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ORIGINAL ARTICLE

# Effects of passive stretching on post-activation potentiation and fibre conduction velocity of biceps brachii muscle

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**Abstract** Stretching is usually part of warm-up routines in many sports, but it affects the subsequent muscle force; therefore, it could negatively influence post-activation potentiation (PAP), one of the warm-up's main effects. The aim of this study was to evaluate the acute effects of passive stretching on PAP and fibre conduction velocity (CV). Seven subjects underwent 2 experimental sessions, control (C) and stretching (S), each consisting of 2 series (7 min resting) of 3 maximal voluntary contractions (MVC) of biceps brachii (5 s isometric contraction, 10 s recovery). During the resting phase of the S session, the biceps brachii was passively stretched (5×45 s stretches, 15 s recovery). Root mean square (RMS), mean frequency (MF) and CV were calculated from electromyography. Peak torque (pT) and half-contraction time (½CT) were measured and normalised by the arm muscular area (pTn). After C, pTn increased and ½CT decreased ($p<0.05$); moreover, MF and CV increased ($p<0.05$). After S, ½CT increased ($p<0.05$) and RMS decreased ($p<0.05$). Passive stretching could blunt the effects of PAP, presumably due to mechanical and neuromuscular changes. The observed changes in CV suggest a possible decrease in $Ca^{2+}$ sensitivity in contractile proteins. Therefore, the use of passive stretching in warm-up routines remains questionable.

**Key words** Electromyogram · Maximal voluntary contraction · Passive stretching · Warm-up

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## Introduction

Warm-up routines are ordinary practices in most sports activities. The main effects of these procedures are related to temperature changes. However, a post-activation potentiation (PAP) has also been proposed among the non-temperature-related mechanisms [1]. PAP represents the increase in muscle force production subsequent to a submaximal or maximal contraction. The mechanism underlying this phenomenon is a higher rate of cross-bridges formation, consequent to an increased sensitivity of the contractile proteins (actin and myosin) to $Ca^{2+}$ [2].

Stretching is a widely used practice in the warm-up routines of most sports, aimed at preventing injuries and improving joints' range of motion [3, 4]. Nevertheless, its effect on subsequent force production is currently under debate. Indeed, there is some evidence that acute static stretching could negatively affect maximal force output during both isometric [5, 6] and isokinetic muscle contractions [7]. Whereas the possible mechanical and neuromuscular mechanisms underlying these phenomena have been extensively assessed [8, 9], the changes in the electrical properties of muscle fibres after acute stretch-

ing have been poorly investigated. One of the possible mechanisms involved in force loss after stretching is a change in [$Ca^{2+}$] sensitivity of myofilaments after muscle fibre elongation [10–12].

To our knowledge, there are no studies evaluating the effects of static stretching on PAP. Hence, the main purpose of this study was to analyse the acute stretching-induced changes in a pre-activated muscle by means of EMG and force output signals analysis. In addition, as some stretching-related changes could occur at the level of fibre conductance, the secondary aim of this study would be to assess the possible modifications in muscle fibre conduction velocity (CV) induced by the stretching procedures [13] and its possible relationship with PAP changes. Indeed, CV seems an appropriate parameter to provide information about the possible alterations in motoneuron firing-rate and ion sensitivity of contractile proteins. However, the influence of stretching on CV is still poorly investigated. Some authors found a decrease of CV when the muscle was contracted at 30% of maximal voluntary contraction (MVC) at different lengths [14], while other authors reported an increase of CV after passive stretching [15].

The data obtained from the changes in PAP, EMG signal and CV induced by stretching in a pre-activated muscles could provide further knowledge on the detrimental mechanisms of stretching on subsequent muscle performance, and could provide useful information to the trainer in programming warm-up protocols.

## Materials and methods

### Subjects

Seven subjects [2 males, 5 females: age 26±5 (SD) years, weight 63.6±12.7 kg, height 169±9 cm], who were recreationally active and had no history of orthopaedic and neurologic diseases affecting the elbow joint and the wrist participated in the study. At the time of the experimental programme none of them was currently involved in any specific training plan for the upper limb muscles. Informed consent was obtained from each subject before participating in the experiments. The study has been approved by the Local Ethics Committee.

