Sympathetic skin response in patients with spinal cord injury

T Ogura, T Kubo, K Lee, Y Katayama
Department of Orthopaedic Surgery, Kyoto Prefectural University of Medicine, Kyoto, Japan

Y Kira, S Aramaki
Department of Orthopaedic Surgery, Kyoto Prefectural Rehabilitation Hospital for the Mentally and Physically Disabled, Kyoto, Japan

ABSTRACT

Purpose. To assess the effectiveness of sympathetic skin response in evaluating peripheral sympathetic nerve activity of patients with spinal cord injury, and to report on the basic properties of sympathetic skin response.

Methods. Sympathetic skin response evoked by electrical stimulation was recorded from the palms and soles of healthy volunteers and patients with spinal cord injury.

Results. Sympathetic skin response was recorded in 17 healthy volunteers and 14 patients with spinal cord injury. Of the 4 waveforms, the shortest latency was obtained from the palm; the sympathetic skin response was 1.2 to 1.4 ms at all stimulated sites, 1.9 to 2.0 ms at the sole, with a difference of about 0.6 ms between the palm and the sole. None of the patients with spinal cord injury responded at either the upper or lower limbs. In patients with a thoracic cord injury, some responded at the upper limbs but none at the lower limbs; some responded at either upper or lower limbs; some responded at neither upper nor the lower limbs; and some responded at both upper and lower limbs. The conducting pathway of sympathetic skin response in the spinal cord for the upper limbs descends to the upper thoracic cord (T4–6), and the conducting pathway for the lower limbs departs from the spinal cord at the lower thoracic cord (T9–10).

Conclusion. It appears that sympathetic skin response should be used for the evaluation and morbid investigation of the functional abnormalities of the sympathetic nervous system in patients with spinal cord lesions such as spinal cord injuries, cervical spondylosis, and spinal canal stenosis.

Key words: nerve fibers; peripheral nervous system diseases; skin/innervation; spinal cord injuries; sympathetic nervous system/physiopathology
INTRODUCTION

Autonomic nerve function is often studied by observing the reaction of effectors at nerve terminals or measuring the metabolic products resulting from neural activity. Shahani et al.\(^1\) reported that waveform analysis of the potential change at the palm and sole, i.e. the sympathetic skin response (SSR), induced by electrical stimulation was useful for evaluating the function of sympathetic nerve postganglionic unmyelinated fibres.\(^2,3\) Recording the SSR is a non-invasive method for evaluating peripheral sympathetic nerve activity, using skin sweat gland function as an index. However, because of habituation and the effects of different psychological states, such as consciousness and emotion, the appearance of the evoked potential varies widely.\(^4-7\) Because of these factors, consensus has not been reached concerning the waveform analysis method.\(^8\)

In this study, we examined the SSR in healthy subjects and patients with spinal cord injury, and reported the findings on the basic properties of SSR.

PATIENTS AND METHODS

14 (8 male and 6 female) patients with spinal cord injury with a mean age of 42.4 years, and 17 (12 male and 5 female) healthy volunteers with a mean age of 29.6 years, were recruited into the study. The subjects were made to lie quietly in a supine position in a room with a room temperature of 25°C to 30°C. The recording electrodes were applied to both palms and the soles of both feet. The forehead, the median nerve at wrist, the umbilical region, and the tibial nerve at ankle were stimulated by applying rectangular electrical stimulation at 15 mA for 0.5 ms 4 times at an interval of 15 s or more at random (Fig. 1).

RESULTS

Responses of all healthy subjects was recorded. Of
the 4 waveforms, the shortest latency was obtained from the palm; the SSR was 1.2 to 1.4 ms at all stimulated sites, 1.9 to 2.0 ms at the sole, with a difference of about 0.6 ms between the palm and the sole. The mean (standard deviation) amplitude at the palm was 5.63 (4.11) mV after stimulation of the forehead; 6.81 (4.04) mV after stimulation of the median nerve; 3.87 (2.01) mV after stimulation of the umbilical region; 4.40 (2.52) mV after stimulation of the tibial nerve.

