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RESEARCH ARTICLE

Jendrassik maneuver effect on spinal and brainstem reflexes

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Abstract

The effect of Jendrassik Maneuver (JM) has been extensively studied on monosynaptic reflexes in numerous muscles below the level at which the maneuver was performed. Here we hypothesize that the effect of JM could be observed also on other reflexes, indicating a widespread influence of performing a motor act such as the JM. We examined polysynaptic reflexes caudal (i.e., the withdrawal reflex of the lower extremities) and rostral (i.e., the blink reflex to supraorbital nerve stimulation) to the level of JM contraction. We have assessed soleus tendon (T) reflex; withdrawal reflex in tibialis anterior and soleus muscle; blink reflex (BR), blink reflex excitability recovery curve (BR-ER) and prepulse inhibition of the blink reflex. Our results showed that (1) T-reflex amplitude increased during JM and decreased just after and 15 min after JM; (2) no change in the withdrawal reflex; (3) R2 area of BR reduced significantly just after or 15 min after JM; (4) Prepulse inhibition in BR reduced significantly during JM; (5) no change in BR-ER. Our results indicate that JM leads to generalized effects on neural excitability at both caudal and rostral levels. Furthermore, JM has a selective effect on excitability of reflex circuitries.

Keywords Jendrassik maneuver · Tendon reflex · Brainstem reflexes · Monosynaptic · Polysynaptic reflexes

Introduction

Jendrassik Maneuver (JM) has been used in clinical setting for reflex reinforcement for more than a century, but there is yet no consensus on the exact mechanisms underlying the maneuver. A decrease of presynaptic inhibition is likely to take place, as there is no change in the background surface EMG activity during the maneuver (Dowman and Wolpaw 1988), and increasing segmental presynaptic inhibition by common peroneal nerve stimulation caused a decrease of reflex facilitation (Zehr and Stein 1999). However, it is not known if such decrease of presynaptic inhibition is a local or a generalized effect. In fact, the neurophysiological correlate of reflex facilitation by JM has been extensively documented in monosynaptic reflex responses (T and H reflexes) of numerous muscles, including triceps surae, tibialis anterior, quadriceps femoris, triceps brachii and rectus abdominis (Hannam 1972; Hagbarth et al. 1975; Tarkka and Hayes 1983; Jabre and Stalberg 1989; Zabelis et al. 1998; Myriknas et al. 2000; Gregory et al. 2001; Nardone and Schieppati 2008) caudal to the level corresponding to the contracting muscles used to execute the maneuver. To our knowledge, though, there is no study on the effects of JM on polysynaptic reflexes integrated above the contracting muscles. Examining such reflexes would enable us to evaluate how widespread is the process underlying reflex facilitation in JM.

Blink reflex, elicited by supraorbital nerve electrical stimulation, is a brainstem circuit with two components; one oligosynaptic (R1) and one polysynaptic (R2)

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component (Kimura et al. 1994; Valls-Sole et al. 1999; Molloy et al. 2002; Kumru et al. 2010). Blink reflex (BR) can be used to show the presence of excitatory or inhibitory pathways via two measures with different physiological bases: One is the blink reflex excitability recovery (BR-ER). This indicates the degree of inhibition caused by one stimulus on the blink reflex response to a second stimulus of the same intensity at a given interstimulus interval. It is thought to reveal the excitability changes occurring at the interneurons mediating the trigemino-facial brainstem circuit (Valls-Sole et al. 2004). The other is prepulse inhibition of the blink reflex (PP-BR). This indicates the degree of inhibition caused by a low-intensity extra-trigeminal stimulus of any modality on a subsequent blink reflex to supraorbital nerve stimuli. PP-BR phenomenon shows the modulation of the blink reflex by sensory input. The PP-BR is thought to be mediated by presynaptic mechanisms of sensory gating (Rossi and Scarpini 1992; Valls-Sole et al. 1999). Therefore, we hypothesized that, JM effects could be seen in polysynaptic reflexes, and that the effect of JM might be different on reflex circuits that are rostral or caudal to the spinal level of contracting muscles during the maneuver. The modulation of central nervous system at different levels during JM may help understand the underlying neuronal mechanisms of the effects of JM on reflex responses.

Methodology

Fourteen healthy subjects (6 women), ranging in age 20–65 years of age participated in the study. All subjects were informed about the complete experimental procedure and gave written consent prior to inclusion. The study was approved by the local ethics committee of the Institute Guttmann of Neurorehabilitation Hospital in compliance with the Declaration of Helsinki.

