Matters arising

The origins of lumbosacral spinal evoked potentials

Sir: We are pleased that Yiannikis and Shahani¹ were able to replicate both our data on the human spinal evoked responses following stimulation of the posterior tibial nerve (PTN)² and the study by Ratto *et al* on the spinal responses following stimulation of the PTN at the popliteal fossa.³ Yiannikis and Shahani¹ failed to cite our study in which we had demonstrated the presence of two distinct negative peaks recorded over the lumbosacral region following PTN stimulation at the ankle. In this study² we had established that the first negativity represents a rostrally travelling waveform with a brief refractory period consistent with a compound nerve action potential. The second event, the N22/P22 complex, is surface negative on the back and surface positive anteriorly; its amplitude is maximal 5–15 cm above the level of the L4 spine and its peak latency remains constant at all levels. The N22/P22 has a long refractory period. These characteristics of N22/P22 indicate that it is a localised, synaptically dependent event conforming to a transverse dipole with a dorsal negativity and a simultaneous anterior positivity. The N22/P22 is therefore probably generated in the dorsal grey of the spinal cord at the root entry zone.²

It has also been shown that following stimulation of the PTN at the popliteal fossa, a third negative peak, the W3 (Ratto *et al*), can be recorded. This is a caudally travelling waveform which represents activity in the ventral roots.³

Yiannikis and Shahani have replicated previously reported studies^{2,3} on the generators of the human spinal somatosensory evoked potentials following PTN stimulation. We however disagree with their contention that the peripheral nerve fibres contributing to the surface recorded spinal potentials following PTN stimulation are primarily muscle afferents. Cutaneous nerve fibres must make substantial contributions to these waveforms as we have shown⁴ that spinal potentials can be readily recorded following stimulation of the saphenous, superficial peroneal and sural nerves.

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References

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- 3 Ratto S, Abbruzzese M, Abbruzzese G, Favale E. Surface recording of the spinal ventral root discharge in man. *Brain* 1983;**106**:897–909.
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Yiannikis and Shahani reply:

We have read the letter from Seyal and Gabor with interest. We do extend our apologies in this regard for this citational oversight but I should point out that our paper¹ in no way replicated the work of Seyal and Gabor² nor that of Ratto *et al*.³ Indeed this work was completed in 1982 and, as it has obviously escaped the attention of the above authors, was published in summary form in a text-book on evoked potentials which I am sure is well known to these authors. *Evoked Potentials in Clinical Medicine* Chiappa; 1983;240–242.

Irrespective of this, our study is indeed unique in that it examines the spinal potentials from stimulation of the posterior tibial nerve in the popliteal fossa and ankle and the sural nerve which up until that point had been done independently. Furthermore scalp responses were also analysed in some of the subjects. By performing such extensive analysis and comparing mixed nerve with H-reflex to one without and then with a cutaneous nerve, disagreements in the literature about the number of travelling waves and their relationship to the H-reflex could be resolved. Clearly in doing so certain findings of previous authors^{3–5} were confirmed and put into context and quoted. The relevant sections of the study by Seyal and Gabor had been found by others previously⁴ and was as such quoted.

Our study confirmed the presence of three waves over the lumbar spine when stimulating the PTN in the PF. A DR negativity travelling rostrally arising from the dorsal roots, a standing SD potential from the lumbar spinal cord and a caudally travelling VR negativity representing ventral root

activity and subserving the H-reflex. These findings are not similar when using mixed nerves that do not subserve the H-reflex and cutaneous nerves where no VR potential was seen.

Furthermore we suggest a major contribution of the Ia afferents to the lumbar spinal potential (SD) produced when stimulating a mixed nerve. We agree that spinal potentials are obtainable from sural nerve stimulation, as has been demonstrated by our study and others. We would argue that they are not “readily” obtainable in all normals of all ages, and require extensive averaging. This is because they are of much lower amplitude than those from the PTN and this difference is not maintained at a cortical level. A simple difference in the number of cutaneous fibres stimulated would not readily explain this and it suggests greater post synaptic activity in the PTN most likely related to the Ia afferents.

References

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The Hyperventilation Syndrome—Research and Clinical Treatment

Sir: Dr Christopher Bass reviewed my book *The Hyperventilation Syndrome—Research and Clinical Treatment*, (1987). His work is well known to me. I have the highest respect for it, and I cite it in the book. I should like, however, to fine-tune his considered remarks.

Dr Bass is quite correct. This book is aimed at clinicians who deal with dyspnoea