



CRANIAL MECHANISM HYPOTHESIS

An investigation into the regulation of intra-cranial pressure and its influence upon the surrounding cranial bones

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KEYWORDS

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Summary The aim of this study is to present a rational coherent hypothesis to explain the palpable involuntary movements of the cranium. The arterial and venous anatomy inside and around the skull and spinal column presents a complete vascular system with the capacity to regulate intra-cranial pressure to a level of equilibrium slightly higher than atmospheric pressure. Variations in cerebrospinal fluid (csf) pressure control the volume of blood draining through the cavernous sinus and hence into the inter-vertebral venous plexus in relation to the jugular vein. Stable intra-cranial pressure is maintained by a controlled release of venous blood through the inter-vertebral venous plexus (slow) and the jugular vein (fast) in the cavernous sinus. Any distortion of the skull from its healthy state will lead to reduced intra-cranial volume. The process of release from the state of compression has been interpreted as “cranial rhythm” but may be a mechanical adjustment increasing the internal volume of the skull, aided by the continual maintenance of stable intracranial pressure. This involuntary movement is capable of being assisted manually.

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Introduction

This paper aims to describe a new hypothesis about the mechanisms underlying the involuntary mechanism on which cranial osteopathic treatment is

based, and to suggest ways in which the hypothesis could be tested.

Current theories about the involuntary mechanism

Sutherland (1939) in his original hypothesis regarding involuntary movement of the skull, along with ideas by Upledger and Vredevoogd (1983) suggested

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bony movement generated by a rhythmic movement of the central nervous system. Williams (1995) in *Gray's Anatomy* suggested that no structures within or surrounding the nervous tissue are strong enough to generate or withstand this movement. Furthermore, the delicate nervous tissue is unlikely to move bony tissue and distort the skull without fatal trauma.

Green et al. (1999) concluded that cranial treatment produced therapeutic improvement but offered little scientific supporting evidence of any anatomical model to generate the movement. Maddick and Korth (2006) supported this view.

Ferguson (2003) presented significant evidence to suggest a vascular origin to the involuntary cranial movement but did not offer a complete anatomically based model to underpin his research. The purpose of this paper is to suggest such a model.

Recent research on fluid dynamics in the brain

The brain is the most important and best-protected organ in the body. It is the only organ completely enclosed by bone with generous blood supply passing in and out of the skull (Williams, 1995). It seems widely accepted that the pressure inside the skull is kept to an equilibrium level but no satisfactory mechanism has been proposed as to how this pressure is maintained.

Schaller (2004) highlighted the importance of the cerebrovenous system as a whole and Han and Backous (2005) presented evidence suggesting a control mechanism based upon blood flow but did not detail any anatomical structures to support this research. A further purpose of this paper is to offer supporting anatomy for this research.

Structures within the cranial circulation with no defined function

Primary osteopathic principles observe the importance of blood flow and the close inter-relationship of the systems of the body as a whole (Still, 1910). The first objective of this paper is to examine the complex unexplained structures within the venous system of the skull and spinal column (The cavernous sinuses, basilar plexus, internal and external vertebral venous plexuses, including the vessels interconnecting the two- the intervertebral and basivertebral veins), and present a model for regulation of intracranial pressure based on documented anatomy primarily from Williams (1995) and simple hydraulic mechanisms.

The first of these complex structures with unexplained anatomy are the two cavernous sinuses, located on each side of the sphenoid bone (Fig. 1). These are part of the venous drainage system of the cranium. Most of the venous outflow from the capillary system of the brain passes through a capacious valveless, free-flowing sinus network (Fig. 2) leading to the large, free flowing jugular veins. Conversely, the design and position of the cavernous sinus restricts blood flow. The cavernous sinuses are different from all the other venous sinuses [open blood filled spaces] in that they present a spongy structure. Furthermore, the sinus is heavily encroached upon by arachnoid granulations in the walls (Fig. 3), so possessing the capacity to restrict blood flow rather than promote it. Butler (1957), Pernkopf (1963) and Parkinson (1973) concluded that it was in fact a venous plexus, draining into the basilar plexus and the transverse sinus via the superior petrosal sinus, and into the internal jugular vein. The two cavernous sinuses communicate with each other above via the anterior and posterior intercavernous sinuses. Again the flow is valveless and reversible.

