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CURRENT OPINION

Non-Muscular Structures Can Limit the Maximal Joint Range of Motion during Stretching

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Abstract Stretching is widely used in sport training and clinical practice with the aim of increasing muscle-tendon extensibility and joint range of motion. The underlying assumption is that extensibility increases as a result of increased passive tension applied to muscle-tendon units. In some stretching protocols, this condition is not always met sufficiently to trigger adaptation within the muscle-tendon unit. For example, there is experimental evidence that both acute and chronic stretching interventions may increase the maximal range of motion in the absence of changes in the passive torque-angle curve. We contend that these results are partly explained by the influence of non-muscular structures that contribute only marginally to the passive torque. The potential candidates are the nervous system and fasciae, which would play an important role in the perception of the stretch and in the limitation of the range of motion of the maximal joints. At least in part, this may explain the lack of a significant effect of some chronic stretching interventions to change passive muscle tension.

Key Points

It is a basic assumption that extensibility increases as a result of increased volume or intensity of passive tension applied to muscle-tendon units.

In some stretching protocols, this condition is not met sufficiently to trigger adaptation within the muscle-tendon unit.

Non-muscular structures may limit the stretching amplitude and thus the magnitude of muscle tension.

The nervous system and fasciae could play an important role in the perception of the stretch and in the limitation of the maximal range of motion.

1 Introduction

Stretching is widely used in sport training and clinical practice with the aim of increasing muscle-tendon extensibility and joint range of motion (ROM). The underlying assumption is that extensibility increases as a result of increased volume or intensity of passive tension applied to muscle-tendon units. In this paper, we contend that in some

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stretching protocols this condition is not always met sufficiently to trigger adaptation within the muscle-tendon unit. This is because other anatomical structures and/or sensory perception may limit the stretching amplitude and thus the magnitude of muscle tension. At least in part, this may explain the lack of a significant effect of some chronic stretching interventions to change passive muscle tension [1, 2]. This may also explain why some studies showed that stretching protocols are ineffective at reducing the risk of muscle-tendon injuries [3, 4]. It may be that such stretching protocols do not load the muscular structures enough to trigger adaptations.

2 Mechanically- and Sensory-Based Theories

Sufficient passive skeletal muscle extensibility/maximal muscle length is thought to be an important outcome for enhancing function [5, 6]. Although the maximal muscle length can be measured by stretching ex vivo muscle-tendon units until tissue tear [7, 8], such investigations are not possible in humans for obvious ethical reasons. Consequently, the maximal muscle length is classically estimated through the maximal joint angle that can be reached [5]. For this measurement, the joint is often passively moved while the participant is instructed to remain relaxed. Various criteria are used to stop the joint motion, but generally they involve a level of discomfort experienced by the participant/patient, and in the literature it has been termed the "sensory endpoint of pain" [6] or maximal "stretch tolerance" [9, 10].

In the case where joint motion is not restricted by bony or articular limitation, a mechanically-based theory of muscle extensibility considers that the joint's maximal ROM is restricted by the tension of the muscle-tendon units being stretched [6]. However, there has long been experimental evidence that this may not always be so. For example, both acute and chronic stretching interventions may increase the maximal ROM in the absence of changes in the passive torque-angle curve (for review see Weppler et al. [6] and Magnusson [11]). Such results underpin the conclusion that sensory mechanisms associated with stretch tolerance might be involved [6, 11].

It is apparent that the aforementioned sensory effect is not limited to the joint being moved. For instance, Chaouachi et al. [12] showed that unilateral stretching of one lower limb was effective in increasing the ROM of the contralateral limb. More recently, Behm et al. [13] showed that an acute bout of stretching of the lower limbs increased the maximal ROM of the distant upper limbs and vice versa. Together, these results suggest that stretching can induce an increase in the "overall stretch tolerance," not limited to the joint where stretching took place. While the exact mechanisms behind these observations are not elucidated, it seems possible that central sensory mechanisms could be involved in the stretching effects. In regard to these findings, it is important to note that, although significant, the stretching effects reported by Behm et al. [13] were of low magnitude (between 5.2 and 9%) with small effect sizes ($0.35 < d < 0.56$). Consequently, the sensory mechanisms involved are unlikely to alone explain the large changes in ROM often reported after stretching.

3 The Important Role of Non-Muscular Structures

What other structures or mechanisms might be responsible for the limitation of ROM change with stretching? This is not discernible from typical measurements such as passive torque, which is generally considered to be representative of the passive tension developed by the muscle-tendon unit during the stretch, but actually represents the global resistance developed by the neuromusculoskeletal complex, including several muscular and non-muscular structures (e.g., tendons, ligaments, joint capsule, skin, fasciae, nerves, vessels, etc.) [14]. While the size and architecture of many of these structures limits their ability to contribute notably to global passive torque, they may be richly innervated with receptors that when strained trigger the central nervous system to limit range of motion. For example, consider peripheral nerves, in which nociceptive fibers are spread throughout the neural connective tissues and become active when exposed to an excess of mechanical, metabolic, or chemical stress [15, 16]. Within this context, a recent case study identified that hamstring stretching exercises induced notable injury to the sciatic nerve of a dancer [17], indicative of the susceptibility for damage to this structure during stretching exercise.