Routine electrodiagnostic equipment (Medelec Synergy, Oxford Instruments, Surrey, UK) was used for recordings. All investigations were carried out by the same examiner (HK) in the same room at similar environmental conditions.

Neurophysiological assessment

Neurophysiological assessment included soleus T-reflex, withdrawal reflex (WR) in the tibialis anterior (TA) and in the soleus muscle (SOL); BR, BR-ER and PP-BR. When testing WR, BR, BR-ER and PP-BR, subjects were asked to assess their perception of pain elicited by the electrical stimulation using numerical rating scale (NRS), in which 0 represented no pain and 10 represented maximal pain.

Soleus T-reflex

For the T-reflex study, subjects had bar electrodes (Medelec) attached to the skin overlying the soleus muscle on the right leg. The T-reflex was obtained by tapping with a reflex hammer (Kawe reflex hammer Trömner, Germany) over the Achilles tendon of right leg in five repeated recordings separated by at least 10 s.

Withdrawal reflex (WR)

Rectified surface EMG was obtained from TA and SOL muscles using bar electrodes. The EMG signal was amplified (0.5 mV/D) and band-pass filtered (50–1000 Hz). The WR was evoked by electrical stimulation through a bipolar electrode fixed over the posterior tibial nerve (PTN) at the medial malleolar fossa. A stimulation train consisting of 5 stimuli of 1-ms rectangular pulses at pain threshold intensity, at a frequency of 100 Hz, was used. Four sequential traces were recorded.

Blink reflex (BR)

Electromyographic (EMG) activity of the orbicularis oculi muscle was recorded on the right side with surface silver/silver chloride 9-mm-diameter recording electrodes attached to the skin, the active electrode in the middle portion of the muscle below the eyes and reference electrode lateral to the outer canthus. The EMG signal was amplified (0.2 mV/division) and band-pass filtered (50–1000 Hz). The BR was evoked by 10 mA electrical stimuli (0.5 ms rectangular pulses) delivered to the right supraorbital nerve (SON) with surface electrodes, the cathode over the supraorbital notch and the anode 3 cm above along the course of the nerve in the forehead. This intensity was used because it was able to induce clear BR in all subjects. Four consecutive sweeps were recorded with an interstimulus interval of 10 s. The subjects evaluated the pain perception induced by each SON stimulation using NRS (0 = no pain, 10 = max pain).

Excitability recovery curve of blink reflex (BR-ER)

Paired pulses were delivered to the right supraorbital nerve at the following interstimulus intervals (ISI): 160, 300, and 500 ms (Kimura and Harada 1976; Kumru et al. 2010). Four traces were recorded for each ISIs. The subjects evaluated the pain induced by each pair SON stimulation using NRS (0 = no pain, 10 = max pain).

Prepulse inhibition of blink reflex (PP-BR)

The electrical stimulus used as prepulse was delivered via ring electrodes to the digital nerves of the right index finger

at 1.5 mA of intensity (0.5 ms rectangular pulses). The prepulse stimulus was applied 100 ms before supraorbital nerve stimulation. Four single sweeps were recorded (Kumru et al. 2009; Kofler et al. 2013).

Experimental design

Experimental design consisted of 4 experimental conditions: (1) baseline (T0); (2) during JM (T1); (3) just after JM (T2) and, (4) 15 min after JM (T3).

All tests were performed while subjects were in supine position. In conditions T0, T2 and T3, subjects were in the relaxed position. In condition T1, subjects pulled a dynamometer with both hands and maintained 80% of their maximal force with the help of the visual feedback from the dynamometer screen. The maximal force was defined as the average of the maximal force that could be maintained for at least 3 s in 3 consecutive trials with 5 s break between each consecutive trial.

In all experimental conditions, T reflex, WR, BR, BR-ER and PP-BR were recorded in a randomized sequence.

Data analysis

For T reflex, peak-to-peak amplitude was measured.

For WR, the latency and area of the responses recorded in the TA and soleus muscles in four recordings were measured and the average for each subject calculated. Absent responses were not taken into account for latency calculations and were given the value of "0" for area.

For BR, the latency and peak-to-peak amplitude of R1 (the early ipsilateral BR component) and the latency and the area of the ipsilateral R2 (the late BR response) were recorded. Absent responses were not taken into account for latency calculations and were given the value of "0" for amplitude for R1 and for area calculations for R2.