The second unexplained structure is the basilar plexus (Fig. 4) which wraps around the brain stem and drains into the internal vertebral venous plexus (ivvp). This ivvp consists of four longitudinal vessels running the length of the spinal column, and horizontally interconnecting vessels at the level of each vertebra (Figs. 5 and 6). The ivvp drains into the external vertebral venous plexus (evvp) as it loops the vertebrae, both anterior and posterior, which in turn drains into the general venous circulation via the vertebral, posterior intercostals and lumbar veins. Hence, these structures present a perforated slow draining tubular cuff of veins within the same space that contains the central nervous system, the meninges and all related circulating fluids. The ivvp and evvp are linked by the intervertebral veins through the inter-vertebral foramen, and the basivertebral veins through the vertebral bodies. The intervertebral and basivertebral veins are the only places where the flow of venous blood in this system is not free flowing and reversible due to partly formed valves which allow for controlled flow in one direction and free flow in the other (Williams, 1995). Experiments conducted by Batson (1957) show the venous blood supply through these vessels can be reversed, with greater volume of blood passing back into the intra-spinal space per second than is allowed to flow out in the normal direction. Although the valves have been observed, no likely function has been assigned to them. The valves located at all the exit points from the intra-cranial and spinal space create a slow

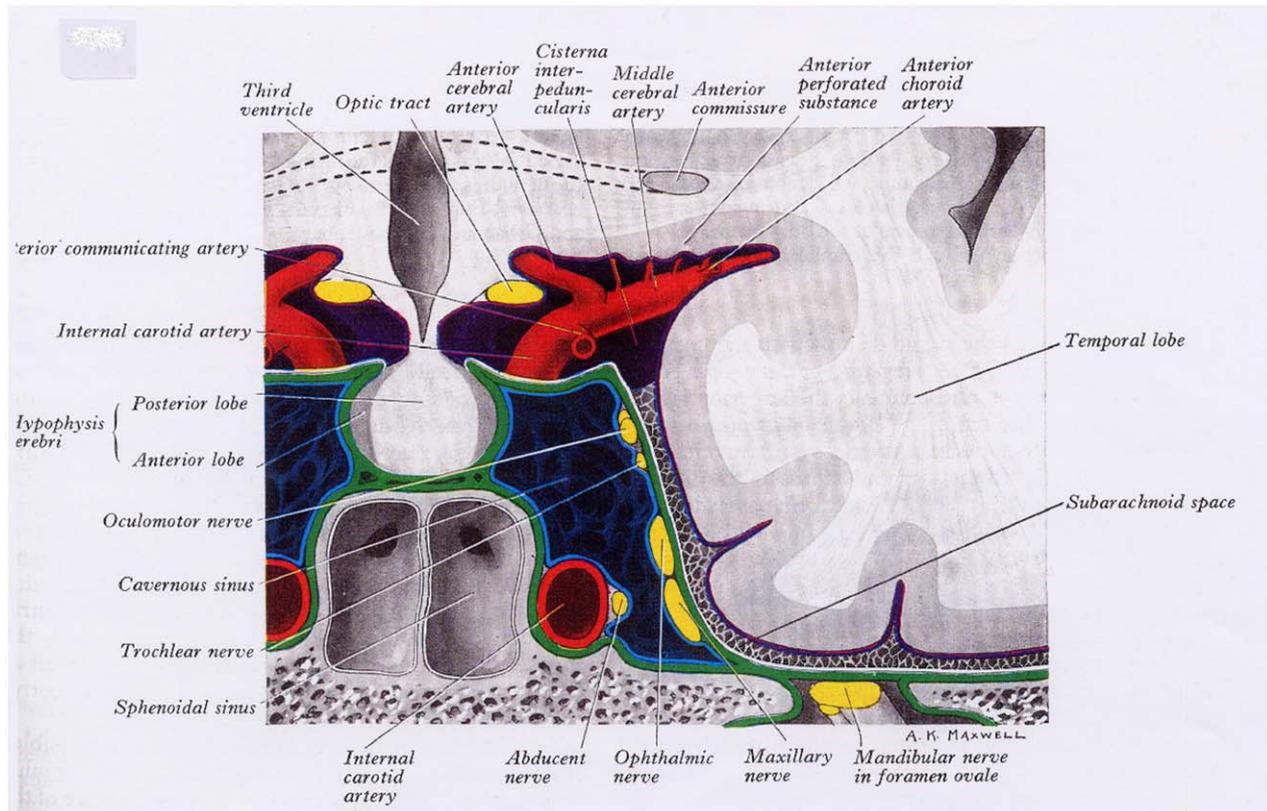


Fig. 1 A coronal section through the middle cranial fossa showing the cavernous sinuses and their surrounding structures.

draining cuff which is unable to be shut off, that allows for rapid refilling of the internal venous plexus depending upon a relative pressure decrease inside the vertebral space. There would seem to be no local control over these valves, suggesting the movement of blood is reactive and passive.

