New hypothesis for the origin of cranio-sacral motion

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Abstract A new hypothesis for the origin of Cranio-Sacral Motion (CSM), has been formulated. Presently accepted concepts are called in to question based on biomechanical studies of cerebro-spinal fluid pulsation waves, neural envelopes and studies of those physiologic factors which influence vessel wall tonus. The difference between the rhythm of venomotion and the Cerebro-Spinal Fluid (CSF) is explained and a new hypothesis is put forwards. It is suggested that CSM is probably the expression of Local Venomotion (LVM) and not of CSF pulsations or the consequences of it. The function of 'rigidity' of the skull sutures is discussed, and finally, a suggestion is made for osteopathic normalization of skull bone restrictions, using the patient's respiration as a powerfull and well controlled means of treatment.

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Introduction

In the early 20th century, WG Sutherland (1939) identified a rhythmic cranial motion, of obscure origin, with a rate of between 6 and 12 cycles per minute (c.p.m.). He named this motion the 'primary respiratory mechanism' and suggested that rhythmical brain movement might be responsible for cerebral ventricular and CSF fluctuations. Other researchers (Magoun 1966, Upledger 1981) proposed that this brain motility, or 'Cranial Rhythmic Impulse',

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originated from the ventricular pumping actions. An opinion concerning CRI, suggesting an entrainment model, has recently been put forward (McPartland 1998). Most manual therapists and osteopaths, employing cranial techniques, who sense the physiological motions at the exocranium, have an interest in knowing what it is that they are palpating, and what lies behind the so called 'cranio-sacral-motion' (Lippincott 1949, Magoun 1966, Frymann 1971, Retzlaff et al. 1975, Mitchell 1976, Roppel et al. 1978, Tettambel et al. 1978, Upledger 1981, De Cock 1982, Altieri 1984, De Cock 1988, Ferguson 1991, Ferre et al. 1991, Norton 1991, Rommeveaux 1993, Chiatow 1997, McPartland & Mein 1997).

Cranio sacral motion and cerebrospinal fluid pulsation

Since many cranial osteopaths support the hypotheses that the origin of CSM is to be found in brain movement, or CSF motions, it is necessary to search what is known about CSF flow pulsation amplitudes in assessment of exocranial dynamics.

Magendie (1842) found that the dynamics of CSF in normal individuals has three characteristics:

- The existence of a slightly positive pressure, in comparison with the atmospheric pressure.
- The behaviour of rapid pulsations, cyclic and synchronous with the arterial pulse.
- There is also evidence of slower waves following the respiratory cycles.

Most authors conclude that the configuration of CSF pulsations are recognized as essentially arterial in nature (O'Connell 1943, Goldenshon et al. 1951). In the sixties, Bering pointed out that CSF, pulse pressure contributes to the intracranial pressure and that arterial pulsations occuring at the choroid plexus were transferred to the cerebral ventricles. Later investigators, (Du Boulay 1966) showed by means of pneumo-encephalography, that CSF propulsion by the choroid plexus pulsations was minimal.

Movements of the third ventricle, caused by the pulsations of the basal arteries, were noted to be of greater value. Those pulsations are probably partly the result of rhythmic fluid displacements of the expanding brain tissues occuring at systole (ter Braak & de Vlieger 1962, Cserr 1971, Martins et al. 1972, Wood 1983, Herndon & Brumback 1989, Schuller 1993).

Some investigators (Hamit 1965) believed the pulsations of CSF depend on venous hydrodynamics. In the case of congestive heart-failure, the pattern of intracranial CSF pulse wave is indeed purely venous, but under physiological conditions the intracranial CSF pulse wave is not primarily venous but arterial in origin (Martins et al. 1972, Hamer 1977, Thomsen et al. 1990, Bhadelia et al. 1997, Ursino & Lodi 1998).