For PP-BR, the mean percentage change of R2 area in trials with prepulse with respect to trials without prepulse were calculated for each subject separately (% change = (mean R2 with prepulse divided by mean R2 without prepulse) X 100). Group means of these ratios were then calculated separately for each experimental condition.

For analysis of the BR–ER, the mean area of all responses to conditioning stimuli per subject (R2: R2 response to the first supraorbital nerve stimulation), and the mean area of all responses to test stimuli per subject (mean of R2b: mean of R2 response to the second supraorbital nerve stimulation) for each ISIs were determined. The percentage recovery was calculated by dividing the mean R2b area by the mean R2 area for each subject and each interstimulus interval tested. Mean values of these ratios were calculated for each interstimulus interval to construct group mean BR-ER curves for each experimental condition (Kimura 1973; Kumru et al. 2010).

Data were expressed as mean and standard deviation. Distribution of data was assessed using the Kolmogorov–Smirnov test. As data were non-normally distributed, non-parametric tests were applied. Friedman test was used to compare the related data at T0, T1, T2, and T3 for each assessment. Wilcoxon test was used for post hoc paired comparisons. Student-*t* test was used to compare NRS for pain perception during the SON stimulation for BR and for PP-BR. The level of significance was set at $p < 0.05$. Statistical analyses were performed with SPSS 17.

Results

14 subjects accepted to participate but 2 male subjects were excluded, one 65 year old and the other 49 year old, because of small, unstable and unreliable T-reflex recording at baseline. The 12 remaining subjects who completed all studies were 5 females and 7 males, aged 21–58 years (mean 33.4 years; SD = 10.3 years). Four of them reported unpleasant sensations during electrical stimulation for withdrawal reflex and 5 for the electrical stimulation during BR tests.

The EMG baseline values did not change over time in any condition.

T-reflex

The T-reflex amplitude increased significantly during JM (T1) with respect to baseline (T0) condition (Wilcoxon test $p = 0.002$; Table 2). However, it decreased significantly just after (T2) and 15 min after JM (T3) compared to the baseline condition (Wilcoxon test; $p = 0.019$ and $p = 0.013$ respectively).

WR

The withdrawal reflex area did not show a significant change between conditions. Perception of pain during stimulation for WR did not change significantly at any point of evaluation ($p > 0.05$ for all comparisons, Table 2).

BR

The latencies of R1 and R2 showed no significant change in any of the study conditions (Friedman test: $p > 0.05$ for latency of R1 and of R2; Table 1). There was no significant change, neither in the amplitude of R1 $p > 0.05$) nor in the area of R2 at T1. However, the area of R2 decreased significantly in T2 and T3 with respect to T0 ($p = 0.012$ and $p = 0.028$, respectively) (Table 1, Fig. 1).

Table 1 Blink reflex and blink reflex with prepulse

	Blink reflex					Blink reflex with prepulse			
	TO	T1	T2	T3		TO	T1	T2	T3
R1 latency (ms)	8.9 (0.3)	9.1 (0.2)	8.9 (0.2)	8.9 (0.3)	R1 latency (ms)	9.1 (0.4)	9.1 (0.2)	8.9 (0.3)	8.9 (0.3)
R1 amplitude (μV)	323.6 (148.7)	306.2 (177.8)	331.7 (170.3)	359.0 (255.7)	R1 amplitude (μV)	380.7 (246.1)	314.0 (236.6)	439.3 (320.8)	420.6 (336.3)
R2 latency (ms)	30.1 (2.6)	32.2 (3.7)	31.3 (3.4)	29.8 (3.5)	R2 latency (ms)	37.7 (6.1)	34.1 (4.1)	36.3 (4.6)	36.7 (5.8)
R2 area (μV ms)	4.3 (2.7)	4.0 (2.3)	2.9 (1.5)*	3.1(1.9)*	R2 area (%)	34.5 (16.5)	54.2 (28.5)*	32.9 (18.3)	29.3 (24.7)
NRS	3.1 (1.9)	1.9 (0.9)**	1.9 (1.4)**	1.8 (1.4)**	NRS	3.3 (2.3)	1.9 (1.1)**	1.8 (1.3)**	1.7 (1.3)**

Data expressed as mean (standard deviation)

T0 baseline condition, *T1* during JM, *T2* just after JM, *T3* 15 min after JM

*$p < 0.05$ in comparison to baseline responses (Wilcoxon test)