### Experimental design

The biceps brachii muscle was chosen as it is very well suited for EMG analysis, having long and parallel fibres with the main innervation zone located at the muscle belly.

Each subject was positioned on a hand-made anatomical ergometer, with the hand kept in a position halfway between pronation and supination. The subjects performed two different experimental sessions in randomised order: a control (C) session and a stretching (S) session, performed with an interval of at least 48 h between each other.

During the first experimental session, biceps brachii skinfold and median arm circumference were measured, in order to assess the arm muscular area (AMA). The elbow flexor muscles of the dominant arm were isometrically tested at an elbow joint angle of 120°. The skin area for the electrode placement was gently scratched with abrasive and conductive gel and then cleaned with ethyl alcohol, to ensure sufficient inter-electrode impedance. An EMG linear 16-electrode array was positioned on the longitudinal axis of the muscle and fixed with a bi-adhesive tape (Fixomull Stretch, Beiersdorf, Germany). The distance from the load cell and the epicondilus was taken in each subject to calculate the moment arm. The neutral EMG electrode was placed on the wrist of the tested arm. At the beginning of each test the subject was familiarised with the ergometer by executing some low-force trials. After a brief warm-up, a resting pause of at least 5 min was observed.

A schematic representation of the experimental design is shown in Figure 1. Each subject was then asked to perform 3 MVCs (5 s each, with 10 s of recovery). This contraction protocol was suggested by Vandervoort et al. [16], in order to emphasise the PAP effect, as concentric MVCs lasting 5–10 seconds cause the greatest PAP. Previous studies showed that the selected protocol did not produce muscle fatigue and did not induce a reduction in MVC [16, 17]. After performing the first MVC series, the subject was released from the ergometer and the arm was allowed to rest in a comfortable position for 7 min. To assure consistent placement of the arm in the anatomical ergometer between the first and the second MVC series, the epicondilus of the elbow was outlined with a marking pen, and the marked point was aligned with the rotational axis of the ergometer. Moreover, the elbow joint angle and the angle between the medial face of the omerus and the supero-external face of the clavicle near to the acromion-clavicle joint were measured by means of a bi-axial electrogoniometer (Biopac System, Inc., Santa Barbara, CA, USA). The EMG signal was monitored on a PC screen to control the complete relaxation of the muscle during the resting period. After this period, the tested arm was re-placed on the ergometer, taking care with the alignment between the skin markers and the rotation axis of the ergometer. Finally, another series of 3 MVCs was performed.

During the S session all the timing and the modalities of the C session were repeated, but the tested biceps

