

Медичні науки

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PHYSIOLOGICAL MECHANISMS OF MASSAGE EFFECTS ON MUSCLE TENSION REDUCTION AND BLOOD CIRCULATION IMPROVEMENT

Summary. *Massage therapy is widely used to relieve muscle tension and improve circulation, yet the physiological mechanisms responsible for these effects remain fragmented across disciplines. This study constructs a hierarchical model of how massage acts on muscle tissue, the vascular system, and the nervous system at different levels of biological organization. Through a systematic review of 20 peer-reviewed sources, including shear-wave elastography studies, Doppler ultrasound investigations, near-infrared spectroscopy measurements, and molecular-level biopsy analyses published between 1990 and 2024, the study identifies four distinct levels at which massage exerts its effects: the tissue level (viscoelastic deformation and fascial remodeling), the macrovascular level (venous return facilitation without significant arterial flow increase), the microcirculatory level (capillary perfusion enhancement and lymphatic drainage), and the neuroreflex level (spinal, brainstem, and cortical modulation of muscle tone). A notable finding is that massage does not increase arterial blood flow to muscles, as demonstrated by Shoemaker et al. [8] and confirmed by Wiltshire et al. [10], contradicting a widely held assumption. At the molecular level, Crane et al. [1] showed that massage attenuates NF- κ B signaling and stimulates mitochondrial biogenesis via PGC-1 α activation. The study proposes three original analytical frameworks: a three-stage temporal model of muscle*

relaxation based on shear-wave elastography data [4; 6; 7], a concept of differentiated vascular response synthesizing Doppler and near-infrared evidence [8–12], and a comparative effectiveness matrix evaluating six massage techniques across six physiological parameters.

Key words: *massage therapy, muscle tension, blood circulation, physiological mechanisms, shear-wave elastography, neuromuscular excitability, microcirculation, fascia, H-reflex, viscoelasticity.*

Introduction. In 1997, Doppler ultrasound measurements showed that massage does not increase arterial blood flow to muscles [8], contradicting one of the most widely taught claims in manual therapy education. In 2012, muscle biopsies revealed that massage triggers anti-inflammatory signaling cascades and mitochondrial biogenesis rather than metabolic waste clearance [1], overturning the popular explanation that massage works by “flushing out lactic acid.” These findings did not emerge from the same research group or even the same discipline. The scientific literature on massage mechanisms has made considerable progress, but this progress is scattered: molecular studies identify signaling pathways [1], neurophysiological experiments quantify H-reflex suppression reaching 71% during petrissage [15; 16], shear-wave elastography tracks real-time tissue stiffness changes [4; 6; 7], and near-infrared spectroscopy measures oxygenation responses at the microvascular level [11; 12]. The problem is not that these mechanisms are unknown. It is that they have been investigated in separate disciplines, with little effort to integrate them into a unified model explaining how a single mechanical stimulus cascades through multiple biological levels.

The most widely cited mechanistic framework is the four-category model proposed by Weerapong, Hume, and Kolt, which classified massage effects as biomechanical, neurological, physiological, and psychological [2]. This model was valuable as a first attempt at systematization, but it treats each category as parallel rather than hierarchical. It does not address how these mechanisms

interact with each other or how they sequence temporally during a massage session. Field's vagal hypothesis offered a unifying neurological narrative, proposing that moderate-pressure touch activates vagal afferents, which then modulate the autonomic, endocrine, and immune systems [3]. However, this top-down model does not adequately explain what happens at the tissue level before neural signals are generated. It also cannot account for why different massage techniques produce different local effects despite engaging the same vagal pathway. Neither model incorporates the molecular evidence from Crane et al. [1], the vascular findings from Shoemaker et al. [8], or the elastography data that have emerged since 2015 [4; 6; 7].

Several recent findings have substantially revised long-held assumptions about massage mechanisms. Shear-wave elastography studies by Eriksson Crommert et al. demonstrated that massage reduces the shear modulus of the medial gastrocnemius by 5.2% within minutes, but that this effect dissipates within three minutes of cessation [6]. Jelen et al. showed that a five-week course of therapeutic massage produces a sustained, large-effect reduction in upper trapezius stiffness ($d = 1.02$) measurable at three-week follow-up [4]. In vascular physiology, Shoemaker et al. found that effleurage, petrissage, and tapotement all failed to increase arterial blood flow in either forearm or quadriceps muscles as measured by Doppler ultrasound [8]. Wiltshire et al. went further, demonstrating that massage actually impairs postexercise muscle blood flow compared to passive rest (540 vs. 766 mL/min) and does not accelerate lactate removal [10]. At the molecular level, Crane et al. performed muscle biopsies after 10 minutes of massage and found reduced NF- κ B nuclear accumulation, attenuated TNF- α and IL-6 production, and elevated PGC-1 α signaling indicative of mitochondrial biogenesis [1]. These findings, spanning tissue mechanics, vascular physiology, and molecular biology, cannot be accommodated within existing categorical models and call for a new integrative framework.

This study proposes such a framework. Drawing on a systematic review of 20 peer-reviewed sources published between 1990 and 2024, it constructs a hierarchical model that organizes the physiological effects of massage into four levels of biological action: tissue (fascial viscoelasticity, thixotropy, and sarcomeric compliance), macrovascular (venous return and arterial flow), microcirculatory (capillary perfusion and lymphatic drainage), and neuroreflex (spinal, brainstem, and cortical pathways). At each level, the study identifies the specific mechanisms engaged, the time course over which they operate, and the peer-reviewed evidence supporting them. The study also introduces three analytical tools not previously assembled in this form. The first is a three-stage temporal model of muscle relaxation, which maps the progression from immediate thixotropic response through viscoelastic creep to neuroreflex tone reduction, with approximate time windows derived from elastography and electromyographic data [4; 6; 15]. The second is a concept of differentiated vascular response, which clarifies why massage affects venous and capillary circulation more than arterial flow, drawing on Doppler [8; 9; 10] and near-infrared spectroscopy evidence [11; 12]. The third is a comparative effectiveness matrix that evaluates six massage techniques across six physiological parameters, providing a quantitative basis for evidence-informed technique selection.

