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Contraction response to muscle percussion: A reappraisal of the mechanism of this bedside test

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HIGHLIGHTS

- We studied the contraction evoked by hammer percussion of muscle in healthy humans.
- Contraction evoked by muscle percussion stems from direct excitation of the muscle.
- Muscle percussion also excites motor axons within the muscle.

ABSTRACT

Objective: To study whether the contraction evoked by muscle percussion stems from the excitation of the muscle or of the nerve and to discuss the changes of this response in neuromuscular disorders.
Methods: In 30 neurologically healthy patients undergoing surgery (for ear, nose, or throat problems unrelated to the study) under general anesthesia with propofol and sufentanil we measured with an electrogoniometer the maximal dorsiflexion of the ankle evoked by reflex hammer percussion of the tibialis anterior muscle before and under neuromuscular junction blockade with rocuronium bromide. In 3 additional healthy volunteers we searched for F-waves to disclose whether percussion excites axons within the muscle.
Results: Responses from 28 neurologically healthy patients (15 women) were analyzed after exclusion of 2 due to technical problems. Mean age (SD) was 28 (9) years. Maximal dorsiflexion of the ankle was not significantly modified by neuromuscular junction blockade (mean difference 0.01 mV [95%CI, −0.07 to 0.08], p = 0.879). Muscle percussion evoked F-waves in the 3 healthy volunteers tested.
Conclusions: Maximal contraction response to muscle percussion has a muscular rather than a neural origin. However, percussion also excites axons within the muscle.
Significance: These findings may provide clues to understand the changes observed in neuromuscular disorders.

1. Introduction

The contraction response to muscle percussion was first described in 1858 by Schiff who called it "idiomuscular contraction" in the belief that it was of muscular origin. Subsequently, it was noted that this response was *diminished* in patients with primary myopathic disorders (Babinski and Jarkowski, 1911; Patel and Swami, 1969) and in case of denervation (André-Thomas and de Ajuriaguerra, 1949), *prolonged* in patients with myotonia (Dejerine, 1914), and *retained* (Guillain et al., 1916) or even *enhanced* (Ropper et al., 1991) in Guillain-Barré patients. More recently it was shown that the response was *increased* in patients with peripheral nerve conduction block and that it could be *diminished* and *prolonged* in case of denervation (Magistris and Kohler, 1996) and in muscle rippling disease (Vorgerd et al., 1999; So et al., 2001; Torbergsen, 2002). Our group quantified further the parameters of the response to muscle percussion with an electrogoniometer in normal subjects and patients, and reported in

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the medical thesis of Schiller (1997) the responses to muscle percussion collected clinically in 1020 patients.

Most neurologists do not include muscle percussion in their standard examination, probably because of the uncertainty of its mechanism and clinical relevance. Contraction to muscle percussion could theoretically relate to: (i) a direct excitation of the muscle sarcolemma (i.e. a true idiomuscular response); (ii) a spinal reflex; (iii) an indirect excitation via the depolarization of intramuscular motor nerve fibers; or (iv) a combination of these mechanisms. The above hypotheses have been evaluated by several groups at different times. Direct muscle depolarization received the experimental support of Brody and Rozear (1970) who showed that the contraction response to muscle percussion persisted in curarized rabbits and in patients undergoing spinal anesthesia. The spinal reflex hypothesis is not tenable, since the delay of the mechanical (Strohl, 1913) or electrical (Brody and Rozear, 1970) response that follows percussion of a limb muscle is too short to involve the spinal arc; furthermore, response to muscle percussion persists after experimental neuromuscular junction blockade (Brody and Rozear, 1970) and in the Guillain-Barré syndrome, whilst the tendon reflex disappears (Guillain et al., 1916; Ropper et al., 1991). Indirect excitation of the muscle via depolarization of motor nerve fibers is likely since axons are known to have a lower threshold to electrical stimuli than muscle fibers; this hypothesis is supported further by the observation that the contraction response to percussion is best obtained in the region of the motor point (André-Thomas and de Ajuriaguerra, 1949; Magistris and Kohler, 1996; Schiller, 1997). The motor point is the region of the muscle that has the lowest threshold to electrical excitation (Walthard and Tchicaloff, 1961); it corresponds to the region where a great density of terminal nerve elements is found (Coërs, 1955). Thus, the response could be indirect, with percussion exciting intramuscular axons and in turn muscle fibers (Magistris and Kohler, 1996; Schiller, 1997). Alternatively, the response could result from the combination of a direct excitation of the muscle and an indirect excitation of axons.

To address further the mechanism and structures involved, we measured the response before and after blockade of the neuromuscular junction. This was done in neurologically healthy subjects undergoing general anesthesia for surgery that required profound muscle relaxation through neuromuscular junction blockade. Our primary hypothesis was that the contraction response would disclose a nervous component and would therefore be reduced with neuromuscular junction blockade. Should the contraction response persist unchanged despite neuromuscular blockade this would disclose a pure muscular origin. Eventually, we added a study to clarify whether intramuscular nerve fibers are depolarized by muscle percussion.