A further structure identified but with no explained function is a small protrusion, containing csf and resembling an arachnoid granulation, which projects into the floor of the straight sinus at the junction with the great cerebral vein. Williams (1995) suggests that this is, in fact, a ball valve whose purpose is to regulate venous blood flow to the sites of csf production, dependant upon the pressure of csf itself.

The new hypothesis

The hypothesis makes certain assumptions: (1) that the cavernous sinuses control venous outflow through the spinal venous network, (2) the ivvp/evvp system allows for a controlled release of venous blood out of the internal spinal/skull vault but can facilitate a dramatic reversible flow of

blood back into the sealed system when there is a sharp drop in intracranial pressure, and (3) a ball valve system operates in the great cerebral vein and the cavernous sinus where the csf filled arachnoid granulations swell to reduce venous blood flow.

Fig. 7 represents a diagrammatic model for reference to support the following explanation.

A suitable starting point will be when the venous blood flow through the cavernous sinus is at its maximum. Large volumes of blood enter the ivvp, the cuff surrounding the spinal cord. The valves in the veins exiting the ivvp restrict the exiting blood to the extent that the ivvp swells as more blood is entering the cuff than is able to leave. In normal posture, like any vessel, it fills from the bottom upwards so the csf is forced upwards as the arachnoid space is pressurised.

Eventually, the pressure wave reaches the level of the skull, where the raised intracranial pressure directly affects the brain. The increased csf pressure backs up to the base of the brain and swell the arachnoid granulations in the cavernous sinus. This swelling reduces the venous flow through the sinus, redirecting the blood to the internal jugular vein and safely out of the skull.