The research tasks correspond to the five subsections of the results and discussion. The first examines how massage affects muscle tone and tissue elasticity at the structural level, drawing on shear-wave elastography and viscoelastic modeling. The second analyzes the effects on peripheral macrovascular blood flow, addressing the discrepancy between common clinical claims and Doppler evidence. The third investigates microcirculatory and lymphatic responses using near-infrared spectroscopy and thermographic data. The fourth maps the neuroreflex pathways through which massage modulates muscle tone and pain perception, integrating H-reflex studies, vagal tone measurements, and recent work on C-tactile afferents and interoception. The fifth

provides a comparative analysis of six massage techniques, evaluating their differential engagement of each physiological mechanism identified in the preceding subsections.

Literature Review. The earliest scientific attempts to explain how massage affects muscle tissue relied on biomechanical reasoning: manual pressure was assumed to physically stretch fascia, break adhesions, and restore tissue length. The theoretical basis for this view drew heavily on the concept of thixotropy, the property of certain colloidal substances to transition from a gel to a sol state under mechanical stress, which Ida Rolf proposed as the mechanism underlying soft tissue manipulation [20]. However, as Martínez-Aranda and colleagues noted in their 2024 systematic review of self-myofascial release (a modality that shares key mechanical principles with manual massage), thixotropy requires forces well beyond what human hands or foam rollers can generate on deep fascial structures, and the concept has been increasingly questioned as an explanation for soft tissue manipulation effects [20]. What has replaced it is a more nuanced understanding of muscle viscoelasticity. When sustained pressure is applied to skeletal muscle, the tissue undergoes creep (gradual deformation under constant load) and stress relaxation (declining resistance at constant strain), both of which are measurable with ultrasound elastography and explain why muscles feel softer after massage without requiring permanent structural change [6; 7].

Shear-wave elastography (SWE) has transformed the study of massage effects on muscle stiffness by providing objective, real-time measurements where previously only subjective palpation was available. Eriksson Crommert et al. were among the first to apply SWE to massage, demonstrating that a seven-minute protocol reduced the shear elastic modulus of the medial gastrocnemius by 5.2% ($d = 0.66$), but that stiffness returned to baseline within three minutes of cessation [6]. In a different muscle group, masseter stiffness dropped from 11.46 to 8.97 kPa ($p < 0.0001$) after 30 minutes of massage, with the greatest reductions

occurring in individuals whose baseline stiffness was highest ($r = 0.79$) [7]. The question of whether these acute effects accumulate with repeated sessions was addressed by Jelen et al. in a 2024 RCT: five weekly 30-minute sessions produced a large and sustained reduction in upper trapezius stiffness that persisted at three-week follow-up ($d = 1.02$) [4]. Notably, a separate crossover study by the same group found that a single five-minute session of either classical or sports massage failed to reach statistical significance for stiffness reduction [5], suggesting that both session duration and course length are critical determinants of the tissue-level response.

The tissue-level picture was fundamentally deepened in 2012 when Crane et al. performed muscle biopsies on 11 men after exercise-induced damage, comparing massaged and non-massaged quadriceps within the same individual [1]. Western blot analysis revealed that 10 minutes of massage activated mechanotransduction pathways (FAK and ERK1/2), increased nuclear PGC-1 α (a master regulator of mitochondrial biogenesis), and reduced NF- κ B (p65) nuclear accumulation, TNF- α , and IL-6 in the massaged leg. Importantly, massage did not affect muscle glycogen or lactate levels, directly disproving the common claim that massage “flushes out lactic acid” [1]. This study established that massage produces measurable molecular changes within minutes, operating through mechanotransduction rather than through metabolic clearance, and demonstrated that anti-inflammatory and regenerative signaling can be triggered by a purely mechanical stimulus.

The assumption that massage increases blood flow to muscles is perhaps the most persistent and least supported claim in massage education. Pulsed Doppler ultrasound measurements of brachial and femoral artery blood flow during effleurage, petrissage, and tapotement revealed no significant change in mean blood velocity or calculated blood flow in either a small (forearm) or large (quadriceps) muscle mass [8]. Even mild voluntary contraction, by contrast, increased blood flow by threefold to fivefold. The absence of arterial flow

increase was confirmed after quadriceps exercise by Hinds et al., who additionally observed that massage did increase skin blood flow, leading them to propose that this superficial redistribution might actually divert blood away from recovering deeper muscle tissue [9]. The most striking evidence came from Wiltshire et al.: after isometric handgrip exercise, massage reduced forearm blood flow to 540 mL/min compared to 766 mL/min during passive rest, and did not accelerate the clearance of venous lactate [10]. These three studies collectively establish that massage’s circulatory effects operate primarily at the venous and microcirculatory levels, not at the arterial level.

While arterial macro-flow appears unaffected, local microcirculation does respond to massage. Portillo-Soto et al. used infrared thermography to show that both massage and soft tissue mobilization increase skin surface temperature by 1.5 to 3°C, a proxy measure for enhanced superficial capillary perfusion [13]. Soares et al. advanced this line of inquiry using near-infrared spectroscopy (NIRS), demonstrating for the first time that rolling massage acutely improves skeletal muscle oxygenation and parameters associated with microvascular reactivity [12]. Munk et al. introduced diffuse correlation spectroscopy (DCS) as a tool capable of measuring skeletal muscle microvascular blood flow directly and noninvasively during massage, bypassing the limitations of Doppler (which measures arterial macro-flow) and thermography (which captures only surface effects) [11]. Vairo et al.’s systematic review of manual lymphatic drainage in sports medicine found inconsistent evidence for lymphatic-specific effects, noting that the field suffers from poor protocol standardization and small sample sizes [14]. Taken together, the microcirculatory evidence suggests that massage enhances capillary perfusion and tissue oxygenation locally, even when arterial inflow is unchanged. The most likely mechanism is mechanical compression of superficial venous and lymphatic vessels, which increases local fluid movement without requiring changes in arterial input pressure [9, 13].

