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COMMENTARY

A hypothesis to explain the palpatory experience and therapeutic claims in the practice of osteopathy in the cranial field

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Compression of the fourth ventricle (CV4)

Abstract A hypothesis is proposed which suggests biomechanical changes affect physiological mechanisms that may explain the therapeutic effects and tissue changes palpated by practitioners of osteopathy in the cranial field (OCF). It is suggested that the subtle application of manual compression between a practitioner's hands may cause a net negative charge in the collagen matrix resulting in a change of state from a gel to a sol. This is attributed to biochemical changes and the thixotropic properties of collagen.

It is also hypothesized that ionic movement results in an electrochemical gradient which causes changes in the cellular/plasma membrane permeability. Altered cation (hydrogen and calcium ions) distribution, present in the extracellular fluid, results in 1) an electrochemical gradient which causes changes in the cellular/plasma membrane permeability and 2) the stimulation of a local vasomotive response. It is postulated that the stimulation of a local vasomotive response within the extracellular matrix (ECM) is perceived by the practitioner of OCF as feeling a change in the quality of the "primary respiratory mechanism" (PRM)[a] and the "cranial rhythmic impulse" (CRI).[b]

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[a]

PRM: Primary respiratory mechanism. There are five proposed components to the PRM which involve the entire body as a unit of physiological function. The rhythmic motion is often referred to as primary respiratory motion or Involuntary Motion (IM) rather than just the CRI and involves the whole body.[88]

[b] CRI refers to "the motion present in the cranial sutures and a rhythmic impulse within the cranium, distinct from any previously known pulsation, as it relates to a basic physiological complex considered to be responsible for many of the essentials of homoeostasis.".[88]

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Restoring a normal electrochemical/ionic gradient across the cellular/plasma membrane, equilibrium is reached and it is hypothesized that this is the point of balanced fluid interchange (BFI), a point practitioners of OCF refer to as a "still point". Other physiological mechanisms and their implications are discussed to 1) explain other palpatory findings felt during the therapeutic response and 2) to explain the reported changes in the quality of the PRM/CRI, made by OCF practitioners, after a still point[1] has been reached. ª 2011 Elsevier Ltd. All rights reserved.

Introduction

Formanyyears,osteopathyinthecranialfield(OCF) has remained controversial. The science behind it was dubious and unproven[1,2] and questions were levelled at practitioners from all quarters and from within the osteopathic profession itself. McGrath[3] points to the lack of substantive evidence and the effectsaslittlemorethanplacebo.Therehavebeen and still remain, continued calls for it to stand up to the rigors of evidence-based medicine. McGrath questions the ethics of continuing such practice when "there is no scientific evidence that justifies the theoretical basis of OCF or refutes that its clinical practice is anything more than placebo." Maddick[4] argues lack of forthcoming scientific evidence should not, however, be a reason to preventitspractice,butequallyshouldnotallowthe practitioners of OCF to become complacent either. Itseemseminentlysensibletotryanddeterminethe scientific basis for OCF, not just to pacify the scientific, medical and osteopathic community, but to prevent OCF practitioners becoming marginalised.

William Garner Sutherland, the founder of the OCF approach, studied under Still's tutelage at the American School of Osteopathy in 1899. He conceived and developed the idea of the primary respiratory mechanism (PRM)[a] and started to work with his ideas for around forty years, before eventually publishing his initial work, The Cranial Bowl, in 1939.[5] There is some research[6] to support Sutherland's model of the PRM and support the claims made by OCF. These contributions to the science behind OCF have not just come from researchers within the osteopathic fraternity, but from other fields of scientific research, especially from discoveries in the field of biochemistry and further understanding of the physiology of the extracellular matrix (ECM). Understanding of the cranial rhythmic impulse (CRI),[b] continues to develop and scientific advances are helping to substantiate and explain osteopathic philosophy.[6] Physiological evidence is emerging that might be used to help explain the concepts of balanced fluid interchange (BFI) and cerebrospinal fluid (CSF) fluctuation. Not only is there less controversy surrounding these conceptual ideas, there is much less controversy surrounding other components of Sutherland's primary respiratory mechanism model; cranial bony motion and reciprocal tension of the dura mater.[6] The concepts of central nervous system motility, cerebrospinal fluid fluctuation and involuntary motion of the sacrum between the ilia, the other three components of the PRM, still remain more controversial.

There seems to be no forthcoming evidence, however, that has advanced our knowledge of how hands on intervention, prior to, during and after treatment, effects a therapeutic change in the physiology of the body or how this might constitute a change in the PRM/CRI. Lucas[7] suggests "with limited research resources we have in osteopathy we should be prioritising research that addresses cardinal questions about 'does this therapy work?' before pursuing questions about 'how does this therapy work?'".

Despite a need for effectiveness data, the problem of mechanism remains, with questions still standing over OCF concerning the "favoured mechanisms being biologically anomalous when compared to long and widely understood human biology",[2] published reports measuring intra/ inter-examiner reliabilities as "tiny and many are less than zero" and with individual practitioners unable to "palpate the same physiological

[1] Still point: The term refers to the brief cessation of the PRM/ CRI during the treatment procedure.[88]

phenomenon",[2] it does seem sensible to make further investigation and gain consensus into how OCF therapy works.

This paper attempts to draw together biochemical and physiological discoveries in an attempt to consider whether there is any credible physiological evidence that could explain what is happening when practitioners treat using OCF methods. Any explanation must address: 1) the clinically palpable phenomenon of the PRM/CRI; 2) How the application of a slight compressive force through an OCF practitioner's hands identify the areas of compression and strain; 3) How does a compressive contact influence the PRM/CRI to explain the phenomenon of a still point[c] and BFI? 4) What accounts for the change in the quality of the PRM/CRI after treatment; and 5) how does this effect a therapeutic change?

The paper starts by describing the OCF approach to treatment, properties of collagen and the properties of piezoelectricity and thixotropy. It is suggested these properties change under the influence of compression/deformation, create ionic movement resulting in an electrochemical gradient which causes changes in the cellular/plasma membrane permeability and local vasomotive changes. The mechanisms of vasomotion are discussed. It is argued that these local vasomotive responses manifest a wave, palpable by the practitioner as the PRM/CRI. Changes felt in the PRM/CRI prior to and during treatment are discussed. Differences are highlighted between TraubeHering-Mayer (THM) oscillations and the vasomotive phenomenon and it is argued that these differences may explain the lack of reliability during intra/inter-examiner reliability testing.

A further attempt is made to show how ionic movement, restoring concentrations across the cellular/plasma membrane, reach equilibrium, a point perhaps recognisable by OCF practitioners as a"stillpoint",apointthatconstitutesbalancedfluid interchange (BFI). The critical role CSF composition and physiology has in this mechanism is discussed. It is intended that these mechanisms show that the favouredmechanismsarenotbiologicallyanomalous to widely understood biology, but integral to it.

Finally, the treatment modalities of 'balanced membranous tension' (BMT), 'compression of the fourth ventricle' (CV4) and 'disengagement' are discussed relative to how they might achieve a still point, the significance of BFI, how this effects a therapeutic change and the changes felt in the PRM/CRI following treatment.