**$p < 0.05$ in comparison to baseline responses (Student's *t* test)

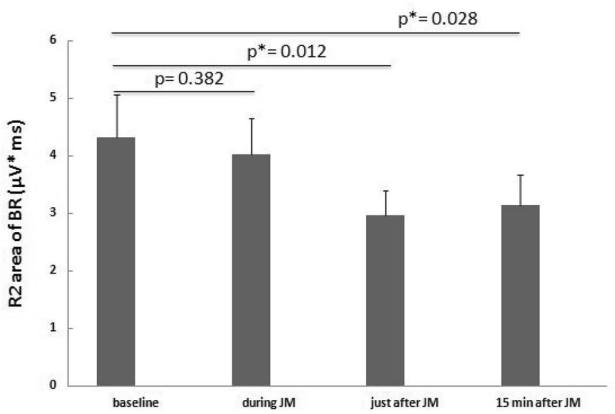

Fig. 1 Changes in the R2 area of BR. Data expressed as mean and standard error. Changes in R2 area of BR at baseline, during JM, just after and 15 min after JM. *P* value <0.05 according to Wilcoxon *t* test) in comparison to baseline response

Perception of pain induced by SON stimulation significantly reduced in T1, T2, and T3 in comparison to T0 ($p < 0.05$ for each comparison; Table 1).

BR-ER

No significant change was observed in the percentage inhibition of R2 at ISI 160 ms, 300 ms or 500 ms in any experimental conditions (Friedman test: $p > 0.05$ for all comparisons; Table 2).

Pain perception did not change between T1, T2 or T3 with respect to T0 (baseline) ($p > 0.05$ for each comparison; Table 1).

PP-BR

No significant difference was found in R1 amplitude or R2 latency in different conditions (Friedman test, $p > 0.05$ for each comparison, Table 1). However, there was significantly less inhibition of R2 at T1 with respect to T0 (Wilcoxon t, $p = 0.001$). Changes were not significant in T2 and T3 compared to T0 (Fig. 2).

Pain perception decreased significantly at T1, T2, and T3 with respect to baseline ($p < 0.05$ for each comparison).

Discussion

Our study illustrated the effect of JM at different levels of the nervous system: (1) the T-reflex amplitude increased during JM, as reported previously, and decreased just after JM, remained decreased 15 min after JM, (2) no change in the withdrawal reflex, a polysynaptic reflex caudal in respect to the level of JM-related muscle contraction at any part of the study; (3) the R2 area of blink reflex did not change during JM, but was found significantly reduced after JM, both, immediately and 15 min after. There was no change in the R1 amplitude of the blink reflex; (4) Prepulse inhibition reduced significantly during JM but returned to baseline values after JM, both immediately and 15 min after; (5) there was no change in BR-ER at any time of the evaluation.

Monosynaptic reflexes: T-reflex

A significant increase in T-reflex amplitude during JM has been reported previously (Ertuglu et al. 2018) but, as far as we know, no previous study has investigated the effects after the maneuver. Our study revealed significant

Table 2 Changes in T reflex, withdrawal reflex (WR) and blink reflex excitability curve

	T0	T1	T2	T3
T reflex Amp (μV)	1873.7 (921.5)	2556.9* (1278.5)	1679.8* (894.5)	1412.6* (709.0)
Withdrawal reflex (WR) (μV*ms)				
Lat of TA (ms)	82.0 (35.4)	79.78 (37.7)	61.97 (33.68)	69.75 (26.71)
Area of TA (μVms)	19.9 (33.2)	23.8 (40.1)	21.4 (45.7)	17.5 (34.5)
Lat of sol (ms)	93.5 (43.4)	97.5 (44.2)	75.46 (50.0)	94.4 (47.2)
Area of Sol (μVms)	25.7 (49.1)	23.3 (42.6)	18.5 (42.6)	25.6 (64.4)
NRS				
NRS- WR	4.9 (3.0)	4.7 (3.3)	4.8 (3.4)	4.7 (3.4)
Blink reflex excitability curve				
% of R2b/R2				
ISI-160 ms	17.8 (15.4)	17.3 (17.1)	14.4 (14.4)	21.4 (19.9)
ISI-300 ms	28.0 (20.6)	26.1 (20.7)	28.2 (22.2)	31.6 (27.0)
ISI-500 ms	54.2 (30.9)	39.1 (25.6)	43.2 (31.2)	36.9 (17.3)
NRS during blink reflex excitability curve				
NRS-ISI-160 ms	4.3 (1.7)	3.0 (1.5)	3.2 (2.0)	3.1 (1.8)
NRS-ISI-300 ms	4.2 (1.8)	2.8 (1.6)	3.4 (2.2)	2.8 (1.7)
NRS-ISI-500 ms	3.8 (2.0)	2.4 (1.4)	3.1 (2.0)	2.8 (1.7)