2. Methods

2.1. Study participants

Recruitment was done at the pre-operative anesthesia visit one week prior to surgery. Eligibility criteria were adult patients, $\leq$50 years of age, requiring general anesthesia with tracheal intubation for elective ear, nose and throat surgery necessitating profound neuromuscular blockade for orotracheal intubation. Non-inclusion criteria were a history of sensory motor deficits (e.g. cerebrovascular accident, myelopathy, peripheral nerve disorder); psychiatric disorders; dysfunction of the ankle joint proposed for testing (e.g. related to osteoarthritis, recent or old fracture with functional sequel); preoperative medication known to influence the function of the neuromuscular junction (e.g. aminoglycosides, phenytoin); electrolyte disorders; hepatic or renal dysfunction; and a body mass index <19 or >28 kg m^{-2}. Written informed consent was obtained from all patients. The protocol was approved by the institutional ethics committee (*commission cantonale d'éthique de la recherche de Genève*, protocol N° 12-071).

Three of the authors (AT, CC, MRM) participated as healthy volunteers in an experiment aimed at disclosing if F-waves were evoked by muscle percussion. The study protocol was approved by the ethics committee (amendment no. 1/PB 2017-00496).

2.2. Preoperative examination, anesthesia and neuromuscular junction blockade

All patients had a preoperative neurological examination by one investigator (AM) to rule out a neurological disease. This examination, that concerned both upper and lower extremities, included standard assessment of muscle strength, tactile and thermal superficial sensation (filaments for fine tactile perception and discrimination of hot/cold), deep sensation (pallesthesia and kinesthesia of proximal and distal joints), gradation of the tendon reflexes, and contraction of the tibialis anterior muscle to direct percussion.

Patients were fasted at least 6 h before anesthesia and did not receive any premedication. They underwent a standardized general anesthesia induction using the intravenous anesthetic propofol and the intravenous strong opioid sufentanil. All anesthetics were administered by one anesthesiologist (CC). Propofol was chosen as it has a negligible influence on neuromuscular transmission and muscle contraction (Suzuki et al., 1999). Monitoring of neuromuscular junction blockade was carried out in the anesthetized neurologically healthy patient according to international guidelines (Fuchs-Buder et al., 2007). The ulnar nerve was stimulated every 15 s by a train-of-four (TOF) stimulation using a TOF-Watch-SX® acceleromyograph (Organon Ltd., Dublin, Ireland). After calibration of the monitoring device and having obtained stable baseline values, rocuronium bromide 0.6 mg kg^{-1}, a non-depolarizing neuromuscular blocking agent of intermediate duration of action, was injected intravenously. This body-weight adjusted regimen corresponds to a conventional intubating dose in adults (Lysakowski et al., 2007). The trachea was intubated when a profound neuromuscular junction block was obtained (zero responses on TOF stimulation). Measurement of the M-wave of the tibialis anterior muscle evoked by supramaximal stimulation of the peroneal nerve assessed depth of the neuromuscular junction blockade of the muscle to be investigated.

2.3. Recordings

The main outcome was the ankle dorsiflexion caused by the contraction of the tibialis anterior muscle after muscle percussion. It was evaluated with a customized electrogoniometer that was previously used at our institution for a medical thesis (Schiller, 1997). The electrogoniometer is converting the deformation of an optical fiber into an electrical signal. The optical fiber, sensor of the electrogoniometer, was attached at both ends to the lateral front of one leg and to the lateral dorsum region of the foot as depicted in Fig. 1. Particular care was taken to ensure free movement of the ankle joint during data acquisition. On the basis of an angular acceleration of the order of 700°/s^2, reflecting the strength of the contraction response to muscle percussion (or the speed of muscle shortening), the calibration of the goniometer was 0.5 mV per 10 deg of angle. The cutoff value for the kinematic response detection was 0.05 mV corresponding to an angle joint displacement of 1 deg after tibialis anterior muscle contraction.

To assess the depth of the neuromuscular junction blockade of the target muscle, we recorded the M-wave of the tibialis anterior muscle to supramaximal electrical stimulation (stimulus duration 0.2 ms) of the peroneal nerve at fibular neck. Electrical surface

