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Conduction velocity of the human phrenic nerve in the neck

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Abstract

Purpose: To measure phrenic nerve conduction velocity in the neck in humans.
Scope: We studied 15 healthy subjects (9 men, 32.4 ± 6.7). We performed bipolar electrical phrenic stimulation in the neck, from a distal and a proximal stimulation site, and recorded diaphragm electromyographic responses on the surface of the chest. The ratio of the between-site distance to the latency difference provided phrenic velocities. Ulnar motor velocity was assessed similarly. In addition, five homogeneous patients with Charcot-Marie-Tooth disease type 1A (CMT1A) were studied for validation purposes. We obtained diaphragmatic responses from the two stimulation sites in all cases. The distal latencies (anterior axillary line recording) were 6.51 ± 0.63 ms (right) and 6.13 ± 0.64 ms (left). The minimal between site distance was 39 mm. Phrenic motor velocity was 55.2 ± 6.3 m s^{-1} (right) and 56.3 ± 7.2 m s^{-1} (left). In CMT1A, phrenic velocities were 17.1 ± 8.1 m s^{-1} (from 7 to 32 m s^{-1}) and were similar to ulnar and median velocities.
Conclusions: Phrenic nerve velocities can be estimated in humans and compare with upper limb motor conduction velocities. This should refine the investigation of phrenic function in peripheral neuropathies.

Keywords: Phrenic nerve; Conduction velocity; Phrenic stimulation; Diaphragm; Humans; Charcot-Marie-Tooth disease

1. Introduction

Damage to the phrenic nerve can cause diaphragm dysfunction and consequently set a requirement for ventilatory assistance. The corresponding prognostic impact makes electrophysiological exploration of the phrenic nerves clinically pertinent. Impairment of phrenic response is common during demyelinating neuropathy. For example, 85% of patients with chronic inflammatory demyelinating polyneuropathy (CIDP) present with phrenic conduction anomalies (Macia et al., 2003) at times responsible for acute or acute on chronic respiratory failure (Henderson et al., 2005). Abnormal diaphragm responses to phrenic stimulation are common during Guillain–Barré syndrome (Durand et al., 2005; Zifko et al., 1996). Phrenic involvement is frequent during Charcot-Marie-Tooth disease, asymptomatic or associated with chronic ventilatory failure (Carter et al., 1992; Sagliocco et al., 2003). Phrenic conduction impairment is common in advanced forms of amyotrophic lateral sclerosis. It can also be revealed in numerous other

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conditions, such as neuralgic amyotrophy (Lahrmann et al., 1999) or uremic neuropathy (Zifko et al., 1995).

Exploration of phrenic nerve conduction, whatever the technique used (electrical or magnetic stimulation in the anterolateral region of the neck (Newsom-Davis, 1967; Delhez, 1965; Mills et al., 1996), cervical magnetic stimulation (Similowski et al., 1989)), is currently limited to the measurement of motor latency. Contrary to conduction velocity, motor latency does not depend solely on the properties of the nerve. For the phrenic nerve, it can be variable depending on subject height (McKenzie and Gandevia, 1985; Mier et al., 1987), the site used to record the diaphragmatic response, the nature of the stimulus (Similowski et al., 1997), and for a given stimulation technique, the precise stimulation site. In the case of electrical stimulation in the neck, approach to the phrenic nerve can be at the thyroid cartilage (Newsom-Davis, 1967; Verin et al., 2002), the cricoid cartilage (Markand et al., 1984; McKenzie and Gandevia, 1985; Sarnoff et al., 1951), or just above the clavicle (Chen et al., 1995; Swenson and Rubenstein, 1992). These variants, combined with variations in recording sites (Newsom-Davis, 1967; Markand et al., 1984; Verin et al., 2002) probably explain the quite wide range of normal values, and make interpretation of phrenic nerve motor latencies difficult.

The phrenic nerve is reputed to be inaccessible for a sufficiently long distance to permit measurement of its conduction velocity (Aldrich et al., 2002). The objective of this study was to test the opposite hypothesis and to devise a technique for measuring phrenic nerve conduction velocity during routine neurophysiologic testing.

2. Methods

The study was conducted after legal and ethical clearance from the appropriate local authority (Comité de Protection des Personnes Ile-de-France 6, Paris, France). All the participants were duly informed of the purpose of the study and the methods used, and provided written consent. They were studied virtually supine on a comfortable couch with the head slightly raised, in a warm room.

2.1. Healthy subjects

Fifteen healthy volunteers (9 men and 6 women, age 32.4 ± 6.7 – from 24 to 45, height 173.7 ± 9.4 cm – from 160 to 187, body mass index 21.8 ± 3.3 kg/m^2 – from 18.6 to 32) participated in the study. They all had a normal neurological examination and had no history of pulmonary or neuromuscular disease.

2.2. Patients

Five patients (all women, age 34–57) (Table 1) were also studied for confirmatory purposes. They were diagnosed with type 1A Charcot-Marie-Tooth disease and drawn from a homogeneous group of patients with duplication of the 17p11.2 chromosomic region.

2.3. Recording of motor diaphragmatic response

Diaphragmatic electromyogram recordings were made using surface electrodes (silver/silver chloride solid gel electrodes, duck foot shaped, sensor area 263 mm^2, Neuroline 700 10-K/C, Ambu, Rugmarken, Denmark) on the anterior axillary line, the active electrode (cathode) situated at the 7–8th intercostal space, the reference electrode 50 mm below and behind the active electrode (“axillary” recording). In the healthy subjects only, a second recording was performed according to a variant known to minimize the risk of surface signal contamination by possible co-contraction of the extra-diaphragmatic thoracic muscles (Verin et al., 2002) (“medial” recording: mid-clavicular line at the 7–8th intercostal space, the two electrodes at a distance of 20 mm). The signal recorded by the two pairs of diaphragmatic electrodes was amplified using a Nihon-Kohden Neuropack Sigma device (Tokyo, Japan) with sampling at 20 kHz, bandwidth 2 Hz–5 kHz, scanning speed 5–10 ms/division, sensitivity of 100–500 microV/division.

2.4. Phrenic stimulation (Fig. 1)

The right and left phrenic nerves were stimulated using a rectangular electrical shock of 0.1 ms duration, using 5-mm diameter bipolar saline–soaked felt electrodes separated by a distance of 20 mm, with the cathode in distal position. Supramaximal stimulation was achieved as follows (Similowski et al., 1997). The phrenic nerve was first spotted with low intensity stimulation (10 mA). A recruitment curve was then built by increasing the stimulation intensity in a stepwise manner with direct visual feedback of the amplitude of the diaphragm potentials. A stimulation intensity 15% greater than the intensity producing the largest potential was retained for the measurements.

Distal stimulation was delivered along the posterior border of the sternocleidomastoid muscle, just above its insertion on the clavicle (Chen et al., 1995; Swenson and Rubenstein, 1992).

Table 1
Characteristics and motor velocities of the patients with Charcot-Marie-Tooth disease studied

#	Sex	Age (y)	Size (cm)	Ulnar[a] velocity (m s^{-1})	Median[a] velocity (m s^{-1})	Phrenic distal latency (ms)		Phrenic velocity (m s^{-1})	
						R	L	R	L
1	F	34	158	21	22	16.3	13.6	19	25
2	F	48	161	15	18	21.1	19.9	9	10
3	F	39	160	26	26	NA	10.4	NA	32
4	F	57	162	23	19	8.3	11.6	7	15
5	F	63	164	17	23	9.5	23.2	20	17

F, female; R, right; L, left; NA, not available.
[a] Right side.