Effects of local hypothermia and tissue oxygen studies in experimental paraplegia

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A controlled series of adult mongrel dogs were rendered paraplegic by traumatizing the thoracic spinal cord. Those animals treated with local hypothermia, whether immediately or after a delay, recovered to a significantly greater degree than the untreated group. Spinal cord pO₂ studies revealed a marked fall in the pO₂ at the area of maximal injury over a 30-min period. The severe hypoxia lasted at least 7 hours. Pathological studies showed the varying degrees of injury produced. It is postulated that local hypothermia may be effective in altering the clinical recovery by decreasing the tissue metabolism at the site of injury.

KEY WORDS • spinal cord • hypothermia • paraplegia • tissue pO₂

Many methods have been used in an attempt to prevent permanent paraplegia in patients with injuries to the spinal cord. There are no consistently effective measures available, however, to retard or arrest those physiological and anatomical events leading to interruption of function in the contused spinal cord. The degree of recovery following spinal cord contusion seems to be primarily related to the degree of the trauma to the spinal cord. The patient's age and general health, and the quality and type of neurological management and rehabilitation, are important but probably secondarily related.

Recently, interest has developed in the protective effects of local hypothermia following spinal cord trauma. Laboratory experiments have shown that the spinal cord can be selectively cooled by local perfusion without affecting the over-all body temperature, and local reduction of temperature for sustained periods of time has been shown to be well tolerated by the normal spinal cord. Albin and his associates and Ducker and Hamit reported excellent functional recovery following nondisruptive trauma to the spinal cord of dogs and subhuman primates treated with hypothermia through extravascular local perfusion.

Numerous experimental and clinical reports have appeared concerning the effect of hypothermia on the central nervous system. However, the therapeutic effectiveness has often been contradictory or not reproducible. Before undertaking a clinical trial we preferred to carry out laboratory studies. We have therefore studied the development and character of spinal cord hypoxia following trauma by the direct method of measuring tissue-oxygen tension.

Study of Neurological Impairment

Methods and Materials

Throughout the study, healthy adult mon-
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grel dogs weighing from 12 to 16 kg were used. The dogs were anesthetized with pentobarbital (32 mg per kg of body weight). Additional pentobarbital (10 mg per kg of body weight) was administered when needed during the experiments. The animals were given lactated Ringer's solution intravenously during the prolonged experiments. Endotracheal tubes were used and a respirator when necessary. Samples of arterial and venous blood were analyzed intermittently for $pO_2$, $pCO_2$, and pH.

The spinal cord and its investing membranes were carefully exposed from T-9 through T-11; a more extensive laminectomy was required for the physiological studies. Care was taken to remove all the layer of epidural fat, which was quite prominent in some animals. After complete hemostasis had been achieved, a light-weight aluminum impounder was positioned over the exposed dura. Free movement of the impounder was assured, care being taken to see that it did not impinge on the bony edges of the laminectomy and that its diameter was equal to the width of the exposed spinal cord. A Teflon tube was placed over the impounder and the surgical table adjusted so that the encased impounder was exactly perpendicular to the spinal cord. Trauma to the spinal cord was produced by dropping a known weight for a known distance through the tube. The amount of force could then be calculated; friction was considered negligible. In most of the experiments, a 20 gm weight was dropped for a distance of 20 cm. Thus, for convenience the force of the injury could be expressed as a 400-gm-cm force (GCF).

Animal Care. All experimental animals were examined by the veterinarian on the day before the experiment. During the immediate postoperative period, the animals were placed on a special plastic screen in a cage located in the animal recovery room. They were turned frequently, and their bladders were expressed every 6 hours. They were bathed daily. Blood count and urinalysis were done frequently. Antibiotics were administered for the first postoperative week. Food supplements and vitamins were given when necessary. The animals were allowed to eat and drink on the day following surgery. They were followed by the veterinary service throughout the period of observation.

