

Review

A review of the physiology of cranial osteopathy

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Abstract

The models generally used to explain the practice of cranial osteopathy have not been supported by reliable research. This paper reviews and explores the relevant physiology and finds much to advance knowledge in this field. Arterial vasomotor waves have a frequency similar to reports of cranial rhythmic impulses; these are controlled by the sympathetic nervous system. Thermoregulation can reverse venous flow through emissary veins of the skull. Cerebrospinal fluid is circulated by arterial pulsations and is partially drained via the cribiform plate into nasal and cervical lymphatics. A model for the practice of cranial osteopathy based on well-researched physiology is proposed, and some clinical implications outlined. Some reasons for poor inter-observer agreement in palpatory studies are discussed. This paper should provide a basis for informed research in this subject in the future.

Key Words: cranial, osteopathy, field, craniosacral, rhythm, vasomotion, sympathetic nervous system, cerebrospinal fluid

INTRODUCTION

Osteopathy was founded in the late 19th century by AT Still. One of his students at the American School of Osteopathy in 1899 was WG Sutherland, who originated and developed his ideas about 'cranial' osteopathy over the next 40 years and published them in 'The Cranial Bowl' in 1939.¹ The key components of the teaching of it, which have not changed much since Sutherland's time, are that:

1. There is 'inherent' motility of the brain and spinal cord.
2. The cerebrospinal fluid fluctuates.
3. There is motility of the intracranial and spinal dural membranes.
4. The bones of the skull are mobile.
5. There is involuntary motion between the sacrum and the ilia that is synchronised with cranial motion by the spinal dural meninges.

Sutherland proposed that this motion was rhythmic and transmitted by fluid or fascia throughout the body so it could be palpated simultaneously at any place. Sutherland originally named this area of practice "Osteopathy in the Cranial Field" (OCF)² so these initials will be used in this paper to include both cranial osteopathy and craniosacral therapy.

Since its beginnings OCF has been a subject of controversy. The lack of evidence supporting Sutherland's model of the underlying physiology has led to a number of alternative

hypotheses, notably the 'pressurestat' model by Upledger³ which suggests that cerebrospinal fluid (CSF) is produced intermittently thus driving the motility of the brain and cranium.

Despite the criticism of the models,⁴ the clinical practice of OCF seems to thrive. Upledger and his followers have taught thousands of non-osteopaths his version of the concept that he calls 'craniosacral therapy'. In 1999 this was the subject of the most thorough review of the relevant literature to date: The Workers' Compensation Board of British Columbia commissioned the study from the British Columbia Office of Health Technology Assessment (BCOHTA) in order to assess whether or not to pay for this treatment. The study searched seven electronic medical, alternative medical and scientific databases, and also searched libraries and requested information from professional associations. Several hundred books, papers and articles were evaluated in three main categories:

- a. Pathophysiological mechanisms of craniosacral dysfunction
- b. Craniosacral assessment
- c. Craniosacral treatment/interventions

The study "did not find valid scientific evidence that craniosacral therapy provides a benefit to patients".⁵

"The beneficial effects of craniosacral therapy (CST) on health outcomes have not been demonstrated using well-designed research protocols. Inter-observer agreement studies have found that assessment... is unreliable. There is little evidence that CST has a valid pathophysiological rationale."⁵

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This is the rational scientific position in the light of the evidence that has been published. However there are thousands of OCF practitioners worldwide and many patients who seem to benefit, so the question is "Is there a credible physiological basis for this practice?"

Rhythmic motion

There are many physiological changes that happen rhythmically or cyclically within the human body. These range from cycles lasting milliseconds, such as some neural reflexes, to cycles lasting hours, days, weeks or months as in some hormonal rhythms. In this study we are investigating what OCF practitioners call the 'Cranial Rhythmic Impulse' (CRI), which is usually described as having a rate of 6 - 15 cycles per minute (cpm).⁶ This is slower than the heart beat, and was claimed by Sutherland¹ not to be linked to breathing because it is not synchronous with it.

Palpating the rate of the CRI has proved to be difficult, with different osteopaths suggesting differing rates, and studies generally showing poor interrater reliability. Also some authors have reported palpating slower rates underlying the usual one; Norton et al.⁷ reported a rate of 3.7 cpm as an average of 12 examiners and 24 subjects and Becker⁸ reported a 'deeper rhythm' of 0.6 cpm. Objective measurements of the CRI have been few and unreliable. This is because researchers, if looking for physical movement in the skull, have had difficulty detecting very small ranges of movement (probably less than 0.3 mm) in living subjects.

As the rate of the CRI is subject to disagreement, and practitioners may be palpating more than one rhythm, it is no surprise that studies of interrater reliability have shown that there is little objectivity in the measurements. Wirth-Pattullo and Hayes⁹ showed that, with 3 examiners and 12 subjects, the CRI was not related to heart rate or breathing, but there was a large variation in the rates reported by the 3 examiners on each subject, suggesting that their palpation of the rate was unreliable. Rogers et al.¹⁰ measured 2 examiners palpating simultaneously the head and feet of 28 subjects and finding no interrater correlation and "did not support claims that cranosacral motion can be palpated reliably". Moran and Gibbons¹¹ have also showed that, with 2 examiners and 11 subjects, there was little intrarater or interexaminer reliability when palpating the head and sacrum.

In the following review and exploration this study will focus on exploring the possibility of rhythmic physical motion of the tissues of the cranium in the range of 0.6 to 15 cpm, which are the limits of the reported range of CRI palpated. In scientific research frequency is usually measured in Hertz (Hz), so 15 cpm is 0.25 Hz and 0.6 cpm is 0.01 Hz.

The physiological basis of cranial rhythmic motion tissue by tissue

Arteries

The blood flow into the brain is approximately 750 ml/min,

or 14% of cardiac output.¹² The most obvious source of rhythmic motion in the cranium is the heartbeat, though at around 72 beats per minute this is outside the range of frequencies linked with OCF. However arterial blood pressure does fluctuate rhythmically within the range ascribed to the cranial rhythm. Measurements by many researchers show two peaks, at 0.25 Hz (HF or high frequency peak) and at 0.1 Hz (Low Frequency or LF peak). These frequencies are 15 cpm and 6 cpm respectively. The HF peak (known as the Traube-Hering wave¹²) is generally linked to respiration (breathing) and vagal parasympathetic activity. The LF peak (known as the Mayer wave) is linked to input from baroreceptors and chemoreceptors in the carotid sinus and aortic arch and to sympathetic nervous system (SNS) control of arterioles throughout the body. There are also peaks at even lower frequencies, around 0.015 Hz (about 1 cpm), which are little researched, but can modulate respiratory and haemodynamic fluctuations,¹³ and may be related to thermoregulation.¹⁴

