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Let's stop saying that a muscle ‘contracts’

by

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Abstract

An argument is presented for discontinuing the term ‘contraction’ when describing the so-called active state of muscles and replacing it with a neutral term such as ‘hardening’. The opposite state of the muscle would be called ‘softening’. Hardening as a so-called active state of a muscle may be accompanied by lengthening, shortening, or no dimensional change in the anatomical muscle, depending on the distribution of internal and external forces. All forces in muscles are distributed between two dominant components: discontinuous carneous (fleshy) fibres and continuous collagenous fibres. The viewpoint in this article re-invigorates the old hypothesis that the fleshy component of a muscle works by its cells tending to expand laterally; the laterally-directed stresses are conveyed to the continuous network of collagen and thus to tendons and bones. Shortening in the anatomical muscle is a consequence of the fleshy fibres maintaining a constant volume when hardening and the lateral forces being relayed to the collagen; lengthening of a muscle could be due to both softening of the fleshy fibres and a decrease in the viscosity of the collagen.

The organs of movement

Since the time of Erasistratos (*ca.* 290 BC), muscles have been identified as the unique, active components of the movement apparatus [1]. Vesalius (1514-64) commented in his *De Humani Corporis Fabrica* (1543):

> "...the flesh of muscles, which is different from everything else in the whole body, is the chief agent, by aid of which (the nerves, the messengers of the animal spirits not being wanting) the muscle becomes thicker, shortens and gathers itself together, and so draws to itself and moves the part to which it is attached, and by help of which it again relaxes and extends, and so lets go again the part which it had so drawn" [2, p. 70].

For decades students have approached the phenomena of muscular action through the disciplines of anatomy, or physiology, or biochemistry. Today the study of muscular function is concentrated in two broad, but essentially divorced, fields. On one hand, macroscopic movements and their electromyograms (EMGs) as determined in the living body are studied in the discipline of kinesiology (i.e.,

muscular macrophysiology) and, on the other, the phenomena of fleshy action is studied using isolated muscle fibres (i.e., muscular microphysiology).

A chasm separates the methodology of these disciplines: this can be readily appreciated by looking in the indices of contemporary textbooks of muscular microphysiology for terms relating to the macroscopic behaviour of muscles, and *vice versa*. Teachers in different departments can hardly bridge the chasm themselves by uniting the disciplines, yet somehow, the student is supposed to synthesize the separate details in order to come to grips with how a muscle is actually working in the body.

Terminology

The author believes that a major barrier to uniting the micro- and the macrophysiology of muscle, is the use of the word ‘contraction’. According to dictionaries [3,4], the principal meaning of the term is ‘to cause to shrink’ or ‘to draw together into smaller compass’. It is unclear who was the first to apply the concept of ‘contraction" to describe the shortening action of muscles: translations of Persian and Greek texts often use current terms retrospectively. Needham's book on the historical development of the biochemistry of ‘muscular contraction’ [5], suggests that it may have been Galen of Pergamon (129–201) in his re-interpretation of Erasistratos' views. Certainly ‘muscular contraction’ was a well-established idea when Thomas Willis (1621–1675) wrote *De Motu Musculorum* (1670) and Alfonso Borelli (1608–79), *De Motu Animalium* (published in 1680-81).

Whatever the history, the term ‘contraction’ is identified with the shortening of an active muscle. In fact, dictionaries often give this as an additional meaning. However we know from self-observation that under different circumstances, the same active muscle may either (i) shorten, or (ii) maintain a constant length, or (iii) increase its length. Since the time of Fick [6], physiologists have described the first two states as ‘isotonic contraction’ and ‘isometric contraction’ respectively, and have frequently ignored the third state. Not so the anatomists and kinesiologists, who have variously described the third state as ‘lengthening contraction’, ‘eccentric contraction’ [7, p.406], ‘pliometric contraction’ [8], ‘active relaxation’ or ‘decontraction’ [9, p.7], or even as ‘the action of paradox’ [10, p.8].

