Matters arising

The origins of lumbosacral spinal evoked potentials

Sir: We are pleased that Yiannikas and Shahani were able to replicate both our data on the human spinal evoked responses following stimulation of the posterior tibial nerve (PTN) and the study by Ratto et al on the spinal responses following stimulation of the PTN at the popliteal fossa. Yiannikas and Shahani 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 study 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.

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.

Yiannikas and Shahani have replicated previously reported studies 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 shown that spinal potentials can be readily recorded following stimulation of the saphenous, superficial peroneal and sural nerves.

Yiannikas and Shahani reply:

We have read the letter from Seyal and Gabor with interest. We do extend our apologies in this regard for this editorial oversight but I should point out that our paper in no way replicated the work of Seyal and Gabor nor that of Ratto et al. 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 were confirmed and put into context and quoted. The relevant sections of the study by Seyal and Gabor had been found by others previously 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 cord 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


Yiannikas and Shahani reply:

We have read the letter from Seyal and Gabor with interest. We do extend our apologies in this regard for this editorial oversight but I should point out that our paper in no way replicated the work of Seyal and Gabor nor that of Ratto et al. 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 were confirmed and put into context and quoted. The relevant sections of the study by Seyal and Gabor had been found by others previously 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 cord 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


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.
Matters arising

typically secondary to emotional disorders, and not physicians who treat dyspnoea. Nevertheless, a careful review of the physical conditions that accompany hyperventilation, and conditions that have been shown to be causally related to it are likewise detailed. And, the extensive research and clinical sources cited are overwhelmingly drawn from medical sources.

The statement that “hyperventilation can be diagnosed on the basis of symptoms alone” is, I regret to say, taken out of context. What I said is that hyperventilation can be determined only by examining alveolar PC02, but that since this procedure is not available to most practitioners, symptoms will have to suffice. And, I then listed the symptoms commonly acknowledged by, among others, the “Dutch” group.

My “aversion” to the use of the hyperventilation challenge arose from my discovery in the medical literature of warnings that it is a hazardous procedure. The sources of these warnings are, of course, cited in the book.

Quite correctly, Dr Bass includes coronary heart disease and epilepsy as the two reasons for avoiding it. Stroke has also been documented. This should give the reader a clue: serious acid-base shifts, blood pressure changes, and both cerebral and peripheral vasospasm have been frequently observed. Since many patients with hyperventilation report chest symptoms, it seems to me imprudent for anyone to employ this technique.

I did not propose the hyperventilation/hypoxia theory of anxiety and panic disorder. I merely cited it and endorse it.

As for the failure to cite recent work, I would like to point out that the book was issued in January 1987. The reader may note that there are 1986 references in it. The work to which Dr Bass refers, if memory serves me, was not available at that time. It is now, and a revised edition, updating references, is in the works.

The work on PC02 biofeedback was aimed at training patients with idiopathic seizures to produce normocapnia, and not to engender a meditative state, contrary to Dr Bass’s contention—although in some instances, such a state was indeed reported by them. And, contrary to his statement, at least four publications reporting controlled studies of the method are cited.

Finally, on a different note, it should be stated in all fairness, that the claim in the foreword, that my book will become a “medical classic,” was made by a prominent physician, partly based on the thoroughness of my citations of medical physiologists. And I hope that Dr Bass did not mean to imply that a psychologist cannot ipso facto contribute to medicine, which is, after all the science and art of healing. Conversely, numerous psychologists have contributed to psychology—behavioural medicine is much in favor of this hybrid.

ROBERT FRIED,
Professor of Psychology,
Head, Respiratory Psychophysiology Laboratory,
Hunter College,
Park Avenue,
New York
NY10021, USA

Reference
1 J Neurol Neurosurg Psychiatry 1988; 51:164.

Book reviews


The established position of Cerullo in the use of the laser in neurosurgery and the development of Chicago as a training centre has enabled him to bring together adequate contributions on most aspects of lasers in neurosurgery. Physicists contribute the prologue, which is an interesting account of the hesitant beginnings of laser applications in medicine, and also the clear chapter on the delivery of laser power from instrument to tissue. The customary account of laser safety in the context of American regulations is followed by the important consideration of anaesthesia for laser surgery and then comes Cerullo’s own chapter on extra-axial tumour removal. This is a fine account of making the entry into the tumour capsule at a point free of other structures (one might add that this can be nicely done with the CO2 laser sharply focused at 5 watts, and should immediately be followed by tumour biopsy) followed by removal of the contents using initially part-defocused low power laser and suction, and then with increased power density. A sharp focused high power density enables tumour removal by morcellation, which is a slow but less haemorrhagic alternative to ultrasound aspiration. The tumour capsule is then treated with a defocused laser to achieve dissection from surrounding structures. Variations of this technique for different tumours is described including the laser dissection of the acoustic neuromenoma from the facial nerve and internal auditory meatus. It would have been informative if this chapter had included some numerical assessment of results of the neurosurgical laser technique.

The paediatric chapter includes McLone’s detailed account of laser excision of spinal cord lipomyelomeningocele, and is less detailed on the removal of intra-cerebral and intra-spinal tumours.

Kelly’s chapter on glial neoplasms begins with an appreciation of the relationship between histology and CT and MRI imaging, based upon 600 stereotactic biopsies; peripheral low density on CT scans, and T2 prolongation on MRI represent brain parenchyma infiltrated by tumour cells, all beyond reasonable tumour excision, whereas CT enhancement represents solid tumour, amendable to resection by laser lobectomy or stereotactic laser excision if deeply placed, the resultant decompression and reduction of tumour burden giving extended survival times to those patients with low-grade gliomas, but making no significant difference to the length of survival in patients with high-grade gliomas. The section by Robertson and Clark on intrapial tumours gives an appropriate detail of the technique of laser/microsurgical excision of intramedullary gliomas which is one of the most significant applications of laser to neurosurgery, but here again analysis of patients treated and follow-up results would have been welcome.

There is a chapter on phototherapy followed by one on photoradiation of malignant brain tumours which together present the principles, technique, and initial results of what is more generally known as photodynamic therapy, the future of which rests with the development of more specific brain glioma sensitisation, the photoactivation being relatively easily achieved.

G BROCKLEHURST


This is an extremely well made, easily managed, book which deserves to be bought and read by postgraduate students preparing for examinations in psychiatry. It covers an important segment of that subject, in par-