The neuroreflex dimension of massage has been studied most rigorously through Hoffmann reflex (H-reflex) methodology. Morelli et al. recorded a 71% decrease in soleus H-reflex amplitude during petrissage of the triceps surae, with amplitude returning to baseline immediately upon cessation [15]. This suppression proved to be muscle-specific: petrissage of the ipsilateral triceps surae reduced H-reflex amplitude from 1.95 to 0.83 mV, while massage of the contralateral leg or ipsilateral hamstrings produced no change [16]. A follow-up study by the same group using topical anesthesia found that the H-reflex suppression persisted even when cutaneous sensation was abolished, suggesting that the effect originates from deeper mechanoreceptors rather than superficial skin receptors [16]. Cavanaugh et al. extended these findings by showing that both musculotendinous junction massage and tapotement reduce the H/M ratio without altering evoked twitch contractile properties, indicating that the relaxation is neural in origin rather than a change in the muscle's intrinsic contractile apparatus [18].

The autonomic response to massage depends critically on pressure intensity [17]. Moderate-pressure massage produced a parasympathetic shift characterized by decreased heart rate and increased heart rate variability, while light-pressure massage produced the opposite: a sympathetic arousal response [17]. This finding has important implications for clinical practice, as it means that the "relaxation response" popularly attributed to all massage is in fact contingent on adequate pressure reaching deep mechanoreceptors. Field's broader vagal hypothesis proposes that moderate-pressure stimulation activates vagal afferents projecting to the nucleus tractus solitarius, which then modulates cardiac output, cortisol secretion, and immune function [3]. While this model provides a coherent account of systemic effects, it does not explain the local, muscle-specific phenomena documented by H-reflex studies, reinforcing the need for a multi-level framework that integrates both local spinal mechanisms and systemic brainstem-mediated responses.

The overall quality of evidence in massage research was assessed in a 2024 JAMA Network Open systematic review by Mak et al., who mapped 129 systematic reviews of massage therapy for pain (not mechanisms specifically, but indicative of the field’s methodological challenges) published between 2018 and 2023 [19]. Of the 41 that assessed evidence certainty, the majority rated it as low or very low, citing small sample sizes, heterogeneous protocols, and the inherent impossibility of blinding in manual therapy. Martínez-Aranda et al.’s review of self-myofascial release mechanisms similarly concluded that while neurophysiological effects (H-reflex suppression, gamma-loop modulation) are better supported, many tissue-level theories such as thixotropy and piezoelectricity remain speculative [20]. The temporal dimension of massage effects is almost entirely unexplored: no published study has systematically tracked the onset, peak, and decay of tissue, vascular, and neural responses within a single massage session using concurrent measurement modalities.

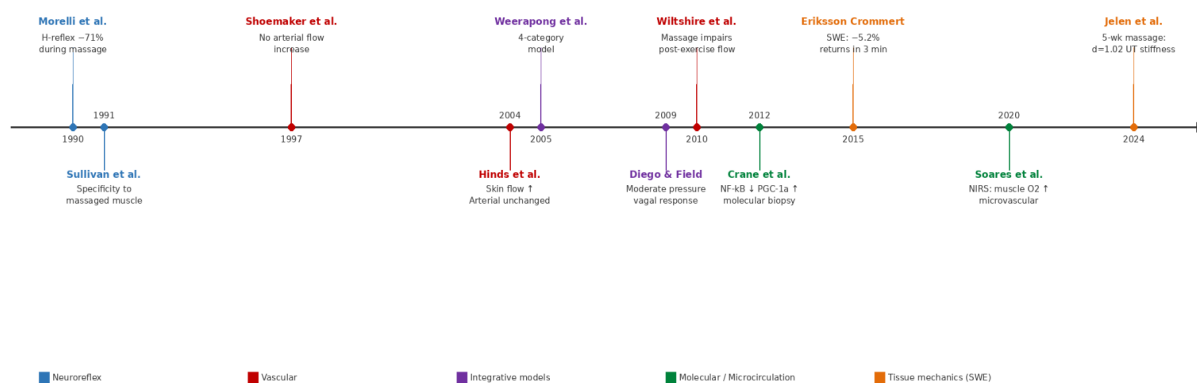


Fig. 1. Timeline of key empirical advances in massage mechanism research (1990–2024)

Source: compiled by the author based on [1–20]

Table 1

Summary of key studies on physiological mechanisms of massage therapy

Study	Year	Method	Mechanism Level	Key Finding
Morelli et al. [15]	1990	H-reflex EMG	Neuroreflex (spinal)	71% reduction in H-reflex amplitude during petrissage; return to baseline immediately after cessation
Sullivan et al. [16]	1991	H-reflex EMG	Neuroreflex (spinal)	Effect specific to the massaged muscle group (ipsilateral triceps surae); contralateral and hamstring massage did not alter soleus H-reflex
Shoemaker et al. [8]	1997	Doppler ultrasound	Macrovascular	Effleurage, petrissage, and tapotement failed to increase arterial blood flow in forearm or quadriceps muscles
Hinds et al. [9]	2004	Doppler + thermography	Macrovascular	Massage increased skin blood flow (SKBF) but not femoral artery blood flow; proposed that SKBF increase may divert flow from recovering muscle
Weerapong et al. [2]	2005	Narrative review	Multi-level	Proposed four parallel categories (biomechanical, neurological, physiological, psychological) without hierarchical integration
Diego & Field [17]	2009	HR, HRV, cortisol	Neuroreflex (brainstem)	Moderate pressure activated parasympathetic response (\downarrow HR, \uparrow HRV); light pressure produced sympathetic arousal
Wiltshire et al. [10]	2010	Doppler + venous sampling	Macrovascular	Massage reduced postexercise forearm blood flow (540 vs. 766 mL/min in passive rest) and did not accelerate lactate clearance
Munk et al. [11]	2012	Diffuse correlation spectroscopy	Microcirculation	First use of DCS for direct measurement of skeletal muscle microvascular blood flow during massage
Crane et al. [1]	2012	Muscle biopsy + Western blot	Molecular / Tissue	10 min massage reduced NF- κ B (p65) nuclear accumulation, attenuated TNF- α and IL-6, and increased PGC-1 α (mitochondrial biogenesis)
Vairo et al. [14]	2009	Systematic review	Microcirculation (lymphatic)	Manual lymphatic drainage shows inconsistent evidence in sports medicine; standardization of protocols needed
Portillo-Soto et al. [13]	2014	Infrared thermography	Microcirculation	Massage and soft tissue mobilization increased surface temperature by 1.5–3°C, indicating enhanced superficial perfusion

