

*Strain et al reply:*

We found the comments of Dr Bowsher on our article very informative, and a suitable starting point for a discussion on the diagnostic value of heat pain sensitivity testing. His main concern was that the mean heat pain threshold in the dermatome ipsilateral to the chronic nerve root compression was 48.1°C, which he found unaccountably high. We wish to point out that our controls' value was 47.3°C in this study and that we had obtained a value of 46.5°C at the same sites in a much younger group of healthy subjects.<sup>1</sup> We therefore think the question is why are our values consistently higher than those reported by Bowsher, which are in the range of 41°C to 43°C.

Even with contact thermodes, the heat pain threshold cannot be considered as a physiological constant given in °C. It is clearly dependent on the physical characteristics of the thermode and the measurement procedures. We will give some examples of our experience. We recently changed from Marstock type thermodes (used also in the study under discussion) to a more advanced model with the same surface area (6 cm<sup>2</sup>). Because of the different characteristics of these thermodes that is, the isolation layer between the Peltier elements and the thermode surface) an average lowering of the

thresholds of about 1.5°C occurred.<sup>2</sup> The heat pain thresholds of three age groups measured at the lateral dorsum pedis were 44.9(1.5)°C (17–29 years), 44.8(1.9)°C (30–44 years) and 45.7(1.2)°C (45–63 years). These values are still considerably higher than those given by Bowsher. We think that there may be two reasons.

First, threshold estimates in the early trials were lower and more variable than in the later ones. For example, the difference between the first and the eighth trial with measurements on the foot was found to be -1.3°C.<sup>3</sup> We therefore disregard the first three trials in evaluation. Second, with the traditional Marstock procedures the temperature increases start from temperatures around 30°C and may lead to what we would call "premature pain responses" at temperatures well below 40°C. This can again be clearly seen in a very recent study by Jensen *et al.*<sup>4</sup> To avoid this, we set the base temperatures to 38°C or 40°C, which are levels that have not been felt painfully by any of our patients or control subjects.

Considering these factors, we think that the pain threshold values we reported, although different from those of Bowsher, should be considered valid and that it is very unlikely that the difference between the pain thresholds measured ipsi- and contralaterally to the nerve root compression is the consequence of an ipsilateral threshold increase rather than a contralateral threshold decrease. (As a reminder, the contralateral value was 45.8°C and significantly smaller than the value of the health controls).

We read the findings of Bowsher on heat pain and warmth sensitivity in different patient groups with interest and have no difficulty agreeing with them. However, hypalgesic phenomena in diabetic, postherpetic and post-stroke patients do not exclude the possibility of hyperalgesic phenomena in

patients with chronic lumbosacral disc diseases as described in our study. We think there are important differences in pathophysiology. So far the testing of neuropathic conditions with heat pain stimulation has rarely resulted in strong evidence for hyperalgesic or hyperpathic changes. In a very recent publication,<sup>5</sup> however, Wall gave a great number of examples of hyperalgesic changes produced by different kinds of neuropathies and he also pointed to the fact that a non-selective blockade of peripheral afferent impulses may lead to a "partial disinhibition" and, in consequence, to hyperalgesia. This is what seemed to have happened in our patients with chronic lumbosacral disc disease. That such an event might produce effects at the contralateral side appears not too speculative when the results of contralateral TENS-effects cited in our paper are considered. Taken together, we still believe that the conclusions drawn from the pilot study described are justified.

Finally, we want to answer the questions raised by Bowsher. Two patients were affected at the L5 root affection and 7 at the S1 root. We measured the thresholds at the medial (L5) and lateral (S1) side of the dorsum pedis, where the peripheral dermatomes are to be found, and verified the location of the dermatomes in the preceding neurological examination. As Bowsher expected, the dermatomes do normally not differ in warmth and pain sensitivity.

