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Proprioceptive Neuromuscular Facilitation Stretching

Mechanisms and Clinical Implications

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Abstract

Proprioceptive neuromuscular facilitation (PNF) stretching techniques are commonly used in the athletic and clinical environments to enhance both active and passive range of motion (ROM) with a view to optimising motor performance and rehabilitation. PNF stretching is positioned in the literature as the most effective stretching technique when the aim is to increase ROM, particularly in respect to short-term changes in ROM. With due consideration of the heterogeneity across the applied PNF stretching research, a summary of the findings suggests that an 'active' PNF stretching technique achieves the greatest gains in ROM, e.g. utilising a shortening contraction of the opposing muscle to place the target muscle on stretch, followed by a static contraction of the target muscle. The inclusion of a shortening contraction of the opposing muscle appears to have the

greatest impact on enhancing ROM. When including a static contraction of the target muscle, this needs to be held for approximately 3 seconds at no more than 20% of a maximum voluntary contraction. The greatest changes in ROM generally occur after the first repetition and in order to achieve more lasting changes in ROM, PNF stretching needs to be performed once or twice per week. The superior changes in ROM that PNF stretching often produces compared with other stretching techniques has traditionally been attributed to autogenic and/or reciprocal inhibition, although the literature does not support this hypothesis. Instead, and in the absence of a biomechanical explanation, the contemporary view proposes that PNF stretching influences the point at which stretch is perceived or tolerated. The mechanism(s) underpinning the change in stretch perception or tolerance are not known, although pain modulation has been suggested.

This article is concerned with proprioceptive neuromuscular facilitation (PNF) stretching techniques that aim to elongate a muscle. In the following text, the muscle or muscle group to be stretched will be referred to as the 'target muscle(s)' (TM) while a muscle or muscle group on the opposite side of the segment or joint will be termed the 'opposing muscle(s)' (OM).[1] For example, in the case where the tricep surae is to be stretched, the gastrocnemius and soleus muscles would be the TM and the pretibial muscles (e.g. tibialis anterior) the OM. While soft tissues other than muscle and its tendon are likely to be influenced by PNF stretching, only the effect on the musculotendinous unit (MTU) will be considered in this article.

In the early 1900s, Sherrington[2] defined the concepts of neuromuscular facilitation and inhibition, which subsequently led to the development of clinical PNF stretching by Kabat.[3] Initially, PNF techniques were used to aid the rehabilitation of clients with spasticity and paresis by either facilitating muscle elongation, supposedly through enhanced inhibitory mechanisms affecting the TM, and/or improving muscle strength through increased excitatory mechanisms affecting the TM.[4,5] The therapeutic use of PNF for clients with conditions other than those of neurological origin soon followed.[6,7]

Today, PNF along with static and ballistic stretching is commonly used to lengthen the MTU and as a result increase the range of motion (ROM) of a specific joint.[8,9] A static (isometric) contraction (traditionally maximal) of a stretched TM and/or a shortening (concentric) contraction of an OM to lengthen the TM, together with a slow and controlled approach to the stretch, is generally what differentiates PNF stretching from both static and ballistic alternatives. Moreover, traditional[10] and often contemporary[11] PNF practices promote movement around a series of joints in more than one plane to achieve diagonal or spiral movements, which differs to single-joint motion in a single plane as often seen in static and ballistic stretching. Unfortunately, most research into PNF stretching has focused on single-joint motion in one plane, thereby giving rise to a lack of concordance between the research and clinical environments.

PNF, static and ballistic stretching are all effective at enhancing joint ROM;[12-15] however, PNF stretching characteristically yields greater gains,[8,9,14,16-21] which may occur at a faster rate than that of static stretching.[22] Furthermore, PNF stretching has been found to improve both passive[18,23-27] and active flexibility,[2,8,15,28-30] with the latter arguably being more functional. Most of the stretching literature has concentrated on static and

PNF stretching and very little attention has been given to ballistic stretching.[12,14,16] There has also been a focus on the short-term changes in ROM stretching produces; however, little interest has been directed towards the comparative efficacy of various stretching techniques on long-term changes in ROM.