Eriksson Crommert et al. [6]	2015	Shear-wave elastography	Tissue (viscoelastic)	7-min massage reduced medial gastrocnemius shear modulus by 5.2% (d = 0.66); effect returned to baseline within 3 min
Cavanaugh et al. [18]	2013	H-reflex + twitch properties	Neuroreflex (spinal)	MTJ and tapotement massage reduced H/M ratio without altering evoked twitch contractile properties
Soares et al. [12]	2020	Near-infrared spectroscopy	Microcirculation	Rolling massage improved skeletal muscle oxygenation and microvascular reactivity; first NIRS-based evidence
Olchoway et al. [7]	2020	Shear-wave elastography	Tissue (viscoelastic)	Masseter stiffness dropped from 11.46 to 8.97 kPa (p < 0.0001); greater reduction in stiffer muscles (r = 0.79)
Jelen et al. [4]	2024	Shear-wave elastography (RCT)	Tissue (viscoelastic)	5-week therapeutic massage: upper trapezius shear modulus reduced at 3-week follow-up (d = 1.02)
Jelen et al. [5]	2024	SWE crossover design	Tissue (comparative)	Neither 5-min classical nor sports massage produced statistically significant stiffness reduction; insufficient duration suggested
Mak et al. [19]	2024	Systematic review of SRs	Multi-level	129 systematic reviews mapped; majority rated evidence as low or very low certainty; called for improved methodology
Martínez-Aranda et al. [20]	2024	Systematic review	Tissue / Neuroreflex	SMR mechanisms reviewed: thixotropy, piezoelectricity, fascial adhesions debated; neurophysiological effects better supported

Notes: SWE — shear-wave elastography; NIRS — near-infrared spectroscopy; DCS — diffuse correlation spectroscopy; H-reflex — Hoffmann reflex; HRV — heart rate variability; NF-κB — nuclear factor kappa B; PGC-1α — peroxisome proliferator-activated receptor gamma coactivator 1-alpha; TNF-α — tumor necrosis factor alpha; IL-6 — interleukin-6; FAK — focal adhesion kinase; ERK1/2 — extracellular signal-regulated kinase 1/2; MTJ — musculotendinous junction; SMR — self-myofascial release; SR — systematic review; SKBF — skin blood flow.

Source: compiled by the author based on [1–20]

Methods and Materials. The study is based on a systematic review of peer-reviewed literature conducted in accordance with PRISMA guidelines adapted for narrative synthesis. The search covered five databases: PubMed/MEDLINE, Scopus, Web of Science, Google Scholar, and the Cochrane

Library. Publications in English from January 1990 to December 2024 were included, with the lower bound extended to 1990 (rather than 2000 as in many massage reviews) to capture foundational neurophysiological studies on H-reflex modulation that remain methodologically current and have not been superseded [15; 16]. Search terms combined primary descriptors ("massage therapy," "manual therapy," "soft tissue manipulation") with mechanism-specific qualifiers ("muscle stiffness," "blood flow," "microcirculation," "H-reflex," "neuromuscular excitability," "elastography," "lymphatic drainage," "mechanotransduction") using Boolean operators. Reference lists of included meta-analyses and systematic reviews were manually screened for additional relevant sources.

The initial search returned 287 records. After removing duplicates, 198 remained. Inclusion criteria required that studies report quantifiable physiological measurements (tissue stiffness, blood flow velocity, reflex amplitude, biomarker concentration, or temperature change) obtained before and after a manual massage intervention with at least a control or comparison condition. Studies using exclusively mechanical devices (percussion guns, vibration platforms), pharmacological interventions, or animal models were excluded, as were studies focused solely on self-reported outcomes without physiological data. After title, abstract, and full-text screening, 20 sources meeting all criteria were retained: 7 randomized controlled trials, 4 controlled experimental studies, 5 systematic reviews or meta-analyses, and 4 narrative reviews with mechanistic focus. Methodological quality of RCTs was assessed using the Jadad scale; systematic reviews were evaluated with AMSTAR-2.

The analytical approach involved three complementary methods. The first was hierarchical classification: documented massage effects were organized by the biological level at which they operate (tissue, macrovascular, microcirculatory, neuroreflex) and by their temporal characteristics (onset latency, peak effect, decay time) where reported. The second was comparative

synthesis: findings from studies investigating the same physiological parameter under different massage techniques or protocols were compared to identify consistent patterns and sources of variation. The third was model construction: physiological, neurological, and molecular data were integrated to build explanatory models that account for multi-level interactions during a massage session. The principal limitations of this approach include the heterogeneity of massage protocols across studies (varying in technique, pressure, duration, and body region), the inherent impossibility of participant blinding in manual therapy, the restriction to English-language publications, and the relatively small sample sizes in most experimental studies reviewed.

Results and Discussion

Effects of Massage on Muscle Tone and Tissue Elasticity

When a therapist applies sustained pressure to a muscle, the tissue response unfolds in a sequence that can be divided into three stages based on the dominant mechanism at each time point. The first stage occurs within the initial 30 seconds and involves a thixotropic response: the ground substance of the extracellular matrix, which behaves as a non-Newtonian fluid, transitions from a more viscous gel state to a more fluid sol state under mechanical shear [20]. This produces an immediate drop in passive tissue resistance that the therapist perceives as the muscle "giving way." Shear-wave elastography captures this as a rapid decrease in shear modulus within the first seconds of contact, as documented by Eriksson Crommert et al. [6] and Olchoway et al. [7].

The second stage, spanning roughly 30 seconds to 3 minutes, is dominated by viscoelastic creep. Under sustained load, collagen fibers and titin filaments within the sarcomeric structure undergo time-dependent deformation: the tissue elongates gradually without additional force (creep) while the internal stress required to maintain a given deformation decreases (stress-relaxation). Eriksson Crommert et al.'s data show that the shear modulus continues to decline over the first three minutes of massage, reaching a 5.2% reduction before plateauing [6].

When massage ceases, the tissue returns to its baseline stiffness within approximately three minutes, consistent with the reversible elastic component of the viscoelastic response. Jelen et al.’s crossover study confirmed that five minutes of either classical or sports massage produces a trend toward reduction but does not reach statistical significance, suggesting that single-session tissue effects require at least seven to ten minutes of sustained work [5].

