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Identification of prolonged phrenic nerve conduction time in the ICU: magnetic versus electrical stimulation

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Abstract *Purpose:* Retrospective study of prospectively collected data to assess the reliability of cervical magnetic stimulation (CMS) to detect prolonged phrenic nerve (PN) conduction time at the bedside. Because PN injuries may cause diaphragm dysfunction, their diagnosis is relevant in intensive care units (ICU). This is achieved by studying latency and amplitude of diaphragm response to PN stimulation. Electrical stimulation (ES) is the gold standard, but it is difficult to perform in the ICU. CMS is an easy noninvasive tool to assess PN integrity, but co-activates muscles that could contaminate surface chest electromyographic recordings. *Methods:* In a first set of 56 ICU patients with suspected PN injury, presence and latency of compound motor action potentials elicited by CMS and ES were compared. With ES as the reference method, CMS was evaluated as a test designed to indicate presence or absence of PN injury. In eight additional patients, intramuscular diaphragm recordings were compared with surface diaphragm recordings and with the electromyograms of possible contamination sources. *Results:* The sensitivity of CMS to diagnose abnormal PN conduction was 0.91, and specificity was 0.84, whereas positive and negative predictive values were 0.81 and 0.92, respectively. Passing–Bablok regression analysis suggested no differences between the two measures. The correlation between PN latency in response to CMS and ES was significant. The "diaphragm surface" and "needle" latencies were close, and were significantly different from those of possibly contaminating muscles. One hemidiaphragm showed likely signal contamination. *Conclusion:* CMS provides an easy reliable tool to detect prolonged PN conduction time in the ICU.

Keywords Phrenic nerve · Respiratory muscles · Diaphragm · Electromyography · Peripheral nervous system diseases · Magnetic stimulation

Introduction

Studying diaphragm function is clinically relevant in various critical care contexts including certain forms of acute respiratory failure of neuromuscular origin [1, 2], certain cases of difficult weaning from the ventilator [3], the assessment of the respiratory impact of critical illness polyneuropathy [4, 5], the prognosis of ventilator dependency [6], and the diagnosis of iatrogenic lesions of the respiratory neuromuscular system [7]. Diaphragmatic dysfunction can arise from muscular damage, phrenic nerve abnormalities, or both.

To assess the function of the phrenic nerve, and thus to establish the responsibility of phrenic nerve abnormalities in a given clinical situation, studying the electromyographic response of the diaphragm to phrenic stimulation is the most appropriate approach [8, 9]. The gold standard to do this is to perform electrical phrenic stimulation in the neck. Yet this technique is technically difficult with a risk of falsely negative results, particularly in the intensive care unit (ICU) setting (edema, presence of a jugular catheter or of a tracheostomy). Easier noninvasive magnetic stimulation techniques alleviate this risk [10, 11], but their lack of focus makes the interpretation of surface-recorded signals difficult and requires the use of needle or esophageal electrodes to eliminate signal contamination from the co-activation of extradiaphragmatic muscles [12, 13].

Our group has previously shown that the risk of signal contamination described above could be minimized by (1) placement of the electrodes in the lowest accessible intercostal space, close to the chondrocostal junction; (2) keeping the two electrodes as close as possible to one another. However, this result was obtained under laboratory conditions [14].

The aim of the present study was to establish, in ICU patients, how cervical magnetic stimulation (CMS) combined with an optimized surface diaphragm electromyography (EMG) recording technique would compare with electrical phrenic nerve stimulation as a simple tool to identify prolonged phrenic nerve conduction time at the bedside.

To fulfil this aim, we first compared the electromyographic diaphragm responses to CMS and to electrical phrenic stimulation in terms of their presence, their latencies, and the right–left symmetry of their amplitudes. Then, because intramuscular electrodes have a very limited sampling volume and hence carry a low risk of signal contamination through volume conduction, we compared the surface diaphragm EMG response to phrenic magnetic stimulation, with concomitant intradiaphragmatic needle electrode recordings, and with surface recordings of possibly contaminating muscles, namely the serratus anterior, latissimus dorsi, and pectoralis major.

