Review Article

Physiological Mechanisms of the Primary Respiratory Mechanism (PRM) and Cranial Rhythmic Impulse (CRI) in Osteopathy: A Narrative Review

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Cranial Rhythmic Impulse (CRI), also referred to as the Primary Respiratory Mechanism (PRM), describes a rhythm perceived by some osteopaths at the surface of the body and used in clinical practice for diagnostic and therapeutic purposes. Despite its widespread use, the physiological basis of this phenomenon remains unclear.

The theoretical model initially proposed by William Garner Sutherland in 1939 has historically shaped the understanding of PRM/CRI. However, advances in physiology and neuroscience have challenged several of its fundamental assumptions. Since the 1990s, alternative hypotheses have emerged, including those highlighted in recent systematic reviews.

In this narrative review, we first re-examine the five classical pillars of Sutherland's model in light of contemporary scientific evidence. After demonstrating the limitations of this framework, we explore a range of physiological mechanisms that may contribute to the PRM/CRI phenomenon, including vasomotion, autonomic nervous system activity, heart rate variability, microcirculation, lymphatic dynamics, and extracellular matrix behavior.

The most plausible interpretation is that PRM/CRI represents an emergent phenomenon arising from the interaction of multiple physiological oscillations, particularly those driven by autonomic regulation, such as Traube–Hering–Mayer waves, respiratory rhythms, and cardiovascular variability. These interactions may induce rhythmic changes in the viscoelastic properties of the extracellular matrix and fascial network.

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Finally, we discuss the clinical relevance of PRM in osteopathic practice. While PRM/CRI may provide useful palpatory feedback, its interpretation should be approached with caution given the influence of multiple physiological and perceptual factors.
This review highlights the need for a paradigm shift in the understanding of PRM/CRI, advocating for a model grounded in contemporary scientific knowledge and for clearer communication within the osteopathic community.

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Abbreviations

- ABP Alpha-band power
- ANS Autonomic Nervous System
- BP blood pressure
- CRI Cranial rhythmic impulse
- CSF Cerebro Spinal Fluid
- ECG electrocardiogram
- EEG Electroencephalogram
- HF High frequency
- IM Intermediate band
- LF Low-frequency
- MRI Magnetic Resonance Imaging
- NMR Nuclear magnetic resonance
- PRM Primary Respiratory Mechanism
- SNS Sympathetic nervous system
- THM Traube Hering and Mayer
- OCF Osteopathy in the Cranial Field
- OMT Osteopathic Manipulative Treatment

I. Introduction

The term *Primary Respiratory Mechanism* (PRM) is commonly used by some osteopaths to describe the palpatory perception of a physiological rhythm at the surface of the skin [1]. It is used both for diagnostic

and therapeutic purposes. This rhythm is typically described as having two phases: an expansion phase (inhalation) and a contraction phase (exhalation) [2][3]. Unlike pulmonary respiration, it remains perceptible even when the patient holds their breath [4].

Historically, the osteopath who first described this phenomenon, William Garner Sutherland (1873-1954), proposed that cranial sutures were designed to allow movement. Based on this idea, he developed a model structured around five principles: the inherent mobility of the central nervous system and spinal cord; the fluctuation of cerebrospinal fluid (CSF); the reciprocal tension membranes; the articular mobility of cranial bones; and the involuntary movement of the sacrum between the iliac bones [2][3].

Sutherland introduced the term “Primary Respiratory Mechanism” to emphasize several key aspects of his concept. The word *mechanism* reflects a coordinated physiological process involving multiple interacting components. The term *respiratory* was chosen by analogy with pulmonary breathing, although it refers to a distinct phenomenon. For Sutherland, the PRM represented an autonomous rhythmic pulsation within the body, essential to life and present even in the absence of lung respiration [2]. He proposed that the body possesses an intrinsic periodic motion, independent of the lungs, which animates both tissues and fluids. The term *primary* suggests that this mechanism originates early in embryonic life and represents a fundamental biological function. Today, many osteopaths continue to use the term PRM to describe the micromovements they work with.

However, to remain precise and faithful to its original meaning, the term *Cranial Rhythmic Impulse* (CRI) should be used when referring specifically to the palpatory perception of this rhythm at the skin surface. This terminology was introduced in 1961 by Woods and Woods, who described a rhythm perceived at the surface of the head. They linked this finding to Sutherland’s PRM and attempted, seven years after his death, to define a normal frequency range—something Sutherland himself had never established [5].

For some osteopaths, CRI and PRM represent the same phenomenon, perceived respectively at the cranial level and throughout the rest of the body. The distinction between the two terms is often blurred [6]. However, this distinction is not essential, as CRI is generally considered to originate from PRM. The frequent conflation of these terms can therefore be seen as a simplification. PRM remains the historical explanatory model for this rhythm, which is the focus of the present review [7][8].

The five components underlying PRM were first described by Sutherland in *The Cranial Bowl* (1939) and later widely disseminated by his student Harold Magoun, who published *Osteopathy in the Cranial Field* in 1951. Magoun sought to systematize Sutherland’s teachings, making them more accessible and

reproducible by formalizing techniques and clinical protocols [2][3]. However, Sutherland himself expressed reservations about this biomechanical formalization, as he feared it might limit a deeper understanding of his approach. While Sutherland viewed cranial motion as a complex, holistic process influenced by multiple factors, Magoun's efforts to popularize these concepts risked reducing this complexity to a predefined set of movements and techniques. This divergence created tension, as Sutherland feared that the essence of his work might be lost [2][3].

Nevertheless, this remains the legacy of cranial osteopathy today: practitioners perceive a rhythm that is assumed to be associated with cranial bone motion and related phenomena. It is precisely this assumption that the present review aims to reassess.

For clarity, we will use the terms PRM and CRI interchangeably, in line with common usage, to refer to the rhythm typically perceived by osteopaths.

PRM/CRI is classically described through three parameters: amplitude, rhythm, and strength. When these parameters are considered normal, the osteopath may conclude that the patient—or at least the area being examined—is well balanced. Some practitioners use PRM/CRI as a feedback tool to assess treatment effectiveness, evaluating it after each technique until it reaches a satisfactory state, which may signal the end of the session. In this context, PRM/CRI is thought to reflect the body's overall harmony and vitality.

A normal amplitude is expected to be large and symmetrical. Strength is considered adequate when it is perceived as clear and consistent. The rhythm is generally described as normal between 6 and 12 cycles per minute (cpm), although the literature reports a much wider range, from 3 to 22 cpm [9]. As illustrated in Figure 1, these frequencies are highly variable and cannot easily be attributed to a single identified physiological rhythm. The perceived rhythm may vary depending on both practitioner and patient, as well as over time [10][11][12][13][14][15], which complicates objective measurement and may explain the difficulty in achieving intra- and inter-examiner reproducibility. Furthermore, different studies may describe distinct rhythms under the same terminology.

Interestingly, rhythms within the PRM frequency range have also been recorded using instrumentation, suggesting that the phenomenon may have an objective basis [10]. Rasmussen and Meulengracht [4], for example, identified a third physical rhythm—distinct from cardiac and respiratory rhythms—on the human head, with a mean frequency of 6.16 cpm (range: 4.25-7.07). However, although their measurement

device was metrologically validated, it is subject to significant limitations. As with earlier work by Frymann (1971), head movements induced by respiration cannot be fully controlled [16].

Other studies have reported correlations between CRI palpation and objective physiological parameters. Nelson et al. [17][18][19][20], and more recently Pelz et al. [9], demonstrated statistically significant relationships between CRI perception and skin blood flow measurements, suggesting that cranial manipulations may influence measurable physiological variables.

Given these findings, and considering the existence of studies suggesting that PRM/CRI may be both perceptible and measurable, we recently conducted a structured systematic review of the literature (MEDLINE, ScienceDirect, Cochrane Library) to explore the physiological mechanisms that could underlie this rhythm [1]. Both this review and the data presented in Figure 1 confirm that the physiological basis of PRM/CRI remains controversial.

The model originally proposed by Sutherland is no longer consistent with current physiological knowledge, and the mechanisms underlying this phenomenon remain unclear. Our previous work highlighted the need for a paradigm shift, as well as for more rigorous evaluation and clearer communication of models aligned with contemporary scientific data.

This led to the present narrative review, which integrates findings from a broader range of life sciences. After first demonstrating the incompatibility of the Sutherland model with current knowledge, we explore the potential roles of vasomotion, the autonomic nervous system, microcirculation, the lymphatic system, the extracellular matrix, the fascial network, and entrainment in the genesis of PRM/CRI. Based on this analysis, we propose an integrative model and outline the need for a conceptual paradigm shift.

