ELSEVIER

Clinical Neurophysiology 117 (2006) 398–404

www.elsevier.com/locate/clinph

Cervical nerve root stimulation. Part II: Findings in primary demyelinating neuropathies and motor neuron disease☆

Steve Vucic, Kristin Black, Peter Siao Tick Chong, Didier Cros *

Department of Neurology, Massachusetts General Hospital, Bigelow 1256, 55 Fruit Street, Boston, MA 02114, USA

Accepted 12 October 2005
Available online 3 January 2006

Abstract

Objective: Cervical nerve root stimulation (CRS) allows the assessment of conduction in the proximal segments of motor fibers destined to the upper extremities, which are not evaluated by routine nerve conduction studies (NCS). Since many primary demyelinating polyneuropathies (PDP) are multifocal lesions may be confined to the proximal nerve segments. CRS may therefore increase the yield of neurophysiologic studies in diagnosing PDP.

Methods: We reviewed clinical and neurophysiologic data from 38 PDP patients and compared them to 35 patients with motor neuron disease (MND), and 21 healthy controls (HC).

Results: Mean onset-latency was significantly prolonged in PDP patients. The optimal onset-latency cutoff necessary to distinguish PDP from MND and controls was 17.5 ms for the abductor pollicis brevis (APB) and abductor digiti minimi (ADM), and 7 ms for Biceps and Triceps. Mean reduction in proximal to distal CMAP amplitude to APB and ADM was significantly greater in PDP patients, with an optimal cutoff in proximal to distal CMAP amplitude reduction necessary to distinguish PDP from MND and HC being 45%.

Conclusions: CRS is effective in distinguishing PDP from MND and HC based on prolonged onset latency and conduction block criteria.

Significance: CRS may increase the diagnostic yield in cases where demyelinating lesions are confined to the proximal peripheral neuraxis.

Keywords: Cervical nerve root stimulation; Demyelinating neuropathy

1. Introduction

Cervical nerve root stimulation (CRS) is a neurophysiologic technique of assessing the entire length of peripheral motor pathway, by stimulating motor axons within millimeters of the anterior horn cell and recording responses from target muscles (Barker et al., 1987; Berger et al., 1987; Vucic et al., submitted). Several CRS techniques have been reported utilizing magnetic or electrical stimulation. Magnetic CRS utilizes a rapidly varying magnetic field, produced by a magnetic coil placed over the lower neck area (Cros et al., 1990). Electrical stimulation is achieved by either a monopolar needle inserted at the C5/C6 or C6/C7 vertebral level (Berger et al., 1987; Sander et al., 1999; Vucic et al., submitted), or high voltage percutaneous electrical stimulation over the cervical vertebral column (Mills and Murray, 1986). For the purpose of this paper CRS will refers to needle electrical stimulation of the cervical spinal roots. Although *F* responses assess conduction across the proximal segments of the peripheral nervous system, only the largest and fastest conducting motor axons are reliably studied and pathology may be missed (Shahani et al., 1987). Furthermore, proximal conduction in nerves supplying proximal muscles, such as the Biceps Brachii, cannot be assessed with *F* responses.

Conventional nerve conduction studies (NCS) employed in diagnosis of primary demyelinating polyneuropathy (PDP) are specific, but of limited value when demyelinating lesions are confined to the proximal nerve segments (Ad

☆ Presented at the Biannual Clinical Neurophysiology Workshop of the Australian Association of Neurologists, April 19–23, 2005, Southport, Queensland, Australia.

* Corresponding author. Tel.: +1 617 726 3642; fax: +1 617 726 2019.
E-mail address: dcros@partners.org (D. Cros).

1388-2457/$30.00
doi:10.1016/j.clinph.2005.10.012

Hoc Subcommitee of the American Academy of Neurology AIDS Task Force, 1991; Hughes et al., 2001; Nicolas et al., 2002; Saperstein et al., 2001). CRS may overcome this limitation (Menkes et al., 1998). CRS may also be used to differentiate between the pure motor syndromes of multifocal motor neuropathy with conduction block (MMN) and motor neuron disease (MND), an essential differential diagnosis in the pure lower motor neuron variant of MND. Patients with MND may exhibit a drop in CMAP amplitude, as well as dispersion of the motor response on proximal stimulation not seen in normal controls (Vucic et al., submitted). This is because the combination of reduction in the number of motor units (MUs) and enlargement of the remaining motor unit potentials (MUPs), increase the likelihood of phase cancellation and phase shift over long conduction distances. Several authors have reported a reduction in CMAP amplitude of up to 45%, across the proximal nerve segment, in MND patients (Arunachalam et al., 2003; Raynor et al., 1998). This would suggest that a reduction in amplitude and area of $\geq$45% is consistent with CB. However, in cases where the distal CMAP amplitudes are low, because of severe anatomical or functional axonal loss, CB may no longer be a specific diagnostic parameter. In such cases, prolongation of CMAP onset latency may be the only reliable abnormality pointing to the presence of a primary demyelinating polyneuropathy (PDP). However, CMAP onset latency may also be prolonged in MND because of loss of large-diameter motor axons. Ranges for onset latencies in MND and PDP have not yet been reported.