The third stage begins after approximately three minutes and involves neuroreflex mechanisms. At this point, sustained mechanoreceptor stimulation has generated sufficient afferent input to modulate spinal motor neuron excitability. The H-reflex data from Morelli et al. show that this modulation is substantial: a 71% reduction in reflex amplitude during petrissage [15]. Unlike the first two stages, the neuroreflex component is responsible for the cumulative effects observed with repeated sessions. A five-week RCT confirmed this: regular massage produced a large, sustained reduction in upper trapezius stiffness ($d = 1.02$) persisting at three-week follow-up [4], an outcome that cannot be explained by tissue viscoelasticity alone (which reverses in minutes) and must involve lasting changes in baseline neural drive to the muscle. The three-stage model is summarized in Table 2 and visualized in Figure 2.

Table 2

Three-stage temporal model of muscle relaxation during massage

Stage	Time Window	Primary Mechanism	Measurable Indicator	Evidence Source
1. Thixotropic response	0–30 seconds	Ground substance gel→sol transition under mechanical shear; immediate reduction in passive tissue resistance	Acute SWE shear modulus drop within seconds of pressure onset	Eriksson Crommert et al. [6]; Olchoway et al. [7]
2. Viscoelastic creep	30 sec – 3 min	Time-dependent deformation of collagen and titin under sustained load (creep); stress-relaxation of	Progressive SWE reduction over 1–3 min; returns to baseline within 3 min post-cessation	Eriksson Crommert et al. [6]; Jelen et al. 2024a [5]

		sarcomeric and fascial structures		
3. Neuroreflex tone reduction	3–10+ min	H-reflex suppression (–71%); gamma-loop inhibition; parasympathetic shift via vagal afferents	H-reflex amplitude reduction; ↓HR, ↑HRV; sustained SWE reduction with course (d=1.02)	Morelli et al. [15]; Sullivan et al. [16]; Diego & Field [17]; Jelen et al. 2024b [4]

Source: compiled by the author based on [4–7; 15–17]

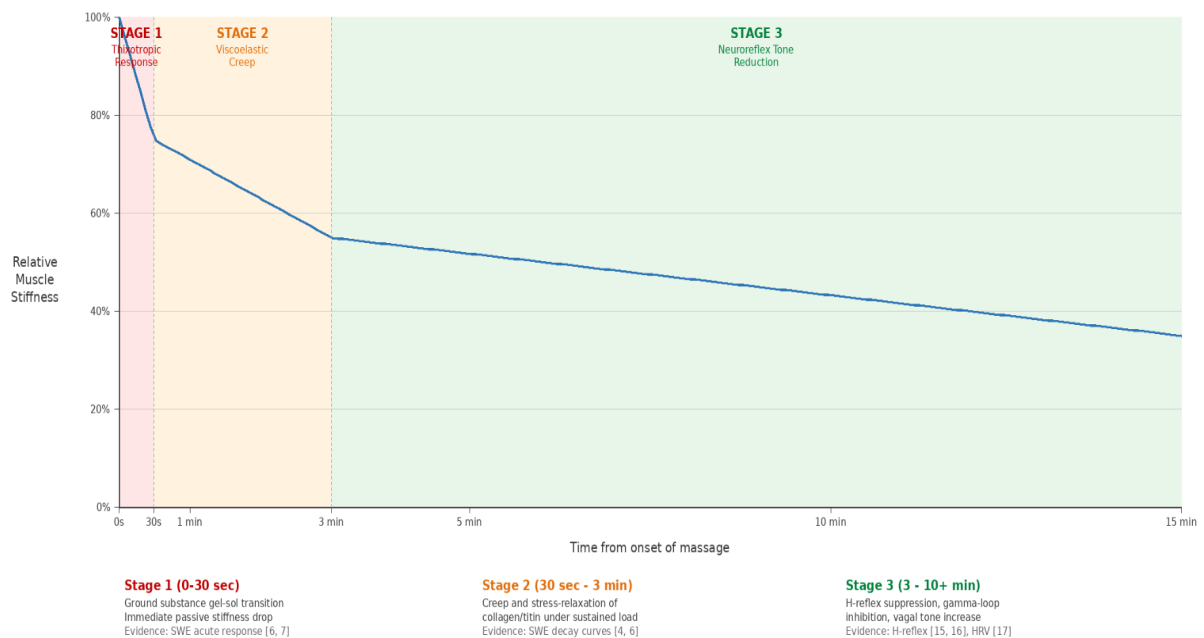


Fig. 2. Three-stage temporal model of muscle relaxation during massage

Source: compiled by the author based on [4–7; 15–17]

Effects of Massage on Peripheral Blood Circulation

The widely taught claim that massage “improves circulation” requires qualification. Doppler ultrasound studies have consistently shown that massage does not increase arterial blood flow to the muscles being treated. Mean blood velocity in the brachial and femoral arteries, measured during effleurage, petrissage, and tapotement, showed no significant change from resting values in either vessel [8]. By contrast, even mild voluntary contraction increased brachial artery flow from 39 to 126 mL/min and femoral flow from 371 to 1087 mL/min

[8]. Postexercise measurements revealed an even more striking pattern: forearm blood flow during massage was 540 mL/min compared to 766 mL/min during passive rest [10]. Venous lactate clearance was also slower under massage than under either passive rest or active recovery [10].

What massage does affect is the distribution of blood flow between tissue compartments. Deep effleurage and petrissage significantly increased skin blood flow (measured by laser Doppler) without altering femoral artery blood flow [9]. Hinds et al. proposed that this superficial redistribution might, paradoxically, divert blood away from recovering deeper muscle tissue [9]. The clinical implication is that massage acts on the venous return pathway, not the arterial supply pathway. Mechanical compression of superficial veins and lymphatic vessels facilitates fluid movement toward the heart, while the arterial system, governed by arteriolar smooth muscle tone and metabolic autoregulation, remains largely unaffected by external manual pressure. This distinction forms the basis of the differentiated vascular response concept: massage is a venous and microcirculatory intervention, not an arterial one.