The absurdity of the phrase ‘isometric contraction’ (literally shortening at the same length) is compounded with the introduction of concepts such as ‘lengthening contraction’. As MacConnail [11] expressed it, no discipline that has achieved a sufficient state of maturity, would tolerate such illogical terminology.

It is taught that muscles produce movements only by shortening. On the other hand, when an active muscle lengthens, it permits movement to take place under an external force. In the so-called ‘action of paradox’, as when the superior fibres of the deltoid muscle control the descent of the elevated upper limb to the side of the body, the limb is moving, but the movement is produced by gravity. Nevertheless, it is most confusing for students to be told that this is a ‘lengthening contraction’, and then to attempt to relate what is happening in their upper limb to their knowledge of muscular microphysiology.

The issue of the use of the term ‘contraction’ is not merely a semantic one: the term is misleading, and misleading language has a habit of confusing our thinking and inhibiting alternative views of a problem. Even if we were to restrict ‘contraction’ to mean ‘shortening in the longitudinal axis’, we would still require a universal term to describe muscular action in general.

Muscle volume

What facts can we cling to in this confusion? Firstly, Jan Swammerdam (1637–80), by causing an isolated frog's muscle to "contract" in a vial of fluid connected to a capillary manometer, found that muscle volume hardly changed during shortening. This experiment was made about 1663, but was not published until after his death. It is likely that Swammerdam's investigation was greatly influenced by Frans de la Boë (Franciscus Sylvius), who was his teacher at Leiden in 1661. As early as 1640–41, de la Boë had given lectures in medicine at Leiden, in which he proposed that "the contraction of a muscle is nothing else than the distension of its fibres swollen with Animal Spirit and hence its shortening" [12, p.202, p.211]. It is possible [5, p.18] that Swammerdam's experiment influenced the views of his friend and fellow student at Leiden, Nicholas Stensen (Steno, 1638–1686) and,

in turn, the views of William Croone, who met with Stensen in 1665 at Montpellier [15].

In 1667 Stensen argued in his book *Elementorum myologiae specimen seu musculi descriptio geometrica*, that muscular shortening proceeded without a change in volume. Indeed for Stensen, Croone, Borelli, and Willis, the prime event in muscular action was lateral expansion, which then caused, because of the constancy of muscle volume, a shortening [5]. By treating anatomical muscles as being composed of small, identical fleshy parallelepipeds, Stensen [ref], and later Croone, offered different geometrical arguments as to how significant decreases in length could be accompanied by only slight amounts of lateral expansion.

Today we know that microscopic changes in muscle volume can occur when isolated muscles are active in organ baths [16]; however the magnitude of these volume changes (an initial small increase followed by a larger decrease) is only of the order of one part in a million [17]. Thus microscopically, the initial event of muscular activation *in vitro* is never a contraction in volume, but rather a miniscule volume increase. However, at the level of direct human experience, there is no change in muscle volume, but only palpable hardening and change in shape. Thus we move away from reality, both microscopic and macroscopic, if we persist in saying a muscle ‘contracts’.

Muscles in the living body

The second raft of muscular facts that we can cling to is based on the observations of clinical anatomists investigating the action of muscles in the living body, in particular Winslow, Duchenne, Beevor, and Wright [18–21].

We may summarize the action of muscles in the body as follows: (i) all muscles cause movement, or allow movement to take place under external forces, or prevent movement; (ii) when a muscle is active it becomes palpably hard; (iii) the length of muscles on hardening, may decrease, or remain constant, or increase, the girth changing conversely. The feature common to all types of muscular activity is a hardening or tensing of the flesh; the hard muscle behaves as a tendon, or rather a ligament. Physiologists have attempted to measure this hardening of an active muscle by determining the rebound of pendulums striking the sides of the muscle

[22], or by measuring the increase in muscle stiffness to short stretches applied during the course of activity [23].

Since muscular action may occur independently of length changes, as in the maintenance of posture, it makes no sense to concentrate scientific attention on changes in length *per se* and on the related quantities such as velocities of shortening, etc. and to ignore the changes that occur in the other major axis, i.e., orthogonally. Indeed, it could be argued that much of our contemporary teaching on the microphysiology of muscles is in a cul-de-sac because we never encourage students to think of the global changes in an active muscle. Although the importance of the earlier studies of Duchenne and Beevor has been stressed [7, 9, 24], it seems that we have lost the forest while picking up the pine needles.