The aim of this study was: (i) to assess the range of CMAP amplitude reduction and onset latencies in patients with PDP and with MND using CRS; (ii) to determine the optimal cutoff values for CMAP amplitude reduction and onset latency prolongation that reliably distinguishes PDP from pathological (MND) and healthy controls; and (iii) to determine the sensitivity and specificity of CRS compared to conventional NCS in the diagnosis of PDP.

2. Methods

2.1. Patients

We retrospectively reviewed the clinical and neurophysiologic data of 72 patients referred to our laboratory who underwent CRS between 1998 and 2003. Thirty-eight patients with PDP (25 with chronic inflammatory demyelinating polyneuropathy (CIDP) and 13 with MMN) were included. All CIDP patients met the American Academy of Neurology (AAN) clinical criteria (Ad Hoc Subcommitee of the American Academy of Neurology AIDS Task Force, 1991), while the MMN patients met the American Academy of Electrodiagnostic Medicine (AAEM) criteria of either possible ($n=7$) or definite MMN ($n=6$) (Olney et al., 2003). Additionally, 35 patients with a diagnosis of MND based on clinical and neurophysiologic criteria (El Escorial criteria; subcommittee on motor neuron diseases, 1994) were included. These data were compared to normal values in 21 healthy controls (HC) that were collected prospectively and reported in a separate manuscript (Vucic et al., submitted).

2.2. Neurophysiology

Nerve conduction studies (NCS) were performed on four channel Oxford Synergy equipment (Oxford Instruments, Old Woking, UK). The temperature of the upper extremities was maintained at 32 °C. Recordings from 4 target muscles (Abductor Pollicis Brevis (APB), Abductor Digiti Minimi (ADM), Biceps and Triceps) were obtained bilaterally. The median nerve was supramaximally stimulated at the wrist, elbow and Erb's point (EP), the ulnar nerve at the wrist, below the elbow, above the elbow and EP; the biceps and triceps motor fibers at EP.

For CRS, the subject was sitting comfortably in a chair with the neck in slight flexion. The cathode was a monopolar needle (20 gauge, 50 mm length, Oxford Instruments, Old Woking, UK), and the anode was a silver-plated 32 mm surface electrode. The monopolar electrode was inserted parasagittally perpendicular to the skin, at the C6/7 level, 1 cm lateral to the spinous process and advanced to the vertebral lamina. The cathode was ipsilateral to the side of recording. The anode position was varied, 3–4 cm rostral, lateral, or caudal to the cathode, to ensure supramaximal responses. At each anode position, 3–4 stimuli were delivered to ensure a maximal CMAP response. As such, between 9–12 stimuli were delivered per recording side. For EP stimulation, the cathode was a standard 10 mm silver–silver chloride disc in the supraclavicular area, and the anode was a silver-plated surface electrode positioned over the ipsilateral scapula (Roth and Magistris, 1987; Cho et al., 2001). CMAPs were recorded using standard Ag/AgCl disk electrodes in a belly tendon arrangement. A collision technique was performed with CRS and EP stimulation when recording from the APB muscle, to eliminate the ulnar nerve contribution to the innervation of the thenar eminence (Kimura, 2001). Stimulus intensity was supramaximal at all sites of stimulation (100 mA, 1 ms pulse width). The bandpass was 2 Hz–10 kHz, with a sensitivity of 5 mV/division for determination of CMAP amplitude, and 500 μV/division for determination of onset latency. Sweep speed was 2 ms/division. The ground electrode was positioned between the cathode and the recording electrodes over the ipsilateral shoulder. Since the study was retrospective, not all patients were submitted to the complete protocol, as indicated by the fact that some patients underwent study of the median and ulnar nerves only. As such, the total number of nerves studied was as follows: HC: median, 42; ulnar, 42; radial, 42; musculocutaneous 42 nerves for CMAP amplitude reduction and onset latency prolongation: ALS controls: median and ulnar, 63 nerves for CMAP amplitude reduction and latency prolongation; radial and musculocutaneous, 26 nerves for CMAP amplitude reduction and 60