The Passive Mechanical Properties of Muscle and Their Adaptations to Altered Patterns of Use

The length and stiffness of a relaxed muscle are determined by the mechanical properties of its intramuscular connective tissue and/or intracellular structures. Viscous deformation of these components of muscle is responsible for the increase in muscle length seen immediately after stretching, but this increase is transient. Lasting changes in muscle length can only be brought about by adaptations of the structure of muscle. An understanding of the nature of the stimulus for muscle to adapt can provide therapists with a theoretical basis for therapeutic intervention aimed at producing changes in muscle length.

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Changes in the length and extensibility of muscles are a major cause of movement dysfunction. These changes occur following immobilization, such as the immobilization imposed by plasters or the effective immobilization that may result from weakness, loss of control of muscles, or pain. Changes in muscle length are also said to occur in people who habitually use their muscles in a shortened or lengthened range.

This paper focuses on two questions. They are (i) what are the anatomical structures which determine the length and stiffness of relaxed muscles; and (ii) what changes occur in these structures when a muscle's length and stiffness change? The paper will present therapists with a theoretical basis from which clinical strategies for the management of muscle length problems can be derived. An important conclusion will be that lasting changes in muscle length can only result from adaptations of the structure of muscle and that therefore intervention should aim to manipulate the mechanical environment of the muscle in order to stimulate or maintain appropriate adaptive processes.

The paper will be limited to the response of adult skeletal muscle length and stiffness to imposed changes in its mechanical environment, and will not deal with changes that result from disease, trauma or congenital conditions, or that are purely a result of ageing. Only changes in the mechanical properties of relaxed muscle will be discussed. The reader is referred to the work of Gossmann *et al* (1982) for a broader review of length associated changes in muscle, including changes in active length-tension properties.

The first part of this paper reviews some of the mechanical properties of relaxed muscles. It includes a brief discussion of the anatomical components that give muscle those properties. The second part of the paper reviews studies on the adaptations of muscle length and adaptations of the structure of muscle that may be responsible for changes in muscle length. Finally some broad implications are drawn for the therapeutic management of adaptive muscle shortening.

Mechanical Properties of Muscle

When an isolated muscle is stretched in the laboratory it is possible to measure the change in muscle length and, indirectly, the tension that develops in the muscle. The results can be plotted on a passive length-tension curve (Banus and Zetlin 1938, Ralston *et al* 1947, Hanson and Lowy 1960, Butler and Little 1976). A typical passive length-tension curve is shown in Figure 1. The passive length-tension curve shows the length at which the relaxed muscle first develops internal tension, designated L_i in Figure 1. This is often called the muscle's resting length, or sometimes simply its length.

Another important mechanical property of muscle is its stiffness. Stiffness is the ratio of the tension developed in the muscle when it is stretched to the amount the muscle lengthens. Thus the slope of the passive length-tension curve represents the muscle's stiffness. The stiffness of the muscle determines how much the muscle will lengthen given a certain change in tension and how much tension will develop in the muscle as it is lengthened.

In some studies the measurements of tension, change in length and stiffness are normalised so that the resulting values reflect only the properties of the muscle tissue and not the size of the muscle. In this case the tension is divided by the cross sectional area of the muscle (called stress) and the change in length is divided by the initial length of the muscle (called strain). The material stiffness (or modulus of elasticity) is the slope of the stress-strain curve.

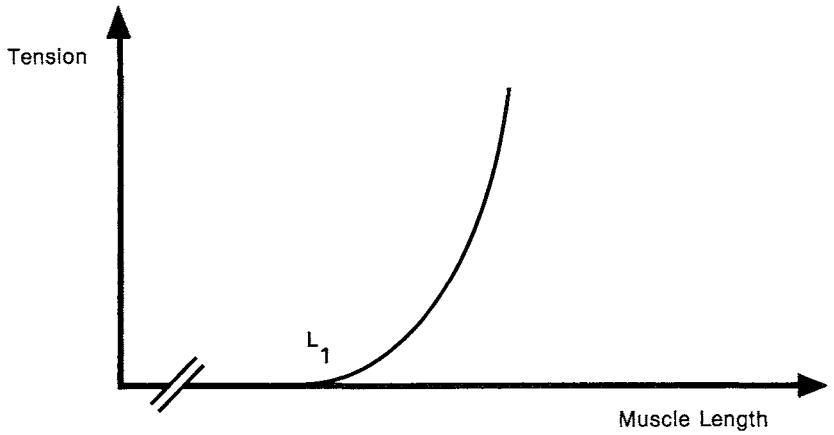


Figure 1: A length-tension curve for muscle. L_1 is the length at which the muscle first develops tension.

The mechanical properties of relaxed muscle may be provided by several of the anatomical structures that make up muscle. Furthermore, it is possible that the structures which provide the properties of relaxed muscle and those which provide its properties when it is actively contracting, are not anatomically distinct. Some structures may contribute to the properties of both relaxed and actively contracting muscle under different conditions of contraction and stretch.

A widely held view is that the intramuscular connective tissue is responsible for the mechanical properties of relaxed muscle. The intramuscular connective tissue is organised in three levels: epimysium, which surrounds the whole muscle; perimysium, which surrounds individual fascicles; and endomysium which surrounds individual muscle fibres (Borg and Caulfield 1980, Rowe 1981). Under an electron microscope the collagen fibres of the perimysium appear crimped (Rowe 1974, Borg and Caulfield 1980). When the muscle is lengthened these fibres become more longitudinally orientated and lose their crimp (Rowe 1974, Williams and Goldspink 1984). As the collagen uncrimps it becomes stiffer (Viidik 1973, Betsch and Baer 1980, Kastelic *et al* 1980). This increase in stiffness of longitudinally orientated collagen fibres suggests a potential load bearing role of the intramuscular connective tissue in relaxed muscle. Also, a study by Kovanen *et al* (1984a) has shown that changing the structure of collagen in rats results in a decreased material stiffness of the soleus muscle. This suggests that collagen, which exists in the intramuscular connective tissue and in the sarcolemma (Mayne and Sanderson 1985) contributes to the stiffness of rat soleus. To summarize, some rather indirect evidence suggests that intramuscular connective tissue is responsible for the mechanical properties of relaxed muscle.

