Lack of Sympathetic Involvement in Dermatitis Conflined to the Median Nerve Territory

A Case Report

ROBERTO CASALE1, MICHELANGELO BUONOCORE1, PATRIZIA TEOFOLI2, MARCO MATUCCI-CERINIC3 and MIKAEL ELAM4

1 Service of Clinical Neurophysiology, Medical Center of Montesacro, Foundation 'Salvatore Maugeri, Clinica del Lavoro e della Riabilitazione' IRCCS Pavia, Montesacro, 2 Department of Dermatology, University of Florence, Florence, 3 Institute of Internal Medicine, University of Cagliari, Cagliari, Italy and 4 Department of Clinical Neurophysiology, Sahlgrens' Hospital, University of Göteborg, Göteborg, Sweden

Both decreased and increased sympathetic nerve activity has been suggested as a possible underlying mechanism in inflammatory skin lesions. Modulation of sympathetic function has been proposed in the treatment of dermatitis. This case report describes the investigation strategy and normal findings in a case of dermatitis strictly confined to the median nerve territory, illustrating the need for specific tests of sympathetic function when pharmacological as well as physical sympatho-modulatory therapies are considered. Key words: skin sympathetic activity (SSA); microneurography; skin temperature.

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R. Casale, M.D., Service of Clinical Neurophysiology, Medical Center of Montesacro, Foundation 'Salvatore Maugeri, Clinica del Lavoro e della Riabilitazione' IRCCS—Pavia, I-27040 Montesacro (PV), Italy.

Dermatitis confined to the median nerve territory has been described in association with local compression of the median nerve in the carpal tunnel and attributed to impaired function of sympathetic vasoconstrictor nerve fibres (1). On the other hand, Bjorna & Kaada (2) in 1987 suggested that increased sympathetic activity to skin may generate and/or maintain dermatitis. They demonstrated a beneficial effect of transcutaneous electrical nerve stimulation (TENS) on dermatitis, which was attributed to improved cutaneous microcirculation due to TENS-induced sympathetic inhibition (2). Thus, both decreased and increased sympathetic nerve activity has been suggested as a possible underlying mechanism for dermatitic lesions. Inflammatory skin lesions are also found in the chronic stage of the reflex sympathetic dystrophy syndrome (RSD), suggesting the possibility of neural involvement in localized dermatitis (3). Therapies modulating sympathetic function should then be considered in cases with localized dermatitis. However, given the complex relation between sympathetic nerve activity and skin sympathetic effector organ function, especially after nerve injury (4), such therapeutic decisions should be based on a thorough investigation of sympathetic neural function.

The present report illustrates clinical and neurophysiological investigation strategy and findings in a case of dermatitis localized within the innervation area of a peripheral nerve.

CASE REPORT

A 49-year-old man complained of spontaneously occurring recurrent eczematous dermatitis, mainly affecting digits I-III of the left hand, with a duration of 10 years. Physical examination showed hyperkerato-...
Lack of sympathetic involvement in dermatitis

Neurophysiological testing was designed to investigate the following pathophysiological possibilities:
(a) entrapment of the median nerve in the carpal tunnel;
(b) subclinical polynuropathy;
(c) regional neurovegetative disturbance.