Nerves have traditionally been suggested in reducing ROM observed in some clinical tests. For instance, knee ROM in extension is significantly reduced when the patient is placed in the slump position (i.e., maximal lumbar and cervical flexion) compared to a neutral head position [18–20]. The change in nerve tension is often proposed to explain the distant decrease in ROM at the knee joint [18], particularly in clinical scenarios in which the nerve in question may already be irritated and there is amplification of the discomfort in response to a limited change in strain. There is some support for this conjecture. Coppieters et al. [21] showed that hip flexion (i.e., straight leg raising) increases the strain in the tibial nerve at the tarsal tunnel, without affecting other musculoskeletal structures. Furthermore, the work of Coppieters et al. [21, 22] eloquently showed that the addition of *sensitizing movements* (i.e., those movements considered to preferentially add load to

the neural tissue but not to adjacent non-neural tissues) during neurodynamic tests (e.g., slump test and straight leg raising) did not alter experimentally-induced muscle and/or myofascial pain. These studies support the validity of *sensitizing movements* preferentially loading neural tissue and cast doubt upon the involvement of muscle and myofascial layers as the sources of pain during these neurodynamic tests, and therefore upon their involvement in the ROM limitation.

Fascia, like peripheral nerves, is continuous from the trunk across the upper and lower limbs and hence has the potential to influence range of motion. In many instances, it is difficult to delineate whether nerves or fascia may be responsible for limiting motion. Here are three examples. First, using shear-wave elastography, Le Sant et al. [23] measured the shear modulus (an index of muscle tension [24–26]) of the hamstring muscles during passive knee extensions performed in several hip positions. Despite similar perception of the stretching, the maximal shear modulus of the muscles reached at the maximal knee extension angle was higher when the thigh was positioned in greater hip flexion positions. This result provides evidence that factors other than the passive muscle tension in the hamstrings contribute to the limitation of the passive knee extension. Second, an acute bout of stretching of the lower limb muscles (plantar flexors or hamstrings muscles) induced an increase in the maximal ROM of the cervical spine [27]. These results were attributed to the involvement of continuous structures connecting the lower limb and the spine, such as the myofascias and the peripheral nervous system. Third, in accordance with Mitchell et al. [28], Andrade et al. [29] showed that the maximal dorsiflexion angle, with the knee fully extended, is $\sim$20° greater with the hip is flexed at 30° (0°: neutral position) compared with the hip flexed at 90°. In addition, neither ankle passive torque nor shear modulus of the gastrocnemius medialis were affected by the hip joint angle (Fig. 1) [29]. These results provide evidence that the ankle ROM may change independently of the passive torque and gastrocnemius medialis muscle tension, which is known to be the most tensioned muscle during such a task [30].

Together, these results strongly suggest that the maximal ROM may be limited by non-muscular structures, and their course of action still needs to be unraveled. The potential candidates are the nervous system and fasciae, which would play an important role in the perception of the stretch and in the limitation of the joints' maximal ROM. Indeed, like the nervous system [15], fasciae [31] have a dense sensory innervation making them highly sensitive to stress/strain that can induce pain, and have been shown to be continuous structures that cross several joints. In regard to the latter, Cruz-Montecinos et al. [32] reported a significant correlation between the pelvic anteversion (forward tilting) in a long sitting position (knees fully extended) and the displacement of the deep fasciae of the gastrocnemius medialis. They explained this result by the presence of non-muscular tissues that might connect the distant hip and ankle joints, not only anatomically, but also mechanically, supporting the concept of *myofascial tissue connectivity*. Thus, fasciae could sustain notable stress levels during stretching maneuvers that involve polyarticular motion such as the slump position or straight-leg raising [20].

It is clear that more direct experimental evidence is required to ascertain the actual contributions of nerves and fasciae to the limitation of the maximal ROM at a joint. For that purpose, localized measurements of the mechanical properties of individual structures among the musculo-articular complex are needed. Such local measurements can be performed by ultrasound shear-wave elastography techniques (for review see [26, 33, 34]) that have been shown to provide a reliable estimate of both muscle [24, 37] and nerve [35] stiffness. Using this non-invasive technique, Andrade et al. [35] reported an increase in sciatic nerve stiffness during passive ankle dorsiflexion, confirming that the sciatic nerve has a biomechanical connection to the ankle joint (Fig. 2).

