Effect of Sympathetic Nerve Block on Acute Inflammatory Pain and Hyperalgesia

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Background: Sympathetic nerve blocks relieve pain in certain chronic pain states, but the role of the sympathetic pathways in acute pain is unclear. Thus the authors wanted to determine whether a sympathetic block could reduce acute pain and hyperalgesia after a heat injury in healthy volunteers.

Methods: The study was made as a randomized, single-blinded investigation, in which the volunteers served as their own controls. A lumbar sympathetic nerve block and a contralateral placebo block were performed in 24 persons by injecting 10 ml bupivacaine (0.5%) and 10 ml saline, respectively. The duration and quality of blocks were evaluated by the sympathetic skin response and skin temperature. Bilateral heat injuries were produced on the medial surfaces of the calves with a 50 × 25 mm thermode (47°C, 7 min) 45 min after the blocks. Pain intensity induced by heat, pain thresholds to thermal and mechanical stimulation, and secondary hyperalgesia were assessed before block, after block, and 1, 2, 4, and 6 h after the heat injuries.

Results: Of the 24 volunteers, eight were excluded because of somatic block or incomplete sympathetic block. The study revealed no significant differences between sympathetic block and placebo for pain or mechanical allodynia during injury, or pain thresholds, pain responses to heat, or areas of secondary hyperalgesia after the injury. The comparisons were done for the period when the block was effective.

Conclusion: Sympathetic nerve block did not change acute inflammatory pain or hyperalgesia after a heat injury in human skin. (Key words: Hyperalgesia, acute pain, sympathetic nerve block, sympathetic nervous system and acute pain, thermal injury.)

THE sympathetic nervous system plays an important role in certain chronic pain states that are relieved by sympathetic nerve blocks, but its role in acute inflammatory pain is unclear. Norepinephrine has been shown to increase hyperalgesia to heat in human skin sensitized by capsaicin and to decrease heat pain thresholds after intradermal injection. Experimental evidence suggests that nociceptors in injured tissue are sensitized by sympathetic activity, and secondary hyperalgesia after capsaicin injection in the rat paw was prevented by sympathectomy and α1-receptor antagonists. Thus norepinephrine and sympathetic nerve stimulation may potentiate nociceptor sensitization in inflamed skin and perhaps sensitization in the central nervous system after injuries. Apart from inhibition of efferent activity, another possible explanation for pain relief by sympathetic nerve blocks is that afferents, which are not related directly to the autonomic nervous system, may travel together with sympathetic efferents, and the afferents become blocked together with the efferent fibers. These afferents normally may be silent, but they are recruited in inflammatory conditions, in which they may be responsible for sympathetic dependent pain. The two hypotheses (efferent and afferent fiber-mediated pain) are not mutually exclusive.

The importance of sympathetic pathways in acute clinical pain and hyperalgesia needs to be clarified, and thus we wanted to determine whether a lumbar sympathetic plexus block could reduce pain and hyperalgesia after a heat injury in healthy volunteers.
PEDERSEN, RUNG, AND KEHLET

Table 1. Timing and Order of Measurements

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Baseline</th>
<th>After Block and Before Injury</th>
<th>1, 2, 4, and 6 h after Injury</th>
<th>Timing (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin temperatures and</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>0–5</td>
</tr>
<tr>
<td>sympatogalvanic-skin response</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Skin erythema intensity</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>5–7</td>
</tr>
<tr>
<td>Warmth detection threshold</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>8</td>
</tr>
<tr>
<td>Cold detection threshold</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>9</td>
</tr>
<tr>
<td>Tactile detection threshold</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>10–11</td>
</tr>
<tr>
<td>Area of mechanical allodynia</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>12–13</td>
</tr>
<tr>
<td>Area of secondary hyperalgesia</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>14–19</td>
</tr>
<tr>
<td>Pain threshold to mechanical stimuli</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>20–22</td>
</tr>
<tr>
<td>Pain threshold to heat stimuli</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>23–24</td>
</tr>
<tr>
<td>Pain responses to heat (VAS) in</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>injured areas: 41, 43, and 45°C</td>
<td>Yes</td>
<td></td>
<td></td>
<td>25–28</td>
</tr>
</tbody>
</table>

Methods

Participants

We studied 24 healthy volunteers ages 22 to 46 yr (21 of them were men). Informed consent was obtained from all participants and the study was approved by the local ethics committee. Volunteers were interviewed about their health and were given a physical examination to ensure that they were healthy.

