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Sensory nerve action potential amplitude is rarely reduced in lumbosacral radiculopathy due to herniated disc

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HIGHLIGHTS

- A previous retrospective study demonstrated abnormalities of sensory nerve action potentials (SNAPs) of the superficial peroneal nerve in 21% of 62 cases of L5 radiculopathy in contrast to the classical neurographic rule.
- Our prospective study showed that abnormalities of SNAP amplitude of sensory nerve originating from compressed root were present only in 7% of 108 patients with lumbosacral radiculopathies due to herniated disc and these anomalies were slight.
- The reduction of SNAP amplitude could be rarely observed in lumbosacral radiculopathies and could be likely due to damage of dorsal root ganglion if located into the foramen or in the spinal canal.

ABSTRACT

Objective: Normal sensory nerve action potential (SNAP) amplitude is a classical neurographic rule whether damage is located proximal to the dorsal root ganglion (DRG) as in radiculopathy. The study's aim is to check SNAP reduction in patients with lumbosacral radiculopathy due to herniated disc (HD).
Methods: A total of 108 consecutive patients with lumbosacral monoradiculopathy were prospectively enrolled. The diagnosis was based on clinical findings and magnetic resonance imaging (MRI). Electromyography of muscles of L4–S1 myotomes, motor neurography of peroneal and tibial nerves and sensory neurography of saphenous, superficial peroneal and sural nerves were performed. Percentage decrease in SNAP amplitude of nerves between healthy and affected sides was calculated.
Results: Significant SNAP amplitude asymmetry was observed in superficial peroneal nerve in seven patients with L5 (12.1%) and in sural nerve in one patient with S1 (2.4%) radiculopathies. All these patients had foraminal HD.
Conclusions: SNAP amplitude reduction of sensory nerve originating from damaged root is present only in 7% of radiculopathies and is likely due to DRG compression when located proximal to the spinal foramen or within the intraspinal canal.
Significance: Preservation of SNAP amplitude in radiculopathy remains an electrophysiological dogma with a little exception. If the reduction of SNAP amplitude affects other nerves, causes other than radiculopathy should be sought.

1. Introduction

One electrodiagnostic rule states that the sensory nerve action potential (SNAP) can be reduced in amplitude or not recordable if the lesion is at level of the dorsal root ganglion (DRG), as in herpes zoster, or distal to DRG, as in the plexopathies or peripheral neuropathies. Decrease in amplitude and absence of SNAP depends upon the number of sensory fibres that degenerate or are blocked. On the contrary, the lesion proximal to DRG does not cause abnormalities of SNAP (Benecke and Conrad, 1980; Wilbourn, 1994). In the radiculopathies, because the damage site is classically considered preganglionic, the amplitude of SNAP of the nerve, which comes from the same segmental level of the damaged posterior root, should be normal. Instead, Levin et al. demonstrated the

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absence or amplitude decrease in the SNAP of the superficial peroneal nerve in 13 of 62 (21%) cases of isolated active L5 radiculopathy resulting from intraspinal and extraspinal causes (Levin, 1998).

The aim of this prospective study is to assess the amplitude of SNAP of the peripheral nerves whose fibres mainly run in the root affected by a compressive monoradiculopathy due to only one type of cause, herniated disc (HD). This is the most frequent cause of the root compression. The term 'HD' refers to localised displacement of nucleus, cartilage, fragmented apophyseal bone or fragmented annular tissue beyond the intervertebral disc space (Fardon and Milette, 2001).

2. Patients and methods

All patients with L4, L5 or S1 monoradiculopathy were prospectively and consecutively enrolled from January 2009 to December 2010 in four electromyography (EMG) labs; two belonged to public outpatient clinics of Local Health Unit No. 7 of Siena and two to the Department of Neurological, Neurosurgery and Behaviour Sciences at 'Santa Maria alle Scotte' Hospital of Siena, Italy – the last two labs admitted in- and outpatients.

The diagnosis of radiculopathy was made on the basis of history and clinical examination; pain that began acutely within 1–12 months respect to our examination at the dermatome, even partial, of L4, L5 or S1 with or without low back pain and of magnetic resonance imaging (MRI) evidence of HD compressing the root corresponding to the dermatome with pain.

Patients aged >65 years, with diabetes, systemic or rheumatic diseases, cancer in the previous 5 years, polyneuropathy, history of radicular pain even in a different dermatome, radiculopathy resulting from causes other than HD, spine surgery at the lumbosacral level for any reason and patients who had lumbar canal stenosis, spondylolysis, spondylolisthesis at MRI or taking drugs toxic to the peripheral nervous system were excluded. The same neurophysiologist of each centre performed physical examination and then electrodiagnostic tests.

