

Myocardial Rigour Trigger Points: A Clinical Anatomists Hypothesis of Focal Contractures in the Heart

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ABSTRACT

Based on over forty years of experience with human body donations, this narrative review proposes that myofascial trigger points represent local rigor contractures in living tissue. Previous descriptions of the so-called “rigor trigger” situate the lesion within the sarcomere. The mechanism under consideration involves persistent actomyosin attachment resulting from inadequate ATP at the contractile apparatus. These attachments may sustain tension even after the initial activity has ceased. An endplate disturbance may initiate the skeletal muscle focus. The rigor hypothesis addresses the sustained state of tension. A myocardial rigour trigger is introduced as a focal ATP-limited contracture in viable heart tissue. Experimental metabolic deprivation has demonstrated that cardiomyocytes can develop contracture. The role of connective tissue in transmitting this tension warrants inclusion in the investigation. Previous fascia-focused analyses of muscle, along with the subsequent paper on fascia and its role in rigor mortis, provide a strong anatomical foundation. This review integrates the clinical characterization of myofascial trigger points with experimental findings on cardiac rigor. This review recommends targeted measurements at the identified focus, including assessment of adjacent tissue responses. Myocardial trigger points have not yet been established in living humans. Identifying a viable focal contracture and quantifying the sustaining attachments would directly test this hypothesis.

Keywords

Myocardial rigour trigger, Myocardium, Rigor contracture, Myofascial trigger points, Trigger point ATP, Fascia.

Introduction

Sensorial touch and skilled palpation help identify unusual topography, taut bands, hyperirritable or densified contractures within myofascial tissues. Referring to a palpable lump as a so-called ‘knot’ lacks anatomical clarity. Examiners must determine the specific tissue depth beneath their fingers and the condition of the tissues responsible for the observed topography. The clinical account [1] describes a localised tender point within a taut band, with pain or altered sensation on provocation. The patient's recognition of this sensation provides clinical context to the specialist. This review advances the hypothesis that a myofascial trigger point consists of a cluster of local rigor contractures within the sarcomere. The term “rigor trigger” has previously been applied to this concept [1]. A myocardial rigour

trigger is defined as a proposed focal contracture, or myofascial trigger point, in viable heart tissue. This bespoke concept is informed by observations of post-mortem muscle stiffness and living myofascial tissues, attributing persistent tension to affected contractile sites. The number and distribution of these sites warrants microscopic investigation. An endplate disturbance could initiate the contracture while calcium availability sustains activation while inadequate ATP at the myosin head would prevent detachment. The energy-crisis hypothesis encompasses metabolic failure [2]. The rigor hypothesis emphasizes the role of attachments that maintain tension after the initiating activity has ceased. Recent research [3] has identified a potential contribution of fascia to post-mortem stiffness. Muscle fibers stiffen within connective tissue, resulting in resistance that is transmitted through this tissue during examination. The paper referenced [1] incorporates the anatomical structures responsible for tension into the analysis of rigor. Given that the heart is a myofascial structure, it warrants examination as well. The actomyosin apparatus of the heart is vulnerable to

energetic failure, and its associated connective tissues transmit load between cells. To date, myofascial trigger-point theories have primarily addressed skeletal muscle. The myocardium raises a specific anatomical question: can a viable region of myocardial tissue exhibit a persistent focal rigor contracture?

Review Method

This narrative review examines focal contracture and myocardial energetics. The author’s latest book [1] was used alongside publicly accessible articles and bibliographic records. Material was drawn from chapters 1, 3, 4 and 5 identifying the cited material. The literature that was considered concerns myofascial trigger points, ATP-dependent actomyosin behaviour and myocardial metabolic deprivation. Papers on connective tissue mechanics and chest pain were included where they addressed the anatomical or clinical interpretation. Foundational experiments were retained because they manipulated the contractile conditions under examination. Some reports were accessible only as abstracts. The selection was narrative; no systematic screening or formal risk-of-bias assessment was undertaken.