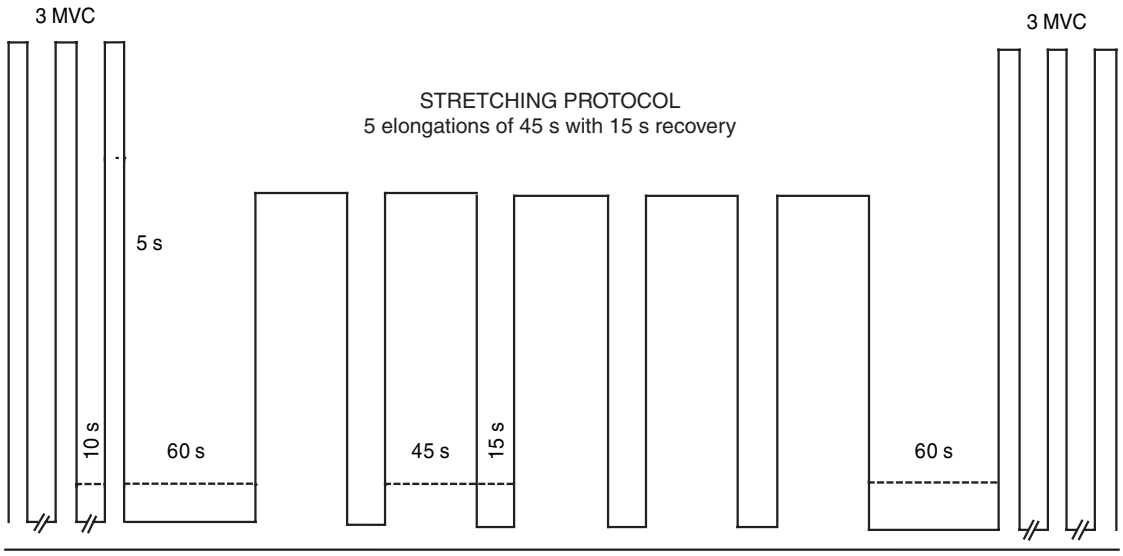


**Fig. 1** Experimental design

brachii was passively stretched by a unique and skilled operator during the 7-min interval occurring between the first and the second MVC series. The arm of the subject was kept horizontally abducted, intra-rotated and retroposed, and biceps brachial was stretched to the point of discomfort. According to Magnusson et al. [18], the stretching protocol consisted of 5 static passive stretches, held for 45 s (stretching period), with 15 s of rest between each stretch (recovery period).

## Force and EMG acquisition

The force output was recorded by a pre-calibrated load cell (SM-1000 N, Interface Inc., UK) connected to the wrist of the subject by a non-elastic strap. The force output was amplified by a differential amplifier (Biopac System, Inc., Santa Barbara, CA, USA), low-pass filtered at 5 Hz and stored (250 Hz) on a hard disk.

EMG signal was detected by a 16-electrodes linear array (200×30 mm), with an inter-electrode distance of 10 mm (16 channels EMG, LiSin - Centre of Bioengineer, Turin, Italy), amplified (1000×) and filtered (band-pass 5–500 Hz), and then stored (2048 Hz) on a hard disk for off-line analysis. The EMG parameters in time and frequency domain were evaluated by ad hoc EMG signal analysis software (EMGacq, LiSin - Centre of Bioengineer, Turin, Italy). To improve the quality of the EMG, the signals from two neighbouring channels (called a "triplet") were averaged and treated as a double-differential signal, according to the method of Hermens et al. [19].

## Signal analysis

The EMG analysis was performed in the time and frequency domains by calculating the root mean square (RMS) and the mean frequency (MF) of the signal. Fibre CV was also calculated (EMGacq, LiSin – Centre of Bioengineer, Turin, Italy). According to the European Recommendations for Surface Electromyography [19], EMG values were analysed during the middle 3 s of MVC, to avoid the analysis during transitional periods. Among the 16 EMG channels of the electrodes arrays, the ones that satisfied the following conditions were selected:

1) displacement of motor point along muscle longitudinal axis (the channels above the surface projection of motor point and the distal tendon were not considered); and
2) high cross-correlation threshold (CC): those channels that showed a CC value under 70% during biceps brachii contractions were excluded from the analysis.

Concerning force analysis, the peak torque (pT) and the half contraction time (½CT) were calculated. pT was considered as the average of the maximum plateau values reached during the 3 MVC trials in both series (plateau phase of the 3 subsequent contractions) divided by the moment at the arm. pT value was then normalised by the AMA of the subject (pTn). AMA was calculated as:

$$(C_A \times 10 - \pi B_S)^2 / 4\pi$$

where $C_A$ is the maximal circumference of the arm and $B_S$ is the biceps skinfold.

½CT was calculated as the average of the times of the 3 MVC trials to reach 50% of pT from the signal onset (con-

ventionally, when the force signal was larger than 3 standard deviations of the baseline noise for at least 3 samples).

### Statistical analysis

Where not otherwise stated, all the EMG and force parameters are shown as mean±standard deviation (m±SD), calculated from the samples obtained during the contraction phases. A Shapiro-Wilks test was used to confirm the normal distribution for all the parameters. A one-way analysis of variance (ANOVA) for repeated measure was performed to compare RMS, MF, CV, pTn and ½CT between basal condition, C and S sessions. The post hoc Newman-Keuls test was applied, as appropriate. The level of significance was set at $p<0.05$. Statistical analyses were performed using commercially available statistical software (Sigma Stat for Windows, v. 3.11, Systat Software Inc., USA).

