Review Article

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

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Cranial Rhythmic Impulse (CRI) or Primary Respiratory Mechanism (PRM), a rhythm felt by Osteopaths at the surface of the skin, is a fundamental concept that some of them in their practice for their diagnosis and treatment. However, the physiological basis of this phenomenon remains unclear. Sutherland, founder of cranial osteopathy, proposed in 1939 a theory that remained dogmatic, despite scientific advances that refuted it. Since 1990, some osteopaths have tried to find better explanations, such as those presented in a previous systematic review. In this narrative review, we first revisit each pillar of Sutherland's theory, analyzing them in light of the latest scientific studies to assess whether contemporary research supports or challenges his ideas. After showing that this model is inconsistent with current scientific evidence, we explore the current knowledge of the physiological mechanisms underlying the PRM/CRI.

The most plausible hypothesis to explain this is based on the variation in extracellular matrix and fascial texture, influenced by rhythmic oscillations in blood pressure (Traube Hering and Mayer waves), and the interplay of other physiological rhythms (ventilation, baroreflex, heart rate variability…). Finally, we discuss the relevance and practical applications of PRM in osteopathy. While Sutherland's model continues to hold sway in mainstream beliefs, the scientific literature has increasingly emphasized autonomic system activity as the leading hypothesis.

This narrative analysis underscores the need for a paradigm shift regarding CRI/PRM in osteopathy, arguing for clearer communication of a model aligned with the latest scientific evidence.

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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 sensation of a physiologic rhythm on the surface of the skin[1]. It can be used for their diagnosis and treatment. It is described as having two phases, an expansion phase (inhalation) and a contraction phase (exhalation)[2][3]. It is different from respiration as it is still perceivable when the patient holds its breath[4].

Historically, the osteopath who first described it, William Garner Sutherland (1873-1954) had the intuition that cranial sutures were made for movement and imagined a model based on five principles: the

inherent mobility of the central nervous system and the spinal cord; the fluctuation of the cerebrospinal fluid (CSF); the Reciprocal Tension membranes; the articular mobility of the bones of the skull; the involuntary movement of the sacrum between the iliac bones[2][3].

Sutherland chose the term “Primary Respiratory Mechanism” (PRM) to highlight several key aspects of his concept. The word *mechanism* indicates that the PRM is a physiological, organized phenomenon where multiple elements interact in a coordinated way. The term *respiratory* was chosen by analogy with pulmonary breathing, but it refers to a different process. For Sutherland, the PRM represented an autonomous rhythmic pulsation in the body, essential to life and present even in the absence of lung respiration[2]. He wanted to emphasize that the body has an internal periodical motion, independent of the lungs, animating tissues and fluids. The word *primary* means that this mechanism would be present from early embryonic life and constitute a fundamental life function. Many osteopaths today use the term PRM to describe the micromovements they work with. [1]

Many osteopaths today use the term PRM to describe the micromovements they work with. However, to be precise and remain true to its original meaning, the term *Cranial Rhythmic Impulse* (CRI) should be used when referring specifically to the palpatory sensation of this rhythm on the skin’s surface. This terminology was introduced in 1961 by Woods and Woods, who perceived a rhythm at the surface of the head. They linked it to Sutherland’s PRM, and attempted, seven years after his death, to define a normal frequency range, something Sutherland had never done himself[5].

For some osteopaths, CRI and PRM represent the same movement, felt respectively on the scalp and throughout the rest of the body. The confusion between these two terms is common[6]. However, this distinction is not critical, as CRI is considered to originate from PRM. Thus, the widespread conflation of these term is essentially a simplification. PRM remains the historical explanation for this particular rhythm, which is the subject of this review[7][8].

The five phenomena 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 then sought to structure Sutherland’s teachings, making them more accessible and reproducible by formalizing techniques and protocols[2][3]. However, Sutherland was somewhat resistant to this biomechanical formalization, fearing it might limit a deeper understanding of his approach. Whereas he viewed cranial movement as a holistic process influenced by multiple factors, Magoun, in his effort to popularize these ideas, risked reducing this complexity to a predefined set of practices,

describing specific movements and techniques. This divergence created some tension, as Sutherland feared his work's essence might be lost[2][3].

Nevertheless, this is the legacy we have today: osteopaths perceive a rhythm believed to be associated with cranial bone movements and other phenomena, and it is this very assumption that we aim to reassess.

With these clarifications in mind, we have chosen to systematically associate the terms PRM and CRI to align with common usage, referring to the typical rhythm perceived by osteopaths.

PRM/CRI is defined by its three components: amplitude, rhythm, and strength. If these three components are correct, the osteopath can conclude that the patient, or at least the area he is focusing on, is well balanced. Some osteopaths think that PRM/CRI can be useful as a tool to verify the effectiveness of each osteopathic treatment, checking it after each technique applied until it is good enough to mark the end of the session. The PRM/CRI would then reflect the harmony and vitality of the body. The amplitude should be large and symmetrical. The force is considered as correct when it feels good and strong. Although in general the rhythm is considered normal between 6 and 12 cycles per minute (cpm), in the literature one can find huge variations from 3 to 22 cpm[9]. Figure 1 shows the wide range of frequencies described in the literature, which are not easily attributable to an identified physiological rhythm. The different perceived rhythms could depend on the practitioner/patient pair and vary against time[10][11][12][2][13][14][15][5], which makes objective measurement and comparison difficult, and may explain why intra- and inter-examiner reproducibility is difficult to achieve. Finally, different studies may report on different rhythms under the same name.

Some rhythms with similar rates to PRM can also be obtained by instrumentation, which could objectify its existence[10]. In a recent study, Rasmussen and Meulengracht[4] demonstrated the existence of a third physical rhythm (different from cardiac and respiratory rates) detected on the human head by their machine with a mean of 6.16 cycles/minute (4.25-7.07). However, the measurement tool, although metrologically validated in their laboratory (Appendix A. Supplementary data in [4]), has a significant bias, similar to the measurement tool used by Frymann in 1971: head movements generated by ventilation are not controllable[16]. Nelson et al.[17][18][19][20], and more recently Pelz et al.[9], demonstrated a statistically significant correlation between skin blood flow measurements and the CRI palpation, showing that cranial manipulations affect these objective parameters. Considering existence of serious studies suggesting that PRM/CRI could exist and that a trained practitioner could feel it, we did recently a

structured systematic literature in MEDLINE, Science direct and Cochrane Library on this topic in order to decipher the various hypothesis of the physiological mechanisms supposed to underlie the PRM/CRI rhythm[1]. This systematic review and Figure 1 show that the question of the physiological basis of this rhythm is still controversial. The initial model proposed by Sutherland is no longer consistent with current knowledge of physiology and physiological explanations behind this phenomenon remains elusive. The results of our previous systematic review highlighted the need for a paradigm shift and for more rigorous evaluation and communication of a model that is in line with the evolution of scientific data. This prompted us to write the present narrative review, which takes into account other articles published in the domain of the life sciences that can contribute to a better understanding of mechanisms underlying the PRM/CRI. After a first part re-affirming that the Sutherland model is no longer compatible with current scientific knowledge, we investigate the putative roles of vasomotion, autonomous nervous system, microcirculation, lymphatic system, extracellular matrix, fascial network, and entrainment on the genesis of PRM/CRI and we propose an integrative model and a paradigm shift taking into account all those parameters.

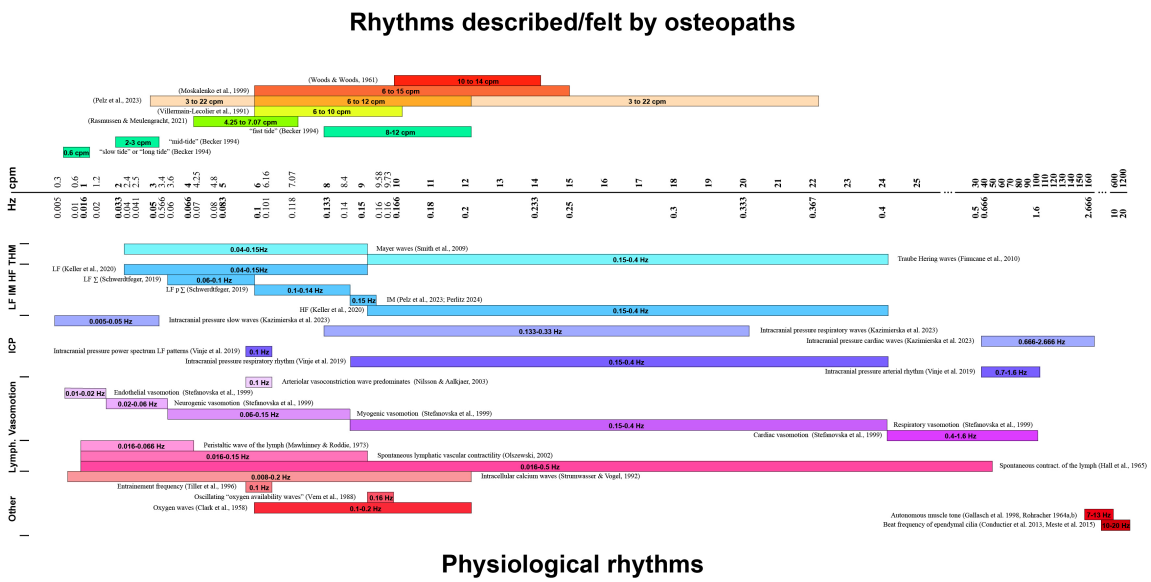


Figure 1. The following presentation will outline the various rhythms and frequencies (expressed in Hz and cpm) described by osteopaths, in comparison to those of physiological rhythms. These rhythms will be grouped into the following categories: Traube Hering and Mayer waves (THM); Low Frequency (LF); 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 developed by Tricco et al. for conducting a narrative review following the PRISMA-AcR Statement[21]. The theoretical framework for this scoping review was developed by a team of three experts (F.M., L.S., A.G.) specialising in scientific research (L.S., A.G.) and the teaching and clinical practice of osteopathy (F.M., L.S.). The present framework is based on the results and conclusions of the article by Mériaux et al.[1].