a) Macroneurography in left median and ulnar nerves showed normal sensory and motor conduction velocities, distal latencies and response amplitudes (Table 1). Electromyography in the median and ulnar muscles of the left hand was normal. b) In addition to a), macroneurography also included recording of motor and sensory conduction velocities in the lower limb which were normal (Table 1). c) Indirect evaluation of sympathetic nerve function included recording of sympathetic skin response (SSR: skin potential changes indicating neurally mediated sweat production) (5) and fingerprint photoplethysmography (PL: changes in capillary pulse volume indicating neural vasomotor function) (6). Direct record of multi-unit sensory afferent and postganglionic sympathetic nerve activity was performed (for details on methodology see Vallbo et al. (7)). Eight skin nerve fascicles with innervation territories which together covered the region of dermatitis were impaled (Fig. 3). In all fascicles normal sensory action potentials were recorded during tactile stimulation (i.e. stroking or stretching the skin within the receptive field) (Fig. 4). In three out of eight fascicles sympathetic activity was recorded, showing the normal characteristics of sympathetic discharges in skin nerves (Fig. 5). Stimuli known to transiently increase skin sympathetic neural discharge (i.e. arousal, mental stress, deep inspiratory gasps) induced normal sudomotor and vasomotor responses in the affected skin region (Fig. 5). Resting skin sympathetic activity was low, whereas inspiratory gasps or arousing stimuli (i.e. sudden noises or painful stimuli) evoked bursts of sympathetic discharge which were consistently followed by sudomotor and/or vasomotor reactions (cf. above).

### Table 1. The normal macroneurographic findings of sensory and motor conduction velocities recorded in the upper (median, ulnar) and lower (peroneal, sural) left limbs

<table>
<thead>
<tr>
<th>Nerves</th>
<th>Dist. Lat. (msec)</th>
<th>Ampl. (uV/mV)</th>
<th>Cond. Vel. (m/s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>motor</td>
<td>3.5</td>
<td>12.8</td>
<td>56.3</td>
</tr>
<tr>
<td>sensory</td>
<td>3.1</td>
<td>9.5</td>
<td>41.9</td>
</tr>
<tr>
<td>Ulnar</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>motor</td>
<td>2.7</td>
<td>16.4</td>
<td>53.1</td>
</tr>
<tr>
<td>sensory</td>
<td>2.4</td>
<td>7.7</td>
<td>43.7</td>
</tr>
<tr>
<td>Peroneal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>motor</td>
<td>3.5</td>
<td>11.6</td>
<td>46</td>
</tr>
<tr>
<td>sensory</td>
<td>4</td>
<td>15</td>
<td>36.2</td>
</tr>
<tr>
<td>Sural</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Fig. 2. Thermography showing the asymmetry in skin temperature between left and right hand (AGA Infrared Telethermovenion System).

Fig. 3. Receptive fields of multi-unit sensory afferents recorded in eight fascicles of the left median nerve.

Fig. 4. Record showing a spontaneously active sensory afferent fibre responding to slight stretching of the skin (•) in the area indicated by ●. The original nerve signal was fed through a band pass filter with a band width of 700–2000 Hz (Discr.). A mean voltage neurogram was obtained by passing the filtered neurogram through an RC integrating network with a time constant of 0.1 s (Rect.). The hatched area indicates the multi-unit sensory receptive field of the impaled fascicle.

**DISCUSSION**

Although the distribution of the dermatitis suggests a neurological involvement, the normal findings with macroneurography and electromyography make a local compression of the median nerve, and/or a subclinical polyneuropathy affecting large diameter myelinated nerve fibres, highly improbable. Macroneurography does not evaluate the function of thin unmyelinated (i.e. sympathetic) nerve fibres, and since experimental models of neuropathy have shown complete loss of sympathetic fibres also after mild compression of a peripheral...
Despite this controversy, regional sympathetic blocks are often performed on patients with pain syndromes associated with regional changes in skin temperature, also regardless of the direction of temperature change (cf. ref. 9). Since inflammatory skin lesions can occur in RSD and a sympathetic involvement was clinically suspected in the present case, both TENS and sympathetic blockade were performed in analogy with the traditional treatment strategy of RSD, both without beneficial effects and thus supporting a lack of sympathetic involvement in skin temperature changes found in this case. In summary, the present case did not show any sign of sympathetic or other nerve fibre dysfunction, arguing against the use of pharmacological or physical treatments modulating sympathetic function. The findings also underline the fact that regional alterations in skin temperature cannot be taken as evidence for altered sympathetic neural drive. Thus more specific tests of sympathetic function are necessary when planning therapeutic strategies in patients with localized pain, itch or other dysfunction such as dermatitis where a neurological disorder can be suspected.

REFERENCES