The intracranial dynamics can be viewed as an interplay between the spatial requirements of four main components: arterial blood, capillary blood (brain volume), venous blood and CSF. These components could be characterized, and the expansion of the arteries and the brain differentiated, by applying the Monro-Kellie doctrine to every moment of the cardiac cycle (see Box 1). The arterial expansion creates the prerequisites for the expansion of the brain by venting CSF to the spinal canal. The expansion of the brain is, in turn, responsible for compression of the ventricular system and hence for the intraventricular flow of CSF (Greitz et al. 1992).

Hypothesis of vasomotion

The so-called 'cranial sacral motion', is possibly either an expression of a general venous vessel wall pattern involving the vessels of the brain and those on sacral level, cyclically contracting at a rate of 6 to 12 c.p.m. (Farasyn 1986b, 1988, 1989).

It is necessary make a clear distinction between something that causes rhythmic pulsation waves in CSF, and consequent passive brain squeezing by each systole, and the automatic rhythmic venomotions of the veins of the head. Norton's theory (1991) assumes that the sensation described as the cranial rhythmic impulse, is related to activation of slowly adapting cutaneous mechanoreceptors by tissue pressures of both the examiner and the subject. The sources of change in these pressures represents a complex interaction of at least four different physiologic rhythms, i.e. the combined respiratory and cardiovascular rhythms of both examiner and subject.

The author wishes to explain the difference between venous cardiovascular rhythmic pattern of the subject and examiner, as suggested by Norton, and palpation of something totally different: venous vasomotion inside the skull. If CSF pulsations follow a physiological venous pattern, as is the case in some normal individuals, it suggests that the venous pattern is caused by cardiac contractions, and not by venous vessel wall contractions. In other words, when palpating the exocranium, the so-called 'cranial sacral motion' may be felt as an indirect expression of the characteristics of the venous vessel walls, at a rate of approximately 10

Box 1 The Monro-Kellie doctrine: CSF and intracranial pressure

The changes in intracranial pressure which occur following a change in one of the constituent volumes within the skull are governed by the Monro-Kellie doctrine, stated in the late 18th century which describes how an increase in one of the constituent volumes must be reflected by a reciprocal decrease in another volume to avoid any change in pressure and that if this does not occur, there is a rapid rise in intracranial pressure. The development of cerebral dysfunction under some experimental conditions is independent of the rate of expansion and only dependent upon a critical volume. This constancy of intracranial volume is the mean sense of the so-called Monroe-Kellie doctrine.

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c.p.m., and not the CSF pulsations themselves.

Palpation of the great or small veins on the body demonstrate these same active characteristics of venous smooth muscle contractions, at a rate of approximately 10 c.p.m.

The physiological behaviour of venous, venular and even arteriolar vessel walls, require examination in relation to the concept of the cranial-sacral motion.

Arguments

In 1852, Jones described for the first time myogenic activity of blood vessels by examination of periodic contractions of veins in the bat wing. In the beginning of the 20th century, Bayliss proved that this behaviour is also common for arterial vessels and made a theoretical explanation for it. In 1961 Funaki recorded, for the first

time, with intracellular micro-electrode techniques, spike-discharges from venular smooth muscle fibre. The sum of trans-membrane potentials of smooth muscle fibre caused bursts of regular spike discharge, explaining the rhythmic and spontaneous 'pace-maker' vascular activity.

Wiedeman (1963) demonstrated that injecting small volumes of physical solutions prolonged the duration of the contracting phase of venomotion. D'Argosa (1970) and Wiederhielm (1973) obtained, with micropunctures in the vascular bed of bat wings, measurements of approximately 9.5 c.p.m.