## Results

### Torque parameter analysis

#### *pTn*

pT normalised by the arm muscular area (pTn) in C and S session are reported in Figure 2 (left column). Although not significantly, pTn tended to decrease in the S session between the first and the second MVC series (51±8 N/cm$^2$ and 49±8 N/cm$^2$, $p=0.15$), respectively. Conversely, during the C session pTn after the resting pause showed a significant increase from 52±3 N/cm$^2$ to 54±4 N/cm$^2$ ($p<0.05$ between 1st and 2nd MVC series and C vs. S).

#### *½CT*

The normalised ½CT values obtained in the C and S sessions are shown in Figure 2 (right column). ½CT significantly increased after stretching from 0.61±0.10 s to 0.69±0.11 s ($p<0.05$ between 1st and 2nd MVC series and C vs. S), whereas it significantly decreased during C session, from 0.636±0.03 s to 0.578±0.03 s pre- and post-resting pause respectively ($p<0.05$ between 1st and 2nd MVC series and C vs. S).

### EMG analysis

#### *RMS*

The normalised RMS values during the C and S sessions are described in Figure 3 (left column). RMS significantly decreased from 387±88 mV to 347±79 mV ($p<0.05$ between 1st and 2nd MVC series and C vs. S)

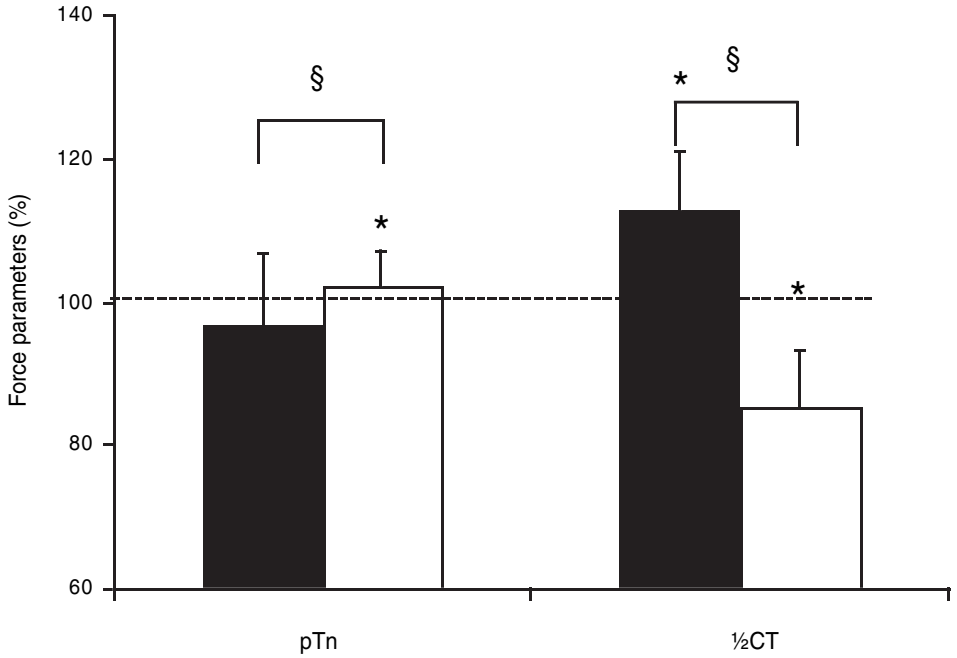


**Fig. 2** Mean (±SD, n=7) values of pTn (*left column*) and ½CT (*right column*) normalised as a percentage of the 1 MVC series value, considered as 100% (*dashed line*) in S (*black bars*) and C (*white bars*). * $p<0.05$ pre vs. post; § $p<0.05$ C vs. S