An alternative theory is that the muscle cells, or parts of the muscle cells, determine the mechanical properties of relaxed muscle. Support for this theory comes from experiments which demonstrate that the isolated muscle cell, stripped of the connective tissue which normally surrounds it, still demonstrates significant stiffness at physiological lengths (Fish *et al* 1984, Magid and Law 1985). Many sources of muscle cell stiffness have been proposed and investigated. These include the sarcolemma (Ramsey and Street 1940, Sichel 1941, Casella 1950, Podolsky 1964, Fields and Faber 1970, Rapoport 1972, 1973, Schmalbruch 1974, Caceci *et al* 1981), hypothetical 'S filaments' (Hanson and Huxley 1955), an interaction between myofilaments (Hill 1968, Moss and Halpern 1977, Magid and Law 1985), intracellular proteins such as connectin (Maruyama *et al* 1977), and the sarcoplasmic reticulum (Herbst and Piontek 1974, 1975). The relative contribution of these structures to the stiffness of whole muscles has not been determined. However Magid and Law (1985) have observed that whole frog semitendinosis muscle has a material stiffness that is similar both to that of an isolated frog semitendinosis muscle cell and to an isolated frog semitendinosis muscle cell after its sarcolemma has been removed. This provides compelling evidence that the major source of the mechanical properties of relaxed muscle resides within the muscle cell, at least for the frog semitendinosis.

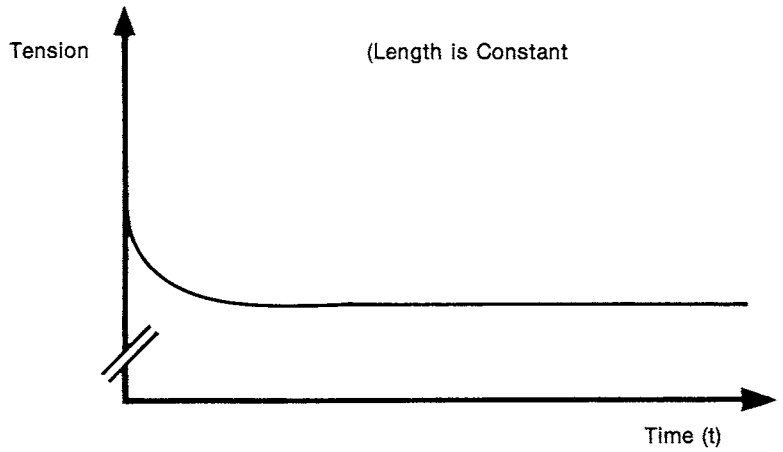
In summary then, the components of muscle that are most likely to determine its length and stiffness are the intramuscular connective tissue and intracellular structures. Their relative contribution in different muscles is yet to be determined.

Muscle length is dependent not only on the mechanical properties of the muscle belly, but also on the mechanical properties of tendon with which the muscle is connected in series. The mechanical behaviour of tendon has been extensively documented in animal models (Viidik 1966, Viidik 1973, Betsch and Baer 1980, Hooley *et al* 1980, Proske and Morgan 1987) and in humans (Walker *et al* 1964, Van Brocklin and Ellis 1965, Benedict *et al* 1968, Schwerdt *et al* 1980, Lamontagne *et al* 1986, Goldstein *et al* 1987). It is clear from these studies that, even at 'physiological' loads, the tendon is still many times stiffer than relaxed muscle (Stolov and Weilep 1966). This means that when the muscle is stretched, most of the increase in length comes from an increase in the length of the muscle belly, not from the relatively inextensible tendon.

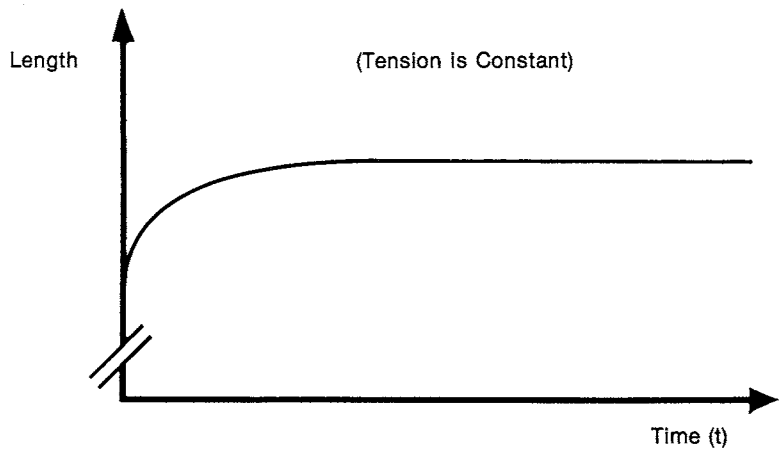
The preceding discussion focussed on the length-tension properties of muscle. The tension in the muscle was consid-

ered to be a simple function of its length, that is, the muscle was assumed to behave 'elastically'. The mechanical behaviour of a muscle that is stretched is, however, more complex than the length-tension curve might suggest. In reality the relaxed muscle also demonstrates time dependent or 'viscous' behaviour under load (Blix 1893 cited in Banus and Zetlin 1938, McCarter *et al* 1971, Butler and Little 1976, Bohannen 1984, Magid and Law 1985). The muscle is said to behave as a 'viscoelastic' material. This means that the relationship between length and tension changes with time if a stretch is maintained. For example, if the muscle is stretched to a given length and that length is maintained, then the tension will be seen to decrease over time until a new steady state force is reached (called stress relaxation, see Figure 2A). Alternatively if the muscle is stretched to a certain tension and the tension is maintained, then the length of the muscle will increase over time until a new steady state length is reached (called creep, Figure 2B). If the muscle is repeatedly stretched each subsequent stretch will demonstrate different length-tension curves (Figure 2C). This viscous deformation is probably the major source of the increases in muscle length seen immediately following muscle stretching.