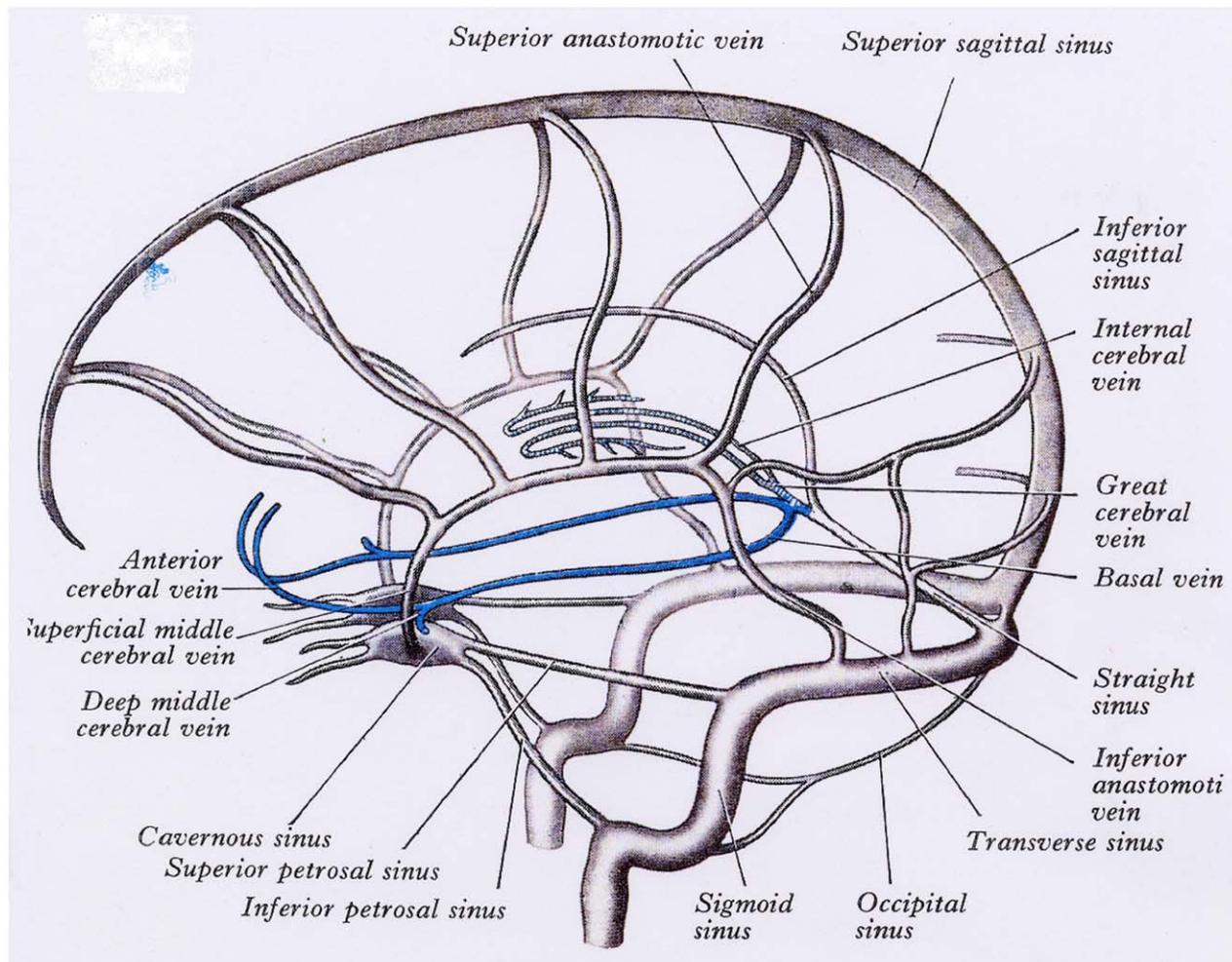


Fig. 2 The venous sinuses of the dura mater and their connections with the cerebral veins.

(Pressure also engorges the granulation at the level of the great cerebral vein and straight sinus, reducing blood flow to the site of csf filtration). The greater csf pressure in the arachnoid granulations around the superior sagittal sinus then promotes re-absorption back into the venous bloodstream.

Reduced blood flow to the ivvp allows it to drain faster than it can be refilled so the blood volume shrinks. The resulting drop in local pressure allows the csf to drain down the cord, relieving pressure on the arachnoid granulations in the cavernous sinus, therefore returning to the start of the cycle.

The cavernous sinuses are positioned below the tentorium with the csf flowing below it and down the spinal column before leaking through the perforations of the tentorium and surrounding the forebrain above it.

This anatomical feature suggests that the switch controlling the blood flow and the drainage mechanism it controls are partially separated from the brain above the tentorium. The superior

compartment will not be subject to the variation in pressure experienced below the tentorium as its csf system is only connected to the csf below by small slow draining apertures.

The proposed mechanism offers a physical mechanism to explain the findings in previous research by Schaller and Graf (2005) concerning the differing pressures inside the cranial compartments.

Effects on cranial movement

The effect of hydraulic auto-regulation is a tendency for expansion of the skull due to it being a fluid filled vessel. This gentle passive involuntary pressure causes the skull to adopt a shape concurrent with the maximum possible internal volume of the cranial vault, i.e. a sphere.

