated by facilitated M-VRP's and interneuronal transmission is depressed, the functional changes observed in the spinal reflex arc are identical to those that earlier studies 7,8,17,18 have suggested occur in the brain-stem reflex arc during the initial period of generalized areflexia following concussive head injury. These studies postulated direct mechanical influences on interneurons as a cause of transmission failure. 18 However, if disruption of interneuronal transmission is a general characteristic of the initial response of the central nervous system to concussion, failure of interneuronal transmission is not necessarily attributable to the direct effects of mechanical stress, since mechanical stress transmitted to the lumbar spinal cord by supraspinal concussion does not appear sufficient to produce such effects. 7,46 Assuming that interneuronal systems may be net-like structures composed of mutually connected excitatory and inhibitory elements, rather than linearly arranged chains of elements, theoretical models of the central nervous system indicate that indiscriminate activation of elements of such structures may transiently abolish their previous input-output characteristics. 1 This hypothesis may help to explain why only interneuronal transmission is profoundly depressed, even though the excitability of primary afferent fibers and motoneuronal soma is not seriously altered.

Previous data from our laboratory 22,23 have provided evidence suggesting that predominance of activity in a pontine inhibitory area may contribute to the comatose state associated with flaccidity of postural muscles following the initial period of generalized areflexia. In contrast, indiscriminate activation of interneuronal systems resulting in nonspecific interneuronal transmission failure of physiological pathways may explain clinical manifestations associated with generalized areflexia during a relatively brief initial period following concussive head injury.

Conclusions

The present study has demonstrated that concussive head injury produces depression of sensory transmission even within the lumbar spinal cord. This depres-
sion of segmental sensory transmission function appears to be due to interneuronal transmission failure. Thus, assessments of sensory function following concussive head injury should recognize that sensory transmission may be depressed even at the segmental level. Depression of segmental sensory transmission following concussive head injury is not observed in spinal cord transected preparations. Thus, the initial clinical symptoms following concussive head injury may not be attributable exclusively to direct mechanical influences on interneuronal transmission. In addition, the present study has demonstrated that the bulbar brain stem is necessary to produce depressed segmental sensory transmission at the level of the spinal cord. Thus, neural processes occurring between the bulbar brain stem and the spinal cord may underlie depression of segmental sensory transmission following concussive head injury.

Acknowledgments

We wish to acknowledge Dr. Peter H. Clamann of the Department of Physiology for his comments on procedures for electrophysiological recordings. We also gratefully acknowledge the excellent technical assistance of Ms. Arletha M. Allen and Ms. Mary C. Hellgeth in the conduct and analysis of this research. We deeply appreciate the careful editorial contributions of Mrs. Rae C. Spivey and Mrs. Fay Akers.

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Head injury suppression of spinal sensory transmission


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Manuscript received December 20, 1983. Accepted in final form January 9, 1985. This research was supported by Grant NS 12587 from the National Institutes of Health.

Address reprint requests to: Yoichi Katayama, M.D., D.M.Sc., Division of Neurosurgery, Box 508, MCV Station, Richmond, Virginia 23298.