S LAUTENBACHER  
Max Planck Institut für Psychiatrie,  
Kraepelinstr 10, D-8000  
Munich, Germany

- 1 Strian F, Lautenbacher S, Galfé G, Hölzl R. Diurnal variations in pain perception and thermal sensitivity. *Pain* 1989;36:125-31.
- 2 Lautenbacher S, Strian F. Similarities in age differences in heat pain perception and ther-

- mal sensitivity. *Funct Neurol* 1991;6:125-31.
- 3 Galfé G, Lautenbacher S, Hölzl R, Strian F. Diagnosis of small fibre neuropathy: computer-assisted methods of combined pain and thermal sensitivity determination. *Hospimedica* 1990;8:38-48.
- 4 Jensen TS, Bach FW, Kastrup J, Deigaard A, Brennum J. Vibratory and thermal thresholds in diabetics with and without clinical neuropathy. *Acta Neurol Scand* 1991;84:326-33.
- 5 Wall PD. Neuropathic pain and injured nerve: central mechanisms. *Br Med Bull* 1991;47:631-43.

Table 1 Mean warmth and heat pain thresholds (°C) in normal feet

Age (mean)	17-30 (22.1)	31-45 (35.1)	46-60 (54.1)	61-74 (68.7)
Number	28	28	28	28
Warmth threshold (SD)	36.75 (3.22)	37.65 (2.89)	37.75 (2.88)	39.4 (4.03)
Heat pain threshold (SD)	41.5 (2.7)	41.75 (2.4)	43.0 (3.3)	43.1 (2.5)

Table 2 Mean (SD) warmth thresholds, warm-cold limen, and heat pain thresholds (°C) in neurogenic pain conditions

		Painful diabetic neuropathy	
64 Patients:	Warm-cold Limen (SD): 19.0 (8.8)	Heat pain threshold 46.6 (2.6)	
Median age: 54	unpaired <i>t</i> test: <i>p</i> < 0.000001	<i>p</i> < 0.00001	
28 Controls:	Warm-cold Limen (SD): 6.1 (2.65)	Heat Pain Threshold: 43.0 (3.3)	
		Postherpetic neuralgia	
39 Patients:	Warm Threshold (SD): 40.8 (0.9)	Heat Pain Threshold (SD): 45.8 (0.5)	
Median age: 69.5	paired <i>t</i> test: <i>p</i> < 0.000001	<i>p</i> < 0.00003	
39 Controls:	Warm Threshold (SD): 35.4 (0.3)	Heat Pain Threshold (SD): 43.1 (0.5)	
		Central post-stroke pain	
38 Patients:	Warm Threshold (SD): 40.5 (0.8)	Heat Pain Threshold (SD): 43.8 (0.4)	
Median age: 64	paired <i>t</i> test: <i>p</i> < 0.000001	<i>p</i> = 0.02	
38 Controls:	Warm Threshold (SD): 33.5 (0.4)	Heat Pain Threshold (SD): 42.7 (0.45)	
		Painful diabetic neuropathy	
64 Patients:	Warm-cold Difference (SD): 19.0 (8.8)	Heat Pain Threshold: 46.4 (2.6)	
Median age: 54	unpaired <i>t</i> test: <i>p</i> < 0.000001	<i>p</i> < 0.00001	
28 Controls:	Warm-cold Difference (SD): 6.1 (2.65)	Heat Pain Threshold: 43.0 (3.3)	
		Postherpetic neuralgia	
39 Patients:	Warm Threshold (SD): 40.8 (0.9)	Heat Pain Threshold (SD): 45.8 (0.5)	
Median age: 69.5	paired <i>t</i> test: <i>p</i> < 0.000001	<i>p</i> < 0.00003	
39 Controls:	Warm Threshold (SD): 35.4 (0.3)	Heat Pain Threshold (SD): 43.1 (0.5)	
		Central post-stroke pain	
39 Patients:	Warm Threshold (SD): 40.5 (0.8)	Heat Pain Threshold (SD): 43.8 (0.4)	
Median age: 64	paired <i>t</i> test: <i>p</i> < 0.000001	<i>p</i> = 0.02	
38 Controls:	Warm Threshold (SD): 33.5 (0.4)	Heat Pain Threshold (SD): 42.7 (0.45)	

