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Muscle Mechanics

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1. Clinical observation of muscle shortening

In clinical practice, a specific phenomenon is frequently observed: muscles, in the absence of congenital or acquired pathologies, neurological disorders, or other specific conditions, may progressively tend to reduce their own length.

This shortening manifests as a reduction in the resting length of the muscle which, while preserving its contractile capacity, leads to an alteration of joint geometry.

This results in a limitation of joint range of motion and, consequently, a modification of the physiological sequence of movement.

To understand the mechanisms underlying this phenomenon, it is useful to analyse the behaviour of the elastic components of muscle tissue according to the principles of material physics.

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2. Physical laws of elastic deformation

Physics teaches that any material subjected to deforming forces undergoes changes according to its elastic modulus.

This universal principle also applies to biological tissues and provides the interpretative key for understanding muscle shortening.

The ideal elastic modulus is equal to 1 and represents a perfectly elastic material that fully restores the accumulated energy, returning exactly to its initial state.

As is well known in physics, such materials do not exist in nature, and a value equal to 1 represents a purely theoretical reference.

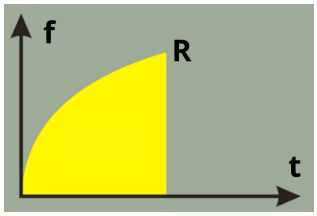

Fig. 1 – The figure shows the deformation curve of ideal elastic materials with elastic modulus E = 1. The yellow area represents complete restitution of stored energy. The variables are force *f* and time *t*.

Real materials present elastic moduli lower than 1.

Consequently, when subjected to deforming forces, they retain residual deformations proportional both to the intensity of the applied force and to the duration of its application.

This means that the greater the force–time product, the greater the permanent deformation of the material.

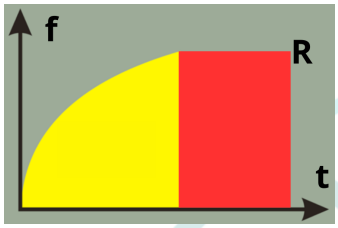

Fig. 2 – The deformation curve of plastic materials (E < 1) shows in yellow the area of partial energy restitution and in red the area of permanent residual deformation. The variables are force *f* and time *t*.

3. Application of physical laws to muscle tissue

Two different elastic materials are present within muscle: the contractile component formed by actin and myosin, and the connective component formed by membranes and tendons.

The contractile component can only contract and relax. It has a very high elastic modulus and is mainly involved in changes in muscle tone rather than permanent structural modifications.

The connective components, on the other hand, having a lower elastic modulus, may remain shortened or lengthened in proportion to the force applied, the duration, and the frequency of the stimulus.

As a result, when subjected to prolonged compressive forces, these tissues retain residual deformations.

This difference in elastic behaviour is confirmed in clinical practice: while muscle relaxation techniques are effective in acting on basal tone, that is on the contractile component, their effect is limited on established connective tissue shortening.

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4. Mechanical classification of elastic elements

From a mechanical standpoint, connective elastic elements of muscle can be divided into two categories based on their behaviour.

Elastic elements in series consist of tendons and their extensions within the muscle belly.

Their function is to absorb stresses produced during muscle contraction, both when the muscle shortens and when it lengthens.

In addition, the presence of protective structures such as Golgi tendon organs prevents injury by inducing muscle relaxation when tension becomes excessive.

An additional advantage of these structures is their ability to restore stored energy, like a spring, according to their elastic properties.

Elastic elements in parallel consist of the sarcolemma, other connective membranes, and the interposed connective tissue.

Their function is to dampen stresses produced by stretching, reducing resistance.

During muscle contraction, these elements undergo direct compression and, depending on the applied force–time product, may retain residual deformation.

It is at this level that permanent muscle shortening develops.

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5. The physical–mathematical model of the muscle fibre

Using a simplified mathematical model of muscle fibre behaviour:

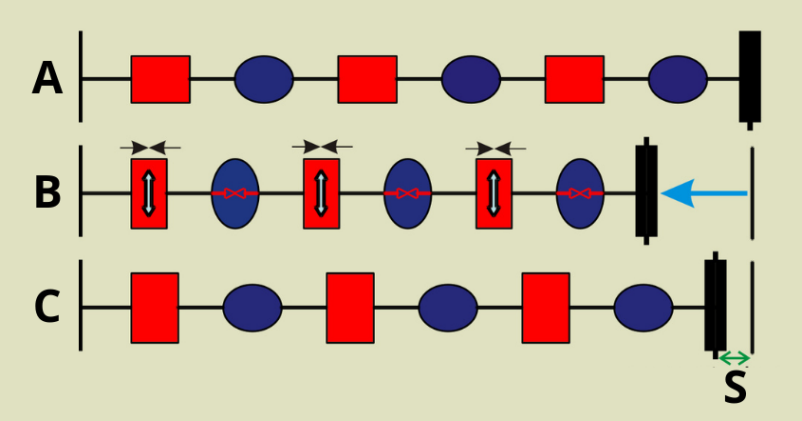

Fig. 3

- A: muscle fibre at rest
- B: contraction
- C: relaxation
- S: summation of residual muscle shortening
- red: connective components arranged in parallel
- blue: contractile components of actin and myosin
- black vertical lines: muscle insertions

During the contraction phase with approximation of insertions (B), the contractile components of actin and myosin actively deform under compression and, by pulling the connective components arranged in parallel, induce further compression.

Connective components arranged in series are not represented, as they do not undergo modifications during the active phase.