4 Conclusion

Our goal is not to claim that the maximal ROM is always limited by non-muscular structures, but we think that such structures could have a strong influence in some multi-joint configurations. In this way, while the aim of chronic stretching interventions is to increase the extensibility of stiff muscles, several studies demonstrated increased ROM but no changes in passive torque-angle relationships after stretching interventions up to 8 weeks [6]. We assume that these results are partly explained by the fact that stretching increases the extensibility (or stretch perception) of non-muscular structures that contribute only marginally to the passive torque, and therefore do not stand out in classic global torque measurements. In addition, it is probable that the contribution of non-muscular structures is very different among individuals. For example, following their analysis in healthy individuals, Ridehalgh et al. [36] highlighted large between-subject variability in both transverse and longitudinal sciatic nerve excursion (as measured *in vivo* with ultrasound imaging) in response to knee extension in varying positions of the hip. Thus, it could be that the structure (i.e., muscle-tendon unit, nerve, or fascia) that limits the maximal ROM of a joint might differ between individuals. As such, individualization of stretching exercises should be considered in order to target the appropriate structure. We contend that the recent

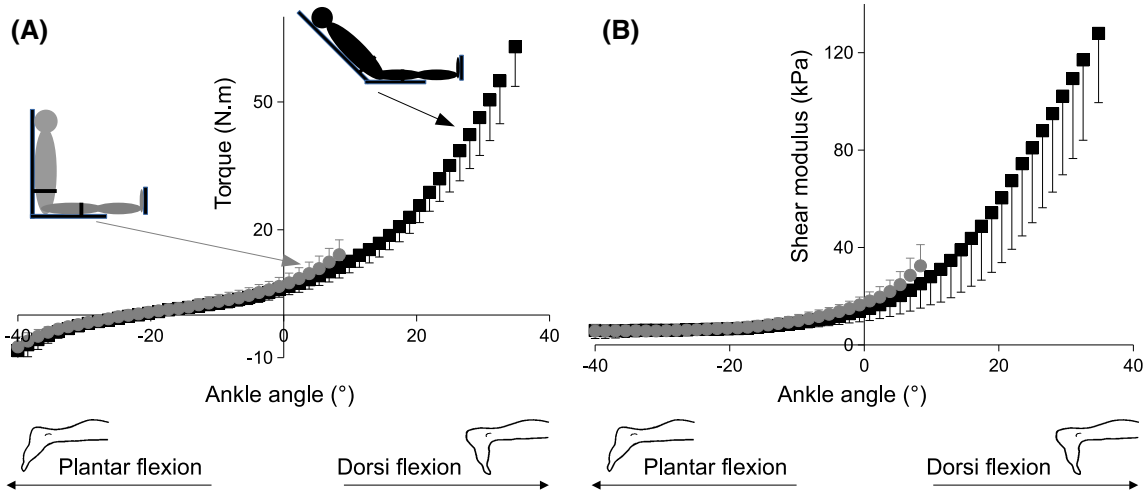

Fig. 1 Averaged **a** passive joint torque–ankle angle, and **b** shear modulus of the gastrocnemius medialis muscle–ankle angle relationships obtained during passive ankle dorsiflexion for two test conditions. *Grey circles* test condition with hip angle at 90°; *black squares* test condition with hip angle at 150°. No significant differences ($P > 0.05$) were observed for each common range of motion between conditions. Adapted from Andrade et al. [29]. Copyright © 2015 John Wiley and Sons. Used with permission

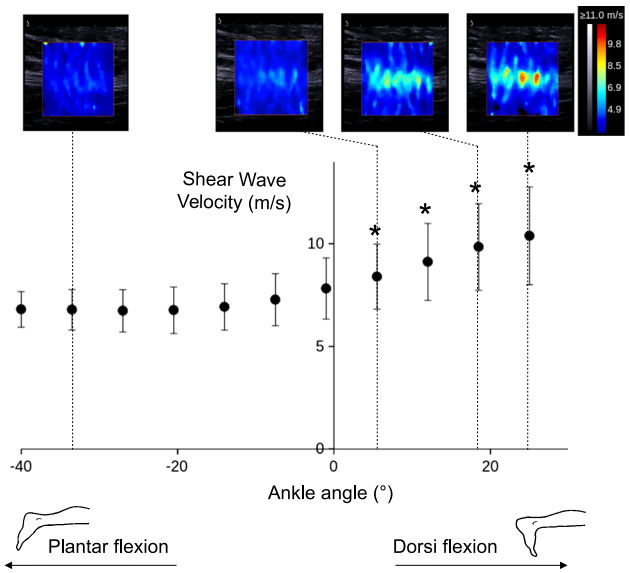

Fig. 2 Averaged relationship between the shear-wave velocity of the sciatic nerve and ankle angle. *Significant differences ($P < 0.01$) in shear wave velocity compared to the most plantarflexed angle (−40°). The *upper panels* correspond to representative examples of shear-wave velocity maps obtained from one participant. Adapted from Andrade et al. [35]. Copyright © 2016 Elsevier. Used with permission

literature strengthens the need for further experiments with localized and simultaneous biomechanical measurements performed on the muscles, nerves, and fasciae during various stretching procedures, in order to provide a better fundamental understanding of the structures and mechanisms associated with limitations to maximal ROM. Furthermore, this greater knowledge and understanding will allow more careful thought before implementing "stretching" programs, where strong techniques imposed upon delicate tissues such as neural tissues may be contraindicated or disadvantageous. Finally, we think that such research will have notable relevance with respect to injury prevention and performance in sport and other physical activities.

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Compliance with Ethical Standards

Conflict of Interest Antoine Nordez, Raphaël Gross, Ricardo Andrade, Guillaume Le Sant, Sandro Freitas, Richard Ellis, Peter J. McNair, and François Hug declare that they have no conflicts of interest.

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