

# The Theoretical Basis of Proprioceptive Neuromuscular Facilitation

DARREN G. BURKE,<sup>1</sup> CHRISTOPHER J. CULLIGAN,<sup>2</sup> AND  
LAURENCE E. HOLT<sup>3</sup>

<sup>1</sup>Department of Human Kinetics, St. Francis Xavier University, Antigonish, Nova Scotia, Canada; <sup>2</sup>Faculty of Medicine, Dalhousie University, Halifax, Nova Scotia, Canada; <sup>3</sup>School of Health and Human Performance, Dalhousie University, Halifax, Nova Scotia, Canada.

## ABSTRACT

Athletic performance, injury prevention, and rehabilitation are areas that have been positively influenced through the intervention of flexibility training programs. The literature already acknowledges that benefits realized through proprioceptive neuromuscular facilitation-type stretching are superior to other methods. Despite the widespread use of proprioceptive neuromuscular facilitation techniques, controversy still exists regarding the underlying mechanisms responsible for the increased range of motion that results when using this type of stretching. Currently, 2 hypotheses exist to account for the resulting gains, one neurophysiological and the other viscoelastic. The purpose of this review is to discuss these hypotheses and suggest a more balanced integrative paradigm.

**Key Words:** flexibility, stretching, PNF

**Reference Data:** Burke, D.G., C.J. Culligan, and L.E. Holt. The theoretical basis of proprioceptive neuromuscular facilitation. *J. Strength Cond. Res.* 14(4):496–500. 2000.

## Introduction

Two general hypotheses pertain to the physiological explanation underlying the flexibility gains that accompany proprioceptive neuromuscular facilitation (PNF) stretching. The first hypothesis focuses on neuromuscular modifications that (a) increase firing thresholds of sensory receptors, thereby allowing greater range of motion (ROM) before triggering reflex shortening (14); (b) cause additional recruitment of alpha motoneurons, creating increased force output (3); and (c) exert inhibitory neural stimuli on the stretched musculature through the activation of the opposing muscles (4).

The second hypothesis postulates that the increased ROM effects seen through PNF manipulation are because of the viscoelastic properties of the mus-

cle-tendon-fascia units. In this approach, ROM increases are said to result from alterations in the connective tissues that envelop the muscle. This process occurs as a consequence of the isometric contractions of the muscle or muscles while in a lengthened state. It is assumed that the muscle-tendon-fascia units, possessing viscoelastic properties, will permit recoverable deformations because of elastic response to stretch forces (25). Viscoelastic theorists suggest that permanent deformations can result from continued flexibility training caused by repeated repositioning of the collagenous and elastin fibers within the connective and contractile tissues (2).

Early proponents of PNF continue to support the theory that advocates neural mechanisms as the singular cause of increases in ROM (8). Recent investigations into the viscoelastic properties of the muscle-tendon-fascia unit suggest most of the ROM gains come from physical changes within the exercised musculature and associated connective tissues (13). Although most researchers recognize the necessary link between the nervous system and the muscular system, it is difficult to qualify or quantify the contribution from both the neural and viscoelastic mechanisms that create the adaptations resulting from PNF training. The purpose of this review was to discuss the current hypotheses for PNF changes and suggest a more balanced integrative paradigm to aid our understanding of this form of exercise.

## *Basic Neural Anatomy and Physiology Associated With PNF*

**Motor.** A cross section of the spinal cord depicts a central region of gray matter and a peripheral region of white matter. The gray matter is further divided into ventral, dorsal, and, in some sections, lateral horns. Motor responses are triggered by cell bodies of alpha and gamma motoneurons, which are found in the ventral horns. Alpha motoneurons innervate the extrafusal fibers of skeletal muscle in response to voluntary

or reflex stimulation. The intensity of the neural stimulation directly affects the number of muscle fibers activated and, thus, force produced. Gamma motoneurons activate intrafusal fibers located deep in the belly of skeletal muscle. The central regions of these fibers contain noncontractile components called muscle spindles. Also found in the ventral horns are Renshaw cells, which are a type of interneuron. Almost immediately after a signal leaves the alpha motoneuron, collateral branches from its axon pass to the Renshaw cells. These cells cause hyperpolarization of the alpha motoneurons, which led to their stimulation, which decreases the sensitivity of the motor cell bodies to subsequent excitatory input (26). Another type of interneuron (type Ia) sends inhibitory signals to opposing muscle or muscles, facilitating the movement of the agonist by decreasing neural activity in the antagonists (7).