The skull, with no direct stresses acting upon it, is as dilated as its anatomy allows due to the internal pressure of its contents pushing the

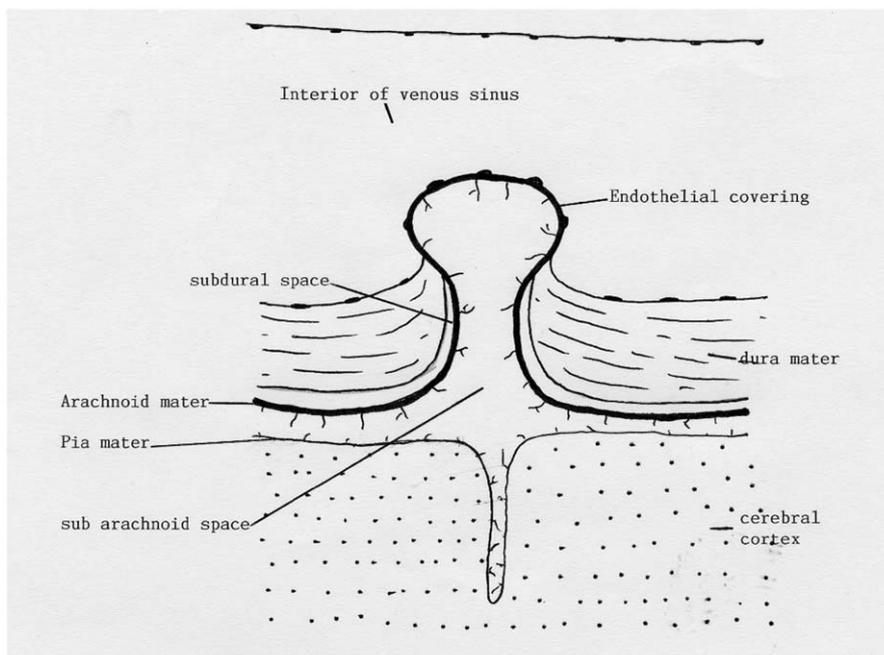


Fig. 3 A diagram illustrating an arachnoid granulation.

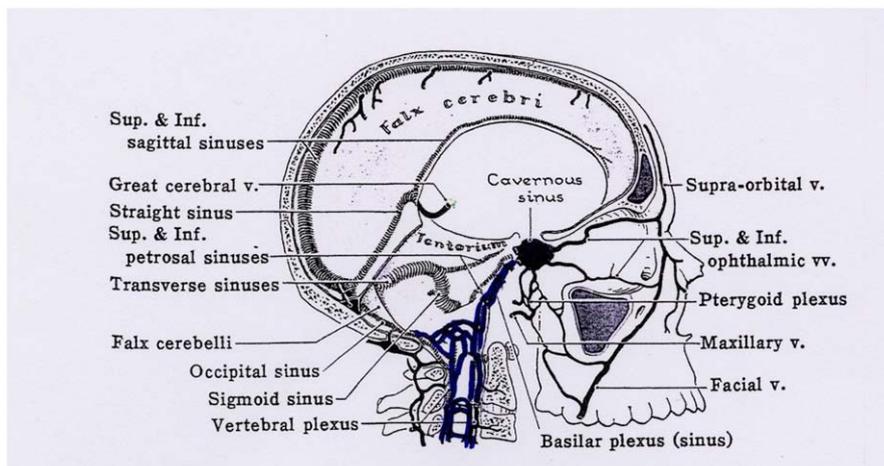


Fig. 4 A diagram illustrating the basilar plexus.

container outwards. As the relaxed skull is held at its maximum internal volume, any stress or torsion applied to the skull, leading it to be distorted will lead to the internal skull volume being reduced. With less internal space, the available volume of fluids (csf and blood) inside the fixed space within the skull and spinal column would be reduced, as the volume of the soft tissues inside is fixed. The slow production and re-absorption of csf means its volume can only vary at a slow pace while blood volume can vary rapidly so a reduction in blood volume inside the skull is the first consequence of torsion in the skull. The reverse is also the case. Improved mechanics of

the skull will primarily benefit blood flow through the brain.

