Validation of surface recordings of the diaphragm response to transcranial magnetic stimulation in humans

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The response of the diaphragm to transcranial magnetic stimulation is chiefly studied in terms of the corresponding motor-evoked potentials recorded by surface chest wall electrodes placed over the anatomical landmarks of the diaphragm. Being nonselective, transcranial stimulation coactivates the diaphragm and several other muscles. The corresponding electrical activity is liable to be detected by “diaphragm” surface electrodes through volume conduction. Such signal contamination, well described with focal phrenic nerve stimulation techniques (16, 17) and cervical magnetic stimulation (15), could limit the use of transcortical magnetic stimulation as a diaphragm tool.

Modification of conventional diaphragm electrode placement [active electrode in the 5th to 7th intercostal space on the anterior axillary line, reference electrode either 5–6 cm apart on the relief of an adjacent rib or extrathoracic (3, 14, 17, 21)] to a lower and more medial site (Fig. 1) improves the reliability of surface electrodes to measure phrenic nerve conduction time (34). The main reason for this finding is probably that, at this site, electrodes do not directly overlay muscles frequently coactivated by phrenic stimulation or cervical magnetic stimulation, such as the pectoralis major, serratus anterior, or latissimus dorsi. It can be postulated that if contamination from these muscles does not occur in response to stimulation techniques that can directly and strongly activate them, it should not occur in response to transcranial stimulation. The pattern of muscle coactivation with transcranial magnetic stimulation is, however, different from what it is with cervical magnetic stimulation and theoretically includes intercostal and abdominal muscles that do lay underneath diaphragm surface electrodes at the modified recording site described above. The present study was thus designed after a previous one by Verin et al. (34) to evaluate, in a distinct set of subjects, how costimulation of extradiaphragmatic muscles with the
diaphragm by transcranial magnetic stimulation could interfere with the measurement of the cortex-to-diaphragm conduction time. Because concentric intramuscular electrodes have a very limited sampling volume and hence carry a low risk of signal contamination through volume conduction, we compared surface recordings performed at a site optimized to record phrenic nerve stimulation-induced diaphragm contractions with concomitant recordings obtained from needles inserted in the diaphragm and with recordings of possibly “contaminant” muscles (latissimus dorsi, serratus anterior, and pectoralis major surface electrodes, and abdominal muscles surface and needle electrodes).

**MATERIAL AND METHODS**

**Subjects**

After successful completion of the French legal procedure for biomedical research in human volunteers, nine subjects participated in the study (6 men, 3 women; age 24–39 yr; height 159–192 cm; weight 48–96 kg). They were informed in detail of the purpose of the study and methods used and gave written consent. They were studied while sitting on a chair with headrests, abdomen unbound. In each subject, the recordings were obtained from the handedness side.

**Measurements**

**Abdominal displacements.** Changes in abdominal circumference were monitored with a mechanical strain gauge (Nichiron Kohden, Tokyo, Japan) attached to an elastic belt placed at the level of the umbilicus.

**Electromyograms.** Surface recordings were obtained at four locations [lower chest (S\textsubscript{ant}); posterior (S\textsubscript{post}), upper chest (S\textsubscript{sup}), and abdominal wall (S\textsubscript{inf})] by using pairs of skin-taped silver cup electrodes filled with conductive paste (Fig. 1). S\textsubscript{post} lay directly over the anterior insertions of the latissimus dorsi and serratus anterior. S\textsubscript{sup} lay over the muscle mass of the pectoralis major. S\textsubscript{inf} aimed at recording the activity from both the obliquus externus and internus muscles, as well as activity of the deeper transversus abdominis muscle. The contribution of the above muscle groups to the signals recorded by the corresponding electrodes was ascertained by the presence of bursts of electromyographic activity during tonic voluntary contractions.

