Shoulder-Hand Syndrome in Hemiparetic Patients: Temperature, Sympathetic Skin Responses, and Nerve Latencies of the Affected and Nonaffected Upper Extremity

S. Hesse, M.D., M.T. Jahnke, M.D., R. Ehret, M.D., K.H. Mauritz, MD

Shoulder-hand syndrome (SHS) is a frequent complication in stroke rehabilitation. Its etiology is not completely understood. We studied twenty-one hemiparetic patients with and eighteen patients without SHS. Variables were palm temperature, sympathetic skin responses (SSR), and proximal nerve latencies of both the affected and nonaffected upper extremity. In patients with SHS, the paretic extremity was significantly warmer and showed enhanced SSR responses when stimulated on the affected side and prolonged proximal nerve latencies in comparison to the nonaffected side. There was no difference in these parameters between both sides in patients without SHS. The altered tonic and reflex mediated sympathetic responses in patients with SHS may be related to neuroapraxia of the axillary plexus due to malalignment of the hemiplegic shoulder. Therapeutic consequences are discussed based on the pathomechanism. Key Words: Hemiparesis—Rehabilitation—Shoulder-hand syndrome.

Introduction

Hemiplegic patients often suffer from a painful shoulder and may develop manifestations of the shoulder-hand syndrome (SHS) (1,2,3).

With its autonomic, motor, and sensory signs in the fully developed stage, SHS can be considered among the reflex sympathetic dystrophy syndromes (RSD) (4). The pathogenesis of RSD is generally explained in terms of a vicious cycle involving hypersensitization of polymodal nociceptors following tissue injury (5). Major symptoms of SHS are a painful shoulder at rest or during mobilization, edema of wrist and hand, vasomotor changes, and tenderness to palpation. The syndrome occurs several days to weeks post-stroke in up to 27% of stroke survivors (3,6) and has adverse consequences for the rehabilitation process as well as for the level of functional independence after treatment. The etiology of SHS in the hemiparetic patient is not completely understood. Its features frequently occur in conjunction with subluxation of the shoulder, paresis of the shoulder girdle muscles, and moderate spasticity (3). The painful shoulder as a possible precursor of SHS is related to a decreased range of shoulder rotation, which can be ascribed to spasticity (in particular to the M. subscapularis) and/or to connective tissue shrinkage (7-9). There are various therapeutic approaches toward both conditions, e.g., corticosteroids, phenytoin, stellate ganglion blockade, transcutaneous nerve stimulation, functional electrical stimulation, slings, and nerve blocks (2,3,10-13). In order to further elucidate the conditions leading to SHS in hemiparetic stroke patients and to provide a basis for a therapeutic approach based on pathophysiology, we measured skin temperature to detect changes in vasomotor activity and performed standard electrophysiological techniques to detect possible proximal nerve lesions.

The study also investigated sympathetic skin responses (SSR) to quantify the sudomotor activity of the sympathetic nervous system. The SSR is a polysynaptic reflex that receives substantial input from supraspinal structures,
including the mesencephalic reticular formation, the posterior hypothalamus, and the cerebral cortex, which can modify the response by the subjects' attention (14–17).

Methods

Two groups of patients who gave informed consent participated in the study.

Group A consisted of twenty-one patients with SHS, which was ascertained by the following clinical criteria:

- pain and tenderness on humeral abduction, flexion, and external rotation;
- pain and dorsal swelling in the hand over carpal bories;
- fusiform edema of metacarpophalangeal and proximal interphalangeal joints;
- changes in temperature, color, or dryness;
- loss of dorsal skin lines and potential change of fingernails.

Fourteen of the twenty-one patients were men; seven were women. Their mean age was 62.3 years (range 43–79). Eight patients suffered from a right and thirteen from a left hemiparesis. Mean stroke interval was 3.3 months. Motor strength of the hemiparetic upper extremity was estimated by the Motricity Index (19); pinch grip, elbow flexion, and shoulder abduction were objectively rated from 0 to 100 points. For the patients of group A, the mean Motricity Index of the affected upper extremity was 41.1 points. Shoulder subluxation was clinically present in all but two patients and mean external shoulder rotation was 41/17° from neutral. Hemiparetic patients in group B did not suffer from SHS. Of the eighteen patients in this group, twelve were men and six were women. Their mean age was 58.6 (range 43–77) years; ten patients suffered from a right and eight from a left hemiparesis. Mean stroke interval was 3.8 months; mean motricity index of the affected upper extremity was 40.3 points; shoulder subluxation was clinically present in eight patients; external shoulder rotation was 69/16° from neutral. The two groups did not differ significantly with respect to age, interval post-stroke, or motor function. In both groups the etiology of stroke was a vascular lesion in the territory of the middle or anterior cerebral artery. Peripheral mono- or polyneuropathies and an anticholinergic medication were exclusion criteria.

