Effects of hyperbaric oxygen therapy on long-tract neuronal conduction in the acute phase of spinal cord injury

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To study the acute effects of hyperbaric oxygen ventilation (HBO) on long-tract function following spinal cord trauma, the authors employed a technique for monitoring spinal cord evoked potentials (SCEP) as an objective measure of translesion neuronal conduction in cats subjected to transdural impact injuries of the spinal cord. Control animals subjected to injuries of a magnitude of 400 or 500 gm-cm occasionally demonstrated spontaneous return of translesion SCEP within 2 hours of injury when maintained by pentobarbital anesthesia and by ventilation with ambient room air at 1 atmosphere absolute pressure (1 ATA). Animals sustaining corresponding injuries but receiving immediate treatment with HBO at 2 ATA for a period of 3 hours following impact demonstrated variable responses to this treatment modality. Animals sustaining injuries of 400 gm-cm magnitude showed recovery of translesion SCEP in four of five cases, while animals sustaining injuries of 500 gm-cm magnitude responded to HBO treatment by recovery of SCEP no more frequently than did control animals. When the onset of HBO therapy was delayed by 2 hours following impact, there appeared to be no demonstrable protective effect on long-tract neuronal conduction mediated by HBO alone.

The observations suggest that HBO treatments can mediate preservation of marginally injured neuronal elements of the spinal cord long tracts during the early phases of traumatic spinal cord injury. These protective effects may be based upon the reversal of focal tissue hypoxia, or by reduction of tissue edema, or possibly by both of these mechanisms. Increasing magnitudes of impact force and delay in the onset of HBO treatment markedly diminished the protective effects of HBO on long-tract neuronal conduction following traumatic spinal cord injury.

KEY WORDS • electrophysiological monitoring • hyperbaric oxygen • spinal cord evoked potentials • spinal cord injury

Despite intensive laboratory investigation of spinal cord injury in recent years, our understanding of basic functional and pathophysiological alterations that occur following trauma remains limited. Detailed histopathological studies have documented a remarkably reproducible pattern of progressive hemorrhagic tissue necrosis.\(^3,4,6,10,23,26\) Perfusion studies have demonstrated a marked reduction of local spinal cord blood flow.\(^5,11,18,20,24\) and biochemical assays have indicated a shift of metabolic pathways from aerobic to anaerobic.\(^2\) All of these pathological changes occur within the first few hours following experimental spinal cord trauma. Other investigators have shown that parallel to these progressive pathological changes, a profound tissue hypoxia develops within and adjacent to experimentally injured spinal cord segments.\(^16,17,21,23\) While it is thought that this state of focal tissue hypoxia contributes to the ultimate irreversibility of traumatic spinal cord lesions over several hours after the initial cord trauma, the extent to which, and the mechanisms by which, the local hypoxia contributes to this irreversibility are unknown.

On the basis of experimental observations demonstrating that focal posttraumatic spinal cord hypoxia can be reversed by conditions of hyperbaric oxygen ventilation (HBO), several chronic functional experiments have been performed in animals.\(^12,17,28,29\) The
results of these experiments indicate that animals subjected to early HBO therapy show more rapid and extensive functional recovery than control animals sustaining similar transdural impact lesions of the spinal cord. Additional therapeutic trials of HBO have rendered similar encouraging results in human patients with spinal cord injury, yet few physiological foundations for such treatment have been established. To study the acute effects of HBO treatments on long-tract neuronal conduction after impact spinal cord injury, the following experiment was performed. Based on preliminary observations, we have employed an objective physiological measurement, the spinal cord evoked potential, to monitor recovery of translesion neuronal conduction during HBO therapy.

**Materials and Methods**

Twenty-five mongrel cats, weighing 2.5 to 3.5 kg, were anesthetized by subcutaneous injection of ketamine hydrochloride, 20 mg/kg, and xylazine, 1.0 mg/kg. Mechanical ventilation was established via tracheostomy using a volume-cycled small animal ventilator.* Arterial and venous cannulation was performed for continuous monitoring of systemic arterial pressure, intermittent sampling of arterial blood, and intermittent administration of a maintenance anesthetic, sodium pentobarbital, to suppress the eyelid reflex. Arterial blood gas and pH analyses were performed for adjustment of ventilatory parameters and documentation of oxygen partial pressures under hyperbaric conditions. Core body temperatures were monitored and maintained as needed by a water-circulating thermal pad.