Intramuscular recordings were obtained by using bipolar concentric needle electrodes (diameter 0.3 mm, length 25 mm, sampling surface 0.03 mm\(^2\); Medelec, Old Woking, UK). One needle electrode was placed in the hemidiaphragm (Fig. 1) adjacent to the lower pair of surface electrode (N\textsubscript{ant}) following the technique described by Bolton et al. (2) and slightly modified. In brief, the needle was inserted in the intercostal space at the upper border of the lower rib and slowly advanced at an upward angle of 45° with continuous auditory and visual monitoring of the signal until the activity from motor units discharging rhythmically with inspiration could be heard and seen. Then the subjects were asked to breathe in slowly but strongly while protruding their abdominal wall. If necessary, the electrode was further advanced so as to record an optimal burst of activity during this predominantly diaphragmatic inspiration. The intradiaphragmatic location of the electrode was checked from electrical phrenic nerve stimulation in the neck. Because, at this site, fibers from the transversus abdominis muscle can possibly be intermingled with diaphragmatic insertions, the signal recorded by N\textsubscript{ant} was also checked during voluntary expiratory efforts. Expulsive efforts with the glottis closed were associated with bursts of electromyographic activity, but N\textsubscript{ant} consistently remained silent during expiratory efforts with the glottis open.

**Fig. 1.** Schematic representation of the electrodes placement. The active electrode in the 8th intercostal space, between the costochondral junction and the midclavicular line and the reference electrode slightly laterally on the above rib (S\textsubscript{ant}) is aimed at recording principally the diaphragm (34). The active electrode in the 6th intercostal space on the posterior axillary line with the reference electrode on the above rib (S\textsubscript{post}) lies over the anterior insertions of the latissimus dorsi and serratus anterior. The two electrodes on the midclavicular line in the 3rd or 4th intercostal space (S\textsubscript{sup}) lie over the muscle mass of the pectoralis major. The two electrodes at the intersection of the midclavicular line and with a horizontal line drawn between the costal margin and the umbilicus (S\textsubscript{inf}) is aimed at recording the activity from both the obliquus externus and internus muscles, as well as activity of the deeper transversus abdominis muscle (31). N\textsubscript{ant}, needle electrode adjacent to S\textsubscript{ant}; N\textsubscript{inf}, needle electrode adjacent to S\textsubscript{inf}.
opened. A mild to moderate degree of abdominal muscle activity thus did not result in contamination of the diaphragmatic signal detected by N\textsubscript{ant}. A second needle electrode was inserted in the abdominal muscles (N\textsubscript{inf}), immediately beside the abdominal wall pair of surface electrodes (Fig. 1) and perpendicularly to the skin, and slowly advanced for 2–3 cm. The appropriate placement of N\textsubscript{inf} was assessed during voluntary expiratory efforts from functional residual capacity, expulsive maneuvers with the glottis closed, and during voluntary in-drawing of the abdominal wall (4). Of note, at both intramuscular recording sites, the amplitude of the cardiac artifact appeared reduced compared with the corresponding surface site by at least a factor of 3.

All signals were fed to a Nihon Kohden Neuropack Sigma electromyograph (Nihon Kohden, Tokyo, Japan), with a 10-kHz sampling rate and a 20 Hz to 5 kHz bandwidth. They were stored on an Apple Macintosh computer for subsequent analysis (PowerLab, AD instruments, Hastings, UK).

**Stimulations**

All the stimulations were carried out by using a Magstim 200 stimulator (Magstim, Whitland, Dyfed, UK) equipped with either a 90-mm circular coil (peak magnetic field 2.5 T) or a double 70-mm figure-eight coil (peak magnetic field 2 T) at the maximal output of the stimulator. Stimulations were delivered at relaxed end-expiration as judged from the abdominal circumference trace, with the glottis closed. For each of the techniques used (Fig. 2), 10 consecutive stimulations were performed.