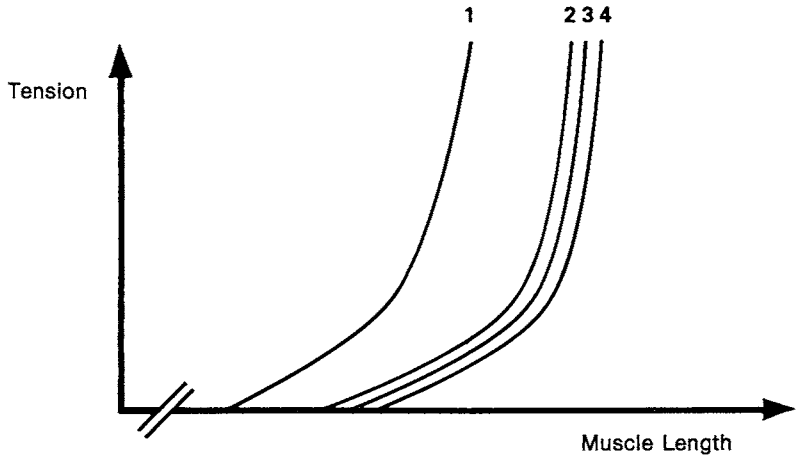
Most of the viscous deformation that is responsible for the increases in muscle length following stretching is lost over time. For example, McCarter *et al* (1971) examined the reversibility of the changes of the length-tension relationship that occurred after six stretch-relaxation cycles of rat soleus muscles through a range slightly greater than the 'physiological' range of the muscle. They found that after a recovery time of about two hours the muscle had returned to its pre-stretch length. If, following stretching, the muscle was electrically stimulated to tetany for three seconds and allowed to shorten actively the length recovery occurred more quickly. In other words, concentric contractions of the muscle ap-



A. Stress relaxation. The muscle is stretched to a constant length (at t = o). The tension in the muscle decreases over time to a new value.



B. Creep. The muscle is stretched to a constant tension (at t = o). The length of the muscle increases over time to a new value.



C. The effect of cyclic loading. Numbers signify order of cycles. Note that the length-tension relationship changes with successive cycles (adapted from McCarter *et al* 1971, with permission).

Figure 2: Viscoelasticity in muscle.

peared to hasten the return to its original length. In all cases the passive length-tension curve of the muscle was observed to return towards its pre-stretch position.

Experiments with humans also demonstrate that the increase in muscle length observed following stretching is gradually lost. For example, a study by Bohannen (1984) showed that the gains in straight leg raising angle (probably indicative of hamstrings length and stiffness) seen immediately after an eight minute stretch were significantly decreased after ten minutes and 'largely lost' after twenty-four hours.

It appears then, that the increases in muscle length seen soon after stretching are largely, if not entirely, a transient phenomena. Presumably this also applies to length gains seen after stretching adaptively shortened muscle, although there is no direct evidence of this. It may be that these transient increases of muscle length have therapeutic value when muscle length needs to be increased for only short periods of time, such as the duration of a sporting activity. However, therapists often need to effect lasting increases in muscle length. Lasting changes in muscle length will probably only result from adaptive remodelling of the structure of muscle, and not simply from mechanical deformation. This has important implications for clinical measurement. The measurement of increases in muscle length made soon after stretching, so often reported in the literature (Tanigawa 1972, Warren *et al* 1976, Medeiros *et al* 1977, Sady *et al* 1982, Wessling *et al* 1987, Madding *et al* 1987) and by clinicians, are probably not valid as a measure of a technique's ability to effect sustained muscle length increases.

Adaptations of Muscle Length

The preceding section argues that mechanical deformation alone is unlikely to produce lasting changes in muscle length, and that lasting changes in muscle length must be mediated by adaptations of the structure of muscle. Changes in muscle length could result from adaptations of any of the structures which determine the relaxed muscle's mechanical properties. This next section reviews the changes in muscle length and stiffness and the associated changes in muscle structure that have been observed using various experimental models.

Imposed Length Changes

The most commonly used method of experimentally inducing adaptive changes in the length and stiffness of muscles involves making a muscle work predominantly in a shortened or lengthened range. One model involves immobilizing an animal's joint with a plaster so that the muscle to be investigated is held either in a shortened or lengthened position. When the limb is immobilized the animal still actively contracts its muscles under the plaster (Fournier *et al* 1983). After a period of immobilization that is long enough for adaptations of the muscle's structure to occur (days or weeks) the mechanical properties and structure of the muscle are investigated.

When the muscle is immobilized in a shortened position the length-tension curve of that muscle shifts to the left and becomes steeper (*ie* the muscle becomes shorter and stiffer; Tabary *et al* 1972, Williams and Goldspink 1978, Goldspink and Williams 1978, Witzmann *et al* 1982, Tardieu *et al* 1982c, Figure 3). The increase in the stiffness of the muscle can probably be explained entirely by its decrease in length (Witzmann *et al* 1982, but see also Goldspink and Williams 1978). In this respect the adaptively shortened muscle can be likened to a short rubber band, which is stiffer than a long rubber band simply because there is less rubber in series to be stretched out.

It appears that the stimulus for the adaptation of muscle length and stiffness results from the imposed length change rather than from movement deprivation. For example, if a muscle is immobilized in a neutral position, that is, if it is deprived of movement without an imposed length change, then its length and stiffness do not change (Tabary *et al* 1972). Therefore, the development of short muscles differs from the development of stiffness in periarticular connective tissues which appears to be directly related to movement deprivation (Akeson *et al* 1974, Akeson *et al* 1980, Akeson *et al* 1987).