There are researchers who call all the rhythmic fluctuations in blood pressure 'Traube-Hering-Mayer' (THM) waves. Recently these have been shown to be synchronous with palpation of the CRI.¹⁵ Given the poor record of palpation of cranial rhythms such findings need to be treated with care, though potentially they open up interesting possibilities for the physiological basis for and measurement of the CRI, which will be discussed later. The same team also found increases in amplitude of the 'Traube-Hering' wave (this may have been incorrectly named and was most likely the 0.1 Hz or 6 cpm Mayer wave) following cranial manipulation in a small trial using two subjects.¹⁶

Corresponding to the rhythmic variations in blood pressure are similar, linked, rhythms in the variability of the heart rate.¹⁷ This heart rate variability (HRV) or heart period can be measured from sequences of R-R intervals from electrocardiographs (ECG) and shows the same two peaks at about 0.25 Hz and 0.1 Hz. It has recently been shown that the 0.1 Hz heart period (R-R) variation is almost entirely accounted for by a baroreflex mechanism.¹⁸

The calibre of arteries and arterioles also fluctuate rhythmically at several rates; the basilar artery has been observed to alter its calibre at a rate of 0.2 cpm¹⁹ and smaller arteries exhibit vasomotion at a rate of 0.74 cpm independent of blood pressure or respiration.²⁰ Significant rhythmic variations in diameter of the radial artery have been recorded at a rate of about 1 cpm.²¹ Researchers using reflected light imaging of neural activity found a pervasive 0.1 Hz (6 cpm) signal present in brain parenchyma and microvasculature and also in the circulation under the fingernails; concurrent measurements of brain vasculature with a laser Doppler flow meter contained an almost identical and synchronous 0.1 Hz signal.²²

It appears that larger arterioles exhibit slower vasomotion rates than smaller vessels. In hamsters it has been found that the largest arterioles (diameter about 30 microns) supplying skeletal muscle had a diameter fluctuation frequency of 0.3 to 3 cpm, whereas the smallest arterioles (diameter about 7.5 microns) fluctuated at between 4 to 15

cpm; these components were superimposed on each other, with the low frequencies transmitted downstream and the high frequencies upstream resulting in a complex superposition of waveforms.²³ Hypoxia (11% O₂) increases the frequency of vasomotion, especially in smaller vessels²⁴. The dominant frequency is usually around 0.1 Hz (6 cpm) generated by medium arterioles (Strahler order 3) which spreads downstream to smaller vessels, and can increase to 0.3 to 0.5 Hz (18 to 30 cpm) with systemic hypoxia, a response that is suppressed by blocking the sympathetic nervous system discharge with the drug phentolamine.²⁵

Further support for an arterial component to rhythmic motion of the cranium has been provided by Moskalenko et al²⁶ in their measurements of rhythmical changes in the shape and volume of the cranium using serial NMR scans. Apart from confirming a rhythm of 6-14 cpm and the physical expandability of the bony cranium, the research also involved injecting 20ml of liquid into the carotid artery which had an immediate expansion influence on the cranium, so adding to the evidence that arterial volume influences intracranial volume.

Veins

Farasyn²⁷ has proposed that the CRI is a result of venomotion. This is unlikely due to the low pressure in most veins, particularly intracranially where the venous pressure can be negative when standing. Researchers have found that venules do not exhibit vasomotion.²⁸ Cabanac and Brinnet²⁹ have demonstrated that venous blood can flow through the emissary veins between the scalp and the intracranial venous vessels. In cadavers after removal of the top (vault) of the cranium venous blood can be made to appear on the inner surface through numerous (valveless) emissary veins by massaging the scalp. Cabanac also demonstrated that at normal or low body temperatures there is little flow in these veins, though if flow was recorded it was from brain to skin. During hyperthermic conditions (induced by exercise) strong flow was demonstrated from skin to brain in parietal and mastoid emissary veins, which helped to cool the core temperature of the brain. The pressure in the veins was very low and could be collapsed by light (4 gram) pressure, which would not happen with an artery. This temperature dependent change of flow direction has also been observed in the ophthalmic vein³⁰ where cool blood from sweating faces was directed toward the cavernous sinus that acted as a heat exchanger to cool warm arterial blood; in hypothermic conditions the flow was from brain to face. It has been proposed that the highly vascular dura mater may transmit venous cooling from the surface of the skin to the CSF and brain parenchyma.³¹ These alterations in venous flow add to the data concerning the fluid dynamics of the human cranium, however they are part of thermoregulation and are probably not part of palpable rhythmic changes.

Lymph

Like arteries the larger lymph channels exhibit pulsatile dilatation and constriction. Measurements of 5.2 cpm (0.09 Hz) have been recorded in the thoracic duct in sheep³² and

6-8 cpm in humans.³³ It has recently been concluded that "lymphatic contractility plays crucial roles in the regulation and generation of lymph transport".³⁴ The capillaries within the brain have tight junctions between their cells, so forming the 'blood-brain barrier', so little lymph is produced within the brain. A small amount of lymph is produced in some areas within the cranium but outside the blood-brain barrier, such as the circumventricular organs.³⁵ This lymph drains through arterial and venous openings in the cranium, particularly around the olfactory nerve as it passes through the ethmoid, en route to the cervical lymph nodes. 70% of lymph is reabsorbed into the venous system at lymph nodes.³⁶ As there are no large lymph vessels within the cranium it is unlikely that the lymphatic system causes the CRI.

Cerebrospinal fluid

About 150ml of cerebrospinal fluid (CSF) fills the ventricles and subarachnoid space in the cranium and spine. The rate of production is usually cited as 550ml per day, thus it turns over about 3.7 times each day,¹² however some research has found that production is 900-1000 ml per day³⁷ and found no differences related to adult age. 50-70% of CSF is formed in the choroid plexuses and the remainder around blood vessels and in the walls of the ventricles.

CSF is circulated within the ventricular system and spinal canal by the heartbeat and slower rhythmic fluctuations of the arterial system as described above. The cranium is viewed, in the 'Monro-Kellie doctrine', as a closed box containing the 1400g brain and about 75ml of blood and 75ml of CSF, all of which are essentially incompressible.¹² So a rise in pressure in arteries or veins causes a rise in intracranial pressure, which immediately triggers a reflex drop in arterial pressure. Cranial arterial blood flow is maintained at a steady state despite significant increases in arterial pressure. CSF is not produced in an intermittent way nor with sufficient pressure or in sufficient quantity to cause expansion of the ventricles and brain, as proposed by Upledger in his 'Pressurestat' model.³

The prevailing view is that CSF is absorbed through the arachnoid villi into veins, particularly the venous sinuses.¹² Research has shown that, in dogs, CSF can flow into the cervical lymph nodes, via the olfactory nerve and around the cribriform plate to the nasal submucosa.³⁸ This was confirmed by a study in rats that found direct drainage of CSF through the cribriform plate to connect with nasal lymphatics.³⁹ Finally recent research has challenged the conventional view of CSF absorption and shown that, not only did CSF drain through the cribriform plate, but this was the primary route at low intracranial pressures, with arachnoid villi and other lymphatic pathways only being recruited at elevated intracranial pressure, this study demonstrated the importance of drainage through the cribriform plate, in sheep at least, by blocking the plate and finding impaired CSF clearance.⁴⁰ A recent commentary on the relationship between CSF and extracranial lymph confirms that in sheep and rats 50% or more of CSF was cleared via the lymphatic system, rather than through arachnoid villi.⁴¹ Evidence also suggests that approximately 25% of CSF is cleared via the spinal subarachnoid space.⁴²

Further research has confirmed that obstructing the drainage of CSF through the cribriform plate to the nasal mucosa leads to reduced CSF clearance and an increase in intracranial pressure.^{43,44} So the lymphatic drainage of the cranium is important, even though there is minimal lymph produced in the brain – CSF drains instead. Although this is an important topic in the hypotheses of the physiology behind OCF, the production and drainage of CSF is unlikely to cause to rhythmic motion.