Cervical magnetic stimulation with the 90-mm coil was performed to measure phrenic nerve conduction time (26, 34). Nonfocal transcranial magnetic stimulation was delivered by using the same coil positioned over the vertex in the position that was associated with the most ample motor-evoked potential from N\textsubscript{ant}. Focal transcranial magnetic stimulation was performed according to the technique described by Maskill et al. (19). In brief, the coil was first centered over a spot located 3 cm lateral and 2 cm anterior of the vertex in the 10- to 20-electroencephalogram electrode placement system, and its position was then optimized according to the amplitude of the motor evoked potential from N\textsubscript{ant}.

**Data Analysis**

**Terminology.** The peripheral motor responses to cervical magnetic stimulation and the motor potentials evoked by transcranial magnetic stimulation were analyzed in terms of their latency, which was defined as the time elapsed from the stimulus to the first departure of the signal from baseline. Individual values were obtained from averaging seven to nine accepted stimulations (see Quality criteria). The results below are the mean values ± SD, with indication of the range in brackets.

**Quality criteria.** Peripheral motor responses and motor evoked potentials were retained for analysis when they met the following three criteria: 1) absence of obvious electrical interference, attested to by a clear return of the electromyographic signal to baseline after the stimulus artifact and before the muscle response; 2) absence of contamination by electrocardiographic complexes; and 3) concomitance with clear abdominal expansion, taken as the mechanical witness of a diaphragm response.

**Statistics.** Statistical analysis was performed by using the StatView 5.0 software (SAS Institute, Cary, NC) on an Apple Macintosh computer. Latencies of the motor-evoked potentials recorded at the various sites were compared by using a nonparametric Friedman analysis of variance followed by the Wilcoxon rank test. Differences were considered significant when the probability of a type I error was <0.05.

**Contamination.** Contamination of the signal recorded at S\textsubscript{ant} from rib cage muscle was considered present if, in two or more of the stimulations retained for analysis, 1) the latency of the S\textsubscript{ant} motor-evoked potential was shorter than the N\textsubscript{ant} one by 0.75 ms (a conservative value since needle electrodes can provide latencies longer than their surface counterparts by up to 2 ms (7, 18)); 2) the S\textsubscript{ant} latency was closer to the S\textsubscript{sup} or S\textsubscript{post} latencies than to the N\textsubscript{ant} one. Conversely, contamination from abdominal muscle was considered present if the latency of the S\textsubscript{ant} motor-evoked potential was longer than the N\textsubscript{ant} one and closer to the S\textsubscript{inf} latency than to the N\textsubscript{ant} latency.

**Additional Studies in Patients**

In addition to the above protocol in healthy volunteers, we report and use the results of transcranial magnetic stimula-

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**Fig. 2.** Schematic representation of the stimulation techniques. **A:** summary of the various techniques used: cervical magnetic stimulation (CMS), with a 90-mm circular coil; nonfocal transcranial magnetic stimulation (nfTMS), with the same coil; focal transcranial magnetic stimulation (fTMS), with a double 70-mm figure-eight coil. **B:** location of the optimal fTMS site. The coil was first centered on a spot located 3 cm lateral and 2 cm anterior to the vertex (Cz) (•) and then slightly displaced until the best possible response was obtained (see MATERIALS AND METHODS for details).
Cervical Magnetic Stimulation

The latency of the motor response recorded at S\text{ant} was 5.4 ± 0.8 ms (4.3–6.5 ms), consistent with usual values in our laboratory (26, 34). The latencies of the motor responses recorded at S\text{sup} and S\text{post} were 3.7 ± 1.1 (2–5.6 ms) and 4.1 ± 0.4 ms (3.5–4.7 ms), respectively, and were consistently shorter than the latency recorded at S\text{ant} \((P = 0.01\) and \(P = 0.008\), respectively). The latency of the motor response recorder by the intramuscular electrode \(N\text{ant}\) was 5.9 ± 0.8 ms (5–6.7 ms) \((P = 0.008\) with \(N\text{ant}\)).