Research question: What is the relevant scientific data to assess whether contemporary research supports or challenges the empirical PRM model described by Sutherland[2][3]?

Research strategy: A systematic literature search was finalised in October 2023 in the indexed electronic databases MEDLINE, ScienceDirect and Cochrane Library[1]. This study identified eight areas of research that could provide scientific explanations for CRI/PMR complementary to Sutherland's five pillars:

Lymph; Vasomotion or vasomotricity; Traube-Hering and Meyer waves; Heart rate variability; Ventilation frequency; Extracellular matrix; Metabolic hypothesis; Embryology. An exhaustive documentary search was conducted between September 2024 and February 2025 in the PubMed, ScienceDirect, Ostmed.DR and Google Scholar databases. The search terms were adapted to each database: 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 systems; extracellular matrix; training; embryological movement; ideomotion; intracranial pressure. The research was conducted without limitation as to the design of the study, the population, the results or the date of publication. The selection of articles was made by examining the abstracts of eligible articles to determine their relevance first, and then by studying the full versions of the articles. The reference lists of the identified articles were also examined and a snowball sampling approach was used to discover other relevant studies.

Eligibility criteria: In order to ensure comprehensive coverage of the available research, no formal assessment of the validity of the selected scientific articles was conducted. The relevance of each article to the subject of the study was critically evaluated during brainstorming sessions between the experts (F.M., L.S., A.G.).The articles were analysed, and the physiological mechanisms explaining and/or invalidating PRM were identified in order to answer the research question. Where necessary, the articles were integrated into several categories.

Following the identification of the relevant studies, a total of 157 articles were analysed and grouped according to their pertinence in the ensuing chapters: The inherent motility of the central nervous system and spinal cord; Fluctuation of the cerebrospinal fluid; Motility of the intracranial and intraspinal membranes; Joint mobility of cranial bones; The involuntary movement of the sacrum between the iliac bones. The 'core link' hypothesis; CRI/PRM and Vasomotion; CRI/PRM and Autonomic Nervous System THM waves and Heart Rate Variability; PRM, microcirculation, lymphatic system, extracellular matrix and fascial network; PRM and Entrainment; PRM and embryologic movement. The articles were identified through a snowballing process, whereby articles identified in the systematic review by Mériaux et al.[1] were expanded upon, based on the authors' knowledge and the references of the scientific articles included[1]. The results suggest that the five pillars described by Sutherland and Magoun to define PRM may be an empirical model without scientific basis. Nevertheless, the extant literature review identifies several rhythmic physiological mechanisms, including vasomotion, heart rate

variability, microcirculation and Traube-Hering and Mayer waves, which have the potential to make a scientific contribution to the development of a new model.

III. The Sutherland's theory Results

Sutherland's model is firmly anchored in the literature. However, we will show in this section, that this model does not align with contemporary scientific understanding.

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

Since 1882, many anatomists observed and described the continuous and cyclic movement of brain mass and medulla corresponding to a systole and diastole, that according to Sutherland could act like a hydraulic pump activating the cerebrospinal fluid (CSF)[12]. Later, Magoun[2] then Upledger[15] thought that the rhythmic production of the CSF by the choroid plexus moved the brain mass and not the opposite. According to this Upledger's "pressurestat" model, the CSF would be secreted twice faster than reabsorbed, considering that some captors in the cranial sutures would detect a maximal pressure threshold, and by a feedback mechanism, would stop secretion, pulling the pressure down until a minimum. Then, secretion would reactivate to restart a new cycle. This model is not valid as the production of the CSF is nor intermittent nor rhythmic[10]. When the pressure increases, the reabsorption increases simultaneously with the exchange surface of the arachnoid villosities in the venous sinuses. This theoretical description is known as the Monro-Kellie "static" doctrine[22][23]. Under normal circumstances, the volumes of the three main intracranial components exist in a state of equilibrium such that increases in the volume of one component are balanced by decreases in the volume of another. However, this equilibrium is not immediate, as volume variations are dynamic, generating intracranial pulse waveforms[24]. Variations in the intracranial pressure waveform (ICP) are used in the monitoring of head trauma and hydrocephalus, and their components can be analyzed at the level of the scalp using non-invasive tools such as mechanical sensors placed in contact. These sensors detect beat-by-beat micrometric deformations of the skull[25][26][27]. The measurement of tiny skull expansions as a reflection of increased ICP was explored by Pitlyk et al. as early as 1985[28]. In the time domain, the ICP signal consists of three overlapping and superimposed components, but they are separated in the frequency domain by different oscillation periods into: slow waves (0.3-3 cpm); respiratory waves (8-20 cpm); and cardiac waves (40-160 cpm)[29][24]. In their study, Vinje et al.[30] measured the intracranial pressure gradient (ICP) obtained between two intracranial pressure transducers. The resulting power

spectrum showed two peaks[30], one corresponding to the arterial rhythm, between 0.7 and 1.6 Hz (42-96 beats per minute) and the other to the respiratory rhythm between 0.15 and 0.4 Hz (9-24 breaths per minute). But interrestingly, Vinje also reported that the ICP power spectrum revealed low-frequency patterns below 0.1 Hz (6 cpm), but contributions to CSF flow were not taken into account in this study[30].

Using magnetic resonance imaging (MRI), Maier et al.[13] already observed a motion of the brain and CSF that seemed related with cardiac and respiratory activity. The brain mass and the medulla moved caudally and medially (2-3 mm) during the systole; the opposite occurred during diastole[11][14]. During an inhalation, the nervous system is pushed towards the skull and the brain is retracting, while during an exhalation, the predominant movement is caudal and the brain is expanding[31]. The origin of the movement of the neuraxis is then cardiorespiratory and its frequency very close to respiration rhythm[32], so a little faster than the PRM rhythm. Overall, the PRM would not be in direct relation with cerebral motion nor respiration.

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

The CSF is a biological transparent liquid surrounding the brain and spinal cord, between pia mater and arachnoid, in the subarachnoid space. It has a role of protection, and transport nutrients and wastes. The CSF is mainly secreted by filtration of the blood at the choroid plexus of the cerebral ventricles[33]. As a liquid, CSF is poorly compressible. Thus, it could push the membranes that would make the cranial bones move. The initial theory was that rhythmic pulsatile circulation of the CSF could be at the origin of the CRI/PRM. CSF flow is driven primarily by the cardiac cycle and is thus pulsatile. CSF movements are also influenced by respiratory movements, posture, pressure of the jugular veins, arterial pressure and physical activity[34][35][30]. The movement of the arterial wall is the main driving mechanism, through a process known as perivascular pumping and thus the velocity of the arterial wall corresponds to that of the CSF.

The volume of total amount is approximately 150 ml, renewed daily three or four times over (approximately 500 ml produced daily)[36]. The prevailing view is that the CSF comes down to the different ventricles, then into the subarachnoid space to be finally reabsorbed mainly at the cranial level by the lymphatic and venous system. We now know that the venous system only drains 30% of the total amount while the nasal lymphatic system is actually a major part of the cranial drainage of the CSF (40% of total amount) along the cranial nerves (cribriform plate of the ethmoid bone)[37]. Around 25% is

drained at the spinal level, by the arachnoid villosities of the spinal veins at the emergence of the spinal nerves. A small amount also diffuses slightly through the arachnoid and dura mater towards surrounding connective tissues and then venous and lymphatic networks[37][38].