Description of OCF treatment approach

Before considering physiological reasons that might explain the changes felt by OCF procedures, it is important to understand and interpret the language and practices used. Most OCF practitioners start the treatment procedure by establishing a 'fulcrum point' with their forearms, or preferably their elbows, resting upon the treatment table. Their hands are introduced around the tissues under examination with a gentle, full and even contact pressure.[8] These tissues can be anywhere around the body and not specifically related to the cranium. A gentle compressive force is introduced by the hands through the fulcrum point, rather than straight through the hands themselves to engage the tissues.[9,10] The amount of compression varies, it is not a set amount and pressure is always changing degree and direction depending on palpatory findings to find the area of dysfunction.[9,11] Pressure is sensitive to palpatory findings; it is not applied blindly but is controlled, by the skilled OCF practitioner.

Authorities[8e11] then describe the activation of "intrinsic forces" and "inherent potencies" within the patient "going to work to manifest the degree, intensity and patterns of the tissue elements in health or disabled states." Although confusing to non-OCF practitioners, these phrases are 'hallmarks' to OCF teaching and practice. Sutherland[8] and Becker[10,11] coined many such phrases in their teaching to convey their palpatory experiences. Physiological evidence must also try and explain what these "intrinsic forces" and "inherent potencies" could be.

Properties of collagen

Collagen is the most abundant protein in the body, making up between 25%[12] and 30% (dry weight)[13] of all proteins. Derived from mesenchyme, it is ubiquitous and present in all the fascias of the body. Along with fluid, cellular and other fibrous components, the fascias and connective tissues continually adapt to the mechanical stresses and strains imposed upon them.[13] The fascias and connective tissues are diverse and dynamic, much has been written about their constituent parts[12e14] and the intricacy and diversity of cellular reactions that occur within them.[15]

The collagen molecule consists of three twisted helixeswhicharebondedtogetherbyhydrogenbonds to form a triple helix.[12,16] The molecules line up side by side and attach to one another, like overlapping bricks, via the same kind of hydrogen bonds.[17] This molecular arrangement of collagen allows a dipolar arrangement,

i.e. the ability of a collagen molecule toreverseitsorientationatasinglelocationalongthe fibril.[17] It is suggested that this property allows collagen to hold a charge and is responsible for its piezoelectric and semiconductor properties.[15]

Thixotropy

Thixotropy is a property of fluids to show a timedependent change in viscosity. It is defined as: "The property of certain gels or colloids to become less viscous, i.e. a change to a more fluid state when shaken or agitated but to revert to their original viscosity upon standing."[18] This is usually a function of time "the longer the fluid undergoes shear stress, the lower its viscosity."[19]

It is suggested that working with the tissues, taking them into a position of least resistance, facilitates this thixotropic effect in the collagen. As discussed later this allows other metabolic responses to occur that may start to explain occurrences during the hands on treatment of an OCF treatment procedure.

It has been known since 1958 that fascias exist in a gel/sol (gelatinous/fluid) equilibrium.[20] At that time it was felt these characteristics were more dependent on the types of fascia rather than it being a dynamic state, where they can change from one form to the other, for all of them. More recent research, outlined below, has explained the metabolic mechanisms behind the gel/sol states, which again is helping to explain and remove controversy around the fascial concept of OCF.

Piezoelectric properties of collagen

Piezoelectric energy is a form of electrical energy produced by certain solid materials when they are deformed (The word "piezo" has its roots in the Greek word "piezein" meaning "to press"). Pierre and Jacques Curie observed in 1880 that certain quartzcrystalsproducedelectricitywhenputunder pressure. At a very simplistic level piezoelectricity is a relationship between shape and charge. It is defined as 1) the generation of a voltage across a solid when a mechanical stress is applied. 2) The dimensional change resulting from the application of a voltage.[18] A biomechanical structure with a shape has a charge, if the shape is changed, the charge changes and if the charge changes the shape changes.[21]

Ferreira[22] studied the effects of piezoelectricity in elastically deformed collagen. Bone was decalcified and the prepared collagen was subjected to deformation. They found that when compression force was applied to the collagen so as to bend it, a net negative charge was induced on the compressed side and a net positive charge was induced on the tensioned or stretched side. This was accredited to the mechanical deformation creating "piezoelectric changes which induce a dipole orientation on the material, as an effect of the internal reorganizationofthecollagenfibers."Marinoetal.[23] looked at the piezoelectric effect in samples of human bone. They reported "at least part and possiblyallofthepiezoelectriceffectfoundinwhole bone arises from the organic component", "bone mineral per se makes no contribution to the piezoelectric effect in bone." Yasuda and Fukada[24] published a detailed study of bone's piezoelectric properties. They also discovered that dry collagen would develop an electric charge when it was stressed or bent and this lead them to propose that the source of piezoelectricity in bone was collagen. Bassett[25]acknowledgedthepiezoelectricproperties of collagen but proposed another way that may give rise to electric charge. The organic component contains not only collagen but also hyaluronic acid, a long-chain molecule from the mucopolysaccharide class. When deformed, a separation of the electric charges in the mucopolysaccharide chains occurs. Bassett proposed electricity could theoretically be generated by bending the mucopolysaccharide molecules. The piezoelectric effect caused by deformation produced an electric charge but this tended to "leak" in a closed short-circuited system, like the fascial system and the energy was quickly released. If the material is not short-circuited, the applied stress/strain induces a voltage across the material, such as in vitro experiments.[21] Basset did report uniformity in one of the pulses obtained after deformation and that was polarity. Whereas the Piezoelectric effect was fleeting, the polarity on the side under compression, the concave side, was negativelychargedandthepolarityonthesideunder tension,theconvexsidewaspositivelycharged.This explainedtheclinicalandexperimentalobservations that on a concave side, bone will be built up over time and on a convex side bone will be torn down over time. Ferreira et al.'s experiments, although in vitro, also found similar results. There was a build up of bone when compression was maintained for varying periods of 3½ and 4 weeks.

Possible effects of deformation

It has been suggested that the polarity changes result within collagen by affecting its dipolar orientation. "Shear stresses develop between adjacent strands of collagen and opposite charges build up on opposite sides of the unit".[25] This results in a net negative charge through the compressed collagen and a net positive

charge through stretched collagen. It is hypothesized that this dipolar change alters the distribution of cations in the extracellular fluids between the tissues. Available research suggests these are most likely to involve H^+, Na^+ and Ca^{2+} ions. There is less available information on the movement of Na^+ ions in the ECM, but more evidence with regards to the physiology of hydrogen and calcium ions to support altered distribution in the ECM. Quinn et al.[26] and Garcia et al.[27] showed "compression of cartilage resulted in the flow of "counterions" such as Na^+ and H^+ which reduced the pH." A Counterion[28] is an ion that accompanies another ion in order to maintain electric neutrality. "Dynamic compression induced pressure gradients, fluid flows and currents within tissues." The oscillations were reported around 0.01e1 Hz (0.6e60 cycles per minute (cpm)).[15]

Further evidence for the likelihood of the migration of H^+ ions is evidenced by the increased presence of H^+ ion concentration having been described around and being associated with chronically compressed tissues. The collagen fibres "thicken and rigidify"[17] with age and chronic stress. This may be due to the hydrogen bonds, between the collagen fibers, packing more and more tightly together and bonding more firmly together over the years.[17] Cathie[29] states "The contractive phase of fascial activity supersedes all other aspects of fascia"and"thisisprecededbyanincreaseinH$^+$ion concentration of articular and peri-articular structures." These may also be responsible for producing vasomotion. Vasomotion, see below, can be generated under conditions of metabolic acidosis, generatedbytheinfusionofdilutehydrochloricacid (HCl)solution.[30]Todate,H^+andCa^{2+}ionshaveboth been reported in vasomotive changes. Gutstein[31] is quoted by Chaitow[32] writing, "Tissue changes which occur in the onset of fibrositis are thought to be initiated by localized sympathetic predominance associated with changes in the hydrogen ion concentration and sodium and calcium balance in the tissues." It is unclear if this is cause or effect.