Those vigorous contractions in muscular venules and veins in the bat wing are also present in other species too (Holman et al. 1968, Wiederhielm & Weston 1973, Morgan 1983, Gustafsson et al. 1994). Contractions of the smooth

muscles of the venous wall cause active displacement of blood towards the heart, and, vasomotion contributes to the maintenance of blood fluidity in the microvessels (D'Argosa 1970, Bevan et al. 1974, Muller-Schweinitzer & Sturmer 1974, Vanhoutte 1977, Siegel et al. 1980, Colantuoni et al. 1984, Folkow 1984, Burnstock et al. 1986, Aggarwall et al. 1994, Davis et al. 1992, Gokina et al. 1996, Yuan et al. 1998). The vessel walls of venules and veins are sensitive to: temperature-changes, extramural pressure (Mellander et al. 1964) and distension (Johanson & Mellander 1975), but some local metabolic factors affect the reactivity of venous smooth muscle, in contrast with the characteristics of the arteriolar one (Bayliss 1902, Nicoll & Webb 1946, Mellander et al. 1964, Wiederhielm 1967, Muller-Schweinitzer & Sturmer 1974, Edwinsson et al. 1983, Colantoni et al. 1984, Vanhoutte & Rimele 1984, Davis et al. 1992, Gustafsson et al. 1994; Aggarwal et al. 1994; Gokina et al. 1996).

Pathological situations such as advanced diabetes mellitus (Bollinger et al. 1983), glucose concentrations (Linke & Betz 1979, Morgan 1983), oxygen and carbondioxide concentrations sympathoadrenergic activity (Bevan et al. 1974, Auer et al. 1981, Edvinsson et al. 1982) and some specific pharmacological agents (Altura 1978) employed in neurology, anaesthesia and angiology (Thulesius et al. 1979, Xiu 1983, Bumstock & Kennedy 1986, Xiu & Intaglietta 1986) all directly influence the vessel wall tonus and vasomotion.

Arterial vasomotion can play an important role in determining oxygen distribution (Colantuoni et al. 1984, Folkow 1984, Ragan et al. 1988). Laser-Doppler flowmetry and electromyography, used simultaneously for measuring

skeletal blood perfusion, show vasomotion, i.e. rhythmic variations in blood flow with a frequency of 5–6 c.p.m.-l (Larsson et al. 1993).

For many practitioners it is hard to accept the concept of 'cranio-sacral motion' being noted simultaneously via contraction waves of CSF, at both occipital and sacral level (Farasyn 1986b, Ferre et al. 1991). Biomechanical studies of neurological envelope-displacement demonstrates elongation and displacement only if strong flexion or extension of the vertebral column occurs (Trolard 1890, Reid 1960, Decker 1961, Kernig 1969, Martins 1972, Bourret & Louis 1974, Breig 1978, Louis 1981, Williams et al. 1989).

CSF-fluctuations, when passively induced, are of such minor value that it is difficult to accept that they are responsible for the stronger displacement-movement noted at the sacral level.

As confirmed by some authors (Bourret & Louis 1974, Decker 1961, Troup 1986) experiments demonstrated, even in a neutral spinal position, that the sheath of the dura-mater spinalis possesses pleats, suggesting that the tissues are relaxed.

Studies of Klein (1986), on cadavers, examining the biomechanical behaviour of the dura-mater spinalis and spinal cord, during flexion of the whole column, showed a downward slip in the upper cervical, an upward slip in the cervico dorsal and a downward movement in the lumbar area. There was no slipping in the area of C4 and T6. The author maintains that it is hard to believe the cranio sacral system could work as proposed in currently accepted models. One requirement for the mechanical expression of cranio-sacral motion would be sufficient tension of the spinal cord between sacrum and occiput, and this seems to be absent.

Little rhythmic (real) respiratory displacement can be palpated on the sacrum of an individual in neutral position. It is consequently difficult to palpate CSF-pulsations on the sacrum. It is suggested by the author that the relatively small degree of flexion–extension of the sacrum at a rate of $+/-$ 10 c.p.m., is probably caused by the venomotions of the vena cava inferior bifurcation situated just anterior to it. Local venomotion associated with sacral motion is not only independent of, but also relatively, stronger than occipital motion (Farasyn 1988).