The changes in length and stiffness that occur when a muscle is immobilized in a shortened position are well documented for slow twitch muscles

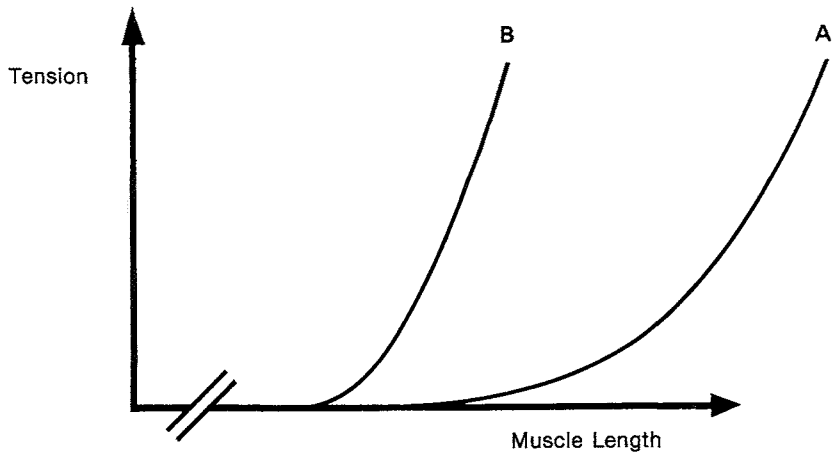


Figure 3: Length-tension curves for (A) mouse soleus and (B) mouse soleus immobilized in a shortened position for three weeks (adapted from Williams and Goldspink 1978, with permission).

such as cat, mouse and rat soleus, but not for other muscles. In one of the few investigations of length changes in immoblized fast twitch muscles, Witzmann *et al* (1982) found a decrease in length and increase in stiffness of rat extensor digitorum longus and superficial vastus lateralis after a long perid (six weeks) of immobilization in the shortened position, but this decrease was not statistically significant. Such a slow response is probably related to the relatively low basal rates of protein synthesis and breakdown in fast twitch muscles (Goldspink and Goldspink 1986).

Several studies have investigated the effect of immobilizing muscle in a lengthened position. Interestingly, it appears that the length-tension curve does not change (Tabary *et al* 1972, Goldspink *et al* 1974, Tardieu *et al* 1977, Williams and Goldspink 1978). Contrary to these findings are those of Tardieu *et al* (1982c), who have shown an increase in muscle length and stiffness using a similar experimental model. The reason for this difference is unclear.

Immobilization of muscle in a shortened or lengthened position also produces changes in the number of sarcomeres in series in the muscle. Thus when a muscle is immobilized in a shortened position it may lose up to 40% of its sarcomeres (Tabary *et al* 1972, Williams and Goldspink 1973, Goldspink *et al* 1974, Williams and Goldspink 1978, Hayat *et al* 1978, Huet de la Tour *et al* 1979a, Witzmann *et al* 1982). Witzmann *et al* (1982) found that the decrease in sarcomere number was proportional to the decrease in muscle length. If the muscle is immobilized in a lengthened position it may produce as much as 25% more sarcomeres in series (Goldspink *et al* 1974, Tabary *et al* 1972; Williams and Goldspink 1973, Tardieu *et al* 1977, Williams and Goldspink 1978) but, as already mentioned, this is probably not associated with a change in its length or stiffness. Increases in sarcomere number have also been observed in an experiment in which muscle was continually stretched but not immobilized (Holly *et al* 1980).

It appears that regulation of sarcomere number occurs in order to preserve an optimal myofilament overlap (see *eg* Williams and Goldspink 1978, Herring *et al* 1984), but the nature of the control mechanism remains obscure. Some experiments suggest sarcomere number is not regulated by neural factors, because models of denervation plus immobilization produce similar adaptations of sarcomere number to immobilization alone (Goldspink *et al* 1974); the only differences in sarcomere number being attributable to the decrease in sarcomere length that results from active shortening of innervated muscles (Hayat *et al* 1978). However, McLachlan and Chua (1983) have shown that innervated tenotomised rat soleus loses sarcomeres more quickly than denervated tenotomised rat soleus. Other authors have shown that chronic active muscle shortening induced by electrical stimulation (Tabary *et al* 1981) or tetanus toxin (Huet de la Tour *et al* 1979b) accelerates sarcomere loss in shortened immobilized muscle. These studies provide the best model currently available of the rapid length changes seen in spastic muscles.

The adaptations of muscle length and sarcomere number appear to be very reversible. When the limb is removed from its plaster cast the muscle quickly regains its original length-tension relationship and sarcomere number (Tabary *et al* 1972, Goldspink *et al* 1974). It is interesting that the attempted return to normal function alone provides an adequate stimulus for recovery of muscle length. The adaptively shortened muscle will also return to its original length and stiffness if it is immobilized in a lengthened or intermediate position.

Immobilization of muscles in a shortened position also produces changes in the intramuscular connective tissue, although these changes have been investigated less thoroughly than the changes in sarcomere number. Goldspink and Williams (1978) and Williams and Goldspink (1984) measured sarcomere number, muscle cross sectional area, collagen fibre diameter and hydroxyproline concentration (hydroxyproline is exclusively an amino acid of collagen) in mouse soleus immobilized in both the shortened and lengthened positions. Their results showed an increase in hydroxyproline concentration and an increase in the ratio of connective tissue cross sectional area to muscle fibre cross sectional area following immobilization in the shortened position. They also found that the muscle did not atrophy in the first few days of immobilization. This must mean that the total amount of connective tissue increased, at least transiently. In addition, Williams and Goldspink (1984) were able to demonstrate that the intramuscular connective tissue became more longitudinally orientated at any given length following immobilization in the shortened position. No changes in intramuscular connective tissue were found in muscles immobilized in a lengthened position. Other authors have reported unquantified observations of intramuscular connective tissue proliferation with immobilization in both shortened and neutral positions (Cooper 1972, Tomanek and Lund 1974, Gossman *et al* 1986).

Changes in the mechanical properties of tendon have generally been considered to be less significant than muscle belly changes in the development of length changes in adult muscle. For example, it has been shown that rabbit and cat soleus tendon does not change its length when the muscle is immobilized in the lengthened or shortened position (Tardieu *et al* 1977, Tardieu *et al* 1982c). A clinical study by Halar *et al* (1978) produced similar findings. They showed that the extramuscular portion of the tendo-achilles was the same length in stroke patients with clinically evident plantarflexor shortening as in a control group. More recently Tardieu *et al* (1983) induced adaptive shortening of both the tendon and the

muscle belly of adult rat soleus by surgically shortening the tibia. Changes in the length of tendon accounted for more than seventy-five percent of the total muscle length adaptations with this model. Further clinical studies may clarify whether adaptations of tendon length commonly contribute to muscle length problems seen clinically.