*Sensory.* Sensory structures in the muscle-tendon-fascia unit include muscle spindles and Golgi tendon organs (GTO). Muscle spindles are found within the belly of skeletal muscles and are specifically located in the central portion of intrafusal fibers. They detect changes in length and rate of changes in length. The GTOs are located in the myotendinous junction of skeletal muscle, connecting in series muscle fibers and tendinous filaments. The GTOs sense tension within the muscle and the rate of change in tension.

### ***Neural PNF Theory***

Neural PNF theory is based on several neurophysiological mechanisms, including facilitation, resistance, inhibition, irradiation, successive induction, and reflexes. Whether isotonic, isokinetic, isometric, or gravity assisted, a positive relationship exists between the magnitude of the resistance and the force of the subsequent muscle activation. This is the basis for progressive resistance training and is fundamental to PNF (14). Resistance to muscular actions cause an increased inflow of electrical activity from the muscle spindles in the agonists and will reflexively facilitate agonist activation while inhibiting the antagonistic motoneurons (11). As well, the resultant central nervous system excitation immediately following either a concentric or isometric agonistic contraction is reported to produce increased antagonistic muscular output, both concentrically and isometrically (8). This enhanced muscular output is referred to as successive induction.

Irradiation is the spreading of excitement in the central nervous system that causes activation of synergist muscles in a specific pattern (9). Knott and Voss (15) defined irradiation as the spreading of muscle activity from 1 body part to another to support a desired movement. They concluded that maximal resistance provides the means for securing this overflow or irradiation from more adequate to less adequate patterns of movement.

The autogenic inhibition reflex causes the relaxation of an activated muscle group through the response of the afferent fibers of the GTO. Afferent signals from the GTO inhibit the alpha motoneurons that innervate the muscle being activated. The GTOs can also override excitatory impulses from the muscle spindles (1), which alters the manner in which the muscle spindles respond to the stretching condition by decreasing afferent flow of impulses from their proprioceptors, in an attempt to avoid damage to the muscle-tendon-fascia unit (9). GTOs respond to high tension, and this is the basis for Kabat's (14) recommendation for maximal intensity of muscle activation during PNF training: to ensure autogenic inhibition.

Thus, PNF is possibly a result of autogenic inhibition, modifications of the muscle spindles, reciprocal innervation, or facilitated muscle activations via successive induction.

### ***Basic Structural Anatomy of the Muscle-Tendon-Fascia Unit***

*Muscle.* Muscle tissue can be divided into several different levels of organization. The entire muscle is made of bundles called fascicles. These bundles are made of muscle fibers in parallel or in series. Muscle fibers or cells are composed of myofibrils in parallel, which are made of sarcomeres connected in series throughout the length of the muscle fiber. Sarcomeres are composed of myofilaments, thick filaments called myosin, and thin filaments called actin. Actin and myosin are organized in an interdigitating manner to form the functional contractile unit of skeletal muscle.

*Tendon.* Tendons are dense fibrous connective tissue, composed almost entirely of tightly packed, parallel, collagen fibers. Little or no stretch occurs in these structures, as they are constructed to withstand great tensile forces. The collagen found in a tendon has several levels of organization similar to the divisions within muscle—for example, bundles, fibers, and microfibrils.

*Fascia.* Fascia is found within and around the muscle and exists in several layers of connective tissue sheaths. The sheaths are called the epimysium, perimysium, and endomysium. The epimysium surrounds the entire muscle, the perimysium surrounds muscle bundles, and the endomysium surrounds individual muscle fibers. This tissue is called dense connective tissue because of the high collagen content. However, some elastic tissue is found in these structures, especially in the endomysium (22). In addition, the collagen in the endomysium is structured differently than that found in tendons. Its arrangement is irregular, and it is called reticular fiber, not collagen. Under tensile loads reticular fibers allow more stretch than do collagen fibers (2).