Brain and neural tissue

There have been many studies of the effect of the heartbeat causing movement in the brain and CSF.^{45,46} Cardiac systole expands the intracranial arteries, which in turn leads to an expulsion of CSF into the compliant spinal subarachnoid space. At the same time the brainstem moves caudally between 0.1-0.5 mm towards the foramen magnum and the cerebral lobes move cephalically.⁴⁷ The ventricles are squeezed so moving the CSF contained within them, with outflow from the aqueduct mirroring the brain expansion in systole.⁴⁸ Cardiac systole also causes an instantaneous increase in the flow in the Superior Sagittal Sinus, which helps venous drainage. The brain stem can also be seen to move in relation to breathing, and the Valsalva manoeuvre produces an initial caudal and subsequent cephalic displacement of 2-3 mm.⁴⁹ The cervical spinal cord also moves caudally just after systole,⁵⁰ a movement that is transmitted down to the lumbar enlargement.⁵¹ CSF flow in the spinal canal is aided by spinal vascular pulsations.⁵² There is no evidence that brain tissue has any inherent contractile ability to move CSF, as originally hypothesised by Sutherland.¹ The force of arterial pulsation is enough to move CSF both within and around the brain and down the spinal canal. It is probable that the slower rhythmic dilatation and contraction of the arterioles within the brain associated with changes in vasomotion and blood pressure, described above, are responsible for the pervasive 0.1 Hz signal captured from the brain parenchyma by reflected light imaging.²² These slower arterial rhythms may also have a role in the circulation of CSF, though it is likely to be a smaller effect than that of the heartbeat.

Bones, joints and dura

There is an increasing body of evidence to support the concept that small amounts of movement are permitted at the cranial sutures throughout life. This mobility, combined with the flexibility of the bones of the cranial vault, leads to a degree of compliance to intracranial volume changes. Retzlaff's work demonstrated that cranial sutures were complex and persistent joints.⁵³ The systematic review of the scientific evidence on craniosacral therapy by the BCOHTA described above did state:

“The research evidence reviewed supports the theory that the adult cranium is not always solidly fused, and that minute movements between cranial bones may be possible. However, no research demonstrated that movement at cranial sutures can actually be achieved by manual manipulation”.⁵

Cranial vault sutures need to remain unossified in children

to allow growth,⁵⁴ as intramembranous bone plates grow at their edges in response to external stimuli – in this case the expanding neurocranium. Their persistence in adults may be related to shock or trauma absorption, as well as improving the compliance of the cranium to changes in intracranial pressure and volume. It is interesting to note that the details of the pattern of cranial sutures is unique to each individual, to the point that they can be used for forensic identification.⁵⁵ Only recently has modern technology made possible reliable measurements of small changes in the position of cranial bones. This has been achieved by several methods:

1. Serial X-rays and NMR tomograms to demonstrate intracranial dimension changes of about 0.38mm, which alternated between sagittal and frontal (AP) expansions.²⁶
2. Movement at the sagittal suture in cats directly linked to changes in intracranial volume.⁵⁶
3. X-ray changes in the positions of several bony landmarks have recently been demonstrated comparing 12 adult patients who had fixed positioned x-rays taken before and immediately after a cranial osteopathic treatment.⁵⁷ This pilot study showed that bony changes could be achieved by manual manipulation.
4. 3-D video imaging to measure rhythmic changes in shape of the human orbit, which have several frequency waves.⁵⁸

In the foetal and infant skull the osseous components are incompletely developed, and in adult skulls there is the possibility of sutural motion and bone compliance, which raises questions about the structural integrity of the skull. Both of these issues can be addressed by including the internal architecture of the cranium in the form of the dural membranes. These stabilise the spherical cranium three-dimensionally to stop it from over-expanding in any direction. The falx cerebri and falx cerebelli divide the brain in a sagittal direction, whilst the tentorium cerebelli provides a lateral component.¹ These are in a state of reciprocal tension so that the cranium tends to alternately expand laterally or in an AP or sagittal direction, which seems to be confirmed by experimental evidence.²⁶ The osseous components of the vault of the skull are effectively formed within the layers of the dura, so the dura has a significant influence on the compliance and mobility of the cranium.

Traditionally in OCF emphasis is placed on a dural link between occiput and sacrum,³ though this is controversial even between practitioners.⁴ It is unlikely that this link is direct enough to transmit perhaps 0.3 mm of movement, as there must be considerable slack in the dura to allow spinal movements; the spinal canal is 5 cm to 9 cm longer in flexion than extension of the spine.⁵⁹ The spinal cord dura is elastic,⁶⁰ and the cervical spinal cord itself elongates by 10% in flexion of the neck.⁶¹ Palpatory studies have shown that there is no link between the rhythms palpated at head and sacrum.¹¹

Whilst the bones of the cranium, together with their internal

membranous (dural) architecture do have the potential to move minutely, they do not generate rhythmic movement themselves.