The effect of a change in tendon length on the mechanical properties of the whole muscle can be predicted. A decrease in tendon length will decrease the length of the whole muscle without changing its stiffness. An increase in tendon length will increase the length of the muscle, again without changing it stiffness (Tardieu *et al* 1982a, Tardieu *et al* 1982b, Figure 4). The decrease in tendon stiffness that occurs with immobilization (Woo *et al* 1982) is unlikely to alter the stiffness of the whole muscle because the tendon remains many times stiffer than the muscle belly.

To summarize the main points of this section: imposed length changes have been shown to alter the mechanical properties and structure of muscle. Immobilization in a shortened position produces short, stiff muscles with fewer sarcomeres and an increase in connective tissue. The connective tissue also becomes more longitudinally orientated. The length-tension properties and sarcomere number return to normal values with remobilization or with plaster immobilization in a neutral or lengthened position. The immobilization of normal muscle in a neutral or lengthened position probably does not change the length or stiffness of the muscle, but it can increase the number of sarcomeres. Most evidence suggests that adaptations of tendon are unimportant in adaptations of adult muscle length.

Training

A series of studies from one laboratory have investigated the effects of training on muscle stiffness and intramuscular collagen (Suominen *et al* 1977, Kovanen *et al* 1980, Kovanen *et al* 1984a, Kovanen *et al* 1984b). This paradigm is of interest because it shows changes in muscle length properties presumably without imposing a condition in which the muscle is required to work at other than optimal lengths. In one of these studies the mechanical properties and hydroxyproline concentrations of rat soleus and rectus femoris were measured before and after four weeks of endurance training on a treadmill (Kovanen *et al* 1984a). The material stiffness of the slow twitch soleus increased significantly with training, although there was no change in hydroxyproline concentration and no change in muscle length. There was no change in the length, stiffness, or hydroxyproline concentration of the fast twitch rectus femoris. Other studies (Suominen *et al* 1977, Kovanen *et al* 1980) have shown that endurance training increases prolyl-hydroxylase activity (which is an enzyme involved in collagen synthesis), but not the amount of collagen, in both a group of sixty-nine year old humans and in rats. The authors suggest that the increase in muscle stiffness with training is a result of increased collagen cross linking but not increased amounts of collagen. However, it is also possible that the increase is stiffness found by these authors was due to changes in structures other than the connective tissue.

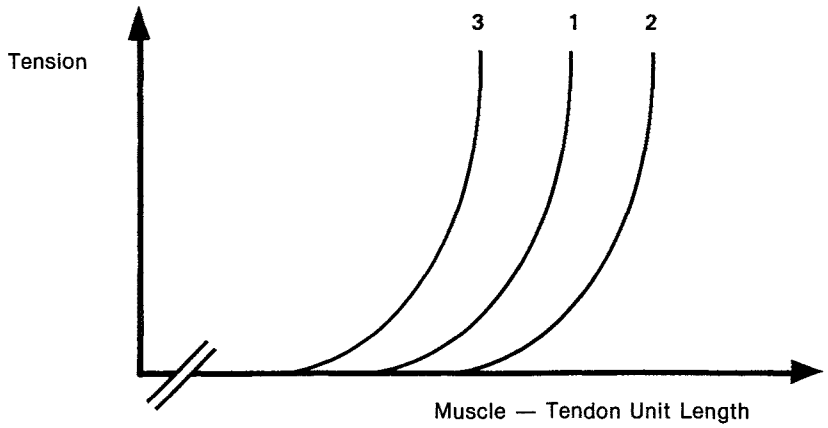


Figure 4: Curve 1: A length-tension curve for muscle. Curves 2 and 3 show the effect on the length-tension curve of increasing and decreasing the length of the tendon respectively.

Increases in the synthesis (Jablecki *et al* 1973, Turto *et al*, 1974), amount and proportion (Williams and Goldspink 1981) of connective tissue in muscle have also been reported to accompany, and even precede, work induced hypertrophy following tenotomy of the synergists of rat plantaris and soleus. As these studies were not accompanied by passive length-tension data the interesting possibility that the increases in intramuscular connective tissue produced stiff muscles remains speculative.

Alnaqeeb, Al Zaid and Goldspink (1984) and Goldspink and Alnaqeeb (1985) have shown an increase in the stiffness of and amount of connective tissue in the muscles of elderly and apparently sedentary rats. Aged rats probably provide a model of relative disuse of muscle because these changes are reversible with exercise (Goldspink *et al* 1988). Why it is that elderly, sedentary rat's muscles become less stiff with exercise, yet younger rat's muscles become stiffer, is unclear.

Concluding Remarks

Information on the manner in which muscle adapts its length and stiffness in response to changes in its mechanical

environment can help therapists design rational, effective and efficient intervention strategies for dealing with muscle length problems. It is clear from the preceding discussion that short muscles are different in structure and composition from muscles of normal length. The task of the therapist is to provide a mechanical environment which stimulates appropriate adaptive changes in the muscle's structure and composition so that its normal mechanical properties are restored. In the short term this may involve plaster immobilization in a neutral or lengthened position or structuring the patients practice of normal function. Evidence from animal studies suggests that these strategies may be effective. It is also possible, but perhaps unlikely, that the commonly used manual techniques for providing intermittent stretch to muscles (such as manual stretching and proprioceptive neuromuscular facilitation techniques) may provide an adequate stimulus for muscle to adapt its length and stiffness. Whether or not these manual techniques on their own provide a stimulus of sufficient duration or frequency for the correction of adaptive muscle shortening remains to be demonstrated. Further research is needed into the nature of the critical stimulus parameters, which perhaps are simply strain or stress and time or number of cycles, but which may be more complex parameters such as those proposed for the adaptation of bone (Carter 1987, Lanyon 1987) or soft connective tissues (Egan 1987). The relationship of the magnitude of these stimulus parameters to the amount or rate of adaptation, may eventually provide information that enables therapists to decide upon the most efficient form of short term intervention.