Patients and methods

Patients

Diaphragm electrophysiological studies performed in our ICU between 1996 and 2008 were carefully reviewed.

During this period 56 patients (43 males, 40 ± 21 years) had both electrical and magnetic phrenic stimulation. These patients constituted group 1. The reasons for diaphragm electrophysiological examination were prolonged weaning failure ($n = 30$), indication for phrenic pacing ($n = 21$), and recent acute respiratory failure with suspicion of neuromuscular involvement ($n = 5$).

During the same period, eight patients (6 males, age 56 ± 20 years) underwent diaphragm function studies involving the use of intradiaphragmatic needle electrodes as well as surface electrode recordings of possibly contaminating muscles. These patients constituted group 2. The reasons for diaphragm electrophysiological examination were prolonged weaning failure ($n = 2$), indication for phrenic pacing ($n = 3$), and recent acute respiratory failure with suspicion of neuromuscular involvement ($n = 3$). In these patients, the use of intramuscular needle electrodes and the EMG recording of possibly contaminating muscles were mostly justified by a strong suspicion of surface EMG signal contamination.

The study was approved by the Institutional Review Board of the Société de Réanimation de Langue Française. The patients or their family received standard information on the nature and motives of the tests.

Stimulations

In all the patients (group 1 and group 2), CMS was carried out using a Magstim 200 stimulator (Magstim, Whitland, Dyfed, UK) at the maximal output of the stimulator. The coil was centered over the spinous process of the seventh cervical vertebra [14, 15] (Fig. 1). In the 57 patients of group 1, electrical stimulation (ES) was also delivered sequentially to the phrenic nerves using a bipolar electrode delivering square-wave pulses of 0.1-ms duration at a supramaximal intensity. The phrenic nerve was stimulated at the posterior border of the sternocleidomastoid muscle, at the level of the cricoid cartilage or slightly lower.

All the patients were studied in the ICU, in recumbent position. For any given patient, the right and left side were studied. For each of the techniques used, 3–5 consecutive stimulations were performed.

Measurements

All signals were amplified, recorded, and analyzed using a Neuropack Sigma® electromyograph (Nihon–Kohden, Tokyo, Japan).

Electromyograms

In both groups, surface diaphragm silver cup electrodes (S*ant*) were positioned in the eighth intercostal spaces, on the mid-clavicular line [14].

In the eight patients of group 2, intramuscular diaphragmatic recordings (N*ant*) were obtained using a

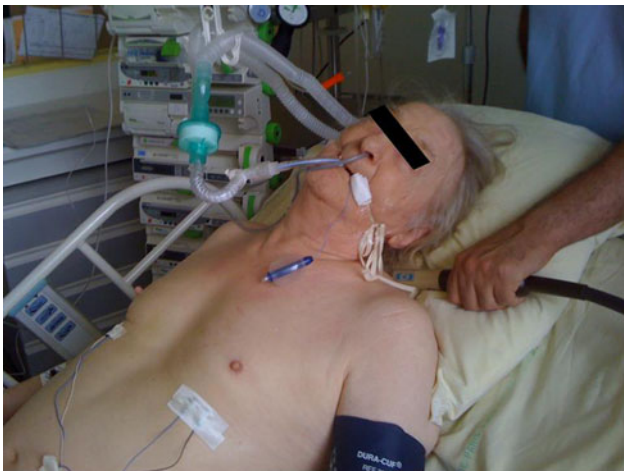

Fig. 1 CMS and surface anterior electrodes placement. The 90-mm circular coil was centered over the spinous process of the seventh cervical vertebra, coil handle directed caudally and held either parallel to the vertebral column or at a 45° angle. Surface anterior electrodes were placed in the eighth intercostal space, between the costochondral junction and the mid-clavicular line