Muscles

The tissue in the body that is usually associated with movement, rhythmic or otherwise, is muscle in its various forms. Indeed even arterial systolic motion is generated by cardiac muscle, with the smooth muscle in the walls of arterioles controlling vasomotion. Much of the body is made of skeletal muscles, and there are a great number attached to the cranium including muscles of the neck, throat, jaw, face and scalp. Of particular interest is one of the suboccipital muscles called rectus capitis posterior minor (RCPM) that was discovered in 1995 to be connected, via a connective tissue bridge, to the dorsal spinal dura at the atlanto-occipital junction.⁶² The RCPM is unlikely to exert much force on the whole dura; the probable purpose of it is to prevent infolding of the dura during head and neck extension. Recent research⁶³ has also shown continuity in the midline between the nuchal ligament and the posterior spinal dura at the atlanto-occipital and atlanto-axial intervals. It is plausible that healthy functioning of RCPM aids CSF circulation in the top of the spinal canal as CSF is ejected into it during cardiac systole, and partially returns in diastole. This may be some support for the, otherwise unlikely, concept in OCF of being able to compress the forth ventricle (CV4),³ most effects of this technique are likely to be due to relaxation of the suboccipital muscles, rather than any direct effect on the ventricles. Suboccipital muscles have a high density of muscle spindles, and give significant proprioceptive input to the balance centres; for example somatic dysfunction in the upper neck has been linked to tinnitus,⁶⁴ and to neck pain, headache and impaired balance.⁶⁵

Skeletal muscle does not appear to contract in a rhythmic manner in the 0.6 -15 cpm range that is the focus of this study; however many of the patterns of movement felt around the cranium in clinical practice are linked to patterns of myofascial tension. Some authorities⁶⁶ assert that, as there is no activity in the efferent motor nerve to a muscle when it is inactive, then resting muscle tone is a result of fluid accumulation or structural connective tissue changes, although that would not explain the flaccidity of a muscle whose nerve supply has been interrupted. There is evidence that this 'myogenic' tone may be due to thixotropic stiffness due to protein cross-bridges.⁶⁷ Whilst the effects of physical therapy, such as massage, could affect myogenic tone, on its own it would not fully explain the capacity for a resting muscle to further relax with self-directed relaxation techniques or emotional therapy. The influence of nerves on resting muscle tone remains controversial,⁶⁸ there are several physiological possibilities for 'neurogenic' influences on resting muscle tone and patterns of tension:

a) Small groups of motor units are stimulated in a variable pattern to maintain some tension in the muscle, yet allowing most of the motor units to rest.⁶⁹ Slow (red) postural fibres within muscles are tonically active. The blood flow to red fibres is three times greater than to white fibres in a resting muscle.

b) Gamma efferent influence on the muscle spindle maintains muscle length,¹² and can be influenced by therapy to allow relaxation and lengthening of tight muscles. Sympathetic nerve fibres have been shown to affect the function of muscle spindles, either by direct action on intrafusal fibres^{70,71} or by influence on the afferent discharge of muscle spindles.⁷²⁻⁷³

c) It has been postulated that 'myofascial trigger points' exist as bands of high sensitivity in abnormally tense muscles. These do not cause motor nerve activity, but are related to physiologically abnormal motor endplate function,⁷⁴ with the release of excessive acetylcholine and localised contraction, which may lead to local inflammation, nociceptor stimulation and pain.⁷⁵ This mechanism could account for localised increases in resting muscle tone.

The possible physiological mechanisms for a neurogenic component to resting muscle tone described above do not seem to have any rhythmic aspects. What does alter rhythmically is the level of muscle sympathetic nerve activity (MSNA), which controls the blood supply to muscles via its influence on the smooth muscle in the walls of arterioles.⁷⁶ There is a measurable 5-6 cpm variation in blood flow to muscles even when they are contracting.⁷⁷ When muscles contract local chemical mediators such as adenosine act as vasodilators.⁷⁸ The vasoconstricting effect of MSNA may have a role in maintaining blood pressure by limiting blood flow to large exercising muscles. MSNA is altered by vestibular otolith stimulation,⁷⁹ exercise,⁸⁰ heart rate variability and blood pressure,⁷⁶ and resting MSNA is decreased by oestrogen.⁸¹

The control of rhythmic fluctuations

Excluding breathing, the principal tissue that changes rhythmically in the range of 0.6 to 15 cpm (0.01 to 0.25 Hz), which is the focus of this study, is in arteries, specifically the smooth muscle in the walls of arterioles that control their diameter. No other tissues have been shown to cause rhythmic fluctuations, though further research is necessary to learn more about the resting tone of skeletal muscle. Any rhythmic motion in the cranial bones, meninges, CSF and CNS is probably secondary to cardiac and arterial influences.

In relation to the rhythmic changes in arterial blood pressure and heart rate variability at 6 cpm (0.1 Hz) the main mode of control is through the sympathetic division of the autonomic nervous system,⁸² when sympathetic activity is decreased by nitroprusside the HF (High Frequency, 0.25 Hz) component predominates, which is linked to parasympathetic (vagal nerve) activity. Synchronous changes in the LF (Low Frequency, 0.1 Hz) and HF rhythms of R-R interval, skin microcirculation and MSNA throughout the body during different levels of sympathetic drive are suggestive of common central mechanisms governing both parasympathetic and sympathetic cardiovascular control.

A study by Bernadi et al.⁸³ found that rhythmic oscillations in skin blood flow were synchronous in both index fingers

in each subject, and also that the LF (0.1 Hz) oscillations in skin blood flow led the synchronised changes in blood pressure – indicating an ‘upstream’ transmission from the microvessels. These synchronous fluctuations were not observed in patients who had had a sympathectomy, where the sympathetic nerve supply to the arm had been severed. By contrast the HF (0.25 Hz) changes in skin blood flow lagged behind the blood pressure fluctuations indicating a ‘downstream’ transmission.

The differences in the rates of autonomic modulation of the heart may reflect the fact that the parasympathetic division produces acetylcholine, which is rapidly broken down by acetylcholinesterase, so allowing repolarisation at a higher frequency (around 0.25 Hz). Sympathetic release of norepinephrine has a slower recovery resulting in a lower frequency of neural activity (around 0.1 Hz).⁸⁴

The link between LF arterial pressure fluctuations and overall sympathetic activity has been challenged by Taylor et al,⁸⁵ who found that LF (Mayer wave) fluctuations were the same in young males, young females and older males despite the great differences in other measures of sympathetic activity, notable MSNA, between these groups.

Breathing affects the higher rate (HF) blood pressure oscillations around 15 cpm (0.25 Hz), which are fully abolished during 1-minute voluntary apnoea,⁸⁶ although at the same time lower frequency oscillations (0.1 Hz and around 0.03 Hz) were enhanced.

There is a clear correlation between oscillations in cerebral blood flow velocity, measured by transcranial Doppler sonography, and oscillations in arterial blood pressure.⁸⁷ Cerebral blood flow is influenced by sympathetic stimulation, being decreased by stimulation of cervical sympathetic nerves,⁸⁸ which also increases the frequency and decreases the amplitude of waves of vasomotion. Generally cerebral blood flow fluctuates at 6 cpm (0.1 Hz) as a result of sympathetic nervous system control, which is linked to electrical activity in the autonomic control areas of the medulla, cerebellum and cortex.⁸⁹ Regional cerebral blood flow changes rapidly in response to local neuronal activity, triggering local vasodilation by bursts of electrocortical activity.⁹⁰ 0.1 Hz oscillations have been observed in electroencephalogram (EEG) recordings during sleep.⁹¹