Studies investigating stretching techniques that elongate a MTU in an efficient period of time are important for athletic and clinical communities, since reductions in ROM may compromise function.[31] Presently, it is unclear what combination of intensity, duration and frequency across all types of stretching techniques is the most beneficial,[23] what the explicit advantages of enhancing ROM are,[32] and whether the stretching response varies between clinical and healthy populations. Moreover, there is a lack of understanding with respect to the mechanisms driving the observed changes in ROM.[32]

1. Descriptions of Proprioceptive Neuromuscular Facilitation (PNF) Stretching Techniques

The terms 'contract relax', 'hold relax' and 'contract relax agonist contract' are commonly referred to in PNF stretching literature.[8,11,23,33] Usually 'contract relax' and 'hold relax' represent a passive placement of the TM into a position of stretch, followed by a static contraction of the TM. The TM is then passively moved into a greater position of stretch.[8,20,27,34,35] 'Contract relax agonist contract' often refers to a technique that is similar to 'contract relax' and 'hold relax' except that following the static contraction of the TM, a shortening contraction of the OM is utilised to place the TM into a new position of stretch, which culminates in additional passive stretch.[34,36]

The above nomenclature and techniques appear regularly in the literature; however, there are also frequent deviations from these terms and descriptions. For example, in some works, 'contract relax' represents a technique that includes a shortening contraction of the TM instead of a static contraction.[35] Furthermore, the fact that 'contract relax' and 'hold relax' are often given to represent the same technique is a problem in itself. Another example adding to confusion in the literature is illustrated by Surburg and Schrader[11] in which reference was made to a technique called 'hold relax contract'. This technique was supposedly included in the work of Nelson and Cornelius,[37] although in the original citation the same PNF stretch technique was actually termed 'slow reversal hold relax'. Furthermore, the frequent inadequate descriptions of the stretching procedures in the literature[10,16,19,33,38-40] creates further problems for the reader. Such disparities and important omissions lead to difficulties in interpreting the research findings and applying these findings with any confidence. It is, therefore, important that a uniformed approach to PNF nomenclature and the way in which each technique is practiced is adopted. In this article, all variations within PNF stretching will be referred to only as 'PNF' in an effort to overcome the lack of uniformity and to avoid confusion.

2. Proposed Mechanisms Underlying the PNF Stretching Response

Autogenic and reciprocal inhibition have traditionally been accepted as the neurophysiological explanations for the superior ROM gains that PNF stretching achieves over static and ballistic alternatives.[41] Whether this pertains to both short- and long-term changes in ROM is unclear in the literature. Attempts have been made to clarify this deficit in the following discussion.

2.1 Autogenic Inhibition

Autogenic inhibition (historically known as the inverse myotatic reflex or autogenetic inhibition) refers to a reduction in excitability of a contracting or stretched muscle, that in the past has been solely

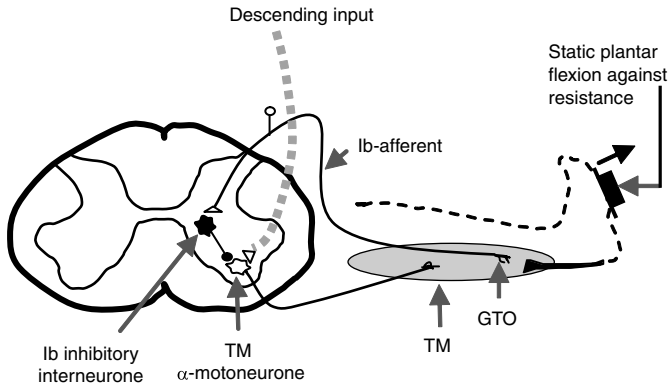

Fig. 1. The mechanism by which autogenic inhibition is purported to contribute to proprioceptive neuromuscular facilitation efficacy. A voluntary static plantar flexion is performed against resistance while the musculotendinous unit (MTU) is on stretch. The plantar flexion developed via descending drive and the existing level of MTU stretch result in an increased firing of tension-sensing mechanoreceptors (Golgi tendon organs [GTOs]) within the same muscle. Increased inhibition from Ib-inhibitory interneurones, a result of the amplified GTO input, results in a reduced level of excitability of the homonymous target muscle (TM), thereby facilitating additional stretch.

