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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