Recovery of vasomotor response in human spinal cord transection


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Vasomotor responses of the skin of the thumb and the big toe were measured in normal subjects and patients with spinal transection at a neutral ambient temperature of 22.0 ± 0.5°C and in a cool (12.0 ± 0.5°C) and warm (32.0 ± 0.5°C) environment. The vasomotor response of the hand and foot to the cooling and warming of part of the opposite upper or lower extremity was also recorded. Spinal transection at T5-11 abolishes acutely all vasomotor responses in the paraplegic lower extremities, but does not alter the responses in the upper extremities. By 4 months, the vasomotor tone in the lower extremities at a neutral ambient temperature returned to normal values as did the response to a cool and warm environment. The crossed vasomotor reflex to cooling and warming one lower extremity recovers more slowly, requiring a period of 18 months for complete recovery. The slower recovery of the vasomotor reflex in spinally transected man than in similarly treated dogs is thought to be due to the greater spinal shock in the former. The recovery of vasomotor responses in the paraplegic limbs to cooling and warming after thoracic transections suggests that these responses are primitive and powerful thermoregulatory mechanisms.

KEY WORDS spinal cord injury vasomotor response

It is well established that cooling or warming of one hand elicits cutaneous vasomotor response of the contralateral hand and both legs in normal subjects. Yet, in patients with spinal transection, Cooper, et al., failed to observe a similar cutaneous vasomotor response, and did not identify temperature-sensitive vasomotor centers in the isolated spinal cord in paraplegia. Johnson and Spalding also failed to find a significant change in blood flow in the hand when the leg was immersed in cold water (4°C) in patients with cervical spinal transection. In addition, Appenzeller and Schnieden and Benzinger all suggested that this kind of reflex depends on brain-driven central impulses passing through an intact pathway above the C6-7 level of the spinal cord.

In contrast, Corbett, et al., observed that cooling of the abdominal skin elicited vasoconstriction on the toes in patients with thoracic spinal transection. Sahs and Fulton demonstrated that cooling of the leg elicited cutaneous vasomotor response of the opposite leg in the thoracic spinal cord-transected monkey. Furthermore, our laboratory has recently observed similar results in dogs with chronic thoracic spinal transection.

As it has been demonstrated that dogs with acute spinal transection failed to exhibit these vasomotor reflexes due to spinal shock, it is reasonable to assume that patients with spinal transection, who suffer greater shock, would require longer recovery periods and may have poorer spinal vasomotor reflexes. It is the purpose of this paper, therefore, to determine the time course and extent of recovery in patients with spinal transection of the cutaneous vasomotor reflexes below the level of complete cord injury.

Clinical Material and Methods

Five normal male subjects aged between 20 and 22 years, and nine adult paraplegic patients, both male and female, were tested in this study. The paraplegic patients had been clinically and surgically verified to have complete spinal transection between T-5 and T-11 (Table 1). They were quite healthy during this period, with no bedsores or genitourinary infection.

The skin temperature of the thumb and big toe was measured by a surface probe connected to an electrical calibrated thermometer to indicate the cutaneous

*Surface probe (Model 409) and thermometer (Model 46 TUC) manufactured by Yellow Springs Instrument Co., Yellow Springs, Ohio.
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The body temperature was measured by an oral thermometer placed sublingually. Two experiments were carried out both in normal and paraplegic persons.

In the first experiment, changes in skin temperature were measured in different ambient temperatures. These studies were carried out in a climate chamber that permitted control of environmental temperatures between 12.0 ± 0.5°C and 32.0 ± 0.5°C. The subjects were kept in a room with a normal ambient temperature of 22.0 ± 0.5°C for at least 1 hour before being moved to the cold or warm environment. The skin temperature of the thumb and big toe was recorded in the normal environment first, then during and after transfer to the cold or warm environment.

In the second experiment, we measured changes in skin temperature in response to peripheral cooling or warming of a part of one extremity. These studies were carried out in a climate chamber controlled at a normal ambient temperature of 22.0 ± 0.5°C. Peripheral cooling or warming of the skin was carried out by immersing one leg up to the knee or one arm up to the elbow in water of different temperatures (13°C to 45°C). The subjects were kept in normal room temperature (22.0 ± 0.5°C) for 1 hour before each experiment.

Results

The changes of the skin temperature in different ambient temperatures in normal subjects and patients with spinal transection are summarized in Table 2. In the normal control group, changes of skin temperature of both thumb and big toe in response to the changes of ambient temperature were clearly seen. The skin temperature of the thumb was always higher than that of the big toe, despite the changes of ambient temperature. In the patients, however, during the acute stage of spinal cord injury, the cutaneous vessels of the lower limb were dilated to some degree. The skin temperature of the big toe was elevated. The patients failed to adjust the cutaneous vasomotor activity below the lesion, but the changes in skin temperature of the thumb in response to the changes of ambient temperature were similar to those observed in normal subjects. After an average of 4 months, the cutaneous vasomotor tonus below the lesion gradually recovered in these patients, and the skin temperature of the big toe responded similarly to that of the normal subjects.