The sympathetic nervous system (SNS) is the main influence on arterial tone and has moved to the centre stage in cardiovascular medicine,⁹² particularly regarding the pathogenesis of hypertension. The SNS affects heart and kidney function in addition to skeletal muscle vasculature, and contributes to ventricular hypertrophy and arrhythmias. Sympathetically mediated vasoconstriction in skeletal muscle vascular beds reduces the uptake of glucose by muscle, and is thus a basis for insulin resistance and consequent hyperinsulinaemia. The relationship between the increased sympathetic tone and decreased parasympathetic tone in hypertension is reciprocal, which strongly suggests that the abnormality emanates from the brain.⁹³ SNS activity tends to increase with age, though

there can be parts of the body that are spared.⁹⁴ There is some evidence that positive and negative emotions can influence the SNS.⁹⁵

To summarise, research has shown that the main rhythmic oscillation in arterial blood pressure and heart rate (R-R) variability is one that has a peak around 0.1 Hz (6 cpm, Mayer wave) and is generated by vasomotion in the medium sized arterioles present throughout the body. The arterioles contract under the influence of the sympathetic nervous system (SNS) and provide resistance to blood flow, which raises the blood pressure ‘upstream’. The rise in blood pressure triggers a baroreflex mechanism that alters the heart rate variability.¹⁸ The SNS control is coordinated centrally, as it is synchronous in the limbs, and is probably largely at a reflex spinal level as it is bilaterally synchronous in tetraplegics.⁹⁶ There is central coordination of parasympathetic (Vagal) and SNS activity in relation to the heart. Breathing influences both heart rate variability and blood pressure at around 0.25 Hz (15 cpm) as a result of physical influences on the heart and circulation, particularly venous return, due to pressure changes in the thorax.

A physiological model for cranial osteopathy

The evidence supporting rhythmic changes in arterial vasomotion, which leads to blood pressure fluctuations and heart rate variability within the cranium, is now strong and the evidence for cranial compliance involving bony movement is growing. It is reasonable to propose that these changes in the arterial system, together with bony mobility, are part of the physiological basis of OCF. Arterial pressure changes, both due to cardiac systole and slower variations due to vasomotor activity, move the brain and cause CSF to flow in the ventricles and around the brain and spinal cord. The dural membranes within the cranium are a necessary part of the internal architecture of the cranium to prevent over-expansion in any direction.

The rates of the peak fluctuations in arterial diameter and pressure correspond to the reported rates of the CRI,⁶ many authors suggest around 6 cpm (0.1 Hz), though some may be palpating the faster circa 15 cpm (0.25 Hz) waves that are more linked to vagal activity and breathing.

These findings would be compatible with an axiom promulgated by the founder of osteopathy, A.T.Still,⁹⁷ for whom “the rule of the artery is supreme”. They would also be compatible with most of the ideas of the originator of OCF, W.G.Sutherland,¹ who described rhythmic motion of the cranium and analysed the arrangement of the sutures and dural membranes, though, in the light of current knowledge it would seem that he incorrectly hypothesised that intrinsic movement of the brain pumped CSF, which in turn moved the cranial bones and the rest of the body.

Clinical implications

If the physiologically based model described above is a more accurate model for representing the phenomena experienced in the practice of OCF then there are clinical implications for that practice, as this model differs from the model that

has been generally used and taught. The traditional model is focused on bones, dura and the flow of CSF, which is assumed to be absorbed into the venous system via arachnoid villi. The new understanding adds elements of arterial function, autonomic control and altered CSF drainage.

Cardiovascular system

If the practice of cranial osteopathy is able to detect and influence the SNS control of arterial vasomotion then consideration of the cardiovascular system as a whole is important. Heart problems may lead to decreased cerebral perfusion, which may cause depression.⁹⁸ Conditions such as uraemia have been shown to make a dramatic reduction in the amplitude of the 0.1 Hz (6 cpm, Mayer wave) fluctuations in blood pressure and heart rate variability,⁹⁹ which may indicate impaired cardiovascular ANS function.

The control of these rhythmic changes is through the autonomic nervous system (ANS). Osteopathic teaching has suggested that the ANS acts as a "mediator between the somatic and supportive processes".¹⁰⁰ Treatment that influences somatic function may have an effect on ANS function. If a healthy balance between SNS and parasympathetic activity can be encouraged then a variety of pathophysiological states may be helped, including hypertension.

The possible influence of cranial therapy on intracranial circulation means that its use should be avoided in cases of haemorrhage. Adverse effects of craniosacral therapy have been reported in patients with traumatic brain injury.¹⁰¹ Physical manipulation should be avoided in any area of the body where there is a risk of aggravating haemorrhage. If cranial osteopathic treatment can influence intracranial circulation then it may be of benefit in cases where blood flow is compromised, such as transient ischaemic attacks or strokes; further research would be necessary to investigate this possibility.

Nervous system

If ANS function is a part of cranial osteopathic diagnosis and treatment then consideration of the pathophysiology of the nervous system is important. Pathological conditions in the central or peripheral nervous systems may influence autonomic function. Longstanding Parkinson's disease can cause reduced sympathetic vasomotor and cardiomotor outflow leading to diminished Mayer waves.¹⁰² Alcoholism can cause autonomic neuropathy,¹⁰³ as can diabetes.

Cerebrospinal fluid drainage

The possibility that the drainage of CSF in humans is to a significant extent through the cribriform plate, as it is in sheep,⁴⁰ raises possibilities of focusing treatment on that area. Disturbance of CSF drainage may help to explain the interference with thought processes that seem to accompany a heavy cold or bad hay fever, when the nasal mucosa is swollen and the drainage of CSF may be impaired. The large number of sutures between the bones of the face suggests that movement is important in this area, which

could be to aid drainage of the sinuses and cribriform plate, and may represent a credible mechanism for the influence of cranial osteopathic treatment on the dynamics of CSF.

Muscular tension patterns

It is a feature of clinical osteopathic practice that many symptoms arise from, and treatment is directed towards, dysfunctional patterns of muscular and fascial tension. Abnormal autonomic activity may be part of the pathophysiology of myalgic encephalomyelitis (ME) or chronic fatigue syndrome, which osteopathic treatment has been shown to help.¹⁰⁴ Diffuse Muscle Coactivation (DMC), an increase in electrical activity and resting tone of a muscle during a movement that does not involve that muscle has been demonstrated in cases of fibromyalgia,¹⁰⁵ which may be similar to the osteopathic concept of 'facilitated segments' proposed by Korr.¹⁰⁶

Breathing

Breathing (respiration) influences brainstem motility⁴⁹, thoracic venous return¹², heart rate variability⁸⁶ and the 15 cpm (HF) vasomotor waves, so it is reasonable to suggest that clinical attention to the encouragement of a good (diaphragmatic) breathing pattern might have some influence on the dynamics of blood, lymph and CSF and also reduce hypertonia in the accessory muscles of respiration.

Palpation and Treatment

Despite the possibilities opened up by this model three important questions need to be addressed:

1. Is it possible to palpate these arterial rhythms manually?
2. Can they be altered by external human intervention (treatment)?
3. Does this alteration have any physiological benefit for the patient?