In each animal, the left sciatic nerve was exposed and fitted with a Silastic cuff containing a bipolar platinum-iridium electrode.† This electrode was in turn connected to the output of a stimulus isolation unit driven by a stimulator unit‡ (Fig. 1).

The spinal canal was exposed for a distance of 2.5 cm through a two-level laminectomy at T-3 and T-4, and this segment of the spine was immobilized between spinal fixation devices affixed to the dorsal spinous processes of T-1 and T-2 rostral to the laminectomy site, and of T-5 and T-6 caudal to that site.

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*Volume-cycled small ventilator manufactured by Harvard Apparatus Co., Dover, Massachusetts.
†Bipolar platinum-iridium electrode manufactured by Avery Laboratories, Farmingdale, New York.
‡Stimulus isolation unit, Model SIU-5, and stimulator unit, Model S88, manufactured by Grass Instruments, Quincy, Massachusetts.
§Bipolar platinum-iridium recording electrodes manufactured by Avery Laboratories, Farmingdale, New York.
¶Model P-15 AC amplifier manufactured by Grass Instruments, Quincy, Massachusetts. Model 565 dual-beam oscilloscope manufactured by Tektronix Corp., Beaverton, Oregon.
**Signal-averaging computer, Model 1170, from Nicolet Instrument Corp., Madison, Wisconsin.
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FIG. 2. Representative pathophysiological changes following impact injury of 400 to 500 gm-cm magnitude in the thoracic spinal cord of the cat. Left: Signal-averaged evoked spinal cord potentials recorded caudal to the lesion site (A), and rostral to the lesion site (B). With electronic amplification of tracing B, note the presence of a low-amplitude, low-frequency injury potential (C). Right: Changes in systemic arterial pressure (SAP) and mean arterial pressure (MAP) following impact injury.

on the cathode-ray screen of the signal averager and stored on Polaroid film. In this fashion, stable signals of 25 to 100 µV amplitude were recorded during sampling periods of 30 to 60 minutes prior to spinal cord injury.

All animals were then subjected to transdural impact injury of the spinal cord at the T3–4 vertebral level, delivered by a standardized weight-drop technique. A tubular weight-drop mechanism mounted on a micromanipulator was positioned so that its impactor pedestal, 6 mm in diameter, rested on the dorsal aspect of the spinal theca. A stainless steel weight (18.9 gm) was released from a variable distance above the impactor to deliver injuries of 400 gm-cm (18.9 gm x 21 cm = 397 gm-cm) and 500 gm-cm magnitude (18.9 gm x 27 cm = 510 gm-cm). Impact injuries of these magnitudes were sufficient to render immediate and complete loss of detectable SCEP at the rostral electrode site in all animals (Figs. 2 and 3) and to cause posttraumatic vasomotor instability. Mean systemic arterial blood pressures averaged 84 ± 18 mm Hg before and 69 ± 16 mm Hg after trauma. This phenomenon persisted for several hours following injury, and no attempt was made to restore blood pressures to preinjury levels. No correlation was noted between signal return and posttraumatic blood pressure changes. In addition, the impact forces selected for these experiments were capable of producing moderate to severe central hemorrhagic necrosis in all animals. Historically, these impact forces are capable of rendering permanent neurological deficit in 90% of experimental animals (cats) subjected to them.

The SCEP were monitored for up to 6 hours following impact for evidence of recovery of neuronal conduc-

tion through the site of injury. Recovery of translesion neuronal conduction was defined as the return of persistent reproducible SCEP of amplitude greater than system noise at the rostral electrode. Control animals were maintained by mechanical ventilation with ambient room air (approximately sea level, 1 atmosphere absolute pressure (ATA)), and treatment group animals were ventilated with 100% oxygen delivered under conditions of 2 ATA within a hyperbaric chamber. Arterial blood samples consistently demonstrated dissolved oxygen partial pressures averaging 91.1 ± 11.4 mm Hg before and 1091 ± 73 mm Hg during HBO administration.

At the conclusion of each experiment, the traumatized cord segment was removed and fixed in 10% formalin for histological preparation. Transverse cord sections 10 µ thick were stained with hematoxylin, eosin, and Luxol fast blue prior to microscopic examination.