The cranial rhythmic impulse

The term 'cranial rhythmic impulse' was first coined by Woods[33] and is described as palpating the PRM on the head. The term was used by Nelson et al.[34] as a term of convenience. The correct term used should be the primary respiratory mechanism (PRM),[35] as the term CRI, although an aspect of cranial motion, is an oversimplification. It is now generally accepted by most observers and increasing research suggests that, practitioners of OCF, palpating the PRM/CRI,[36,37] are palpating Traube-Hering (TH) oscillations. The modern interpretation is that the PRM/CRI is synonymous with the TH oscillations and that this is the rhythm most consistently felt by OCF practitioners.

Traube-Hering-Mayer oscillations

THM oscillations have been described as: rhythmical variations in blood pressure, usually extending over several respiratory cycles, with a frequency varying from 6 to 10 cpm.[38e40] THM oscillations have also been associated with heart rate, cardiac contractility, pulmonary blood flow, cerebral blood flow and movement of cerebrospinal fluid and peripheral blood flow, including venous volume and body temperature regulation.[40] They are related to variations in vasomotor tone and arise from oscillations in nervous tissue, efferent vasoconstrictor, i.e. sympathetic nerve innervation of smooth muscle in the arterioles.[37] As peripheral arterioles vasodilate and vasoconstrict, there is a swelling and receding of the tissues. Due to the fact that these oscillations arise from variations in neural discharge, they would be synchronous in large parts of the body.[30]

The phenomenon was discovered by Ludwig Traube in 1865[38] and was attributed to intrathoracic pressure fluctuation of pulmonary respiration. Traube noted the oscillations continued after the cessation of respiratory motion. Ekbert Hering[39] confirmed Traube's discovery in 1869. In 1876 Siegmund Mayer[42] observed similar oscillations, but according to some writers, Mayer's oscillations are a separate entity, described as oscillations of low frequency, similar to Traube and Hering (TH) oscillations, but non-synchronous with respiratory cycles.[43]

Nelson et al.[40,44] investigated the TH oscillations and the CRI using Lazer-Doppler flowmetry (LDF). Light from a strong, non invasive, monochromatic light source undergoes a frequency shift (Doppler shift) when itisbackscattered from movingobjects; analysis of this shift in backscattered light from living tissue gives a relative measure of blood cell movement, thus average blood flow in a volume of tissue. The accuracy, sensitivity and specificity of the LDF device was validated against an ECG (Electrocardiography) device.[45] The LDF device measuredtheTHoscillations andheart rate.TheTH component represented baroreceptor activity and reflected change in autonomic balance. Data gathered from spectral analysis was analysed. Using a paired-samples t-test, HRV (Heart rate variability) calculated from ECG and cardiac components of LDF waves demonstrated a correlation of 0.97 (P

0.001);thisreflectedLDF's abilitytodetectRRI (RR interval) with accuracy. The TH component of the LDF waves (0.08e0.15 Hz) when compared with the low frequency components of ECG/HRV
(0.08e0.15 Hz) demonstrated a correlation of 0.712 (P 0.001); this reflected simultaneous changes between the TH component of the LDF wave and HRV. LDFwas95.45%sensitiveand90.91%specificin detecting changes in autonomic balance.

It was concluded LDF was sensitive and specific in detecting autonomic balance changes. Cardiac and autonomic (TH) components of LDF wave accurately assess and quantify changes in autonomic balance.

Nelson et al.[40] demonstrated a statistically significant correlation between the palpated CRI and the 0.10e0.15 Hz TH oscillation, (6e9 cpm). The measured frequency of the TH oscillations was 6.75 4.5 cpm and that the CRI was

4.5 2.08 cpm. They concluded that the primary respiratory motion and the TH 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 ascribe the phenomenon of a 'still point' as a brief cessation in the rhythm of the PRM in 79% of cases with diminished TH amplitude.

This conclusion was not a new theory. McGrath[3] reviewing Ferguson's paper[46] proposed a similar explanation. McGrath suggests there is evidence to suggest that the practitioner of OCF is palpating "a manifestation of an extracranial blood flow phenomenon" and suggests this is the CRI. Reference is made to "Absence of reliability and validity are consistently associated with attempts to detect the CRI, and this may be because the CRI, as an inherent biological entity, does not exists. The observed inconsistency of this finding may lie in the previous explanation that the CRI phenomenon is an artifactual extra-cranial phase interaction of TH oscillations between the examined and subject."

It would therefore seem logical to conclude TH oscillations might account for the conceptual idea of involuntary motion throughout the body, i.e. the PRM and not just the CRI. This, however, leads to another question: if the TH oscillations are consistent with the PRM/CRI throughout the body, why is the PRM/CRI not more easily palpated and reproducible through inter/intra-examiner reliability testing?[47e49]

One suggestion may be that the TH oscillations are constantly changing around an autonomic mean, rather than staying as a static wave, due to the smooth muscle sympathetic efferent innervations in the vascular wall, continuously changing to maintain homeostasis. This may also explain variations in a palpable wave around and in different parts of the body. Although most researchers[36,37,40,44,50] are now suggesting the most likely oscillations attributable to the PRM/ CRI are the neurologically generated TH oscillations rather than the vasomotion itself, a second suggestion may be explained by the phenomenon of vasomotion in its own right.

Ferguson[46] concluded, in his paper reviewing the possible physiological basis of cranial osteopathy, that "It is unlikely the CSF production and drainage accounts for CSF fluctuation" and that CSF is circulated by arterial pulsation, "rhythmic changes in arterial vasomotion, leading to blood pressure fluctuations within the cranium" and that this had a frequency similar to the frequency of the CRI, 0.1 Hz (6 cpm) in medium sized arterioles. Although he alludes to vasomotion as a possible mechanism for the CRI, he does not provide us with the evidence for it. With regard to the recent evidence, outlined above, it could be that arterial vasomotion is responsible for CSF fluctuation and has a similar mechanism to that which produces the TH oscillations, i.e. do the two processes have the same physiological basis?

Vasomotion

At first sight vasomotion has many of the characteristics attributed to the PRM/CRI.

Nilsson and Aalkjaer[30] reviewed the hypothesized mechanisms of vasomotion and discussed some of the physiological and pathophysiological roles discovered over the past 20 years.

Of note, they state, "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 of time and in vivo experiments fail because vasomotion cannot be replicated." They conclude "The presence as well as the intensity or quantity of vasomotion is highly variable."

These characteristics could explain many of the criticisms levied at OCF with regard to inter/intraexaminer reliability testing.