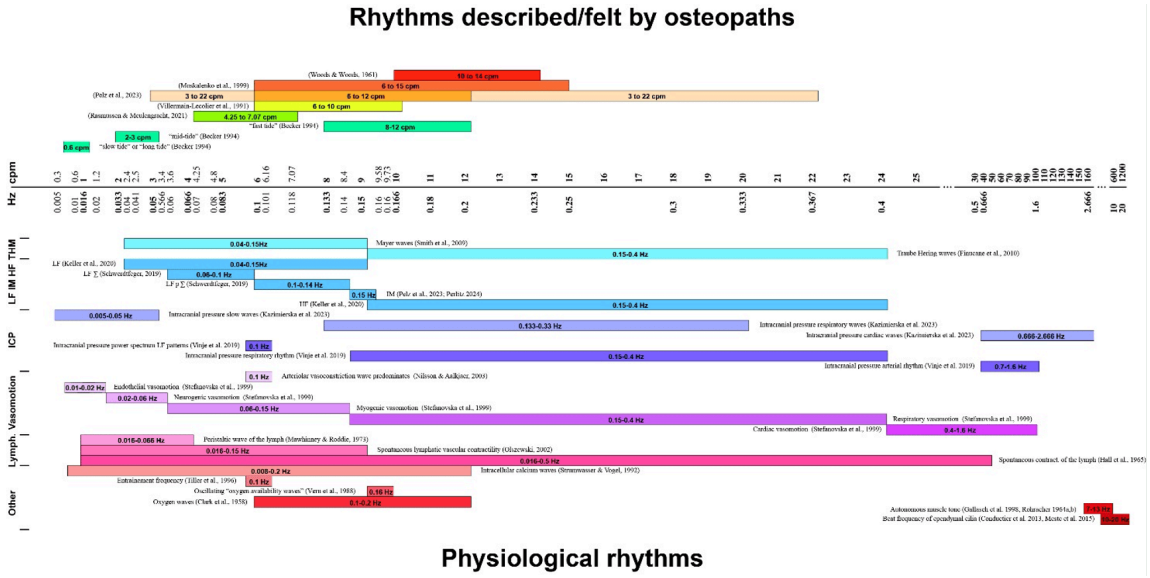

Figure 1. The following presentation will outline the various rhythms and frequencies (expressed in Hz and cpm) described by osteopaths, in comparison to those of physiological rhythms. These rhythms will be grouped into the following categories: Traube Hering and Mayer waves (THM); Low Frequency (LF); Intermediate Frequency (IM); High Frequency (HF); Intracranial Pressure Wave (ICP); Vasomotion; Spontaneous Lymphatic Contractility (Lymph.) and others. For Pelz et al.[8], the frequency range between 3 and 22 Hz represents the total range of measurements, the more restricted ranges between 6 to12 Hz represent the range of the most frequently found values.

II. Methods

This review was conducted in accordance with the guidelines proposed by Tricco et al. for narrative reviews, following the PRISMA-ScR Statement [21]. The theoretical framework for this scoping review was developed by a team of three experts (F.M., L.S., A.G.) with complementary expertise in scientific research (L.S., A.G.) and in the teaching and clinical practice of osteopathy (F.M., L.S.). This framework is based on the results and conclusions of the study by Mériaux et al. [1].

Research question

What scientific evidence is currently available to determine whether contemporary research supports or challenges the empirical PRM model described by Sutherland [2][3]?

Research strategy

A systematic literature search was completed in October 2023 using the indexed electronic databases MEDLINE, ScienceDirect, and the Cochrane Library [1]. This initial search identified eight main areas of research that could provide complementary physiological explanations for CRI/PRM beyond Sutherland's five pillars: lymphatic system; vasomotion; Traube–Hering and Mayer waves; heart rate variability; ventilation frequency; extracellular matrix; metabolic hypotheses; and embryology.

An extended and comprehensive literature search was then conducted between September 2024 and February 2025 using PubMed, ScienceDirect, Ostmed. DR, and Google Scholar. Search terms were adapted to each database and included: *cranial rhythmic impulse; primary respiratory mechanism; Traube–Hering waves; Mayer waves; cranial osteopathy; osteopathy in the cranial field; craniosacral therapy; cranial osteopathic manipulative medicine; heart rate variability; cranial bone mobility; central link; vasomotion; autonomic nervous system; ventilation; muscle tone; microcirculation; lymphatic system; glymphatic system; extracellular matrix; entrainment; embryological movement; ideomotion; intracranial pressure*.

No restrictions were applied regarding study design, population, outcomes, or publication date. Articles were first screened based on their abstracts to assess relevance, followed by full-text analysis. Reference lists of selected articles were also examined, and a snowballing approach was used to identify additional relevant studies.

Eligibility criteria

To ensure broad coverage of the available literature, no formal quality assessment of the included studies was performed. Instead, each article was critically appraised for its relevance to the research question during expert discussions among the authors (F.M., L.S., A.G.).

The selected studies were analyzed to identify physiological mechanisms that could either support or challenge the PRM model. When appropriate, individual studies were included in multiple thematic categories.

Data synthesis and categorization

A total of 157 articles were included and organized according to their relevance within the following thematic areas:

- The inherent motility of the central nervous system and spinal cord

- Fluctuation of the cerebrospinal fluid
- Motility of intracranial and intraspinal membranes
- Joint mobility of cranial bones
- The involuntary movement of the sacrum between the iliac bones (“core link” hypothesis)
- CRI/PRM and vasomotion
- CRI/PRM and the autonomic nervous system (including Traube–Hering–Mayer waves and heart rate variability)
- PRM and microcirculation, lymphatic system, extracellular matrix, and fascial network
- PRM and entrainment
- PRM and embryological movement

Articles were primarily identified through a snowballing process, expanding upon those included in the previous systematic review by Mériaux et al. [1], as well as through the authors’ domain expertise and reference tracking.

Interpretation framework

The analysis suggests that the five pillars described by Sutherland and later formalised by Magoun may represent an empirical model that is not fully supported by current scientific evidence. However, the literature identifies several physiological rhythms—particularly vasomotion, heart rate variability, microcirculation, and Traube–Hering–Mayer waves—that may contribute to the development of a more scientifically grounded explanatory model.

III. Results: Reappraisal of Sutherland’s Theory

Sutherland’s model is deeply rooted in the osteopathic literature. However, in this section, we demonstrate that it is not consistent with current scientific knowledge.

III.1. Inherent motility of the central nervous system and spinal cord

Since 1882, several anatomists have described continuous, cyclic movements of the brain and medulla, corresponding to systolic and diastolic phases. Sutherland interpreted these movements as a hydraulic mechanism capable of driving cerebrospinal fluid (CSF) circulation [12].

Later, Harold Magoun [2], followed by John Upledger [15], proposed an alternative interpretation. They suggested that rhythmic CSF production by the choroid plexus drives brain movement, rather than the reverse.

According to Upledger's "pressurestat" model, CSF secretion would occur at a rate twice that of reabsorption. Mechanoreceptors located in cranial sutures were hypothesized to detect a maximal pressure threshold and, via a feedback mechanism, inhibit secretion until pressure decreased to a minimum, after which secretion would resume, generating a cyclical process.

However, this model is not supported by current evidence. CSF production is neither intermittent nor rhythmic [10]. Instead, increases in intracranial pressure are accompanied by a simultaneous increase in CSF reabsorption, mediated by an expansion of the exchange surface of arachnoid villi within the venous sinuses. This concept is described by the Monro–Kellie doctrine [22][23].

Under physiological conditions, intracranial volume remains in dynamic equilibrium: increases in one component (brain tissue, blood, or CSF) are compensated by decreases in another. However, this compensation is not instantaneous, and transient variations generate intracranial pressure (ICP) waveforms [24].

These ICP variations are clinically relevant and are routinely monitored in conditions such as head trauma and hydrocephalus. Their components can be detected non-invasively at the scalp using mechanical sensors, which measure beat-to-beat micrometric deformations of the skull [25][26][27]. Early work by Pitlyk et al. (1985) demonstrated that these minute cranial expansions reflect changes in intracranial pressure [28].

In the time domain, the ICP signal consists of overlapping components. In the frequency domain, however, they can be distinguished as:

- slow waves (0.3-3 cpm),
- respiratory waves (8-20 cpm),
- cardiac waves (40-160 cpm) [29][24].

Vinje et al. [30] measured intracranial pressure gradients using dual transducers and identified two dominant frequency peaks: one corresponding to arterial pulsations (0.7-1.6 Hz; 42-96 bpm) and the other to respiratory activity (0.15-0.4 Hz; 9-24 breaths per minute). Interestingly, they also reported low-

frequency oscillations below 0.1 Hz (~6 cpm), although their contribution to CSF flow was not assessed in this study [30].

Using magnetic resonance imaging (MRI), Maier et al. [13] observed movements of both brain tissue and CSF that were closely linked to cardiac and respiratory activity. During systole, the brain and medulla move caudally and medially (2-3 mm), with the opposite movement occurring during diastole [11][14]. During inspiration, the central nervous system is displaced cranially while the brain retracts; during expiration, the predominant movement is caudal, accompanied by brain expansion [31].

These findings indicate that neuraxis motion is primarily driven by cardiorespiratory dynamics, with a frequency close to respiratory rate [32], which is slightly faster than the PRM rhythm. Overall, these observations suggest that PRM is not directly related to intrinsic brain motion or respiration.

III.2. Fluctuation of the cerebrospinal fluid (CSF)

Cerebrospinal fluid (CSF) is a clear biological fluid that surrounds the brain and spinal cord within the subarachnoid space, between the pia mater and arachnoid. It plays a protective role and is involved in the transport of nutrients and metabolic waste. CSF is primarily produced by filtration of blood at the choroid plexus within the cerebral ventricles [33].

As a fluid, CSF is poorly compressible. Early theories proposed that its pulsatile circulation could generate forces sufficient to move cranial membranes and bones, thereby producing the PRM/CRI rhythm. However, CSF flow is now known to be primarily driven by the cardiac cycle and is therefore pulsatile rather than intrinsically rhythmic.

CSF dynamics are also influenced by respiration, posture, jugular venous pressure, arterial pressure, and physical activity [34][35][30]. Arterial wall motion plays a key role in driving CSF movement through a mechanism known as perivascular pumping. As a result, CSF flow velocity closely follows arterial wall motion.