Sutherland's theory posited that the CSF was in continuity with the rest of the organism, extending towards the lymphatic system, perivascular and perineural pathways. This hypothesis suggests that the perception of the rhythm of the CSF may be experienced anywhere on the body[2]. However, according to Sakka et al.[38], there is no continuity between the CSF and the periphery as the subarachnoid space narrows near the proximal pole of the ganglion and terminates in a clearly anatomically defined cul-de-sac. The continuity happens with the perivascular or Virchow-Robin spaces, which seem rather to drain the interstitial fluid of the brain. The trabeculations and ligaments of the arachnoid slow down and impede the flow of the CSF, making the movement rather anarchic, and the circulation very slow (1cm/hour) and neither constant nor linear. However, advances in technical and methodological performance have now demonstrated that CSF diffuses in the peripheral nervous system (PNS). A study by Ligocki et al. demonstrated that CSF follows the distribution of 1.9 nanometer gold nanoparticles from the CNS into the PNS at root attachment/transition zones and distributes into the perineurium and endoneurium, eventually reaching the axoplasm of distal peripheral nerves[39]. Pessa et al. [40] demonstrated that ventricular perfusion and nanoprobes can be used to identify CSF flow and glymphatic circulation within the neural sheaths of human nerves. CSF flow within nerves can be conceptualized as an open circulatory system[40]. There seems to be no truly continuous, palpable flow, no ebb and flow as familiar to osteopaths, no rhythm in CSF production or drainage. Within the cranium and spinal canal, the circulation of the cerebrospinal fluid (CSF) is subject to fluctuations in intrathoracic pressure, which is induced by ventilation. During ventilation, CSF flow serves to counterbalance venous blood flow. Inspiration, whether voluntary or forced, leads to an increase in the volume of CSF flowing towards the head, with subsequent expiration resulting in a caudal shift in flow[41][32]. These observations testify to a close coupling between the CSF and the venous system, which reacts to pressure variations induced by respiration[41][32]. The velocity varies depending on the location: it is slowest at points farthest from the production site and the cranial arteries, with minimal or no flow observed at the lumbar level[36].

In conclusion, an expansion/retraction movement of the brain is related to respiration[30] rather than to variations in CSF pressure that are actually too low to influence cranial system[10]. On top of that, there is

no fluid continuity between the CSF reservoir and the periphery of the body at any point. It therefore seems difficult to attribute CSF movements to the CRI/PRM perceived by osteopaths. The CSF is of significant importance in the philosophy of cranial osteopathy and, by increasing fluctuations, could play a crucial role in improving a patient's condition. However, it does not appear to be at the origin of the PRM discussed here.

III.3. Motility of the intracranial and intraspinal membranes

The dura membranes are a part of membranous envelopes called the meninges that surround the brain and CNS. The dura mater attaches to certain cranial bones intracranially and can have external portions through the sutures of the skull[42]. In addition, the dura attaches to upper cervical bones and the sacrum, and there are attachments all along the spine[43][44][45]. In the skull, the cerebral falx and the tentorium cerebelli are part of the dura mater system and form a structure that holds all the bones together. Sutherland called this the 'reciprocal tension membrane' of the human skull. Sutherland believed that variations in the tension of the dura mater would play a role in the displacement of the bones to which it is attached and that there could be no reciprocal mechanism if it were not continually taut[3]. The cranium would move in rhythm, through a so-called inhalation and exhalation phases, driven by the motion of the shifting tensions in the reciprocal tension membranes. The dura mater has an average thickness of one millimeter, with anisotropic viscoelastic properties (therefore, it is not inextensible as some osteopathic theories claim)[46].

The findings of Al-Habib et al.[47] are consistent with fundamental anatomical knowledge that the dura mater possesses the greatest degree of elasticity, followed by the pia mater and finally the spinal cord[47]. They also demonstrate that the compressed spinal cord exhibited significantly greater rigidity in comparison to its uncompressed counterpart[47]. These results are consistent with the findings of Royo-Salvador et al.[48][49] on craniocervical growth conflict, where it was demonstrated that stretching of the filum terminal can result in compression of the spinal cord, as well as traction, thereby pulling the cerebellum into the foramen magnum. The detachment procedures for the filum terminal have been associated with cranial displacement of the conus medullaris[48][49].

The meninges transmit mechanical forces from the outside to the inside (trauma), and from the inside to the outside[3]. Moreover, they are structures that reflect the mechano-metabolic variables of the environment in which they reside, changing their ability to handle stress and modifying their intrinsic

structure over time[50]. According to Bordoni et al., meninges would therefore be able to adapt to stresses and strains, and would not appear to be rigid enough to pull the bones, except when they tend to ossify (which can happen with age, especially in the sinuses of the dura) affecting the transmission of mechanical forces[50]. However, looking at the current data, it could be assumed that if a tension (pressure or traction) comes from the brain to the skin, the different layers should dampen and slow down the propagation speed of the tension forces produced[51].

According to Bordoni[50], the viscoelastic properties of the fascia would dampen the wave from the skull to the periphery in the same way as the wave from the inside to the outside of the skull. Consequently, the wave of the CRI/PRM should also decrease with distance from the vertebral axis. However, osteopaths using the PRM/CRI usually observe and describe an equal, even synchronous, frequency at all points of the body, thus this hypothesis appears to be wrong.

III.4. Joint mobility of cranial bones

The first hypothesis proposed in 1939 by Sutherland was that PRM felt at the cranial level was due to movement of the cranial bones pulled by the membranes, themselves pushed by the fluctuation of cerebrospinal fluid. He thought that the skull could keep a relative flexibility at the level of skull sutures in the adult. Historically, cranial bone motion was considered as an anatomic impossibility and it is still the most controversial phenomenon of the PRM[52].

The main argument of the defenders of the skull bone mobility theory is that the sutures that articulate the different parts of the skull never ossify completely[53][54]. Although suture closure is a gradual process, bony spicules appear well before complete suture fusion[54]. The stiffness of the human skull increases with age with a huge variability between individuals and between sutures[55]. Calcification of most sutures begins at age 20 or 30 years[56] and the end of this process is extremely variable according to the individuals and the suture types[57][58][59]. Beyond the age of 60, most sutures are completely ossified[60]. By contrast, some osteopaths do not report any particular difficulty in perceiving or reviving CRI /PRM in the elderly[61]. The amplitude and strength of the PRM do not seem to diminish progressively with age, as would be expected if it were indeed linked to a decrease in articulation mobility within the sutures as they progressively fuse over time.

In the osteopathic cranial model, the joint that represents the engine of cranial biomechanics and to which the different dysfunctions imagined by Sutherland are attributed, is the synchondrosis between

the occipital bone and the sphenoid bone, the famous sphenobasilar synchondrosis[2][3]. The sphenobasilar synchondrosis begins to undergo an ossification process before puberty, with an intracranial departure, to end within the pubertal cycle[46]. Sutures in the human chondrocranium fuse progressively during infancy and adolescence, reaching complete fusion once the head is fully grown[60][62][63]. Thus, the adult skull has an ossified sphenobasilar synchondrosis and, from a scientific point of view, it is not possible to think of this joint as the principle of cranial movement or as the cause of the various dysfunctions described in osteopathy and explanations for manual approaches to release this ossified joint should be reconsidered[46].

Some authors have attempted to prove that common pressure applied by the practitioner (5 to 10g) could move the sutures. Downey et al.[64] hypothesized that low loads applied to the frontal bone of anesthetized rabbits, simulating the frontal lift osteopathic technique, would cause significant changes and movement at the coronal suture but failed to prove it. A force of 50kg should be applied to the rabbit skull to move a 1 mm suture, for a level of ossification comparable to a human aged 20-30[65], and 15kg for a child human, which is much more than the 5 to 10g recommended in cranial osteopathy[66]. Scientists have long assumed that the cranial bones are fused and cannot move, but there is now considerable evidence that there is a cranial flexibility[67][55] like the rest of the body tissues. Bone tissue has viscoelastic properties as it contains elastin and collagen and together they ensure a capacity of mechanical deformation of about 10%-15%[46]. Starkey[61] describes the anatomy of sphenoïd clivus and show that it is spongy and therefore malleable, hard and thick and therefore immobile. Cook[68] evokes a flexibility rather than mobility of the skull bones while Seimetz et al.[55] suggest a possible motility of cranial bones.

Even though it is among the hardest materials in the body, bone distorts during normal function and more obviously during trauma. In many studies, cranial motion was induced by various internal and external stresses on the cranium across animal and human specimens. Using MRI, Crow et al.[69] indeed observed the variation of measurements of different points of cranium without human intervention/intention. They found statistically different values for area, width, height and major axis measurements, but not for perimeter and minor axis, which is not matching with the expansion and retraction felt by the osteopaths.

Sutherland's cranial model makes little reference to the action of muscles. This is possibly because he analyzed dry bones, as was the practice in other early osteopathic models. However, according to Gabutti

et al.[70], a large amount of research into the mechanical properties of cranial bones and sutures confirms that muscle contraction is one of the principal causes of cranial bone deflection[71]. Cranial sutures, due to their viscoelastic properties and to the constitution of the extracellular matrix surrounding them, rich in collagen fibers, proteoglycan and water, are obviously more flexible than the adjacent bone[70]. Rather than mobility, the sutures play a role in energy absorption and force transmission[72][73]. The biomechanical properties of the human skull/cranial suture differ according to age and layers (diploe, inner and outer plates)[74]. Sutures may affect the way the head distorts but the head still distorts even if they are fused and the density of the sutures (depending on their closure) could be a limiting factor in the amplitude of the movement[70].

To conclude, there is indeed a movement of the bones of the skull, but not necessarily between them. The measurements of the range of movement at the sagittal suture in the studies by Adams and Heisey are of the order of 300 μm[75][52]. Cranial mobility would thus be due to a deformability of the skull as a whole, under the influence of the intracranial pressure, the fluctuation of the CSF or the tensions of the membranes and of external forces such as the muscles of the head and their connective tissues, crossed by the blood vessels whose motility could deform the skull[76]. Indeed, Moskalenko et al.[76] measured rhythmic changes in skull shape and volume using serial NMR scans. They confirmed a rate of 6 to 15 cpm and the physical extensibility of the bony skull and observed an expansion of the skull of 0.2 to 0.4 mm immediately after the injection of 20 ml of liquid into the carotid artery, and thus verified the effectiveness of the increase in cranial volume[76].