Mechanisms of vasomotion

Vasomotion is defined as "the rhythmic oscillations in vascular tone caused by local changes in smooth muscle constriction and dilation."[30] Tortora[12] states vasomotion "is an intermittent contraction and relaxation, which may occur 5e10 times per minute" as "blood flows intermittently through a capillary bed due to alternating contraction and relaxation of smooth muscle." It was first detailed 150 years ago in the bat wing. Semien et al.[51] found vasomotion contributed to the perfusion of oxygen, regulated capillary pressure and improved flow in capillaries. They found that by applying sodium nitroprusside, vasomotion stopped. Sodium nitroprusside is a potent vasodilator and relaxes smooth muscle. It works by potentiating calcium ion reuptake by inhibiting activity of the endomembrane $Ca^{2þ}$ ion pump. This suggests vasomotion is calcium driven. Other researchers have found similar results.[52] Zacharia et al.[53] also suggests vasomotion can be interrupted by interrupting $Ca^{2þ}$ ion flow. Fujii et al.[54] found vasomotion in the basilar artery isdependentonextracellular$Ca^{2þ}$ions. Kim etal.[55] showed how extremely low electric and magnetic fields may induce changes to $Ca^{2þ}$ ion transport through plasma membrane ion channels (adrenergic $Ca^{2þ}$) in sympathetic nerve fibres and results show low intensity electric fields can alter calcium distribution inside cells, most probably due to the effect on receptor-operated $Ca^{2þ}$and/or ion channels.

Whereas TH oscillations arise from variations in neural discharge and are synchronous in large parts of the body, vasomotion can also be a local phenomenon. Although sympathetic tone is a prerequisite for vasomotion, either by providing a base level of tone necessary for oscillation or by stimulating the oscillation directly.[56,57] It can still occur when sympathetic input to the smooth muscle is decreased or blocked.[58] Localized changes indicate that not all changes in tone are caused by nerves from external signals to the vessels but are generated locally.[59] Thus if only a local area is under study, it becomes difficult for the practitioner to distinguish a systemic vascular oscillation from a local oscillation.[60]

It may offer an explanation to the unexplained results Nelson et al. observed when conducting their experiments.[44] Although examiners palpating the CRI maintained precise register with blood flow oscillations measured instrumentally, some examiners palpated the CRI/TH rate at a 1:2 ratio. A small number of osteopathic examiners palpated the CRI/TH rate at a 1:1 ratio.

It may also start to explain why inter/intraexaminer reliability studies, with two practitioners palpating one model at two different bodily locations, show poor results. One, or both operators, may be palpating a locally produced vasomotive response rather than the true autonomic, baseline TH oscillations that are synchronous across the entire body. Each practitioner believing they are palpating the CRI.

This may be explained in two different ways. The first may be due to the amount of contact pressure the OCF practitioner is applying with his palpating hands or as Bollinger et al.[61,62] suggest, mechanical oscillations from the movement of the heart and respiratory movement may confound vasomotion measurements by the Lazer-Doppler technique. It was found that the low frequency components (1e10 cpm) are caused by local vasomotion. The prevalence of higher frequency flux waves (15e25 cpm) might be induced by respiration.

Nilsson and Aalkjaer[30] claim that vasomotion "remains incompletely understood" with regards to "the cellular mechanism responsible" and "the physiological consequence of vasomotion."

Its "generation seems dependent on synchronization of a multitude of individual cellular oscillations. The concept of calcium release and uptake by the smooth muscle calcium stores, synchronised by sarcolemmal ion channels, and sometimes influenced by endothelial factors, may provide further understanding of this little-understood phenomenon."[63e65]

Nilsson and Aalkjaer[30] claim, "It is generated from within the vascular wall and is not a consequence of heartbeat, respiration or neural input." They then suggest a model.

Their conceptual model has "the basic oscillator as the sarcoplasmic reticulum, intermittently releasing calcium traversing the cell. In the presence of cyclic GMP (guanosine monophosphate) this calcium is able to activate a chlorine channel in the plasma membrane. If this current is activated in a sufficient number of cells at the same time, cells will depolarize. Cells that are not contracting at that moment will also depolarize because they are electrically coupled." (Coupling is present between the vascular smooth muscle cells, the endothelial cells and the endothelial to vascular smooth muscle cells.) "Depolarization will cause calcium influx through L-type calcium channels, which not only triggers contraction but also calcium release in quiescent cells, thus resetting their intracellular oscillators. In this way, the likelihood for a sufficient number of cells becoming active together in the next cycle is very high and the cells in effect become phase locked. cAMP (adenosine monophosphate) promotes vasomotion by its permissive action on the ion channel, most likely via protein kinase G-mediated phosphorylation of the channel, but inhibits vasomotion by action on the

sarcoplasmic reticulum by influencing intracellular calcium availability, uptake and/or release." Nilsson and Aalkjaer[30] vasomotion requires coordination of the activity of the vascular smooth muscle cells in the vessel wall and this is electrically linked.

This phenomenon may also start to explain the theory of entrainment.[66] Coordination between electrically linked cells oscillating asynchronously may be able to oscillate and through the interconnections, eventually entrain. Chaytor et al.[67] found this interconnectedness could be blocked by introducing gap-junction blockers.

Nilsson and Aalkjaer conclude, "The physiological significance of vasomotion remains elusive".

It would be easy to construe that the mechanisms of vasomotion are present under normal physiological conditions and that they may explain the PRM/CRI. One overriding problem still existsd that vasomotion is not consistently present under 'normal' physiological conditions.[30] "It is generally most prevalent under conditions of reduced perfusion", "is absent under resting conditions" and is "initiated by metabolic stress."[58,68,69]

Its prevalence, when critically reviewed,[30] suggested "that vasomotion might have been present only for brief periods." There is also some debate as to whether the oscillations seen in the diameter of large arteries should be denoted as
vasomotion.[70]

This means, despite the behavioural similarities in its characteristics to the PRM/CRI and the fact it is an intermittent, local rather than global, total body phenomenon, it is less likely that vasomotion, in its own right, is the mechanism that explains the PRM/CRI.

An alternative hypothesis to Nilsson and Aalkjaer's conceptual hypothesis[30] has also been suggested by Lee,[15] who states "It is my belief that what we feel in the tissues as the tide is related to the waves of calcium ions and the accompanying water, which is associated with alterations in the viscosity and charge of the matrix."

Lee[15] describes how the microstructure of the fascia is made up of a network of proteoglycans (PGs), glycoproteins (GPs) and glycosaminoglycans (GAGs). They form 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 extracelullar matrix to act as an essentially unstable semiconductor, a liquid crystal. Electrons freely move through the collagen and waves of ions can freely move through the matrix. This can cause depolarization of cell membranes, or the activation of secondary messengers like cAMP, inositol triphosphate (ITP) or calcium ions. Adjacent cells are in turn stimulated via the integrins, (receptors that mediate attachment between a cell and the tissues surrounding it).