The total CSF volume is approximately 150 mL and is renewed three to four times per day (around 500 mL produced daily) [36]. CSF circulates from the ventricles into the subarachnoid space and is then reabsorbed primarily at the cranial level via venous and lymphatic pathways.

Current evidence indicates that the venous system accounts for approximately 30% of CSF drainage, while the nasal lymphatic system plays a major role, accounting for about 40% of outflow via pathways along cranial nerves, particularly at the cribriform plate of the ethmoid bone [37]. Approximately 25% is

drained at the spinal level via arachnoid villi associated with spinal veins. A small fraction diffuses through the arachnoid and dura mater into surrounding connective tissues before entering venous and lymphatic networks [37][38].

Sutherland proposed that CSF was continuous with the rest of the body via lymphatic, perivascular, and perineural pathways, suggesting that its rhythm could be perceived throughout the body [2]. However, anatomical studies contradict this hypothesis. Sakka et al. [38] demonstrated that the subarachnoid space narrows near the proximal pole of nerve ganglia and ends in a clearly defined cul-de-sac, indicating no direct continuity with the peripheral tissues.

Continuity instead occurs via perivascular (Virchow–Robin) spaces, which appear to be involved in interstitial fluid drainage rather than direct CSF flow. Additionally, arachnoid trabeculae and ligaments slow and disrupt CSF circulation, resulting in slow (~1 cm/h), non-linear, and non-uniform flow.

Recent advances have shown that CSF can diffuse into the peripheral nervous system. Ligocki et al. [39] demonstrated that CSF follows the distribution of nanoparticles from the central to the peripheral nervous system, reaching the perineurium, endoneurium, and ultimately the axoplasm of distal nerves. Similarly, Pessa et al. [40] described CSF flow within nerve sheaths and glymphatic pathways, suggesting an open circulatory system within neural tissues.

Nevertheless, there is no evidence of a continuous, rhythmic “ebb and flow” of CSF as traditionally described in osteopathy. CSF production and drainage are not rhythmic processes.

Within the craniospinal system, CSF dynamics are closely coupled to intrathoracic pressure variations induced by respiration. During inspiration, CSF shifts cranially, while expiration promotes caudal flow [41] [32]. These dynamics reflect a strong interaction between CSF and venous circulation.

CSF flow velocity varies depending on location, being slowest at sites distant from production and arterial pulsation, with minimal or absent flow observed at the lumbar level [36].

In summary, brain expansion and retraction are primarily linked to respiration [30], rather than to CSF pressure variations, which are too small to drive cranial motion [10]. Furthermore, there is no anatomical continuity between CSF and peripheral tissues. It is therefore unlikely that CSF dynamics alone explain the PRM/CRI perceived by osteopaths.

The CSF is of significant importance in the philosophy of cranial osteopathy and, by increasing fluctuations, it could play a crucial role in improving the patient's condition. However, it does not appear

to be the cause of the PRM/CRI discussed here.

III.3. Motility of the intracranial and intraspinal membranes

The dura mater is part of the meningeal system, a set of membranous envelopes surrounding the brain and central nervous system. Intracranially, the dura attaches to specific cranial bones and may extend externally through the cranial sutures [42]. It also attaches to the upper cervical vertebrae and the sacrum, with additional attachments along the spine [43][44][45].

Within the skull, the falx cerebri and tentorium cerebelli are part of the dural system and contribute to a structure that mechanically links the cranial bones. Sutherland referred to this structure as the "reciprocal tension membrane" of the human skull. He believed that variations in dural tension contributed to the displacement of the bones to which the dura is attached, and that a reciprocal mechanism could only exist if the dura remained continuously under tension [3].

According to this model, the cranium moves rhythmically through so-called inhalation and exhalation phases, driven by shifting tensions within the reciprocal tension membranes. However, the dura mater has an average thickness of approximately one millimetre and displays anisotropic viscoelastic properties. It is therefore not inextensible, contrary to what some osteopathic theories have suggested [46].

The findings of Al-Habib et al. [47] are consistent with basic anatomical knowledge: among these tissues, the dura mater shows the greatest elasticity, followed by the pia mater and then the spinal cord. Their study also showed that compressed spinal cord tissue is significantly more rigid than uncompressed spinal cord tissue [47].

These findings are consistent with those of Royo-Salvador et al. [48][49] on craniocervical growth conflict. Their work suggests that stretching of the filum terminale may contribute to spinal cord compression and traction, thereby pulling the cerebellum towards the foramen magnum. Procedures involving sectioning of the filum terminale have been associated with cranial displacement of the conus medullaris [48][49].

The meninges transmit mechanical forces from the outside inward, as in trauma, and from the inside outward [3]. They also reflect the mechano-metabolic conditions of their surrounding environment. Over time, they can modify both their ability to respond to stress and their intrinsic structure [50].

According to Bordoni et al. [50], the meninges are therefore able to adapt to mechanical stress and strain. They do not appear rigid enough to pull cranial bones, except when they tend to ossify, which may occur with age, particularly in the dural sinuses. Such ossification may alter the transmission of mechanical forces [50].

Based on current data, if a tension force—whether pressure or traction—were transmitted from the brain to the skin, the intervening tissue layers would be expected to dampen it and slow its propagation [51]. Bordoni [50] also suggests that the viscoelastic properties of fascia would dampen a wave travelling from the skull to the periphery, just as they would dampen a wave travelling from inside the skull outward.

Consequently, if the CRI/PRM were transmitted mechanically from the cranial region, its amplitude should decrease with distance from the vertebral axis. However, osteopaths who use PRM/CRI commonly report an equal, or even synchronous, frequency at different points throughout the body. This observation makes a purely mechanical transmission hypothesis unlikely.

III.4. Joint mobility of cranial bones

The first hypothesis proposed by Sutherland in 1939 was that the PRM perceived at the cranial level resulted from movement of the cranial bones. In this model, the bones were thought to be pulled by the membranes, themselves influenced by fluctuations in cerebrospinal fluid. Sutherland considered that, even in adults, the skull could retain some flexibility at the sutures.

Historically, cranial bone motion was considered anatomically impossible. It remains one of the most controversial aspects of the PRM [52].

The main argument advanced by proponents of cranial bone mobility is that the sutures joining the different parts of the skull never completely ossify [53][54]. Although suture closure is gradual, bony spicules appear well before complete fusion [54]. Skull stiffness increases with age, but with considerable variability between individuals and between sutures [55].

Calcification of most cranial sutures begins around 20 to 30 years of age [56], although the endpoint of this process varies greatly according to the individual and the type of suture [57][58][59]. After the age of 60, most sutures are completely ossified [60]. In contrast, some osteopaths do not report any particular difficulty in perceiving or restoring CRI/PRM in older patients [61]. If PRM were directly linked to sutural mobility, its amplitude and strength would be expected to decline progressively as the sutures fuse with age. This does not appear to be consistently observed.

In the osteopathic cranial model, the synchondrosis between the occipital and sphenoid bones—the sphenobasilar synchondrosis—is considered the central structure of cranial biomechanics. It is also the joint to which Sutherland attributed several cranial dysfunctions [2][3].

However, the sphenobasilar synchondrosis begins to ossify before puberty, starting intracranially, and this process is completed during puberty [46]. More broadly, sutures of the human chondrocranium fuse progressively during infancy and adolescence, reaching complete fusion once cranial growth is complete [60][62][63].

Thus, in the adult skull, the sphenobasilar synchondrosis is ossified. From a scientific standpoint, it can no longer be considered the primary driver of cranial movement or the source of the various dysfunctions described in cranial osteopathy. Explanations for manual approaches intended to “release” this ossified joint should therefore be reconsidered [46].

Some authors have attempted to determine whether the light pressure commonly applied by practitioners (5-10 g) can produce sutural movement. Downey et al. [64] hypothesised that low loads applied to the frontal bone of anaesthetised rabbits, simulating the osteopathic frontal lift technique, would induce measurable changes and movement at the coronal suture. However, they were unable to demonstrate this effect.

For a degree of ossification comparable to that of a 20- to 30-year-old human, a force of approximately 50 kg would be required to move a rabbit cranial suture by 1 mm [65]. In a human child, the estimated force would be approximately 15 kg, far greater than the 5-10 g typically recommended in cranial osteopathy [66].

Scientists have long assumed that cranial bones are fused and immobile. However, there is now substantial evidence that the skull displays a degree of flexibility, as do other tissues of the body [67][55]. Bone tissue has viscoelastic properties because it contains both elastin and collagen, which together allow mechanical deformation of approximately 10-15% [46].

Starkey [61] describes the anatomy of the sphenoid clivus as spongy and therefore potentially malleable, but also hard and thick, and therefore relatively immobile. Cook [68] refers to cranial bone flexibility rather than mobility, while Seimetz et al. [55] suggest the possibility of cranial bone motility.

Although bone is one of the hardest tissues in the body, it deforms during normal function, and even more clearly during trauma. In several studies, cranial motion was induced by various internal and

external stresses in both animal and human specimens. Using MRI, Crow et al. [69] observed changes in measurements taken at different cranial points without human intervention or intention. They found statistically significant differences in area, width, height, and major-axis measurements, but not in perimeter or minor-axis measurements. This pattern does not correspond to the expansion and retraction described by osteopaths.

Sutherland's cranial model makes little reference to muscular action, possibly because he studied dry bones, as was common in early osteopathic models. However, according to Gabutti et al. [70], a substantial body of research on the mechanical properties of cranial bones and sutures indicates that muscle contraction is one of the main causes of cranial bone deflection [71].