Cranial mobility would thus be more a consequence than a component of the PRM, an effect rather than a cause, and we have to reconsider Sutherland's model. From the point of view of osteopathic treatment, we do not question the possibility of releasing, like any other fascia, a cranial suture from its tension, whatever its level of ossification. One could imagine, as in the article by Hamm[77], that a densification of the tissues by polymerisation of the collagen fibres solidifying the two edges of a suture and causing a fixation[77]. The cranial osteopathic treatment would then consist in softening the area as at other levels of the body but not necessarily in restoring a movement of the bones of the skull between them.

III.5. The involuntary movement of the sacrum between the iliac bones. The «core link» hypothesis

Sutherland suggested the dura mater as inextensible, so that the traction from the cranium in relation with the cranial bone motion would drive the sacrum motion[2][3]. However, if the dura mater were so rigid, could we bend over? In reality, it has an elasticity and experiments show that in a neutral position of the spine, the spinal hard-sheath has folds, suggesting that the tissues are relaxed[78]. The study by Al-Habib et al.[47] demonstrates that the dura mater exhibits a degree of elasticity that is double that of the pia mater, and threefold that of the spinal cord[47]. The length of the spinal canal is observed to vary between flexion and extension of the spine by 19.4 mm ± 6.4 mm[79]. Therefore, there are no concrete studies to prove this theory. Cella et al.[80] investigated the occiput–sacrum connection from a neurophysiological perspective. The sacral technique did not produce immediate changes on occipital brain alpha-band power. Conversely, the cranial technique "compression of the fourth ventricle" (CV4), as previous evidence supported[81] generated immediate effects, suggesting a different biological basis for osteopathic therapy's connection between the head and sacrum.

From this pillar emerges the idea of a unity and synchronization of frequencies observed at the level of the cranium and pelvis, and by extension throughout the body. Not all osteopaths agree on this point. Moran and Gibbons[82] were unable to demonstrate synchronization between palpated rhythms of the head and sacrum. Rogers et al.[83] showed no correspondence between head and foot rhythms when two examiners simultaneously palpated the PRM. It may be hypothesized that the results might have been different if they had first "rebalanced" their patients. It would be interesting to conduct further experimental studies measuring the PRM at different body sites, before and after osteopathic sessions, with the same and different practitioners, in a blinded fashion.

In view of results of Al-Habib et al.[47] and of Royo-Salvador et al.[48][49], in the case of a transmission of a movement propagating from the skull to the sacrum, this should be transmitted through the medula and the filum terminal, which is attached to the coccyx and not to the sacrum[47][48][49]. This does not correspond to Sutherland's model[2][3].

In conclusion of this part, we have shown that Sutherland's 1939 conception of the physiological basis of the CRI/PRM, still taught as a dogma, is challenged by the current scientific knowledge. We will now

review various hypotheses or new explanatory models that could be taken into consideration and confronted with scientific data to see a future consensus model emerge.

IV. CRI/PRM and Vasomotion

The PRM is a mechanism that produces a cyclical movement, perceptible under hands everywhere on the body, not only around the skull (CRI). Jones[84] first demonstrated in 1850 a vascular rhythmicity on the ventral surface of the wing of a bat, independent of the systolic and respiratory rhythms, corresponding to a spontaneous rhythmic variation in the diameter of the veins, induced by intermittent contractions of the smooth muscles of the walls of the micro-vessels, causing an active displacement of the blood in the form of a slow oscillation. He called this oscillation flowmotion, but we now use the term of vasomotion or vasomotricity.

Vasomotion corresponds to the capacity of blood or lymphatic vessels to contract or dilate, in order to modulate the flow, in response to pressure variations[85]. At the arterial level, the large vessels (aorta, large caliber arteries) present essentially passive responses because of the abundance of elastic fibers in their tunics (called compliance or arterial distensibility), which serve to mitigate the consequences of ventricular systole on blood pressure (BP)[86]. In diastole, the large trunks regain their diameter like a deflating balloon, which limits the decrease in blood pressure[86].

Two other mechanisms, linked to the presence of Vascular Smooth Muscle Cells (VSMC) are present only in small resistance arteries and arterioles whose media (medial layer) is rich in VSMC, contrary to capillaries and large arterial trunks which are devoid of them. The first mechanism is myogenic and the second neurogenic often source of confusion in the literature when authors do not precise which type they are referring to. Vasomotion is of particular importance in the study of vascular disorders, as evidenced by the findings of Fredriksson et al.[87][87]. Their research, which utilized near-infrared spectroscopy (NIRS) to measure weak and very weak spontaneous haemodynamic oscillations (0.003-0.15 Hz), corroborated the results of Stefanovska et al.[88]. The latter study demonstrated a distribution of vasomotion frequencies as follows: 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]. First, let us talk about the myogenic mechanism, which seems to be the most considered in the study of vasomotion. The small diameter arteries contract in response to an acute increase in blood pressure and conversely relax when blood pressure lowers[89][90]. At the arteriolar bifurcations, cells show a pacemaker activity generated by the

spontaneous oscillation of the intracellular Ca^{2+} ion concentration in the smooth muscles located in the vessel walls themselves[91][90][92]. These cells also respond to various signals, of adrenergic (vasoconstrictors), cholinergic (vasodilators), metabolic (pH, CO_2, ATP, NO and O_2), humoral (prostaglandins, EDRF, etc.) and myogenic (temperature and local muscle pressure) origin[90]. In the article by Colantuoni et al.[93] on the study of arteriolar vasomotion and blood flow regulation, the main characteristic of arteriolar microvascularisation was identified as variations in diameter as a function of time, the fundamental frequencies of which were determined by spectrum analysis. The fundamental frequency was found to range from approximately 4 to 15 cpm in arterioles of rank 1 (8 micrometers) and from 2 to 11 cpm in arterioles of rank 2 (10 to 13 micrometers). Vessels of orders 3 and 4 (20 and 30 micrometers, respectively) exhibited a fundamental frequency ranging from 0.5 to 6 cpm and from 0.3 to 3 cpm, respectively[93]. The oscillations are synchronized by electrical phenomena related to the oscillation of the cell membrane potential (polarization-depolarization), which is transmitted to all gap junctions coupled cells, even those without a pacemaker function[94][95]. Longitudinal conduction of vasomotor responses is a vital process that enables the orchestration of alterations in diameter and flow distribution among vessels. It is also instrumental in the coordination of vascular resistance by integrating the functions of proximal and distal vascular segments within the microcirculation[96]. Neurogenic influences are not essential to it, but adrenergic stimulation reinforces them. This variation is therefore neither rhythmic nor cyclic nor synchronous with neighboring vessels. It is rather a localized and intermittent phenomenon, making it difficult to directly associate with the PRM. Nilsson and Aalkjaer[91] observed 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] concludes: “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 under conditions of reduced perfusion and is initiated by metabolic stress but is usually absent under resting conditions[94]. Vasomotion may be present only for brief periods[77]. As a conclusion, the vasomotion due to myogenic mechanism could influence the CRI/PRM but cannot be its only explanation. If it does play a role, its unpredictable characteristics may explain many of the criticisms regarding inter/intra-examiner reliability testing.

However, Nilsson & Aalkjaer[91] observed rhythmic vasoconstrictions, probably of neurogenic origin, the largest transverse arterioles having the lowest frequencies, ranging from 0.3 to 3 cycles/min with a diameter variation amplitude of between 5% and 20% of their mean diameter[91]. Arterioles of around 18μm diameter have amplitudes of variation of 15% to 50% of the mean diameter and frequencies of 0.5 to 6 cycles/min. Arterioles of around 11 μm in diameter have amplitudes of variation of 50% to 100% of the mean diameter and frequencies of 2 to 11 cycles/min, and, the smallest so-called terminal arterioles, the most superficial, have the fastest frequencies (4 to 15cpm), and the largest amplitude since they vary from 60% to 100% of the mean diameter. Interestingly, in their study, carried out in hamsters, the authors Nilsson & Aalkjaer,[91] underline a predominance of 0.1Hz waves, transmitted at the beginning of medium-sized arterioles (type 3 according to Strahler's classification) and reverberating into the smallest arterioles (type 2 and 1), a frequency similar to the PRM[91]. This suggests that there is a superposition of different arteriolar rhythms, the easiest to perceive by the osteopath's hand being probably the most superficial ones impelled by the smallest arterioles.

Villermain-Lecolier et al.[98][99][98] described the existence of periodic micro-movements perceived at the level of the skin with an amplitude of about 20 to 50μm, with rhythms differing from the cardiac and respiratory rates, also measured on the subject. The frequency of these movements was 9.73, ± 3.48 cpm. According to them, the PRM was the manifestation of local vasomotricity, and it was not necessary for it to be synchronous at any point in the body. Like previously mentioned, there is obviously no consensus among osteopaths on this issue.