The sliding filament concept

Let us examine the specific example of muscular hardening accompanied by shortening. This is often described as 'isotonic contraction' but, as has been pointed out [25], this term is nonsense as muscle fibres exert their greatest strength or tone when they are longest; they therefore cannot maintain this same tone (i.e., remain isotonic) as they shorten.

During muscular shortening, the intracellular protein filaments of actin and myosin are believed to slide longitudinally between one another: this is the sliding filament hypothesis, which was proposed in 1954 [26, 27]. Even if the filaments do telescope between each other, the force or mechanism responsible for this is unresolved. The additional hypothesis, which dominates contemporary textbooks, stems largely from the work of A.F. Huxley [28] and holds that the shortening is generated by a longitudinal force, that is, a force acting in the direction of the filaments. In particular, the force is considered to arise partly from the rotation of the heads of the myosin molecules [29] – the cross-bridge hypothesis of force generation.

Why have we forgotten the views of Erasistratos, Stensen, Borelli, Croone, Willis, and others, namely that shortening was a consequence of lateral expansion of a muscle with constant volume? Needham [5] sheds no light on this problem.

Singer [1] states that the ideas of Borelli were “rebutted" by Swammerdam's experiment, but this is clearly not the case, as Swammerdam's experiment is consistent with any mechanism that does not require the volume of an active muscle to increase. Even now one still reads the nonsensical claim that the 'lateral expansion theories' require a muscle to increase in volume during shortening and that Swammerdam's experiment refutes this [30].

The sliding filament and cross-bridge hypotheses, with their emphasis on longitudinal changes, have overshadowed facts from earlier experiments, both microscopic and macroscopic. In particular, the invention of cross-bridges to generate longitudinal forces and shortening leaves completely unexplained the concomitant increase in muscular girth. Morel [31] has commented that, instead of the palpable, transverse expansion of a muscle shortening in activity, the cross-bridge hypothesis would lead one to expect that such a muscle should shrink transversely. This is because the resolution of the oblique vector of each hypothetical cross-bridge into its longitudinal and transverse components would result in a net radial force directed inward. This is opposite to experience. With the emphasis mostly on longitudinal events, the transverse phenomena of muscular action are being ignored.

Despite the apparent universal acceptance of the cross-bridge hypothesis, the older swelling theories are occasionally resurrected in various molecular-mechanistic forms [e.g., 31–35]. To date, the major rebuttal for these proposals is that propounded by A.F. Huxley in his 1974 Review Lecture where he states:

> "Theories... which depend on lateral expansion as the primary event are made unlikely by a recent observation by Matsubara & Elliott (1972)." [36]

This rebuttal is so frequently quoted by supporters of cross-bridges that it demands scrutiny: the particular observation of Matsubara and Elliott [37] is that, in muscle fibres which have been ‘skinned’ by removal of the sarcolemma and which are undergoing shortening, there is no change in the lateral spacing between protein filaments as the sarcomere length decreases. From this it is concluded that there could be no change in lateral dimensions of the whole muscle fibre, and thus that it is

"very improbable that lateral expansion is a causal link in the chain of events between chemical reaction and shortening" [36].

Yet this interpretation and conclusion is itself not immune from criticism: after all, skinned fibres are not normal muscle cells and it is quite unclear why conclusions about their properties can be translated to describe the properties of normal muscle fibres. Moreover, such skinned fibres are swelling steadily during the experiment and are therefore not in a state of rest prior to the superimposed shortening [38]. In all, there appears to be no contemporary evidence from the study of normal muscle, which refutes the idea that lateral expansion is the prime event in muscle shortening. On the other hand there is much evidence, both from everyday experience and physiological experiments [31], which suggests that we should at least retain the lateral expansion hypothesis, until it is refuted.