attributed to the increased inhibitory input arising from Golgi tendon organs (GTOs) within the same muscle.[42] The reduced efferent (motor) drive to the muscle by way of autogenic inhibition is a factor believed to assist TM elongation[8,19,22,43] (figure 1) and as such, most PNF stretches include a static contraction (traditionally maximal) of the lengthened TM in order to take advantage of autogenic inhibition. A maximal contraction has historically been used because it was thought that GTOs only respond to high forces but, in fact, GTOs are also sensitive to very low forces.[44]

The role of the GTOs in PNF stretching efficacy is, however, unclear.[45] Whilst, there is no doubt that GTOs can have an inhibitory effect upon the homonymous motoneurone pool,[42,46,47] in some circumstances pathways are available that enable GTO input to excite the same muscle[48,49] and inhibit or excite the heteronymous motoneurone pool.[42,46] Furthermore, during PNF stretching, any change in excitability brought about by GTO activity is likely to be limited to the period of tension within the muscle, as both animal[50] and human studies[44,51] have demonstrated that GTO activity *following* a contraction is either nonexistent or at very low levels. Taken together, autogenic-induced reductions in TM activity along with TM lengthening and longer lasting changes in ROM must be due to a more complex central and peripheral neurological organisation.

2.2 Reciprocal Inhibition

Voluntary contraction of the OM can lead to reduced activation levels in the TM through the development of reciprocal inhibition. The descending commands that activate the motoneurones of the OM, also provide excitatory input to Ia-inhibitory interneurones that synapse onto the motoneurones of the TM. The resulting inhibition of TM motoneurones can be further augmented by increased excitatory input arising from OM Ia-afferents converging onto the same Ia-inhibitory interneurones (figure 2), particularly during contractions with high fusimotor drive.[42,52-55] The increased Ia-afferent input from the OM is commonly reported in PNF stretching literature as the major contributor to TM elongation. Little consideration is given to descending influences and input from other sources such as recurrent inhibition[56] and presynaptic inhibition of the TM Ia-afferent.[57]

Several studies have demonstrated that PNF stretches that incorporate a shortening contraction of

the OM to lengthen the TM achieve greater gains in ROM and this effect is often attributed to reciprocal inhibition.[8,19,20,24,58-61] Although there is evidence to suggest that greater activation of the OM will result in greater levels of presynaptic inhibition of Ia-afferents targeting the TM motoneurone pool,[57] we are not aware of any clinical research that has examined this.

To understand the increase in ROM as a result of reciprocal inhibition, several studies have assessed TM activation through electromyography (EMG)[10,18,59-61] and/or Hoffman-reflexes (H-reflexes)[10,62,63] at the completion of the stretching procedure. The H-reflex is an artificially induced reflex brought about by electrically stimulating a mixed peripheral nerve,[64] which, in this context and like EMG, is used to estimate the excitability of the motoneurone pool in the TM.

