Myofascial Trigger Points: From Metabolic Failure to Recovery

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Abstract

Background: Conventional treatment of myofascial pain has historically focused on symptom mitigation (pain relief), which may explain the high recurrence rates observed in clinical practice. Myofascial trigger points (MTrPs) represent a structural metabolic dysfunction characterized by a state of tissue hypoxia, localized "rigor mortis", and fascial restriction.

Methods: This article aims to integrate the energy crisis model with molecular findings of tissue acidosis and ischemia, proposing a sequential non-invasive treatment triad consisting of radial pressure waves/focused shockwaves/radial pressure waves application (preparation/intervention/drainage).

Discussion: The chronological evolution of the MTrP concept is reviewed, from the integrated energy crisis hypothesis proposed by Travell and Simons to the molecular confirmation of Jay Shah's "biochemical soup" and cytoskeletal blockade model. Shockwave therapy is introduced as a cellular "mechanical defibrillator" capable of restoring ATP synthesis and sarcomere homeostasis within a tensegrity-based framework. Treatment outcomes are further assessed using pressure algometry as an objective measure of functional recovery.

Conclusion: The BIOLOGICAL RESET protocol redefines the therapeutic approach, shifting from passive analgesic management toward active biological engineering, achieving clinical success through pure mechanotransduction.

Keywords: Myofascial pain syndrome, Trigger points, Shock waves, Radial pressure waves

Section I. Introduction and Chronological Pathophysiological Framework

Short-term relapse is a constant challenge in the field of musculoskeletal rehabilitation. It is common for a patient to be discharged symptom-free, only to return a few weeks later with the same clinical presentation. This pattern suggests that conventional treatments frequently mask superficial symptoms without addressing the underlying cellular metabolic crisis.

Manual therapy is inherently limited by its depth of penetration and energy-transfer capacity, preventing it from reaching the core of the dysfunction. Consequently, direct intervention at the cellular level is required, with pressure wave therapies serving as a mechanism for metabolic reactivation.

Over recent decades, the concept of the myofascial trigger point (MTrP) has evolved from the subjective palpation of a "muscle knot" to molecular confirmation [1,2]. Today, it is understood as a failure of sarcomere self-regulation, in which the contractile unit becomes trapped in a vicious cycle of contraction, ischemia, and pain, ultimately losing its metabolic homeostasis [1]. The origin of this biochemical arrest can be better understood through the contributions of four key theories:

The energy crisis hypothesis of Travell and Simons

The original integrated hypothesis proposes that dysfunction begins at the motor endplate, where excessive acetylcholine release occurs [1]. This neurochemical overload maintains persistent depolarization of the postsynaptic membrane, resulting in continuous calcium (Ca^{2+}) release from the sarcoplasmic reticulum. Sustained intracellular calcium concentrations maintain actin-myosin cross-bridge formation [3].

The force generated by this focal contraction is sufficiently intense to compress adjacent capillaries, producing critical ischemia and depriving the tissue of oxygen. In the absence of oxygen, mitochondrial oxidative phosphorylation ceases, leading to ATP depletion. Without this energy source, the cell can no longer power ion pumps required to recapture calcium against its concentration gradient. Thus, muscle relaxation becomes impossible because ATP is required for relaxation, while ischemia simultaneously prevents ATP synthesis.

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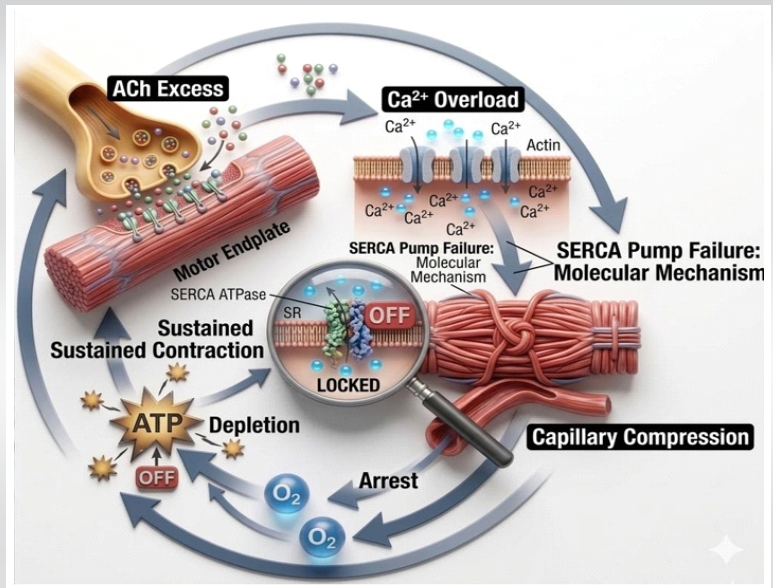