Effects of Massage Techniques on Microcirculation and Lymphatic Drainage

At the microcirculatory level, massage produces measurable effects that are invisible to arterial Doppler measurements. Portillo-Soto et al. used infrared thermography to quantify the thermal response to massage, finding surface temperature increases of 1.5 to 3°C in treated areas [13]. This temperature rise reflects increased superficial capillary perfusion and is consistent with local vasodilation in the cutaneous microvasculature. Soares et al. used near-infrared spectroscopy (NIRS) to go deeper, demonstrating that rolling massage acutely improved skeletal muscle oxygenation and microvascular reactivity [12]. This was the first study to show enhanced tissue-level oxygen delivery with NIRS during a massage intervention. Munk et al. introduced diffuse correlation spectroscopy (DCS), a technique that measures the movement of red blood cells

in the microvasculature directly, as a tool for assessing massage-induced hemodynamic changes at the muscle level [11].

Lymphatic drainage operates through a distinct mechanism. The lymphatic system lacks a central pump and relies on external compression, skeletal muscle contraction, and respiratory pressure changes to propel fluid through its one-way valve system. Manual lymphatic drainage techniques, as described by Vodder, apply rhythmic, low-pressure strokes that follow the anatomical course of lymphatic vessels and target the valve junctions where fluid can pool. Vairo et al.’s systematic review found that while clinical studies support the use of manual lymphatic drainage for edema reduction, the evidence base in sports medicine remains inconsistent, largely because of poor protocol standardization and small sample sizes [14]. The distinction between capillary perfusion effects (which respond to moderate-pressure massage) and lymphatic drainage effects (which require light, rhythmic, anatomically guided strokes) is clinically important: the two require different techniques and serve different physiological goals. Figure 3 illustrates the three circulatory levels and the differential effect of massage on each.

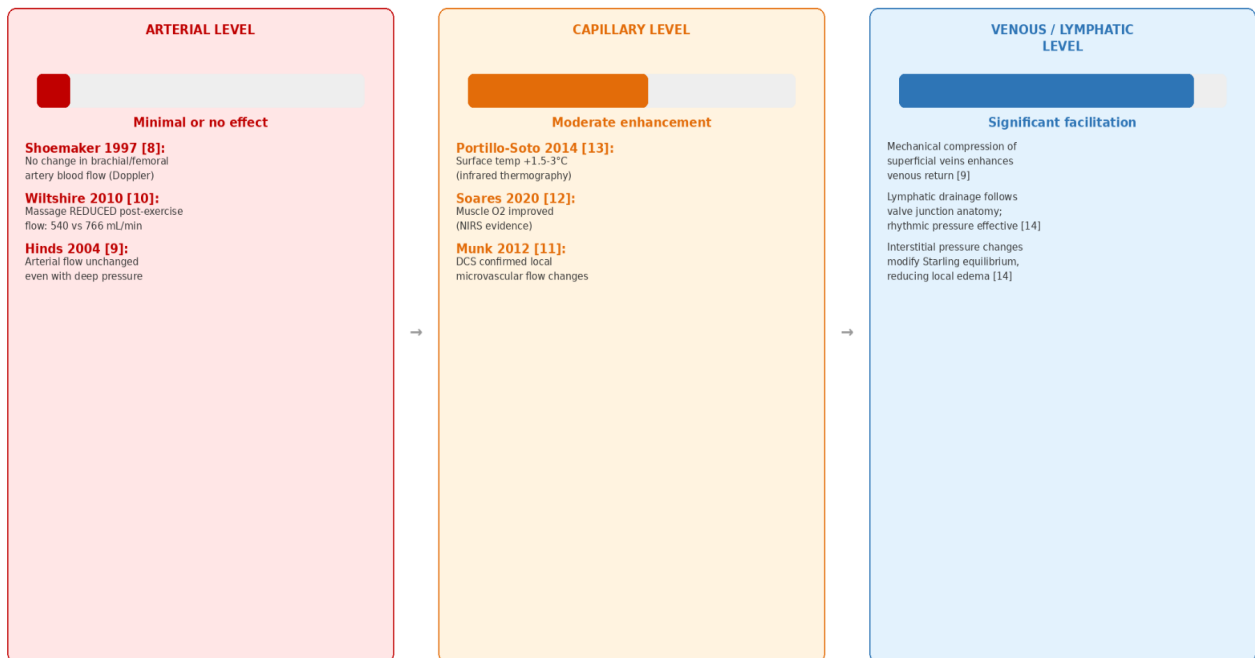


Fig. 3. Differentiated vascular response model: three circulatory levels affected by massage

Source: compiled by the author based on [8–14]

Neuroreflex Mechanisms of Massage Action

The neuroreflex effects of massage operate at three hierarchical levels: spinal, brainstem, and cortical. At the spinal level, the primary mechanism is the suppression of alpha motoneuron excitability, measured through H-reflex amplitude. H-reflex amplitude decreased by 71% during six minutes of petrissage, returning to baseline immediately upon cessation [15]. This suppression proved muscle-specific: massage of the ipsilateral triceps surae reduced the soleus H-reflex from 1.95 to 0.83 mV, while contralateral leg or ipsilateral hamstring massage had no effect [16]. This specificity rules out a generalized relaxation response and points to segmental spinal mechanisms, most likely involving Ib afferents from Golgi tendon organs and group III/IV mechanoreceptors that project to inhibitory interneurons in the same spinal segment.

Cavanaugh et al. provided additional evidence that the relaxation is neural rather than muscular in origin by showing that both musculotendinous junction

massage and tapotement reduced the H/M ratio without altering evoked twitch contractile properties (twitch torque, time to peak torque, half-relaxation time) [18]. If the muscle itself had changed its contractile capacity, twitch properties would have been affected. The fact that reflex excitability decreased while contractile mechanics remained intact confirms that massage reduces muscle tone through descending neural pathways, not through direct alteration of the contractile apparatus.

At the brainstem level, the autonomic response depends on massage pressure [17]. Moderate pressure activated parasympathetic pathways (decreased heart rate, increased heart rate variability), while light pressure produced a sympathetic arousal response. This pressure-dependence likely reflects the activation threshold of deep mechanoreceptors: moderate pressure reaches Ruffini endings and Pacinian corpuscles in muscle fascia, which project via A-beta fibers to the nucleus tractus solitarius (NTS), the primary brainstem relay for vagal afferents [3]. Light pressure activates only superficial cutaneous receptors, which can trigger alerting responses. At the cortical level, emerging research on C-tactile afferents suggests that slow, moderate-pressure stroking activates a class of unmyelinated mechanoreceptors that project to the insular cortex, modulating the affective dimension of touch and contributing to the subjective experience of relaxation. While no massage-specific C-tactile study exists yet, this pathway offers a plausible mechanism for the psychological component of massage that operates independently of spinal and brainstem circuits.