It is known that ITP causes release of $Ca^{2þ}$ ions from intercellular stores. This intracellular $Ca^{2þ}$ ion concentration propagates the length of the cell producing "periodic oscillations in vascular smooth muscle cells."[71] Vasomotion is synchronised by the depolarization of adjacent cells and the voltage gated model suggested by Nilsson and Aalkjaer.[30]

Cerebrospinal fluid composition and physiology

The total volume of CSF in and around the brain is around 150 ml, of which 20 ml (15%) is in the ventricles. The total volume is turned over five to six times a day meaning there is approximately 500e600 ml of CSF produced daily. Of the 150 ml in and around the brain, some of this is draining into the blood sinuses via the pacchionian granulations and some CSF drains from the spinal and cranial sub-arachnoid space down through the collagen tubules of the fascias.[72,73] The total volume of CSF within the collagen is unknown. Supposition suggests if 500e600 ml is secreted within ventricles, some drains into the blood sinuses and there will be an onward movement of the remaining volume through the body to the extracellular fluid (ECF) and lymphatic system. CSF will be continuously replenished at the same rate of loss through the day. It is unknown if and to what extent it changes composition.

CSF basically consists of ions and water.[12] It is made up of 98.5% water. The other 1.5% is made up of glucose, oxygen, lactic acid, uric acid, urea, and waste products in a dissolved form and white blood cells and proteins, in low concentrations. There is a high concentration of hydrogen ions due to the water content, and a high concentration of sodium and chlorine ions. CSF is also high in calcium and magnesium but it has low concentrations of potassium, all dissolved or in ionised forms.

Erlingheuser[72] discovered that CSF was present in the microtubules that make up the structure of the collagen. The collagen is surrounded by ECM which provides a fluid medium through which substances are exchanged between the blood and cells. Lee postulates that the ionic composition of the CSF, within the collagen microtubule, allows an action potential of around 90 mV, to exist across the collagen microtubule between the CSF and the surrounding ECF (Ionic concentrations and concentration gradients between the CSF and ECM are maintained through $Na^{þ}/K^{þ}$ ion pumps and facilitated

$Na^{þ}/Ca^{2þ}$ ion pumps. The inside is negative relative to the outside). Lee[15] suggests this electrochemical potential drives a calcium wave/flux, "fluctuating redox potentials", towards the electronegative cell membrane, taking with it a flux of water, i.e. calcium moves from areas that are less negative, or more positive, to areas that are more negative or less positive. $H^{þ}$ ions (counterions) move in a counter direction as the $Ca^{2þ}$ ions depolymerise the bound water on the PGs. As the unbound water effectively creates a lowering in the $Ca^{2þ}$ ion concentration and a more positive charge, the calcium will move to an area that is more negative, the water behind will then repolymerise or rebind to the PGs and the process can continue. Lee reports that Nordenstrom found these waves cycle at a rate of seven times a minute, a similar rate to the TH oscillations and a similar rate reported of the CRI.

It seems that the depolymerisation/repolymerization oscillations within the mucopolysaccharide chains and the associated shape changes within the molecules could be responsible for the piezoelectric effects outlined by Bassett.[25]

Although Lee's hypothesis reinterprets Sutherland's clinical observations, he offers no explanation as to how treatment effects cause a change in this mechanism. Although he does discuss changes associated with pathological change, it is left unclear as to what exactly the OCF practitioner is doing to this process that justifies the therapeutic claims.

It is unclear if this is the same process by which TH oscillations are generated but supposition suggests that Nilsson and Aalkjaer and Lees' two proposed mechanisms are similar. Lee indicates, quoting Korr, that it is the autonomic nervous system that mediates these mechanisms in the ECM. Norepinephrine, from the sympathetic nervous system, can directly affect calcium oscillations via the sarcoplasmic reticulum, as Aalkjaer and Nilsson hypothesized. Lee suggests norepinephrine directly changes the viscosity in the GAGs making them more negative.

Charman,[74] citing other authors, referring to the benefits of soft tissue manipulation, acknowledges the charge in collagen and proteoglycans in the ECM are changed by mechanical pressure which affects the state of the ground substance. Charman cites Schmitt et al, 1955, Athenstaedt, 1974 and Linsenmeyer, 1983. "This provides the opportunity for electrical signal processing locally and globally throughout the somatic system. Due to the web-like structural matrix of the soma, piezoelectric phenomena can be local or spread to distal regions along the uninterrupted connective tissue mechanical and bioelectrical network throughout the body." Charman continues, "Indicative of these phenomena is the appearance of vasomotor responses, fasciculation of the neuromuscular elements and heat. These responses can be seen in the region of the body being treated or in other regions of the body." He quotes Athenstaedt, "The entire organism is interwoven with chains of piezoelectric dipolar molecules which are capable of oscillation due to their spiral nature. Thixotropy is the change of phase state of the ground substance to become more fluid when stirred up and more solid when left sedentary. Mechanical energy and subsequent friction from myofascial release or exercise has the effect of changing the gel portion of connective tissue to fluid from a dehydrated crystalline state." Athenstaedt suggests treatments can improve polarity potential in the tissues but there is no explanation as to how.

Charman[74] suggests "the molecular form of proteoglycans is particularly suitable for binding water, creating the viscoelastic, shock-absorbing and energy-absorbing behaviour of the extracellular matrix."

He acknowledges that energy flow is most apparent at this level and "Functional morphology is based in the interaction between water and the ECM. More specifically, the liquid-crystalline water and the sugar molecules (supposition suggests he is referring to the present names for the PGs, GPs and GAGs) determine the degree of organization and structural proportions in the ECM. All vital functions are mediated by the ECM."

Kleeman et al.[75] state that "calcium ions in the intracellular and extracellular fluids and in cellular and sub-cellular membranes are maintained with remarkable consistency." Calcium ions play a "critical role in many fundamental biologic processes". "The integrity, electrical properties and permeability of these cellular and sub-cellular structures are critically dependent on the calcium ion." As well "as being an essential coupling factor or 'biologic transducer' in the depolarization of cell membranes and conversion of electrical activity into contraction of skeletal, cardiac and smooth muscle, membranes depleted of calcium ions become increasingly porous and lose their selective permeability characteristics."

Calcium physiology is intricate, complex and multi-faceted with interconnectedness with many other ions and hormonal interactions. Of the 60% (5%) of the calcium in a diffusible, ultrafilterable state, 94% (5%) is in the form of ions[75] $Ca^{2þ}$ ion concentration is controlled by the dynamic equilibrium between the metabolically active component of the skeleton, i.e. osteoclasts and osteoblasts and the extracellular fluid bathing the components. Calcium in the ECF and the bony skeleton is

in dynamic equilibrium, 1% of skeletal calcium is freely exchangeable with the ECF, and the purpose is to maintain homeostasis of plasma calcium levels. Calcium, however, has a natural tendency to flow up its concentration gradient towards bony matrix and all other cells and therefore there would be a tendency for the calcium ions toleavetheECMandbecomedepositedinthebone.
To counteract this effect there are active transport systems to pump large amounts of calcium from bone back to the ECM. This is hormonally controlled as well by parathyroid hormone. This was discovered by Newman and Newman[76] over 40 years ago. Calcium in the mineral phase of bone comes into equilibrium with the calcium in the ECF at about one-third of the concentration level of that in the ECF and indicates the ECF, compared to the bone, is supersaturated. Due to the calcium's natural tendency to move up its concentration gradient, where it would all end up in the bone, active transport of these ions work against their natural concentration gradient to maintain the relatively higher concentrations in the ECF. Newman and Newman also report that these processes are
"apparently continuous."

