The benefits of experimental localized spinal cord hypothermia to prevent secondary damage following spinal injury have been reported by some and questioned by others. The technique has also been safely carried out in a few human cases, although the total anesthesia time for exploration is prolonged. The beneficial effects may be due to retardation of local metabolic reactions that produce ischemia, edema, and vasospasm in the injured cord.

The present experiments were designed to measure tissue temperatures achieved within injured and intact spinal cords during localized cooling.

Methods

Eighteen healthy mongrel dogs weighing 25 to 34 lbs were anesthetized with pentobarbital (30 mg/kg), intubated, and placed in the prone position. End-tidal CO₂ was kept between 4.5% to 5.5% utilizing a Harvard respirator and room air. In each animal a wide laminectomy was performed over T7-9, creating a reservoir of about 50 cc. In each case the control spinal cord temperature was identical with the deep body temperature. The inflow stream of refrigerated saline was directed at a level approximately two spinal segments from the outflow catheter. In half of the dogs the dura...
Spinal cord temperature during local hypothermia in dogs

TABLE 1

Spinal cord parenchymal temperatures during local hypothermia

<table>
<thead>
<tr>
<th>Dog No.</th>
<th>Dura Open</th>
<th>Trauma Bath</th>
<th>Alcohol Bath</th>
<th>Inflow 30 Minutes</th>
<th>Outflow 30 Minutes</th>
<th>Inflow 60 Minutes</th>
<th>Outflow 60 Minutes</th>
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</table>

was left intact, and in the other half the dura was opened and sutured to the adjacent exposed paravertebral muscles (Table 1). A twin roller pump* with volume gauges and a modified Harrison heat exchanger† (250 ml priming volume) delivered and recirculated refrigerated saline (100 to 150 cc/min) via two separate circuits to the reservoir and spinal cord, as previously described.5,6,8 A two-gallon container was used as an ice bath. In Dogs 1-6 and 16-18, ice water was the coolant used in the bath. In Dogs 7-15, isopropyl alcohol was combined with ice so that bath temperatures could be lowered to −4°C or below. Cooling was continued for 1 hour. The spinal cord was injured in Dogs 14-18 by a 300 to 500 gm-cm force applied to the dura (Table 1).2,4 These are severe injuries resulting in permanent paralysis in most dogs.3,4

Reservoir and bath temperatures were monitored by Fisher model FT15 thermometers.‡ Continuous deep body temperature and inflow and outflow spinal cord parenchymal temperatures were recorded on Yellow Springs Instrument Company (YSI) telethermometers model 41TD.§ Thermistor needle probes (YSI No. 524§) were introduced into spinal parenchyma under direct vision. Esophageal and rectal probes (YSI No. 401§), thermistors, and thermometers were calibrated according to manufacturers’ specifications both before and after procedures. Deep body temperature was kept at 36.5°C (±2°C) by heating blankets. The mean aortic arterial pressure (MAP) and electrocardiogram were continuously monitored on a Sanborn polygraph.

Results

Injured cord temperatures varied from 1.0°C to 3.8°C at inflow sites and 2.0°C to 13.7°C at outflow areas. Inflow tissue temperatures approximated reservoir temperatures. A drop in cord temperature was achieved within 60 seconds in the 500 gm-cm animals and 150 seconds in the 300 gm-

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*Twin roller pump manufactured by International Medical Instrument Corporation, Stoneham, Massachusetts 02180.
†Heat exchanger model 69051 manufactured by Harrison Radiator Company, Division of GMC, Lockport, New York 14094.
‡Fisher ASTM thermometer made by Fisher Scientific Company, 26401 Miles Avenue, Warrensville Heights, Ohio 44128.
§Telethermometers and thermistor probes made by Yellow Springs Instrument Company, Yellow Springs, Ohio 45387.

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cm group (Table 1). The stream of cold saline was directed at the injury site, and outflow was two segments higher in a grossly undamaged area; it is interesting that cord temperatures two segments away from the site of injury showed this change both in the speed and extent of the temperature reduction. Temperatures in non-injured cords varied between 5.4° and 23.5°C, and took 10 to 15 minutes to stabilize at the lowest possible level. Once achieved, the temperatures remained reduced for 1 hour (Table 1).

In alcohol-cooling experiments, bath temperatures varied between −18° and −4°C, and in water-cooling experiments, between 0° and 2°C. Reservoirs in all animals varied from 1° to 5.5°C independent of bath temperatures, although alcohol-cooling showed a tendency to lower the reservoir temperature. In all experiments, spinal inflow parenchymal temperature was either lower or about equal to the outflow tissue temperature (Table 1). No significant variation in cord temperature could be attributed to an open dura, although there was a trend toward a slightly lower parenchymal temperature.

Control MAP varied between 120 and 165 mm Hg. During cooling, MAP varied between 110 and 160 mm Hg. There was no relationship between spinal cord temperature reduction and MAP.

Discussion

The variation in the spinal cord parenchymal temperature in uninjured animals could not be attributed to differences in reservoir temperature, low systemic blood pressure with decreased flow, low body temperature, or dural protection. Variations in actual spinal cord blood flow in conjunction with individual physical factor differences such as the width of the neural canal, size of the spinal cord, etc., may be of great importance. The rapid and marked reduction in the temperature of the injured spinal cord to that of the reservoir indicates impaired blood flow with lack of heat transport into the damaged area. Spinal cord ischemia implying stagnation of blood in areas of contusion has been previously demonstrated by metabolic, anatomic, and tissue oxygen studies. The slower rate of temperature drop in intact compared to injured cord is due to retained heat conduction properties in the circulating blood. A temporal relationship between heat reduction and magnitude of injury was noted. Local spinal cooling causes little or no fall in deep body temperature. The documentation of actual tissue temperatures in this study verifies the magnitude of the temperature reduction assumed previously; it seems likely that the findings would be similar in humans.

Local cooling of both brain and spinal cord have been the subject of previous investigations. Negrin described techniques of localized brain and spinal cord perfusion without tissue temperature measurements. Albin, et al., achieved spinal cord temperatures of 11.8° ± 2.3°C in dogs utilizing similar perfusion techniques. Although temperatures recorded in noninjured areas approximate their results, our experiments show a marked reduction in temperature in injured areas. Ommaya and Baldwin in studies on dogs, cats, and monkeys showed that 20°C could be reached 1.5 cm from the cerebral surface utilizing perfusate at 12°C. Similar thermal characteristics were recorded in humans.

In the sustained perfusion cooling of subarachnoid space reported by Albin, et al., spinal cord temperatures of 5.2° to 7.8°C were reached. Silastic catheters were introduced at T-4 and L-5, and the perfusate gradually warmed as it passed from inflow to outflow site. Meyer and Hunter measured changes in the size of the cerebral cortical arteries and veins during surface cooling in animals and noted a reduction in the caliber of vessels beginning at 32°C. Changes were most evident in arteries having a diameter of 200 μ. Abolition of evoked cortical response to sciatic stimulation occurred at spinal temperatures between 12° and 15°C, indicating considerable retention of physiological function at levels of hypothermia considered therapeutic.

References


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