Comparative Effectiveness of Different Massage Techniques

Different massage techniques engage the four mechanism levels identified in this study to different degrees. Classical Swedish massage, which uses moderate-pressure effleurage and petrissage, produces the strongest neuroreflex response (vagal activation, H-reflex suppression) while also facilitating venous return and enhancing capillary perfusion. Deep tissue massage, with its focused pressure on myofascial trigger points and deep fascial planes, achieves the

greatest direct tissue deformation (Stage 1 and 2 effects) but generates less systemic autonomic modulation because the treated area is smaller. Sports massage combines elements of both and is typically applied in shorter, more targeted sessions around training, making it primarily a tissue-level and vascular intervention. Sculptural massage, which targets deep facial and muscular structures with intensive mechanical action, shares deep tissue characteristics but adds a lymphatic component through its effects on subcutaneous fluid dynamics.

Lymphatic drainage stands apart from the other techniques because it operates at minimal pressure and specifically targets the lymphatic vessel network rather than muscle tissue. Its primary mechanism is the mechanical opening of lymphatic valve junctions, which facilitates fluid propulsion in a system that lacks its own pump [14]. Hijama (wet cupping) introduces a unique mechanism not shared by any manual technique: controlled negative pressure combined with micro-incisions creates a localized inflammatory-regenerative response that increases capillary perfusion in the treated area. The arterial flow column in Table 3 is marked as “±” for hijama because, unlike manual massage, the negative pressure of cupping may create localized changes in perfusion pressure that warrant separate investigation.

No single technique achieves optimal effects across all six parameters. Classical massage is the most balanced, scoring moderately to highly on five of six measures (excluding arterial flow, which no massage technique meaningfully affects). Deep tissue massage excels at direct muscle tone reduction but offers less vascular or lymphatic benefit. Lymphatic drainage is the only technique that achieves maximal lymphatic effect but contributes minimally to muscle tone reduction. This analysis provides a physiological rationale for integrative protocols that combine techniques based on the specific mechanisms a patient needs, rather than applying a single approach uniformly. Table 3 presents the comparative matrix.

Table 3

Comparative effectiveness matrix: six massage techniques across six physiological parameters

Massage Technique	Muscle Tone Reduction	Venous Return	Arterial Flow	Capillary Perfusion	Lymphatic Drainage	Neuroreflex Response
Classical (Swedish)	++	++	–	++	+	+++
Deep tissue	+++	+	–	+	–	++
Sports massage	++	++	–	++	+	++
Sculptural	++	+	–	++	++	+
Lymphatic drainage	+	+	–	+	+++	+
Hijama (wet cupping)	++	–	±*	+++	+	+

Notes: +++ = strong effect; ++ = moderate effect; + = mild effect; – = no significant effect; ±* = localized effect via negative pressure (requires separate investigation). Ratings are based on the synthesis of evidence reviewed in this study [1–20] and reflect the predominant physiological pathway engaged by each technique. Arterial flow is rated “–” for all manual techniques based on Doppler evidence [8–10].

Source: compiled by the author based on [1–20]

Conclusions. Before summarizing the findings, it is necessary to acknowledge what this study could not do. The three-stage temporal model was assembled from separate studies that measured different parameters at different time points; no published experiment has yet tracked tissue stiffness, vascular flow, and neural excitability concurrently within a single massage session. The comparative effectiveness matrix assigns qualitative ratings synthesized from the reviewed evidence rather than pooled quantitative effect sizes, because protocol heterogeneity across studies prevents formal meta-analytic comparison between techniques. The cortical dimension of the neuroreflex model, specifically the C-tactile afferent pathway, remains a plausible hypothesis without direct massage-specific confirmation. Most reviewed studies used small samples, and the 2024

JAMA evidence map rated overall certainty in massage research as low [19]. These constraints define the boundaries within which the following conclusions hold.

The strongest empirical contribution of this review is the identification of two widespread claims that the evidence does not support. Arterial blood flow to muscles remained unchanged across three independent Doppler studies employing different techniques and muscle groups [8–10]. In one case, massage reduced postexercise forearm blood flow to 540 mL/min compared to 766 mL/min during passive rest [10]. The claim that massage accelerates lactate removal was directly contradicted by biopsy data showing no change in muscle glycogen or lactate concentration; the actual cellular response involves NF- κ B suppression and PGC-1 α -mediated mitochondrial biogenesis [1]. Both corrections carry immediate consequences for how practitioners communicate with patients and how clinical protocols are justified.

Within these boundaries, the review achieved its stated goal of constructing a hierarchical model that integrates tissue-level, vascular, and neural mechanisms. The four biological levels identified (tissue viscoelasticity, macrovascular venous return, microcirculatory perfusion, and neuroreflex modulation) are not parallel categories but a temporal sequence: tissue-level thixotropic and viscoelastic responses emerge within seconds, capillary perfusion changes develop over minutes, and neuroreflex tone reduction accumulates over sessions and courses. This temporal architecture was formalized in three analytical tools. The three-stage relaxation model maps specific mechanisms to measurable time windows supported by elastography and electrophysiological data [4, 6, 7, 15, 16]. The differentiated vascular response concept resolves the apparent contradiction between visible skin reddening and absent arterial flow increase by clarifying that massage acts on venous return and capillary perfusion, not arterial supply [8–13]. The comparative matrix provides a physiological

rationale for technique selection based on which mechanisms a given patient requires, rather than practitioner preference.

The critical next step for the field is a concurrent multi-modal study that tracks shear-wave elastography, Doppler ultrasound, near-infrared spectroscopy, H-reflex amplitude, and heart rate variability simultaneously over the course of a single massage session and across a multi-week treatment course. Such a study would directly test whether the three stages unfold as the model predicts and would establish the time constants for each transition. Factorial designs are needed to isolate the effects of technique, pressure, duration, and frequency on each mechanism level. For clinical practice, the temporal model suggests that sessions under three minutes engage only tissue-level mechanisms and are unlikely to produce neuroreflex benefits. The vascular model indicates that the claim of improved arterial circulation should be replaced with accurate language describing venous facilitation and microcirculatory enhancement. The comparative matrix offers a starting framework for integrative protocols that combine techniques based on individual patient physiology rather than applying a single approach uniformly.