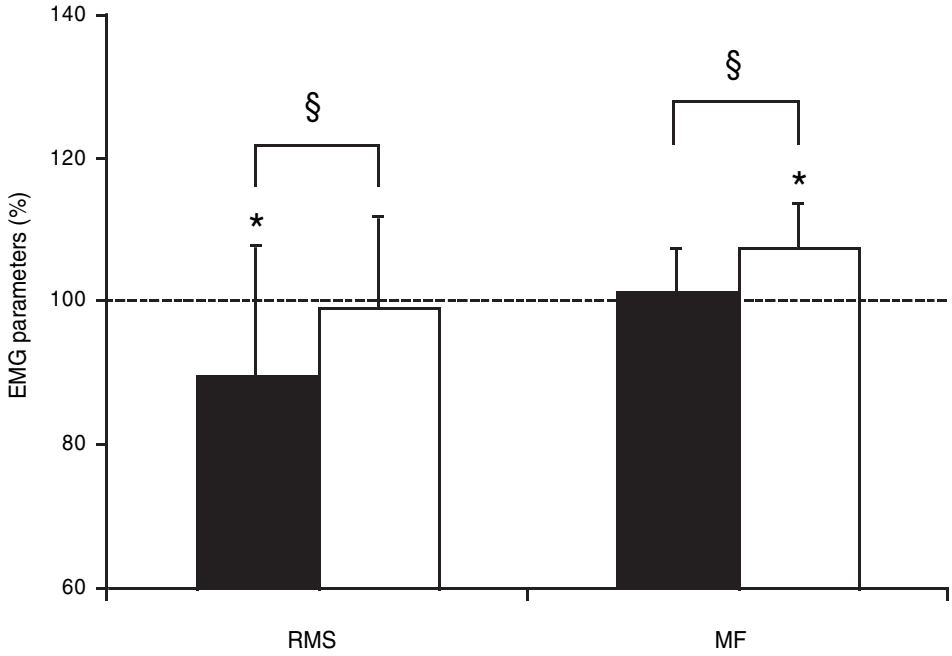


**Fig. 3** Mean (±SD, n=7) values of EMG RMS (*left column*) and MF (*right column*) normalised as a percentage of the 1 MVC series value, considered as 100% (*dashed line*), in S (*black bars*) and C (*white bars*). * $p<0.05$ pre vs. post; § $p<0.05$ C vs. S

during the 2nd MVC series during the S session only, whereas it remained unchanged in the C session, from 377±53 mV to 368±51 mV ($p=0.12$), before and after resting pause.

*Mean frequency*

The normalised MF values in the C and S sessions during the 1st and the 2nd MVC series are shown in Figure 3 (right column). MF significantly increased during the C session from 92±4 Hz to 99±5 Hz ($p<0.05$ between 1st and 2nd MVC series and C vs. S), before and after resting pause, respectively. Conversely, the difference between pre- and post-stretching MF values during the S session was not statistically significant (from 91±8 Hz to 92±6 Hz, $p=0.78$).

*Conduction velocity*

The normalised changes in CV during the C and S sessions are shown in Figure 4. CV significantly increased in the C session from 4.3±0.1 m/s to 4.7±0.1 m/s ($p<0.01$ between 1st and 2nd MVC series and C vs. S). However, during the S session CV remained stable from 4.2±0.3 m/s to 4.2±0.2 m/s ($p=0.59$) before and after stretching respectively.

## Discussion

The main finding of the present study is that an acute stretching bout, similar to those included in the warm-up routines of several sports activities, significantly modifies some electromyographic and force output features during isometric MVC in biceps brachii muscle, and blunts the PAP phenomenon.

Effects of stretching on torque parameters

In this protocol we observed a significant increase of pT during the C session, and a decrease, although not significant, of pT during the S session, suggesting that stretching procedure may impair the subsequent isometric maximal force production. These data confirm those of several previous works [7, 20, 21], despite other Authors found no effects [22, 23]. In particular, Behm et al. [17] and Nelson et al. [24] observed a pT depression after repeated passive stretching in the quadriceps muscles during maximal, voluntary isometric leg extensions of –11.4% and –7.2% respectively. Whether this stretching-induced decrease in pT may be related to changes in the mechanical properties of the muscle (such as an altered length–tension relationship) or to a central and/or reflex neural inhibitory mechanism is still a matter of debate [25]. The design of this study did not allow eluci-