Inhibition and cross bridge release

Within the context of muscle activity, inhibition is central to the assessment of movement [1, ch. 3]. Inhibition is defined as the regulation that restrains or reduces contractile activation. ATP binding is the molecular event necessary to release an attached myosin head [4], and a rigor attachment may persist after the activating input has been inhibited. Calcium ion availability is critical for activation of the contractile apparatus. The referenced account [1, chs. 3–4] incorporates calcium dynamics into the explanation of persistent sarcomere shortening. Elevated cytosolic calcium can sustain cycling if ATP remains available [place the Gordon AM, Homsher E, Regnier M. reference here]. The rigor state discussed here involves attachments maintained

under inadequate ATP support. Measurements at the focus should determine which state maintains force. Rigor mortis refers to the post-mortem phenomenon, while rigor contracture describes a similar mechanically resistant state resulting from energetic failure. This review examines a localised occurrence in viable tissue. Sampling must target the contracted focus and consider phosphocreatine buffering. The relevant physiological conditions are present at the contractile apparatus.

Endplate activity and persistent contracture

Gerwin [5] proposed a failure of protective feedback, with persistent calcium accumulation at some trigger points. Continued activation in a shortened focus increases demand on local ATP supply. Inadequate calcium clearance sustains activation. More severe energetic failure can impair actomyosin detachment. The proposed failure is persistent attachment after the initiating activity has been inhibited. Endplate recordings would document electrical activity at the site. Cross-bridge measurements would establish whether impaired detachment maintains its tension. Pain recorded during the same investigation would link the tissue state to the patient’s symptoms. Cardiomyocytes lack the motor endplates found in skeletal muscle. Sympathetic nerves communicate with them at neurocardiac junctions [6]. Initiating a myocardial focus therefore requires examining cardiac physiology. Figure 1 locates the proposed ATP-dependent detachment failure in each tissue.

What has been measured at myofascial trigger points Shah et al. [7] sampled an altered extracellular chemical environment around active trigger points. Their findings place the focus within a local environment capable of influencing nociception. Intracellular measurements at the same site would allow examination of ATP availability and actomyosin attachment alongside that chemistry. The examination needs a consistently located focus. The clinical account [1, ch. 4] describes active and latent myofascial trigger

Persistent contracture in skeletal and cardiac muscle

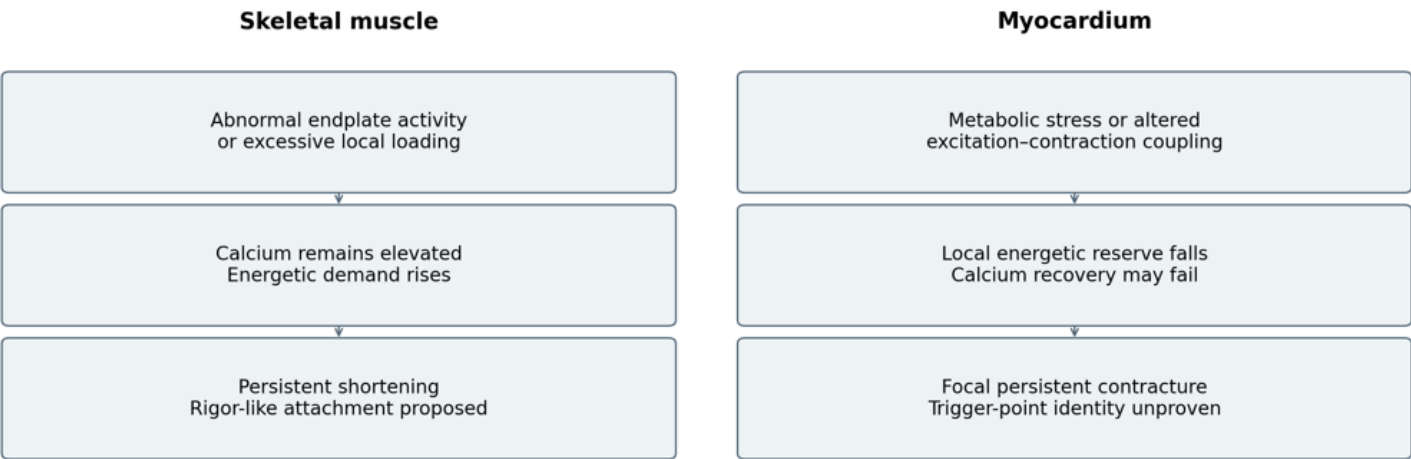


Figure 1: Proposed sites of persistent contracture in skeletal muscle and myocardium. ATP-dependent failure of actomyosin detachment is the mechanism investigated in each tissue.

