

A MODERN PERSPECTIVE ON SOME OF THE MOST HIGHLY CITED JNNP PAPERS OF ALL TIME

Sensory nerve action potentials in patients with peripheral lesions

Thomas Sears



Dr Thomas Sears recalls his 1958 paper and discusses the clinical, physiological and technical background from the perspective of a basic scientist

Graduating in 1952, I could hardly have known that 60 years on I would be writing about this 1958 paper!¹ Had he lived, Roger Gilliatt would have provided a secure clinical perspective. Of necessity, my commentary reflects on the clinical, physiological and technical background from the perspective of a basic scientist. Initially my post at 'Queen Square' was physiologist in the EEG department under Dr 'Bill' Cobb, a distinguished electroencephalographer and electromyographer. In Stockholm, Bill had collaborated with Eric Kugelberg studying the post-ischaemic repetitive firing of motor nerve fibres after a pressure cuff is released.² Their use of two cuffs with pressure first released in the proximal one proved, (a) that the repetitive discharges arose in axons at the local site of compression induced ischaemia; (b) that conduction velocity slows *through* that region; and (c) that such repetitive activity of sensory fibres would explain and support Merrington and Nathan's view³ that post-ischaemic paraesthesiae arise in sensory nerve axons and not in the nerve terminals, as others believed. For paraesthesiae in the carpal tunnel syndrome, Brain, Wright and Wilkinson (1947) showed that surgical incision of the flexor retinaculum led to rapid relief of symptoms,⁴ as confirmed by Kremer, Gilliatt, Golding and Wilson in 1953, also using a pressure cuff

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to exaggerate symptoms, examining the more general problem of 'acroparaesthesiae'.⁵

Although motor nerve conduction velocity measurements had previously been made in diffuse neuropathic states (eg, in regeneration, Hodes, Larrabee and German 1948; poliomyelitis, Hodes, 1949; and peroneal muscular atrophy, Lambert, 1956 and Henriksson, 1956; see Lambert, 1956 for references⁶), it was our colleague Dr J 'Iain' Simpson who studied conduction through a discrete site of *nerve* and *vascular* compression, the carpal tunnel. He stimulated the median nerve at the wrist just proximal to the tunnel and also at the antecubital fossa and made EMG recordings from the abductor pollicis brevis using Adrian–Bronk concentric needle electrodes. In this way he demonstrated a marked slowing of motor conduction velocity distal to the wrist stimulation site, but otherwise not localisable. However, for a focal ulnar nerve lesion at the elbow, he showed that such slowing actually occurred *through* the lesion site and deduced that this must also be so at the carpal tunnel.⁷

In the mid-50s, Roger Gilliatt, a senior registrar at the Middlesex Hospital, came to help Bill Cobb with the EMGs, continuing to do so following his appointment as consultant neurologist at the 'Square' and the beginning of our collaboration. His aim was to measure sensory nerve conduction in patients with peripheral neuropathies, including carpal tunnel, by recording nerve action potentials through the skin, as described by Dawson and Scott in 1949.⁸ These low amplitude signals required 'averaging' and in that pre-computer age, the photographic method used depended on the fact

that any stimulus time locked signals would summate as 'latent images' in the photographic emulsion to form a clear and discrete image, whereas random electrical or biological 'noise' would not. We recorded compound nerve action potentials over the median or ulnar nerves at the wrist and elbow following electrical stimulation of the digital nerves. Excellent instrumentation by 'Bert' Morton coupled with good techniques was paramount for successful recording of the 5–20 μ V signals, of still lower amplitude, dispersed or absent altogether with pathology. We also studied patients with ulnar nerve lesions, chronic polyneuritis, peroneal muscular atrophy and brachial plexus lesions. Some patients returned for post-operative measurements and it was rewarding to obtain objective evidence of recovery. I also recall scepticism at the time from some clinicians averse to the work because of mismatch between clinical and laboratory findings. However, they did not appreciate that such dissociations helped to advance understanding of recovery processes dependent on remyelination, or axon regeneration and remyelination. Our discussion clearly emphasised that the test itself did not examine conduction most distally in the sensory axons or nerve terminals. I circumvented this problem by recording antidromically conducted nerve impulses through ring electrodes on the digits themselves. And these electrodes, or ones at the wrist, could record the highly synchronised digital nerve volley evoked by tapping the finger nail with an electromechanical device time locked to the oscilloscope trace. Dr Roger Bannister and I used these two approaches to investigate sensory nerve conduction as well as motor nerve conduction in a patient with Guillian–Barre syndrome, who was paralysed, ventilated and had impaired sensibility and numbness of 'stocking glove' distribution. We made serial measurements through to his good clinical recovery some 60 days later. Because conduction was slowed or blocked over extended lengths of nerve, this precluded axonal regeneration as the basis

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of his early recovery. We therefore invoked demyelination/remyelination as the most likely underlying basis of the clinical sequelae, the pathohistology and aetiology of the acute phase of Guillain–Barre syndrome being then uncertain, if not unknown.⁹ This experience was formative in my interest in demyelination, furthered during my time with Sir John Eccles in Canberra when Ian McDonald visited on his way to present his PhD work on experimental diphtheritic demyelination to the Australian Physiological Society. Ian was to further his training in neurology at Queen Square so we resolved then to collaborate in London if his work allowed it; fortunately, that proved to be the case and led to scientific collaborations, for example,¹⁰ to electrophysiological studies

in multiple sclerosis and, most importantly, a lifelong friendship.

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