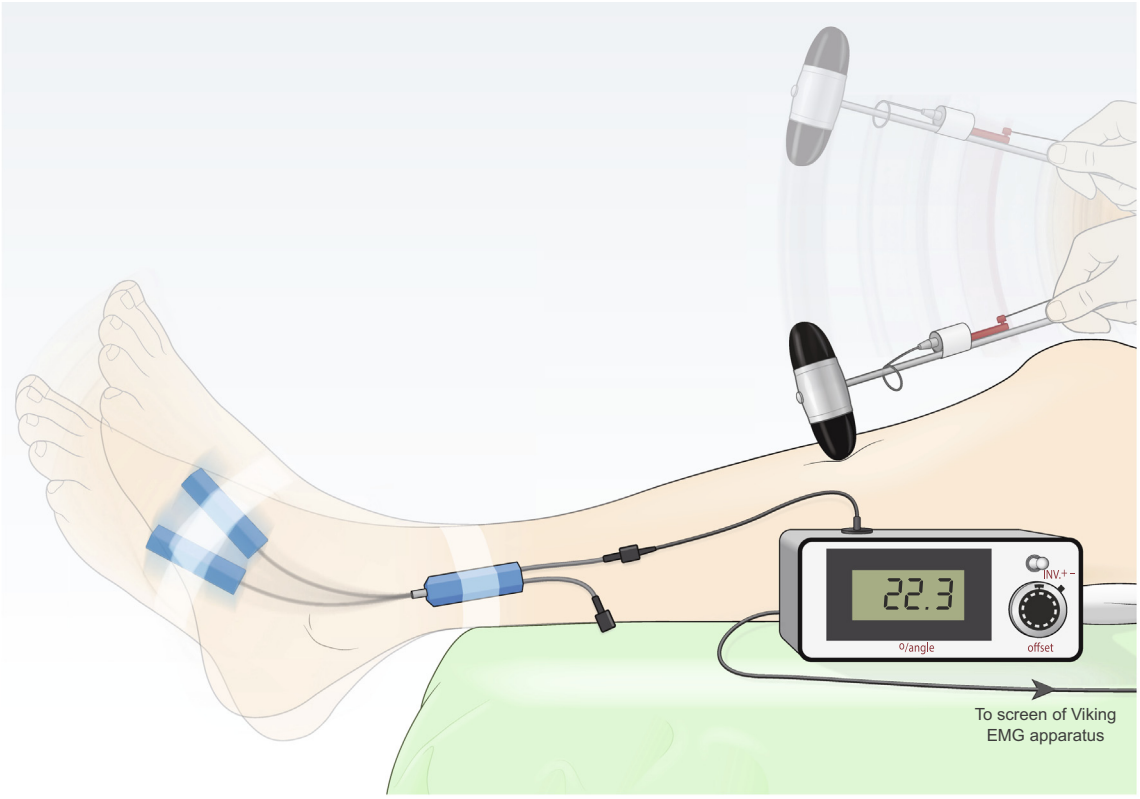

Fig. 1. Experimental setup for recording of the contraction response to muscle percussion. The tibialis anterior muscle is hit on its motor point with a reflex hammer triggering the sweep of the oscilloscope fed by the signal of the electrogoniometer measuring the angular ankle dorsiflexion (kinematic response).

recording was used with an active recording electrode placed over the motor point of the tibialis anterior muscle and connected to the negative pole of the amplifier, and a reference electrode placed over the anterior tibial tuberosity and connected to the positive pole of the amplifier. This montage yields large amplitude maximal M-waves (unpublished multicenter study). Muscle electrical response to percussion was not recorded because: percussion site and active electrode location were identical, and the percussion caused an artifact that interfered with the recording of the M-wave. Also, the main aim of our study was to address the kinematic contraction response to muscle percussion.

We also measured the dorsiflexion with the goniometer to electrical stimulation.

The electrical surface recording and goniometer signals were fed into the amplifier of an EMG apparatus (Nicolet VikingSelect, Nicolet Biomedical, Inc., Madison, WI) with bandpass set at 2 Hz to 5 kHz for surface M-wave recordings and at 1 Hz to 30 Hz for goniometer signal. Because the electrical response has a much shorter duration than the kinematic response (in the order of 1/20–1/40), the spectra of frequencies of these responses differed. For each response, we measured latency (ms), amplitude (mV), duration (ms), and area (mV ms) under the curve of the negative peak. The latency was measured to the onset of the negative deflection. It was often difficult to determine because the percussion generated a skin displacement that was detected by the goniometer and appeared as a small negative peak just before the onset of the main negative deflection produced by the dorsiflexion. We called it "skin artifact". The amplitude and duration measured were those of the negative peak. Because of the variability of the dorsiflexion due to unavoidable minor changes in percussion strength and location of contact around the motor point, measures of angular size parameters after muscle percussion were performed on the curve displaying the largest amplitude among a series of five percussions. Thus they were maximal responses to muscle percussion. The reflex hammer used for percussion was a Medelec model with a ring contact triggering the sweep of the Viking apparatus with a variable delay up to 5 ms.

Incomplete neuromuscular junction blockade of the target tibialis anterior muscle was defined as a M-wave to supramaximal electrical peroneal nerve stimuli >0.5 mV if a contraction response could still be detected by the goniometer after administration of the neuromuscular blocking agent, and also $\geq$0.1 mV if no contraction response could be detected.

All measurements were done by a single investigator (AM) under 3 conditions: at rest before induction of anesthesia (baseline), during general anesthesia before administration of the non-depolarizing neuromuscular blocking agent, and during anesthesia after administration of the non-depolarizing neuromuscular blocking agent.