Nonfocal Transcranial Magnetic Stimulation

The average latency of the motor-evoked potential recorded at S\text{ant} was 17 ± 1.3 ms (15.6–19.1 ms), similar to data obtained in our laboratory with the same technique (28). The latencies of the motor-evoked potentials recorded at S\text{sup} (8 subjects) and at S\text{post} (all subjects) were consistently shorter than the latencies recorded at the other sites [12.4 ± 1.8 ms (10.1–15.7 ms) and 13.8 ± 1.5 ms (11.5–15.9 ms), respectively; \(P = 0.01\) with \(S\text{ant}\)]. The latencies of the motor-evoked potentials recorded by the abdominal surface electrodes \(N\text{inf}\) (7 subjects) were longer [19.1 ± 1.9 ms (15.6–20.4 ms)] than the latencies recorded at the other sites \((P = 0.03\) with \(S\text{ant}\)).

The average latency of the motor-evoked potential recorded by the lower chest needle electrode \(N\text{ant}\) was 17.2 ± 1.1 ms (15.7–18.7 ms). It was significantly shorter than the latency of the motor-evoked potential recorded at the abdominal needle electrode \(N\text{inf}\) [19.7 ± 1.4 ms (17.2–21.5 ms); \(P = 0.02\)].

The latencies of the motor-evoked potentials recorded at \(S\text{ant}\) tended to be slightly shorter than the latencies measured from the corresponding needle electrode \(N\text{ant}\). Similarly, the latencies of the motor-evoked potentials recorded by the abdominal surface electrodes \(N\text{inf}\) tended to be slightly shorter than the latencies measured from the corresponding needle electrode \(N\text{inf}\), but these differences did not reach statistical significance.

Focal Transcranial Magnetic Stimulation

Focal transcranial magnetic stimulation elicited a motor-evoked potential at \(S\text{ant}\) in seven subjects [16 ± 1 ms (14.3–16.8 ms)], whereas no response could be recorded at this site in two subjects. A motor-evoked potential was simultaneously recorded at \(S\text{post}\) in five cases [12.5 ± 0.8 ms (10.1–14.9 ms)], at \(S\text{sup}\) in four cases [11.9 ± 2.1 ms (11.7–13.7 ms)], and at \(S\text{inf}\) in four cases [19.3 ± 3.4 ms (16.1–24 ms)].

Focal transcranial magnetic stimulation elicited a motor-evoked potential at the diaphragm needle electrode \(N\text{ant}\) in the same seven subjects [16.5 ± 1.3 ms (13.9–17.6 ms)]. Here again, the motor-evoked potential latencies measured at the surface electrodes \(S\text{ant}\) tended to be slightly shorter than the latencies measured at the corresponding needle electrode \(N\text{ant}\), without statistical significance.

Focal transcranial magnetic stimulation elicited a response at \(N\text{inf}\) in only five subjects [19.7 ± 2.8 ms (15.9–23.4 ms); \(P = 0.04\) with \(N\text{ant}\)].

Contamination

Contamination of the signal recorded at \(S\text{ant}\) by a signal arising from rib cage muscles occurred, according to our convention (see MATERIALS AND METHODS), in two of nine subjects with nonfocal transcranial magnetic stimulation (from upper rib cage muscles in one case and from abdominal muscle in the other case). One case of contamination was observed with focal stimulation. Figure 3 shows the recordings obtained in response to nonfocal transcranial magnetic stimulation in one subject where there was no contamination. Figure 4 shows the comparison between the \(N\text{ant}\) and the \(S\text{ant}\) trace in all subjects, also in response to nonfocal stimulation.

Patients with Phrenic Nerve Paralysis

Among the five patients with unilateral phrenic paralysis, the \(S\text{ant}\) electrode on the paralyzed side remained silent in response to nonfocal transcranial magnetic stimulation in four cases. In the remaining one, a motor-evoked potential was present, with a 19.2-ms latency that was markedly longer than the latency measured at the nonparalyzed side (16.1 ms). This suggested that the response recorded at the paralyzed side took its origin in the abdominal muscles.

Among the four patients with bilateral diaphragm paralysis, the electrode at \(S\text{ant}\) was silent on both sides in two cases. In the two other ones, a motor-evoked potential could be observed on both sides. However, its latency was always above 20 ms, and it was associated with a decrease in abdominal circumference; both elements suggest again that the response recorded took its origin in the abdominal muscles.