Figure 1: Gerwin's active transport failure model: As ATP concentrations decline, the sarco/endoplasmic reticulum Ca^{2+}-ATPase, responsible for calcium reuptake into the sarcoplasmic reticulum, becomes inactive due to insufficient energy availability.

Gerwin's active transport failure model

Robert Gerwin demonstrated that MTrPs fundamentally represent a failure of the active transport systems of the cellular membrane [4]. As ATP concentrations decline, the sarco/endoplasmic reticulum Ca^{2+}-ATPase (SERCA), responsible for calcium reuptake into the sarcoplasmic reticulum, becomes inactive due to insufficient energy availability [5].

The tissue consequently enters a state of biochemical arrest in which muscle relaxation cannot be achieved through passive stretching or manual compression alone. Without adequate ATP reserves, cross-bridge dissociation is not possible unless an external biophysical stimulus restores ionic homeostasis (Fig. 1).

Jay Shah's biochemical soup

Using in situ microdialysis techniques, Shah et al. demonstrated that the core of an active MTrP exhibits a profoundly pathological biochemical environment, with pH values decreasing to approximately 4.0 [2]. This severe acidosis alters the physicochemical properties of hyaluronic acid, increasing its viscosity and impairing fascial gliding.

Furthermore, Shah et al. documented substantial accumulation of nociceptive mediators and pro-inflammatory cytokines, including substance P, bradykinin, serotonin, interleukin-1β, and tumor necrosis factor-alpha [6].

At this stage, manual therapy encounters a fundamental biophysical limitation: manual pressure alone is incapable of clearing a biochemical burden of this magnitude. Effective removal of these metabolites requires a deep hydrodynamic stimulus capable of facilitating tissue drainage and metabolic clearance.

Localized rigor mortis model

Simons identified the ultimate consequence of this acidic environment. When tissue pH decreases below 6.5, SERCA activity becomes profoundly inhibited [3]. In the absence of ATP and calcium reuptake, the cross-bridges of contractile proteins become permanently fixed.

Simons described this condition as a state of localized "rigor mortis" (Fig. 2). In this context, the sarcomere is not actively contracting but rather immobilized by a molecular kinetic failure. Reversal of this structural blockade requires an external intervention capable of inducing mechanotransduction, restoring perfusion, and promoting ATP resynthesis.

Section II. Fascial Tensegrity and the Concept of Mechanical Defibrillation

Localized rigor mortis does not occur in isolation; rather, it develops within a three-dimensional network governed by the principles of tensegrity, a concept of biological architecture extensively described by Ingber [8]. The human body is not maintained through continuous compression, such as a brick column, but through a three-dimensional distribution of forces in which bones function as discontinuous compression struts and the myofascial system acts as the continuous tension network [9].

When a group of sarcomeres becomes trapped within the chronic contraction cycle described by Travell and Simons [1, 3], the resulting traction forces are transmitted to the extracellular matrix through integrins and costameres [9]. The tissue acidosis described by Shah et al. [2, 6], together with prolonged ischemia, increases collagen density and elevates hyaluronic acid viscosity, causing the extracellular matrix to transition from a fluid state to a semi-solid gel state.

This transformation converts the fascia into a rigid structure that entraps the trigger point, restricts fascial gliding, and disrupts local tensegrity [8]. Consequently, the MTrP becomes a fascial prison that perpetuates inflammatory and ischemic processes (Fig. 3).