Discussion

A hypothesis is proposed to try and explain how these physiological mechanisms produce the therapeutic effects described by practitioners of OCF. It is suggested that the subtle application of manual compression between the practitioner's hands to specific tissues and through specific techniques causes an ionic movement through the fluids and fibers of the ECM. A net negative charge in the collagen matrix may result in a change of state from a gel to a sol. This would be attributed to the thixotropic properties of collagen and to the polarity changes resulting from dipolar rearrangement in the collagen molecules generated in the manually compressed collagen fibers.

The "structure governs function" premise is core to osteopathic principle.[8] When tissues undergo direct or indirect trauma, physiology, healthy mechanical function and normal tissue arrangement is altered.[77] If Ferreira[22] and Bassett[25] are correct and an external compression stimuli induce polarity changes in the collagen fibers, it would seem a possibility that a change in the polarity of the collagen could be generated from hands on contact from the OCF practitioner alone. This is highly conjectural and unsubstantiated. There is no data available as to the magnitude of compression used by OCF practitioners and it is certainly an area where future research needs to be conducted.

The contact pressure of craniosacral practitioner's hands has been quoted as being around 5 g,[78] the "weight of a nickel." A criticism often levied against craniosacral practitioners, however, is that this contact pressure is too small to have any effect.[78] Certainly the contact pressures used by OCF practitioners can be significantly greater. Reference is only made to the use of compression rather than to the amount of compression.[10,11] The contact pressure is dependent upon the depth and degree of dysfunction and the type of tissue and is constantly changing. To evoke a substantial change in the PRM/CRI slightly more compression is often required and this has been described as "pounds to many pounds"[9] or up to or beyond 1 kg for very
compressed tissues.[79]

There is no mention of the amount of compression force needed to bend collagen referenced by Ferreira[22] in her study. Ferreira does report the force used in their experiments was "more than can be possible in the normal body conditions."[80] Bassett[25] plotted electrical output against the increase in deformation and found a linear, or one to one relation, but no mention is made of the compression loads used to achieve it. The evidence available does seem to suggest that the direct force required to evoke a polarity change directly in collagen would be either too great for the body to endure and may be too great for the practitioner to apply. Other physiological mechanisms may still offer some explanation as to how gentler compression may evoke a response within the ECM and affect the quality of the PRM/CRI. Literature review suggests most research has been conducted on collagen extracted from cartilage and bone but is still of the type 1 collagen common in the fascias of the body. Palpatory contact on the skin may still cause a change in cation distribution via mechanochemical transduction processes which may lead to changes in ionic distribution, changes in plasma membrane permeability and a vasomotive response. No evidence seems available on the effects of heat from the practitioner's hand.

Vos et al.[81] proposed living human skin generates an increase in the skin potential when compressed. The potential increased with pressure until it reached a maximum (It is suggested that this may start to offer an explanation as to why it is unnecessary to match the compression within a tissue strain to the compression applied by the OCF practitioner). Human cadaver skin did not show these potential increments. Neither did pads of collagen, paper tissue soaked in a KCl solution, nor layers

of cultured keratinocytes. Three theories were described to explain the origin of the measured skin potentials. The first is based on the piezoelectric characteristics of proteins in the skin.

The second theory assumes that the skin is a charged membrane which generates a streaming potential when deformed. This theory is disputed by McDonald[82] who reports these are of less consequence in in vivo situations. A third theory is proposed in which deformation of absorbed charged protein layers on structures in the skin changes the alignment of Donnan potentials in the surroundingtissue.TheDonnaneffect[83]isthename given for the behaviour of charged particles near a semi-permeable membrane, often referred to as a platen, to sometimes fail to distribute evenly across the two sides of the membrane. The usual cause is the presence of a different charged substance that is unable to pass through the membrane and thus creates an uneven electrical charge. This is because small cations are attracted, but are not bound to the proteins and small anions will cross capillary walls more readily than small cations. The solutions may be gels or colloids, like the ECM, as well as solutions of electrolytes, like the CSF and act as the phase boundary between gels, or a gel and a liquid, like a selective barrier. It may be possible that this uneven electrical charge may be produced by much lighter contact pressure, than that seemingly required to compress collagen and the resultant ionic movement may produce changes in the cellular/plasma membrane as well as produce a vasomotive response. It could also be possible the vasomotive response maythen possibly entrain.

Pienkowski and Pollack,[84] investigating strain generated potentials (SGP) in fluid saturated bone, agreed that whilst piezoelectric properties were retained, the properties of the fluid in bone have a greater influence on the magnitude and time dependence of the SGP. Especially notable was the observation that solutions of high NaCl concentration, (similar to CSF) consistently reversed the polarity of the SGP. No note is made of rate minute and supposition suggests a similar effect between the fluid and cellular content of the other fascial tissues may be worthy of investigation. Pienkowski et al. concluded these results were consistent with streaming potential theory.

Silver et al.[85] suggested internal tension in the dermis also gives rise to active cell-extracellular matrix and cellecell mechanical interactions that may be an important part of the homeostatic processes. Silver et al. reviewed how internal and external mechanical loads are applied at the macromolecularandcellularlevelsintheepidermis and dermis. A review of the literature suggested that internal and external forces applied to dermal cells appear to be involved in mechanochemical transduction processes involving both cellecell and cell-ECM interactions. Internal forces present in dermis are the result of passive tension that is incorporated into the collagen fiber network during development. Active tension generated by fibroblasts involves specific interactions between cell membrane integrins and macromolecules found in the ECM, especially collagen fibrils. Forces appear to be transduced at the cell-ECM interface via rearrangement of cytoskeletal elements, activation of stretch-induced changes in ion channels, cell contraction at adherens junctions and activation of cell membrane associated secondary messenger pathways. Silver et al. concluded that internal and external mechanical loading appears to affect skin biology through mechanochemical transduction processes.

Chiquet et al.[86] acknowledge mechanical forces are important regulators of connective tissue homeostasis. Their experiments in vivo indicated that externally applied mechanical load can lead to the rapid and sequential induction of distinct extracellular matrix (ECM) components, especially the fibroblasts. Thus, ECM composition seems to be adapted specifically to changes in load regulating the production of ECM proteins indirectly, by stimulating the release of a paracrine growth factor, or directly, by triggering an intracellular signalling pathway that activates the gene. Chiquet et al. found evidence that tenascin-C, an ECM component was directly regulated by mechanical stress: induction of its mRNA in stretched fibroblasts is rapid both in vivo and in vitro, does not depend on prior protein synthesis, and is not mediated by factors released into the medium. Fibroblasts sense force-induced deformations (strains) in their ECM. Integrins within cell-matrix adhesions can by acting as ‘strain gauges’, trigger MAPK and NF-kappaB pathways in response to changes in mechanical stress. Their results indicated that cytoskeletal ‘pre-stress’ is important for mechanotransduction to work: relaxation of the cytoskeleton (e.g. by inhibiting Rho-dependent kinase) suppresses induction of the tenascin-C gene by cyclic stretch, and hence desensitizes the fibroblasts to mechanical signals.

It is suggested, by engaging the tissues and introducing a contact pressure to the skin, the OCF practitioner is introducing a pre-stress.