DISCUSSION

Mostly because of a difficult experimental design, this study concerns a relatively small number of subjects, which certainly limits the extent of the conclusions that can be drawn from it. However, it suggests that surface electrodes placed on the lower chest in such a way as to reliably measure the latency of the motor response of the diaphragm to phrenic nerve stimulation can also give access to a reliable measure of the latency of the diaphragm motor potential evoked by transcranial magnetic stimulation in the majority of cases.
cases. This is reassuring regarding the interpretation of studies, published or to come, that rely on the combination of transcranial magnetic stimulation with surface electrodes to diagnose central diaphragm dysfunction.

Comparison with Previous Data

The latencies of the motor evoked potentials recorded at $S_{\text{ant}}$ match those previously reported (28). Being obtained without the facilitating effect of an underlying contraction (28), they are expectedly longer than in other studies (6, 10, 35).

Signal Contamination

Relevance and reality of the problem. All electrical signals within the body are radiated in all directions from their source. A pair of electromyographic electrodes placed on the skin can thus record signals arising not only from the muscle that lies immediately under it, but, theoretically, from anywhere in the body (cross talk). The degree of cross talk at a given recording site is a matter of the spatial summation of signals that are produced close to the electrodes and of signals that are coming from far away. It involves the initial amplitudes of the contributing signals, which depend on the mass of the corresponding muscles, their activation, and the degree of synchronization of their contracting fibers. Indeed, close-to-synchronous mass action potentials are a major source of cross talk, with the electrocardiographic complex being the best example. Cross talk also involves the initial frequency content of the signals, because a signal will lose power and do more so in the high- than in the low-frequency range in proportion to the distance between its source and the recording electrodes. Reduction of the distance between differentially recorded electrodes reduces signal power and acts as a high-pass filter. It thus decreases the risk of cross talk. Similarly, the orientation of the recording electrodes relative to muscle fiber direction can affect the power spectrum of the recorded signal (30) and, therefore, modify the pattern of cross talk (29).

Transcranial magnetic stimulation is not capable of selectively activating the precise motor cortical area governing the contraction of a given muscle, particularly if the area in question is small. The coactivation of various muscles is thus unavoidable and occurs under the form of close-to-synchronous mass action potentials. This makes cross talk at the level of surface electrodes an obvious risk, either from muscles layered beneath the electrodes or from more distant ones through volume conduction. To use transcranial magnetic stimulation as a tool to study the cortical command of a given muscle, it is thus necessary to reckon with the question of cross talk. This issue does not seem to have been addressed in a systematic manner regarding the diaphragm, although several transcranial magnetic studies have been published. In a comparison of the responses of the diaphragm and of hypothenar muscles to cervical and transcranial magnetic stimulation, Zifko et al. (35) mentioned, in two subjects, the concordance between motor-evoked potentials recorded from a surface diaphragm electrode and from an intramuscular electrode. Conversely, Gea et al. (6) found dissimilarities between the motor-evoked potentials recorded from a diaphragm esophageal electrode and from surface electrodes, and raised signal contamination as a possible explanation for their results. Another explanation of this discrepancy could lie in the fact that esophageal electrodes and

Fig. 3. Examples of motor-evoked potentials obtained in response to ntTMS in subject 4, as recorded by, from top to bottom, $N_{\text{ant}}$, $S_{\text{ant}}$, $S_{\text{post}}$, $S_{\text{sup}}$, $N_{\text{inf}}$, and $S_{\text{inf}}$. The first vertical line indicates the stimulus, and the second vertical line marks the point of measurement of the response at the $N_{\text{ant}}$ site, which was taken as a reference. Responses at $S_{\text{ant}}$ and $N_{\text{ant}}$ begin synchronously; they occur way after the responses recorded by $S_{\text{post}}$ and $S_{\text{sup}}$ and before the response recorded by $N_{\text{inf}}$. This pattern validates the uncontaminated diaphragmatic nature of the $S_{\text{ant}}$ signal.
surface electrodes record electrical activity arising from different sources, namely the crural and the costal diaphragm, respectively.