Tissue release therefore requires a biophysical intervention capable of modifying substrate properties. When a specific mechanical stimulus is applied, a thixotropic effect is generated whereby hyaluronic acid decreases in viscosity and transitions from a gel state to a sol state, restoring three-dimensional elasticity and facilitating fluid movement

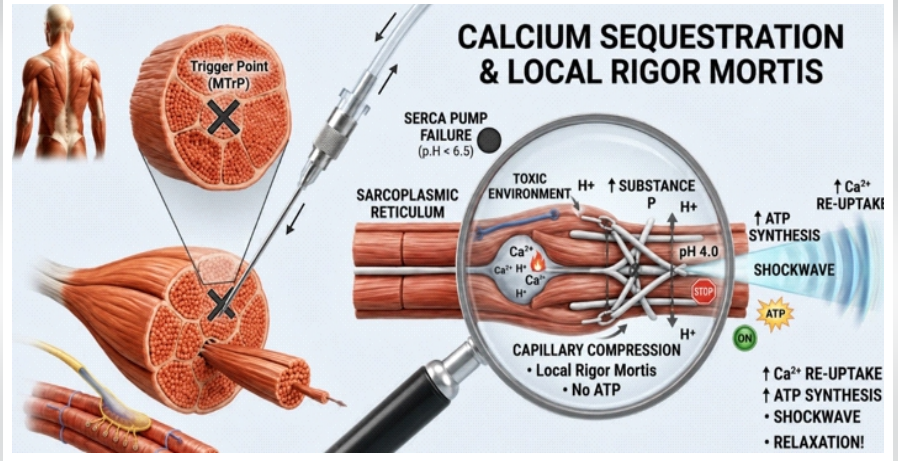

Figure 2: "Rigor mortis" model: The sarcomere is not actively contracting but rather immobilized by a molecular kinetic failure.

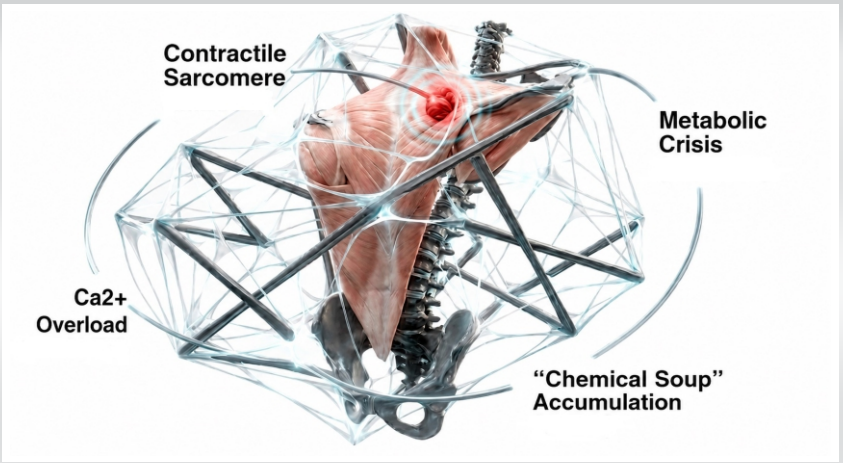

Figure 3: The myofascial trigger points becomes a fascial prison that perpetuates inflammatory and ischemic processes.

Shockwaves as cellular mechanical defibrillators

To understand the effects of shockwaves at this stage, an MTrP may be functionally compared to a state of metabolic fibrillation. Similar to cardiac fibrillation, in which the myocardium exhibits electrical activity and disorganized contraction without effective function or relaxation, the sarcomere may be viewed as undergoing metabolic fibrillation.

In the presence of cardiac fibrillation, emergency medicine does not rely on friction or massage but instead delivers an electrical shock to depolarize and reset the membrane.

Analogously, focused shockwave therapy acts as a mechanical defibrillator within muscle tissue [10,11]. It does not function as a conventional mechanical stimulus or superficial massage. Rather, the focused acoustic pulse generates an abrupt pressure change that penetrates directly into the core of the energy crisis.