Physical examination included manual evaluation of the segmental muscle strength with Medical Research Council (MRC) scale, knee and ankle reflexes and sensitivity. The touch sensation was evaluated with cotton wool, pinprick sensation with a pin and two-point discrimination with a discriminator disc, comparing the affected with the contralateral healthy side. There are various representations or maps to localise specific dermatomes and Foerster's map was used to identify dermatomes with sensory symptoms (Foerster, 1933).

In addition, the patient quantified the pain with the 1–10 visual analogue scale (VAS) and the 'Douleur Neuropathique en 4 questions' (DN4) questionnaire for neuropathic pain was filled in. This questionnaire contains seven items related to symptoms and three related to clinical examination. A total score $\geqslant 4$ out of 10 suggests neuropathic pain (Bouhassira et al., 2005; Cruccu et al., 2010).

All the neurophysiologists and technicians had received the same training and they agreed on the clinical and electrophysiological methods before the beginning of the study. The neurophysiological protocol was the same and the technical equipment was similar for all the labs.

The electrodiagnostic protocol was based on a human experimental study on the determination of segmental sensory and motor innervation of the lumbosacral spinal nerves and was used for another electrophysiological study (Liguori et al., 1992; Mondelli et al., 2002).

Standard needle EMG was performed with disposable concentric coaxial needle electrodes (outside diameter of 0.46 mm, recording area 0.07 mm^2) in the muscles supplied by femoral and sciatic nerves belonging to the myotome corresponding to the dermatome with pain and was extended to one myotome above and below (if possible). The filter setting was 20 Hz–5 kHz. EMG was carried out in the following muscles in all the labs: at least two heads of quadriceps femoris (between vastus lateralis, vastus medialis and rectus femori), tibialis anterior, peroneus longus (and/or extensor hallucis longus and/or extensor digitorum brevis), gastrocnemius medialis and lateralis and abductor hallucis. When necessary, other muscles, including paraspinal, could be examined and varied from lab to lab and from patient to patient in relation to the patient's history to exclude other pathologies or confirm radiculopathy. EMG included observation of abnormal spontaneous activity at rest (positive sharp waves, fibrillations and high-frequency repetitive discharge), qualitative evaluation of motor unit action potentials (MUAPs) and recruitment at maximum effort. The spontaneous activity was explored in at least five sites; each of these insertion sites could be examined at two to four different depths (depending on the muscle size), allowing 10–20 discrete areas of the investigated muscle to be examined. The EMG of a muscle was considered abnormal when denervation activity at rest in at least two separate areas of the investigated muscle and/or neurogenic decreased recruitment at full effort (fewer MUAPs firing at higher rate) were recorded (Fisher et al., 1978). The muscles that needed to be abnormal to define the myotome affected by HD compression were identified according to the above-cited human experimental study of Liguori et al. (1992). In view of the aim of the study, only the results of abnormal EMG of the muscles supplied by the sciatic and femoral nerves, the same from which 'target' sensory nerves originate, were reported (see below for 'target' sensory nerves).

Motor nerve conduction studies of femoral, deep peroneal and tibial nerves were performed with surface Ag/AgCl disc recording electrodes, 9 mm in diameter, placed in the 'tendon-belly' arrangement. Compound muscle action potential (CMAP) amplitude was always measured from baseline to the following negative peak. Motor latency of the femoral nerve was obtained stimulating at the groin just lateral to the femoral artery and recording from vastus lateralis muscle at a fixed distance of 18 cm. Motor conduction velocity (MCV) of the deep peroneal nerve was calculated from the lateral border of the popliteal fossa to below caput fibulae at a fixed distance of 10 cm and below caput fibulae–flexor retinaculum segment recording from the extensor digitorum brevis muscle. MCV of the tibial nerve was measured from midline of the popliteal fossa to above and posterior to the medial malleolus recording from the abductor hallucis muscle. Distal motor latency (DML) was calculated at a fixed distance of 9 cm (for deep peroneal nerve) and 14 cm (for tibial nerve), from the point of stimulation to the muscle from which CMAP was recorded.

Sensory nerve conduction studies were performed with recording rubber suction electrodes and included antidromic sensory conduction velocity (SCV) and SNAP amplitude measured from the first positive peak (where latency was calculated) to the following negative peak. The distance between stimulator and recording electrodes was always set at 14 cm apart from the sensory nerve investigated. The 'target' sensory nerve was that mainly served by the root corresponding to the dermatome affected by radicular pain and injured by HD. The 'target' nerves were saphenous for L4, superficial peroneal for L5 and sural for S1 radiculopathy (Liguori et al., 1992). The saphenous nerve was stimulated along the medial aspect of the leg with the stimulator pressed between the tibia and the gastrocnemius muscle, recording just anterior to the medial malleolus. The superficial peroneal nerve was stimulated at the anterolateral aspect of the leg against the anterior edge of the fibula recording just medial to the lateral malleolus at the ankle. The sural nerve was stimulated along the posterior aspect of the leg immediately lateral to midline recording slightly above and posterior to the lateral malleolus. In all the cases, the ac-