Eventually, an experiment was carried out in 3 healthy authors in search for F-waves to electrical stimuli and to percussion performed at the motor point. This was done to detect whether percussion caused ectopic excitation of axons. First, a search for the region of the tibialis anterior muscle disclosing the lowest threshold to monopolar stimuli (motor point) was performed with a small hand-held surface cathode probe (area 30 mm^2) and a large plate anode (30 cm^2) placed on the calf in a region opposite to the motor point. Then, 16 (or more) maximal electrical stimuli (0.2 ms duration) were followed by 16 (or more) hammer percussions performed over the motor point. Recording of M- and F-waves used a tibialis anterior muscle belly-tibial tuberosity montage. A ground electrode was taped between the motor point and the muscle belly.

For the evaluation of the safety of the procedure the occurrence of any adverse event was recorded.

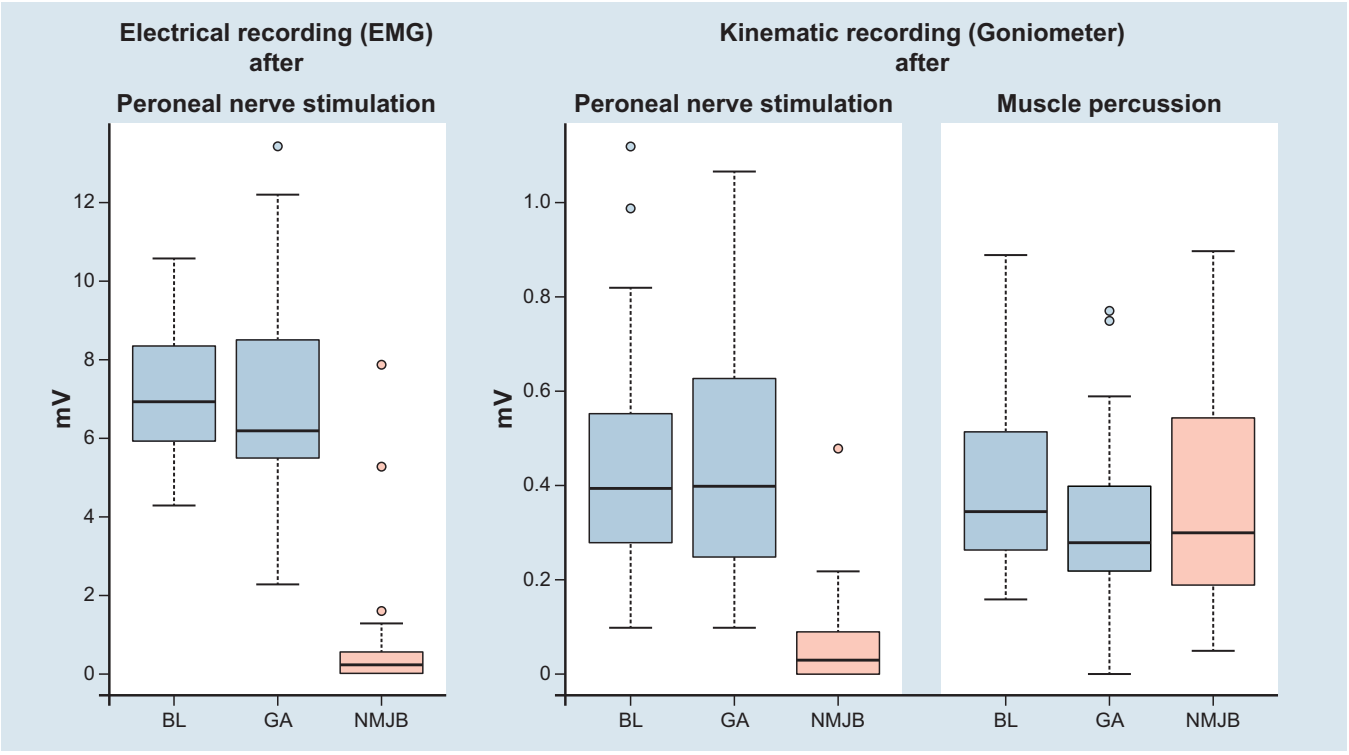

Fig. 2. Boxplot of electrical (EMG) and kinematic (Goniometer) recordings of the ankle dorsiflexion. Results of response amplitude of 28 subjects under 3 conditions: baseline (BL); during general anesthesia before neuromuscular junction blockade (GA); during general anesthesia with neuromuscular junction blockade (NMJB). Red colored boxes: electrical stimulation of the peroneal nerve at fibular neck shows that the neuromuscular junction blockade is nearly complete, while the kinematic response to percussion of the tibialis anterior muscle remains virtually unchanged. Median values are represented as the bold horizontal line and first and third quartiles are represented as a box (lower horizontal line of the box = 25th percentile, upper horizontal line of the box = 75th percentile). End of whiskers indicate minimal and maximal values, excluding the outliers (circles).

Table 1
Results of electrical (EMG) and kinematic (Goniometer) recordings. NMBA = Neuromuscular blocking agent. CI = Confidence interval. SD = Standard deviation.