At this point, it is important to clearly separate two issues. One is the interpretation of the whole motor-evoked potential recorded at Sant as reflecting the activity of the diaphragm, which implies that unwanted signals be insufficiently strong to cause a deflection at the recording site. This is clearly not an easy question to address, and the design of the present study does not allow us to answer it. The second issue, which is specifically addressed by our study, is the interpretation of the latency of the said motor-evoked potential as a reflection of the cortex-to-diaphragm conduction time. **Possibility to measure a reliable diaphragmatic motor-evoked potential latency.** Our study shows that, in normal individuals, this possibility exists in the majority of cases. It is mainly supported by the concordance between the latencies measured at Sant and at the corresponding intramuscular site. This argument relies namely on the main assumption that concentric needle electrodes inserted in the diaphragm at Sant do not pick up close-to-synchronized mass action potentials from the intercostal or abdominal muscles. Several arguments support this contention. The first argument lies in the major reduction in amplitude of the cardiac artifact recorded by the intramuscular electrodes compared with that observed on surface record-

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**Fig. 4. Motor-evoked potentials obtained in response to nTMS in the 9 subjects studied.** For each subject, the tracings obtained from Nant and Sant are shown. Each number corresponds to a subject.
ings (see MATERIALS AND METHODS). In regard to the intercostal muscles, the absence of electromyographic activity at \( N_{\text{ant}} \) during voluntary expiration makes a contribution of intercostal muscles to the recorded signal unlikely (2, 32). This does not rule out cross talk due to an intercostal close-to-synchronized mass action potential, but the very existence of such potentials in response to transcranial magnetic stimulation is questionable in view of a probably very small motor cortical representation. The small muscle mass involved sets a limit to the power of the corresponding signal, if any. Of note, the observations made in our patients with unilateral phrenic nerve palsy (absence of response to transcranial magnetic stimulation on the paralyzed side in four of five cases; late response of probable abdominal origin in one case) are reassuring about a putative contribution of intercostal muscles to the signal recorded at the \( S_{\text{ant}} \) site. In regard to the abdominal muscles as a source of cross talk, the absence of electromyographic activity at \( N_{\text{ant}} \) during voluntary expiration is again reassuring. However, contrary to what is the case with intercostal muscle, the possibility to evoke a close-to-synchronized mass action potential in abdominal muscles from transcranial stimulation is a certitude (11, 22), and the muscle mass involved can confer a non-negligible power to this signal. The fact that the latencies measured by the abdominal intramuscular electrode \( N_{\text{inf}} \) were longer than the latencies measured by the diaphragmatic intramuscular electrode \( N_{\text{ant}} \) in all but one case suggests that the activation of abdominal muscles follows that of the diaphragm. Hence, abdominal muscle activity should not contribute to the first detectable deflection of the signal measured with surface electrodes over the eighth intercostal space. Here again, the observations made in the four patients with bilateral diaphragm paralysis that we studied are reassuring (absence of response to transcranial magnetic stimulation in two cases; latency of a response of obvious abdominal origin far longer than the expected diaphragm latency in the two others).

The recording technique used is probably important to our results. Indeed, the location chosen for the surface electrode \( S_{\text{ant}} \) provides maximal proximity to the diaphragm together with maximal distance from interfering upper rib cage muscle, and the small interelectrode distance should reduce the risk of cross talk from distant muscles. We have previously shown that, compared with the traditional diaphragm recording technique, these modifications could suppress contamination from thoracic muscles in response to cervical magnetic stimulation (13, 15, 16, 34). In regard to the abdominal muscles, one additional explanation of the small amount of cross talk between the \( S_{\text{ant}} \) and \( S_{\text{inf}} \) electrode could lie in differences in the relative direction of the muscle fibers respective to the interelectrode axis. Indeed, the fibers of the obliquus internus are oriented upward and midward and those of the transversus abdominis run downward and midward, and are not parallel with the diaphragm fibers. The spatial relationship between the abdominal fibers and the \( S_{\text{ant}} \) interelectrode axis and between the diaphragm fibers and the \( S_{\text{ant}} \) interelectrode axis should thus be different. This factor is known to modify the power spectrum of the signal recorded, which in turn can influence its distant transmission.