In the long term therapy must be dictated by the principle that normal movement provides the stimulus for normal muscle length. For the patient with motor control deficits following stroke or head injury, or the patient with habitually poor posture, poor sporting technique or inappropriate working environment, the cause of muscle length changes will persist even after short term therapy has temporarily reversed the problem. Ultimately therapy for these people must involve retraining normal movement, altering the task or structuring the environment (Carr and Shepherd 1987) so that the stimulus for normal muscle prevails.

This review has presented information on the adaptations of muscle length and stiffness. The information can provide the basis from which clinically testable hypotheses concerning the correction of muscle length can be derived. With the use of appropriate measurement tools these hypotheses can be tested both on individual patients and in clinical trials.

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References

Akeson WH, Woo SL-Y, Amiel D *et al* (1974), Biochemical and biomechanical changes in the periarticular connective tissue during contracture development in the immobilised rabbit knee, *Connective Tissue Research* **2** (4), 315-23.

Akeson WH, Amiel D, Woo SL-Y (1980), Immobility effects on synovial joints. The pathomechanics of joint contracture, *Biorheology*, **17**, 95-110.

Akeson WH, Amiel D, Abel MF *et al* (1987), Effects of immobilisation on joints, *Clinical Orthopaedics and Related Research*, **219**, 28-37.

Alnaqeeb MA, Al Zaid NS and Goldspink G (1984), Connective tissue changes and physical properties of developing and ageing skeletal muscles, *Journal of Anatomy*, **139** (4), 677-689.

Banus MG and Zetlin AM (1938), The relation of isometric tension to length in skeletal muscle, *Journal of Cellular and Comparative Physiology*, **12**, 403-420.

Benedict JV, Walker LB and Harris EH (1968), Stress-strain characteristics and tensile strength of unembalmed human tendon, *Journal of Biomechanics*, **1**, 53-63.

Betsch DF and Baer E (1980), Structure and mechanical properties of rat tail tendon, *Biorheology*, **17**, 83-94.

Blix M (1893), Die lange und spannung des muskels. Skand. Arch. F. Physiol, Bd. **4**, S 199., cited in MG Banus and AM Zetlin (1938), The relation of isometric tension to length in skeletal muscle, *Journal of Cellular and Comparative Physiology*, **12**, 403-420.

Bohannen RW (1984), Effect of repeated eight minute muscle loading on the angle of straight leg raising, *Physical Therapy*, **64** (4), 491-497.

Borg TK and Caulfield JB (1980), Morphology of connective tissue in skeletal muscle, *Tissue and Cell*, **12** (1), 197-207.

Butler DL and Little RW (1976), Constitutive equation for skeletal muscle in the passive and active states, in P Komi (Ed), *International Series on Biomechanics 1A*, University Park Press, Baltimore.

Caceci T, Orenstein JM and Bloom S (1981), Surface cables of vertebrate muscles, *Scanning Electron Microscopy*, **3**, 115-123.

Carr JH and Shepherd RB (1987), A motor learning model for rehabilitation, in J Carr and R Shepherd (Eds), *Movement Science: Foundations for Physical Therapy in Rehabilitation*, Aspen Systems, Rockville.

Carter DR (1987), Mechanical loading history and skeletal biology, *Journal of Biomechanics*, **20** (11/12), 1095-1109.

Casella C (1950), Tensile force in total striated muscle, isolated fibre and sarcolemma, *Acta Physiologica Scandinavica*, **21**, 380-401.

Cooper RR (1972), Alterations during immobilisation and regeneration of skeletal muscle in cats, *The Journal of Bone and Joint Surgery*, **54-A** (5), 919-953.

Egan JM (1987), A constitutive model for the mechanical behaviour of soft connective tissues, *Journal of Biomechanics*, **20** (7), 681-692.

Fields RW and Faber JJ (1970), Biophysical analysis of the mechanical properties of the sarcolemma, *Canadian Journal of Physiology and Pharmacology*, **48**, 394-404.

Fish D, Orenstein J and Bloom S (1984), Passive stiffness of isolated cardiac and skeletal myocytes in the hamster, *Circulation Research*, **54**, 267-276.

Fournier M, Roy RR, Perham H *et al* (1983), Is limb immobilization a model of muscle disuse?, *Experimental Neurology*, **80**, 147-156.

Goldspink DF and Goldspink G (1986), The role of passive stretch in retarding muscle atrophy, in WA Nix and G Vrbova (Eds), *Electrical Stimulation and Neuromuscular Disorders*, Springer-Verlag, Berlin.

Goldspink G, Tabary C, Tabary JC *et al* (1974), Effect of denervation on the adaptation of sarcomere number and muscle extensibility to the functional length of the muscle, *Journal of Physiology*, **236**, 733-742.

Goldspink G and Williams PE (1978), The nature of the increased passive resistance in muscle following immobilisation of the mouse soleus muscle, *Journal of Physiology*, **289**, 55P.

Goldspink G and Alnaqeeb MA (1985), Aging of skeletal muscle, in VJ Cristofalo (Ed), *CRC Handbook of Cell Biology and Aging*, CRC Press Inc., Florida.

Goldspink G, Howells K, Williams P *et al* (1988), Age-related accumulation of connective tissue in muscle and the influence of regular exercise, in P Russo and R Balnave (Eds), *Muscle and Nerve, Factors Affecting Motor Performance*, Cumberland College of Health Sciences, Sydney.

Goldstein SA, Armstrong TJ, Chaffin DB *et al* (1987), Analysis of cumulative strain in tendons and tendon sheaths, *Journal of Biomechanics*, **20** (1), 1-6.