Venomotion: independent contraction waves

Veins and venules exhibit marked active vasomotion that sweeps centripetally as a peristaltic wave with the segment of any vessel between two valves forming an automatic responsive unit (Nicoll & Webb 1946, 1955, D'Argosa 1970, Siegel et al. 1980, Ragan et al. 1988, Larsson et al. 1993). This means that veins coming from the head (without valves) and those coming from the limbs and trunk, demonstrate nearly the same frequency of vasoconstriction, but have independent cycles. Palpation for only a brief time leads to a

suggestion of the same frequency of pulsation at both occipital and sacral level.

On the other hand, if palpation continues for more than 5 minutes, the contraction wave which is noted is felt to occur independently at occipital and sacral levels. Just as it is possible to visualize venomotion influence at the sacral level, so can vasomotion of the superior sagittal sinus, the great cerebral vein, the straight sinus, the transverse sinus and their confluence, posterior and inferior in the head, be seen to influence motion in the cranium. It is possible, on the exocranium, to palpate these physiological pulsations independentally on the frontal and sagittal bones (Fig. 1).

The laws of hydrodynamics suggest that the force of venomotion, in structures lying just under the skull, should be able to move, albeit slowly and with very small displacement ranges, the different bones of the skull (Farasyn 1986b). The existence of a cavernous nodule at the confluence between the straight sinus and the great vein of Galen (Gray's Anatomy 1973) might possibly play a role in CSM as suggested in Upledger's pressurstat model (1981). The validity of this has been difficult to accept because no muscular or elastic tissue
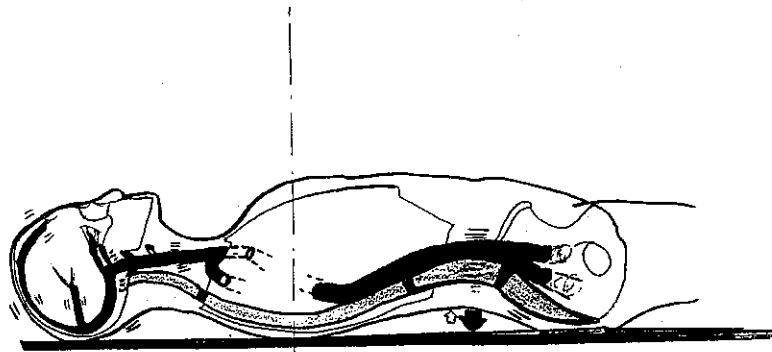

Fig.1 Vasomotric pulsations of Vena cava inferior and Vena iliaca communis the primary motor for rhythmic impulse at cranial and sacral level with an independent frequency of 6 to 10 c.p.m.

components are noted in this mechanism (Bergquist 1974). Research may demonstrate that venomotion plays an active role in this hypothesized mechanism. The morphological organization of cerebral veins, with a rich supply of collagenous material and only a few pericytes, supports the evidence of weak contractions, regularly observed in isolated vein preparations in vitro. These small contractions might, however, be of importance in vivo, since cerebral veins only have to constrict against a low pressure (Edvinsson et al. 1983).

Rigidity of the skull

It is important to note that the rigidity of the skull of an adult serves particular needs and functions. Just as negative pressure exists in the thorax, produced by its osseous cage, pleura and a film of liquid, so in the head there exists an osseous box, different soft tissue layers and films of liquid, between the periostium and the dura-mater, assuring a negative pressure in sinus areas of the head. Thus, a perfect venous return is guaranteed by the pressure gradient from a high (arterial) pressure to a low (venous) pressure system.
In support of this consider:

- Young people under the age of 11, do not yet have skull rigidity (Parsons 1905, Ericson & Myrberg 1973, Moss 1975), and there is no negative pressure in the jugular vein system at this age (Hamit et al. 1965, Edvinsson et al. 1983).
- After ossification of the skull bones at the age of $+/- 11$, negative pressure is always present (normally) in the jugular vein system.
- Trepanation – the greater the surface of skull bone involved in trepanation, the more the patient

complains of headaches, possibly due to malfunction of vessel tonus, resulting from decreased negative pressure (Fodstad et al. 1984).