Localized Hypothermia of the Spinal Cord

After laminectomy and traumatization of the spinal cord, the dura was incised and retracted with silk sutures. A sterile tubular coil system was placed in the alcohol bath of a hypothermia unit (Temptrol 2R6-G) with pump (Sigmamotor 2-6S). Inflow and outflow tubes were then placed within the surgical defect over the exposed spinal cord. With the surgical field used as a reservoir, cold saline was circulated at a rate of 100 ml/min. Remote censors from the Temptrol unit maintained an inflow temperature of 5°C with a reservoir temperature of 7° to 9°C. Spinal cord temperature was monitored with a No. 26 needle thermister. The flow system was closed off from the cooling unit and the pump so that sterility could be maintained throughout the period of local perfusion.

Experimental Groups

Normothermic Control Group. Ten dogs were subjected to 400 GCF. The dura was incised and left open. The spinal cord was covered with Gelfoam and the wound closed. These animals were followed when possible for 3 months. To complete the control group of animals for Test Group 2, an additional four dogs had the dura left open for 4 hours.

Test Group 1 (Immediate Cooling Following Trauma). After laminectomy, the spinal cords of 10 dogs were subjected to 400 GCF. As in the control group, the dura was then incised and retracted with silk sutures. Spinal cord cooling was started immediately and continued for a period of 23 hours; a mean cord temperature of 12.1°C, ± 3.1°C, was maintained throughout the period of cooling. After this period, the wound was closed aseptically, and the animals were followed for a period of 3 months.

Test Group 2 (Delayed Cooling Following Trauma). Fourteen dogs were subjected to standard laminectomy from T-9 through T-11. The spinal cord was contused with 400 GCF at T-10, and, as in the control group, the dura was open immediately. After a delay of 4 hours, local cooling of the spinal cord was carried out for a period of 3 hours; a mean cord temperature of 12.8°C, ± 2.9°C, was maintained during cooling. The wound was closed, and the animals followed neurologically for 3 months.

* Yellow Springs Instrument Co., Inc., Yellow Springs, Ohio.
Postoperative Neurological Examinations

The test and control animals were examined daily for 3 weeks postoperatively and twice weekly thereafter until the end of the observation period. Their ability to walk and jump up was tested on a closed runway. Judgment was made concerning motor strength as well as motor coordination of the hind limbs. The scale for judging recovery of motor function proposed by Tarlov\(^\text{16}\) and adapted by Albin, et al.,\(^\text{1,3}\) was used: 0 = no voluntary movement; 1 = perceptible movement at the joints; 2 = good movement at the joints but inability to stand; 3 = ability to stand and walk; 4 = complete recovery. The animal’s ability to perceive pain was judged by evaluation of turning of the head or growling; simple withdrawal of a limb was not considered a reliable indicator of pain perception. Position sense of the joints was tested by observing the ability of the animal to correct an upside-down position of the foot. Placing reaction was tested by thrusting the animal’s foot over the edge of the examining table. The presence of adequate motor function was necessary to test for both touch and position sense. Black-and-white photographs and moving pictures on 16-mm color film were made during the observation period to document neurological changes.

Results

Normothermic Control Group. In the 14 animals subjected to 400 GCF, the spinal cord appeared contused, with subpial hemorrhage appearing within 15 min after trauma (Table 1). In one dog, cord substance extruded through a small pial tear. Five animals remained completely paraplegic with no motor or sensory function. Seven animals developed some movement at the joints but recovered very little sensation. Only two dogs could stand and walk at the end of the observation period.

Test Group 1. Of the 10 dogs subjected to cord trauma and immediate cooling, two remained completely paraplegic (Table 2). Three animals recovered completely. The remaining five dogs were moderately impaired: their gait remained uncoordinated, they maintained stance in slight flexion, had involuntary function of the bladder, and a large volume of residual urine.