Because of their viscoelastic properties and the composition of their surrounding extracellular matrix—rich in collagen fibres, proteoglycans, and water—cranial sutures are clearly more flexible than adjacent bone [70]. Rather than enabling mobility, sutures appear to play a role in energy absorption and force transmission [72][73].

The biomechanical properties of the human skull and cranial sutures vary according to age and anatomical layer, including the diploë and the inner and outer tables [74]. Sutures may influence the way the skull deforms, but skull deformation can still occur even when sutures are fused. Suture density, which depends on the degree of closure, may also limit the amplitude of deformation [70].

In conclusion, the bones of the skull do move, but not necessarily relative to one another. In the studies by Adams and Heisey, the measured range of movement at the sagittal suture was approximately 300 μm [75][52].

Cranial mobility may therefore be better understood as deformability of the skull as a whole. This deformability may occur under the influence of intracranial pressure, CSF fluctuations, membrane tensions, and external forces such as contractions of the head muscles and their associated connective tissues. Blood vessels crossing these tissues may also contribute, as their motility could deform the skull [76].

Moskalenko et al. [76] measured rhythmic changes in skull shape and volume using serial NMR scans. They reported a rhythm of 6-15 cpm, confirmed the physical extensibility of the bony skull, and observed a cranial expansion of 0.2-0.4 mm immediately after injection of 20 mL of fluid into the carotid artery. These findings support the possibility of changes in cranial volume [76].

Cranial mobility should therefore be considered more as a consequence than as a component of the PRM —an effect rather than a cause. This requires a reconsideration of Sutherland's model.

From an osteopathic treatment perspective, this does not exclude the possibility of releasing tension in a cranial suture, just as one might address tension in any other fascial structure, regardless of its degree of ossification. As suggested by Hamm [77], local tissue densification may occur through polymerisation of collagen fibres, which could solidify the two edges of a suture and produce fixation.

In this view, cranial osteopathic treatment would aim to soften the area, as it does elsewhere in the body, rather than necessarily restoring movement between individual cranial bones.

III.5. Involuntary movement of the sacrum between the iliac bones: the "core link" hypothesis

Sutherland considered the dura mater to be inextensible. In this model, traction generated by cranial bone motion would be transmitted through the dura mater and drive sacral motion [2][3]. However, if the dura mater were truly rigid, spinal flexion would be difficult to explain.

In reality, the dura mater is elastic. Experimental studies show that, in a neutral spinal position, the spinal dura forms folds, suggesting that the tissue is relaxed rather than continuously under tension [78]. Al-Habib et al. [47] showed that the dura mater has approximately twice the elasticity of the pia mater and three times that of the spinal cord. In addition, the length of the spinal canal varies by 19.4 ± 6.4 mm between spinal flexion and extension [79]. These data do not support the idea of an inextensible dural link mechanically driving sacral motion.

Cella et al. [80] investigated the occiput–sacrum relationship from a neurophysiological perspective. A sacral technique produced no immediate changes in occipital alpha-band brain activity. Conversely, the cranial technique known as compression of the fourth ventricle (CV4), in agreement with previous findings [81], produced immediate effects. This suggests that the osteopathic relationship between the head and sacrum may rely on a biological mechanism different from a direct mechanical dural link.

This pillar of Sutherland's model also gave rise to the idea of unity and frequency synchronization between the cranium and pelvis, and by extension throughout the body. However, not all osteopaths agree with this view. Moran and Gibbons [82] were unable to demonstrate synchronization between palpated rhythms at the head and sacrum. Rogers et al. [83] found no correspondence between head and foot rhythms when two examiners simultaneously palpated the PRM.

One could argue that the results might have differed if the subjects had first received an osteopathic "rebalancing" treatment. Further blinded experimental studies would be useful to measure PRM at different body sites, before and after osteopathic treatment, using both the same and different practitioners.

In light of the findings of Al-Habib et al. [47] and Royo-Salvador et al. [48][49], if a movement were transmitted mechanically from the skull to the sacrum, it would be expected to pass through the spinal cord and the filum terminale. However, the filum terminale attaches to the coccyx, not the sacrum [47][48][49]. This does not correspond to Sutherland's model [2][3].

In conclusion, Sutherland's 1939 model of the physiological basis of CRI/PRM, although still often taught as a dogma, is challenged by current scientific knowledge. We will now examine alternative hypotheses and explanatory models that may be considered in light of current evidence, with the aim of contributing to the emergence of a future consensus model.

IV. CRI/PRM and vasomotion

PRM is described as a cyclical movement perceived by the hands throughout the body, not only at the cranial level, where it is usually referred to as CRI. In 1850, Jones [84] first demonstrated vascular rhythmicity on the ventral surface of a bat wing. This rhythm was independent of systolic and respiratory rhythms and corresponded to spontaneous rhythmic changes in venous diameter. These changes were induced by intermittent contractions of the smooth muscle within the walls of microvessels, producing active blood displacement in the form of a slow oscillation. Jones referred to this phenomenon as *flowmotion*; today, it is more commonly termed vasomotion or vasomotricity.

Vasomotion refers to the ability of blood or lymphatic vessels to contract and dilate in response to pressure changes, thereby modulating flow [85]. At the arterial level, large vessels such as the aorta and large-calibre arteries respond mainly passively because their walls contain abundant elastic fibres. This property, known as compliance or arterial distensibility, helps buffer the effect of ventricular systole on blood pressure [86]. During diastole, large arterial trunks recoil, like a deflating balloon, thereby limiting the fall in blood pressure [86].

Two additional mechanisms are present only in small resistance arteries and arterioles, whose medial layer is rich in vascular smooth muscle cells (VSMCs). These mechanisms are absent from capillaries and large arterial trunks, which lack such muscular walls. The first mechanism is myogenic, and the second

is neurogenic. Confusion may arise in the literature when authors do not specify which mechanism they are referring to.

Vasomotion is particularly relevant in the study of vascular disorders. Fredriksson et al. [87] used near-infrared spectroscopy (NIRS) to measure weak and very weak spontaneous haemodynamic oscillations (0.003-0.15 Hz), confirming previous findings by Stefanovska et al. [88]. Stefanovska et al. described the following frequency bands: endothelial (0.0095-0.02 Hz), neurogenic (0.02-0.06 Hz), myogenic (0.06-0.15 Hz), respiratory (0.15-0.4 Hz), and cardiac (0.4-1.6 Hz) [88].

The myogenic mechanism is the most commonly considered mechanism in studies of vasomotion. Small-diameter arteries contract in response to an acute increase in blood pressure and relax when blood pressure decreases [89][90]. At arteriolar bifurcations, some cells display pacemaker activity generated by spontaneous oscillations in intracellular Ca^{2+} concentration within vascular smooth muscle cells [91][90][92]. These cells also respond to adrenergic vasoconstrictor signals, cholinergic vasodilator signals, metabolic factors (pH, CO_2, ATP, NO, O_2), humoral factors (prostaglandins, EDRF, etc.), and myogenic influences such as temperature and local muscle pressure [90].

In their study of arteriolar vasomotion and blood flow regulation, Colantuoni et al. [93] identified time-dependent diameter variation as a major feature of arteriolar microvascularisation. Spectral analysis showed that the fundamental frequency ranged from approximately 4 to 15 cpm in rank 1 arterioles (8 μm), and from 2 to 11 cpm in rank 2 arterioles (10-13 μm). Third- and fourth-order vessels (20 and 30 μm, respectively) showed lower frequencies, ranging from 0.5 to 6 cpm and from 0.3 to 3 cpm, respectively [93].

These oscillations are synchronized by electrical phenomena related to oscillations in cell membrane potential, namely polarization and depolarization. This activity is transmitted through gap junctions to coupled cells, including cells without pacemaker function [94][95]. Longitudinal conduction of vasomotor responses allows coordinated changes in vessel diameter and flow distribution. It also contributes to the regulation of vascular resistance by coordinating proximal and distal segments within the microcirculation [96].

Neurogenic influences are not essential for this process, although adrenergic stimulation can reinforce it. This variation is therefore not necessarily rhythmic, cyclic, or synchronous with neighbouring vessels. Rather, it is a localized and intermittent phenomenon, which makes it difficult to directly associate with PRM.

Nilsson and Aalkjaer [91] noted that: “In many experimental contexts vasomotion is problematic; for example, it is difficult to define specific amounts of tone in an oscillating vessel. Furthermore, vasomotion is frequently unpredictable and difficult to reproduce; occasionally, experimental animals cease to exhibit vasomotion for varying periods and in vivo experiments fail because vasomotion cannot be replicated.” Hamm [77] similarly concluded that: “The presence as well as the intensity or quantity of vasomotion is highly variable.”

Vasomotion is not systematically present under normal physiological conditions [97]. It is generally more frequent when perfusion is reduced, is often triggered by metabolic stress, and is usually absent at rest [94]. It may also occur only for brief periods [77]. Myogenic vasomotion may therefore influence CRI/PRM, but it cannot be its sole explanation. If it does play a role, its unpredictable nature may help explain the poor inter- and intra-examiner reliability reported in palpatory studies.

However, Nilsson and Aalkjaer [91] also observed rhythmic vasoconstrictions, probably of neurogenic origin. The largest transverse arterioles showed the lowest frequencies, ranging from 0.3 to 3 cycles/min, with diameter variations of 5-20% of the mean diameter [91]. Arterioles of approximately 18 μm in diameter showed variation amplitudes of 15-50% and frequencies of 0.5-6 cycles/min. Arterioles of approximately 11 μm showed larger variation amplitudes, from 50-100%, with frequencies of 2-11 cycles/min. The smallest and most superficial terminal arterioles showed the highest frequencies, from 4-15 cpm, and the greatest amplitudes, ranging from 60-100% of their mean diameter.