To date evidence has tended to suggest that cranial rhythms are not palpated with any degree of reliability.^{9,10,11} There are several possible explanations for the differing rhythms palpated by different practitioners:

1. Arterial vasomotion occurs at different rates in different sized arteries and arterioles. The predominant frequency is 6 cpm (0.1 Hz), which occurs in medium-sized arterioles, however larger arteries exhibit spontaneous vasomotion at slower rates for example 0.2 cpm (0.0033 Hz) in the Basilar artery,¹⁹ and 1 cpm (0.02 Hz) in the Radial artery.²¹ The smallest arterioles exhibit diameter fluctuations up to 15 cpm (0.25 Hz).²³ It is possible that individual practitioners focus on different rates of vasomotion generated by different sized arteries.
2. Practitioners may palpate differing depths of movement; the scalp is highly vascular so those utilising light finger pressure, as is encouraged by teachers of 'craniosacral therapy',³ may be detecting vasomotion in the scalp.
3. Norton¹⁰⁷ has proposed that the sensation described as

the cranial rhythmic impulse (CRI) is related to activation of slowly adapting cutaneous mechanoreceptors by tissue pressures of both the subject and the examiner, and that the sources of change in these tissue pressures are the combined respiratory and cardiovascular rhythms of both examiner and subject.

4. McPartland¹⁰⁸ takes Norton's model further and has proposed that the CRI is the palpable perception of entrainment, a harmonic frequency that incorporates the rhythms of multiple biological oscillators, not only heart rate and respiration, but also heart-rate variability and arterial vasomotion with Traube-Hering-Mayer (THM) modulation, and oscillations in lymphatic vessels, glial cells and cortical metabolism.⁴ It is derived primarily from signals between the sympathetic and parasympathetic nervous systems. This model includes the examiner's own rhythms so could explain the differing rates of CRI palpated on a subject by two examiners at the same time.

The concept of entrainment is utilised in the treatment of stress and emotional states by measuring 0.1 Hz heart rate variability and teaching subjects to achieve it through the expression of positive (happy, calm) emotions.¹⁰⁹ Physiologically the 0.1 Hz frequency can be entrained by 0.1 Hz leg movements, which stimulate local vasomotor reflexes and baroreceptor outflow, and can also be entrained by periodic neck suction, which stimulates baroreceptors.¹¹⁰

Whether or not treatment can affect these rhythms has yet to be convincingly researched. A recent small study has suggested that treatment can increase the amplitude of 0.1 Hz fluctuations,¹⁶ though more rigorous research will be needed to confirm this. Even if it is demonstrated that cranial practitioners can palpate and influence these rhythms, further studies are needed to determine whether or not this external influence has any physiological health benefits.

Further research

Considerable physiological and clinical research needs to be undertaken to clarify and support (or challenge) the theory and practice of OCF. In the past the problem has been that clinical practice seems to be successful, yet the theories behind it have been controversial and reliable research has not been able to validate the concepts and practice. One of the reasons for this has been the limitation of available technology that can make reliable and reproducible measurements of the phenomena involved. Various methods have been tried over the years, but generally they have been one-off experiments with questionable methodology or unreliable technology. Recent improvements in technology mean that it is now possible to reliably measure changes in cranial bones, for example using functional Magnetic Resonance Imaging or pulsed phase-lock loop ultrasound.¹¹¹ Blood pressure (BP) fluctuations can be measured in a clinical setting using a 'Finapres' device attached to a finger which, when combined with the appropriate software, can be used to analyse arterial BP waveforms which correlate well with changes in cerebral blood flow in the 0.1 Hz range.¹¹² Changes in the HRV can

be measured with electro-cardiograms (ECG). So if the model proposed in this paper is feasible then reliable research should be able to support or refute it.

CONCLUSION

This discussion has reviewed current research concerning aspects of the physiology behind the theories and practice of cranial osteopathy. There is evidence of movement between and compliance of cranial bones, and there is evidence that arterial vasomotion occurs at rates usually associated with the CRI. It was proposed that these might form part of the basis of the practice of OCF. The clinical implications of this model were discussed. Several clinically important aspects of physiology have been brought into general osteopathic awareness by this study:

1. An understanding of the properties of arteriolar vasomotion. There is a pervasive rate of 6 cpm (0.1 Hz) that corresponds to most reports of the CRI, though there are differing rates depending on the diameter of the artery. The effects of synchronous vasomotion on arterial blood pressure and heart rate variability were discussed.
2. The possibility of reversible venous flow through emissary veins of the cranium as part of thermoregulation.
3. The possibility that a significant proportion of CSF is drained via the cribriform plate to the cervical lymphatic system. The mechanisms for the circulation of CSF were reviewed.
4. Clarification of the role of the sympathetic nervous system (SNS) in its control of vasomotion and effects on blood pressure, with the possibility that there may be direct SNS influence on skeletal muscle.

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Viewpoint

McGrath MC.

This review¹ echoes the position that there is no scientific justification for osteopathy in the cranial field (OCF), a position which has been previously well described in the literature.^{2, 3, 4, 5, 6, 7} An alternative theoretical model is proposed. This proposed model theorises that cranial bone movement is the result of "rhythmic changes in arterial vasomotion, leading to blood pressure fluctuations within the cranium." It is proposed that arterial pressure changes due to cardiac systole and vasomotor activity move the brain and cause CSF to flow. This, coupled with evidence of slight sutural movement and cranial bone compliance, is presented as offering the potential for a more likely theoretical basis. New theoretical model notwithstanding, there remains an absence of demonstrated validity in OCF theory and clinical practice that reaches well beyond merely tinkering with a theoretical model.

It is stated that there is evidence for compliance and movement of the cranial bones.¹ It is however, biologically implausible to suggest a link between this and the explanation postulated by the new model. That slight cranial bone movement occurs under certain conditions is undeniable: it is the conditions by which such movement occurs that is the critical point. Cohen and MacLean⁸ give a detailed description of cranial sutural biology. Cranial sutures interlock to prevent the calvarial bones from separating due to external forces. With advancing age, the sutures become increasingly bound together by bony spicule formation and eventually, though not necessarily, close completely. A particular feature of this process is the formation of bony spicules (bridges) within the suture between the two adjacent bones. Spicules may bridge the

suture either completely or partially along its entire length. Alternatively, irregular, acellular calcified masses appear singly or as coalesced aggregates within the sutural gap, providing attachment to bone spicules bridging the sutural gap. Eventual bony fusion may occur endo- or ectocranially. A lack of sutural movement is a desirable characteristic between the cranial bones, whether or not complete sutural closure occurs. The adequate protection of the CNS is at stake. Any sutural movement or bony compliance that may occur, does so only in association with considerable externally applied loads. In a study by Losken et al.,⁹ an applied tensile load of 50kg produced a sutural displacement of 1mm in rabbits with delayed onset cranial synostosis, equivalent to human sutural closure in a 20 - 30 year old. Normal rabbit sutures, regarded as equivalent to human children, require a 15kg tensile loading to produce a sutural displacement of 1mm. These forces are many orders of magnitude greater than those advocated in OCF, where 5g manual pressure is said to be necessary to produce a movement of 50microns.¹⁰ Additionally, knowing that the calvarial bones must move at a rate and amplitude sufficient to breach the threshold of the examiners human tactile perception, it is straightforward to estimate the necessary intracranial pressure required to cause the alleged cranial bone motion, which turns out to be several orders of magnitude greater than the established values of intracranial or CSF pressure. Such a value is not only biologically implausible but it would be quite incompatible with life.