Gossman MR, Rose SJ, Sahrmann SA *et al* (1986), Length and circumference measurements in one-joint and multijoint muscles in rabbits after

immobilisation, *Physical Therapy,* **66** (4), 516-520.
Gossman MR, Sahrmann SA and Rose SJ (1982), Review of length associated changes in muscle, *Physical Therapy,* **62** (12), 1799-1808.
Halar EM, Stolov WC, Venkatesh B *et al* (1978), Gastrocnemius muscle belly and tendon length in stroke patients and able-bodied persons, *Archives of Physical Medicine and Rehabilitation,* **59,** 476-484.
Hanson J and Huxley HE (1955), The structural basis of contraction in striated muscle, *Symposia of the Society for Experimental Biology,* **9,** 228-264.
Hanson J and Lowy J (1960), The structure and function of the contractile apparatus in the muscles of invertebrate animals, in GH Bourne (Ed), *The Structure and Function of Muscle, Vol I: Structure,* Academic Press, New York.
Hayat A, Tardieu C, Tabary JC *et al* (1978), Effects of denervation on the reduction of sarcomere number in cat soleus muscle immobilised in shortened position during seven days, *Journal de Physiologie,* **74,** 563-567.
Herbst M and Piontek P (1975), Studies on the origin of latency relaxation and resting tension of skeletal muscle: the effect of hypotonic solutions, *Life Sciences,* **16,** 891-902.
Herbst M and Piontek P (1974), Studies on the origin of resting tension of skeletal muscle, *Biochemical and Biophysical Research Communications,* **57** (4), 1293-1300.
Herring SW, Grimm AF, Grimm BR (1984), Regulation of sarcomere number in skeletal muscle: A comparison of hypotheses, *Muscle and Nerve,* 161-173.
Hill DK (1968), Tension due to interaction between the sliding filaments in resting striated muscle: the effect of stimulation, *Journal of Physiology,* **199,** 637-684.
Holly RG, Barnett JG, Ashmore CR *et al* (1980), Stretch-induced growth in chicken wing muscles: A new model of stretch hypertrophy, *American Journal of Physiology,* **238,** 062-71.
Hooley CJ, McCrum NG and Cohen RE (1980), The viscoelastic deformation of tendon, *Journal of Biomechanics,* **13,** 521-528.
Huet de la Tour E, Tabary JC, Tabary C *et al* (1979a), The respective roles of muscle length and active muscle tension in sarcomere number adaptation of guinea-pig soleus muscle, *Journal de Physiologie,* **75,** 589-592.
Huet de la Tour E, Tardieu G, Tabary JC *et al* (1979b), Decrease of muscle extensibility and reduction of sarcomere number in soleus muscle following a local injection of tetanus toxin, *Journal of the Neurological Sciences,* **40,** 123-131.
Jablecki CK, Hueser JE and Kaufman S (1973), Autoradiographic localization of new RNA synthesis in hypertrophying skeletal muscle, *Journal of Cell Biology,* **57,** 743-759.
Kastelic J, Palley I and Baer (1980), A structural mechanical model for tendon crimping, *Journal of Biomechanics,* **13,** 887-893.
Kovanen V, Suominen H, Heikkinen E (1980), Connective tissue of "fast" and "slow" skeletal muscle in rats — effects of endurance training, *Acta Physiologica Scandinavica,* **108,** 173-180.
Kovanen V, Suominen H and Heikkinen E (1984a), Mechanical properties of fast and slow skeletal muscle with special reference to collagen and endurance training, *Journal of Biomechanics,* **17** (10), 725-735.
Kovanen V, Suominen H and Heikkinen E (1984b), Collagen of slow twitch and fast twitch muscle fibres in different types of rat skeletal muscle, *European Journal of Applied Physiology,* **52,** 235-242.
Lamontagne M, Dore R, Drovin G *et al* (1986), Mechanical properties of tendons measured by strain gauges for different loading angles, in B Jonsson (Ed), *International Series on Biomechanics (XB),* Human Kinetics Publishers.
Lanyon LE (1987), Functional strain in bone tissue as an objective and controlling stimulus for adaptive bone remodelling, *Journal of Biomechanics,* **20,** 1083-1093.
Madding SW, Wong JG, Hallum A *et al* (1987), Effect of duration of passive stretch on hip abduction range of motion, *Journal of Orthopaedic and Sports Physical Therapy,* **8** (8), 409-416.
Magid A and Law DJ (1985), Myofibrils bear most of the resting tension in frog skeletal muscle, *Science,* **230,** 1280-1282.
Maruyama K, Matsubara S, Natori R *et al* (1977), Connectin, an elastic protein of muscle, *Journal of Biochemistry,* **82,** 317-337.
Mayne R and Sanderson RD (1985), The extracellular matrix of skeletal muscle, *Collagen and Related Research,* **5,** 449-468.
McCarter RJM, Nabarro FRN, Wyndham CH (1971), Reversibility of the passive length-tension relation in mammalian skeletal muscle, *Archives Internationales de Physiologie et de Biochemie,* **79,** 469-479.
McLachlan EM and Chua M (1983), Rapid adjustments of sarcomere length in tenotomized muscles depends on an intact innervation, *Neuroscience Letters,* **35,** 127-133.
Medeiros JM, Smidt GL, Burmeister LF *et al* (1977), The influence of isometric exercise and passive stretch on hip joint motion, *Physical Therapy,* **57,** (5), 518-523.
Moss RL and Halpern W (1977), Elastic and viscous properties of resting frog skeletal muscle, *Biophysical Journal,* **17,** 213-228.
Podolsky RJ (1964), The maximum sarcomere length for contraction of isolated myofibrils, *Journal of Physiology,* **170,** 110-123.
Proske V and Morgan DL (1987), Tendon stiffness: methods of measurement and significance for the control of movement. A review, *Journal of Biomechanics,* **20** (1), 75-82.
Ralston HJ, Inman VT and Strait LA *et al* (1947), Mechanics of human isolated voluntary muscle, *American Journal of Physiology,* **151,** 612-620.
Ramsey RW and Street SF (1940), The isometric length tension diagram of isolated skeletal muscle fibres of the frog, *Journal of Cellular and Comparative Physiology,* **15,** 11-34.
Rapoport SI (1972), Mechanical properties of the sarcolemma and myoplasm in frog muscle as a function of sarcomere length, *Journal of General Physiology,* **59,** 559-585.
Rapoport SI (1973), The anisotropic elastic properties of the sarcolemma of the frog semitendinosis muscle fibre, *Biophysical Journal,* **13,** 14-36.
Rowe RWD (1974), Collagen fibre arrangement in intramuscular connective tissue. Changes associated with muscle shortening and their possible relevance to raw meat toughness measurements, *Journal of Food Technology,* **9,** 501-508.
Rowe RWD (1981), Morphology of perimysial and endomysial connective tissue in skeletal muscle, *Tissue and Cell,* **13** (4), 681-690.
Sady SP, Wortman M and Blanke D (1982), Flexibility training: Ballistic, static or proprioceptive neuromuscular facilitation? *Archives of Physical Medicine and Rehabilitation,* **63,** 261-263.
Schmalbruch H (1974), The sarcolemma of skeletal muscle fibres as demonstrated by a replica technique, *Cell and Tissue Research,* **150,** 377-387.
Schwerdt H, Constantinesco A and Chambron J (1980), Dynamic viscoelastic behaviour of the human tendon in vitro, *Journal of Biomechanics,* **13,** 913-922.
Sichel FJM (1941), The relative elasticity of the sarcolemma and of the entire skeletal muscle fibre, *American Journal of Physiology,* **133,** P446.
Stolov WC and Weilepp TG (1966), Passive length-tension relationship of intact muscle, epimysium and tendon in normal and denervated gastrocnemius of the rat, *Archives of Physical Medicine and Rehabilitation,* Sept, 612-620.
Suominen H, Heikkinen E and Parkatti T (1977), Effect of eight weeks physical training on muscle and connective tissue of the M. Vastus Lateralis in 69-year-old men and women, *Journal of Gerontology,* **32** (1), 33-37.
Tabary JC, Tabary C and Tardieu G *et al* (1972), Physiological and structural changes in the cats soleus muscle due to immobilisation at different lengths by plaster casts, *Journal of Physiology,* **224,** 231-244.
Tabary JC, Tardieu C and Tardieu G *et al* (1981), Experimental rapid sarcomere loss with concomitant hypo-extensibility, *Muscle and Nerve,* **4,** 198-203.
Tanigawa MC (1972), Comparison of the hold-relax procedure and passive mobilisation on increasing muscle length, *Physical Therapy,* **52** (7), 725-734.
Tardieu C, Tabary JC, Tabary C *et al* (1977), Comparison of the sarcomere number adaptation in young and adult animals. Influence of tendon adaptation. *Journal de Physiologie,* **73,** 1045-1055.
Tardieu C, Huet de la Tour E, Bret MD *et al* (1982a), Muscle hypoextensibility in children with cerebral palsy: I. Clinical and experimental observations, *Archives of Physical Medicine and Rehabilitation,* **63,** 97-102.
Tardieu C, Huet de la Tour E, Bret MD *et al* (1982b), Muscle hypoextensibility in children with cerebral palsy: II. Therapeutic implications, *Archives of Physical Medicine and Rehabilitation,* **63,** 103-107.
Tardieu C, Tabary JC, Tabary C *et al* (1982c), Adaptation of connective tissue length to immobilisation in the lengthened and shortened positions in cat soleus muscle, *Journal de Physiologie,* **78,** 214-220.
Tardieu C, Blanchard O, Tabary JC *et al* (1983), Tendon adaptation to bone shortening, *Connective Tissue Research,* **11,** 35-44.
Tomanek RJ and Lund DD (1974), Degeneration of different types of skeletal fibres II: Immobilisation, *Journal of Anatomy,* **118** (3), 531-541.
Turto H, Lindy S and Halme J (1974), Protocollagen proline hydroxylase activity in work-induced hypertrophy of rat muscle, *American Journal of Physiology,* **226** (1), 63-65.
Van Brocklin JD and Ellis DG (1965), A study of the mechanical behaviour of toe extensor tendons under applied stress, *Archives of Phys-*