Considering the many different levels of tissue organization, it would seem plausible that even the

slightest deformation realized at each of these levels or layers could yield additive effects, allowing an overall increase in length.

### ***Viscoelastic Properties of the Muscle-Tendon-Fascia Unit***

A recent move has occurred away from the singular view that neural manipulation alone accounts for the ROM changes that result from PNF training. The alternative explanation is dependent on the viscoelastic properties of the muscle-tendon-fascia unit. Elasticity implies that length changes are directly proportional to the applied forces or loads. Viscous properties are characterized as time dependent and rate change dependent, where the rate of deformation is directly related to the applied force. Certain properties are characteristic of viscoelastic materials. If a viscoelastic material is stretched and then held at a constant length, the stress or force at that length gradually declines. It is viscous because the tension decreases with time and elastic because the material maintains some degree of tension (5). This is referred to as stress relaxation. Creep is the term used to describe the changes that occur when a fixed load is applied to a viscoelastic material. When this process occurs, the material will experience continual deformation until it reaches a new length based on the applied load (25). Hysteresis is the variation between the load-deformation relationship of loading and unloading (16). Another property of viscoelastic materials is strain-rate dependence. Strain-rate-dependent materials exhibit higher tensile strength at faster strain rates; therefore, the final length of a material being stretched is related to the speed at which it is elongated (5).

Taylor et al. (25) studied these properties on rabbit extensor digitorum longus and tibialis anterior muscle-tendon-fascia units under physiological conditions by leaving the neurovascular attachments intact. The study was designed to simulate widely used stretching techniques, including PNF. The conclusion was that muscles respond to stretch by viscoelastic properties alone, exclusive of reflex effects. This statement was based on the fact that denervated muscles responded similarly to the innervated muscle for all parameters evaluated, and that no significant force contributions were observed from a stretch reflex.

McHugh et al. (17) demonstrated viscoelastic stress relaxation independent of detectable muscle activity using electromyographic analysis. A straight leg raise was performed from the supine position to 5° less than the point where maximal electromyographic activity was previously noted. The stretch was held over 45 seconds with a significant decrease in force and no observed change in muscle activity.

### ***Interaction of Collagen and Elastin***

Collagenous tissue is regarded as nonextendible (2), in that individual fibrils can only stretch to 3% of their

original length without permanent deformation. Surburg (23) implied that when a relaxed muscle is stretched, resistance to the stretch is derived from the extensive connective tissue framework and sheathing within and around a muscle, and not the myofibril elements. Surburg highlighted the importance of connective tissue to stretch, but surprisingly, he ignored elastin when discussing the apparent viscoelastic properties displayed by muscle-tendon-fascia units. Instead, effects were assumed to be caused primarily by collagen. Elastin's low number of cross-links, compared with collagen's high degree of cross-linkage, allow it to stretch up to 150% of its original length before permanent deformation. Although elastin is almost always found in close association with collagenous tissue, its nonpolar hydrophobic heads ensure the relationship is not a fixed one with the polar hydrophilic collagen fibers, but a viscous one. Elastin is found in all sheaths, including the epimysium, perimysium, and endomysium (22). It is within these sheaths that the semiplastic changes seen with flexibility training, such as ballistic, static, or PNF exercises, may be found (12).

Because collagen is a tightly cross-linked structure and relatively nonextendible, tensile forces are unlikely to cause a significant transient change in its molecular structure and interfiber cross-linkage relationship. Elastin, which is not so extensively cross-linked, seems like a more probable molecule for potential modification of a molecular cross-linkage structure. Thus, herein could lie the elasticity inherent to any muscle-tendon-fascia unit, not neglecting collagen's small contribution to elongation and large contribution to resistance to stretch, through its cross-linkage to elastin fibers. The cross-links could be staggered in a manner that best suits the normal flexibility needs with ROM regularly used. When a muscle-tendon-fascia unit is repeatedly used beyond the range of regular motion, the cross-linkages between elastin molecules and between elastin and collagen molecules are broken and reformed at a different mean spacing, allowing an increased elongation before tautness occurs. Therefore, the transient short- and long-term changes in flexibility could be caused by the semipermanent elastin cross-linkages and elastin-collagen linkages that are constantly broken and reformed with the natural degradation and assimilation of new molecules.