In everyday experience, the shortening and the widening of an active muscle would appear to be synchronous. However, if the lateral expansion is a causal event in muscle action, it suggests that, on a microscale, the expansion should precede the shortening, or that transverse changes in muscle properties should precede the longitudinal changes. Unfortunately, too few 'muscle microphysiologists' have examined transverse changes in an active muscle, and fewer still have compared longitudinal and transverse changes in the same muscle [39–42]. [add section here on ultrasound measurements] The evidence is inconclusive.

The work of active muscles and 'paradoxes'

If the lateral expansion of a muscle is not a causal event in the process of shortening, how are we to account for the work the muscle fibre does in expanding laterally? For example in 1890, Fick [6] found that in the hindlimb muscles of a frog, the muscles could do as much work by thickening as by shortening. The literature appears to be silent on this issue until 1965, when reviewers commented that if an increase in muscular cross-sectional area is opposed by a reactive force, then the work done in thickening will be unavailable to the work performed on an axial (longitudinal) load [43]. Thus we have a so-called paradox of wasted work. As Wainwright [44] commented in 1986:

"This forceful radial expansion of contracting muscle does no useful work in the familiar locomotor systems of running lizards, birds and mammals."

In reality, the ‘wasted work paradox’ is a by-product of the ‘longitudinal view’ of muscle action. The circular reasoning vanishes the moment we consider the hypothesis that radial expansion is the prime event of muscle action. Radial expansion against a fibrous muscular sheath would allow potential energy to be stored in the stretched muscular sheath, which energy would then be available during subsequent movements. In this context, lateral expansion of muscle fibres (cells) in an anatomical muscle can be interpreted as contributing to the total distribution of energy between active (flesh) and reactive (collagenous) elements.

Similarly, the ‘zero work paradox’ stems from the idea that the work performed by a muscle can be measured by the product of the longitudinal distance and the longitudinal force exerted by the muscle: thus in ‘isometric contraction’, a muscle performs no external work and its mechanical efficiency (if it were a machine) is zero. When it is appreciated that real muscles have both active fleshy and passive collagenous components and that a change in the dimensions of the former can be compensated by a change in the form of the latter (so that the length of the anatomical muscle between its attachments does not change), we see the fallacy of relating the work done by the fleshly component to the distance that, say, the limb moves. A similar consideration applies to the concept of ‘negative work’, which a muscle is supposed to perform when the distance between its attachments is being increased by an external force, as in ‘lengthening contraction’.

Finally the lateral expansion view makes it possible to comprehend how certain muscle cells with their peripheral myofibrils arranged at *right* angles to the direction of the fibre's long axis, e.g., the *Ringbinden* fibres of the extra-ocular muscles [refs], could transfer a force to the terminal tendons.

Recommendations

a. Discontinue the use of the word ‘contraction’ when describing the active state of muscles. Use the term ‘hardening’.

b. Remind our students that activity in a muscle may be felt as a hardening of the muscle and may be accompanied by lengthening, shortening, or no dimensional change of the anatomical muscle, depending on the distribution of internal (i.e., fleshy) and external forces, noting in this that muscles consist of two dominant components.

c. Reinstate the hypothesis that the fleshy part of a muscle works by its cells tending to expand laterally.

Postscript

In the light of the above, the following correspondence between a Sydney orthopaedic surgeon, the late Dr. Crawford McKellar and the anatomist, the late Prof. F. Wood Jones of Liverpool University might be instructive. McKellar to Wood Jones (8.6.1944):

> "The expression 'contraction' annoys me intensely for it implies shortening. Can we not have some word which implies action and so would cover the case where the acting muscle is actually lengthening, etc. e.g., the deltoid when the arm is lowering weight?"

Wood Jones to McKellar (3.9.1944):

> "I know well how wrong the hard and fast use of the word 'contraction' as applied to muscles is. I get my students to lengthen the biceps by extending the elbow and at the same time feeling it 'contract' when they supinate. .... But you must realize that to alter a nomenclature largely established by Sherrington's classical work is no easy matter. One can give corrective lectures to students – but even that may get them into trouble with their examiners. ....Those who would effect changes in accepted terminology must ever have one eye on publishers, examiners and reviewers."

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