This mechanical impact disrupts the cross-bridges fixed by localized "rigor mortis", normalizes cellular membrane potentials, and alters sarcolemmal permeability. The resulting mechanotransduction response activates microcirculation, enhances oxygen delivery, and promotes endogenous ATP synthesis [10,12].

By restoring this metabolic engine, SERCA-mediated calcium reuptake resumes [5,7], interrupting the Travell-Simons cycle [1] and re-establishing cellular homeostasis within the affected tissue.

Figure 4: Pressure algometry is a quantitative assessment tool of choice in daily clinical practice.

Section III. Precision Diagnosis, Quantitative Metrics, and Technological Synergy

The evolution of evidence-based medicine requires objective and quantifiable clinical data. Historically, the diagnosis of myofascial pain syndrome has relied heavily on manual palpation; however, the medical literature has consistently demonstrated limited objectivity and poor inter-rater reliability when digital palpation is used as a standalone diagnostic tool [4]. To validate the outcomes of advanced biophysical interventions, it is essential to incorporate quantitative assessment methods capable of documenting actual changes in tissue homeostasis [13].

The clinical duality of MTrPs and pressure algometry

In clinical practice, it is crucial to distinguish between the two operational manifestations of MTrPs [1]:

- Active MTrP: Represents a persistent focus of altered metabolic activity that produces spontaneous, continuous, and referred pain, accompanied by functional impairment and reduced muscle flexibility [1,2,3,6].
- Latent MTrP: Remains clinically silent at rest but exhibits genuine motor dysfunction, localized stiffness, and altered neuromuscular recruitment patterns that often go undetected until physical examination [1,2].

To minimize the subjective bias associated with patient-reported improvements, such as "it hurts a little less," pressure algometry emerges as the quantitative assessment tool of choice in daily clinical practice (Fig. 4), whereas elastography remains primarily confined to research settings.

Based on the thresholds validated by Fischer, an active MTrP mechanically collapses and elicits pain at pressures below 2.5 kg/cm2 [13], whereas healthy homeostatic muscle tissue typically tolerates approximately twice this amount of pressure [14]. Pre- and post-treatment algometric measurements therefore provide an objective and reproducible assessment of pressure pain threshold, enabling clinicians to document functional restoration of the sarcomere using quantitative data.

Biophysical synergy: Complementary biological roles of focused shockwaves and radial pressure waves

Once dysfunction has been identified and quantified, the therapeutic strategy should not be framed as a choice between competing technologies but rather as a biologically complementary approach. Physical and clinical evidence suggests that focused shockwaves and radial pressure waves possess distinct physical characteristics that translate into complementary biological functions within the tissue.

Focused Shockwave (The "Sniper")

The biophysical design of focused shockwave therapy concentrates maximum energy density at a deep convergent focal point without significant dissipation in superficial tissues. Its biological role is primarily structural and regenerative. It penetrates directly into the core of localized rigor mortis, promoting cavitation phenomena, disrupting fibrotic tissue, stimulating deep angiogenesis through growth factor release, and reactivating cellular energy metabolism [10,12].

Radial pressure wave (The "Purifier")

Radial pressure waves are generated pneumatically and propagate in a divergent pattern, delivering their greatest mechanical impact at superficial tissue levels. Their biological role is predominantly hydrodynamic and metabolic. They perform a mechanical sweeping action across superficial fascia and muscle bellies, facilitating the mobilization and clearance of Jay Shah's "biochemical soup."

This pneumatic stimulus disperses accumulated metabolites, elevates local tissue pH, promotes tissue alkalinization, and facilitates lymphatic drainage pathways [11,12].

The sequential combination of these two modalities should not be regarded as redundant; rather, it represents a planned tissue-level synergy in which focused shockwaves repair deep cellular ultrastructure while radial pressure waves optimize the surrounding metabolic microenvironment [15].

Section IV. Integrated Mechanisms of Action and Sequential Clinical Dosing

The clinical success of shockwave therapy in myofascial pain syndrome cannot be attributed to a single isolated mechanism. Restoration of tissue homeostasis results from the simultaneous activation of four principal biological pathways, collectively constituting what may be described as a "Biological RESET" [10,12].