If the bones of the skull are under no state of tension, they have been expanded to their maximum volume so there would be no potential for the involuntary movement of expansion.

If, however, there has been a traumatic event creating torsion within the skull and its internal volume has been decreased, the outward pressure generated by the internal contents of the skull will create a latent desire for the bones of the skull to realign to their greatest level of expansion. This natural process of inflation will be prevented if the skull or articulating vertebrae are traumatised with

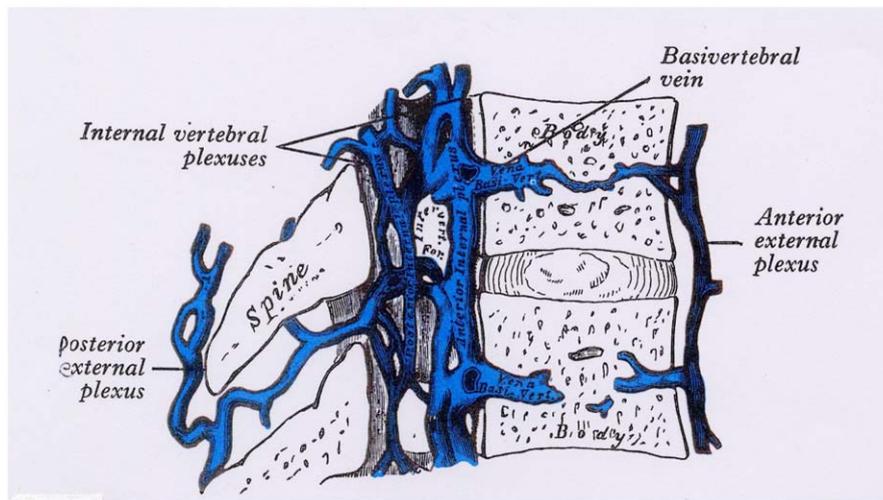


Fig. 5 A median saggital section through two vertebrae showing vertebral venous plexuses and basivertebral veins.

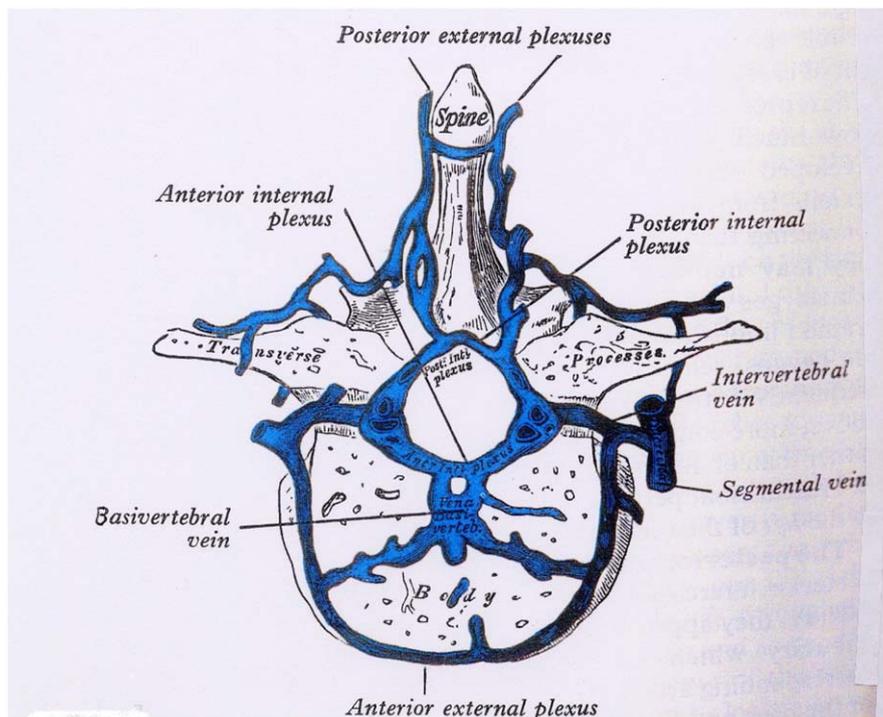


Fig. 6 A transverse section through the body of a vertebra showing the vertebral venous plexuses and basivertebral veins.

greater force than the tendency for expansion can overcome. Treating the cause of restriction and improving the mechanics of the skull in such a way that encourages its expansion aids this process.