Vasomotricity ensures a better blood circulation and a better perfusion of the tissues and thus contributes to a better vitality of the organism. Exploring a link between PRM/CRI and vasomotion aligns with a fundamental principle of Osteopathy: the importance of circulation in maintaining health, as emphasized by Still[100][100].

Vasomotion could be part of the PRM/CRI underlying mechanism, but likely not the only one as we will see in the next paragraph.

V. CRI/PRM and Autonomic Nervous System

V.1. CRI/PRM and THM waves

Vasomotor waves or Traube-Hering Mayer (THM) waves are normal physiologic waves that are generated by spontaneous pulsations of arterial, venous and lymphatic vessels. Traube–Hering waves are formed by interaction of respiratory sinus arrhythmia and pulse pressure modulation in healthy men[101]. THM waves are mediated by the autonomic nervous system and along with increased heart rate variability, are considered to be markers of good autonomic balance[36].

Neurogenic mechanisms influencing vasomotion are present under normal conditions. This mechanism generates rhythmic and synchronous contractions activated by the SNS and causes these oscillations in blood pressure, called Mayer waves[102]. These waves are also baroreflex responses to blood pressure variations[103], and are therefore the result of a resonance phenomenon in the sympathetic baroreflex loop[104].

Afferent signals from the conductance arteries would initiate a baroreflex feedback loop, relayed through the Nucleus Tractus Solitarius (NTS) and transmitted via sympathetic vasoconstrictor nerves (ganglionic chain) to the arterioles, thereby triggering vasomotion associated with Mayer waves[91]. The NTS is located in the floor of the IVth ventricle, interestingly, the same location as the so-called "occipital compression" or CV4, a well-known technique in cranial osteopathy to reviving the PRM. In regional circulations, Mayer waves are transmitted to the conductance arteries[102], although attenuated by local compliance, and are detectable in certain peripheral vascular beds, for example in the fingers, which can be measured by photoplethysmography (PPG)[105]. These Mayer waves band (0.04-0.15Hz) correspond to the low-frequency (LF) component of heart rate variability, associated with stimuli coming from baroreceptors and chemoreceptors in the carotid sinus circulating through the SNS to the vascular smooth muscle cells (VSMCs), which is added to the high-frequency (HF) component or sinus respiratory arrhythmia, also known as the TH (Traube Hering) wave band, synchronous with respiratory movements (0.15-0.4 Hz)[106].

Mayer waves show a strong, significant coherence (strength of linear coupling between fluctuations of two variables in the frequency domain) with efferent sympathetic nervous activity[107]. Within a given biological species, their frequency is quite stable; in humans it has been shown that this frequency does not depend on gender, age or posture[107], but to a large extent, on the delay in the vascular response to a

change in sympathetic nerve activity. This delay depends itself on the length of the post-ganglionic sympathetic neurons (lower conduction A-myelinated fibers), and therefore, *in fine*, on the size of the animal considered. The larger the animal, the longer the length of the fibers, the longer the delay, the lower the frequency (e.g. 0.1 Hz in humans, 0,3 Hz in rabbits and 0.4 Hz in rats)[108][102]. This is also an observation made in animal osteopathy with regard to the frequency of the PRM: the larger the animal, the lower the frequency.

Fernandez & Lecine were likely the first to record the vasomotor wave while simultaneously comparing it to cranial palpation[109].

The group of Nelson et al.[17][17] also showed a good correlation (N 328 data pairs; correlation, 1.00; sig., 0.00) between CRI palpation and the recording of the THM wave (2:1 ratio) by means of a laser-doppler, which measures the speed of blood flow by change of speed of erythrocytes (hemoglobin) in the subcutaneous capillaries[110]. Like other authors, they confused the frequencies of Traube-Hering waves, Mayer waves, and both mixed (THM) and it was not obvious to report clarity on the subject. They concluded that the PRM/CRI and the THM oscillations were simultaneous, if not the same phenomenon.

It opened new possible explanations for the basic theoretical concepts of the PRM/CRI and cranial therapy. They ascribed the phenomenon of a 'still point' as a brief cessation in the rhythm of the PRM in 79% of cases with diminished TH amplitude. Hamm explains that McGrath, reviewing Ferguson's paper, proposed a similar explanation[77], suggesting that CRI is "a manifestation of an extracranial blood flow phenomenon".

Christ et al.[111][111] measured cyclic changes in limb volume in the CRI range and suggested that they were due to changes in blood pressure, arteriolar vasomotion, and possibly lymphatic diameter. They found a correspondence between cyclic increases in arterial volume and changes in the volume of the limb (<0.177 ml per 100 ml tissue). Muscles are essentially incompressible[112] and there are no empty spaces in surrounding tissues that could absorb these changes in volume, which means that a cyclic dilation and contraction of these fascial tubes in sympathy with changes in vascular volume (both intra- and extra-muscular) might be able to influence the crossed-helical fiber configurations within their walls in a particular way[113]. If this is indeed linked, it could explain the helical sensation (external and internal rotation) that osteopaths have when they perceive PRM on the limbs.

At a cranial level, Abenavoli et al.[114][115] concluded that osteopaths could detect a change in CRI amplitude after applying the Q test (Queckenstedt test, a bilateral compression of the internal jugular

veins that produced an increase in intracranial pressure), showing that the flow and pressure of veins and arteries could be part of the origin and perception of CRI/PRM.

Mayer waves are abolished, or at least strongly attenuated, by pharmacological blockade of alpha-adrenoreceptors[102], thus the hemodynamic basis of Mayer waves would be oscillations of the sympathetic vasomotor tone of arterial blood vessels. Conversely, acute beta-adrenoceptor blockade has minor, if any, effects on these oscillations[108][116] suggesting a low impact of the vagal control.

Uremia would also result in a dramatic reduction in Mayer wave amplitude, which could indicate altered cardiovascular autonomic nervous system function[117]. It might be interesting to study the potential variation in PRM in patients under such conditions.

In conclusion, Traube Hering and Mayer waves have to be differentiated one from another, and they both could contribute to the CRI/PRM.

V.2. CRI/PRM and Heart Rate Variability (HRV)

The heart does not beat with the regularity of a metronome: the time between two consecutive heartbeats varies continuously and is known as HRV[118]. HRV is a recognized indicator of an individual's autonomic nervous system health[119]. In general, a high HRV can be considered a sign of a healthy, adaptive autonomic nervous system, which readily adjusts heart rate in response to changes in the internal or external environment[120]. Conversely, low HRV is found in a variety of pathological conditions, including cardiovascular disease and diabetic neuropathy, and indicates a decreased ability of the autonomous nervous system to respond to environmental changes[121]. HRV analysis is a noninvasive objective measure that can be used in experimental settings to measure the modulation of the ANS, thus providing information about the levels of sympathetic and parasympathetic activity. In contrast to the concept that the sympathetic and parasympathetic systems act holistically and antagonistically, it is now recognized that the two systems work in a coordinated manner, sometimes synergistically, 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, there is variability in the definitions of these HRV band boundaries, which describe cardiac ANS regulation. HF-HRV, associated with respiratory sinus arrhythmia, increases with heightened parasympathetic (vagal) activity[123]. In contrast, LF-HRV reflects both sympathetic and parasympathetic influences and may represent an independent physiological

mechanism in addition to its dominant parasympathetic contributions[123]. Some studies propose that the LF band reflects subharmonic activity of a central pacemaker located in the lower brainstem[123].

The LF/HF ratio (low frequency to high frequency) was long regarded as an indicator of sympatho-vagal balance. However, this stance has faced significant criticism. Notably, since the LF band does not reliably reflect sympathetic activity, there is now a consensus that the physiological mechanisms underlying the LF/HF ratio remain unclear, making the interpretation of LF/HF data highly problematic[124]. Commonly reported measures of vagally mediated HRV, the high frequency HRV (HF-HRV), the low frequency HRV (LF-HRV). More recently, an intermediate (IM) frequency band (an activity emerging in various peripheral systems in humans during hypnoid relaxation), was included in systemic physiological ANS analyses of forehead skin perfusion (SP), ECG, and respiration[123][125]. Preliminary evidence suggests that those 0.15 Hz waves originate in the brainstem and travel to skin microvasculature through parasympathetic nerves[123][125]. Schwerdtfeger et al.[126] also suggest two rhythms in the LF band, one lower between 0.06 and 0.1 Hz and one higher between 0.1 and 0.14 Hz. They propose that the lower band mainly reflects the regulation of blood pressure by the baroreflex (sympathetic nerve fibres) and that the upper band could provide information about the complex interaction between parasympathetic and sympathetic efferences that signal interactions between the heart and the brain. Pelz et al.[9] showed that skin blood flow exhibits parallels with the PRM/CRI using photoplethysmography (PPG) measurements, suggesting that CRI/ PRM could be a manifestation of this '0.15 Hz rhythm band' physiology, also referred to as intermediate (IM) band. Therefore, HRV could also contribute to CRI/PRM[127].