So what is it that the practitioner of OCF feels? I suggest that there is evidence to show that cranial rhythmic impulse (CRI) is a manifestation of an extra-cranial blood flow phenomenon and an observational artifact of no clinical significance. The cranial literature states that it is difficult

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to palpate CRI^{11, p. 905} and indeed many cannot. Absence of reliability and validity are consistently associated with attempts to detect the CRI^{2, 4} and this may be because the CRI, as an inherent biological entity, does not exist. The observed inconsistency of this finding may lie in the previous explanation that the CRI phenomena is an artefactual extra-cranial phase interaction of Traube-Hering-Mayer (THM) oscillations between the examiner and the subject.¹² Unless there is an extra-cranial THM phase coincidence (positive interference pattern) between the subject's extra-cranial blood flow and blood flow in the hands of the examiner that is of sufficient amplitude to breach the human pressure perception threshold¹³ then nothing will be felt by the examiner. The numerous variabilities associated with THM in two individuals would provide explanation for the absence of consistency, reliability and validity. This hypothesis remains to be tested, but an early indication¹² in which extra cranial blood flow is measured at the ear lobe with laser doppler flowmetry lends credibility.

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Viewpoint

McGrath MC.

This review¹ echoes the position that there is no scientific justification for osteopathy in the cranial field (OCF), a position which has been previously well described in the literature.^{2, 3, 4, 5, 6, 7} An alternative theoretical model is proposed. This proposed model theorises that cranial bone movement is the result of "rhythmic changes in arterial vasomotion, leading to blood pressure fluctuations within the cranium." It is proposed that arterial pressure changes due to cardiac systole and vasomotor activity move the brain and cause CSF to flow. This, coupled with evidence of slight sutural movement and cranial bone compliance, is presented as offering the potential for a more likely theoretical basis. New theoretical model notwithstanding, there remains an absence of demonstrated validity in OCF theory and clinical practice that reaches well beyond merely tinkering with a theoretical model.

It is stated that there is evidence for compliance and movement of the cranial bones.¹ It is however, biologically implausible to suggest a link between this and the explanation postulated by the new model. That slight cranial bone movement occurs under certain conditions is undeniable: it is the conditions by which such movement occurs that is the critical point. Cohen and MacLean⁸ give a detailed description of cranial sutural biology. Cranial sutures interlock to prevent the calvarial bones from separating due to external forces. With advancing age, the sutures become increasingly bound together by bony spicule formation and eventually, though not necessarily, close completely. A particular feature of this process is the formation of bony spicules (bridges) within the suture between the two adjacent bones. Spicules may bridge the

suture either completely or partially along its entire length. Alternatively, irregular, acellular calcified masses appear singly or as coalesced aggregates within the sutural gap, providing attachment to bone spicules bridging the sutural gap. Eventual bony fusion may occur endo- or ectocranially. A lack of sutural movement is a desirable characteristic between the cranial bones, whether or not complete sutural closure occurs. The adequate protection of the CNS is at stake. Any sutural movement or bony compliance that may occur, does so only in association with considerable externally applied loads. In a study by Losken et al.,⁹ an applied tensile load of 50kg produced a sutural displacement of 1mm in rabbits with delayed onset cranial synostosis, equivalent to human sutural closure in a 20 - 30 year old. Normal rabbit sutures, regarded as equivalent to human children, require a 15kg tensile loading to produce a sutural displacement of 1mm. These forces are many orders of magnitude greater than those advocated in OCF, where 5g manual pressure is said to be necessary to produce a movement of 50microns.¹⁰ Additionally, knowing that the calvarial bones must move at a rate and amplitude sufficient to breach the threshold of the examiners human tactile perception, it is straightforward to estimate the necessary intracranial pressure required to cause the alleged cranial bone motion, which turns out to be several orders of magnitude greater than the established values of intracranial or CSF pressure. Such a value is not only biologically implausible but it would be quite incompatible with life.

So what is it that the practitioner of OCF feels? I suggest that there is evidence to show that cranial rhythmic impulse (CRI) is a manifestation of an extra-cranial blood flow phenomenon and an observational artifact of no clinical significance. The cranial literature states that it is difficult

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to palpate CRI^{11,p.905} and indeed many cannot. Absence of reliability and validity are consistently associated with attempts to detect the CRI^{2,4} and this may be because the CRI, as an inherent biological entity, does not exist. The observed inconsistency of this finding may lie in the previous explanation that the CRI phenomena is an artefactual extra-cranial phase interaction of Traube-Hering-Mayer (THM) oscillations between the examiner and the subject.¹² Unless there is an extra-cranial THM phase coincidence (positive interference pattern) between the subject's extra-cranial blood flow and blood flow in the hands of the examiner that is of sufficient amplitude to breach the human pressure perception threshold¹³ then nothing will be felt by the examiner. The numerous variabilities associated with THM in two individuals would provide explanation for the absence of consistency, reliability and validity. This hypothesis remains to be tested, but an early indication¹² in which extra cranial blood flow is measured at the ear lobe with laser doppler flowmetry lends credibility.

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Viewpoint

Chaitow L.

This review of the mechanisms associated with cranial mobility is comprehensive and illuminating, although there remain a few areas where amplification and reinterpretation of the evidence may be useful.¹

By systematically evaluating the participation of different tissues and processes Ferguson has offered a clearer view of what is possible and probable, when the cranial hypothesis is set alongside current physiological knowledge.

What emerges is the strong likelihood that, rather than there being a 'primary' process that 'drives' cranial motion, palpable accommodating degrees of sutural motion occur, in response to rhythmic fluid movements, involving a synthesis of physiological pulsations (e.g. the Traube-Hering-Mayer oscillations, representing blood pressure, heart rate, cardiac contractility, pulmonary blood flow, cerebral blood flow and movement of CSF).¹

The amount of cranial sutural motion is minute, but it is both palpable and has been measured. Utilising infrared markers and a kinematic system Lewandoski et al.² demonstrated a rhythmic degree of motion at the saggital suture of approximately 250 microns (approximately 100th of an inch).