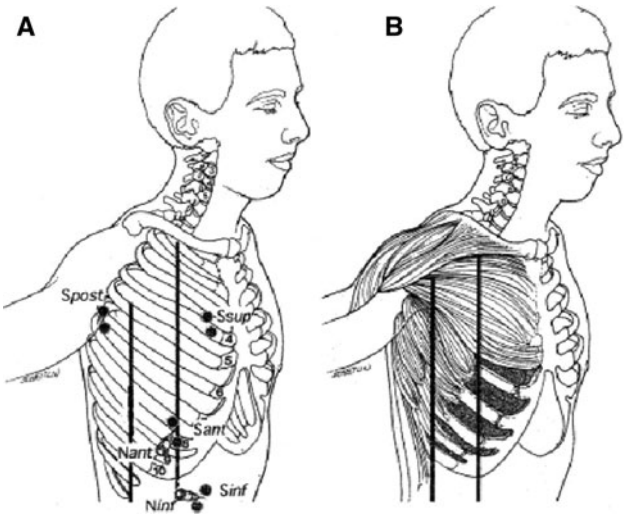

Fig. 2 Schematic representation of electrode placement. S*ant* (active electrode in the eighth intercostal space, between the costochondral junction and the mid-clavicular line; reference electrode slightly laterally on the above rib), aimed at principally recording the diaphragm (by hypothesis). S*post* (active electrode in the sixth intercostal space on the posterior axillary line, reference electrode on the above rib), lies over the anterior insertions of the latissimus dorsi and serratus anterior. S*up* (two electrodes on the mid clavicular line, in the third or fourth intercostal space), lies over the muscle mass of the pectoralis major. N*ant* is the needle electrode adjacent to S*ant*

bipolar concentric needle electrode (Medelec, Old Woking, UK) inserted in the hemidiaphragm adjacent to S*ant* (N*ant*) [14, 16]. In addition, the EMG response of possibly contaminating muscles (serratus anterior, latissimus dorsi, and pectoralis major) was studied using two additional pairs of surface electrodes: S*post* were positioned in the sixth intercostal space, on the posterior axillary line, while S*up* were positioned in the third or fourth intercostal spaces, on the mid-clavicular line (Fig. 2).

Abdominal displacements and airway pressures

Abdominal displacements (mechanical strain gauge, Nihon-Kohden, Tokyo, Japan) and airway pressures (differential transducer Validyne, Northridge, CA, USA) were recorded during CMS and taken as the mechanical proof of an actual diaphragm response to CMS ("Quality criteria").

Analysis and statistics

Quality criteria

The motor EMG response to ES or CMS is called the compound motor action potential (CMAP). CMAPs were retained for analysis when they met the following three criteria: (1) absence of obvious electrical interference, evidenced by a clear return of EMG signal to baseline after the stimulus artifact and before muscle response; (2) absence of contamination by electrocardiogram QRS complexes; (3) concomitance, in response to the stimulation, of a negative intrathoracic pressure swing and of an abdominal expansion, taken as the mechanical proof of an actual diaphragm response to CMS.

Signal interpretation criteria

In group 1, the diaphragm EMG response to phrenic nerve stimulation was considered to be contaminated if CMAP latency was less than 5 ms in response to CMS, abolished if no signal was detected, and delayed if CMAP latency was at least 7.0 ms in response to CMS, and at least 8.5 ms in response to ES. In addition, the amplitudes of the potentials, when present, were measured from peak to end of CMAP. Because amplitudes in response to CMS are extremely variable, they were not interpreted in terms of absolute value, but rather in terms of a ratio between the signals from the right and left side. The CMAPs were considered symmetrical when the right and left amplitudes were between 80 and 120% of one another, asymmetrical otherwise.

In group 2, contamination of the surface diaphragm signal (S*ant*) from rib cage muscles was deemed present in a given subject if (1) the latency of the S*ant* CMAP was shorter than the N*ant* one by more than 0.75 ms (a conservative value as needle electrodes can provide latencies longer than their surface counterparts by up to 2 ms [17, 18]); (2) the S*ant* latency was closer to the S*sup* or S*post* latencies than to the N*ant* one.

