of the technique and may, in part, account for the large intra-individual variability of the results that the authors have found on repeated determinations.

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References


Bertelsmann et al reply

Thank you for allowing us to reply to the comment of Dr Jamal. Although there are differences in cold and warm perception, detailed morphological studies of cold and warm receptors and fibre pathways are still lacking. Moreover there is no proof that neuropathy affects cold and warm perception separately. It is generally accepted that both cold and warm perception is conducted by thin myelinated and unmyelinated nerve fibres. With our equipment we are able to investigate cold and warm sense separately but this procedure is time consuming and gives information on the same type of nerve fibres. Therefore we determine a cold-warm index: thermal discrimination threshold. We agree with Dr Jamal that it is worth exploring this theme.

The topic of our article was to present a method to investigate thermal cutaneous sensation in normal subjects and in patients with diabetic neuropathy. In other studies we investigated different groups of diabetics to assess the sensitivity of our method. We found that a group of diabetics without complaints of neuropathy had a significantly increased thermal discrimination threshold in comparison with healthy volunteers.

Because thermal perception is related to the temperature of the skin, skin temperature is included in our test procedure. At the beginning of the test, skin temperature is measured and the first stimulator is set and maintained at this temperature. The temperature difference is adjusted and thus the temperature of the second stimulator is always related to the temperature of the first stimulator. It is our experience that after the subject is acclimatised in the examination room skin temperature does not change during the test.

Although on theoretical grounds a pure thermal stimulus is preferred, this manner of stimulation has some practical limitations. Using a spring mechanism application pressure of the stimulators is reproducible. We agree with Dr Jamal that automated application of the stimulators would be ideal.

In our opinion technical modifications would not result in a smaller variability of thermal discrimination thresholds. It has been argued elsewhere that the main part of variation in sensory thresholds is caused by central processing factors.

References


Simulated paraplegia: an occasional problem for the neurosurgeon

Sir: In their recent article on simulated paraplegia RS Maurice-Williams and H March stress that an important criterion for the diagnosis is the demonstration of inconsistencies. At the bedside one may note the absence of sphincter problems, and either normal reflexes and a non-anatomical distribution of sensory changes. Nurses may note movement when the patient is unaware of being observed.

An objective test of spinal cord function independent of the patient’s co-operation is necessary. To record somatosensory evoked potentials from the scalp following stimulation below the level of the lesion, the posterior tibial nerve at the ankle being a useful site. A normal response obtained from someone whose movement of feeling is a very important inconsistency. The somatosensory evoked potential tests the dorsal column, medial lemniscal system only and therefore is normal in pure spino-thalamic sensory loss. Pain and temperature loss with normal touch and pressure sense is not a pattern one sees simulated though it may become so as patients become more knowledgeable.

Another physiological test which may be of value, though I have no experience with it in simulated paraplegia, is stimulation of the motor cortex. A muscle twitch after stimulation gives an estimate of cortico-spinal tract function and conduction velocity. Simulated paraplegia is an obvious indication for the use of a non-invasive and objective test of spinal cord function which can aid diagnosis. Somatosensory evoked potentials is one such test and is now widely available and should be used.

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