Procedures

The volunteers participated in a training session, where a heat injury was induced and all measurements were explained and performed. On the day of the study, baseline measurements were made and heat injuries were later induced in the same marked areas. Table 1 shows the timing and the order of the measurements. The right or left leg was randomly chosen for sympathetic block. All measurements were repeated when effectiveness of the block was confirmed. About 45 min after the block, a heat injury was induced on each leg, starting with the unblocked leg. During the injury, pain was assessed every min (visual analog scale), and the area of mechanical allodynia around the injury was mapped in the period 4 to 6 min after start of the injury. All measurements were made again 1, 2, 4, and 6 h after the injuries. The skin temperature was standardized to 35°C with the thermode when possible, because nociception and sensory thresholds may depend on skin temperature, which is elevated by sympathetic block. The experiments were performed in a quiet room at a temperature of 24–26°C, with the participants reclining in a relaxed position. They were instructed to keep their eyes closed during all measurements, which ensured that they did not know the results of the measurements. The participants were blinded to the treatment and they were not medically knowledgeable. The person assessing the response to the block and injuries could not be blinded to the treatment, because the changes caused by the sympathetic block are obvious to any investigator with medical knowledge.

Sympathetic Nerve Block. A unilateral lumbar sympathetic nerve block was performed according to a conventional technique. The skin was anesthetized with 1 ml 0.5% lidocaine injected through a 27-G needle 10–12 cm lateral to the third lumbar (L-3) spinous process. The L-3 spinous process was identified by the T-12 spinous process (the 12th costa) and the iliac crest. A 7-inch, 22-G spinal needle was passed through this point in a medial direction in the sagittal plane at a 45° angle to the skin. When the needle touched the lateral side of the L3 vertebral body, it was withdrawn to subcutaneous tissue, redirected slightly anterior, and advanced again until the tip of the needle passed just beyond the anterior edge of the vertebral body. Bupivacaine (0.5%, 10 ml) was injected at this location after aspiration tests. The identical procedure was performed on the contralateral side except that 10 ml normal saline was injected. Intravenous access and electrocardiographic monitoring were instituted before the blockade.

The duration and quality of the blocks were evaluated by the sympatogalvanic skin response (SGR) and clinical signs of block, such as differences between placebo and blocked leg in skin temperature, vein congestion,
erythema, and sweating. The blocks were evaluated at the same times as the other measurements. A sympathetic nerve block was identified by complete suppression of SGR, clear clinical signs of block, or both. Clear clinical signs of block were defined as a temperature difference between the placebo leg and the sympathetic nerve block leg of more than 1.5°C on the proximal part of the calf, along with other clinical signs of block.

The SGR was recorded by two electrocardiograph electrodes, which were located in the arch and the dorsum of the foot. The SGR signal was recorded synchronously from both feet during the entire study and stored by a Holter monitor. Symmetrical deflections in the SGR on the feet are seen after stimuli such as pain, deep breathing, and yawning. Absence of any SGR deflections is seen after a complete sympathetic block. Skin temperature was measured on the distal part of the foot (only in the last half of the study), in the injured areas, and just outside the injured areas. The temperatures were measured with an accuracy of ±0.1°C (Viking 3000; Frode Petersen & Co. AS, Alleroed, Denmark). Erythema was assessed with a skin reflectance spectrophotometer (explained later). Vein congestion and sweating were evaluated by inspection and rated (0, none; 1, little; 2, moderate; 3, heavy). Somatic nerve block was identified by increases in detection thresholds of warm, cold, and tactile stimuli.