Statistics

The results are expressed as mean ± SD. In group 1, the performance of CMS to detect phrenic nerve abnormalities was assessed as follows. In order to calculate sensitivity, specificity, and predictive values, we defined true positives as abnormal conduction (delayed or abolished) in response to both ES and CMS, false positives as abnormal conduction in response to CMS but normal in response to ES, true negatives as normal conduction in response to both ES and CMS, and false negatives as normal conduction in response to CMS but abnormal in response to ES. The agreement between the surface diaphragm latency in response to CMS and ES was further tested using Passing–Bablok linear regression [19]. Finally, correlations between latency in response to CMS and ES were measured using Spearman's rank correlation coefficient. In group 2, the CMAP latencies recorded at the various sites were compared using nonparametric Kruskal–Wallis one-way analysis of variance, followed, if significant, by Dunn's post test.

The statistical analysis was performed using StatView 5.0® software (StatView®, SAS Institute, Cary, NC), except for Passing–Bablok linear regression that was performed using MedCalc Software® (Mariakerke, Belgium).

Results

Group 1

In the 56 patients in group 1, 112 phrenic nerves were studied. Figure 3 depicts the results of magnetic and electric stimulation. In seven cases, the latency of diaphragm EMG response to CMS was less than 5 ms and was therefore classified as contaminated. In 57 cases, the diaphragm EMG response to CMS was normal. Among them, the diaphragm EMG response to ES was normal in 49 (true negatives) and prolonged or abolished in four (false negatives). In 48 cases, the diaphragm EMG response to CMS was abnormally prolonged or abolished. Among them, nine had a normal response to ES (false positives) while the response to ES was abolished or prolonged in 39 (true positives). Finally, in the four remaining phrenic nerves, while there was no response to ES, a normal EMG response to CMS in conjunction with abdominal expansion and negative thoracic pressure swing were observed, suggesting normal diaphragm conduction. These four cases were therefore considered as ES failure. The sensitivity of CMS to diagnose abnormal PN conduction was 0.91 [95% confidence interval (CI) 0.77–0.97] and its specificity was 0.84 (95% CI 0.72–0.92), whereas its positive predictive value was 0.81 (95% CI 0.67–0.91) and its negative predictive value was 0.92 (95% CI 0.81–0.98). The results of Passing–Bablok analysis are provided in Fig. 4. They indicate the lack of systematic differences between the two measures. Finally, the correlation between phrenic nerve latency in response to CMS and ES was significant for both right ($n = 40$ nerves, rho = 0.80; 95% CI 0.65–0.89; $p < 0.0001$) and left ($n = 46$ nerves, rho = 0.82; 95% CI 0.69–0.90; $p < 0.0001$) phrenic nerves.

In the 39 patients who exhibited a bilateral response to CMS, the right-to-left amplitudes of the diaphragm CMAPs were symmetrical in all cases. The corresponding ES-elicited CMAPs were also symmetrical. In response to ES, there were six cases where the right-to-left amplitude ratio was below 0.8 or above 1.2. A concordant value of the CMS-elicited CMAPs right-to-left amplitude was found in five occurrences.

Group 2

Intradiaphragmatic recordings (N*ant*) were compared to surface diaphragm recordings (S*ant*) and possibly contaminating muscles (S*up*, S*if*) in the eight patients in group 2, providing 15 sets of data; comparison was not possible on the left side of one patient in whom both surface and needle responses to CMS were abolished because of severe phrenic nerve injury.