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

VI.1. Microcirculation

Approximately 10 to 20% of the fluid from the capillary bed that enters the interstitial space is returned to general circulation through the lymphatic system, while the majority (80 to 90%) re-enters the capillaries and exits the region through the venous system[128]. Venules have a larger diameter and thinner muscular walls compared to arterioles. These venules are controlled by the sympathetic nervous system, allowing their walls to contract and relax. This action plays a significant role in the vascular system's capacitance, the regulation of tissue perfusion, and serves as a physical mechanism for the

frequency modulation observed in the TH and M waveforms[129][130][128]. As the vascular system fluctuates with the TH and M oscillations and, in concert with arterial resistance, the venous capacitor is contracting slowly and regularly. This fluctuation facilitates simultaneously the fluid movement through the interstitium, the lymphatic circulation, and the return of venous blood to the heart[131][128]. Vasomotion resulting from the TH and M oscillations accounts for the negative interstitial pressure of Starling's equilibrium for capillary exchange[128].

Oscillating "oxygen availability waves" were also described in a study by Vern et al.[132] with an average frequency of 9.58 ± 0.117 cpm and in a study by Clark et al.[133] with a frequency of 6-12 cpm. These data suggest that the cyclic increases in cortical oxidative metabolism represent the primary local oscillatory process, followed by reflex hemodynamic changes that effect local tissue perfusion, and intracranial blood volume. Although this process does not occur synchronously throughout the brain, the relatively close frequencies of the cytochrome oxidase redox state and the THM oscillations could also allow the two processes to become entrained, thus linking local and central control of tissue perfusion[128][134].

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

Tissue fluid pressure is a determinant of fluid transfer between the blood and tissue spaces and between tissue spaces and terminal lymph vessels[135]. The lymphatics present rhythmic contractions (smooth muscles of the lymphangion's) at the origin of a peristaltic wave allowing the progression of the lymph, of variable frequency: mean of 2-2.5 cpm and that the value range from 1 to 4 cpm for Mawhinney and Roddie[136] [136]; 4 to 5 cpm for McHale et al. in bovine mesenteric lymphatics[137]; 4.8 cpm in the popliteal vessels of sheep[138]. Lymph vessels demonstrate spontaneous contractions varying from 1 to 30 cpm[139]. Olszewski[140] reported spontaneous lymphatic vascular contractility at a rate of 1 to 9 (average 4) cpm that was independent of arterial pulse rate, respiration, and body movements. Each lymphangion is also capable of spontaneous independent contractility, by a same myogenic response mechanism as the one described before for arteries[128]. Although lymphangions may contract randomly through a pacemaker located in the lymphangion wall just proximal to the valve, they function more efficiently when contracting synchronously and lymphatic vessels tend to develop synchronous activity easily[141] [128]. Again, THM driven entrainment probably insures optimal efficiency of this aspect of the mechanism. The authors describe human and animal research showing that a rhythmic spontaneous lymphatic contraction occurs in many species, including humans. For example, the thoracic duct in

humans contract once every 10-15 seconds[142] roughly four pulsations per minute, close to the CRI rate. Perrin[143] also suggests that the PRM could correspond to a rhythmic contraction of lymphatic vessels.

Ferguson[10] claimed “As there are not large lymph vessels within the cranium it is unlikely that the lymphatic system causes the CRI”. However, since then, the group of Kipnis showed that there are lymphatic vessels surrounding the brain[144]. In any case, it is worth seeking the origin of the PRM in the skull? Indeed, patients with a weak or imperceptible CRI/PRM at the skull level but well-expressed at the sacrum or elsewhere in the body would suggest the opposite.

Glymphatics is the study of the process of circulation, accumulation, and clearance of glymph, a fluid that circulates in the brain interstitium. The glymphatic system is a complex physiological process involving three distinct phases. Firstly, cerebrospinal fluid (CSF) enters the perivascular spaces. Secondly, the CSF is then mixed with the interstitial fluid (ISF). Finally, the fluid that has travelled through the brain tissue is cleared[145]. Brain cells, both glia (oligodendrocytes and astrocytes) and neurons, have been shown to present rhythmic swelling/deflating initiated by variations in intracellular Calcium ion concentration and the accompanying water[16][7][132]. However, the oligodendrocytes contraction measured by Pomerat et al. [146] that were referred to in Retzlaff and Mitchell[147] to had a much slower frequency (of 4 to 18 mn) [147] [146]. The cycles of these intracellular calcium waves were recorded at frequencies ranging from 0.5 to 12 cpm in concordance also with the rhythm orchestrated by the THM waves and thus the autonomic nervous system[148], both in vitro and in vivo. These ion transfers are also associated with alterations in the viscosity and charge of the matrix.

In addition, the capillary perfusion pressure, influenced by the THM waves, cause a rhythmic variation of the gel/sol viscosity of the extracellular matrix, which is a colloid with viscoelastic properties. Indeed, Lee[7] describes how the microstructure of the fascia is made up of a network of proteoglycans (PGs), glycoproteins (GPs) and glycosaminoglycans (GAGs) forming a sieve through which all dissolved metabolites pass from the capillary to the cell and vice versa. The PGs are negatively charged, bind water and naturally, due to their unstable thermodynamic properties, form a gel. The whole structure allows the extracellular matrix to act as an essentially unstable semi-conductor, a liquid crystal, which, according to Hamm[77] could be a hypothesis to explain the palpatory experience and therapeutic claims in the practice of osteopathy in the cranial field through a cyclic texture change of the ECM, which could be altered and densified by localized fascial tensions.

In summary, blood flow is also modulated by vascular compliance and resistance but also ultimately that of the surrounding tissues, namely the local density of the ECM. The ECM can sclerotize under the strain of fascial tensions, which lead to densification and polymerization of collagen fibers and voluminous hyaluronic acid aggregates[149]. These processes can also be part of the PRM underlying mechanism.

VII. PRM and Entrainment

Although Ferguson[10] and Perrin[143] indicated that cerebral veins have neither valves nor muscles in their thin walls, and therefore no vasomotion, the rhythm perceived by osteopaths could come from superficial and subcutaneous vessels rather than deep and cerebral ones.

Within the range of those described by the osteopaths, there is a great variety of different rhythms, and there is a possibility that some osteopaths could feel the resultant of whole rhythms while others could feel only some of them. All these rhythms are likely superimposed and probably influence each other, and what the osteopath perceive with his fingertips is likely the resultant of all these oscillations. This is the theory developed by McPartland and Mein[150], suggesting that the PRM is a rhythm produced by the synchronization, or entrainment, of multiple biological oscillations of both patient and practitioner. This idea was also explored by Norton[151] with his tissue pressure model, who proposed that the CRI is associated with slowly adapting cutaneous mechanoreceptors of both the patient and practitioner, and that the sources of change in these tissue pressures are the combined respiratory and cardiovascular rhythms of both examiner and subject. This could explain why different examiners often do not agree in their determination of the CRI frequency on the same subject, as the resting heart rates and respiratory frequencies of the examiners may be quite different[151]. Tiller et al.[152] [152] describe a significant event arising in biological oscillators when sympathetic and parasympathetic systems become balanced. The body's myriad rhythms, including HRV, THM, respiration rate, pulse transit time and even brain waves, all coordinate into harmonics with each other. Together they form a primary, fundamental frequency, which the authors termed the entrainment frequency[150]. The entrainment frequency measured in healthy human subjects with balanced sympathetic and parasympathetic systems is about 0.1 Hz (6 cpm), and could correspond to the PRM[152].

If balance is present within the autonomic nervous system, then the body’s many rhythms harmonize into a strong, coordinated, sinusoidal fluctuating entrainment frequency, which can be palpated as a strong, healthy CRI/PRM. In a healthy state one would expect synchrony of all tissues in that all are

entrained to a common innate rhythm. Therefore, vitality, as assessed by evaluating the CRI, would depend on sympatho-vagal balance[153].

Dysfunction of the autonomic nervous system may change the entrainment frequency, causing perturbations in the PRM/CRI, altering its rate, strength and amplitude[150]. In the presence of severe dysfunction, the body's rhythms may not synchronise, resulting in an undetectable PRM/CRI. It should be possible to determine experimentally if entrainment frequency corresponds to the PRM rhythm.

VIII. PRM and embryologic movement

Another model called Biodynamic has been developed by Rollin Becker in the 1930s, and later on James Jealous[150]. When studying the writings of the embryologist Blechschmidt, he was impressed by Blechschmidt's conclusion that embryonic function (fluid motion) creates form and precedes structure[154][155]. Blechschmidt carefully observed the cells of the developing embryo, which migrate as they differentiate. He used the term "Biodynamics" to describe these movements. Jealous hypothesized that the forces of embryologic development did not cease functioning at birth, but were maintained throughout our lives as forces of growth and development, and were also involved in healing processes. He described a rhythm slower than PRM/CRI, which he simply called the "$2^{1/2}$ cpm rate", with a frequency of 2.5 cpm[150]. In their article, often use Sutherland's termination, the "Breath of life" or the « therapeutic force », without mentioning a specific frequency nor origin, but emphasizing that it is different from CRI/PRM. This is why we chose not to further develop this approach, which is off-topic since we are focusing on the origin of PRM/CRI and not its harmonics or other rhythms outside its frequency range. However, it is interesting to notice that PRM may not be the final, fundamental frequency. Sutherland alluded to deeper, more subtle, harder-to-detect rhythms besides CRI[3]. His student, Rollin Becker[156], stated that CRI consists of different components, a "fast tide" (8-12 cpm), and a "slow tide" (0.6 cpm)[156]. We also find in biodynamic literature, the "mid-tide" (2-3 cpm), and the "long tide" (6 cycles every 9/10 minutes) that could be related or similar to the "Breath of Life". Thus, there could be different rhythms, which makes objective measurement and comparison complex, and may explain why intra- and inter-examiner reproducibility is difficult to achieve.