Mechanical pathway

The acoustic pulse disrupts intracellular fibrotic bands and physically breaks the persistent cross-bridges within the immobilized sarcomere, restoring mobility to contractile proteins.

Chemical pathway

The pneumatic and hydrodynamic stimulus promotes extensive clearance of Shah's et al. "metabolic soup," actively removing accumulated hydrogen ions (H^+), substance P, and pro-inflammatory metabolites, thereby reversing local acidosis [2,6].

Neurological pathway

Energy delivery to nociceptors induces transient hyperstimulation of afferent fibers. This process disrupts the pain reflex arc through habituation and saturation mechanisms consistent with gate control theory, resulting in immediate analgesic effects [10,16].

Biological pathway

Mechanotransduction stimulates the expression of vascular endothelial growth factor and endothelial nitric oxide synthase, promoting angiogenesis, restoring oxygen delivery to ischemic tissue, and supporting long-term tissue repair [12,15].

The non-negotiable dosing triad: The clinical protocol

To translate this physiological cascade into a reproducible and safe therapeutic outcome, treatment should be structured according to a three-phase sequential protocol [15]:

Phase 1: Preparation and thixotropic modulation

The primary objective is to reduce the viscosity of the densified extracellular matrix (transition from gel to sol state) while simultaneously desensitizing superficial tissues [10].

A low-energy, high-frequency stimulus is applied using radial pressure waves at low pressure settings or high-intensity pulsed laser therapy across the tendinous and myofascial regions. This phase decreases the mechanical resistance of the collapsed tissue and improves patient tolerance for the subsequent intervention [15,16].

Phase 2: Structural intervention (the cellular defibrillator)

Once the fascial matrix has regained adequate fluidity, focused shockwave therapy is applied directly to the MTrP core previously identified through pressure algometry.

This stage delivers the highest energy density of the treatment session, aiming to stimulate ATP resynthesis and reactivate SERCA function [7,12].

Biological dosing parameters

According to international recommendations [12], the energy flux density applied to muscle tissue should remain within the low-to-medium energy range of 0.05–0.15 mJ/mm2.

Technical rationale

Muscle tissue is highly innervated and mechanically sensitive. Direct application of high-energy shockwaves (>0.25 mJ/mm2) may induce microstructural damage in healthy fibers and trigger protective myotatic reflexes, causing muscle contraction that counteracts the intended therapeutic effects [16].

The 0.05–0.15 mJ/mm2 range provides an optimal therapeutic window, sufficient to activate mechanotransduction and enhance cellular permeability while minimizing tissue damage and reflex muscle contraction [12,15].

Phase 3: Drainage and tensegrity recalibration

The final phase aims to eliminate metabolic by-products mobilized during the molecular reset process [15].

Using low-pressure, high-frequency radial pressure waves, long decompressive sweeps are performed along the entire muscle belly and adjacent fascial structures [10]. This procedure restores intertissue gliding, stimulates superficial microlymphatic channels, and re-establishes physiological tensegrity throughout the musculoskeletal system [8,9], thereby reducing the likelihood of new ischemic foci developing.

Section V. Conclusion

1. Biological approach: The treatment of myofascial pain syndrome should move beyond temporary symptomatic analgesia and focus on restoring cellular metabolism and reoxygenating ischemic tissue [1,2].
2. Technological synergy: The sequential combination of focused shockwaves and radial pressure waves represents a new

methodological gold standard in biophysical therapy, enabling deep cellular ultrastructural repair while simultaneously optimizing the tissue microenvironment [15].

3. Movement and function: In chronic ischemic muscle disorders, prolonged rest perpetuates fascial stiffness. Consequently, biophysical interventions should be immediately complemented by progressive loading strategies and neuromuscular re-education [12,16].

4. Clinical judgment: The effectiveness and safety of advanced therapeutic technologies depend fundamentally on rigorous differential diagnosis, detailed anatomical knowledge, and the use of objective outcome measures, such as quantitative pressure algometry to guide evidence-based treatment decisions [4,13].

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given the consent for his/ her images and other clinical information to be reported in the journal. The patient understands that his/ her names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Conflict of interest: Nil **Source of support:** None

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