Technique	Parameter	Baseline		Anesthetized without NMBA		Anesthetized with NMBA		Baseline – Anesthetized with NMBA		
		N	Mean ± SD	N	Mean ± SD	N	Mean ± SD	N	Mean difference [95%CI]	p*
Kinematic (Goniometer) recordings to muscle percussion	Amplitude (mV)	28	0.39 ± 0.18	25	0.33 ± 0.20	28	0.38 ± 0.24	28	0.01 [−0.07; 0.08]	0.879
	Duration (ms)	28	216.3 ± 62.8	25	196.0 ± 55.2	28	197.3 ± 42.7	28	18.9 [−3.2; 41.1]	0.090
	Area (mV ms)	28	44.3 ± 28.1	24	38.2 ± 25.3	28	44.9 ± 35.6	28	−0.6 [−12.2; 10.9]	0.911
	Latency (ms)	27	59.9 ± 24.5	24	51.0 ± 19.1	25	54.2 ± 12.5	25	3.9 [−3.9; 11.6]	0.313
Electrical (EMG) recordings after peroneal nerve stimulation	Amplitude (mV)	28	7.03 ± 1.69	25	7.06 ± 2.84	27	0.79 ± 1.75			
	Duration (ms)	28	9.4 ± 1.4	25	10.8 ± 4.1	18	12.5 ± 5.3			
	Area (mV ms)	28	36.3 ± 7.8	25	41.7 ± 17.8	27	6.4 ± 17.5			
	Latency (ms)	28	2.6 ± 0.9	25	3.0 ± 1.2	18	4.3 ± 2.5			
Kinematic (Goniometer) recordings to peroneal nerve stimulation	Amplitude (mV)	28	0.43 ± 0.26	25	0.43 ± 0.25	26	0.06 ± 0.11			
	Duration (ms)	28	204.8 ± 62.5	25	189.6 ± 35.7	15	173.7 ± 59.3			
	Area (mV ms)	28	52.4 ± 32.2	25	49.9 ± 34.1	26	7.1 ± 14.5			
	Latency (ms)	28	29.3 ± 8.8	25	29.3 ± 8.6	13	42.5 ± 9.2			

* Paired Student *t* test.

2.4. Sample size calculation

We assumed a 50% reduction of the mean amplitude of the goniometer signal between baseline condition and complete neuromuscular blockade; we expected an effect size of 1.5. Under these hypotheses we needed 7 subjects to achieve 90% power at an alpha level of 5%. Similarly, assuming a 30% reduction in the duration of the response between physiological conditions and complete neuromuscular junction blockade, we expected an effect size of 1.2. The number of subjects required to achieve 90% power at an alpha level of 5% was 10. Due to the uncertainty in the differences variability and in order to reach a sufficient statistical power in case of less optimistic effect sizes and to compensate for possible dropouts, we eventually included 30 patients.

2.5. Statistical analyses

The objective of this analysis was to determine whether neuromuscular junction blockade significantly modifies the contraction response to muscle percussion in humans.

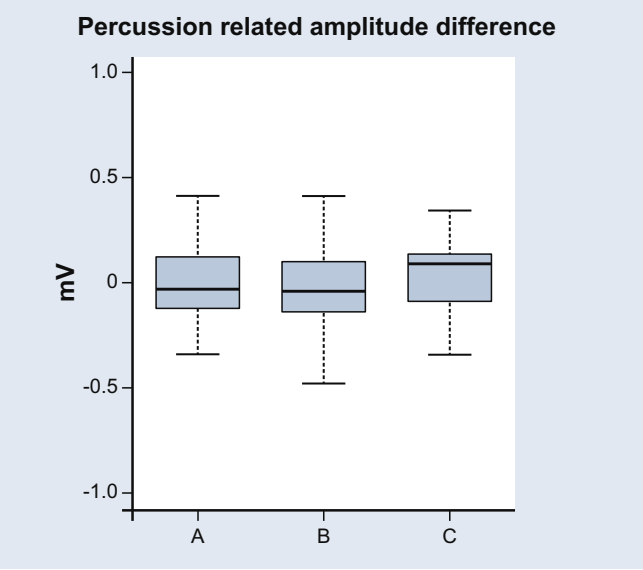

Fig. 3. Boxplot of the percussion related amplitude differences between neuromuscular junction blockade and baseline conditions. A. All neurologically healthy patients (N = 28). B. Patients with incomplete neuromuscular junction block (N = 15). C. Patients with complete neuromuscular junction block (N = 13). Differences were calculated as [amplitude under neuromuscular junction blockade] – [amplitude at baseline].

Categorical data are presented as frequencies and percentages; continuous variables are expressed as means with standard deviation (SD) or 95% confidence interval (CI). Differences in contraction response to muscle percussion (amplitude, duration, area and latency) between the baseline and anesthetized with neuromuscular junction blockade conditions were assessed using the paired t-test. The differences in amplitude between the 3 conditions baseline, anesthetized with and without neuromuscular junction blockade were assessed using a one-way repeated measures ANOVA. To explore if the difference in the degree of the neuromuscular junction block could have biased the results, we performed a posthoc stratified analysis: we assessed differences in contraction response to muscle percussion between baseline and neuromuscular junction blockade conditions in neurologically healthy patients with (a) complete and (b) incomplete neuromuscular junction blockade.