Data Collection

Patients were seated comfortably in a room of constant temperature (23–25°C) and low noise level. They had emptied their bladders before the procedure. The skin temperature of both palms was measured (temperature resolution was 0.1°C). The sympathetic skin responses (SSR) were provoked by an electric single square pulse (0.5 millisecond, intensity adjusted to 20%-30% above the motor threshold) to stimulate the median nerve at the wrist first of the nonparetic and then of the paretic side. On both sites stimulation was repeated ten times and presented at irregular rates with intervals of at least sixty seconds in between to prevent habituation. Recordings were carried out using an electromyograph (Medelec Msystro MS 25) with an amplifier gain of 0.1 to 2 mV per division and filter settings at 0.2 to 50 Hz. Sweep duration was ten seconds. One disc Ag/AgCl electrode was attached to the palm and the second electrode to the dorsum of the hand. The SSR were recorded simultaneously on both hands. Mean amplitudes (peak to peak) and rectified areas (time interval from zero to ten seconds) of six consecutive SSR recordings of each side were included in the analysis.

F- and M-waves of the median nerve on both sides were measured in the conventional way and the corresponding F/M ratios were calculated.

Statistical comparisons were performed separately within each of the two patient groups using the Wilcoxon test. It compared temperatures, F/M ratios, and amplitudes and areas of the SSR of the paretic side with those of the nonparetic side. For this analysis the SSR values after stimulation of the nonparetic side were used. Additionally, the SSR responses of the paretic extremity obtained after stimulation of the nonaffected were compared with those after stimulation of the affected side.

A corrected significance level alpha of 0.01 was chosen and statistical standard software (SYSTAT™) was used for the calculations.

Results

The paretic extremity was significantly warmer in group A, while there was no notable difference between affected and nonaffected side in group B (Table 1).

The mean SSR amplitudes and areas of the paretic side were significantly larger compared to the nonparetic side in group A. There was no difference in group B (Table 1, Figures 1,2). The F/M ratio of the affected side was larger than the same ratio of the unaffected side in group A. Again, there was no difference in group B (Table 1).

The SSR responses (amplitudes and areas) of the paretic extremity obtained after stimulation of the nonparetic side were larger than those obtained after stimulation of the paretic side in group A, whereas in group B there was no notable difference between the two modes of stimulation (Table 2).
Table 1. Temperature, amplitudes, and areas of the sympathetic skin responses (SSR) and P/M ratios of the paretic and nonparetic upper extremity in group A with and group B without shoulder-hand syndrome

<table>
<thead>
<tr>
<th></th>
<th>Group A</th>
<th>Group A</th>
<th>Group B</th>
<th>Group B</th>
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<tbody>
<tr>
<td></td>
<td>Paretic</td>
<td>Nonparetic</td>
<td>Paretic</td>
<td>Nonparetic</td>
</tr>
<tr>
<td>Temperature (°C)</td>
<td>28.8 ± 0.55</td>
<td>27.9 ± 0.45</td>
<td>29.9 ± 1.02</td>
<td>29.8 ± 1.05</td>
</tr>
<tr>
<td>SSR Amplitude (mV)</td>
<td>1.69 ± 0.37</td>
<td>0.96 ± 0.22</td>
<td>1.40 ± 0.36</td>
<td>1.36 ± 0.30</td>
</tr>
<tr>
<td>SSR Area (mVa)</td>
<td>5.01 ± 1.11</td>
<td>2.73 ± 0.50</td>
<td>4.35 ± 0.89</td>
<td>3.96 ± 0.91</td>
</tr>
<tr>
<td>P/M ratio</td>
<td>7.48 ± 0.19</td>
<td>6.88 ± 0.19</td>
<td>7.39 ± 0.19</td>
<td>7.20 ± 0.16</td>
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Means ± SD; SSR = sympathetic skin response; * = significant at p < .05.

![Figure 1](image1.png)  
Figure 1. Sympathetic skin response (SSR) of a left hemiparetic patient with a definite shoulder-hand syndrome (male, 63 years, stroke interval 3 months); the SSR were recorded both from the paretic (upper) and nonparetic upper extremity (lower row) after electrical stimulation of the nonparetic (left) and paretic side (right column).