Responses of Extradiaphragmatic Muscles

To our knowledge, the responses to transcranial magnetic stimulation of the serratus anterior and pectoralis major have not been described before. The latencies that we measured at the corresponding recording sites (namely \( S_{\text{post}} \) for the latissimus dorsi and serratus anterior, and \( S_{\text{sup}} \) for the pectoralis major) appear reasonable in view of the location of the concerned muscles and of published data about neck muscles [6.5 ms with transcranial electrical stimulation (5) and 7 and up to 9.4 ± 0.6 ms with nonfocal stimulation (1) and focal stimulation (33), respectively]. Of note, the latencies of the motor-evoked potentials recorded at \( S_{\text{post}} \) and \( S_{\text{sup}} \) (13.8 ± 1.5 and 12.4 ± 1.8 ms, respectively) in our subjects are close to the latencies of diaphragm responses facilitated by an underlying contraction (13.5 ms on average in Ref. 35). As a result, the risk of signal contamination interfering with the determination of the latency of diaphragm motor-evoked potentials should increase with diaphragmatic facilitation if it occurs without an identical facilitation at the level of the coactivated muscles. The possibility to obtain a diaphragm response independently of facilitation (28) is thus not only important to keep transcranial magnetic stimulation a nonvolitional test but also to increase the reliability of the derived information.

Of note, in our study, the coactivation with the diaphragm of possibly contaminating muscles was inconsistent, particularly with focal transcranial magnetic stimulation. This indicates that it is probably, at times, possible to obtain a pure or close to pure diaphragm motor-evoked potential.

Practical Consequences and Conclusions

From the present observations, we submit that surface electrodes placed in the lowest accessible intercostal space close to the costo-chondral junction and <2 cm apart from one another are appropriate to determine the latency of the response of the diaphragm to transcranial magnetic stimulation in the absence of facilitation, namely under relaxation conditions. Two safeguards to detect possible signal contamination can be proposed. The first consists in checking that the considered potential is actually associated to a diaphragmatic contraction, as attested to by a mechanical indicator such as abdominal expansion. In the absence of such expansion, a motor-evoked potential should not be interpreted in terms of diaphragm response. The second safeguard consists of comparing the response obtained at the diaphragm recording site with that recorded by electrodes to 1) optimally pick up close-to-synchronous potentials arising from the latissimus
dorsi and serratus anterior (e.g., 2 electrodes in the 5th to 6th intercostal space, on the posterior axillary line) and 2) record activity from the upper abdominal muscles (e.g., 2 electrodes at the intersection of the midclavicular line and of a horizontal line drawn between the costal margin and the omblicus). A latency of the motor-evoked potential read at the lower anterior chest site <15 ms and not longer than the latency of the concomitant response at \( S_{\text{post}} \) should raise a strong suspicion of signal contamination from rib cage muscles. Similarly, a latency of the motor-evoked potential at \( S_{\text{lat}} \) of >20 ms and not shorter than the latency of the concomitant response at the abdominal site should raise a strong suspicion of signal contamination from abdominal muscles. In rare occurrences, it is conceivable that an abnormal closeness between the latencies at the diaphragm site and at the abdominal site is due to the conduction delay affecting the path to the diaphragm but leaving intact that to the abdominal site. Study of the effects of inspiratory facilitation on the diaphragm but leaving intact that to the abdominal site. to the conduction delay affecting the path to the diaphragm site and at the abdominal site is due that an abnormal closeness between the latencies raise a strong suspicion of signal contamination from abdominal muscles.

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