Comparison of latencies

The diaphragm surface (S*ant*) CMAP latency was 6.0 ± 1.0 ms [5.4–7.0 ms], and not significantly shorter than its counterpart needle (N*ant*) latency (6.6 ± 1.1 ms [5.6–9.7 ms]) (Fig. 5). A response of significantly shorter latency occurred at S*up* in 13 cases (3.8 ± 0.5 ms

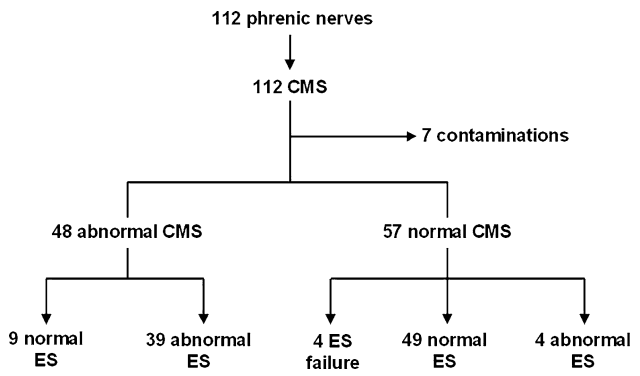

Fig. 3 Results of magnetic (CMS) and electrical (ES) phrenic nerve stimulation. The diaphragm electromyographic response to phrenic nerve stimulation was considered as contaminated if CMAP latency was no greater than 4 ms in response to CMS. The response was considered as abnormal if no signal was detected or if the signal was delayed, which was defined as a CMAP latency of at least 7.0 ms in response to CMS and at least 8.5 ms in response to ES

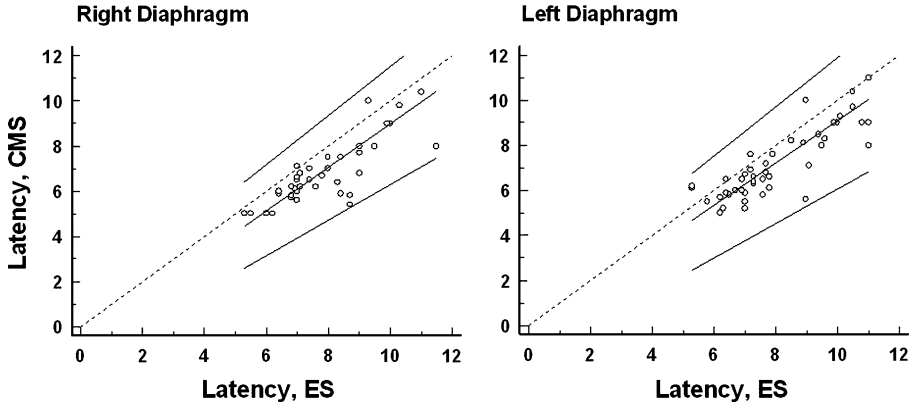

Fig. 4 Passing–Bablok linear regressions. The *left panel* shows the comparison of the right hemidiaphragm electromyographic surface response to magnetic (CMS) and electrical (ES) phrenic nerve stimulation. The intercept *A* of the regression equation was −0.65 with a 95% CI (−1.58 to 0.68) including zero, and the slope was 0.96 with a 95% CI (0.79–1.08) including one, indicating the lack of difference between the two measures. The *right panel* shows the comparison of the left hemidiaphragm electromyographic surface response to magnetic (CMS) and electrical (ES) phrenic nerve stimulation. The intercept of the regression equation was −0.29 with a 95% CI (−1.62 to 0.95) including zero, and the slope was 0.94 with a 95% CI (0.77–1.09) including one, indicating the lack of difference between the two measures