Results

Five experimental groups consisting of five animals each were defined (Table 1). Groups I and III sustained 510 and 397 gm-cm injuries, respectively, following which no treatment other than maintenance pentobarbital anesthesia and controlled ventilation with ambient room air was administered. Two other groups of animals, Groups II and IV, sustained 510 and 397 gm-cm impacts, corresponding to the two control groups. These animals, however, were treated with hyperbaric oxygen ventilation (100% inspired O₂) at 2 ATA within 15 minutes of trauma and continuing for 3 hours. Animals in Group V sustained impact injuries of 397 gm-cm force, following which
no treatment other than maintenance was adminis-
tered for 2 hours. During this time, no detectable
return of neuronal conduction was observed in any of
the animals within Group V. At the end of 2 hours,
each Group V animal was administered 100% oxygen
ventilation at 2 ATA for an additional 2 hours.

Recovery of translesion neuronal conduction was
observed in both treatment and control groups. How-
ever, quantitative differences were noted between
comparable groups. One animal from each of Groups
I and III demonstrated spontaneous recovery of de-
tectable and reproducible SCEP at the recording elec-
trode rostral to the site of injury by 90 minutes follow-
ing the transdural impact (Fig. 4). These signals were
abnormal in latency, amplitude, and morphology;
however, they persisted and grew in amplitude
throughout the 6-hour observation period following
impact. The remaining animals in these control groups
demonstrated no evidence of returning translesion
neural conduction for the duration of the SCEP
monitoring.

Among the animals of Group II, again, one of five
animals demonstrated detectable return of rostral
SCEP by 100 minutes following impact (Fig. 4). The
remainder of Group II failed to demonstrate any
detectable return of translesion neuronal conduction
for the duration of SCEP monitoring.

Among the animals of Group IV, four of five
demonstrated detectable persistent return of neuronal
conduction between 60 and 120 minutes following
impact (Figs. 3 and 4). The remaining animal in
Group IV failed to demonstrate any detectable con-
duction return throughout the period of observation.
Returning signals observed in animals of this group
and in those of Group II persisted for at least 1 hour
following cessation of HBO and did not appear to
deteriorate in that time. None of the animals in Group
V, the delayed treatment group, demonstrated any
detectable return of neuronal conduction throughout
the period of observation.

Analysis of the SCEP signals monitored throughout
each experiment revealed that in all animals SCEP
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TABLE 1
Summary and analysis of SCEP recordings following transdural impact injury*

<table>
<thead>
<tr>
<th>Group</th>
<th>Cat No.</th>
<th>Trauma Impact</th>
<th>Treatment</th>
<th>SCEP Return</th>
<th>Amplitude (µV)</th>
<th>Percent Return</th>
<th>Latency P1 (msec)</th>
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<tr>
<td></td>
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<td></td>
<td></td>
<td>Re-turning Signal</td>
<td>Control Signal</td>
<td>Control Signal</td>
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<tr>
<td>I</td>
<td>9</td>
<td>510 gm-cm</td>
<td>maintenance anesthesia, FIO2 = 0.2</td>
<td>yes (90 min)</td>
<td>1.2</td>
<td>7.3</td>
<td>16</td>
</tr>
<tr>
<td></td>
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<td>II</td>
<td>14</td>
<td>510 gm-cm</td>
<td>immediate FIO2 = 1.0, 2 ATA x 3 hrs</td>
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<td>no</td>
<td>no</td>
<td>yes (100 min)</td>
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<td>397 gm-cm</td>
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<td>397 gm-cm</td>
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<td>25</td>
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<td></td>
<td>yes (120 min)</td>
<td>5.3</td>
<td>85.2</td>
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<tr>
<td>V</td>
<td>35</td>
<td>397 gm-cm</td>
<td>2-hr delay, FIO2 = 1.0, 2 ATA x 2 hrs</td>
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* SCEP = spinal cord evoked potentials; ATA = atmosphere absolute pressure; FIO2 = fraction of inspired oxygen.
† Percentage of the returning signal amplitude of the control signal amplitude obtained before impact.

recorded caudal to the lesion site remained reasonably stable in amplitude and latency throughout the monitoring period and did not appear to be affected by either the traumatic lesion or by HBO treatment (Fig. 3). The persistence of this stable caudal SCEP signal following cord injury indicates that the immediate postimpact interruption of ascending long-tract conduction is focal and limited to the site of injury. The caudal SCEP were not used for standardization or comparison to the amplitude of returning SCEP recorded from the electrodes rostral to the lesion site, because of the variability of physical parameters between recording sites. Latency and amplitude of returning signals were compared with signals obtained from the rostral epidural electrodes prior to impact (Table 1).