Statistical analyses were performed using R software (Vienna, Austria, http://www.R-project.org/). A two-tailed P-value of 0.05 was considered significant for all analyses.

3. Results

3.1. Patients

Thirty neurologically healthy patients were included between October 9, 2012 and June 21, 2013. All had a normal preoperative neurologic examination. Due to technical problems impeding proper data recordings and interpretation, data from 2 patients were excluded from all analyses. The mean age (SD) of the remaining 28 neurologically healthy patients was 28 (9) years, 15 (54%) were women, the mean body mass index was 22 (3).

3.2. Outcomes

There was no significant difference in the maximal dorsiflexion of the ankle after percussion of the tibialis anterior muscle under the 3 conditions: baseline, during general anesthesia before and after administration of the non-depolarizing neuromuscular blocking agent ($F[2,51] = 0.79$, $p = 0.461$).

The results after peroneal nerve stimulation or muscle percussion, under the 3 conditions: baseline, during general anesthesia before and after neuromuscular junction blockade are given in Fig. 2. The mean amplitude, duration and area under the curve of

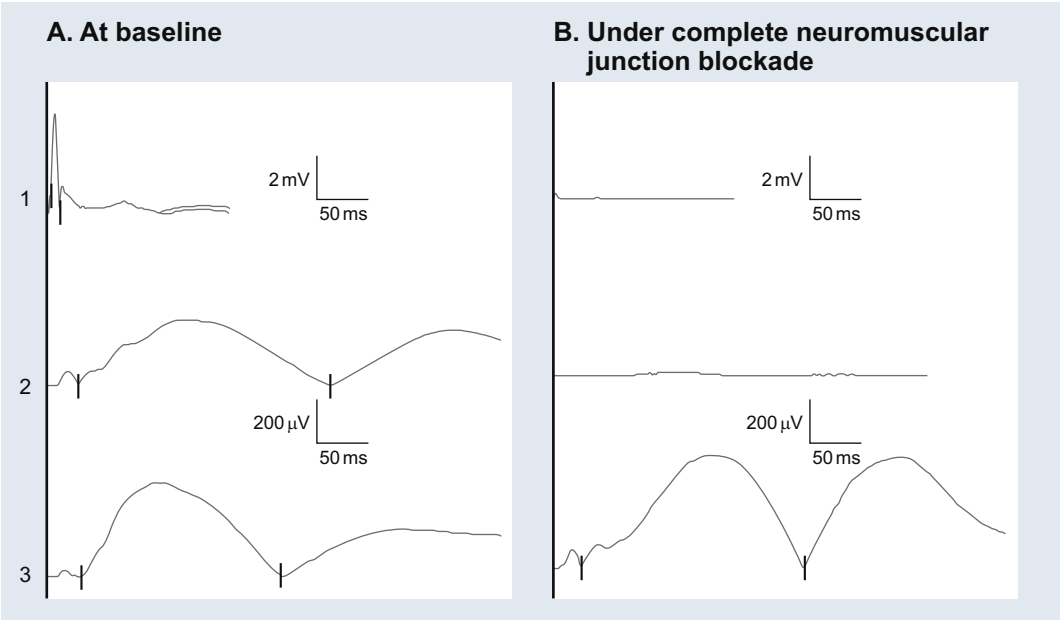

Fig. 4. Example of recordings obtained in a single neurologically healthy patient. 1. Surface EMG electrical response after electrical peroneal nerve stimulation at fibular neck (2 curves are superimposed). 2. Kinematic (Goniometer) response after electrical peroneal nerve stimulation at fibular neck. 3. Kinematic (Goniometer) response after percussion of the tibialis anterior muscle at motor point. Note disappearance of electrical and kinematic responses to peroneal nerve stimulation after neuromuscular junction blockade, but persistence of the kinematic response after muscle percussion, similar to that observed before neuromuscular junction blockade (baseline). Traces of kinematic responses (2 and 3) are rectified for easier assessment of the duration of the responses.

the dorsiflexion converted into an electrical signal at baseline, and after neuromuscular junction blockade are given in Table 1.

By contrast with the unchanged dorsiflexion to muscle percussion, M-wave and dorsiflexion of tibialis anterior after maximal electrical stimulation of the peroneal nerve were significantly reduced after neuromuscular junction blockade (Table 1; Fig. 2).

After administration of the neuromuscular blocking agent rocuronium bromide, a zero response to TOF stimulation using the acceleromyograph on the ulnar nerve was obtained, and the patellar tendon reflex disappeared in all 28 neurologically healthy patients. However, at the same time, in 13 (46%) of them an M-wave and sometimes a dorsiflexion to peroneal nerve stimulation remained detectable, indicating that the tibialis anterior muscle was incompletely paralyzed. Our posthoc analysis disclosed no statistical difference in the 2 groups of neurologically healthy patients with (a) complete (N = 13) and (b) incomplete (N = 15) neuromuscular junction blockade (Fig. 3; Supplementary Tables S1 and S2). Example of recordings obtained at baseline and under complete neuromuscular junction blockade are provided in Fig. 4.