points by the sensation produced on provocation and its relationship to the patient's complaint. The same chapter describes the local twitch response and spontaneous electrical activity as additional findings. Recording how a site was identified would allow later measurements to refer to the clinical finding examined. The diagnostic consensus of Fernández-de-las-Peñas and Dommerholt [8] provides criteria for locating that site. Agreement among examiners would ensure physiological measurements target the same tissue. The pathogenesis reviewed by Steen et al. [9] remains unresolved. Reproducible localisation is the first requirement for investigating it. Tsai et al. [10] measured the mechanical response of low-back myofascial trigger points. Their work helps locate and quantify resistant tissue. Chen et al. [11] compared ultrasound findings with endplate noise in rat masseter muscle and reported second-stage specificity of 40%. These studies provide methods for locating a candidate focus. A study of rigor would add measurements of its energetic and cross-bridge state. Cao et al. [12] reported five biopsies from a study of 49 participants. The samples showed remodelling without so-called identifiable contraction knots.

Tissue remodelling deserves direct comparison with persistent actomyosin attachment as a source of the resistance found at a clinical trigger point. Quintner et al. [13] questioned whether the clinical findings identify a distinct lesion. A study that samples a consistently located focus and measures the state maintaining its resistance would address that criticism directly.

Imaging the identified focus

Ultrasound gives the examiner an excellent way to return to the tissue identified by palpation. In lower-back skeletal muscle, grayscale images recorded the depth and dimensions of the focus. Measurements before and during applied pressure quantified its deformation [10]. Figure 2 shows this approach. Probe force and the depth of the focus belong in the interpretation of the image.

Shear-wave elastography and Doppler add measurements of tissue stiffness and the local vascular environment. A recent upper-trapezius study combined these methods with grayscale imaging and a small biopsy series [12]. For the rigour-trigger hypothesis, imaging should guide physiological measurements to the same focus. Establishing persistent ATP-limited actomyosin attachment requires those measurements. A myocardial investigation must also account for the changing load and motion of the beating heart.

The connective tissue carrying the tension A primary focus is on tissue that maintains its intrinsic tension. The anatomical account [1, chs. 1 and 3] places named muscles within continuous connective tissue relationships. That account asks the examiner to follow those relationships beyond the site of resistance. At the location of a myofascial trigger point, the palpable finding includes the focus and the tension transmitted into adjacent tissue. Chapter 4 describes shortened sarcomeres at the nodule and lengthened sarcomeres on either side, producing the associated taut band [1]. This provides a specific arrangement to investigate. Sarcomere lengths could be mapped across the focus and compared with deformation in the surrounding matrix. The tissue felt beneath the examiner's fingers would then have a microscopic account. The referenced book [1] also distinguishes between central and attachment trigger points. Its account relates an attachment-site finding to tension carried from a central contracted band [1, ch. 4]. That relationship directs attention to where resistance is generated and where it is encountered. In the heart, local cell-to-matrix relationships require their own anatomical description. Recent research [3] places post-mortem stiffness within the anatomy of muscle and fascia: the candidate mechanisms concern matrix material properties and the distribution of pre-existing tension. Cardiac fibroblast experiments show that matrix mechanosensing affects fibrosis [14]. Measuring the adjacent matrix alongside the contractile focus would allow assessment of its contribution to resistance.