TABLE 1

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Test Group 2. Of the 14 animals subjected to cord trauma and delayed cooling, only one remained completely paraplegic (Table 3). Four animals were markedly impaired. Five dogs were neurologically normal. Four others were moderately impaired, and also had abnormal function of the bladder.

Study of Spinal-Cord pO\(_2\)

To understand better the events that occur in the spinal cord following nondisruptive trauma, studies of the pO\(_2\) of spinal-cord tissue were carried out. Because of errors inherent in the instrumentation available for such studies, two separate but related methods for determination of tissue pO\(_2\) were used.

Methods and Materials

To study the pO\(_2\) of surface tissue, an IL 17026 reservoir electrode* was used for the polarographic determination of tissue pO\(_2\) through an IL 113 oxygen analyzer.* The electrode was first calibrated at 37°C as in the usual calibration procedure for determinations of blood gas. The electrode was then dismounted and calibrated in normal saline solution at 37°C saturated with a mixture of oxygen-free gas and room air. After stabilization up-scale and at zero slope, the electrode was checked in a saline solution of known

* Instrumentation Laboratory, Inc., 113 Hartwell Ave., Lexington, Massachusetts 02173.

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pO₂ content. At the completion of each experiment, the electrode was rechecked in vitro; if a greater than 5% error was noted, the observations were discarded.

To study the pO₂ of subpial tissue of the spinal cord, an IL 17365 pO₂ needle electrode* was modified for amperometric determination of tissue pO₂ through the IL 113 oxygen analyzer. Calibration was accomplished as with the surface electrode. At the completion of the study, the electrode was checked in vitro for sustained performance.

Experimental Groups

Laminectomy from T-6 through T-11 was performed on 10 dogs. The animals were respired with room air through an endotracheal tube. Arterial blood pressure and pulse rate were recorded from the lower aorta through a catheter connected to a strain gauge leading to a polygraph. Arterial and venous pO₂, pCO₂, and pH were monitored throughout the experiments. The 10 dogs were divided into two experimental groups.

Group 1. In five animals (Fig. 1) the dura was opened and retracted. With the reservoir, electrode surface readings of pO₂ were made at various points on the cord until stable readings were obtained. The dura was then closed and the cord subjected to a trauma of 400 GCF. The dura was reopened, and continuous readings were made with the same electrode over the next 2 hours. When the tissue pO₂ again stabilized in the traumatized area, recordings were obtained at a distance of 5 mm above and below the center of impact. Local hypothermia was instituted: the reservoir temperature was 7°C to 9°C. This was continued over a 3-hour period. When hypothermia was stopped and the cord allowed to rewarm to 37°C, additional measurements of tissue pO₂ were obtained.

Group 2. In five additional animals (Fig. 2), the dura was incised and retracted. With the in vivo needle electrode, the tissue pO₂ of the substance of the dorsal columns was recorded until stable readings were obtained. The dura was then closed and the cord subjected to trauma of 400 GCF. The dura was

* Instrumentation Laboratory, Inc., 113 Hartwell Ave., Lexington, Massachusetts 02173.

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TABLE 2
Neurological findings in Group 1 subjected to 400 GCF followed by immediate hypothermia

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TABLE 3
Neurological findings in Group 2 subjected to 400 GCF, 4-hour delay, and 3 hours of local hypothermia

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FIG. 1. Mean tissue pO₂ in dogs subjected to 400 GCF and hypothermia. Shaded area represents extremes of values obtained at each point in time.
reopened and the needle electrode repositioned in the dorsal columns in the area of trauma. Readings of tissue \( pO_2 \) were obtained over the next 7 hours. Following this period, recordings were obtained at 5-mm distances above and below the center of impact (Fig. 3).