The function of bone structures may be necessary for impact protection by means of local absorption of kinetic energy (Moss 1975, Dörheide & Hoyer 1984, Spetzler et al. 1980), and relative rigidity may be a need for a normal, good working venous return in the head. On the other hand, abnormal fixations of skull bones could hypothetically cause locally decreased venous blood circulation, leading to increased circulation elsewhere, in compensation.

Examination of patients

Suggested examination on a person lying supine

1. Put one hand supine under the occipital bone and with your index-finger of the other hand, successively on:

   - Facial vein;
   - External jugular vein;
   - Anterior jugular vein.

2. Put one hand under the occipital bone and with your other hand supine under L5-S1 level.
3. Put one hand under L5-S1 level and with the index-finger of the other hand on the great saphenous vein, or if possible on the common femoral vein. Observe :

   - The local force of venomotion
   - That the greater the segmental distance the more obvious the independent rhythm and the common frequency rate of 6 to 12 c.p.m. of venomotion
   - That the force of contraction is greater at sacral level than on occipital level as the sacral motion depends on the strong venomotion activity of the

vena cava inferior and vena iliaca communis
- That the pulsations at the occipital level are independent of those on the sacral level
- Cranial rhythmic pulsations are the strongest at the level of the mastoid process, caused by the passage of the vena jugularis interna (Fig. 2).

Try also to palpate 'ascending–descending' sensations of the liver or kidneys. Besides respiration and arterial pulsations, an apparently 'intrinsic' force of swelling is to be felt each $+/- 10$ seconds, possibly caused by the rhythm of portal vasomotion.

The concept of cranial rhythmic motion suggests that the rhythmic movements of $+/- 10$ c.p.m. of 'cranial bone mobility' is palpable all over the body. The concept proposed by the author, suggests that the primary motor is local venomotion (LVM).

It is possible that arterioles take part in the motion of CRM, but it is unknown to what degree this occurs. The author hypothesises that veins are responsible for palpated 'CRM' pulsations in the fascia, bodywide, including organs.

When palpating for 'CRM', by placing the hands, for example, on the legs of a patient, local venomotion may be what is being noted, rather than reflection of cranial motions.

Treatment for sutural rigidity

The 'local venomotion' under the cranial bones, as described above, is necessary for diagnosis of 'cranio sacral motion' and is commonly employed therapeutically in classical cranial osteopathic treatment. If respiratory forces produce pressure within the cranial bowl these can be employed in treating cranial restrictions.
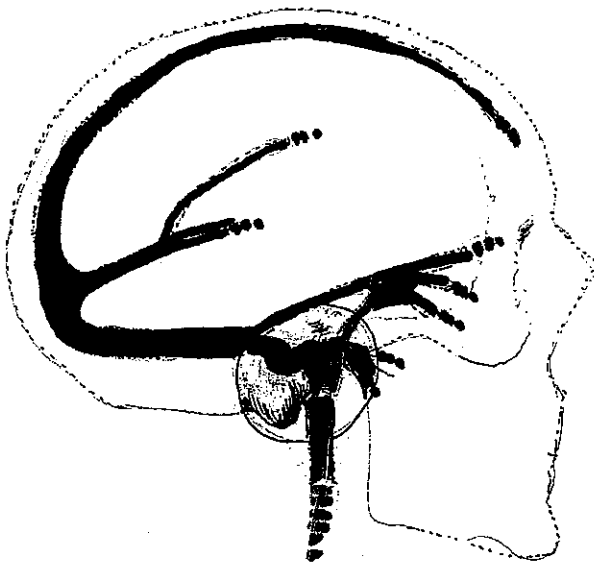
Fig. 2 Cranial rhythmic pulsations are the strongest at the level of the processus mastoideus caused by the passage of the Vena jugularis interna.