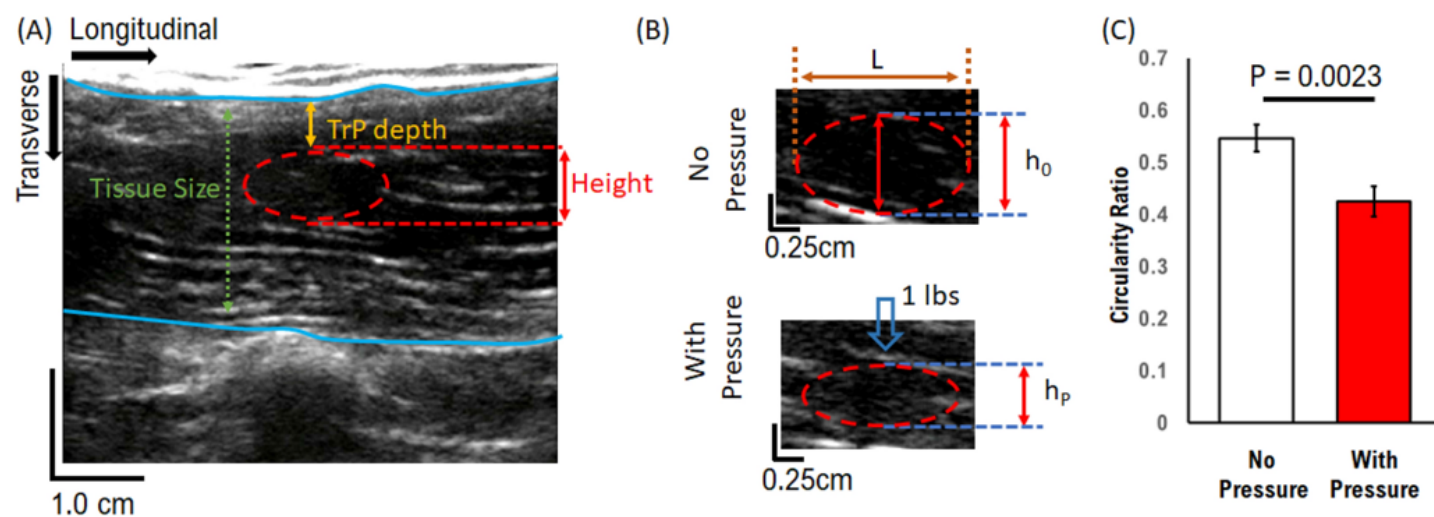


Figure 2: Ultrasound measurement of a clinically identified focus in lower-back skeletal muscle. (A) Location and dimensions. (B) Change in transverse height under applied pressure. (C) Circularity ratio before and during pressure. Reproduced from Figure 1 of Tsai et al. [10]. Scientific Reports. 2024;14:7562. <https://doi.org/10.1038/s41598-024-57733-4>. Licensed under CC BY 4.0 (<https://creativecommons.org/licenses/by/4.0/>). No changes made.

Contracture in cardiac muscle

Haworth et al. [15] produced contracture in metabolically deprived adult rat cardiomyocytes. Severe ATP loss accompanied the contracture. Cardiac cells can enter this mechanically resistant state during energetic failure. Ventura-Clapier and Veksler [16] manipulated energetic conditions in skinned rat ventricular fibres. Phosphocreatine and ADP altered rigor tension and stiffness. Local buffering and metabolite availability therefore belong in an investigation of the proposed myocardial focus. Studying viable myocardium requires preserving local tissue relationships. The proposed lesion is a focal contracture in this setting. Its energetic state and response to restored ATP support would provide the first measurements. Investigation of nociceptive effects would address its possible relationship to chest pain.

Locating the source of chest pain

Travell and Rinzler [17] identified pain originating in the chest muscles that closely resembles angina and myocardial infarction. Their findings establish the chest musculature as a potential anatomical source of angina-like pain. The clinical account emphasizes the importance of the patient's description of referred sensations and their identification during examination [1, chs. 4–5]. The tissue responsible for these sensations should be the primary focus of investigation. In the context of a proposed myocardial origin, this investigative approach begins within the heart. Figure 3 presents the chest-wall findings in relation to experimental myocardial rigor and the hypothesized myocardial lesion.

Unrevealing cardiac investigations Vrints et al. [18] discuss angina and ischaemia with non-obstructive coronary arteries in the ESC guidelines. Coronary functional disease is recognised as a potential explanation for symptoms that routine investigations may not clarify. According to “Understanding Fascia, Tensegrity and Myofascial Trigger Points” [1, ch. 5], medical history and scope of practice are foundational to patient assessment. This approach

is particularly pertinent when symptoms involve the chest. Chest-wall tenderness and cardiac disease may coexist, and any new or changing chest pain warrants medical evaluation. Investigating microscopic myocardial contracture requires a method that can identify the specific focus and quantify the tension that sustains it.

Measuring the proposed lesion

The investigation should begin with a clinically identified skeletal-muscle focus, located independently by multiple examiners before physiological assessment. It is essential to document palpation findings and the patient's reported sensations. Consistency in pressure application and anatomical location is vital for reliable examination [1, chs. 4–5]. One should compare the identified focus with a non-tender site in the same muscle and with sites from participants experiencing diffuse muscle pain. It is important to measure force and sarcomere length during controlled changes in ATP availability and calcium activation. Cross-bridge assessments can determine whether persistent attachment or continued cycling is present. Restoration of ATP support under controlled calcium conditions will clarify the energetic contribution to resistance. Measure the adjacent matrix concurrently. Map the shortened region relative to its taut band and the deformation transmitted through the tissue. Excision delay and fixation may introduce contracture artefacts; therefore, control sample handling before attributing microscopic findings to the original clinical site. In cardiac investigations, use viable tissue that preserves local cell-to-matrix relationships. The focus must be localised and assessed for injury, with its energetic state mapped against diastolic behaviour and conduction. Preparations containing fibroblasts enable measurement of the matrix response. Assessing nociceptive effects requires a model that incorporates relevant sensory pathways. Human studies should be based on evidence of the mechanism in viable preparations. Imaging may help localise regional behaviour. Tissue obtained for independent clinical indications may allow structural examination with appropriate consent.