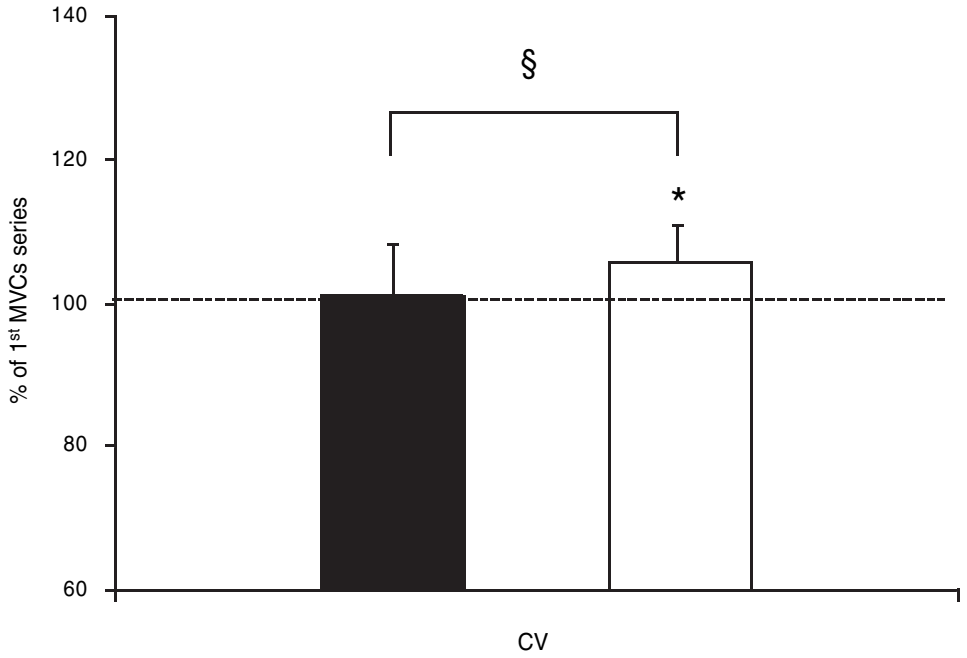


**Fig. 4** Mean (±SD, $n$=7) values of CV as a percentage of the 1 MVC series value, considered as 100% (*dashed line*), in S (*black bars*) and C (*white bars*). * $p$<0.05 pre vs. post; § $p$<0.05 C vs. S

dation of this point. However, in our protocol stretching caused a significant increase in ½CTn (+13%), whereas during the C session we observed a significant decrease of the same parameter (–7.9%). Similar results were reported by Avela et al. [25], who demonstrated a significant lengthening (+14%) of the total duration of the twitch contraction after stretching under both voluntary and electrical elicited isometric contractions. A possible reason for this phenomenon could be an inhibitory neural mechanism activated by the stretching procedures, as suggested by Behm and coworkers [17]. Passive stretching may cause a decreased motoneuron excitation, but this inhibition has been demonstrated to be lost immediately when the muscle recovers its neutral position [26]. Indeed, Rassier and MacIntosh [27] demonstrated on a rat gastrocnemius muscle that stretching significantly slows the twitch contraction and, accordingly, Rosenbaum and Hennig [28] showed a significant decrease in the force rise rate in the human triceps surae after passive stretching. Alternative explanations could be found in the possible changes induced by stretching on the aponeurosis-tendon complex and in a decreased musculo-tendineous stiffness [25, 29, 30].

Whatever the mechanism underlying the reduction of muscle torque and the changes in the rate of force development after a passive stretching bout, from a practical point of view the negative influence of stretching on maximal isometric contraction observed in this study may suggest that such a technique could be detrimental to subsequent muscle performance, at least when practised in the warm-up procedures of those sports activities that require powerful and brief contractions.

## Effects of stretching on EMG features

EMG amplitude value (RMS) was influenced in our study only by the stretching procedure, whereas it remained unchanged between the 1st and the 2nd MVC series during the C session. Conversely, the EMG spectral component MF was significantly increased during the C session, but not during the S session. A decreased RMS value after stretching was reported also by Avela et al. [25] in gastrocnemius muscle after a session of repeated passive stretching. The Authors suggested that these reductions could depend on a modification of the aponeurosis-tendon system, such as a relaxation and/or a plastic deformation. However, other studies did not find any difference in EMG amplitude in biceps brachii muscle after passive stretching under concentric isokinetic contractions [7].