### Disturbances of C-fibre-mediated sensibility in lumbosacral disc disease

I was interested to read the communication by F Strian *et al.*<sup>1</sup> There is difficulty, however, in accepting that heat pain threshold is lowered in the foot *contralateral* to the sciatic root compression, rather than raised in the foot *ipsilateral* to the lesion (as is the warmth threshold).

With JA Campbell, AW Chan, G Leijon and T Nurmiikko, thresholds to most somatosensory modalities have been measured, by the method of limits, at five body sites on each side in a large number of volunteers, as well as in patients with neurogenic pain conditions. In the case of the foot, a Marstock Peltier thermode measuring 25.0 × 50.0 mm (12.5 cm<sup>2</sup>) was applied just below the medial malleolus, where the skin is neither thickened nor hairy. Our results for warmth and heat pain in (1) the normal foot; (2) the diabetic foot, (3) postherpetic neuralgia, and (4) central post-stroke pain ("thalamic syndrome") are shown in table 1. In cases 3 and 4, comparison is made (paired *t* test) with the unaffected mirror-image area on the other side of the body, while in (2) age-matched normal subjects were used (unpaired *t* test). Our results suggest that while there is a large rise in the warmth threshold (average about 6°C), or twice this for the warm-cold limen, on the affected side, there is only a very small (average about 2°C), but significant, rise in the heat pain threshold, also on the affected side.

Strian *et al.* used a thermode measuring 6 cm<sup>2</sup>. With J Giewald, we have performed experiments using both large (12.5 cm<sup>2</sup>) and small (1.3 cm<sup>2</sup>) thermodes, and found that while warmth thresholds vary according to the surface area of the thermode, heat pain thresholds do not. We find the ipsilateral mean (SD) heat pain threshold of 48.1 (1.6)°C<sup>1</sup> unaccountably high compared with our normal thresholds (see table 2) and those found by others, and would suggest that the ipsilateral heat pain threshold in the patients of Strian *et al.* may be raised.

It would be helpful to know how many of their 9 patients had L5 root lesions and how many S1; where on the foot they measured heat pain thresholds for the two roots; and whether there is any difference according to

site, in both patient population and control subjects—although it must be admitted that our own results on the unaffected side of postherpetic neuralgia patients suggest that site makes little difference.

DAVID BOWSHER  
Pain Research Institute  
Walton Hospital  
Liverpool L9 1AE

1 Strian F, Lautenbacher S, Karlbauer G, Galfe G. Disturbances of C-fibre-mediated sensibility in lumbosacral disc disease. *J Neurol Neurosurg Psychiatry* 1991;54:1013-14.

#### Strian *et al.* reply:

We found the comments of Dr Bowsher on our article very informative, and a suitable starting point for a discussion on the diagnostic value of heat pain sensitivity testing. His main concern was that the mean heat pain threshold in the dermatome ipsilateral to the chronic nerve root compression was 48.1°C, which he found unaccountably high. We wish to point out that our controls' value was 47.3°C in this study and that we had obtained a value of 46.5°C at the same sites in a much younger group of healthy subjects.<sup>1</sup> We therefore think the question is why are our values consistently higher than those reported by Bowsher, which are in the range of 41°C to 43°C.