The anatomical site determines the evidence required

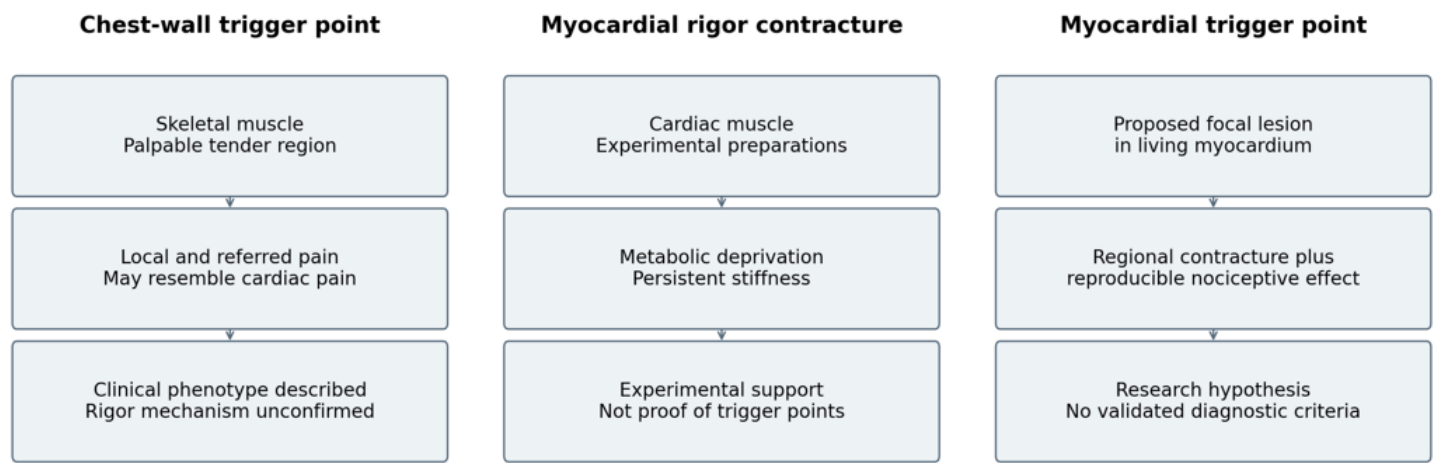


Figure 3: Chest-wall trigger points, experimental myocardial rigor and the proposed myocardial trigger point. Each requires evidence from its own anatomical site.

Table 1 identifies results that would challenge the hypothesis. Figure 4 sets out the measurements required.

Table 1: Results for evaluating the local rigor hypothesis at a consistently identified focus.

Finding to test	Evidence supporting rigor	Evidence requiring another explanation
Persistent contracture at the identified focus	Local energetic deficit with persistent actomyosin attachment	Adequate energetic support and no rigor attachment
ATP dependent release	Resistance falls when ATP support is restored under controlled calcium conditions	Continued activation or matrix resistance explains the force
Force transmitted into adjacent tissue	Matrix deformation changes as the focal contracture resolves	Resistance persists independently of the contractile focus
Focal contracture in viable myocardium	Localised energetic failure and contracture in viable tissue; nociceptive effects tested separately	Diffuse deprivation, irreversible injury or recognised disease accounts for the findings

Limitations

The “rigour trigger” developed from the earlier anatomical account [1] is an anatomical hypothesis. The book also proposes an electromagnetic account of shortening and a needle-related phase change [1, ch. 4]. These proposals require their own measurable variables and experiments. The molecular test developed in this review concerns persistent actomyosin attachment under inadequate ATP support. The approximate number of affected sarcomeres in the book is a proposed scale, not a measured count at a clinical trigger point. No study examined here demonstrates that mechanism at a clinically identified human trigger point. The cardiac experiments used isolated cells under metabolic deprivation or skinned fibres. A focal lesion in a functioning heart

remains to be identified. The proposed fascia contribution [3] also awaits experimental confirmation. Palpation and imaging help locate a site for investigation. The diagnosis of a cardiac myofascial trigger point or cardiac rigor contracture requires evidence of the attachments maintaining their tension and the energetic conditions at that site. Matrix remodelling and altered nociception remain possible explanations for similar clinical findings. Myocardial trigger points remain unestablished in living humans. Negative routine investigations cannot identify the proposed lesion or exclude coronary functional disease. The hypothesis currently provides no basis for myocardial needling or attempted manual release.