**Heat Injury.** Heat injuries were produced on the medial surface of the right and left calf with a 50 × 25 mm thermode (Thermostat, Somedic A/B, Stockholm, Sweden). The thermode (47°C) was applied to the skin for 7 min under standardized pressure and caused a first-degree or a mild second-degree burn injury. Pain intensity was rated with a visual analog scale at the beginning of the heat injury and every minute afterward for 7 min, except for the fifth minute, when mechanical allodynia was assessed. The burn caused immediate intense stinging pain, which was followed by a moderate burning pain with a more diffuse quality during the rest of the injury. Spontaneous pain after the injury was not observed.

**Primary Hyperalgesia and Thermal and Tactile Sensation.** The mechanical pain detection threshold within the injured area was determined by pinprick with nine progressively rigid von Frey hairs (Lafayette Instrument Co., Lafayette, IN). We examined the force produced by each hair and confirmed that the steps from 1 to 9 represented logarithmic increases in force (hair 1 = 7 mN, 2 = 13 mN, 3 = 20 mN, 4 = 39 mN, 5 = 59 mN, 6 = 98 mN, 7 = 128 mN, 8 = 133 mN, and 9 = 514 mN). The mechanical pain detection threshold was defined as the lowest force (pinprick) that produced a sensation of pain or discomfort. Eight to ten pinpricks in the area of the injury were made with each hair from the thinnest until at least one half of the stimulations with one hair caused pain or discomfort. The threshold assessment was repeated three times at each measurement point, and the median was reported as the mechanical pain detection threshold. If hair 9 did not produce any pain or discomfort, we assigned the value 10 to that observation.

Tactile thresholds were also assessed using von Frey hairs. The measurements started with the thinnest hair, which was pressed against the skin until it flexed slightly and the participant was asked if he or she could feel it. If not, the next hair was applied and so on until the participant reported feeling the hair. The procedure was repeated in the reverse direction, in which the person reported when he or she could no longer feel the hair. Three ascending and three descending thresholds were determined and the median was calculated.

Thermal thresholds were determined with a computerized thermostat (Somedic A/B, Stockholm, Sweden). Heat pain detection threshold was the lowest temperature perceived as painful. Warm and cold detection thresholds were defined as the smallest changes from baseline that the participant could feel. The volunteers were instructed to press a button as soon as they perceived the specified sensation. All thermal thresholds were determined as the average of three trials performed at 9-s intervals, from a baseline temperature of 35°C, and with a rate of change of 1°C s⁻¹. The upper cutoff limit was 52°C.

Pain responses (visual analog scale) to heat were evaluated by stimuli of 41°C, 43°C, and 45°C lasting 5 s, preceded by 40°C for 5 s. This initial heating was necessary because the thermode cannot warm faster than 4°C s⁻¹.

**Secondary Hyperalgesia.** The area of mechanical hyperalgesia that developed around the burn injury was assessed with a rigid von Frey hair (314 mN). In most cases, pinprick with this hair is painful on distinct points of the skin but not universally uncomfortable. Thus hyperalgesia, not allodynia, was measured with the von Frey hair. Allodynia was assessed by gently stroking the skin with a fingertip. The skin was stroked perpendicular to the stimulation path for a distance of about 3 cm one time per second. Borders of hyperalge-
sia and allodynia were determined by stimulating along eight linear paths angled 45 degrees and meeting in the center of the injury. Stimulation along each path began well outside the hyperalgesic area and continued slowly toward the center of the heat injury, until the participant reported a definite change in sensation. With the von Frey hair, this was most often a change to a more intense pricking sensation with a burning after-sensation. Stroking was associated with a change from touch to a slight burning or smarting. The points of change were marked and later traced onto a clear acetate sheet. The areas of secondary hyperalgesia and allodynia were calculated from the marks using a vector algorithm.

**Inflammation.** To estimate the severity of inflammation, the intensity of erythema inside the injury was assessed using a skin-reflectance spectrophotometer (Dermaspectrometer; Cortex Technology, Hadsund, Denmark). The spectrophotometer provided a skin erythema index based on the absorption characteristics of green and red light in the skin. Measurements were made in six spots both inside and just outside the heat injury. All values were recorded and the mean was calculated.