Depending on the force–time characteristics of contraction, upon relaxation the contractile component, having a very high elastic modulus, may return to its initial condition or retain a residual compressive deformation expressed as increased basal tone.

The parallel connective component, having a lower elastic modulus, undergoes residual compressive deformation.

At the end of contraction, therefore, all connective components have undergone compressive modification, and their summation determines residual muscle shortening (S).

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6. Muscle as a compressive force

Muscle acts as a compressive force and is not able, by itself, to separate its own insertions.

This principle of muscle mechanics has direct consequences for understanding skeletal alterations: since the skeleton lacks autonomous movement capacity, modification of the physiological joint sequence, in the absence of specific pathologies, is a consequence of shortening muscle forces.

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7. Skeletal consequences of muscle shortening

Muscle contractions involving approximation of insertions and isometric contractions performed outside maximal physiological or relative length, depending on force–time characteristics, lead to loss of muscle length in the connective component and increased basal tone in the contractile component.

At skeletal level, bones on which muscles insert progressively undergo vector traction forces capable of modifying physiological joint sequencing.

At muscular level, progressive connective shortening and increased basal tone of the contractile component increase muscle resistant force and simultaneously reduce its capacity for Work (force × displacement) and Power (Work per unit time).

This mechanism explains clinically observable alterations in physiological joint sequencing in the absence of specific pathology.

The skeleton adapts passively to muscular forces according to mechanical laws.

In this sense, osteoarthritis is considered an effect rather than a cause.

Muscle traction acting on shortened bony ends alters physiological force distribution, concentrating force lines in restricted areas of joint surfaces.

This vector imbalance generates abnormal and chronic intra-articular overcompression which, depending on force and time, induces the tissue and cartilage damage characteristic of osteoarthritis.

Internal mechanical load and external load

In clinical language, the term “load” is often implicitly associated with body weight or external resistance.

From a biomechanical perspective, however, this equivalence is inaccurate.

The musculoskeletal system may be subjected to high mechanical loads even in the absence of relevant external forces, when internal forces generated by shortened muscles produce persistent joint compression.

In these cases, load derives not from what acts on the body externally, but from how forces are distributed internally.

This distinction is essential to understand why misalignments, joint conflicts, and degenerative processes may develop even under conditions of apparent functional unloading or minimal activity.

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8. Relationship between resistant force, Work, and Power

Progressive connective shortening and increased basal tone of the contractile component lead to increased muscle resistant force.

Resistant force refers to the resistance the muscle opposes to traction, that is, to lengthening.

This increase simultaneously reduces the muscle's capacity for Work and Power.

This means that a muscle with shortened connective components, while preserving contractile capacity, must expend part of its energy overcoming internal resistance before producing useful movement.

The principle is analogous to a mechanical system with internal friction: the motor functions correctly, but part of the energy is dissipated overcoming resistance before effective movement occurs.

The consequence is that the muscle, while not truly weak, is mechanically inefficient.

This principle is clinically evident in prolonged immobilisation.

For example, after removal of a cast following humeral fracture, the elbow often presents in flexion.

This posture is determined by connective shortening of the flexor muscles, which have undergone sustained compression during immobilisation and developed increased resistant force.

In this condition, elbow flexors resist extension both during passive stretching and during active extension attempts via triceps contraction.

Paradoxically, evaluation of their dynamic capacity reveals reduction, because available contractile energy is partially dissipated overcoming internal connective resistance.

Differentiating resistant force, Work, and Power is key to understanding how muscle shortening affects both static posture and movement dynamics.

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9. Systemic implications of muscle shortening

Progressive connective shortening does not remain an isolated phenomenon but affects the entire musculoskeletal system.

Statically, altered muscle length forces joints to adapt along new axes, concentrating loads in restricted areas and generating asymmetric stress.

Over time, this altered force distribution may promote degenerative intra-articular processes.

Dynamically, muscle shortening mechanically limits joint excursion, increasing energy expenditure due to internal resistance.

As a result, the system adopts alternative movement patterns that preserve function while deviating from physiological models.

These adaptations perpetuate the phenomenon: altered patterns generate further compensatory shortening in other muscle districts, and the nervous system, through body schema plasticity, normalises these alterations as a new baseline condition.

A self-reinforcing loop is thus established.

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10. Reversibility of muscle shortening

The same physical laws explaining muscle shortening also define the principles of its reversibility.

Connective tissue, having an elastic modulus lower than 1, retains the ability to deform in both shortening and lengthening depending on applied force and time.

Reversibility requires the application of specific therapeutic techniques for durations compatible with the properties of the biological material.

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11. Operational validity of the hypothesis

Muscle shortening, understood as reduction in available muscle length, is an empirical clinical finding observed daily through joint range-of-motion assessment.

The hypothesis proposed—namely that shortening is primarily due to viscoelastic deformation of connective components caused by compressive forces as a function of force–time application—represents a logical interpretative model derived from material physics principles.

Direct histological verification is not currently feasible in vivo, and traction-based mechanical tests do not replicate chronic compressive conditions.

Therefore, the validity of the hypothesis rests on clinical operation:

- it provides a coherent explanation for altered joint sequencing, predicting vector dominance and cascade compensation
- it is indirectly confirmed by the success of interventions aimed at restoring connective length and rebalancing vector forces (Resistant Force / Working Force), leading to symptom resolution

In line with Popper's philosophy, the model remains valid as long as it withstands clinical verification, while remaining open to falsification by new evidence.

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