Yao and Gu[87] investigating mechanoelectrochemical mixture theory analyzed the mechanical, chemical and electrical signals within cartilage underdynamicunconfinedcompression (5% dynamic

strain).Theeffectsofthepermeableloadingplaten, loading frequency, and fixed charged density on the physical signals and the transport of fluid, ions, and uncharged solutes were investigated. Numerical analyses showed that a permeable platen will increase the rate of dynamic fluxes of fluid, ion, and uncharged solute in the region near the permeable platen, but not the fluid pressure and electrical potential in the central region of the tissue at 0.1 Hz (6 cpm). The increase in fixed charge density (FCD) will decrease the dynamic fluxes of fluid, ion, and uncharged solute, but increase the fluid pressure and electrical potential within the tissue. Again conjecturally, this may start to explain how a lighter contact pressure, may evoke a feeling of expansion from the increased fluid pressure at 6 cpm. A light but definite contact pressure is indeed sufficient for palpation of the PRM/CRI or TH oscillations provided proper fulcrum pressure is maintained around the practitioner's elbows or forearms and may start to evoke a change in the PRM/CRI.

With a light contact pressure, it will be difficult for the OCF practitioner to know if they are feeling TH oscillations or whether the dynamic fluxes of fluid and ions are evoking a vasomotive response. The OCF practitioner may just be feeling the increase in fluid pressure. The OCF practitioner will feel they are tuning into the PRM/CRI. Ionic movement, altering the distribution of cations in the surrounding extracellular fluids between the fibers and cells of the tissues, may increase electrical potential and change the alignment of Donnan potentials in the surrounding tissue. This may affect the polarity of the plasma/cellular membrane.

It has been suggested these are most likely to be $Na^{þ}$, $H^{þ}$ and $Ca^{2þ}$ ions. It is unknown, unlikely and can only be speculated if the compression creates polarity changes in the collagen or whether the net negative charge in the collagen attracts these cations present in the extracellular fluids resulting in the stimulation of a local vasomotive response.

It is postulated that the stimulation of local vasomotive responses and dynamic fluid fluxes, produces a feeling of expansion from the increased fluid pressure within the ECM, from the increased FCD and is perceived by the practitioner as "tuning into the CRI." So although we have a baseline oscillation via the TH oscillations, which maybe palpable by some practitioners as flexion, external rotation and extension, internal rotation movements almost as soon as they put their hands on, compression through the tissues also induces a vasomotive response. Could this be what practitioners of OCF construe as "feeling innate forces"?

It is unclear if the ionic movement either directly induces the movement of $Ca^{2þ}$ ions towards the negative charge in one direction and the $H^{þ}$ ions, counterions, in the opposite direction as Lee proposes, or if the polarity changes directly affect $Ca^{2þ}$ ion distribution in the ECM, as Nilsson and Aalkjaer's model suggests, through changes in the cellular/plasma membrane, leading to an ongoing oscillation through the adjoining cells, effectively alteringpH(higher$H^{þ}$ionconcentration),volumeor temperature through linear thermodynamic effects, but a vasomotive response is initiated. It is hypothesized that this is felt as a change in the quality of the PRM/CRI, possibly as the "tuning in phase" of the OCF treatment where flexion and extension is more readily palpable. Practitioners would now be feeling a wave which is associated with the flexion/extension components of the PRM. With the Donnan effect, an ionic concentration gradient would result with the net effect of ions moving back down their concentration gradients, and as equilibrium is approached there would be a gradual lessening in the amplitude of the flexion/ extension tissue motion until equilibrium is reached.[12] It is hypothesized that this might equate to a "still point".

It is further hypothesized that, following equilibrium at the still point, this freely allows the movement of all ions across membranes against their usual gradients, a point known by OCF practitioners as BFI.

Nelson et al. identified the still point with LDF and Lee suggests these fluctuations are palpable. When a still point is reached, the redox potentials are reset everywhere simultaneously, electrical potentials are reset, free electrons recharge the fluids, raising the pH which recollects minerals. Hypothetically, even though the compression between the practitioner's hands is still present, these "calcium ion pumps" may actively restore the calcium to the optimal supersaturated levels. With the active free ionic movement possible through all the plasma cell membranes at the point of BFI all other ionic levels might return to optimal levels. The baseline TH oscillations return to normal for that patient and the OCF practitioner registers a change in fluid dynamics.

With ionic concentrations returning to their optimal levels within the intracellular, extracellular and lymphatic fluids, ionic concentrations within the CSF in the microtubules[72] would also be optimised, as would the action potentials, outlined by Lee, across the collagen microtubules. Optimal ionic compositions would be produced in the ECM. Optimal tissue perfusion would most likely mean that vasomotion would cease. As previously stated, vasomotion is not common in normal physiological conditions and a baseline, optimised, TH

oscillation would result. This, it is hypothesized, would be palpated by the OCF practitioner as an improved quality in the PRM/CRI wave and would be felt, most likely as a slower wave.

This hypothesis is highly conjectural, scientifically unproven and unsubstantiated. Research needs to be conducted to test these hypotheses.

OCF practitioners need to gain consensus concerning the magnitude of compression used and whether different oscillations are produced with differing magnitudes of compression. Investigation needs to be made into what OCF practitioners are feeling and into whether OCF practitioners are feeling the same thing. Investigation is required into whether in vivo compression applied to fascial tissues by the OCF practitioner can evoke dipolar changes in collagen or whether ionic movement is created via mechanochemical transduction and whether, as suggested, these cause a vasomotive response and/or changes in cellular/plasma membrane permeability. More investigation needs to be conducted into the events occurring at a still point and ionic changes associated with BFI.

Treatment

Conditions claimed to be amenable to OCF treatment are well documented in OCF texts.[8,10,11,88] Can the physiological mechanisms outlined above explain the changes in relation to applied OCF techniques? Charman suggests on the back of the work of Trincher (1981) that "False information stored within the liquid crystals could be cancelled with temperature increase, piezoelectric events, and transferred back to a homogenous fluid." "The process should ideally depolarize the interstitial tissue and reset the ground regulation system to be more efficient in information transmission and eliminate any false signals produced by the crystalline dehydrated matrix."

It is arguable that OCF may be in a unique position to achieve these therapeutic changes.

Lesions may affect bony, fascial or fluid structures[9] directly or indirectly and alter articular mobility, vascular circulation, arterial supply, venous and lymphatic drainage, affect nerve trophism within the motor and sensory nerves of the central nervous system (CNS) and autonomic nervous system (ANS).[77] CNS imbalance will lead to musculoskeletal problems and ANS imbalance may result in a degree of excessive tone in the sympathetic nervous system (SNS).[89,90] This will disrupt normal vasomotive tone in the vascular structures and ECM, which in turn will compromise immunology and disrupt cellular physiology across the cellular/plasma membranes. The aim of osteopathic treatment is to correct the structure to improve function and improve function to improve structure. Can the physiology of the therapeutic response explain how these lesions are amenable to OCF treatment through bony, fascial and fluid techniques?

Balanced membranous tension (BMT) and balanced ligamentous tension (BLT)

Oncecompressionhasengagedtherequiredtissuein the body, in the case of BMTand BLT it is at a fascial level, piezoelectric change within the collagen matrix discharge. It is unknown if compression creates a direct polarity change in the collagen and a net negative charge through the compressed tissues or if, as is more likely, compression to the fasciascreatemechanochemicaleffectsthroughthe ECM. Altered cation distribution alters cellular/ plasma membrane permeability and a vasomotive response is produced through the adjoined cells. Do practitioners detect the calcium oscillation, or vasomotion, within the tissues?