### ***An Integrative Paradigm of the Mechanisms Associated With PNF***

Most PNF protocols start with a muscle or muscle group in a maximum actively (not ballistically) stretched position. Restriction of this movement is attributed to the muscle tissue length itself, neural reflex mechanisms, and the fascia sheathing within and around the many levels of organized structures associated with the muscles involved. When the muscle or



muscle group is at its maximum active attainable length, a voluntary motor impulse to stimulate muscle activation is initiated against an immovable object. After a gradual increase, the force is sustained for a period of time (about 6 seconds), with the intensity remaining constant at a predetermined level. The tension created within the muscle-tendon-fascia unit of the contracting muscle causes an adjustment of the muscle spindle sensitivity and prolonged stress on the length restrictive aspects of this unit. Connective tissue accommodates the stress created by myosin cross-bridge attachments to actin during the isometric contraction, maintaining the overall length of the stretched muscles. These physical components are believed to respond viscoelastically and elongate. This increased length and the neural adaptations allow greater elongation before triggering a reflex response, promoting increased overall length (5). The above-mentioned process is one sequence of contractions, isometric for the lengthened muscle, concentric for its antagonist. This sequence is immediately followed by 3 or 4 additional sequences from the new and lengthened positions. If similar training conditions persist or intensify, elongation will be maintained or gradually increased (20). However, if treatment stops, tissue length returns to that of ranges encountered through regular activity and subsequent joint motions (6).

## Discussion

A logical assumption is that PNF techniques are superior to static and ballistic training because of the combined assistance of neural and viscoelastic modifications. Despite being brief, neural manipulations are a factor, especially in those techniques incorporating agonist-antagonist activation sequences. In static and ballistic training, proprioceptors will be stimulated by an increase in tensile forces within the muscle-tendon-fascia unit and respond with reflex contraction. In these 2 techniques, muscle fibers will not relax at the lengthened position. However, with PNF, voluntary motor impulses sent to the length sensitive receptors cause them to adapt and decrease afferent signals of stretch, which permits further elongation. Moore and Kukulka (19), in a study testing depression of Hoffman-reflex amplitudes with PNF exercises, found that a phase of relaxation followed voluntary activation.

The neural adjustments occurring with PNF (successive induction, reciprocal inhibition, etc.) are not the sole reason for increases in ROM; however, they allow the physical structures of the muscle-tendon-fascia units to experience viscoelastic changes. By decreasing neural restrictions, the physical structures can experience greater flux in cross-linkage formation, supporting the semiplastic modification of elastin-collagen cross-linkages and elastin-elastin cross-linkages.

## Practical Applications

It is important to recognize that both neural and viscoelastic adaptations contribute to the gains experienced during PNF training. The intensity of muscular activation necessary to create neural and viscoelastic modifications that lead to greater elongation are still poorly understood. However, preliminary unpublished research at Dalhousie University seems to indicate that a lower effort yields significant flexibility increases and that progressive increases in intensity not only equal the results of higher intensity, but also provide a safer approach to the protocol. Coaches, athletes, and therapists should sustain muscle activations longer than 6 seconds to ensure neural accommodation for subsequent physical changes in the viscoelastic structures within the muscle-tendon-fascia units.