At a given joint angle, before the end ROM, the EMG activity in the TM following PNF stretching is similar to that following a static stretching procedure.[17] This result casts doubt over the notion that PNF stretching is more effective than other stretching procedures due to reciprocal inhibition. While other studies have found that electrical activity is much greater in the TM following PNF stretching compared with static stretching,[10,18,59-61] these studies have assessed EMG amplitude at disparate muscle lengths, which is a known confounding variable.[17]

H-reflex data have demonstrated a greater reduction in motoneurone excitability in the TM following PNF stretching than that seen following static stretching, although this change in motoneurone excitability is limited to the duration of the stretching procedure (both static and PNF).[63] H-reflexes used in isolation[10,62,63] are, however, a poor indicator of motoneurone excitability. H-reflexes can be influenced by many factors, including presynaptic inhibition of the Ia-afferents, which can be mediated by a number of central and peripheral sources, including changes in joint angle.[64] All PNF stretching studies utilising the H-reflex have done so at dissimilar muscle lengths[10,62,63] and hence the results comparing motoneurone excitability are not convincing.

2.3 The Passive Properties of the Musculotendinous Unit

A muscle and its tendon has both viscous and elastic mechanical properties.[32,41] The viscous properties within a MTU will elongate in response to a slow sustained force and will resist rapid

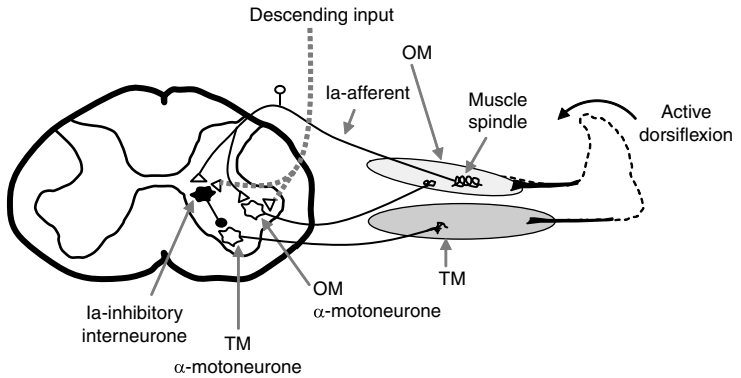

Fig. 2. The mechanism by which reciprocal inhibition is purported to contribute to proprioceptive neuromuscular facilitation efficacy. A shortening contraction of the dorsiflexors (the opposing muscles [OM]) results from descending input onto the OM α-motoneurone. In addition to exciting the OM, descending input and the OM Ia-afferent branch to excite the Ia inhibitory motoneurone. The consequent inhibitory input onto the target muscle (TM) α-motoneurone reduces the activation levels within the same muscle, thereby facilitating additional stretch.

changes in length.[65] While the MTU is under stretch, the amount of force generated by the viscous material to resist the elongation decreases over time ('stress relaxation').[65-67] As a result of this property, if the force attempting to lengthen the MTU is sustained, the MTU will gradually elongate, a property known as 'creep'.[68] The amount of force required to elongate the MTU is mostly dictated by the elastic properties of the MTU.[65]

Researchers have considered the passive properties of the MTU in order to explain how stretching can alter ROM. Of particular interest have been the relationships between stretching and viscoelastic stress relaxation, passive torque and muscle stiffness. Most research within this area has focused on static stretching; however, as PNF stretching techniques generally include a static stretching component, several findings from studies on static stretching are applicable.

Investigations into the passive properties of the human MTU have found that there is an increase in passive torque (the passive resistance of the MTU to stretch) and muscle stiffness (change in torque divided by the change in joint angle) as elongation of the TM increases.[32] When the passive torque response has been compared across static and PNF stretching, the PNF stretch not only yields greater gains in ROM but also greater passive torque measures at end ROM, although at a given angle (not at final ROM) passive torque measures are similar.[17] Longer term (3-week) static stretching programmes have also demonstrated enhanced passive torque measures at end range when compared with initial measures of end range, although improvements in ROM occurred.[32,69]

However, when a TM is held in a stretched position, passive torque and muscle stiffness decrease, that is, the MTU demonstrates viscoelastic stress relaxation.[32,70] Reductions in passive torque measures due to stretch are relatively short term[17,32] and last for approximately 1 hour after an 80-second bout of either a static or PNF stretch.[17] Even following a static stretching regime that was conducted twice daily over a 3-week period, the viscoelastic response was transitory.[32]