Interestingly, in their hamster study, Nilsson and Aalkjaer [91] reported a predominance of 0.1 Hz waves. These waves were transmitted from medium-sized arterioles (type 3 in Strahler’s classification) and reverberated into smaller arterioles (types 2 and 1), a frequency similar to that of PRM. This suggests a superposition of different arteriolar rhythms, with the most superficial rhythms, generated by the smallest arterioles, probably being the easiest for the osteopath’s hand to perceive.

Villermain-Lecolier et al. [98][99] described periodic micromovements perceived at the skin surface, with an amplitude of approximately 20-50 μm. These rhythms differed from both cardiac and respiratory rhythms, which were also measured in the same subjects. The mean frequency of these micromovements was 9.73 ± 3.48 cpm. According to the authors, PRM reflects local vasomotricity and does not need to be synchronous throughout the body. As noted above, there is no clear consensus among osteopaths on this point.

Vasomotricity improves blood circulation and tissue perfusion and may therefore contribute to the vitality of the organism. Exploring the link between PRM/CRI and vasomotion is consistent with one of the fundamental principles of osteopathy: the importance of circulation in maintaining health, as emphasized by Still [100].

Vasomotion may therefore contribute to the physiological basis of PRM/CRI, although it is unlikely to be the only underlying mechanism.

V. CRI/PRM and the autonomic nervous system

V.1. CRI/PRM and Traube–Hering–Mayer waves

Vasomotor waves, also known as Traube–Hering–Mayer (THM) waves, are physiological oscillations generated by spontaneous pulsations of arterial, venous, and lymphatic vessels. Traube–Hering waves result from the interaction between respiratory sinus arrhythmia and pulse pressure modulation in healthy individuals [101]. THM waves are mediated by the autonomic nervous system and, together with increased heart rate variability, are considered markers of good autonomic balance [36].

Neurogenic mechanisms influencing vasomotion are present under normal physiological conditions. They generate rhythmic, synchronous contractions activated by the sympathetic nervous system (SNS), leading to blood pressure oscillations known as Mayer waves [102]. These waves are also baroreflex responses to blood pressure variations [103] and are thought to result from a resonance phenomenon within the sympathetic baroreflex loop [104].

Afferent signals from conductance arteries may initiate a baroreflex feedback loop. These signals are relayed through the nucleus tractus solitarius (NTS) and transmitted via sympathetic vasoconstrictor fibres of the ganglionic chain to the arterioles, thereby triggering vasomotion associated with Mayer waves [91].

The NTS is located in the floor of the fourth ventricle. Interestingly, this corresponds anatomically to the region targeted by so-called “occipital compression”, or CV4, a well-known cranial osteopathic technique used to stimulate or restore PRM.

In regional circulations, Mayer waves are transmitted to conductance arteries [102]. Although attenuated by local vascular compliance, they remain detectable in some peripheral vascular beds, such as the fingers, where they can be measured by photoplethysmography (PPG) [105].

The Mayer wave frequency band (0.04–0.15 Hz) corresponds to the low-frequency (LF) component of heart rate variability. This component is associated with stimuli arising from baroreceptors and chemoreceptors in the carotid sinus, which are transmitted through the SNS to vascular smooth muscle cells (VSMCs). It is distinct from the high-frequency (HF) component, or respiratory sinus arrhythmia, also known as the Traube–Hering (TH) wave band, which is synchronous with respiratory movements (0.15–0.4 Hz) [106].

Mayer waves show strong and significant coherence with efferent sympathetic nervous activity, meaning that there is a strong linear coupling between fluctuations in these variables within the frequency domain [107]. Within a given species, their frequency is relatively stable. In humans, this frequency does not appear to depend on sex, age, or posture [107]. Rather, it largely depends on the delay between changes in sympathetic nerve activity and the resulting vascular response.

This delay is itself related to the length of post-ganglionic sympathetic neurons, particularly slower-conducting myelinated A fibres, and therefore ultimately to the size of the animal. The larger the animal, the longer the fibres, the longer the delay, and the lower the frequency. For example, Mayer wave frequency is approximately 0.1 Hz in humans, 0.3 Hz in rabbits, and 0.4 Hz in rats [108][102]. A similar observation is made in animal osteopathy regarding PRM frequency: the larger the animal, the lower the perceived frequency.

Fernandez and Lecine were likely the first to record vasomotor waves while simultaneously comparing them with cranial palpation [109].

Nelson et al. [17] also reported a strong correlation between CRI palpation and THM wave recordings obtained using laser Doppler flowmetry (328 data pairs; correlation = 1.00; significance = 0.00). Laser Doppler flowmetry measures blood flow velocity by detecting changes in erythrocyte movement, mainly through haemoglobin, within subcutaneous capillaries [110]. However, like other authors, they did not clearly distinguish between Traube–Hering waves, Mayer waves, and combined THM oscillations, which makes interpretation of these findings difficult. They concluded that PRM/CRI and THM oscillations were simultaneous, if not the same phenomenon.

These findings opened new perspectives for interpreting the theoretical basis of PRM/CRI and cranial therapy. Nelson et al. attributed the “still point” phenomenon to a brief cessation of the PRM rhythm, observed in 79% of cases together with reduced TH amplitude. Hamm [77] notes that McGrath, in his

review of Ferguson's paper, proposed a similar interpretation, suggesting that CRI may be "a manifestation of an extracranial blood flow phenomenon".

Christ et al. [111] measured cyclic changes in limb volume within the CRI frequency range and suggested that these changes were related to blood pressure variations, arteriolar vasomotion, and possibly changes in lymphatic diameter. They observed a relationship between cyclic increases in arterial volume and changes in limb volume (<0.177 mL per 100 mL of tissue).

Because muscles are essentially incompressible [112], and because surrounding tissues contain no empty spaces capable of absorbing these volume changes, cyclic dilation and contraction of fascial tubes in response to vascular volume changes, both intra- and extramuscular, may influence the crossed-helical fibre arrangements within their walls [113]. If this mechanism is involved, it could help explain the helical sensation, described as external and internal rotation, perceived by osteopaths when palpating PRM in the limbs.

At the cranial level, Abenavoli et al. [114][115] concluded that osteopaths could detect changes in CRI amplitude after the Queckenstedt test, which consists of bilateral compression of the internal jugular veins and produces an increase in intracranial pressure. These findings suggest that venous and arterial flow and pressure may contribute to both the origin and perception of CRI/PRM.

Mayer waves are abolished, or at least strongly attenuated, by pharmacological blockade of alpha-adrenoceptors [102]. This suggests that their haemodynamic basis lies in oscillations of sympathetic vasomotor tone in arterial blood vessels. Conversely, acute beta-adrenoceptor blockade has little or no effect on these oscillations [108][116], suggesting that vagal control plays only a limited role.

Uraemia also appears to markedly reduce Mayer wave amplitude, which may indicate altered cardiovascular autonomic nervous system function [117]. It would therefore be of interest to investigate whether PRM varies in patients with such conditions.

In conclusion, Traube–Hering and Mayer waves should be clearly distinguished from one another. Both may contribute to CRI/PRM, but they are not identical phenomena.

V.2. CRI/PRM and heart rate variability (HRV)

The heart does not beat with metronomic regularity: the interval between successive heartbeats varies continuously. This variability is known as heart rate variability (HRV) [118]. HRV is widely recognized as an indicator of autonomic nervous system (ANS) function [119].

In general, high HRV reflects a healthy and adaptive ANS, capable of adjusting heart rate in response to internal and external stimuli [120]. Conversely, low HRV is associated with various pathological conditions, including cardiovascular disease and diabetic neuropathy, and indicates reduced adaptability of the autonomic nervous system [121].

HRV analysis provides a non-invasive and objective measure of ANS modulation, offering insight into the relative contributions of sympathetic and parasympathetic activity. Rather than acting purely in opposition, these two branches are now understood to operate in a coordinated manner, sometimes synergistically and sometimes reciprocally [122].

Spectral analysis of HRV is traditionally divided into low-frequency (LF; 0.04–0.15 Hz) and high-frequency (HF; 0.15–0.4 Hz) bands. However, the exact boundaries of these bands vary across studies. HF-HRV, associated with respiratory sinus arrhythmia, increases with parasympathetic (vagal) activity [123]. In contrast, LF-HRV reflects both sympathetic and parasympathetic influences and may represent an independent physiological mechanism beyond its parasympathetic contribution [123]. Some authors suggest that the LF band reflects subharmonic activity of a central pacemaker located in the lower brainstem [123].

The LF/HF ratio was long considered an index of sympathovagal balance. However, this interpretation has been widely challenged. Because the LF band does not reliably reflect sympathetic activity, there is now general agreement that the physiological meaning of the LF/HF ratio remains unclear, making its interpretation problematic [124].

Commonly reported HRV metrics include HF-HRV and LF-HRV. More recently, an intermediate (IM) frequency band has been described. This band appears during states of hypnoid relaxation and has been identified in analyses of forehead skin perfusion, electrocardiographic signals, and respiration [123][125]. Preliminary evidence suggests that oscillations around 0.15 Hz originate in the brainstem and propagate to the skin microvasculature via parasympathetic pathways [123][125].

Schwerdtfeger et al. [126] further proposed that the LF band may include two distinct rhythms: a lower-frequency component (0.06–0.1 Hz), primarily associated with baroreflex-mediated blood pressure regulation via sympathetic pathways, and a higher-frequency component (0.1–0.14 Hz), which may reflect more complex interactions between sympathetic and parasympathetic efferent activity and heart–brain communication.

