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CEREBROSPINAL FLUID STASIS AND ITS CLINICAL SIGNIFICANCE

James M. Whedon, DC
An instructor at The Dartmouth Institute for Health Policy and Clinical Practice in Lebanon, New Hampshire, and trauma registrar at Dartmouth-Hitchcock Medical Center, also in Lebanon. He maintains a private chiropractic practice in Grantham, New Hampshire

Donald Glassey, MSW, DC, LMT
A member of the postgraduate faculty of Life University and the founder of Cerebrospinal Fluid Technique Seminars. He was formerly an instructor of Network Chiropractic and a faculty member of Pennsylvania College of Straight Chiropractic. He has retired from the private practice of chiropractic in Newtown, Pennsylvania

Abstract

We hypothesize that stasis of the cerebrospinal fluid (CSF) occurs commonly and is detrimental to health. Physiologic factors affecting the normal circulation of CSF include cardiovascular, respiratory, and vasomotor influences. The CSF maintains the electrolytic environment of the central nervous system (CNS), influences systemic acid-base balance, serves as a medium for the supply of nutrients to neuronal and glial cells, functions as a lymphatic system for the CNS by removing the waste products of cellular metabolism, and transports hormones, neurotransmitters, releasing factors, and other neuropeptides throughout the CNS. Physiologic impedance or cessation of CSF flow may occur commonly in the absence of degenerative changes or pathology and may compromise the normal physiologic functions of the CSF. CSF appears to be particularly prone to stasis within the spinal canal. CSF stasis may be associated with adverse mechanical cord tension, vertebral subluxation syndrome, reduced cranial rhythmic impulse, and restricted respiratory function. Increased sympathetic tone, facilitated spinal segments, dural tension, and decreased CSF flow have been described as closely related aspects of an overall pattern of structural and energetic dysfunction in the axial skeleton and CNS. Therapies directed at affecting CSF flow include osteopathic care (especially cranial manipulation), craniosacral therapy, chiropractic adjustment of the spine and cranium, Network Care (formerly Network Chiropractic), massage therapy (including lymphatic drainage techniques), yoga, therapeutic breathwork, and cerebrospinal fluid technique. Further investigation into the nature and causation of CSF stasis, its potential effects upon human health, and effective therapies for its correction is warranted.

In 1927 Cushing described CSF motion as the third circulation (the first and second circulatory systems being the cardiovascular and lymphatic systems).[1] CSF flow is characterized by both circulatory and fluctuant movement. Circulatory movement due to hydrostatic pressure gradients occurs primarily with CSF secretion at the choroid plexuses, and with CSF resorption at the arachnoid granulations. The CSF also fluctuates in a rhythmic ebb and flow in both the cranium and the spinal canal. Sutherland ascribed great significance to the CSF fluctuation, describing it as the *Primary Respiration* (thoracodiaphragmatic breathing being the *Secondary Respiration*).[2, 3]

Disclosure: Author Donald Glassey is the owner of Cerebrospinal Fluid Technique Seminars.

THE THIRD CIRCULATION

On average, approximately 150 ml of CSF circulates around the central nervous system at any one time, yet the total amount of CSF produced daily, approximately 500 ml, is enough to completely replace the circulating volume three to four times over.[4] The CSF is formed and secreted primarily by the choroid plexus of the cerebral ventricles, particularly the lateral ventricles; smaller amounts are secreted by the ventricular ependyma, the arachnoid membranes, and by the brain itself via the perivascular spaces.[5]

CSF secreted in the lateral ventricles flows through the interventricular foramina of Monro into the third ventricle in the mid brain, between the right and left halves of the thalamus. Flow continues through the cerebral aqueduct of Sylvius into the fourth ventricle, in the area of the middle and lower brain stem. The fourth ventricle narrows to form the central canal of the spinal cord, which extends through the medulla and the entire length of the cord. From the fourth ventricle, a portion of the CSF flows inferiorly through the central canal. Most of the CSF escapes through the median foramen of Magendie and the two lateral apertures of Luschka into the subarachnoid space (SAS). CSF in the SAS communicates with the cisterns at the base of the brain and percolates through the arachnoid trabeculae, diffusing superiorly around the brain and inferiorly around the spinal cord. The spinal SAS is divided into anterior and posterior compartments by the dentate ligaments, which anchor the cord laterally to the spinal dura mater. CSF circulates within the spinal canal through the SAS and potentially through the central canal. In most adults over the age of 30, however, the central canal is probably not patent.[4] The central canal communicates inferiorly with the subarachnoid space at the conus medularis, at the superior aspect of the lumbar cistern, a reservoir of CSF that also contains the cauda equina and filum terminale.