Only one reviewed study[69] concluded that structural changes within the TM were responsible for enhancements in ROM following a static stretching programme conducted 5 times per week over a 6-week period. Because of an increase in muscle stiffness at the new end ROM, it was concluded that changes to the material properties of the MTU had taken place. Although speculative, the authors suggested that such changes may be due to an increase in the number of sarcomeres in series. However, suggesting the presence of a structural adjustment is only valid when there is a reduced level of muscle stiffness at the same joint angle or when a greater ROM can be achieved with the same level of muscle stiffness[32] combined with an unaltered level of EMG activity.

Accordingly, the collective consideration of the biomechanical responses to stretching does not support the notion that changes in the passive properties of the MTU are responsible for more lasting increases in ROM.[32]

2.4 Other Proposed Mechanisms

Since more lasting changes in ROM cannot be convincingly attributed to autogenic and reciprocal inhibition, nor to changes to the passive properties of the MTU, it is considered by some that stretching alters the point at which stretch is perceived or tolerated and that PNF stretching may influence this to a greater extent than other stretching techniques.[13,17,32,71,72] The mechanism(s) behind a change in stretch perception or stretch tolerance are not known,[17] although an interruption to the transmission of pain is a plausible suggestion,[17,67] which may be centrally or peripherally mediated.[32] This is an important research direction because of its potential application, not only to standard stretching

methods, but also to other therapeutic practices that apply stretch to tissues, e.g. massage and mobilisation techniques.

3. Evidence-Based Recommendations

Although further investigation is required into the mechanisms underlying the PNF response, there have been sufficient applied studies conducted on this topic to support its efficacy. The fact that most of the research has focused on the PNF stretching response in healthy populations is a deficit in the literature.

3.1 Repetitions, Frequency and Duration of Intervention

One repetition of PNF is sufficient to increase ROM[12,21,23,60,73-75] with an expectant change in ROM from anywhere between 3 and 9°, depending on the joint.[12,21,23] Subsequent repetitions appear to produce relatively minor gains.[59,61] Conducting PNF twice per week,[21,22,36] even with a single repetition,[21] effectively augments ROM. For example, in Etnyre and Lee[21] there was a 21° change over a 12-week period in the direction of long-lever hip flexion when conducting one repetition of PNF stretching 2 times per week.

Regardless of the duration of the stretching intervention (e.g. 1 day to 12 weeks), changes in ROM will occur.[15,16,21,26,29,36,58] There is some evidence to suggest that the greatest gains in ROM will occur in the first half of the intervention period.[21,36] For example, in a 6-week programme, one PNF stretching group achieved a 20° change in passive long-lever hip flexion in the first half of the intervention period followed by an additional 12° in the final 3 weeks.[36]

3.2 PNF and Plasticity (Long-Term Range of Motion Changes)

There are mixed messages in the literature with respect to what the lasting effects PNF stretching has on ROM. For example, in one study, ROM improvements were no longer significant 6 minutes after five repetitions of a PNF stretch.[30] However, elsewhere, even after one repetition of a PNF stretch, ROM was still significantly higher than baseline values 90 minutes following the intervention in all but one of several muscle groups stretched.[73] In this study, subjects were allowed to walk around between the 30-minute testing intervals, which may have facilitated the lasting change in range. McCarthy et al.[39] demonstrated that ROM gains last for approximately 7 days after 1 week of twice-daily stretching. An additional study found that after conducting five repetitions of PNF stretching, performed 3 times per week over a 30-day period, it was necessary to continue with stretching once per week in order to maintain ROM improvements, although 3 times per week was necessary to further increase ROM.[14] Three studies aforementioned[14,30,73] utilised a similar PNF stretch approach, while it was not possible to determine the type of procedure used by McCarthy et al.[39] due to insufficient detail provided.