In all animals, immediate and complete disappearance of rostral SCEP was noted at the time of impact and, in the animals described above, progressive return of rostral SCEP was observed. In cats which demonstrated return of neuronal conduction through the site of impact, the characteristics of all returning SCEP were remarkably similar (Table 1 and Fig. 4). Signal recovery occurred by 90 minutes after impact in untreated animals and by 60 to 120 minutes under HBO treatment. Conduction recovery was, in all cases, only partial in that signal amplitudes were in the range of 1 to 5 µV, compared to control signals of 10 to 50 µV (6% to 24% of control signal amplitude). Despite marked complexity and high frequency, the returning signals always demonstrated prolonged latency and loss of early large amplitude peaks seen in control signals. These findings signify both reduction in numbers of, and desynchronization of, conducting neural fibers.

To discriminate between returning signals originating from primary afferent fiber pathways and those originating from synaptic pathways, the frequency response of translesion-conducted signals to varying rates of sciatic nerve stimulation was recorded (Fig. 5). At higher rates of stimulation, alterations in SCEP were evident, constituting conduction failure in syn-
aptic pathways, and indicating the contribution of both synaptic and primary afferent pathways to the returning signals.

Another noteworthy observation is the development of a low-amplitude, low-frequency injury potential recorded from the rostral electrodes in all animals (Figs. 2 and 3). This potential appeared immediately following impact in all cases, and persisted throughout the duration of observation regardless of the return of SCEP conduction through the site of injury. It occurred only during sciatic nerve stimulation and its latency at onset corresponded to 3.3 msec, compared to 4.0 msec for the onset of preinjury rostral signals, 3.0 msec for the onset of caudal signals, and 4.8 msec for the onset of returning rostral signals recorded following injury. The amplitude of this low-frequency potential was 1 to 3 μV, and its duration 6 msec. When translesion neuronal conduction was observed to return following impact injury, these signals were always superimposed on a baseline consisting of the injury potential.

In contrast to injuries that cause focal blockade of spinal cord-conducting pathways, diffuse ischemia of the spinal cord produced by cardiac arrest is followed by a progressive deterioration of SCEP. Over a period of 3 to 20 minutes after cardiac arrest, SCEP amplitude is lost and signal latency is prolonged without the development of a distinct injury potential (Fig. 6). Similar effects have been noted by Cracco and Evans\(^7\) following induced respiratory arrest. Kobrine, et al.\(^19\) have followed spinal cord neuronal conduction by monitoring SCEP during controlled ventilation with low concentrations of inspired oxygen. Their investigation has demonstrated that long-tract neuronal conduction fails only after a 10- to 20-minute period of an 8% to 10% inspired gas O\(_2\) content with a resultant arterial pO\(_2\) of 20 to 30 torr. The difference in time course for signal failure under these various experimental conditions may reflect the tissue-toxic effects of metabolic acidosis which develop more rapidly following circulatory failure than during controlled systemic hypoxia. However, the time course for either type of hypoxic or asphyxic deterioration of long-tract conduction is different from the immediate onset of conduction failure produced by impact injury. The absence of development of a distinct injury potential...
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![Graph](image)

**Fig.** 5. Recovery of spinal cord evoked potentials (SCEP) following 510 gm-cm impact injury. Tracings prior to impact injury (I), 15 minutes following impact (II), and 105 minutes following injury (III). IV: Frequency response of returning rostral signal to varying rates of sciatic nerve stimulation. Recordings from caudal (A) and rostral (B) electrodes are seen at same amplification. Electronic amplification of rostral signal is shown in tracing C. Note deterioration of specific peaks with high-frequency stimulation.

During the course of systemic conduction failure suggests that such an injury potential is a volume-conducted phenomenon associated with focal conduction block.

Histological examination of injured spinal cord segments revealed changes consistent with progressive central hemorrhagic necrosis (Fig. 4). These findings of petechial and coalescent gray matter hemorrhage, with associated hemorrhage and edema within adjacent white matter, appeared in all specimens. Animals subjected to 510 gm-cm impact demonstrated more extensive tissue injury than did animals subjected to 397 gm-cm impact. No quantitative differences were noted in the extent of tissue injury between treatment and control groups.

**Discussion**

The application of hyperbaric oxygen ventilation (HBO) in the acute treatment of spinal cord injuries was reported by Maeda in 1965. His study of tissue oxygen levels in the spinal cord before and after transient crush injuries in dogs largely defines the development of posttraumatic tissue hypoxia within the injured cord segment. Within 1 to 2 hours following injury, spinal cord tissue oxygen falls to subvital levels and remains depressed for prolonged periods, up to 72 hours in Maeda’s experiments. During this time, Maeda was able to reverse subvital tissue oxygen levels within the injured cord segments by administration of 100% oxygen under conditions of 2 and 3 atmospheres absolute pressure (ATA).