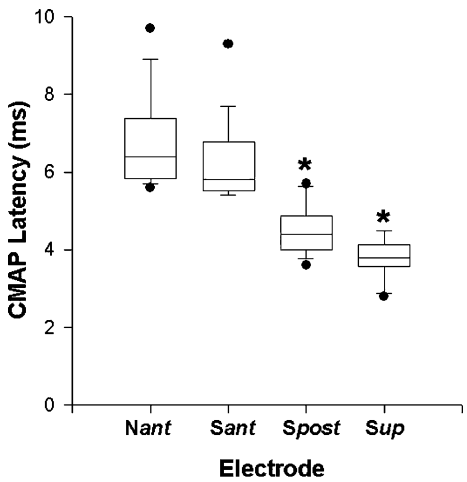

Fig. 5 CMAP latencies (*vertical axis*) in response to CMS, in the 15 simultaneous EMG studies of both the diaphragm and the possibly contaminating muscles. S*ant*, anterior surface lower chest electrodes; N*ant*, adjacent needle electrode; S*post*, posterior surface electrode; S*up*, upper chest anterior electrode (Fig. 2). The *ends of the whiskers* represent the 10th and the 90th percentile. The *points outside of the whiskers* are outliers. The *asterisk* symbol denotes a significant difference ($p < 0.05$) from the S*ant* value

[2.8–4.5 ms], $p < 0.05$ with S*ant*) and at S*post* in 11 cases (4.5 ± 0.7 ms [3.6–5.7 ms], $p = 0.05$ with S*ant*).

Contamination

Contamination of the signal picked up at S*ant* by a signal arising from rib cage muscles occurred, according to our definition (see "Patients and methods"), in one out of the 15 hemidiaphragms studied, and seemed to arise from S*post* (latissimus dorsi and serratus anterior). Figure 6 shows an example of uncontaminated recordings obtained with S*ant*.

Discussion

The present study shows that CMS combined with an optimized surface diaphragm EMG recording technique is a simple and reliable way to identify prolonged phrenic nerve conduction time.

Methodological issues

Before discussing the results and their implications, some methodological points warrant mention. Firstly, we took care to analyze only EMG responses for which the reality of a diaphragm contraction in response to phrenic nerve stimulation was always ascertained by the conjunction of a negative pressure response with abdominal expansion. This pattern rules out confusion factors such as a purely extradiaphragmatic inspiratory response or a predominant contraction of the pectoralis major [20]. Secondly, operating values of CMS were calculated in reference to ES, which is very selective for phrenic nerves and thus unlikely to produce a contaminated EMG surface signal [8, 9]. Thirdly, as in previous studies [14, 15], CMS provided shorter CMAP latencies than ES. This time difference does not involve signal contamination, but is rather attributed to either the preferential activation of fast fibers by magnetic stimulation or a more distal nerve depolarization site, as suggested by previous reports in the diaphragm [14, 15] and other nerves [21, 22].

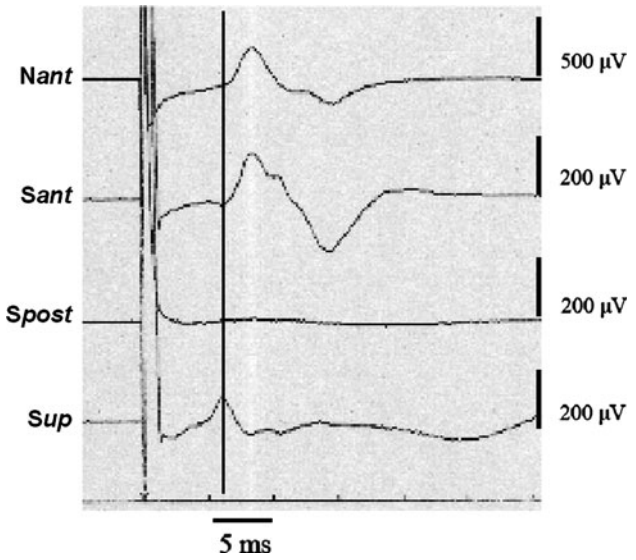

Fig. 6 Example of uncontaminated S*ant* tracings in response to CMS in patient 12. The traces correspond to the M waves recorded by, from *top* to *bottom*, the anterior lower chest needle electrodes (N*ant*), the corresponding surface electrode (S*ant*), the posterior surface electrodes (S*post*), the upper chest anterior electrode (S*up*). The *vertical bar* shows CMAP latency recorded at S*ant*. It can be seen that the S*ant* and N*ant* CMAP are synchronous or practically synchronous, and that they occur long after the CMAP recorded by S*up*, validating the diaphragmatic, uncontaminated nature of the S*ant* signal. Of note, S*post* remained silent in this patient