The decrease in intracranial pressure due to an external force expanding the skull is neutralised by the cavernous sinus continually diverting the venous blood into the slowly releasing channel that is through the basilar plexus until the available

expansion of the skull has reached its maximum and the intra cranial pressure reaches homeostasis.

The areas of the skull not affected by the trauma would be freer to expand than the locked or traumatised areas and would present greater flexibility as the skull wishes to expand. This would offer a palpatory tool for diagnosis as to the site and nature of trauma within the skull and a guide for treatment, although its nature would be difficult to test.

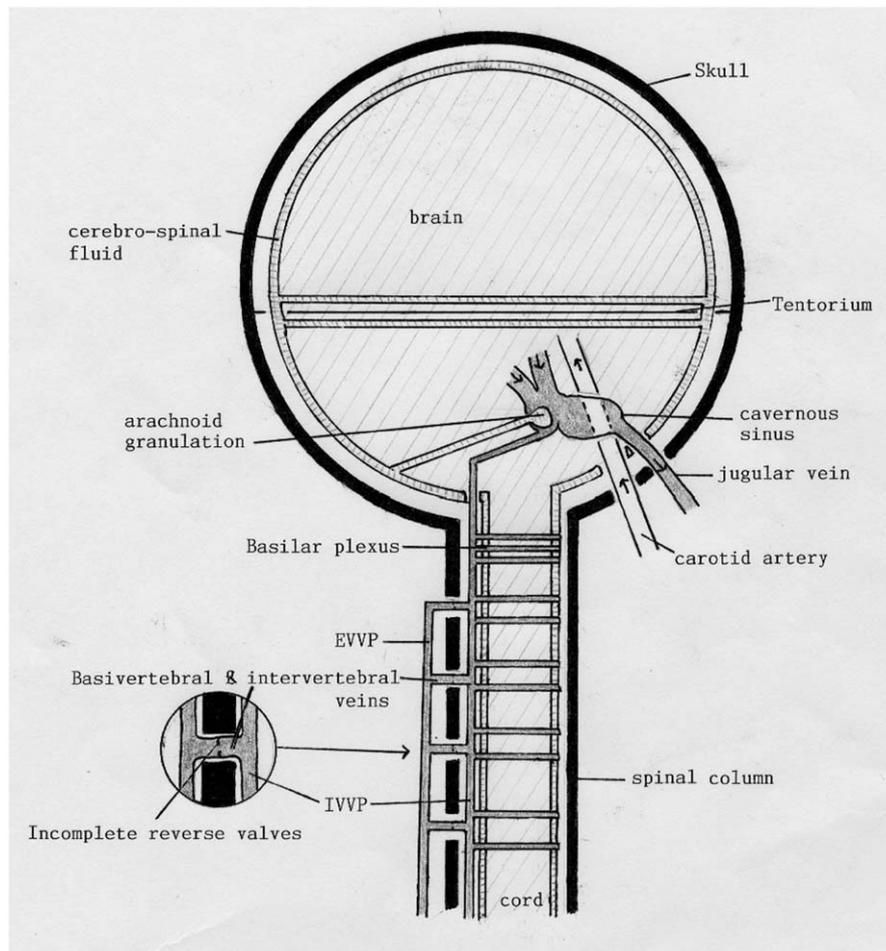


Fig. 7 A diagram illustrating the vascular mechanism which regulates the intracranial pressure.

It does, however offer an explanation as to the variable movements of the skull interpreted in different ways by Sutherland (1939), Upledger and Vredevoogd (1983). This is a further purpose of this paper.