**Results**

The partial pressure of oxygen as recorded through the reservoir surface \( pO_2 \) electrode in the traumatized spinal cord declined in a linear fashion over a period of 1 hour. After 1 hour, the mean \( pO_2 \) was 7 mm Hg, which represented a severe state of hypoxia. The \( pO_2 \) continued to deteriorate over the next hour, and at the end of 2 hours the mean \( pO_2 \) in the injured area was 3 mm Hg (Fig. 1). With the *in vivo* needle electrode positioned in the dorsal white columns, a decline in tissue \( pO_2 \) was noted over a 30-min period; at 30 min after trauma the mean \( pO_2 \) was 3 mm Hg (Fig. 2). Because of the extreme sensitivity to temperature of the \( pO_2 \) electrodes, measurements of tissue \( pO_2 \) could not be obtained during hypothermia. However, when the cord was allowed to rewarmed to 37°C, no change in tissue \( pO_2 \) was noted. The duration of extreme hypoxia in the traumatized area could not be assessed by the methods used. There was no significant change in tissue \( pO_2 \) in the injured area in the 7-hour period following trauma.

By both electrodes it was demonstrated that tissue \( pO_2 \) above and below the level of trauma was consistently normal (Fig. 3). The mean values for tissue \( pO_2 \) were 19 mm Hg at 1 cm above the center of injury and 18 mm Hg at 1 cm below the center of impact. At 2 cm above and below the center of impact, the mean values were 27 mm Hg and 32 mm Hg respectively. These values approach what is considered to be a normal level for tissue \( pO_2 \) of the spinal cord. At 3 cm above and below the lesion, the mean values were 29 mm Hg and 33 mm Hg respectively. In all animals, the blood pressure, pulse rate, arterial and venous \( pO_2 \), \( pCO_2 \), and pH remained essentially unchanged throughout the period of recording tissue \( pO_2 \) from the spinal cord.

**Pathological Examination**

**Method**

The spinal cords of all dogs used in these experiments were removed in their entirety, examined grossly, and fixed in neutral, buffered 10% formalin. A total of 11 spinal cords were selected for histopathological study. The spinal cord from a dog subjected only to laminectomy was chosen for one control; a second spinal cord from a dog subjected to laminectomy and cooling only was selected for a second control specimen. In the three groups of animals used for experimental evaluation, three spinal cords were selected from each group (Group 1: trauma only; Group 2: trauma and immediate cooling; Group 3: trauma and delayed cooling) for a total of nine specimens. Every cord studied histologically was cross-sectioned at the level of maximum impact (presumably the site of major injury), and sections were also made at 1-cm and 3-cm in-

*Fig. 2. Mean tissue \( pO_2 \) measured by needle electrode in five dogs subjected to 400 GCF. Shaded area represents extremes of values obtained at each point in time.*

*Fig. 3. Mean tissue \( pO_2 \) at 0.5 mm distances above and below center of impact injury in 10 dogs subjected to 400 GCF trauma.*
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tervals rostral and caudal to the center of the lesions.

All tissues selected for microscopic study were dehydrated in alcohol in the usual manner, embedded in paraffin, cut at 6 μ and stained by the following methods: hematoxylin and eosin for general cellular reactions, luxol blue and eosin for myelin, Bielschowsky silver for axons in a few cases, and Holzer’s method for astrocytes.

All microscopic sections were initially examined without knowledge of the experimental techniques or the clinical results. After recording the results and preparing tracings of each case, we reexamined the slides for correlative purposes but no readings were changed. In addition to the usual microscopic evaluation, myelin-stained sections from the point of maximum impact were traced at X5 magnification with a Bausch & Lomb microprojector.

Results

Gross Alterations. Figure 4 illustrates the range of alterations in the nine cases from the three study groups. The gross alterations of all cases in the three study groups were similar and consisted of varying degrees of focal epidural hemorrhage, fibrosis, and nodular loculation of epidural fat. In the multiple cross sections, the major change was an extremely variable central cavitation, which in the large lesions often extended 1 to 1.5 cm caudal and rostral to the site of major impact. In a few specimens, minimal cavitation with blurring of the internal architecture of the cord was present. The extent of the lesions can be assessed by referring to Fig. 4.