We did not assess the value of CMS in terms of the amplitudes of the CMAPs. Indeed, the amplitudes of diaphragmatic CMAPs evoked by phrenic nerve stimulation are generally not used for diagnostic purposes, contrary to what is commonly done in the interpretation of limb muscles electromyograms. This is because the spatial relationship between the muscle and surface electrodes is highly variable from one subject to another, which lowers considerably the value of diaphragm CMAPs [9]. This implies that using phrenic nerve stimulation to identify phrenic nerve lesions is liable to miss the diagnosis of nondemyelinating axonal neuropathies (decreased motor potential amplitude without latency lengthening). However, in severe forms of axonal neuropathies, the CMAP amplitude reduction correlates with a velocity slowing that is probably explained by a predominant loss of large, fast-conducting fibers [23]. Of note, the right-to-left amplitude comparison that we could perform in a small subset of our study population is reassuring regarding the ability of CMS to identify unilateral axonal defects.

Possibility of recording an uncontaminated signal

Several studies have called attention to the issue of signal contamination regarding diaphragm recordings from reference surface electrodes [12, 13]. This is because a pair of EMG electrodes placed on the skin records the summative activity of the various muscle layers that lie underneath it. Whatever the efforts made to focus the stimulus, CMS is not able to selectively activate the phrenic nerves, and thus the co-activation of various muscles is unavoidable. The present study provides additional arguments against signal contamination with CMS. Indeed, in a quite large group of ICU patients, CMS and ES provided quite similar information regarding the presence of a phrenic nerve conduction anomaly. Indeed, in reference to ES, the performance of CMS as a test to diagnose phrenic nerve injury was satisfactory. In addition, Passing–Bablok linear regression analysis suggested the lack of systematic differences between the two measures. Along the same lines, a strong correlation was found between latency in response to CMS and ES, which would be unlikely in the presence of systematic contamination. Finally, needle and surface electrode diaphragm recordings showed similar latencies, quite different to possibly contaminating muscle latency responses.

As previously suggested [14, 16], the reasons for the rarity of signal contamination in our patients probably lie in the recording technique. Indeed, we positioned the two electrodes very close to one another and located them low and medially on the chest on a spot relatively free of contaminating fibers (Fig. 2). The closer the surface electrodes are to the contaminating muscles, and the further apart from one another, the higher the risk of contamination.

Practical consequences

This study brings clinical information immediately relevant to the care of ICU patients. Indeed, it shows that it is possible to gather valid electrophysiological responses of the diaphragm to phrenic magnetic stimulation in this setting, and this with surface electrodes. These electrodes are easy to use, their positioning is totally noninvasive, and they allow the separate study of the right and left side, even in response to bilateral stimulations (which is not the case for esophageal electrodes). Phrenic nerve magnetic stimulation is also noninvasive, and is faster and easier to perform than ES, especially in the ICU setting. From a practical point of view, we recommend the following precautions: the surface electrodes should be carefully placed in the lowest accessible intercostal space close to the costochondral junction, and less than 2 cm apart from one another. The reality of a diaphragm response to the stimulation should be ascertained before interpreting the EMG responses; the combination of tracheal pressure and abdominal circumference provides a simple means to do this. If the latency of diaphragm CMAP in response to CMS is less than 5 ms, contamination is likely, and a surface electrode placed over the mass of a possibly

contaminating muscle should be used as a control. Provided these precautions are taken, stimulation techniques should allow clinicians to more easily diagnose the underlying mechanisms of diaphragm dysfunction [24]. This should particularly be the case in diseases interrupting or slowing conduction through the phrenic nerve (e.g., demyelinating neuropathies) since interpretation of CMAP amplitude in response to CMS is not as reliable as CMAP latency. These results pave the way for a study of how prolonged phrenic nerve conduction relates to clinical outcome.

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