![Figure 2](image2.png)  
Figure 2. Sympathetic skin response (SSR) of a right hemiparetic patient without a definite shoulder-hand syndrome (female, 59 years, stroke interval 2.5 months); the SSR were recorded both from the paretic (upper) and nonparetic upper extremity (lower row) after electrical stimulation of the nonparetic (left) and paretic side (right column).
**Table 2. Amplitudes and areas of the sympathetic skin response of the paretic extremity obtained after stimulation of the affected and nonaffected side in group A and B.**

<table>
<thead>
<tr>
<th></th>
<th>Group A Stimulation</th>
<th>Group B Stimulation</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Nonparetic</td>
<td>Paretic</td>
</tr>
<tr>
<td></td>
<td>1.69 ± 0.37</td>
<td>0.98 ± 0.31*</td>
</tr>
<tr>
<td>SSR Amplitude</td>
<td></td>
<td>1.40 ± 0.36</td>
</tr>
<tr>
<td>(mV/A)</td>
<td></td>
<td>1.48 ± 0.36</td>
</tr>
<tr>
<td></td>
<td>5.01 ± 1.11</td>
<td>2.59 ± 0.74*</td>
</tr>
<tr>
<td>SSR Area</td>
<td></td>
<td>4.35 ± 0.89</td>
</tr>
<tr>
<td>(mV/A)</td>
<td></td>
<td>4.11 ± 0.81</td>
</tr>
</tbody>
</table>

Means ± SD; SSR = sympathetic skin responses; * = significant at p < .05.

**Discussion**

In agreement with previous reports, the paretic extremity was significantly warmer than the nonparetic one in patients with a definite shoulder-hand syndrome (20,21). Increased cutaneous temperature is generally attributed to reduced tonic sympathetic outflow (21). At the same time, the activity of cutaneous vasomotor reflexes (wave of vasoconstriction following deep inspiration) can be increased on the paretic side in hemiparetic patients (21).

Sympathetic skin responses were suppressed in brain infarct patients without SHS in comparison to normal subjects (22). There was no difference between the paretic and nonparetic side when the nonparetic extremity was stimulated (22) (cf. Table 1). This is in agreement with the results of the present study, in which there was no difference in hemiparetic patients not suffering from shoulder-hand syndrome.

In contrast, hemiparetic subjects with SHS showed enhanced responses on the paretic side when stimulated on the nonparetic side. This stimulation site corresponds to the protocol of Korpelainen and coworkers (22).

Consistent with the concept proposed by Herbaut et al. (21), disinhibited spinal mediatized sympathetic reflexes could perhaps explain this phenomenon. Since autonomic reflex responses have the tendency to generalize over several segments and to both sides, one would not have expected the asymmetry actually observed.

An alternative explanation would be supersensitivity to sympathetic neurotransmitters on the affected side. This hypothesis is supported by the finding that the concentration of noradrenaline and its intracellular metabolite (3,4-dihydroxyphenylethylenglycol) was reduced on the affected side as compared to the nonaffected side in patients with RSD (23).

In patients with SHS, the SSR responses of the paretic side were smaller when a stimulus was applied on the same side compared to the nonaffected one. In the hemiparetic patients without SHS there was no notable difference. Since both groups had comparable central lesions, this finding could be related to a defect in peripheral conduction. A lesion of the efferent sudomotor fibers is excluded because there was a normal or even enhanced response following stimulation of the nonaffected side. The prolonged F/M ratio on the affected side in the group of patients with SHS is consistent with the presence of a proximal lesion of the myelinated nerve fibers, although only motor efferent fibers are assessed by the F-wave. A recent study was unable to diagnose a brachial plexus injury or proximal mononeuropathy in hemiplegic patients, but the study did not exclusively include patients with acute and definite SHS (24).

Neuromagnasia of the axillary plexus could develop as the result of malalignment of the hemiplegic shoulder. Trauma to nerve sheaths is usually prevented by the locking mechanism of the shoulder (upward slope of the glenoid fossa, tension in the superior part of the capsule and the conchohumeral ligament) and the rotatory cuff muscles as the "guardians" of the shoulder (25). In hemiplegic patients the muscles are weak and the glenoid fossa slopes downward as a consequence of the dropping of the shoulder and winging of the scapula (26), resulting in a subluxed shoulder, which was present in all but two of our patients with SHS. The study does not firmly establish whether the neuropathia of the axillary plexus is the primary reason for, or the consequence of, the shoulder-hand syndrome.

Our hypothesis that peripheral nerve injury is a determining factor of the SHS is supported by the recent report of Braus et al., who also observed previous trauma of the affected shoulder in autopsies of seven patients and concluded that shoulder-hand syndrome is initiated by peripheral lesions (3).
The major therapeutic consequence of this study is the importance of preventing trauma to plexus fibers including surrounding connective tissue. Theoretical considerations of reflex sympathetic dystrophy syndrome imply a vicious cycle maintained by sensitization of nociceptor fibers following peripheral tissue lesion (5).

To prevent recurring pain injury, it is important to restore the natural locking mechanism by correcting the position of the scapula and to stimulate activity of the rotatory cuff muscles. Braus et al. showed that careful handling, avoiding damage to the shoulder, lowered the incidence of the shoulder-hand syndrome (3).

Unsupported elevation of the arm can aggravate this injury mechanism, as patients trained with an overhead pulley program had a markedly higher risk of developing a painful shoulder (27).

Acknowledgments. The study was supported by Maria-Sonnenfeld Gedachtnistiftung. The authors thank Ms. Daniela Lucke for technical assistance.

References