Eventually F-waves were observed in response to both electrical stimulus and percussion performed at motor point in the 3 healthy volunteers tested. The M-wave to percussion was often smaller than to electrical stimuli, but the frequency of occurrence of F-waves and the ratio of the "maximal size F-wave/maximal size M-wave" were similar with both types of stimuli (Fig. 5).

No major, nor minor adverse events occurred.

4. Discussion

We investigated the contraction response to muscle percussion in neurologically healthy patients at baseline and under general anesthesia prior to and under pharmacological neuromuscular

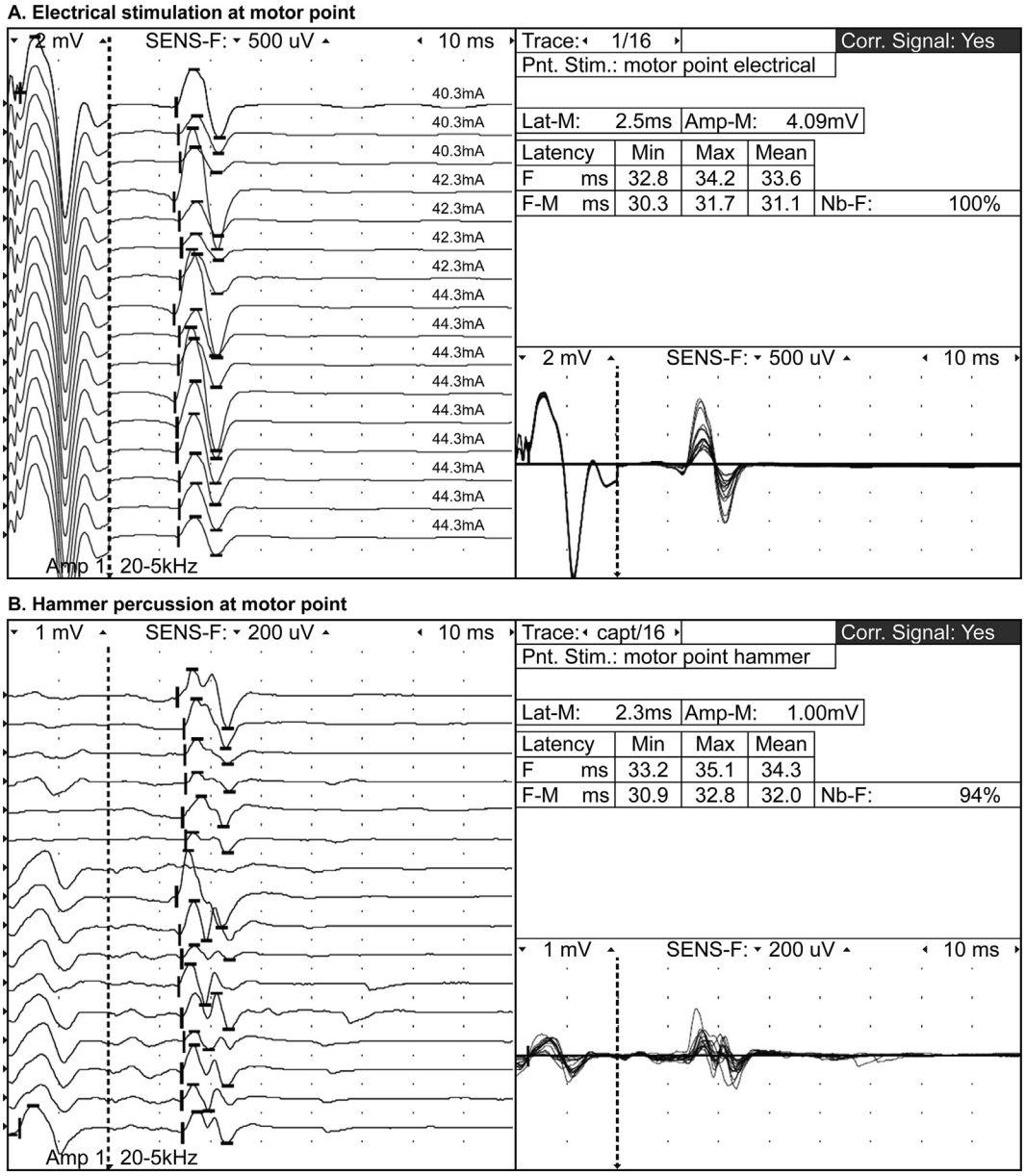

Fig. 5. Example of F-waves obtained in one healthy volunteer (CC) in response to (A) electrical stimuli, and (B) reflex hammer percussion applied at the motor point of tibialis anterior muscle. Recordings were from the muscle belly, circa 7 cm distally from the motor point. It used a muscle belly-tibial tuberosity montage with a ground electrode taped between motor point and muscle belly. Stimulus artifacts were attenuated by the "stimulus artifact suppression" function of the VikingSelect EMG apparatus, as indicated in the upper right corner of A and B "Corr. Signal: Yes".