**Statistical Analysis**

Distribution of data and differences between active block and placebo were evaluated using the Shapiro Wilk test. The aim of the study was to evaluate the effects of a sympathetic nerve block, and thus comparisons between block and placebo were based on summary measurements for the period with effective block. We considered the mean of measurements from the period with effective block as the best summary. The summary measurements were compared using the Student's t test for differences in a paired design, because all differences in presented results showed normal distribution. Differences in skin temperatures between blocked and placebo-blocked legs were evaluated separately at each measurement point, and the repeated Student's t tests for paired observations were adjusted with the Bonferroni correction to prevent mass significance from multiple comparisons.

The smallest differences between block and placebo that we could detect with the present variation, a power of 90% and a type I error of 5%, were calculated for all summary measures. The calculations are based on the Student's t test for differences in a paired design. The power of a study is the probability of detecting a real difference as statistically significant. Probability values less than 0.05 were considered significant.

**Results**

Of the 24 volunteers, eight did not fulfill the inclusion criteria because of incomplete sympathetic blocks (n = 4) or signs of somatic block (n = 4). Incomplete sympathetic block was defined by the absence of clinical signs of block and normal SGR, whereas somatic nerve block was identified by increases in detection thresholds of warm, cold, and tactile stimuli. Sixteen participants had obvious sympathetic blocks based on clinical signs, but in five of these the SGR was never completely suppressed. Thus analyses were made for the period with clinical block (n = 16) and for the period with completely suppressed SGR (n = 11). The results from the two sets of analyses were fully concordant. We present probability values from comparisons based on the period with clinical block. The median duration of the blocks according to clinical criteria was 5 h (range, 3–7 h) and 3 h according to SGR (range, 1–5 h). The duration of blocks according to clinical signs was significantly longer than according to the SGR criterion (P = 0.0008). Figure 1 shows the time course of skin temperature changes in the injured areas and in the distal parts of the feet. Temperatures on the feet were only measured in the last half of the study, which is why only 7 of 16 included volunteers have data concerning temperatures on the feet. The temperature difference between the legs at the last measurement before clinical recovery from block was a mean of 1.8°C on the calves and 5°C on the feet.

The study revealed no significant differences between sympathetic block and placebo as regards pain (P = 0.61; fig. 2) and mechanical allodynia (P = 0.80) during injury, or thermal (P = 0.23; fig. 3A) and mechanical pain thresholds (P = 0.25; fig. 3b), pain responses to heat (41°C, P = 0.57; 43°C, P = 0.72; and 45°C, P = 0.64; fig. 3c), or areas of secondary hyperalgesia (P = 0.50; fig. 3d) after the injury.

Erythema in the injury tended to be more intense in sympathetic-blocked legs than in placebo-blocked legs, but the difference was not significant (P = 0.08; fig. 4). The injury increased erythema significantly in the area of flare (sympathetic block, P = 0.0001; placebo, P = 0.009 for baseline vs. 1 h after injury), but no difference in erythema intensity was found between placebo and active block (P = 0.83; fig. 4).
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![Graph showing skin temperature changes in the injured areas and the distal parts of the feet.](image)

Fig. 1. Skin temperature changes in the injured areas and the distal parts of the feet. Presented values are means for 16 participants with clinically effective blocks. Temperatures on the distal part of the feet are from seven participants (see Methods). Dotted lines = legs with sympathetic block; solid lines = placebo-blocked legs. Filled dots represent the feet, and open dots represent injured areas. Time - 0.75 h indicates observation after block but before injury. Asterisks indicate significant temperature differences between sympathetic block and placebo in the injured areas. Comparisons were performed using Student's t test with Bonferroni correction. The Bonferroni correction implies that P must be less than 0.008 to be considered significant.

Table 2 shows the smallest differences between placebo and sympathetic block that we could detect.

Discussion

We examined the effects of a unilateral lumbar sympathetic nerve block on inflammatory pain after a standardized heat injury in healthy volunteers and compared these effects with the effects of a contralateral placebo block. The study revealed no significant differences between sympathetic block and placebo as regards pain or mechanical allodynia during the injury, or pain thresholds, pain responses to heat, or areas of secondary hyperalgesia after the injury.