Data expressed as mean (Standard deviation)

Lat latency, *Amp* amplitude, *T0* baseline condition, *T1* during JM, *T2* just after JM, *T3* 15 min after JM, *ISI*: interstimulus interval, *TA* tibialis anterior, *Sol* soleus, *NRS* Numerical rating scale for pain perception of electrical stimulation

*$p < 0.05$ in comparison to baseline responses (Wilcoxon test)

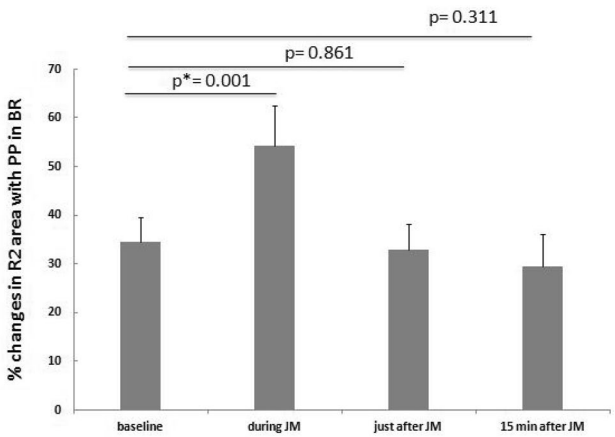

Fig. 2 Changes in the R2 area of BR with prepulse (PP). Data expressed as mean and standard error. Percentage changes in R2 of BR with prepulse at baseline, during JM, just after and 15 min after JM. *P value < 0.05 according to Wilcoxon t test) in comparison to baseline response

inhibition of the T-reflex that began just after the maneuver and was still present 15 min after. As we mentioned at the introduction, the mechanisms underlying reflex reinforcement by JM are not yet clearly understood. The facilitatory effect during JM on the stretch reflex led to the hypothesis of activation of gamma motoneurons (Burg et al. 1974; Murthy et al. 1978; Ribot-Ciscar et al. 2000) and/or other neuronal circuits, including those leading to decrease of presynaptic inhibition (Hagbarth et al. 1975; Dowman and Wolpaw 1988; Zehr and Stein 1999; Ertuglu et al. 2018). Bussel et al. (1978) showed that the increase in H and T reflexes during JM was resistent to ischemic block of Ia fibers, indicating that sensory inputs from muscle afferent fibers do not play a crucial role in reflex facilitation and questioning the role of the gamma loop.

The increase in the T-reflex amplitude during JM may be explained by presynaptic disinhibition; however, the decrease after JM may be associated to a separate mechanism. Given the frequency of the taps and the time interval between each condition, the effect of a possible habituation seems unlikely. We think that the cessation of the intense muscle contraction associated to JM might have caused a general decrease of motoneuronal and axonal excitability. Indeed, it has been shown that prolonged voluntary activity can produce a significant depression in excitability of the innervating motor neurons (Rossi et al. 2012). Possibly, a similar mechanism of post-contraction excitability depression may account for the decrease in size observed in the blink reflex, as an expression of widespread effect on the whole body's motoneuron pool. Further studies are needed to examine the late inhibitory effects of a strong muscle contraction.

Polysynaptic reflexes: withdrawal reflex

Withdrawal reflex showed no change during or after JM, suggesting that JM does not modulate this particular polysynaptic reflex, which is caudal to the level of JM muscle contraction. The withdrawal reflex (also known as nociceptive flexion reflex or flexor withdrawal reflex) is a spinal reflex intended to protect the body from damaging stimuli and it is mediated by a polysynaptic circuit resulting in activation of many motor neurons to generate a quick response (Rhudy and France 2007). The stimulus intensity at which a reflex response is evoked is often the intensity at which the subject reports pain sensation, and the strength of the withdrawal reflex is correlated with the intensity of experienced pain. In our study, with and without JM, subjects referred painful sensation but the JM neither changed the pain perception, nor the reflex size at any point. We interpret that the WR is a strongly routed protection reflex that cannot be modulated easily by other stimuli. Absence of WR modulation has been reported with somatosensory prepulse stimuli in upper and lower limbs by Alvarez-Blanco et al. (2009). We think that the withdrawal reflex does not respond to the changes in activity in somatosensory pathways induced by JM.