Cross sections through the spinal cords of the dogs subjected only to laminectomy and to laminectomy and cooling without trauma are shown in Fig. 5. Hypothermia produced no significant alterations. Also in Fig. 5 are

![FUNCTIONAL RECOVERY](image)

Fig. 4. Tracings of cord cross-sections at the trauma area from selected animals in the normothermic control group, Test Group 1, and Test Group 2.

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cross sections of spinal cords of animals which had been traumatized and cooled. CL showed only minimal central cavitation and persistent myelin edema, whereas C demonstrated marked alterations in the architecture of the cord. Tracings of the cross sections of the cords demonstrated the wide variations in gross alterations produced by the same degree of trauma. There was a correlation between the degree of central cavitation produced and the degree of recovery, particularly in the totally paraplegic animals. Hypothermia, whether immediate or delayed, did not seem to alter the results microscopically.

**Histological Changes.** In all cases the histological changes were qualitatively similar. The major changes were: 1) cavitation, often but not invariably central in location; 2) edema, pallor, and fragmentation of myelin sheaths in the white matter; 3) swelling and fragmentation of axons; 4) astrocytosis in and about the cystic lesions; 5) mononuclear phagocytic infiltration; 6) dural adhesions and meningo spinal scars; and 7) neuronal loss when the cavities destroyed gray matter extensively, although neurons that survived showed no morphological alterations in their perikarya.

**Discussion**

A 400 GCF applied to the spinal cords of adult mongrel dogs regularly produced permanent paraplegia but not with uniformity. Mechanical disruption occurred with a 480 GCF; even a 420 GCF produced disruption of the pia with extrusion of the cord substance in many animals.

Not all animals were rendered totally and permanently paraplegic with the 400 GCF delivered to the spinal cord. Two of our 14 control animals were completely normal neurologically at the end of 3 months. Many others had minor degrees of motor preserva-
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...tion, but only one dog had useful function. This variation in neurological deficit after trauma does not seem to be related to the size of the animal and is more likely a factor of chance.

All of the animals were paraplegic for a minimum of 48 hours after trauma. Thereafter, progress was rapid up to about 3 weeks, with only a few animals later improving measurably. Motor function proved to be the most reliable indication of improvement; some animals, even preoperatively, did not respond consistently to pinprick or pinching of the toes.

The eventual functional status of animals treated by hypothermia differed significantly from that of the controls. Eight of the 10 animals cooled immediately after trauma had significant recovery of function ($p < 0.01$). However, only three of these were completely normal. The other five showed ataxia of gait, inability to leap normally (weakness), and bladder disturbance (a large amount of residual urine). Two animals remained completely and totally paraplegic, even with immediate cooling.

Nine of the 14 animals treated by hypothermia after a 4-hour delay showed good functional recovery. This result is also significant but at a lower level ($p < 0.05$). Five of the good results were considered normal, and the other four displayed disorders similar to those in the immediately cooled group. Five animals in this delayed cooling group had no appreciable function following treatment.

Our results confirm those of Albin, and his associates, and of Ducker and Hamit that hypothermia improves the chances of recovery following experimental trauma to the spinal cord in dogs. Although we attempted to use precisely the same technique of injury, we could not uniformly produce complete and total paraplegia, even in the control animals, without mechanical disruption of the cord. All of our animals remained paraplegic for 48 hours, even if they eventually made a good recovery; this is contrary to the rapid recovery reported by Albin, et al. Those dogs cooled immediately after trauma recovered at a highly significant rate, but those cooled after a 4-hour delay recovered at a rate barely significant statistically. Results of the same order of significance have been obtained with the posterolateral longitudinal cordotomy by Freeman and Wright, and by Allen.