junction blockade. We used the anesthetic propofol, which has only minor effects on neuromuscular transmission compared to volatile anesthetics and is the preferred anesthetic for clinical studies on neuromuscular blocking agents (Suzuki et al., 1999). As per visual observation and kinematic measurement, anesthesia and neuromuscular junction blockade had no relevant effect on the maximal muscle contraction response and ankle dorsiflexion. This was observed in both groups of neurologically healthy patients in whom neuromuscular junction blockade was complete or incomplete. This finding confirms that Schiff (1858) correctly supposed that the contraction response to muscle percussion is of muscular origin. It also confirms for the first time in humans the observation made by Brody and Rozear (1970) who performed a similar study in rabbits. These authors induced a "curariform" block of myoneural junctions through the intravenous administration of gallamine, a non-depolarizing neuromuscular blocking agent that is no longer used in clinical practice. Under neuromuscular junction blockade, the rabbits could no longer react with movements in response to painful stimuli and their blink reflex was abolished, whereas a brisk plantar movement of the foot was still obtained in response to muscle percussion. The latter disappeared only after sarcolemmal depolarization had been abolished by tetrodotoxin. The authors concluded that the muscle response was sarcolemmal in origin, and did not appear to be explained by stimulation of intramuscular nerve fibers or terminal axons (Brody and Rozear, 1970).

In our study, muscle percussion with the reflex hammer was performed manually, with a force that was not standardized. We tried to limit its variation by having only one investigator performing all measurements. Our study was done in neurologically healthy patients, such that our findings may not be extrapolated to patients with neuromuscular disorders. The latter may induce changes of excitability of both muscle and nerve fibers. These changes may consist in *hyperexcitability of the muscle fibers* that causes fibrillations observed in denervation and myositis (Katz and Miledi, 1964), myotonic discharges in myotonic disorders, and *hypoexcitability* observed in myopathies. *Hyperexcitability of axons* has been suspected to explain the fasciculations and myokymia observed in case of persistent conduction blocks (Roth et al., 1986; Roth and Magistris, 1987), and has been demonstrated by nerve excitability measurements in multifocal motor neuropathy (Kiernan et al., 2002). On another hand, axonal lesion is accompanied by a reduced response to the excitation of nerves.

To assess a possible excitation of axons by muscle percussion, we performed an experiment in the search for F-waves. F-wave is a muscle response that follows ectopic excitation taking place on the length of axons. Whereas orthodromic action potentials give rise to a direct response of the muscle, the antidromic action potentials depolarize a number of motor neuron cells that backfire and give rise to an indirect response of the muscle called F-wave. This response occurs with a delay that relates to the distance between the site of stimulation and the motor neuron; its shape varies depending on the motor units involved in the F-wave. This part of our study will benefit from a more thorough quantitative evaluation, but the observation that F-waves readily followed percussion, with a similar latency than those obtained by electrical stimuli performed in the same region (Fig. 5), demonstrates unequivocally that percussion excites axons. Such demonstration has not been reported previously to the knowledge of the authors. The role of this excitation of axons appears to be limited in our neurologically healthy patients in whom the response to percussion did not notably change under neuromuscular junction blockade. However, percussion that aims at obtaining the strongest possible contraction is probably sufficient to depolarize muscle fibers similarly before and under neuromuscular junction blockade. This may have obscured a response evoked by the excitation of axons. The existence of the latter may explain that responses to percussion of muscles are (i) best obtained from the motor point, (ii) increased in relation to conduction block, and (iii) decreased in case of axonal degeneration (cf. discussion in Magistris and Kohler, 1996). Early electrical studies have shown that in case of denervation the motor point to electrical excitation disappears (for a review see Walthard and Tchicaloff, 1961) whereas the muscle can still be excited, then by direct sarcolemmal depolarization and over its entire surface, if a stimulus of higher intensity and longer duration is used (Fischer, 1961). It is not surprising that responses to percussion follow the same rule.

Direct percussion of muscles with a reflex hammer enables to address a number of questions at bedside. The test is rather coarse and does not allow for a precise site of percussion, nor for a fine-tuning of the intensity and duration of the stimulus. Therefore, muscle percussion will not replace electrophysiological testing. Nevertheless, on many occasions information given by muscle percussion is in agreement with that provided by electrodiagnostic means and may foresee the result of the latter. Therefore this simple "mechano-diagnostic test" may orientate, or even suffice in a number of clinical situations (André-Thomas and de Ajuriaguerra, 1949; Brody and Rozear, 1970; Lehn et al., 2014; Magistris and Kohler, 1996; Schiller, 1997).

In the era of computerized imagery of the nervous system, the hammer of the neurologist remains a useful tool for the clinician and neurophysiologist. This is particularly true if one adds muscle percussion to tendon reflex testing.

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Conflict of interest

None.

Appendix A. Supplementary material

Supplementary data associated with this article can be found, in the online version, at https://doi.org/10.1016/j.clinph.2017.10.013.

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