While for diagnosis of cranial bone mobility, the LVM is the best indicator of sutural restrictions of the skull, for treatment the force of respiration of the patient is most helpful. Asking the patient to inhale and exhale offers a greater degree of controllable force than the more subtle venous motion (Fig. 3).

Following correction, employing respiration tools, palpation of LVM of cranial bones is necessary to check whether treatment has been successful.

Discussion

The medical literature reveals experimental studies concerning human arteriolar vasomotion. It seems is that it is experimentally difficult to examine human venomotion. Flowmetry of venules, and veins in vivo, show direct artefacts of cardioarteriolar rhythmic contraction waves. To register venomotion alone, researchers need dissected preparations. The only direct venomotion measurements in vivo

which are currently possible, involve bat wings.

Recent literature of human evidence of arterial vasomotion:

- Experiments on segments of human basilar arteries with Caprate (C10) indicates that these lipids could influence directly vasomotion as vasodilators (White et al. 1991);
- Percutaneous measurement by Laser-Doppler flowmetry of human skeletal muscle microcirculation at varying levels of contraction force showed vasomotion, i.e. rhythmic variations in the blood flow, with a frequency of 5–6 c.p.m.-1 (Larson et al. 1993);
- Human pial arteries obtained during surgery frequently exhibit spontaneous periodic contractions. Fifty-three segments from 38 patients were studied and spontaneous depolarization reached levels of −40 to −35 mV. It was concluded that these periodic depolarization and action potentials underlie the periodic spontaneous contractions of human pial arteries (Gokina et al. 1996).

A theory can be constructed based on clinical evidence concerning the relation between vasomotion and CSM requiring proof of:

- The existence of human CRI
- The existence of human venomotion in vivo
- The correlation between rhythmic venomotion and rhythmic cranio sacral motion.

Conclusion

It is proposed that the intrinsic movement of cranial bones, fascia and organs may be caused by local venomotion pulsation, the reflection of which we may palpate at the surface.
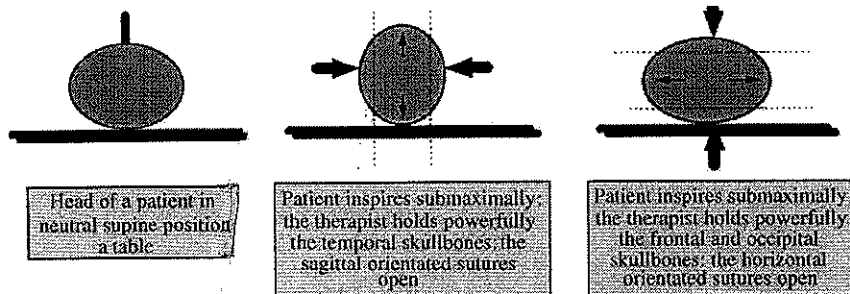

Head of a patient in neutral supine position a table

Patient inspires submaximally the therapist holds powerfully the temporal skullbones: the sagittal orientated sutures open

Patient inspires submaximally the therapist holds powerfully the frontal and occipital skullbones; the horizontal orientated sutures open

Fig. 3 Treatment for sutural rigidity.

Arterial and venous vasomotoricity is to be found throughout the body, including the head where local venomotion is probably the prime motor of cranio-sacral motion.

It remains to be demonstrated conclusively that factors such as temperature (fever), transmural-pressure (hydrocephalus), glucose concentration (diabetic patients) are able to change the patterns of venomotion and, therefore, of CSM. The question is not the belief or otherwise of CSM, which exists as a measurable physiological phenomenon. The problem is to explain the origin of it and this paper has offered a possible answer.

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