Pelz et al. [9] showed that skin blood flow exhibits patterns similar to PRM/CRI when measured using photoplethysmography (PPG). They suggested that PRM/CRI may correspond to this ~0.15 Hz physiological rhythm, also referred to as the intermediate (IM) band. HRV-related oscillations may therefore contribute to the PRM/CRI phenomenon [127].

VI. PRM, microcirculation, lymphatic system, extracellular matrix and fascial network

VI.1. Microcirculation

Approximately 10-20% of the fluid filtered from capillaries into the interstitial space is returned to the systemic circulation via the lymphatic system, while the majority (80-90%) re-enters the capillaries and exits through the venous system [128].

Venules have a larger diameter and thinner muscular walls than arterioles. They are under sympathetic control, allowing their walls to contract and relax. This contributes significantly to vascular capacitance and the regulation of tissue perfusion. It also provides a physical basis for the frequency modulation observed in Traube–Hering and Mayer waveforms [129][130][128].

As the vascular system oscillates under the influence of TH and M waves, and in conjunction with arterial resistance, the venous capacitance system undergoes slow and regular contractions. These oscillations facilitate fluid movement within the interstitial space, promote lymphatic circulation, and support venous return to the heart [131][128]. Vasomotion associated with TH and M oscillations also contributes to the maintenance of negative interstitial pressure, as described in Starling's equilibrium for capillary exchange [128].

Oscillatory "oxygen availability waves" have also been described. Vern et al. [132] reported a mean frequency of 9.58 ± 0.117 cpm, while Clark et al. [133] reported frequencies between 6 and 12 cpm. These findings suggest that cyclic variations in cortical oxidative metabolism may represent a primary local oscillatory process. These metabolic oscillations are followed by reflex haemodynamic changes that influence local tissue perfusion and intracranial blood volume.

Although these processes are not synchronized across the entire brain, the relatively close frequencies of cytochrome oxidase redox oscillations and THM waves may allow these processes to become entrained.

This coupling could link local metabolic regulation with central haemodynamic control of tissue perfusion [128][134].

VI.2. Lymphatic and glymphatic systems, brain cell volume, and extracellular matrix

Tissue fluid pressure is a key determinant of fluid exchange between the vascular and interstitial compartments, as well as between interstitial spaces and terminal lymphatic vessels [135].

Lymphatic vessels exhibit rhythmic contractions driven by smooth muscle cells within lymphangions. These contractions generate peristaltic waves that propel lymph forward. Their frequency is variable: Mawhinney and Roddie reported a mean of 2-2.5 cycles per minute (range 1-4 cpm) [136]; McHale et al. observed frequencies of 4-5 cpm in bovine mesenteric lymphatics [137]; and a frequency of 4.8 cpm was reported in the popliteal lymphatic vessels of sheep [138]. More broadly, lymphatic vessels can show spontaneous contractions ranging from 1 to 30 cpm [139]. Olszewski [140] described spontaneous lymphatic contractility between 1 and 9 cpm (mean ≈ 4 cpm), independent of cardiac, respiratory, or body movements.

Each lymphangion is capable of spontaneous, independent contractility through a myogenic mechanism similar to that described for arterioles [128]. Although lymphangions may contract asynchronously under the influence of local pacemaker activity located near the valves, they function more efficiently when contractions become synchronized. Lymphatic vessels also tend to develop synchronized activity relatively easily [141][128]. Entrainment driven by THM oscillations may therefore optimize the efficiency of lymphatic transport.

Experimental and clinical observations indicate that rhythmic lymphatic contractions occur across multiple species, including humans. For example, the human thoracic duct contracts approximately once every 10-15 seconds, corresponding to about four pulsations per minute, a frequency close to that described for CRI [142]. Perrin [143] has therefore suggested that PRM could correspond, at least in part, to rhythmic lymphatic contractions.

Ferguson [10] argued that the lymphatic system is unlikely to be responsible for CRI, based on the absence of large lymphatic vessels within the cranial cavity. However, subsequent work by Kipnis and colleagues demonstrated the presence of lymphatic vessels associated with the meninges [144]. This raises the question of whether the origin of PRM should necessarily be sought at the cranial level. Indeed, some

patients present with a weak or imperceptible CRI at the skull but a clearly perceptible rhythm at the sacrum or elsewhere in the body, suggesting a more distributed origin.

The glymphatic system refers to the circulation, mixing, and clearance of fluid within the brain interstitium. This system involves three main phases: (1) entry of cerebrospinal fluid (CSF) into perivascular spaces; (2) mixing of CSF with interstitial fluid (ISF); and (3) clearance of this fluid from brain tissue [145].

Brain cells, including neurons and glial cells such as astrocytes and oligodendrocytes, exhibit rhythmic swelling and shrinking associated with intracellular calcium fluctuations and water movement [16][7][132]. However, oligodendrocyte contractions described by Pomerat et al. [146], and later cited by Retzlaff and Mitchell [147], occur at much lower frequencies (4–18 minutes per cycle).

More generally, intracellular calcium oscillations have been recorded at frequencies ranging from 0.5 to 12 cpm, both in vitro and in vivo, and are consistent with rhythms influenced by THM oscillations and the autonomic nervous system [148]. These ionic exchanges are also associated with changes in the viscosity and electrical charge of the extracellular matrix.

Capillary perfusion pressure, modulated by THM waves, may induce rhythmic transitions between gel and sol states in the extracellular matrix (ECM), which behaves as a viscoelastic colloid. Lee [7] describes the ECM as a network of proteoglycans (PGs), glycoproteins (GPs), and glycosaminoglycans (GAGs), forming a sieve-like structure through which metabolites diffuse between capillaries and cells.

Proteoglycans, which carry negative charges, bind water and tend to form gel-like structures due to their thermodynamic instability. This organization gives the ECM properties similar to those of a liquid crystal or a semi-conductive medium. According to Hamm [77], cyclic changes in ECM texture may provide a plausible hypothesis to explain both the palpatory sensations and some of the therapeutic effects reported in cranial osteopathy. These properties may be altered by local fascial tensions, leading to increased density or stiffness.

In summary, blood flow is influenced not only by vascular compliance and resistance but also by the mechanical and structural properties of surrounding tissues, particularly the local density and organization of the ECM. Under conditions of sustained fascial tension, the ECM may undergo densification through collagen fibre polymerization and the accumulation of hyaluronic acid aggregates [149]. These changes may contribute to the mechanisms underlying PRM.

VII. PRM and Entrainment

Although Ferguson [10] and Perrin [143] noted that cerebral veins lack valves and contain minimal smooth muscle—therefore exhibiting little to no vasomotion—the rhythm perceived by osteopaths may instead originate from superficial and subcutaneous vascular structures rather than from deep intracranial vessels.

A wide range of rhythms has been described by osteopaths. It is therefore plausible that some practitioners perceive the composite signal resulting from multiple overlapping oscillations, while others detect only specific components of this signal. These rhythms are likely superimposed and may interact with one another. Consequently, the palpatory perception of PRM/CRI may represent the resultant of several physiological oscillations rather than a single, discrete phenomenon.

This perspective aligns with the theory proposed by McPartland and Mein [150], who suggested that PRM arises from the synchronization—or entrainment—of multiple biological rhythms originating from both the patient and the practitioner. Norton [151] further explored this concept through his tissue pressure model, proposing that CRI perception involves slowly adapting cutaneous mechanoreceptors in both individuals. According to this model, fluctuations in tissue pressure result from the combined respiratory and cardiovascular rhythms of the practitioner and the subject.

This framework may help explain the variability observed in inter- and intra-examiner reliability studies. Differences in baseline physiological parameters, such as heart rate and respiratory frequency between practitioners, could influence the perceived rhythm [151].

Tiller et al. [152] described a phenomenon in which biological oscillators become synchronized when sympathetic and parasympathetic activities are balanced. Under such conditions, multiple physiological rhythms—including heart rate variability (HRV), Traube–Hering–Mayer (THM) waves, respiration, pulse transit time, and even brain activity—can become coordinated and form harmonic relationships. This coordination gives rise to a dominant oscillatory pattern referred to as the *entrainment frequency* [150].

In healthy individuals, this entrainment frequency has been measured at approximately 0.1 Hz (≈6 cpm), which falls within the commonly reported range for PRM/CRI [152]. This convergence suggests that PRM/CRI could correspond to an emergent property of coordinated physiological rhythms rather than to a single underlying mechanism.

From this perspective, a balanced autonomic nervous system would favor the emergence of a coherent, stable, and sinusoidal oscillation that could be perceived as a strong and regular PRM/CRI. Conversely, autonomic dysregulation may disrupt this coordination, leading to alterations in PRM characteristics such as frequency, amplitude, and regularity [150]. In cases of severe dysregulation, the absence of synchronization between physiological rhythms could result in a weak or imperceptible PRM/CRI.

Further experimental studies are required to determine whether the entrainment frequency can be reliably measured and whether it corresponds consistently to the palpated PRM.

VIII. PRM and Embryologic Movement

An alternative model, often referred to as the biodynamic approach, was initially developed by Rollin Becker in the 1930s and later expanded by James Jealous [150]. This model draws on the work of the embryologist Erich Blechschmidt, who proposed that function—particularly fluid motion—precedes and shapes anatomical structure during embryonic development [153][154].

Blechschmidt observed that cellular migration and differentiation are driven by dynamic fluid processes, which he described as "biodynamics." Building on this concept, Jealous hypothesized that these embryological forces persist throughout life and continue to influence growth, adaptation, and healing processes.