Mechanisms for continuous secretion and resorption of CSF effectively maintain intracranial and intrathecal pressure equilibria. The Monro-Kellie Doctrine, first postulated by Alexander Monro in the eighteenth century, describes the cranium as a rigid box filled with the incompressible contents of brain, blood and CSF. These three space-occupying components of the cranium must co-exist in a state of dynamic equilibrium, wherein a change in volume in one component requires a change in volume in either or both of the other two.[6] Any increase in total volume of the cranial contents must result in elevated intracranial pressure. The major site of resorption of CSF into the venous blood stream is through the arachnoid villi of the superior sagittal sinus.[5]

CSF is also in communication with the extracellular spaces of the brain, and mixes with the extracellular fluid within perivascular extensions of the subarachnoid space, the Virchow-Robin spaces.[7] Much of the resorption of CSF may occur directly from the extracellular matrix,[8,9] and significant amounts of CSF may pass into the lymphatic system.[10] CSF crosses the cribriform plate and enters lymphatic vessels of the nasal submucosa,[10] and animal studies have demonstrated that some cranial CSF drains into cervical lymph nodes.[10] Not all of the CSF in the lumbar cistern returns to the cranium; some is absorbed into lymphatics where the spinal nerve root sleeves merge with the epineurium.[10] Investigators in an animal study found that 25% of all CSF resorption was from the spinal SAS.[11]

Spinal Circulation of CSF

It is something of a misnomer to speak of CSF “circulation”, particularly in the spinal canal, as there is no continuous loop circulation of CSF as in the cardiovascular system. The spinal CSF fluctuates in biphasic tides of cephalic ebb and caudal flow. The fluid dynamics of CSF fluctuation in the spinal SAS are complex, and paradoxical flow patterns have been observed. Levy and Di Chiro noted that opposing flow patterns sometimes occur in the anterior and posterior subarachnoid spaces.[12] Radionuclide cisternography has shown that CSF follows the

path of least resistance as it flows through the spinal SAS. Centrifugal forces appear to propel the flow of CSF toward the convexities of the lumbar, thoracic and cervical curves.[14]

We focus upon the spinal flow or "circulation" of CSF because spinal CSF appears to be particularly prone to stasis due to the relatively high capacitance of the lumbar cistern and lower intrathecal pressures in the distal spinal canal,[12] as well as the apparent absence of a strong active mechanism for cephalad flow and the gravitational effect of upright posture. The relatively high pressure pulse waves and velocities in the cranium and cervical SAS normally diminish significantly with caudal flow, with little or no flow at the system's periphery in the relatively capacious SAS of the lumbar cistern.[13] In addition, the arachnoid trabeculae and denticulate ligaments likely constitute physical barriers to cephalad spinal flow, which does not benefit from the relatively strong cranial pressure pulse wave that propels caudad spinal flow. Like the venous circulation, CSF circulation depends upon indirect and passive mechanisms to propel cephalad flow. But unlike the veins, the SAS contains no valves to prevent backflow, and both caudad and cephalad flow of CSF may occur simultaneously in the spinal SAS.[13]

Cardiovascular Influences on CSF Fluctuation

Calling into question the validity of the "rigid box" of the Monro-Kellie Doctrine, Sutherland was the first to propose that most cranial sutures do not ossify, but remain mobile throughout life.[15] Although there is evidence for expansibility of the cranial sutures in adults,[16] the subject remains controversial.[17] The Monro-Kellie Doctrine remains the basis for our understanding of changes in intracranial pressure, and explains how expansion of the brain drives the CSF circulation. MR imaging has demonstrated pulsatile motion throughout the brain, particularly in the diencephalon and brain stem.[8, 17–20]

Sutherland correctly predicted in 1939 that rhythmic movements of the brain and associated compression of the cerebral ventricles generate the CSF pressure pulse wave. However, he described a system of spontaneous oscillations of the brain, spinal cord and cerebrospinal fluid.[2,3,15] Magoun later proposed rhythmic secretion of CSF by the choroid plexus as a cause of brain motility and the CSF pulse wave.[21] Building upon the ideas of Sutherland and Magoun, Upledger and Vredevoogd hypothesized that the cranial sutures possess a stretch reflex, which when activated by cranial expansion, feeds back to the ventricular system to reduce production of CSF, thus maintaining intracranial pressure equilibrium. Their `pressurestat' model attributed CSF fluctuation to the coordinated rhythmic movements of the choroid plexus, the cranial sutures, and central nervous system structures.[22]

These hypotheses of inherent brain motility as the engine of CSF fluctuation were inaccurate. Rhythmic brain motion is not primarily endogenous, but is propagated by the cerebrovascular pulse wave. The oscillatory brain movements are synchronous with cardiac systole.[18–20] The oscillatory brain movements extend through the length of the spinal cord, with a strong caudal motion tied to systole, followed by a weaker cephalad recoil.[18] Systolic arterial expansion causes a pulsatile expansion of the brain, resulting in a piston-like action that compresses the ventricles and propels CSF into the subarachnoid space and the spinal canal.[19,20] The spinal SAS thus allows for pulsatile decompression of the rigid box of the cranium with each systolic expansion. CSF aqueductal flow as well as outflow from the cranial cavity is related to systolic phase brain expansion.[9] In the systolic phase the midbrain and brainstem move caudally and medially toward the foramen magnum. In diastole, decreased arterial blood volume causes a reduction in overall brain volume, the brainstem retreats cephalad, and CSF flow reverses.[13]