Several studies reported stretching-induced decreases in muscle activation through the use of surface [5, 31, 32] and fine-wire EMG, as well as twitch interpolation technique [5]. These Authors showed that elongation of the muscle affects proprioception, through an increase in the activation threshold of muscle spindles [25, 33] and

Golgi tendonous organs (GTO) [34]. Moreover, mechanical and nociceptive reflex-circuits also seem to be involved [26, 33].

Reduced reflex assistance or "spindle support" as a result of stretch could explain the reduced performance; however, this mechanism acts mainly during the stretching procedures and it recovers immediately [33], even though the intrafusal muscle fibre could also be influenced by elongation, which leads intrafusal fibres to make rather compliant connections with the surrounding extrafusal fibres. That, in turn, lowers background levels of activity in spindles and reduces spindle stretch sensitivity [12]. The GTO reflex is an inhibition that occurs when the GTO, located at myotendon junctions, detect high force combined with muscle lengthening. The GTO feedback inhibits agonist activation to lower force production and reduce potentially injurious strain on the muscle. However, an extremely intense stretch is necessary to activate GTO [35]; moreover, their discharge rarely persists during maintained muscle stretch, and the inhibitory effects are transient [36].

Finally, mechanoreceptor (type III afferent) and nociceptor pain feedback (type IV afferent) may have reduced the central drive [26, 37]. However, in the present study the perception of pain was not present during the post-stretch, so these sensations as a cause of temporary activation failure of muscle fibres remained questionable.

### Effects of stretching on conduction velocity

A significant increase of CV occurred during the C protocol, but not during the S protocol. This increment was concomitant with the increase in MF. Therefore, CV seems to be influenced by passive stretching, as after stretching it remained unchanged. This effect could be ascribed to changes in motoneuron excitability and firing rate after passive stretching, as suggested by Guissard et al. [26]. Since CV is related also to ion concentration [38], a reduced sensitivity in [$Ca^{2+}$] in the contractile proteins due to passive stretching is presumable.

An alternative mechanism has been proposed by Jones et al. [11], who found a stretch-induced decrease in troponine-C sensitivity in binding $Ca^{2+}$ as a basis for the reduction in force output after muscle stretching.

### Overall effects of stretching on post-activation potentiation

In the present study the significant increase of MF and CV and force parameters during C are the results of PAP. On the contrary, pTn, MF and CV during S showed no differences.

Overall, our data seem to suggest that stretching could negatively depress the phosphorylation of myosin and the subsequent increased actin-myosin sensitivity to $Ca^{2+}$ typical of PAP. Moreover, it seems that stretching might be involved in some pre- and postsynaptic neuromuscular circuit that could influence the motor unit recruitment strategy [36].

### Limitations of the study

In this study we used an isometric contraction protocol. However, although isometric contraction allows surface EMG signal to be measured in a well known and standardised condition, it may be less appropriate to represent a typical sport action, where dynamic contractions are normally involved. It would be interesting to evaluate the effects of passive stretching on PAP during dynamic tasks, such as isotonic or isokinetic contractions or jump tests.

## Conclusions

In conclusion, our study seems to suggest that passive stretching could blunt the effects of PAP. This effect could be due to mechanical and neuromuscular changes that occurred in the muscle as a consequence of passive stretching. In particular, the observed changes in CV suggest a possible decrease in $Ca^{2+}$ sensitivity of contractile proteins.

Therefore, the use of passive stretching in warm-up routines, aimed at improving muscle performance, seems to be questionable, especially when a maximal contraction or brief and powerful tasks are required.

Finally, CV seems to be a valuable parameter to investigate the effects of passive stretching on the PAP mechanisms, and its use could be appropriate to elucidate the changes of muscle electro-mechanical and contractile characteristics secondary to muscle stretch.

**Conflict of interest statement** The authors declare that they have no conflict of interest related to the publication of this article.

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