The sympathetic nervous system plays an important role in some chronic pain states, in which pain is relieved by sympathectomy, sympathetic nerve blocks, and adrenergic antagonists. Pain in these conditions can be aggravated by applying adrenergic agents to the skin and perhaps by increases in sympathetic activity. The mechanisms of sympathetic pain are not clear, but efferents of and afferents related to the sympathetic nervous system may be important. The efferents may increase sensitization of nociceptors in acute inflammatory conditions. Efferent sympathetic activity may also cause pain by exciting nociceptors, mechanoreceptors, or both, especially in states with preexisting central sensitization such as nerve injuries, which can maintain central sensitization by ongoing ectopic nociceptive activity. The afferents that probably travel within the sympathetic nerves, which may normally be silent, may be recruited during injury. The afferents may also become a source of ongoing ectopic nociceptive activity with nerve injuries. In addition, ectopic activity in afferents could be evoked by sympathetic efferents because primary afferents in dorsal root ganglia may develop adrenergic sensitivity after nerve injuries, and nerve injury has been shown to trigger sprouting of noradrenergic axon terminals in dorsal root ganglia. Adrenergic excitation of cutaneous nociceptors also has been shown within days of nerve injuries. The development of supersensitivity to adrenergic substances in peripheral afferent neurons or other cells may also be an important mechanism in sympathetic-mediated pain, because plasma catecholamine concentrations were lower in limbs with reflex sympathetic dystrophy than in the contralateral unaffected limbs, and α1-adrenoceptor density has been shown to be significantly increased in hyperalgesic skin of patients with reflex sympathetic dystrophy compared with the skin of the pain-free limb. Correspondingly, sympathetic nerve activity has been

![Graph showing pain score (VAS) over time after start of injury.](image)

Fig. 2. Pain during heat injury (47°C, 7 min; medians). Dotted lines = legs with sympathetic block; solid lines = placebo-blocked legs. Pain was assessed using the visual analog scale 0–100. The response from each participant was summarized in the mean value of measurements during the injury. No difference between sympathetic block and placebo was found (P = 0.61 using Student’s t test; n = 16).

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shown to be normal in a patient with reflex sympathetic dystrophy with marked skin vasoconstriction.25 Finally, Bossut et al.26 found the development of adrenergic excitability of cutaneous C-fiber nociceptors and signs of denervation supersensitivity after sympathectomy.

Adrenergic excitation of cutaneous nociceptors does not occur in normal skin, because neither norepinephrine nor sympathetic efferents excite C-fiber nociceptors.4,29 A microneurographic study in healthy volunteers indicated that there is no evidence for a sympathetic modulation of C-fiber afferent transmission,14 and sympathectomy in five patients did not affect thermal perception thresholds, including heat pain thresholds.30 Together these data indicate that sympathetic activity does not excite or modulate nociceptors in normal skin, nor
SYMPATHETIC NERVE BLOCK AND ACUTE INFLAMMATORY PAIN

Fig. 4. Skin erythema intensity in the areas of injury and in the flare around the injured areas (means). Dotted lines—legs with sympathetic block; solid lines—placebo-blocked legs. Filled dots represent injury, whereas open dots represent flare. The response from each participant was summarized in the mean value of measurements during effective block. Comparison revealed no difference between sympathetic block and placebo (P values are shown in the figure; Student's t test; n = 16).

do sympathetic fibers transmit afferent information from the skin of limbs.

Injured tissues may react differently than normal tissues because nociceptors may become sensitized by sympathetic activity in skin and other tissues after inflammatory changes, and secondary hyperalgesia after capsaicin injection in the rat paw was prevented by sympathectomy and α1-receptor antagonists. Human studies have shown that norepinephrine increased hyperalgesia to heat in skin that was sensitized by capsaicin, and intradermal norepinephrine injections (50 μl, 10 μM) decreased the heat pain thresholds and increased the pain ratings of a heat stimulus. The heat pain thresholds in these studies were decreased in the range of 1–1.5°C and were just significant. In contrast to these findings, two experimental studies have shown that sympathetic stimulation had no effect on the responses to heat of sensitized polymodal C-fiber nociceptors.