Grossly, all of the spinal cords developed subpial hemorrhage and discoloration after trauma. It could not be predicted, however, from gross observations of the cord following trauma, which animals would eventually show a good result and which ones would show very little recovery. The appearance of the cord also was not altered appreciably by cooling, either immediately or after a 4-hour delay.

Davies and Brink were the first to describe determinations of oxygen tension in living tissues. Since then, the physical and electrochemical properties of oxygen electrodes have been extensively studied. Three different but related methods of measuring oxygen tension in tissue have evolved: 1) the bare-tipped “open” electrode, usually made of platinum, gold, or stainless-steel; 2) the surface reservoir electrode; and 3) the membrane-covered needle electrode, which is a further modification of the principle of the surface reservoir electrode.

The bare-tipped electrode allows chronic implantation, but it remains doubtful that an open electrode system measures absolute oxygen tension in living tissues. Measurements of oxygen tension in tissue by means of the surface and needle modifications of the surface reservoir electrode have been made from many organs including the brain. Problems of calibration, control of temperature, and surface changes at the electrode’s interface introduce many potential errors. The introduction of the needle electrode into neural substance also modifies the architecture of the capillary bed, even in the absence of formation of hematoma.

In view of the limitations imposed by available instrumentation, it is apparent that no claim to absolute measurement of oxygen tension in tissue can be made in our own experiments. Because of the consistency of our results, however, we believe that our data reflect relative quantitative changes in the tissue $pO_2$ measured. Similarity of data obtained by surface and subsurface measurements of tissue $pO_2$ further support the validity of these quantitative changes.

The biochemical changes in the contused spinal cord on other than a histochemical basis have not been investigated extensively.
Using the polarographic determination of tissue pO₂ with a bare-tipped electrode, Maeda\textsuperscript{13} demonstrated that the fall in tissue pO₂ preceded the change in histopathological appearance. He reported a linear decline in tissue pO₂ in traumatized spinal cords over a 3-hour period.

In our experiments, the traumatized spinal cord became extremely hypoxic very shortly after injury and remained so for the extended period measured. This hypoxia was a local phenomenon occurring just at the site of injury; the tissue pO₂ was approaching a nearly normal level 1 cm above and below the center of impact. We have not determined how long the hypoxia persisted. Maeda,\textsuperscript{13} using chronically implanted electrodes, reported persistent hypoxia up to 72 hours following injury.

Photographs made at $\times 9$, $\times 16$, and $\times 25$ magnifications revealed subpial hemorrhage but no apparent vasospasm of external vessels of the injured cord. Figure 6 shows $\times 16$ photographs of the dorsal cord before and after injury. No change in the size and number of large and small vessels of the cord was observed. At 4 hours after injury, the cord usually measured only 1 mm wider than the width before trauma. An appearance of fullness could also be appreciated 4 hours after trauma. These gross observations suggest that the tissue hypoxia is probably more related to tissue edema with impaired O₂ tissue diffusion than to ischemia secondary to vasospasm.

In all animals in which it was measured, the spinal cord tissue pO₂ dropped precipitously after trauma. There was no correlation, therefore, with the pathological changes in the spinal cord or with the degree of re-

![Figure 6](image-url)
Hypothermia and pO₂ of spinal cord recovery of the animals. Possibly more refined recording or measurements over a longer interval might show differences in the tissue pO₂ of the animals that recovered and those that did not. Tissue pO₂ measurements under these circumstances would have some value.

The rationale for the use of hypothermia to modify the degree of recovery following trauma lies in the fact that tissue metabolism is reduced by hypothermia. This reduction in the need for O₂ might therefore decrease the permanent physiological and pathological alterations following trauma.

Summary
An experimental study of dogs subjected to a nondisruptive force to the spinal cord verifies that, in a significant number, paraplegia can be prevented by the use of local hypothermia. The tissue pO₂ of the spinal cord was found to be greatly decreased over a short segment of injured cord. It is postulated that local hypothermia offers some protection to the traumatized cord by decreasing metabolic demands.

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References

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