The data from Maeda’s experiments indicate a linear relationship between ventilatory oxygen pressure and tissue oxygen levels (between 1 and 3 ATA, 100% inspired oxygen) analogous to the linear relationship between ventilatory oxygen pressures and dissolved oxygen partial pressures in blood following saturation of blood hemoglobin. According to Henry’s law, the amount of gas dissolved in a liquid with which it is in contact, but does not chemically combine, is directly proportional to the partial pressure of the gas above the liquid. For conditions of 1 ATA (sea level) ambient pressure and 20% inspired oxygen (fraction of inspired oxygen (FIO₂) = 0.2), arterial oxygen partial pressure (PaO₂) will approximate 100 mm Hg. In blood containing 15 gm of hemoglobin per 100 cc, oxygen saturation of hemoglobin occurs at PaO₂ of approximately 100 mm Hg, and maximum
heme oxygen-carrying capacity is approximately 20.1 vol\%. Theoretical considerations indicate that ventilation with 100% oxygen (FIO2 = 1.0) at 1 ATA can increase PaO2 to 673 mm Hg and increase total blood oxygen content from 20.4 to 22.1 vol\%. Ventilation with FIO2 at 2 ATA can yield a maximum PaO2 of 1433 mm Hg, and total blood oxygen content of 24.3 vol\%. Similarly, ventilation with FIO2 = 1.0 at 3 ATA can yield a maximum PaO2 of 2193 mm Hg and total blood oxygen content of 27.0 vol\% through a 22-fold increase in dissolved oxygen content. Practically, however, ventilation of our experimental animals with FIO2 = 1.0 at 2 ATA increased total blood oxygen content by about 15% from that under conditions of FIO2 = 0.2 and 1 ATA by delivering an average PaO2 of 1091 mm Hg.

Although a 15% increase in blood oxygen content may not appear physiologically important, all of this additional oxygen is free to diffuse from the intravascular space, greatly increasing the blood-to-tissue oxygen gradient. Kelly, et al.,16,17 found that traumatic spinal cord injuries in dogs produced profound segmental spinal cord hypoxia which could not be reversed by 100% oxygen ventilation at 1 ATA. This was confirmed by Hukuda, et al.14 However, ventilation with 100% oxygen at 2 and 3 ATA did restore tissue oxygen at the lesion site to supravital levels, according to Kelly's measurements. In addition to increasing tissue oxygen availability, HBO has been shown to have constrictive effects on the normal central nervous system vasculature and indirect effects which reduce tissue edema following cerebral injury.25

Kelly, et al.,17 also conducted chronic functional studies of dogs sustaining impact spinal cord injuries and found that functional hindlimb recovery was earlier and more extensive in dogs treated with 100% oxygen ventilation at 2 ATA than in control animals. A brief report on experimental HBO therapy by Hartzog, et al.,12 included small numbers of baboons subjected to transdural impact injuries. Hindlimb functional recovery appeared to be more extensive in animals treated with HBO at 3 ATA. Yeo, et al.,28,29 conducted chronic experiments on sheep that had sustained experimental impact injuries of the spinal cord, and demonstrated significant differences in functional recovery between control and treatment groups (HBO, 3 ATA) from the 1st week following injury. Histopathological examination of spinal cord sections removed from these animals showed less extensive cystic degeneration at the lesion site 8 weeks after injury than did those of control animals.

In 1975, Holbach, et al.,13 reported the use of HBO administered at 1.5 ATA to three patients with compressive spinal cord lesions. In each case, improvements of neurological deficits were observed. Subsequently, Yeo, et al.,27 and Jones, et al.,15 reported HBO treatment results for 10 and nine additional patients, respectively. These series included patients with acute traumatic spinal cord injuries who commenced HBO treatments within 10 to 14 hours following injury. Treatment protocols included HBO ventilation at 2.5 ATA for 90 minutes, interrupted by rest periods of variable duration where ventilatory oxygen content was reduced to 20%. Although no significant statistical differences are claimed by these authors, they report that no patient deteriorated as a result of...
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HBO therapy and that, in their opinion, several patients improved during HBO treatments. An additional group of five patients with spinal cord injury treated with HBO at 2 ATA is reported peripherally by De Jesus-Greenberg, although no details of clinical outcome are included in this report.