Within this framework, a slower rhythm than PRM/CRI has been proposed, often referred to as the "2.5 cycles per minute" rhythm [150]. Biodynamic literature also introduces broader and less precisely defined concepts such as the "Breath of Life" or "therapeutic force," which are described as fundamental organizing principles but are not associated with a clearly defined physiological origin or frequency.

Because these concepts extend beyond the scope of the present review—focused specifically on the physiological basis of PRM/CRI—they are not examined in detail here. Nevertheless, they highlight the possibility that PRM/CRI may not represent the most fundamental biological rhythm.

Sutherland himself alluded to the existence of deeper and more subtle rhythms underlying CRI [3]. Becker later described CRI as comprising multiple components, including a "fast tide" (8-12 cpm) and a "slow tide" (~0.6 cpm) [155]. Additional rhythms have been described in biodynamic literature, such as the "mid-tide" (2-3 cpm) and the "long tide" (approximately 6 cycles every 9-10 minutes), which may correspond to broader regulatory processes.

The coexistence of multiple rhythms across different frequency ranges adds further complexity to the study of PRM/CRI. It also provides a plausible explanation for the variability observed in both subjective perception and objective measurement, as well as for the limited reproducibility reported in inter- and intra-examiner studies.

IX. Perspective

In this final section, we discuss the potential clinical relevance of CRI/PRM in osteopathic practice.

IX.1. PRM as an Osteopathic Tool

If PRM oscillations reflect variations in the mechanical properties of the fascial network, they may represent a valuable clinical tool for manual therapists aiming to assess and release myofascial tension. In this context, PRM could serve both as a diagnostic indicator—reflecting the level of tissue tension—and as a guide for therapeutic intervention. Its qualitative features, such as amplitude and strength, may improve as tissue viscoelasticity and deformability increase.

To further investigate this hypothesis, objective measurements could be compared with subjective palpatory findings. For example, Myoton-based assessments performed at high temporal resolution could be correlated with PRM palpation to determine whether cyclic variations exist within the same frequency range. Such approaches could help clarify whether PRM corresponds to measurable biomechanical oscillations.

While this perspective primarily supports a loco-regional approach, understanding the underlying mechanisms of PRM also reinforces a core osteopathic principle: the functional unity of the body. In particular, interventions that modulate autonomic nervous system (ANS) activity—especially through brain–heart interactions—may have systemic effects.

Osteopathic manipulative treatment (OMT) has long been proposed to restore physiological function by modulating autonomic activity, promoting vasodilation, reducing smooth muscle tone, and improving circulation [156]. If PRM is indeed related to ANS dynamics, its use as a clinical feedback tool may help practitioners assess the immediate effects of their interventions.

Most studies report a decrease in sympathetic activity and an increase in parasympathetic modulation following OMT [124][157], which is consistent with a relaxation response. However, it is important to acknowledge that such effects are not specific to osteopathy. Any intervention capable of inducing

relaxation—including non-specific manual contact or contextual factors—may influence ANS regulation [158]. This highlights the importance of controlled studies using appropriate placebo or sham conditions.

For example, Abenavoli et al. [115] evaluated the effects of the CV4 technique on serum amyloid A (sAA) levels and observed changes that were not significantly different from those obtained with a sham procedure. This finding underscores the need for cautious interpretation of physiological effects attributed to specific techniques.

The CV4 technique has nevertheless been associated with decreased sympathetic tone and increased parasympathetic activity [81][159][19]. Nelson et al. reported that CV4 enhanced THM oscillations, whereas simple cranial contact did not, suggesting a possible specific effect beyond placebo [160][19]. Similarly, Sergueef et al. [20] showed that cranial techniques targeting global cranial motion and the cranio-cervical junction influenced THM oscillations.

More recently, Pelz et al. [9] demonstrated significant ANS responses to the cranial vault hold (CVH), including changes in LF-HRV, the intermediate frequency band, and photoplethysmography-derived parameters. They also reported an increase in RMSSD following CV4 [161], a commonly used index of parasympathetic activity.

Other studies have used RMSSD [162], skin conductance [163], or HF-HRV [164] as objective markers to evaluate ANS modulation following OMT. Fornari et al. [165] further showed that a single osteopathic session improved recovery of heart rate and sympathovagal balance after acute stress in healthy individuals.

Overall, these findings suggest that OMT may influence ANS regulation, particularly by enhancing parasympathetic activity and reducing sympathetic dominance. Within this framework, PRM could potentially serve as a clinical indicator of autonomic modulation.

Traditionally, a “balanced” PRM is described as having a strong amplitude, regular rhythm (around 6 cpm in humans), and symmetrical expression between expansion/flexion/external rotation and retraction/extension/internal rotation. Symmetry between body regions, including the limbs, is often interpreted as a sign of global coherence.

However, this concept of systemic synchrony remains debated. Palpatory studies have failed to demonstrate consistent correlations between rhythms perceived at different anatomical sites, such as the

head and sacrum [166][82][83]. While it is possible that synchrony may be more apparent in well-regulated physiological states, current evidence does not support it as a universal feature.

IX.2. Ideomotion

Ideomotor phenomena refer to involuntary micro-movements generated by cognitive processes, such as expectation, attention, or intention, rather than by conscious motor control [167].

In the context of osteopathic palpation, it is possible that part of the perceived PRM arises from such ideomotor responses. The practitioner's expectations, attentional focus, and interpersonal interaction with the patient may influence perception and contribute to "expectancy confirmation effects" [168].

Shin et al. [169] reviewed historical perspectives on ideomotion, highlighting early concepts proposed by William Benjamin Carpenter (1874), Johann Friedrich Herbart (1825), and William James (1890). These authors suggested that mental representations and expectations can directly influence motor output, even in the absence of conscious intention.

Applied to PRM perception, this implies that actively searching for a rhythm may contribute to generating or amplifying it. However, this does not necessarily invalidate the clinical relevance of the phenomenon. Tissue response remains constrained by its intrinsic mechanical properties. Relaxed, compliant tissues may allow greater transmission or expression of micro-movements, whereas stiff or restricted tissues may dampen them.

From this perspective, PRM palpation may still provide meaningful information about tissue state, even if the perceived motion is partially influenced by the practitioner. The symmetry and amplitude of the response would depend on the biomechanical properties of the tissues rather than on the sole existence of an objective rhythm.

Extremes in interpretation should therefore be avoided. The claim that PRM is purely illusory is not supported, as measurable physiological rhythms exist within the relevant frequency range. Conversely, the assumption that palpation provides a fully objective measure is also questionable, given the potential influence of ideomotor processes.

Similarly, the search for perfect synchrony across the body should be interpreted with caution. While it may reflect a state of reduced tissue tension and improved responsiveness, it is unlikely to represent a universal physiological norm. If PRM results from the interaction of multiple oscillatory systems, complete and systematic synchronization would not be expected.

IX.3. Cardiac Coherence, Respiration and PRM

Osteopathic manipulative treatment (OMT) aims to enhance the body's capacity for self-regulation, potentially through modulation of the autonomic nervous system (ANS). Within this framework, PRM/CRI may serve as a clinical indicator reflecting changes in autonomic balance.

Pelz et al. [170] reported rhythmic responses within the intermediate (IM) frequency band following the Cranial Vault Hold (CVH), a commonly used technique in osteopathy in the cranial field (OCF). These responses were comparable to those observed during autogenic relaxation training, suggesting that similar physiological mechanisms may be involved.

An increase in the amplitude of perceived PRM oscillations has been hypothesized to reflect improved coordination between cardiovascular, neural, and respiratory rhythms. More broadly, physiological entrainment is increasingly recognized as a key mechanism underlying mind–body interactions. Interventions based on entrainment principles have shown potential benefits for cognitive, motor, and emotional functions, and are being explored in rehabilitation and mental health contexts [171].

Some authors have proposed more speculative models. For example, Blacklaw-Jones suggested that piezoelectric phenomena and endogenous electromagnetic fields could contribute to the effects observed in cranial osteopathy [172]. According to this hypothesis, oscillations in bioelectrical activity could generate coherent vibratory patterns within tissues. It is further proposed that practitioners entering a meditative state may enhance coherence within their own physiological systems and, through physical contact, influence the patient's state. However, these mechanisms remain theoretical and are not currently supported by robust experimental evidence.

Mind–body practices such as yoga, meditation, and breathing exercises have been associated with increases in HRV and improved autonomic balance [173][36]. A common feature of many of these approaches is the induction of cardiac coherence through controlled breathing patterns.

Cardiac coherence breathing typically consists of inhaling for approximately five seconds and exhaling for five seconds, resulting in a respiratory frequency of about 0.1 Hz (6 cycles per minute) [174]. This breathing pattern modulates autonomic activity: inhalation transiently reduces parasympathetic influence and increases heart rate, whereas exhalation enhances parasympathetic activity and decreases heart rate. When practiced regularly, this leads to increased heart rate variability and improved autonomic adaptability [175][152].

Given that PRM/CRI may reflect the integration of multiple physiological rhythms, it is plausible that its amplitude and clarity increase during states of autonomic coherence. Under such conditions, oscillatory systems may become more synchronized, potentially facilitating both palpatory perception and physiological regulation. This could also contribute to improved circulation of blood, lymph, and other fluids.

It can therefore be hypothesized that synchronizing the breathing patterns of both patient and practitioner during treatment may enhance these effects. However, this remains speculative and requires experimental validation. Further research is needed to establish the relationship between PRM/CRI and well-characterized physiological processes such as cardiac coherence.