IX. Perspective

In this last part, we will discuss the use of this CRI/PRM in the osteopathic practice.

IX.1. PRM as an osteopathic tool

In summary, if PRM oscillations correspond to variations in the texture of the fascial network, it represents a valuable tool for manual therapists aiming to release myofascial tensions. Indeed, PRM can serve as a diagnostic tool by reflecting the level of tension in the area, and it can guide the practitioner's techniques towards tissue release, as its quality (strength and amplitude) would improves with the viscoelastic properties and deformability of the tissue. To validate this concept, could we compare objective Myoton measurements in different regions of a subject with subjective PRM palpation to assess their correlation? And what if we took Myoton measurements every second? Would we observe a cyclic variation? A frequency within the PRM range? This theory concerns loco-regional approach, but understanding the mechanisms and origins of PRM highlights the importance of treating the body as a unified whole-another core osteopathic principle. By first releasing the brain-heart connection and enhancing autonomic nervous system (ANS) function, the entire body benefits.

The osteopathic community has long theorized that osteopathic manipulative treatment (OMT) can restore body functions disrupted by musculoskeletal imbalances, by inducing autonomic activation, and leading to vasodilation, smooth muscle relaxation, and enhanced blood flow[157]. If PRM is related to autonomic activation, using it as diagnostic tool like many osteopaths do could indeed be useful feedback to make sure the technique just applied have made a significant change. Most studies generally indicate a reduction in sympathetic activity, coupled with enhanced parasympathetic modulation following OMT[124][158], which fosters relaxation and decreases sympathetic nerve activity. However, it is important to consider that any form of manual therapy capable of inducing relaxation, regardless of the specific method used, will influence ANS regulation. Furthermore, the psychological anticipation and experience associated with osteopathy can produce physiological effects, complicating assertions that a particular manual therapy technique directly causes specific outcomes[159]. Therefore, it is always better to compare with a placebo group, like Abenavoli et al.[115] did: trying to measure the physiological response of the sAA (Serum Amyloid A) levels after CV4 technique, they showed a positive effect that was not significantly different from the sham procedure[115].

CV4 is used for enhancing PRM and associated with decreased sympathetic tone and parasympathetic activation[81][160][19]. Nelson and al. demonstrated that CV4 amplified THM waves while simple cranial palpation didn't affect them so that it could serve as placebo[161][19]. Before that, Sergueef and al.[20] also showed that cranial manipulations, consisting of equilibration of the global cranial motion pattern and

the cranio-cervical junction, affect THM oscillations[20]. Pelz et al.[9] showed significant autonomic nervous system responses to the cranial vault hold (CVH), a specific osteopathic intervention, observing its effects on LF-HRV, IM band, and momentary frequency of highest amplitude (MFHA) of PPG signals. More recently, they observed a significant rise in the root mean square of successive differences (RMSSD) of HRV following CV4[162].

Others recent studies used RMSSD[163], skin conductance[164], or HF of HRV[165] to prove the effects of OMT on ANS.

Finally, Fornari et al. showed that a single osteopathic session to healthy participants induced a faster recovery of heart rate and sympathovagal balance after an acute mental stressor by substantially dampening parasympathetic withdrawal and sympathetic prevalence[166].

Cranial and/or cervical spine manipulation could lead to an enhanced autonomic balance through a reduction in sympathetic nerve activity and an improvement in heart rate variability, a measure of parasympathetic activity[167][168][169][36].

Collectively, these studies suggest that OMT may modulate the ANS by enhancing parasympathetic function and reducing sympathetic dominance and that PRM could be a useful diagnostic tool to make sure the technique just applied have made a significant ANS change.

When the body is balanced, the PRM characteristics of strength, amplitude and rhythm should theoretically be within the norm. It is therefore perceived as strong, around 6 cpm in humans, and symmetrical in its amplitudes of expansion/flexion/external rotation, and retraction/extension/internal rotation (globally, the tissues oscillate between opening and closing the body). Symmetry should also be sought between the limbs, suggesting synchronicity at all points of the body. If this is not the case, the body is then considered unbalanced, and osteopathic treatment is relevant; if it is, the patient is then considered balanced, and osteopathic treatment is over. This synchrony could make sense, considering that all tissues, harmonized on the tensegrity level, oscillate in unison at the entrainment frequency, as claimed by the principle of the unity of the body, so dear to osteopaths. However, this alleged synchronization between different parts of the body is not universally accepted by practitioners. Indeed, palpatory studies have failed to demonstrate a clear link between palpated rhythms at the head and sacrum[170][82][83]. Nevertheless, we can assume that this synchronization would be easier to demonstrate in balanced subjects who have undergone a prior osteopathic session.

IX.2. Ideomotion

Ideomotor movements can be described as micro-movements that we unconsciously and involuntarily perform, which are automatic expressions of dominant ideas rather than the result of voluntary efforts[171].

Applied to osteopathic manual medicine, the perceived movement may actually be generated by the therapist during palpation. The therapist's unconscious perceptions (such as expectations, degree of empathy, etc.) can influence decision-making and leave open the possibility for the therapist to fall into a self-fulfilling prophecy or "expectancy confirmation effects"[172]. In their literature review, Shin et al.[173] [173] explore several historical perspectives on the concept of ideomotion, which could offer valuable insights into understanding PRM perception. Although these early works may lack the rigor expected in contemporary scientific standards, they highlight foundational ideas: according to Carpenter in 1874, merely expecting a result would suffice to produce even involuntary movements. Similarly, according to Herbart in 1825, actions can be initiated by anticipation or the desire for sensory effects to be produced. Finally, James in 1890, argued that imagining a movement activates the corresponding motor response, underscoring the interplay between mental intention and physical action[173]. Extrapolating to the practice of osteopathy and the perception of the PRM, we might imagine that by looking for these micro-movements in the patient's tissues, the osteopath is provoking them, thereby misleading his own perception. However, if this is the case, it may not be a major problem, since tissues can only respond according to their own mechanical properties. If they are tension-free, their viscoelastic properties will enable them to absorb ideomotor movements, which the osteopath perceives as good PRM. If, on the other hand, the tissues are too tense, they will respond less readily to this stimulus, which may translate perceptually into a less optimal PRM. The test may still be relevant, as may be the assumed directions. Even when influenced by the ideomotor actions of the practitioner, the tissue can only respond based on its own resilience, and the amplitude will only be symmetrical if the structure is free from restrictions.

Sceptics who think that the PRM/CRI does not exist (but would only be a palpatory hallucination) are partly mistaken because there is indeed a rhythm detectable by both trained hands and machines. Osteopaths who are convinced that their sensations are entirely objective are also mistaken. This is because the ideomotor response could influence their perception, leading them to experience an inconsistent superposition of numerous rhythms at varying depths, likely interfered with by their own fluctuating rhythms. Osteopaths who think that synchronization should not be searched are mistaken

because when it is reached, it could be a sign that the tissues are sufficiently relaxed to respond to the ideomotor action (intention) of the practitioner. On the other hand, osteopaths who believe that synchronization is physiological are partially wrong as, if PRM is the summation of so many rhythms at different levels, it cannot be systematic.

IX.3. Cardiac coherence respiration and PRM

OMT is focused on enhancing the body's self-regulation putatively by stabilizing sympathetic and parasympathetic branches of the ANS, with PRM/CRI possibly serving as a witness to that effect. Pelz et al.[174] report rhythmic IM band responses to Cranial Vault Hold (CVH), a common technique in OCF, which was comparable with dynamics reported for IM activity in response to autogenic relaxation training.

An increase in amplitude of perceived oscillations is supposed to be linked to harmonization of heart-brain-circulatory rhythms and to allow a fluid circulation in the organism.

Physiological entrainment is recognized as a fundamental mechanism underlying the mind–body connection. Entrainment-based interventions may be used to promote well-being by improving cognitive, motor, and emotional functions, offering promising rehabilitative strategies for mental health enhancement[175].

Blacklaw-Jones presents a model that identifies piezoelectric activity as a potential mediator of responses to osteopathy in the cranial field (OCF) and explores the role of magnetic fields generated by the body's electrical activity. According to this theory, these magnetic fields oscillate in synchronization with electrical waves, resulting in coherent vibrations of the molecules and, consequently, of the body's tissues[176]. Blacklaw-Jones further suggests that OCF practitioners, when entering a meditative or contemplative state during treatment, can induce coherent vibrations within their own tissues. Through physical contact, the practitioner's harmonized electromagnetic field is believed to transfer energy to the patient's less coherent field, promoting therapeutic changes[176]. This increased coherence is then proposed to enhance cellular function, ultimately supporting self-regulation and homeostasis.