Our experimental design allowed us to monitor the integrity of neuronal conduction in the ascending long tracts of the cat spinal cord during the acute phase of spinal cord injury. This enabled us to observe the process of spontaneous conduction recovery as well as to test the effects of HBO therapy on SCEP conduction through injured and uninjured spinal cord segments. It also allowed us to test the efficacy of delayed HBO therapy in comparison to immediate administration of HBO therapy. Previous experiments by Campbell, et al., using similar techniques determined that, in cats subjected to transdural impact injuries of the thoracic spinal cord, return of cortical evoked potentials within 2 hours of impact strongly predicted eventual functional hindlimb recovery, whereas absence of these signals for longer periods of time predicted permanent paraplegia. The end point of our own experiment was the return of detectable and reproducible SCEP within 6 hours following impact injury.

Preliminary SCEP recordings obtained from each animal displayed waveforms characteristic in morphology and in amplitude for the mid- and upper thoracic electrode locations, respectively (Fig. 3). These signals consisted of an initial positive deflection (P1) followed by a larger-amplitude negative deflection (N1) and subsequent polyphasic high-frequency waveforms of diminishing amplitude. Latency differences between the appearance of P1 at the caudal electrode and at the rostral electrode allowed calculation of the conduction velocities for the most rapidly conducting neural elements in these preparations. These were usually in the range of 50 m/sec. Signal amplitudes measured in the high thoracic region were smaller than those measured in the midthoracic region. It is believed that the origin of the earliest conducted signals in the cat lies in the primary afferent fibers of the dorsal columns, and that later signals originate in the synaptic pathways which also follow the course of the dorsal columns. As such, the SCEP sampling performed throughout each experiment served primarily as a physiological monitor for the integrity of the dorsal funiculus ipsilateral to peripheral nerve stimulation.

Evidence of spontaneous partial recovery of rostral SCEP in control animals indicates recovery of neuronal conduction within certain elements of the ascending long tracts which are rendered nonconductive by the initial impact. Subsequently, these fibers complete sufficient reparative processes to conduct again in response to peripheral nerve stimulation. The return of translesion SCEP in one of five animals of Group II indicates no difference between Group II and its untreated counterpart, Group I. However, the partial recovery of SCEP in four of five animals of Group IV indicates a substantial quantitative difference in response between treatment Group IV and its corresponding control, Group III.

By introducing a 2-hour delay between the onset of impact injury and the onset of HBO treatment in Group V, spontaneous return of translesion SCEP conduction was precluded. At the same time, efficacy of delayed HBO treatment was tested. No animal in this delayed treatment group demonstrated return of SCEP conduction across the site of injury during the 6-hour period of observation.

Failure of signals to return in HBO-treated animals of Group II with greater frequency than in control animals suggests that this intensity of trauma-induced tissue injury is so extensive that long-tract conduction could not be preserved or restored by HBO alone. The failure of any animal in Group V to recover detectable SCEP conduction implies that the combined damage of impact and subsequent tissue hypoxia, edema, ischemia, and central hemorrhagic necrosis could not be reversed by HBO administration later than 2 hours following impact. The return and persistence of SCEP conduction in most Group IV animals may indicate the presence of a protective effect of HBO on elements of the ascending long tracts which were not permanently impaired at the moment of impact yet would succumb to progressive tissue hypoxia within the first few hours following injury. However, preservation of long-tract conduction could be demonstrated only in those animals sustaining lower magnitudes of trauma and subjected to immediate HBO therapy.

The findings discussed here are consistent with a bimodal mechanism of spinal cord injury due to impact trauma. At the time of impact, neuronal conduction is blocked at the site of injury by disruption of certain fibers and by transient impairment of membrane function in other fibers of the long tracts. Transient conduction block lasts for approximately 90 minutes in the cat, following which gradual return of conduction occurs in these marginally injured fibers. Such transient conduction block may be related to the loss of extracellular-intracellularionic gradients at the time of impact, followed by gradual restoration of these gradients by energy-dependent processes. Over a period of 1 to 4 hours after impact, however, secondary changes occur within the spinal cord that
can progress to total and permanent loss of long-tract conduction on the basis of local metabolic compromise. It is within this critical time period that the protective and therapeutic effects of HBO exert their maximum benefit by providing substrate for oxidative metabolism within the injured spinal cord segment, reducing tissue edema as it does in cerebral injuries, or possibly by direct vascular effects. To correlate the observations based on this acute experiment with long-term functional recovery, additional experiments are needed, including a long-term follow-up study of SCEP and functional hindlimb recovery of injured animals.

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