The mean amplitude at the sole was 4.50 (2.14) mV after stimulation of the forehead; 4.04 (2.06) mV after stimulation of the median nerve; 3.14 (1.44) mV after stimulation of the umbilical region; 3.66 (1.46) mV after stimulation of the tibial nerve. The amplitude tended to be smaller at the umbilical region than at other stimulated sites (Table 1). After stimulation of the median nerve, the shortest latency was 1.31 (0.21) ms at the stimulated sites, and 1.30 (0.19) ms at the non-stimulated sites, exhibiting no significant difference. The mean amplitude was 7.57 (4.53) mV at the stimulated sites, and 6.41 (4.58) mV at the non-stimulated sites, exhibiting significantly larger amplitude at the stimulated sites.

None of the patients with cervical cord injury responded at either the upper or lower limbs. In patients with a thoracic cord injury, some had response at the upper limbs but not at the lower limbs; some had response at neither the upper nor lower limbs; and some had response at both the upper and lower limbs (Fig. 2, Table 2). The shortest latency did not differ from that of healthy subjects after stimulation of the forehead, the median nerve, and the umbilical region. In patients with hypaesthesia in the umbilical region, the mean amplitude tended to be smaller after stimulation of the umbilical region.

DISCUSSION

Galvanic skin reflex (GSR) has been used to evaluate
the function of the autonomous nervous system since the late 19th century, and SSR, which is included in GSR in a broad sense, has also been used. In the present study, we examined the SSR in healthy subjects and patients with spinal cord injury, to determine the basic properties of SSR, especially factors affecting the magnitude of the mean amplitude and presence or absence of response.

In healthy subjects, the mean amplitude at all recording sites tended to be smaller after stimulation of the umbilical region than after stimulation of the other sites. After stimulation of the site where sensation was decreased due to thoracic cord injury, the amplitude decayed. Based on the above, the magnitude of amplitude seemed to be affected by the number of medullated nerve fibres effectively stimulated by electricity. Comparison of the stimulated sites and the non-stimulated sites in the palm revealed significantly greater mean amplitude at the stimulated sites (p<0.01) after stimulation of the median nerve. This may be partly attributed to the changes in the vascular flow surrounding the recording electrodes caused by contraction of the abductor muscle of the thumb at the stimulated site.

Next, we examined the SSR in patients with spinal cord injury, to determine the factors affecting the presence or absence of SSR. In all patients with cervical cord injury, all recording sites were paralysed, and thus neither the upper limbs nor the lower limbs responded. As for the patients with thoracic cord injury, those patients with an injury to the T1 thoracic cord (cases 7 and 8) did not respond at the healthy and paralysed sites after stimulation of these sites. Of the 3 patients with an injury to the T9–10 thoracic cord, 2 (cases 11 and 13) responded at the paralysed sites after stimulation of the normal sites. These results could be explained by the anatomy of the conducting pathway in the spinal cord—the conducting pathway of SSR of the upper limbs descends to the upper thoracic cord (T4–6) while the conducting pathway in the lower limbs departs from the spinal cord at the lower thoracic cord (T9–10), as stated by Fuhrer in 1971. However, case 10, an injury to the T7 thoracic cord, and case 14, an injury to the T10 thoracic cord, did not respond at the upper limbs or at the lower limbs. These 2 cases cannot be explained by the above-mentioned conducting pathway. These 2 patients had no paralysis in the upper limbs, but they had a long history of the disease, and were depressive and poor in response to stimuli. Thus, responsiveness to stimulation of the normal sites was decreased in the superior central nervous system, which might have resulted in non-responsiveness in the normal sites.

**CONCLUSION**

It appears that SSR should be used for the evaluation and morbid investigation of the functional abnormalities of the sympathetic nervous system in patients with spinal cord lesions such as spinal cord injuries, cervical spondylosis, and spinal canal stenosis. The following properties of SSR were derived from this study:

**Figure 2** Pattern of SSR in patients with spinal cord injury. (a) SSR in healthy volunteers was recorded at upper and lower limbs. (b) SSR in patients with thoracic cord injury was recorded at the upper limbs but not at the lower limbs. (c) SSR in patients with cervical cord injury was not recorded at upper or lower limbs.
The amplitude of SSR induced by electrical stimulation may be affected by the number of medullated nerve fibres stimulated effectively. It seems that the conducting pathway of SSR in the spinal cord in the upper limbs descends to the upper thoracic cord (T4–6), and the conducting pathway in the lower limbs departs from the spinal cord at the lower thoracic cord (T9–10). The superior central nervous system may have a role to play in determining the presence or absence of SSR in patients with spinal cord injury.

REFERENCES