The suggestion is that a harmonic of multiple pulsations, interacting with sutural accommodation, allows an apparently significant degree of palpable motion to occur, involving the bones of the skull and associated fascial structures. Therefore, rather than being called a cranial rhythmic impulse (CRI), the palpated motion could more accurately be renamed the cranial rhythmic response (CRR). And if respiration together with multiple fluid pulsations

are the combined driving force of cranial motion, then this is the true *primary respiratory mechanism*, and palpable cranial sensations, previously credited as such (e.g. cerebrospinal fluid fluctuations), should be seen as associated features, rather than as being 'primary'.

Additionally, by challenging the concept of synchronous cranial and sacral motion, Ferguson has taken us closer to reality, and away from physiologically improbable linkages.

What remains to be satisfactorily demonstrated is whether restrictions in cranial motion negatively influence health, and how cranial treatment might normalise such changes. Physiotherapists it appears have already begun to systematically undertake this task.³

It seems to this observer that the sheer volume of clinical experience associated with cranial manipulation (of whatever variety) supports the usefulness of these methods, even if the explanations for their efficacy remain unclear.

It is suggested that cranial practitioners give renewed attention to a major feature of the real primary respiratory mechanism, breathing, and to the demonstrable structural, functional and biochemical impact on cranial function and dysfunction deriving from habitual breathing pattern disorders (BPDs) -of which hyperventilation is an extreme. On a structural level an altered breathing pattern, such as upper chest breathing (as opposed to diaphragmatic respiration) inevitably alters accessory respiratory muscle (and fascial) function and structure.⁵ Many of these muscles attach to the cranium, some capable of applying enormously powerful loads that cross sutures (sternomastoid, upper trapezius). How much this inhibits normal sutural flexibility remains to be established, but it is clear that attempting to 'release' a sutural restriction that is affected by such muscular loads, without prior and appropriate attention to the muscular status, is likely to be relatively ineffective.^{6,4}

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Respiratory alkalinity effect on smooth muscle

The result of an increased ventilation rate (as occurs with BPDs e.g. upper chest breathing) during which the rate of CO₂ exhalation exceeds the rate of its accumulation in the tissues, is respiratory alkalosis, characterised by a decrease in CO₂ (and therefore of carbonic acid) and an increase in pH.

Lum⁷ notes that, ‘Cerebral vascular constriction, a primary response to hyperventilation, can reduce oxygen available to the brain by about *one-half*. Significant amounts of CO₂ can be lost in a few minutes of over-breathing, immediately causing respiratory alkalosis. Compensation, by excretion of bicarbonate, is relatively slow and may take hours or days.’ Goldstein⁸ concurs, stating that reduced cerebrovascular flow (rCBF) is the result of “*dysregulation of the vascular response to hyperventilation*”.

Respiratory alkalosis induces vascular constriction, decreases blood flow, and is commonly accompanied by reduced oxygen transfer from haemoglobin to tissue cells, due to the Bohr effect.⁹ The Bohr effect states that an increase in alkalinity (decrease in CO₂) increases the affinity of haemoglobin for O₂. Note: the lungs are more alkaline than the rest of the body - enhancing O₂ uptake. The haemoglobin carrier molecule is therefore less likely to release its oxygen in an alkaline environment.^{10,11}

Lum¹² describes resulting symptoms: “cortical inhibition, emotional instability, generalized body tension and chronic inability to relax, proneness to tetany (spasm) in muscles involved in ‘attack posture’ - they hunch their shoulders, thrust head and neck forward, scowl and clench their teeth”.

The potential effect of smooth muscle contraction on the rate of vasomotion (and therefore the rate of the CRI) is made clear by Ferguson’s discussion of the changes in flow rate when blood vessel calibre reduces. However there are further cranial implications because of the presence, in fascia, of large numbers of smooth-muscle cells.

Staubesand and Li^{13,14} studied fascia in humans with electron photomicroscopy and found smooth muscle cells widely embedded within the collagen fibres. They describe a rich intrafascial supply of capillaries, autonomic nerves and sensory nerve endings, and concluded that these smooth muscle cells enable the autonomic nervous system to regulate a fascial pre-tension, independently of muscular tonus.

They suggest that this understanding of fascia, as an actively adapting organ, may have far reaching clinical implications. Schleip¹⁵ notes that elevated pH, resulting from over-breathing, produces smooth muscle contraction and even spasm in fascial tissues so altering general fascial tone. The circulatory (and therefore cranial) implications of this are obvious.¹⁶

In summary, Ferguson has described, in some detail, the possible influence of the rate of arterial (and to a much lesser degree, venous) motion on cranial rhythms, and states that the rate of vasomotion increases in smaller calibre

vessels, as well as where systemic hypoxia is a feature.¹⁷ Narrowing of blood vessel calibre (for example as a result of smooth muscle contraction – as described above), as well as relative hypoxia, occurs automatically and rapidly, as a sequel to a disordered breathing pattern. It is therefore important that breathing patterns be evaluated, and if possible rehabilitated when dysfunctional, as a precursor to attempting to interpret or normalize cranial rhythms.

The motions and functional influences of the structures of the face are less controversial, compared to those at the cranial sutures. Recent animal evidence, as discussed in the paper^{18,19} points to CSF drainage via the cribriform plate. This adds to the importance we need to give to the bones and soft tissues of the face. The cribriform plate, lying as it does immediately anterior to an anchorage point for the falx cerebri, is clearly vulnerable to increases in local cranial fascial tension and behaviour. Since fascia also forms the venous sinuses, via a folding of the dura, increases in fascial tension via respiratory alkalosis is of major potential significance. Can cranial treatment modify fascial structures such as the falx? Studies in 1992 by Kostopoulos and Keramides²⁰ measured the forces required to lengthen the falx-cerebri when anteriorly directed traction was applied to the frontal bone of an extremely fresh cadaver. Elastic response began at 140 grams and ended when viscous changes were noted at 642 grams traction on frontal bone. Falx cerebri lengthened by 1.097 mm with 642 grams traction. 140 grams – enough force to begin falx cerebri elongation – is equivalent to just over 4 ounces, an amount of pressure/effort commonly applied in cranial interventions.

How widespread are breathing pattern disorders?

Chronic hyperventilation can present with a myriad of respiratory, cardiac, neurologic, or GI symptoms, without any clinically apparent over breathing by the patient. In the US as many as 10% of patients in general internal medicine practices are reported to have HVS as their primary diagnosis.^{7,21}

The number who are progressing toward that status is demonstrably far higher, suggesting that features such as those described above are active in a large proportion of the general public, symptomatic or asymptomatic, with an unknown degree of influence on cranial function.⁶

Final thought – pray

Recent research has shown that the effects of rosary prayer (‘Ave Maria’ in Latin) or recitation of a yoga mantra, results in a slowing of respiration to approximately 6cpm, along with synchronization of the Traube-Hering-Meyer oscillations. This influence on autonomic activity, represented by THM oscillations, may therefore be seen as having a profound influence on the so-called cranial rhythmic impulse (CRI).²²

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