## References

1. ALTER, M.J. *Science of Stretching*. Champaign, IL: Human Kinetics, 1988.
2. BORYSENKO, M., AND T. BERINGER. *Functional Histology*. Boston: Little, Brown, and Company, 1989.
3. CAVAGNA, G., B. DUSMAN, AND R. MARGARIA. Positive work done by a previously stretched muscle. *J. Appl. Physiol.* 24:21-32. 1986.
4. ETNYRE, B.R., AND L.D. ABRAHAM. Antagonist muscle activity during stretching: A paradox re-assessed. *Med. Sci. Sports Exerc.* 20:285-289. 1988.
5. FUNG, Y.C. Elasticity of soft tissues in simple elongation. *Am. J. Physiol.* 213:1532-1544. 1967.
6. GORDON, G.A. P.N.F.: Proprioceptive neuromuscular facilitation: 'The super stretch.' *Nat. Strength Cond. Assoc. J.* April-May: 26-28. 1982.
7. GUYTON, A. *Basic Neuroscience*. Philadelphia: W.B. Saunders, 1987.
8. HELLEBRANDT, F., S. HOUTZ, M. PARTRIDGE, AND C. WALTERS. Tonic neck reflexes in normal human subjects. *Am. J. Phys. Med.* 4:144-156. 1956.
9. HOLT, L. *Scientific Stretching for Sport (3S)*. Dalhousie University, Halifax, Nova Scotia: Sports Research Limited, 1974.
10. HOLT, L., H. KAPLAN, T. OKITA, AND M. HOSHIKO. The influence of antagonistic contraction and head position on the responses of agonistic muscles. *Arch. Phys. Med. Rehabil.* 50:336-345. 1969.
11. HOLT, L., T. TRAVIS, AND T. OKITA. Comparative study of three stretching techniques. *Perceptual Motor Skills* 31:611-616. 1970.
12. HUBLEY-KOZEY, C.L., AND W.D. STANISH. Can stretching prevent athletic injuries? *J. Musculoskeletal Med.* 7(3).
13. JANSSEN, K., L. EICHINGER, P. JANMEY, A. NOEGEL, M. SCHLIWA, W. WITKE, AND M. SCHLEICHER. Viscoelastic properties of F-actin solutions in the presence of normal and mutated actin-binding proteins. *Arch. Biochem. Biophys.* 325:183-189. 1996.
14. KABAT, H. Studies of neuromuscular dysfunction XV: The role of central facilitation in restoration of motor function in paralysis. *Arch. Phys. Med.* 9:667-672. 1952.
15. KNOTT, M., AND D. VOSS *Proprioceptive Neuromuscular Facilitation*. New York: Hoeber Medical Division, Harper and Row, 1956.
16. MCHUGH, M.P., S.P. MAGNUSON, G.W. GLEIM, AND J.A. NICHOLAS. Differentiation of viscoelastic and contractile responses to stretch of human skeletal muscle in vivo. In: *Combined Meeting of the Orthopaedic Research Societies of USA, Japan, and Canada*. Banff, Alberta, Oct. 21-23, 1991.

17. McHUGH, M.P., S.P. MAGNUSSON, G.W. GLEIM, AND J.A. NICHOLAS. Viscoelastic stress relaxation in human skeletal muscle. *Med. Sci. Sports Exerc.* 24:1375-1382. 1992.
18. MOORE, M., AND R. HUTTON. Electromyographic investigation of muscle stretching techniques. *Med. Sci. Sports Exerc.* 12:322-329. 1980.
19. MOORE, M., AND C. KUKULKA. Depression of the Hoffman reflexes following voluntary contraction and implications for proprioceptive neuromuscular facilitation therapy. *Phys. Therapy* 71:321-333. 1991.
20. NELSON, K.C., AND W.L. CORNELIUS. The relationship between isometric contractions, durations, and improvement in shoulder joint range of motion. *J. Sports Med. Phys. Fitness* 31:385-388. 1991.
21. OSTERNIG, L., R. ROBERTSON, R. TROXEL, AND P. HANSEN. Differential responses to proprioceptive neuromuscular facilitation (PNF) stretch techniques. *Med. Sci. Sports Exerc.* 22:106-111. 1990.
22. SNELL, R. *Clinical and Functional Histology for Medical Students*. Boston: Little, Brown and Company, 1984.
23. SURBURG, P. New perspectives for developing range of motion and flexibility for special populations. *Adapted Phys. Activity Quart.* 3:227-235. 1986.
24. TANIGAWA, M.C. Comparison of hold-relax procedure and passive mobilisation on increasing muscle length. *Phys. Therapy* 57:725-735. 1972.
25. TAYLOR, D., J. DALTON, A. SEABER, AND W. GARRETT. Viscoelastic properties of muscle-tendon units: the biomechanical effects of stretching. *Am. J. Sports Med.* 18:300-309. 1990.
26. THOMSON, D., AND A. CHAPMAN. The mechanical response of active human muscle during and after stretch. *Eur. J. Appl. Physiol.* 57:691-697. 1988.