PRM amplitude is supposed to increase during mind-body approaches such as Yoga, mindfulness based therapy/meditation, breath therapy and others[177][36]. A common aspect of those practices is the occurrence of cardiac coherence breathing, leading to a state of wellbeing, plenitude and receptivity[178].

Cardiac coherence breathing consists inhaling for five seconds, then exhaling for the same amount of time (for a 10-second or 0.1 Hz respiratory cycle), thus it can be also achieved by simply controlling one's breathing[179]. Breathing influences the way the autonomic nervous system regulates heart rate: inhalation temporarily inhibits the influence of the parasympathetic system and increases heart rate, while exhalation stimulates the parasympathetic system and decreases heart rate. During cardiac coherence state, there is an ideal sympathetic/parasympathetic balance leading to a coherence between cardiac and breathing rhythms, with a maximum heart rate variability, which allows increasing the adaptability of the autonomic nervous system, providing subtle control over many organ function in the face of stimuli or stress[180][152].

Since the CRI/PRM is probably the result of summing up all the rhythms outlined above, we can hypothesize that the CRI and the PRM, underpinned by the ANS activity, are at their maximum intensity during cardiac coherence respiration, allowing the osteopaths to perceive them better and favoring an optimal circulation of all fluids (blood, lymph, CSF, air) in the body. This would lead to a state of wellbeing and equilibrium. We could hypothesize that synchronizing the patient's and practitioner's breathing with the perception of PRM during the session could have an additional benefit. Further research is needed to link PRM to other, more studied practices.

X. General conclusion

Our review questioned the physiological mechanisms underlying the PRM/CRI. We discussed the various hypothesis proposed to explain this rhythm. Since the Sutherland's theory, thanks to science progress, the knowledge about the physiological mechanisms underlying PRM has evolved[3]. Although Sutherland's hypothesis are still taught, the most plausible hypothesis explaining PRM/CRI is based on rhythmic changes in fluidity of the extracellular matrix induced by an intertwine of various physiological rhythms mostly driven by ANS activity, favoring metabolism and assuring nutrients and waste products an efficient transit through the extracellular space[181]. An increase in amplitude of these oscillations would lead to synchronization of heart-brain-circulatory rhythms, which would allow an optimal circulation of every fluid in the organism. Figure 2 illustrates an overview of the different mechanisms that could underlie the PRM/CRI rhythm, which we have presented in this review.

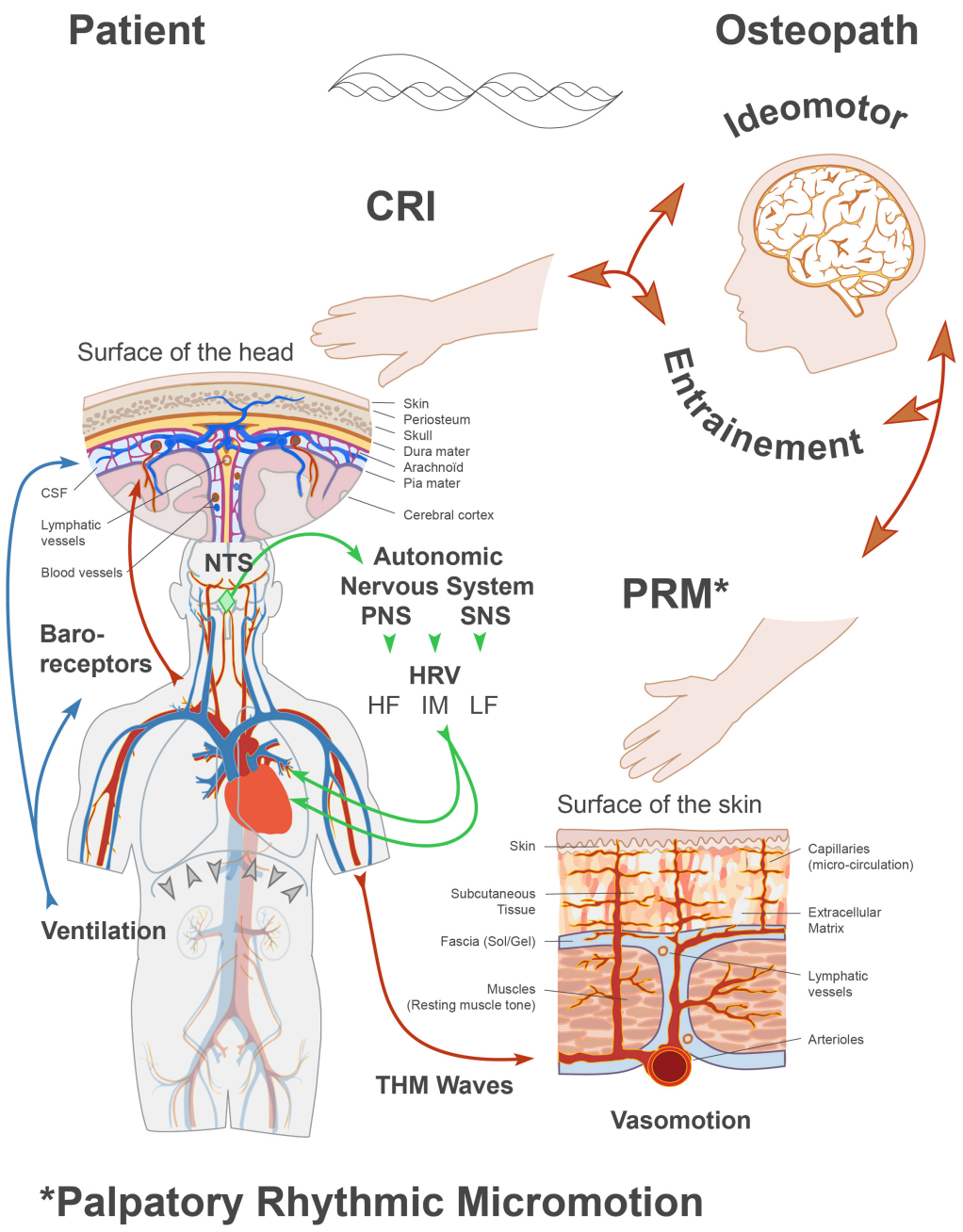
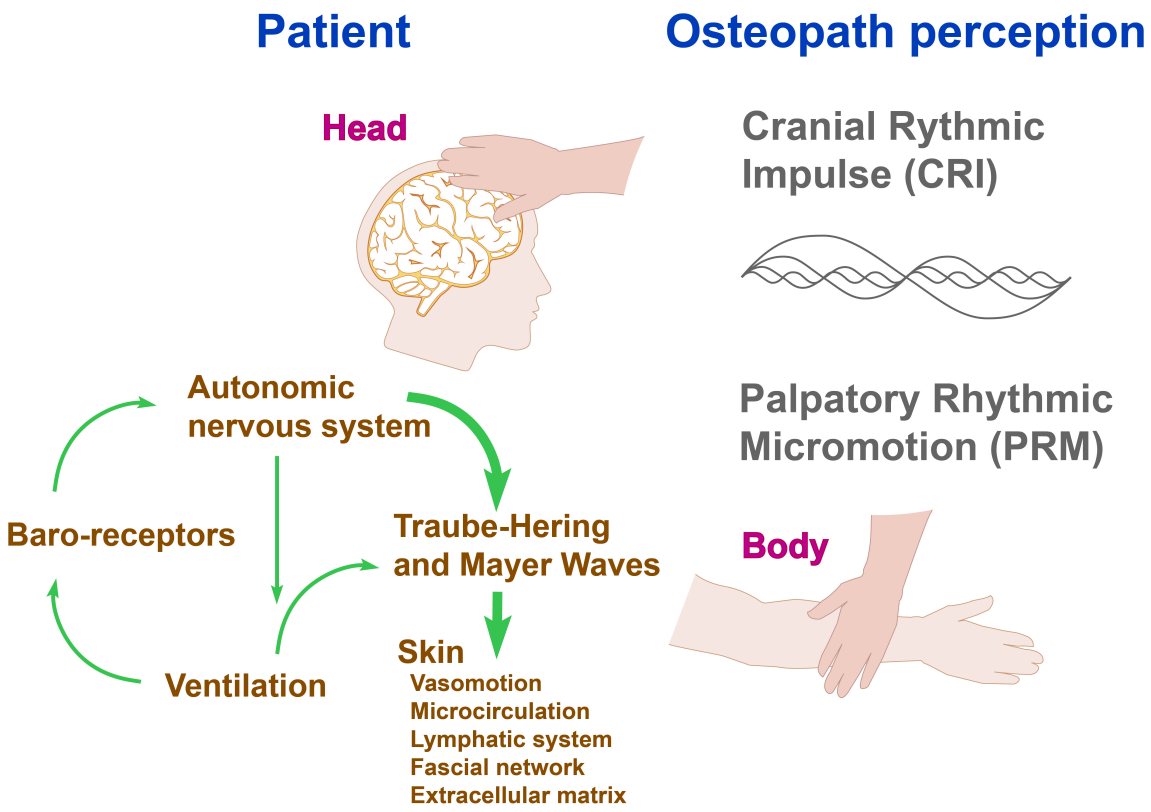


Figure 2. overview of the different mechanisms underlying the PRM/CRI.

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



Statements and Declarations

Conflict of interest

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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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