Polysynaptic reflexes: blink reflex

Our result showed that R2 area of blink reflex did not change during but it reduced significantly after JM. This effect was not accompanied by any change in R1 amplitude. The reduction in the size of the polysynaptic component had a similar pattern as the decrease in the T reflex. We can therefore speculate that both phenomena may share similar physiological mechanisms. However, the decrease in R2 with no accompanying decrease in R1 implies participation of trigemino-facial interneurons before affecting the alpha motoneurons. This points out to the possibility that rebound changes in presynaptic inhibition (increase after a transient decrease) could account for the decrease in reflex excitability in both levels. Obviously, the effects of presynaptic inhibition should be much more evident in polysynaptic (R2) than in oligosynaptic (R1) responses. Although there can be other explanations for the decrease of R2 (habituation, attention-related changes, etc.), we believe that they express a generalized post-contraction depression effect in reflex responses throughout the whole somatosensory pathway following strong contraction during JM.

Prepulse inhibition in blink reflex and BR-ER curve

In our study, prepulse inhibition of the BR reduced significantly during JM while it was unaffected at just after or 15 min after JM. PP-BR is an operational measure of sensorimotor gating, and the amount of gating is reflected by the degree to which the reflex response is suppressed by a weak prepulse. A prepulse is any low-intensity stimulus which is capable of inducing changes in the response to a subsequent suprathreshold stimulus. Prepulse inhibition may serve to protect the brain from sensory overload. Hence, reduced PP-BR, as seen during JM, means less filtering of the sensory information that reaches the brain, and as a consequence, more information arriving simultaneously at higher centers.

Here we hypothesize that JM caused a decrease in interneuronal inhibition at the brainstem level and therefore increased the responsiveness of nervous system during the maneuver. Although we cannot exclude the effect of sensorimotor gating of JM, the absence of changes in perception of supraorbital nerve electrical stimuli claims against a crucial impact of gating. However, another form of gating should also be considered to explain the transient decrease of PP-BR: afferent inputs derived from muscle contraction during JM might have masked the perception of the prepulse stimulus applied to the finger, which would then become less effective.

BR-ER is used to investigate the excitability of human interneurons at brainstem level (Kimura 1973; Basso et al. 1996; Esteban 1999; Valls-Sole et al. 2004). JM did not induce any change during or after the maneuver, suggesting that muscle activity at sites caudal to the BR did not change brainstem interneuronal excitability. This may appear to contradict the hypothesis put forward above that the changes seen in R2 may derive from presynaptic inhibition in trigemino-facial interneurons. However, the BR-ER reveals excitability changes occurring at the interneurons mediating the trigemino-facial brainstem circuit after a stimulus has circulated in the same reflex pathway, i.e., the change in reflex excitability after the passage of an excitatory volley (Kimura 1973). Therefore, the results of testing reflex excitability with the BR-ER may differ from those of testing the reflex on its own as a probe for changes in excitability elsewhere. An example of this is, for instance, in Huntington's disease, where the R2 response is usually larger than in control subjects, while the BR-ER shows decreased excitability (Esteban and Giménez-Roldan 1975; Muñoz et al. 2003; Valls-Sole et al. 2004). Differences have been also shown between changes in excitability tested with BR-ER and those tested with PP-BR (Valls-Sole et al. 2004). Therefore, we conclude that the effect of JM on the blink reflex induced by supraorbital nerve stimulus could occur at a presynaptic level in the trigemino-facial interneurons activated by inputs generated during muscle contraction.

Our study has many limitations: We did not select subjects according to age, gender or other demographic factors and, therefore, our results cannot be generalized to all groups of healthy subjects. We did not control for the

relatively long period of supine posture that subjects had to keep for the evaluation done at time period 15 m after JM. Reflex excitability might have changed during that period due to many factors, including attention, boredom and even electrode impedance. However, it is unlikely that the changes observed were due to random changes in excitability as all of them could be explained according to the physiological mechanisms that we hypothesized could be implicated in the maneuver.

In conclusion, this study shows for the first time that JM leads to generalized effects on neural excitability at both caudal and rostral levels generated during and following voluntary muscle contractions associated with JM. These effects are mostly characterized by early increase and later decrease of reflex excitability.

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Compliance with ethical standards

Conflict of interests The authors declare that they have no competing interests.

Ethical standards We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

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