Several studies have noted that ROM increases do drop off relatively quickly once intervention ceases[22,30,39,58] and, therefore, PNF stretching should be conducted at least once or twice weekly, with ROM being regularly reassessed in order to better guide the parameters required to induce long-term ROM changes as indicated.

3.3 Static Contraction Duration of the Target Muscle

The studies reviewed used a static contraction duration of the TM between 3[76] and 15 seconds,[77] which in the majority of cases ROM increased when

any duration between this range was included.[14,18,23-26,61,76-78] In some cases, a longer static contraction duration is positively correlated with increased ROM.[36,58] For example, using a PNF stretching procedure that combined passive stretch, a static contraction of the TM and a shortening contraction of the OM, at the end of 6 weeks of intervention the mean change in the group that utilised a 5-second static contraction was 28° compared with the 10-second group that gained 33°. The influence of the static contraction duration, however, may be limited to techniques that include a shortening contraction of the OM following a static contraction of the TM.[58] Conversely, several studies[24,25,37,76,79] have demonstrated that ROM gains are independent of the static contraction duration, whether a shortening contraction of the OM is included or not and, therefore, we recommend that the static contraction should be held for 3 seconds, which is effective[24,37,76] and time efficient.

3.4 Static Contraction Intensity of the Target Muscle

ROM gains appear to be independent of static contraction intensities[26] and, thus, a low intensity (e.g. 20% of a maximum voluntary contraction [MVC] as used by Feland and Marin[26]) should be adopted preferentially in order to minimise risk of injury. There is some evidence to suggest that progressively increasing the contraction intensity (in this case over a 2-week period from 30% to 70% MVC) may produce larger increases in ROM as compared with a constant intensity (e.g. 50% MVC) over the same period.[79]

3.5 Opposing Muscle Shortening Contraction Intensity

To our knowledge, no studies have investigated the relationship between the level of contraction torque of the OM and ROM. As discussed in section 2.2, in relation to reciprocal inhibition, those PNF stretching techniques that include a shortening contraction of the OM have a greater impact on augmenting ROM.[8,20,24,58-61] The fact that the influence of various shortening contraction torques on ROM has not been investigated is a deficit in the literature.

3.6 Overall Recommendations

A PNF technique combining a shortening contraction of the OM and a static contraction of the TM is most effective. A minimum of one repetition, conducted twice per week is required to augment ROM. Only 20% of a maximal static contraction of the TM is required for ROM gains and should be held for at least 3 seconds. Where possible, a shortening contraction of the OM should be used to place the TM on stretch and if following a static contraction of the TM, it should be initiated immediately to make use of any inhibitory effects present within the TM.[80] When moving the TM into a position of stretch, this should be conducted at a low velocity in order to avoid an increase in TM stiffness due to muscle spindle excitation (also known as the stretch reflex)[41] and to prevent enhanced viscous resistance.[65] In light of the findings that augmented ROM occurs from stretching due to an altered stretch perception or tolerance, we recommend that the stretch component of the PNF procedure is maintained until the sensation of stretch abates. Once the desired ROM is achieved, PNF should be conducted a minimum of once per week or as required to maintain the changes.

4. Conclusion

The literature clearly supports that PNF is the most effective means to increase ROM by way of stretching, particularly in respect to short-term gains in ROM. Aside from being safe and time efficient, the dramatic gains in ROM seen in a short period of time may also promote compliance with the exercise and/or rehabilitation programme. Why PNF stretching is so effective has not been substantiated but

stretch perception or tolerance modulation seems likely. If this is the case, then the appropriateness of the term 'PNF' will be in question. Furthermore, the heterogeneity across the applied PNF stretching research has been referred to by many authors. If PNF is to be further positioned as an evidence-based practice, then a uniformed approach to the classification and implementation of PNF stretching is critical, first and foremost. Good quality clinical studies that are comprehensively detailed and combine objective methodologies within a functional context are required in this research domain.

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