Conclusion

The phrase “rigour trigger”, as previously proposed, [1] places persistent trigger-point tension within the sarcomere. An endplate disturbance could initiate the skeletal-muscle focus while failure of ATP-dependent actomyosin detachment could maintain it after the initiating activity has been inhibited. This is the molecular claim to test. The book, “Understanding Fascia, Tensegrity, and Myofascial Trigger Points” [1] places focus within the muscle fiber and within the connective tissue carrying its tension. The nodule and taut band become characteristics of the same anatomical investigation. Research on the possible role of fascia in rigor mortis [3] gives further reason to measure the contribution of the surrounding tissue. The heart deserves examination on these grounds. Cardiomyocytes are susceptible to contracture during energetic deprivation. A viable myocardial focus should be sought and the state maintaining its tension measured. Its effect on adjacent tissue and its relationship to pain can then be investigated. The hypothesis now has a defined anatomical site and a direct experimental task.

Significance statement

The “rigour trigger” hypothesis locates persistent myofascial tension at the sarcomere. This review develops a test of ATP-

Locate the focus and measure its contractile state

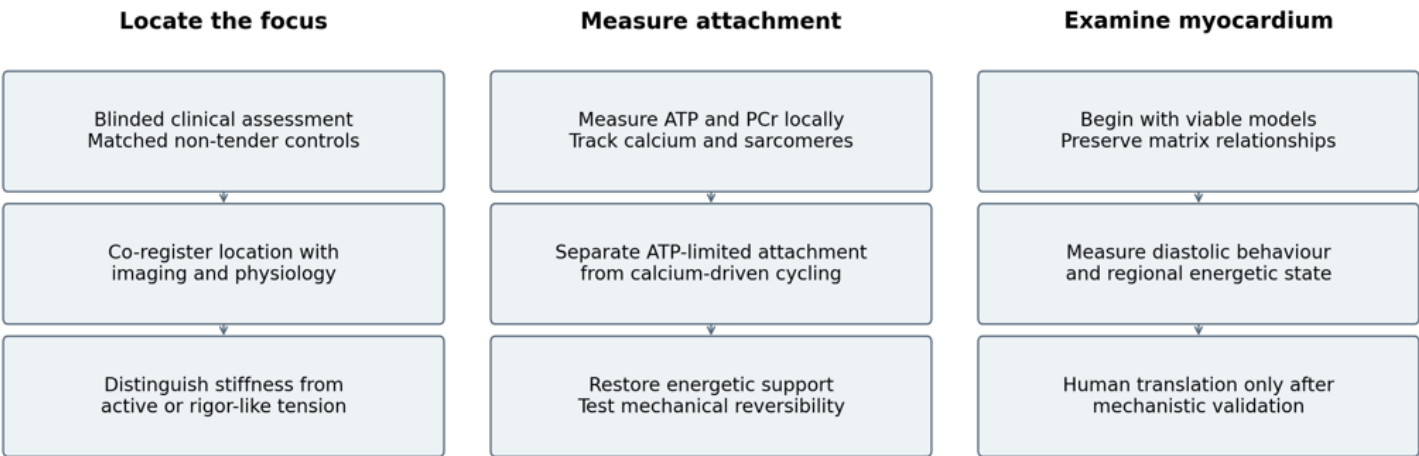


Figure 4: Measurements needed to identify the focus, establish its contractile state and investigate myocardium.

dependent failure of actomyosin detachment and brings the connective tissue carrying the tension into the same investigation. Focal contracture in viable myocardium is proposed as a further anatomical target.

Ethical and consent statement

This narrative review reports no new human or animal experiments. Figures 1, 3 and 4 are original conceptual schematics. Figure 2 reproduces published ultrasound images and quantitative results under CC BY 4.0, with attribution in the caption. No new donor or patient material is reported.

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Conflicts of interest

The author is the author of Understanding Fascia, Tensegrity, and Myofascial Trigger Points and a contributing author to the fourth edition of Travell, Simons & Donnelly's Myofascial Pain and Dysfunction.

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