The OCF practitioner may further engage this vasomotive response by utilizing the thixotropic propertiesofcollagen.Subtlecontrolledmovement of the practitioner's hand contact is initiated through further compression, torsion, side bending andshearingmovementstogentlytakethecollagen towards its "maximal shear potential" or in OCF terms towards a point of "greatest ease". This is known by OCF practitioners as a point of "balanced membranous tension". At the point of balanced membranous tension the amplitude of the oscillation reduces until flexion/extension ceases. A "still point" is possibly reached as ions move back along their concentration gradients to a point of equilibrium. Thermodynamic changes are occurring within the ECM, through pH, volume, heat and ionic change acrossthe cellular/plasmamembranes.The fluid viscosity of the collagen matrix is optimised due to its maximal shear potential at its point of balance. The fluid viscosity can take between a few seconds to a few minutes to occur. It is hypothesized that this is the feel of 'release' within the tissues. As the "still point" is reached, balanced fluidinterchangeoccurs;thisisassociatedwithfree ionic and solute movement across the plasma membranes. As already stated, Lee suggests "When astillpointisreachedtheredoxpotentialsarereset everywhere simultaneously, electrical potentials are reset, free electrons recharge the fluids raising the pH." The calcium pumps are reversing the movement of calcium ions to return the supersaturated concentrations

in the ECF. Vasomotion stops as normal tissue perfusion has resulted and normal calcium oscillations are felt as the TH oscillations return. Ionic balance is optimal across the cellular/ plasma membranes, through $Na^{þ}/K^{þ}$ion pumps and facilitated $Na^{þ}/Ca^{2þ}$ ion pumps and within intracellular and extracellular spaces. Ionic concentrations are optimal within the CSF and action potentials have been optimised across the collagen microtubules, in turn optimising the electromotive motion and calcium/water fluxes in the ECM. The piezoelectric effects, the temperature changes associated with the thermodynamic events and the return of the ECM back to a more homogenous fluid, suggested by Charman, cancels the false information stored in the liquid crystal matrix. OCF practitioners often refer to this "false information" as a strain pattern. The process of balanced fluid interchange has "depolarized the interstitial tissue and reset the ground regulation system to be more efficient in information transmission, eliminating any false signals produced by the crystalline matrix" as suggested by Trincher.

Compression of the fourth ventricle (CV4)[88] and fluid treatment

As compression is applied to the squama of the occiput, the collagen in the bony matrix is engaged. Similar thixotropic and piezoelectric effects are possibly induced as for BMT. As the vasomotive response is initiated, further compression is introduced in an attempt to resist rather than push against the vasomotive force. This introduces further compression into the collagen matrix. Rather than utilising the thixotropic properties of the collagen to maximise fluid viscosity into a direction of ease, it is hypothesized that fluid viscosity is reached by slowly compressing the collagen by the slow application of controlled pressure and waiting for the fluid viscosity to respond. If too much pressure is applied too quickly the body responds protectively and a resistant holding patternmayresult.Ifnotenough pressureis applied the thixotropic effects are not engaged and only the elastic tissues are moved. It has been suggested, but seems unlikely, the piezoelectric effect "must be held for a minimum of 90e120 s, perhaps even as much as 3e5 min before the piezoelectric effect is elicited and real change begins."[91] It is proposed a similar physiological mechanism as outlined for BMT then takes place. A "still point" in the fluid is secured, BFI occurs and the therapeutic response takes place within the tissues. The fluid viscosity within the collagen matrix is similarly optimised. The piezoelectric influences affect the total network of fibers throughout the body simultaneouslyandarethereasontheydischargequickly. This may also explain why other 'hand holds' can still be used to induce a "still point" within the fluid other than at the head.

It is not currently known if a CV4 technique has any further benefit when performed on the fourth ventricle as opposed to a "still point" secured anywhere else on the body. Some OCF practitioners consider that performing a CV4 affects the autonomic centres in the floor of the fourth ventricle. This is anecdotal and as yet remains unproven within the parameters measured.[92] There are further theories that the TH oscillations may originate from autonomic centres in the medulla but again no known research as yet seems to support this idea and no references seem available. Physiologically there is also much debate as to the exact location of the origins of the autonomic centres.

In the caseof BMTand BLT, the viscosity becomes more fluid and flows three dimensionally until an older underlying restriction arises. Through subtle application of pressures and movements a similar process is repeated through the treatment process.

Disengagement

Another physiological phenomenon may take place duringtheconsideredreleaseofcompressionduring the CV4 hold and during the procedures collectively described as 'disengagements'. This may be the phenomenon of reverse piezoelectric charge. In Ferreira et al.'s experiments a positive charge was created on stretched tissues. Reverse piezoelectric charge,asthenamesuggests,istheoppositeeffect. Rather than negative ions concentrating in the compressed collagen tissue, positive ions concentrate in the relatively stretched collagen tissue. A similar response may occur in tissues that have a subtle tensioning or traction force applied.

Conclusion

A hypothesis is proposed suggesting biomechanical and biochemical changes may offer a physiological explanation into some of the therapeutic claims and tissue changes palpated by OCF practitioners.

It is hypothesized that the OCF treatment techniques of BMT/BLT, fluid (CV4 and other 'fluid techniques') as well as disengagement, may effect changes within the bony, fascial and fluid tissues of the body. Through subtle compression, utilizing the physical properties of thixotropy, which are attributable to the molecular configuration of the collagen, ionic concentrations can be made to stimulate powerful vasomotive responses, a phenomenon researchers have reported as "a

mechanism of substantial pathophysiological importance".

Research in the areas of biochemistry and physiology, with regard to ionic pumps, calcium oscillations, concentration and electrochemical gradients, may offer insight to observations made by practitioners of OCF.

A scientific hypothesis is offered to explain the conceptual ideas of treatment, still points and BFI and suggestions are offered to explain the scientific reasoning behind the change in fluid quality experienced after treatment.

William Garner Sutherland dedicated a large part of his life conceiving and developing his principles of the primary respiratory mechanism, suffering scepticism, condemnation and reservation from his colleagues for many years. Sutherland believed he was uncovering a fundamental truth and that one day science would substantiate his claims.

Science is unveiling Still and Sutherland's truths. A greater scientific understanding could bring many positive benefits to the cranial model, addressing many of the inconsistencies and misconceptions, the controversy and confusion held amongst OCF practitioners and sceptics of OCF alike. Science can start to offer explanation, seek agreement and consensus to the PRM/CRI, BFI, and the therapeutic changes experienced by OCF practitioners. Scientific explanation can provide consensus as to what OCF practitioners are palpating. Biochemistry is enlightening the mechanisms of vasomotion and offers an explanation to the inconsistencies of inter/intra-examiner reliability testing amongst OCF practitioners and the theory of entrainment.

Further scientific research is needed to offer new insight and explanations for the existence and characteristics of the PRM/CRI and TH oscillations and offer explanation of their origins, control mechanisms and continued insight into their physiological behaviour with regard to the therapeutic response.

The hypotheses offered here are conjectural and do not address all the questions but information is available in increasing amounts and it is in the interest of all OCF practitioners, that would like to see OCF accepted into mainstream osteopathy, that OCF becomes scientifically validated. As Dr Sutherland often quoted "If you understand the mechanism, the treatment is easy".

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