Cranial osteopathic techniques

The hydraulic model may explain the action of commonly used cranial techniques. An example would be the technique known as fourth ventricular compression, the CV4 (Upledger and Vredevoogd, 1983). It would seem implausible that gentle external compression would compress deep internal spaces without significant damage to the brain. It does, however, provide gentle deep pressure to the sub-occipital muscles which will lead to a functional relaxation which allows for the occipito-atlantoid joint to extend more comfortably. It also dramatically reduces the tension holding the occipital bone in a flexed position, allowing the

cranial base to flatten into expansion (the “bowl” becoming a “saucer”).

When the temporal bones are encouraged to move away from each other by direct bilateral lateral traction applied to the mastoid processes, supported by structural techniques to relax the tension in the upper neck, the occipital bone extends in relation to C1. The temporal bones, influenced by this movement, posteriorly rotate, widening the cranial base. Upledger and Vredevoogd (1983) refers to this movement as the flexion phase. Ultimately, the extension of the occipital bone will be limited by its contact with the sphenoid, preventing further movement. Upledger and Vredevoogd (1983) identifies a “still point” where the cranial rhythm shuts down. A similar feeling of immobility may be due to a mechanical blocking of a range of movement. In order for further expansion of the skull to occur, the sphenoid and frontal bones have to be encouraged to move forward. Direct techniques to the frontal and facial bones to increase the anterior expansion

of the skull may achieve this and the skull experiences the “extension” phase. Once again, the range of this movement is limited by the position of the temporal bones. Another “still point” is reached and the process starts again. The movement palpated as a cranial rhythm may therefore be the above mechanical interplay within the bones of the cranial base, influenced by a desire to expand.

Further “cranial” movements and treatment modalities such as discussed by [Kok Weng Lim \(2006\)](#) can be explained within the proposed model.

Testing the model

Examining the effect of treatment on babies over a period of time presents some opportunity for studies to support the hypothesis. Measuring the circumference of the baby’s skull horizontally above the ears and along the cranial baseline as well as the length and width of skull before, during and after treatment will demonstrate a faster convergence to a normal stable fully expanded skull shape than a control baby. A successfully treated baby would have the bones of the skull feeling as if they were free floating with no desire for movement rather than the skull flexing or extending as in the model proposed by Upledger and Vredevoogd (1983).

Furthermore, a small fontanelle size suggests there is less free space in between the skull bones so limiting the ability for them to move and expand in relation to each other. The absolute size of the fontanel, as well as the increase in its size over treatment can be assessed, which in turn can be measured relative to the success of treatment outcomes.

The pain and aura of migraine can be explained in this model. Muscular tension at the sub-occipital level can lead to head and neck pain. Chronic restriction of the upper neck, in particular O/C1 will alter the function of the occipital bone and the increased arterial pulsation in the cavernous sinus wall may irritate the cranial nerves (including the oculomotor, ophthalmic, and maxillary nerves) that are embedded in the sinus wall and give rise to the aura symptoms of migraine.

The predisposition of some individuals to migraine may be due to an anomaly within the vessel containing the arachnoid granulation at the level of the great cerebral vein and straight sinus preventing the closure of the vessel, fixing csf production at its maximum level. The only way to study this hypothesis is by examination of brain scans accu-

rate enough to demonstrate the anatomy around the great cerebral vein and straight sinus.

Conclusion

The structures outlined within this paper suggest a vascular mechanism whose function is to regulate the pressure inside the skull. This mechanism causes a physical action upon the bones of the cranium and spinal column whereby there is a potential for expansion of the cranium to its maximum possible internal volume.

This involuntary expansion of the skull presents a credible foundation for movement palpated by osteopaths exploring cranial movement.

With a plausible foundation behind it, the relevance and limitations of “cranial” work can be explored within the context of osteopathic philosophy and treatment as a whole. A model based on accepted anatomy will assist in promoting the credibility of treatment based on involuntary movement to a wider audience within and beyond the osteopathic profession. As this mechanism has a vascular component, it is in accordance with A.T. Stills’ primary principles.

Ultimately, the best techniques and treatments for each individual patient will be based on the benefit to the patients themselves rather than the preference of a practitioner for particular techniques.

Acknowledgement

Figs. 2–6 reprinted from Williams; GRAY’S ANATOMY, 38th edition, copyright 1995 with permission from Elsevier.

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