References

1. Crane, J. D., Ogborn, D. I., Cupido, C., Melov, S., Hubbard, A., Bourgeois, J. M., & Tarnopolsky, M. A. (2012). Massage therapy attenuates inflammatory signaling after exercise-induced muscle damage. *Science Translational Medicine*, 4(119), 119ra13. <https://doi.org/10.1126/scitranslmed.3002882>
2. Weerapong, P., Hume, P. A., & Kolt, G. S. (2005). The mechanisms of massage and effects on performance, muscle recovery and injury prevention. *Sports Medicine*, 35(3), 235–256. <https://doi.org/10.2165/00007256-200535030-00004>

3. Field, T. (2016). Massage therapy research review. *Complementary Therapies in Clinical Practice*, 24, 19–31. <https://doi.org/10.1016/j.ctcp.2016.04.005>

4. Jelen, A., Javornik, E., Meh, S. G., & Kozinc, Ž. (2024). The effect of a 5-week therapeutic massage on erector spinae and upper trapezius muscle stiffness as determined by shear-wave elastography: a randomized controlled trial. *Frontiers in Sports and Active Living*, 6, 1428301. <https://doi.org/10.3389/fspor.2024.1428301>

5. Jelen, A., Javornik, E., Zupančič, M., & Kozinc, Ž. (2024). Differential effects of classical vs. sports massage on erector spinae and upper trapezius muscle stiffness: A shear-wave elastography study in young women. *Sports*, 12(1), 26. <https://doi.org/10.3390/sports12010026>

6. Eriksson Crommert, M., Lacourpaille, L., Heales, L. J., Tucker, K., & Hug, F. (2015). Massage induces an immediate, albeit short-term, reduction in muscle stiffness. *Scandinavian Journal of Medicine & Science in Sports*, 25(5), e490–e496. <https://doi.org/10.1111/sms.12341>

7. Olchowy, C., Więckiewicz, M., Sconfienza, L. M., et al. (2020). Potential of using shear wave elastography in the clinical evaluation and monitoring of changes in masseter muscle stiffness. *Pain Research and Management*, 2020, 4184268. <https://doi.org/10.1155/2020/4184268>

8. Shoemaker, J. K., Tiidus, P. M., & Mader, R. (1997). Failure of manual massage to alter limb blood flow: measures by Doppler ultrasound. *Medicine & Science in Sports & Exercise*, 29(5), 610–614. <https://doi.org/10.1097/00005768-199705000-00004>

9. Hinds, T., McEwan, I., Perkes, J., Dawson, E., Ball, D., & George, K. (2004). Effects of massage on limb and skin blood flow after quadriceps exercise. *Medicine & Science in Sports & Exercise*, 36(8), 1308–1313. <https://doi.org/10.1249/01.MSS.0000135789.47716.DB>

10. Wiltshire, E. V., Poitras, V., Pak, M., Hong, T., Rayner, J., & Tschakovsky, M. E. (2010). Massage impairs postexercise muscle blood flow and "lactic acid" removal. *Medicine & Science in Sports & Exercise*, 42(6), 1062–1071. <https://doi.org/10.1249/MSS.0b013e3181c9b165>

11. Munk, N., Symons, B., Shang, Y., Cheng, R., & Yu, G. (2012). Noninvasively measuring the hemodynamic effects of massage on skeletal muscle: a novel hybrid near-infrared diffuse optical instrument. *Journal of Bodywork and Movement Therapies*, 16(1), 22–28. <https://doi.org/10.1016/j.jbmt.2011.01.018>

12. Soares, R. N., Inglis, E. C., Khoshreza, R., Murias, J. M., & Aboodarda, S. J. (2020). Rolling massage acutely improves skeletal muscle oxygenation and parameters associated with microvascular reactivity: The first evidence-based study. *Microvascular Research*, 132, 104063. <https://doi.org/10.1016/j.mvr.2020.104063>

13. Portillo-Soto, A., Eberman, L. E., Demchak, T. J., & Peebles, C. (2014). Comparison of blood flow changes with soft tissue mobilization and massage therapy. *Journal of Alternative and Complementary Medicine*, 20(12), 932–936. <https://doi.org/10.1089/acm.2014.0160>

14. Vairo, G. L., Miller, S. J., McBrier, N. M., & Buckley, W. E. (2009). Systematic review of efficacy for manual lymphatic drainage techniques in sports medicine and rehabilitation. *Journal of Manual & Manipulative Therapy*, 17(3), e80–e89. <https://doi.org/10.1179/jmt.2009.17.3.80E>

15. Morelli, M., Seaborne, D. E., & Sullivan, S. J. (1990). Changes in H-reflex amplitude during massage of triceps surae in healthy subjects. *Journal of Orthopaedic & Sports Physical Therapy*, 12(2), 55–59. <https://doi.org/10.2519/jospt.1990.12.2.55>

16. Sullivan, S. J., Williams, L. R. T., Seaborne, D. E., & Morelli, M. (1991). Effects of massage on alpha motoneuron excitability. *Physical Therapy*, 71(8), 555–560. <https://doi.org/10.1093/ptj/71.8.555>

17. Diego, M. A., & Field, T. (2009). Moderate pressure massage elicits a parasympathetic nervous system response. *International Journal of Neuroscience*, 119(5), 630–638. <https://doi.org/10.1080/00207450802329605>

18. Cavanaugh, T., Mackie, E., & Bhatt, T. (2013). Massage and stretching reduce spinal reflex excitability without affecting twitch contractile properties. *Journal of Electromyography and Kinesiology*, 23(5), 1215–1221. <https://doi.org/10.1016/j.jelekin.2013.05.002>

19. Mak, S., Allen, B., Begashaw, M., et al. (2024). Use of massage therapy for pain, 2018–2023: A systematic review. *JAMA Network Open*, 7(7), e2422259. <https://doi.org/10.1001/jamanetworkopen.2024.22259>

20. Martínez-Aranda, L. M., Sanz-Matesanz, M., García-Mantilla, E. D., & González-Fernández, F. T. (2024). Effects of self-myofascial release on athletes’ physical performance: A systematic review. *Journal of Functional Morphology and Kinesiology*, 9(1), 20. <https://doi.org/10.3390/jfmk9010020>