X. General Conclusion

This review aimed to re-examine the physiological mechanisms underlying the Primary Respiratory Mechanism (PRM) and Cranial Rhythmic Impulse (CRI).

The analysis of the literature indicates that the original model proposed by William Garner Sutherland is not fully consistent with current scientific knowledge. Advances in physiology, neuroscience, and biomechanics challenge the five classical pillars historically used to explain PRM.

At present, no single mechanism can fully account for the characteristics of PRM/CRI. Instead, the available evidence supports a multifactorial and integrative interpretation. The most plausible hypothesis is that PRM/CRI emerges from the interaction of multiple physiological oscillations, including vasomotion, autonomic nervous system activity, heart rate variability, microcirculation, and respiratory dynamics.

In this context, rhythmic variations in the viscoelastic properties of the extracellular matrix—modulated by these interacting systems—may play a central role. These fluctuations could facilitate the transport of nutrients and metabolic waste products, thereby contributing to tissue homeostasis [176].

An increase in the amplitude of these oscillations may reflect improved coordination between cardiovascular, neural, and respiratory systems. Such coordination could enhance global fluid circulation and support the organism's adaptive capacity.

However, several limitations must be acknowledged. The relationship between palpatory perception and measurable physiological processes remains incompletely understood. In addition, the influence of practitioner-related factors, including ideomotor effects and interpersonal dynamics, cannot be excluded.

These findings support the need for a paradigm shift. Rather than relying on a fixed and mechanistic interpretation derived from Sutherland's original model, PRM/CRI should be considered as an emergent phenomenon arising from complex, dynamic, and interacting biological systems.

Future research should aim to:

- develop objective measurement tools capable of capturing PRM-related oscillations,
- clarify the relationship between palpation and physiological signals,
- and evaluate the specific effects of osteopathic interventions using rigorous experimental designs.

Figure 2 provides a schematic overview of the different physiological mechanisms that may contribute to the PRM/CRI phenomenon.

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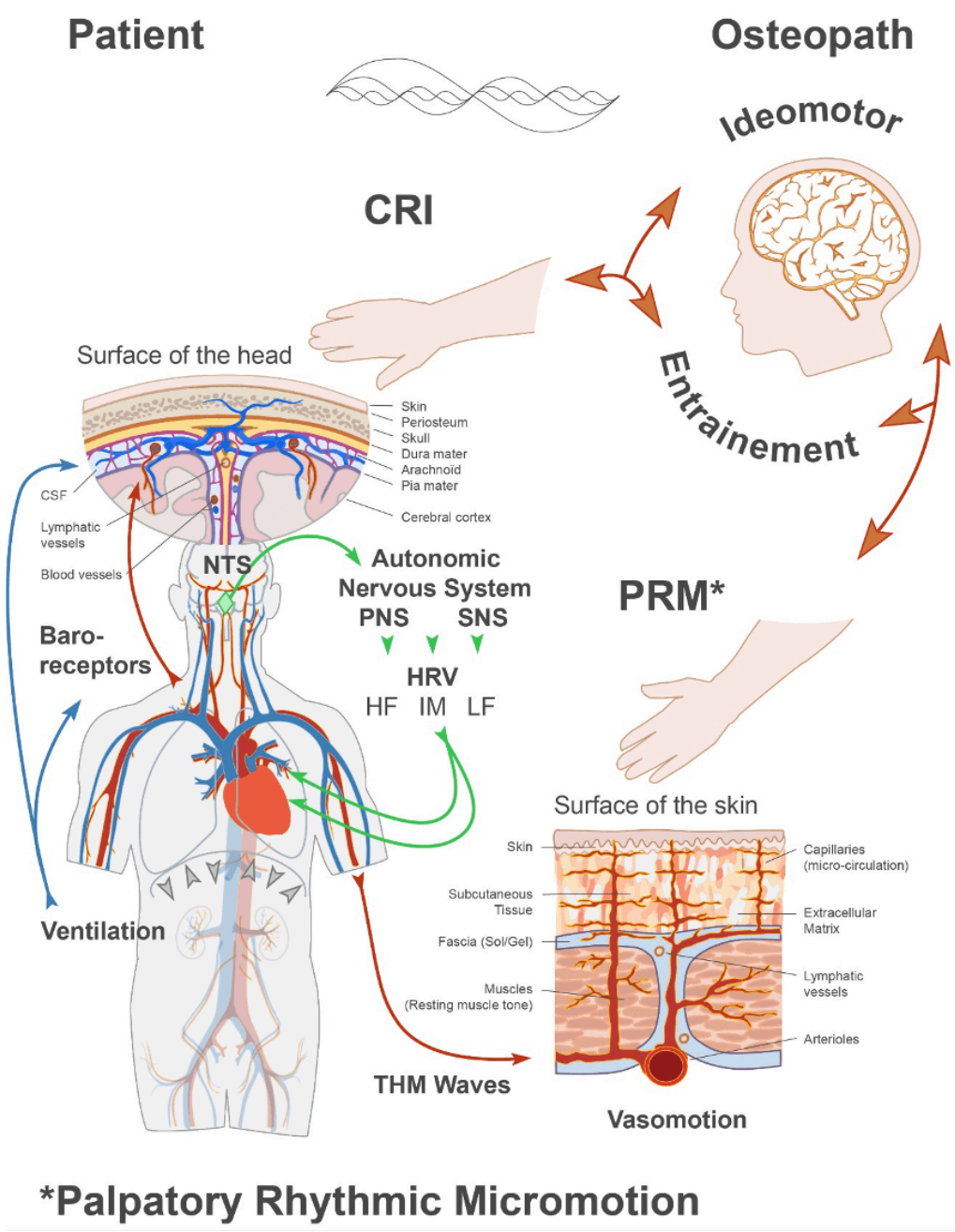
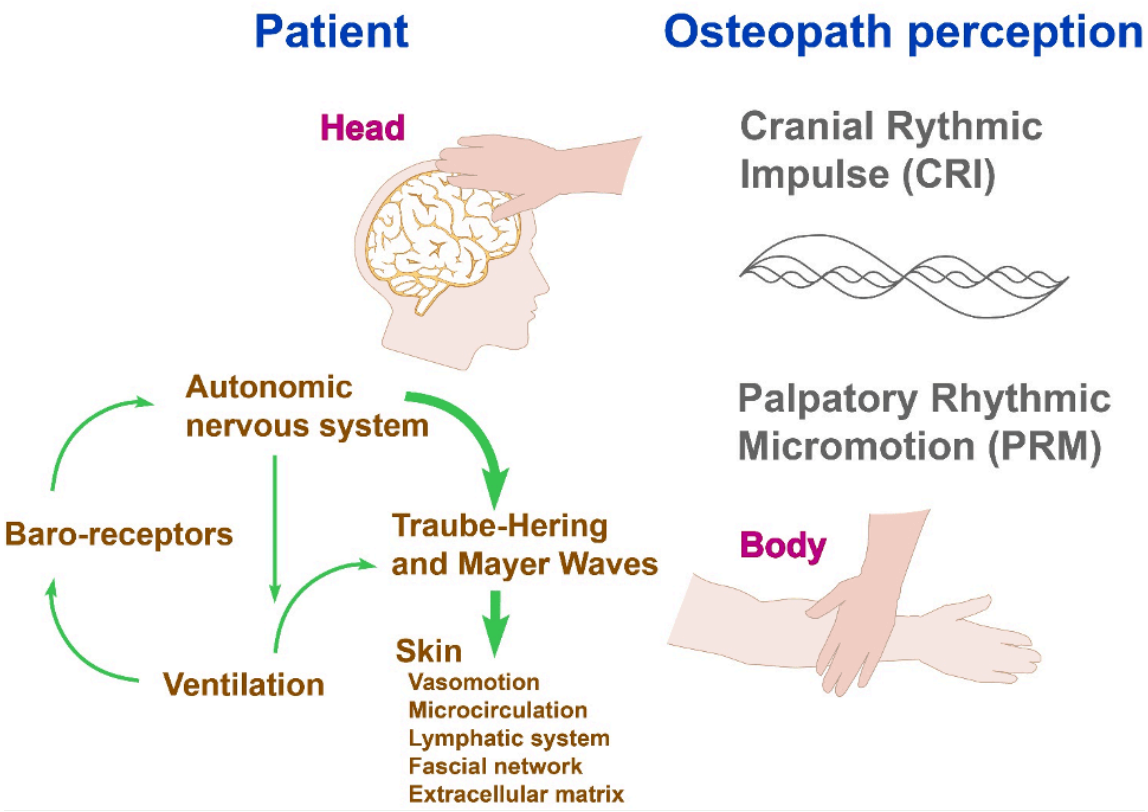


Figure 2. Overview of the different mechanisms underlying the PRM/CRI.

As a consequence of paradigm shifts, and in order to accompany this evolution, the authors propose replacing the dogmatic term 'Primary Respiratory Mechanism' with a factual term, 'Palpatory Rhythmic Micromotion' (PRM).

Graphical abstract PRM/CRI



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All authors contributed to the redaction of the narrative review. AG and FM had the idea for the article, LS, AG and FM performed the literature search and data analysis, FM wrote the main draft and LS, AG drafted and critically revised the work, LS did a major work on the figures. All authors read and approved the final manuscript.

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Notes

Short title: Physiological mechanisms underlying the PRM/CRI in osteopathy

Footnotes

[1] Note that in France, a semantic shift can be observed, as many osteopaths use the term 'movement' instead of 'mechanism' when referring to PRM.

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