Our study indicates that sympathetic pathways are not clinically important in acute inflammatory pain from skin in humans. Apparently, sympathetic activity did not contribute to sensitization of nociceptors in injured tissue because the sympathetic nerve block did not change hyperalgesia or pain after the heat injury. Similarly, if any silent afferents travel within sympathetic nerves from the skin, they did not contribute significantly to pain and hyperalgesia after the injury. Our model, however, does not preclude the possibility that epinephrine from the adrenal medulla contributes to sensitization of nociceptors after injury, because this humoral component of the sympathetic response is not controlled by the nerve block. To our knowledge, no evidence suggests that epinephrine in physiologic concentrations can sensitize nociceptors. A recent study with healthy volunteers by Liu et al. supports our conclusion. Capsaicin-evoked ongoing pain, areas of pin-prick hyperalgesia, and areas of mechanical allodynia were not changed by phenolamine (a nonspecific adrenergic antagonist), although significant differences in mechanical allodynia were found in 4 of 12 time points.

Acute postoperative pain in humans is reduced by epidural and intravenous clonidine, an α2 agonist, which induces a general decrease in sympathetic outflow. This suggests that the sympathetic nervous system may be involved in acute postoperative pain. However, the general decrease in sympathetic outflow is not a likely mechanism of postoperative pain relief, because the analgesic effects of α2 agonists are probably induced by spinal postsynaptic α receptors, which are.

Table 2. The Minimal Differences between Sympathetic Block and Placebo that Could Be Detected with the Present Variation, a Power of 90%, and a Type I Error of 5%

<table>
<thead>
<tr>
<th>Pain during Injury (VAS)</th>
<th>Mechanical Allodynia during Injury (cm²)</th>
<th>Primary Thermal Hyperalgesia (°C)</th>
<th>Primary Mechanical Hyperalgesia (v. Frey no.)</th>
<th>Heat Pain after Injury 41°C (VAS)</th>
<th>Heat Pain after Injury 43°C (VAS)</th>
<th>Heat Pain after Injury 45°C (VAS)</th>
<th>Secondary Hyperalgesia (cm²)</th>
<th>Erythema Injury (arbitrary unit)</th>
<th>Erythema Flare (arbitrary unit)</th>
</tr>
</thead>
<tbody>
<tr>
<td>7</td>
<td>21</td>
<td>1.2</td>
<td>0.5</td>
<td>4</td>
<td>7</td>
<td>8</td>
<td>14</td>
<td>1.5</td>
<td>1.1</td>
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</table>

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stimulated by analgesic descending fibers from the brain stem under physiologic conditions. Thus the well-established analgesic effects of clonidine seem more related to endogenous pain control systems than to the sympathetic nervous system.

Ineffective sympathetic blocks are an unlikely explanation of our results, because we assessed the effectiveness and duration of the blocks with the SGR in addition to clinical signs. The SGR is a measure of changes in skin potentials evoked by pain and deep breathing. The changes in potentials are caused by sympathetic innervation of eccrine sweat glands, and the response is absent (flat line) in persons with a congenital absence of sweat glands and after sympathectomy, complete sympathetic blocks, and treatment with atropine. The relation between SGR amplitude and skin sympathetic activity is a complex, nonlinear function, so SGR cannot be used as a quantitative estimate of the degree of block. For that reason we used SGR as an all-or-none response. The changes in skin potentials are symmetrical on the feet of healthy persons, and we monitored the responses from the feet synchronously and continuously during the entire study period. Some sympathetic efferent axons to the leg do not pass through any part of the sympathetic trunk, and this may explain why SGR was never eliminated in a few volunteers despite clear clinical signs of sympathetic block, including increased skin temperature, vein congestion, and erythema of toes. For that reason we made comparisons between sympathetic block and placebo for both clinical and SGR criteria, and the results were fully concordant.

The relatively small number of participants (16) may have caused us to miss some real differences. Table 2 shows the smallest differences between sympathetic and placebo-block that could be detected with a power of 90%. Such differences, however, appear to be clinically insignificant.

Our data show that a sympathetic nerve block does not reduce acute clinical pain in healthy skin, nor does it reduce induction of primary and secondary hyperalgesia after a heat injury.

References