Even with contact thermodes, the heat pain threshold cannot be considered as a physiological constant given in °C. It is clearly dependent on the physical characteristics of the thermode and the measurement procedures. We will give some examples of our experience. We recently changed from Marstock type thermodes (used also in the study under discussion) to a more advanced model with the same surface area (6 cm<sup>2</sup>). Because of the different characteristics of these thermodes that is, the isolation layer between the Peltier elements and the thermode surface) an average lowering of the

thresholds of about 1.5°C occurred.<sup>2</sup> The heat pain thresholds of three age groups measured at the lateral dorsum pedis were 44.9(1.5)°C (17-29 years), 44.8(1.9)°C (30-44 years) and 45.7(1.2)°C (45-63 years). These values are still considerably higher than those given by Bowsher. We think that there may be two reasons.

First, threshold estimates in the early trials were lower and more variable than in the later ones. For example, the difference between the first and the eighth trial with measurements on the foot was found to be -1.3°C.<sup>3</sup> We therefore disregard the first three trials in evaluation. Second, with the traditional Marstock procedures the temperature increases start from temperatures around 30°C and may lead to what we would call "premature pain responses" at temperatures well below 40°C. This can again be clearly seen in a very recent study by Jensen *et al.*<sup>4</sup> To avoid this, we set the base temperatures to 38°C or 40°C, which are levels that have not been felt painfully by any of our patients or control subjects.

Considering these factors, we think that the pain threshold values we reported, although different from those of Bowsher, should be considered valid and that it is very unlikely that the difference between the pain thresholds measured ipsi- and contralaterally to the nerve root compression is the consequence of an ipsilateral threshold increase rather than a contralateral threshold decrease. (As a reminder, the contralateral value was 45.8°C and significantly smaller than the value of the health controls).

We read the findings of Bowsher on heat pain and warmth sensitivity in different patient groups with interest and have no difficulty agreeing with them. However, hypalgesic phenomena in diabetic, postherpetic and post-stroke patients do not exclude the possibility of hyperalgesic phenomena in

patients with chronic lumbosacral disc diseases as described in our study. We think there are important differences in pathophysiology. So far the testing of neuropathic conditions with heat pain stimulation has rarely resulted in strong evidence for hyperalgesic or hyperpathic changes. In a very recent publication,<sup>3</sup> however, Wall gave a great number of examples of hyperalgesic changes produced by different kinds of neuropathies and he also pointed to the fact that a non-selective blockade of peripheral afferent impulses may lead to a "partial disinhibition" and, in consequence, to hyperalgesia. This is what seemed to have happened in our patients with chronic lumbosacral disc disease. That such an event might produce effects at the contralateral side appears not too speculative when the results of contralateral TENS-effects cited in our paper are considered. Taken together, we still believe that the conclusions drawn from the pilot study described are justified.

Finally, we want to answer the questions raised by Bowsher. Two patients were affected at the L5 root affection and 7 at the S1 root. We measured the thresholds at the medial (L5) and lateral (S1) side of the dorsum pedis, where the peripheral dermatomes are to be found, and verified the location of the dermatomes in the preceding neurological examination. As Bowsher expected, the dermatomes do normally not differ in warmth and pain sensitivity.

S LAUTENBACHER  
Max Planck Institut für Psychiatrie,  
Kraepelinstr 10, D-8000  
Munich, Germany

- 1 Strian F, Lautenbacher S, Galfe G, Hölzl R. Diurnal variations in pain perception and thermal sensitivity. *Pain* 1989;36:125-31.  
2 Lautenbacher S, Strian F. Similarities in age differences in heat pain perception and ther-

- mal sensitivity. *Funct Neurol* 1991;6:125-31.  
3 Galfe G, Lautenbacher S, Hölzl R, Strian F. Diagnosis of small fibre neuropathy: computer-assisted methods of combined pain and thermal sensitivity determination. *Hospimedica* 1990;8:38-48.  
4 Jensen TS, Bach FW, Kastrup J, Deigaard A, Brennum J. Vibratory and thermal thresholds in diabetics with and without clinical neuropathy. *Acta Neurol Scand* 1991;84:326-33.  
5 Wall PD. Neuropathic pain and injured nerve: central mechanisms. *Br Med Bull* 1991;47:631-43.