In normal subjects, immersion of one leg in hot (45.0 ± 0.5°C) or cold (13.0 ± 0.5°C) water elicited an abrupt elevation or drop in skin temperature of the contralateral leg. The average increase and fall in skin temperature was 1.3 ± 0.25°C and 2.5 ± 0.36°C, respectively, in 10 minutes (Table 3). Corresponding changes in skin temperature of both thumbs were also produced. In the acutely paraplegic patient, peripheral cooling or warming of one leg failed to induce this kind of vasomotor response. Approximately 18 months after injury, a significant crossed thermal response below the lesion returned. However, the skin temperature of the thumb no longer showed any significant changes in response to thermal stimulation applied to the leg. When one forearm was immersed in cold or hot water, vasomotor reactions were observed similar to those mentioned above.
TABLE 3  
Changes of skin temperature induced by immersion of one leg in water of different temperatures in normal and spinally transected cases*  

<table>
<thead>
<tr>
<th>No. of Cases &amp; Water Temperature</th>
<th>Normal Subjects</th>
<th>Cases with Spinal Transection</th>
<th>Normal Subjects</th>
<th>Cases with Spinal Transection</th>
</tr>
</thead>
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<td></td>
<td>Thumb</td>
<td>Acute Stage</td>
<td>Recovery Stage</td>
<td>Thumb</td>
</tr>
<tr>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>no. of cases</td>
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<td>3</td>
<td>7</td>
<td>5</td>
</tr>
<tr>
<td>water temp</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>45 ± 0.5°C</td>
<td>11.2 ± 0.20</td>
<td>10.2 ± 0.01</td>
<td>10.2 ± 0.03</td>
<td>11.3 ± 0.25</td>
</tr>
<tr>
<td>22 ± 0.5°C</td>
<td>10.2 ± 0.02</td>
<td>10.2 ± 0.03</td>
<td>10.2 ± 0.02</td>
<td>10.3 ± 0.04</td>
</tr>
<tr>
<td>13 ± 0.5°C</td>
<td>12.6 ± 0.35</td>
<td>10.2 ± 0.03</td>
<td>10.2 ± 0.03</td>
<td>12.5 ± 0.36</td>
</tr>
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</table>

*Skin temperature changes are mean ± standard error of the mean. Arrows indicate increase or decrease.
†Measurements are significantly different with p < 0.05, calculated from the Student’s t-test.

Discussion

The present study indicates that in patients with chronic spinal cord injury, changes of cutaneous vasomotor response to changes of ambient temperature are noted below the level of trauma. Also, a crossed thermal response is produced by the peripheral cooling or warming of one leg. However, during the acute stage of the injury, these patients failed to exhibit these vasomotor activities. It is obvious from these experimental findings that, after a period of recovery, patients with spinal transection are able to evoke a cutaneous vasomotor response to peripheral thermal stimulus below the level of spinal cord injury.

The acute failure of vasomotor responses below the level of the spinal cord lesion may be due to the abrupt loss of supraspinal influences and paralysis of vasomotor tone. During this acute period, which lasts nearly 4 months, the skin temperature below the level of trauma was higher than that above the lesion. The resolution of this skin temperature difference indicated the recovery of vasomotor tone and the return of vasomotor reflexes of the lower extremities in response to changes in the ambient temperature.

Leriche and Fontaine demonstrated the recovery of vasomotor tone in patients with thoracic (T8–10) cord transection, and suggested that the recovery was attributed to the existence of vasomotor centers in arterial walls. Sahs and Fulton, however, failed to confirm this hypothesis; they were unable to elicit a cutaneous vasomotor response below the level of transection in monkeys when the isolated spinal cord was removed. The present results agree with those of Corbett, et al., who found that cooling the abdominal skin in patients with cervical cord transection decreased the blood flow in the calf, while cooling the skin above the lesion did not.

In the present study, as in our experiments with dogs, it was noted that the vasomotor activity of the lower extremities of paraplegic patients recovered earlier in response to changes in ambient temperatures than to peripheral thermal stimulation delivered to one leg. This difference in recovery time may be due either to the size of the surface area cooled or warmed, or to the involvement of different sets of interneurons for the crossed vasomotor reflex, or to a combination of both factors.

The slower recovery time for vasomotor reflexes in response to changes of ambient temperature in the paraplegic patients (4 months), as compared with the spinally transected dog (1 week), correlates with the difference of severity in spinal shock to other reflexes in these two species. However, it is surprising that the vasomotor reflexes showed an equal amount of recovery in both species. The mechanism that causes this equal recovery is unknown.

The spinally transected patient, like the dog, failed to exhibit reflex shivering, while the appropriate vasomotor reflex responses to cooling and warming of the skin were readily elicited. This finding indicates that spinal vasomotor reflex activity is a primitive and powerful mechanism for thermoregulation.

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References


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