ical Medicine and Rehabiliation, **48,** 369-373.

Viidik A (1966), Biomechanics and functional adaptation of tendons and joint ligaments, in FG Evans (Ed), *Studies of the Anatomy and Function of Bone and Joints,* Springer-Verlag, Berlin.

Viidik A (1973), Functional properties of collagenous tissues, *International Reviews of Connective Tissue Research,* **6,** 218-252.

Walker LB, Harris EH and Benedict JV (1964), Stress-strain relationship in human cadaveric plantaris tendon: A preliminary study, *Medical Electronics and Biological Engineering,* **2,** 31-38.

Warren CG, Lehman JF and Koblanski JN (1976), Heat and stretch procedures: An evaluation using rat tail tendon, *Archives of Physical Medicine and Rehabilitation,* **43,** 461-466.

Wessling KC, DeVane DA and Hylton CR (1987), Effects of static stretch versus static stretch and ultrasound combined on triceps surae muscle extensibility in healthy women, *Physical Therapy,* **67** (5), 674-679.

Williams PE and Goldspink G (1973), The effect of immobilisation on the longitudinal growth of striated muscle fibres, *Journal of Anatomy,* **116** (1), 45-55.

Williams PE and Goldspink G (1978), Changes in sarcomere length and physiological properties in immobilised muscle, *Journal of Anatomy,* **127** (3), 459-468.

Williams PE and Goldspink G (1981), Connective tissue changes in surgically overloaded muscle: Cell and Tissue Research, **221,** 465-470.

Williams PE and Goldspink G (1984), Connective tissue changes in immobilised muscle, *Journal of Anatomy,* **138** (2), 343-350.

Witzmann FA, Kim DH and Fitts RH (1982), Hindlimb immobilisation: length-tension and contractile properties of skeletal muscle, *Journal of Applied Physiology,* **53** (2), 335-345.

Woo SL-Y, Gomez MA, Woo Y-K *et al* (1982), Mechanical properties of tendons and ligaments. II: The relationships of immobilisation and exercise on tissue remodelling, *Biorheology,* **19,** 397-408.