CSF outflow into the cervical spine SAS correlates with cord motion and cardiac systole.[18, 19,23] Caudad flow in the subarachnoid space at C2 has been measured at up to 2.91 cm per second,[24] but flow velocity decreases inferiorly through the spinal SAS, and in the distal lumbar

sac drops essentially to zero.[12] As the systolic pressure wave propels CSF into the SAS, the elevated fluid volume increases strain on the distal dural sac, and at the end of systole, the built up dural tension powers a rebound pressure wave that propels the CSF cranially.[25] The resistance of the dentate ligaments also probably contribute to rebound motion of the cord following diastole.[26] Paradoxical flow patterns have also been observed in the spinal SAS, perhaps due to pulsations of the spinal arteries and epidural venous plexus that compete with cranial pulse waves.[13] A study on laboratory animals found that 77% of the lumbar CSF pulse wave was caused by arterial and venous pulses in the spinal canal, and 23% was caused by spinal transmission of the intracranial pressure pulse wave.[27] The strength of pulsations from the spinal vessels appear to provide a component of the pressure gradient required to propel cephalad flow against gravity in this low pressure system, but cephalad flow of CFS is significantly slower than caudad flow in the spinal SAS.[12] Tracer substance has been detected over the medulla within minutes following lumbar intrathecal injection, but diffusion likely accounts for at least part of this result.[28]

Respiratory Influences on CSF Fluctuation

The systolic pulse wave is well established as the primary impetus for CSF circulation, but there is evidence that the respiratory rhythm also plays a significant role. A recent study of CSF pressure oscillations in anesthetized rats found that the strongest oscillations were coincident with ventilatory chest movement (both spontaneous and mechanically assisted) rather than arterial pulse pressure. CSF pressure oscillations were found to be only weakly entrained to heart rate.[29]

Maier et al observed that normal respiration may induce periodic brainstem motion as strongly as systolic phase arterial expansion. The respiratory cycle drives a low frequency oscillation of the brainstem, in which caudal brain displacement corresponds with expiration and cephalad rebound corresponds with inspiration. The effect is more pronounced with forced respiration. This respiratory phase pulse, superimposed upon the higher frequency cardiovascular pulse, appears to influence cerebrospinal fluid fluctuation. Modified respiratory efforts affect CSF fluctuation in peculiar ways. Valsalva maneuver quickly causes caudad and then cephalad brainstem movement. Coughing causes a cephalad impulse in CSF flow.[30]

Vasomotor Influences on CSF Fluctuation

In addition to pulse and respiration, vasomotor activity also appears to influence CSF fluctuation. Vasomotor waves, or Traube-Herring Mayer (THM) waves are normal physiologic waves that are generated by spontaneous pulsations of arterial, venous and lymphatic vessels. THM waves are independent of the respiratory and cardiac cycles, and occur at variable frequencies, but with generally longer wavelengths than those of the respiratory and cardiac cycles. THM waves are mediated by the autonomic nervous system and along with increased heart rate variability, are considered to be markers of good autonomic balance.[31] THM waves of the intracranial arteries cause the propagation of waves of elevated intracranial pressure known as C waves, first described by Lundberg. Lundberg described C waves as rhythmic oscillations with a frequency of 4–8 per minute (most frequently 6 per minute) and an amplitude of up to 20 mm Hg. Because of the close association between C waves and THM waves, any effect of C waves is probably a healthy one.

CLINICAL SIGNIFICANCE OF CSF STASIS

Pathological disorders associated with disturbance of CSF circulation include hydrocephalus, intracranial hypertension (including brain edema due to trauma, infection or other pathology), and intracranial hypotension. CSF circulation can also be impeded by space occupying lesions, syringomyelia, and subarachnoid cysts. It has been suggested that reduced rates of CSF

diffusion through the extracellular spaces of the brain may be a cause of degenerative and age related disease.[32] Aging is associated with a 50% reduction in CSF production in healthy people,33 and age-related pathology of the cardiovascular and cerebrovascular circulation, coupled with calcification of the choroid plexus and decreased brain tissue compliance may also contribute to impaired CSF circulation.34 Aging is also associated with occlusion of the central canal and increased damping of systolic CSF flow4,[18] but causation has not been established. Rubenstein hypothesized that aging can lead to stagnation of the CSF circulation, which in turn may contribute to the development of some age-related dementias.[34]

We hypothesize that functional CSF stasis, a physiologic impedance or cessation of CSF flow, is also a common occurrence in the absence of degenerative changes or pathology, and that CSF stasis is detrimental to health. The concept of a physiologic CSF stasis is not entirely new. Its occurrence has been suggested by Rubenstein and Perrin[34,35] and alluded to by Sutherland, Upledger and Epstein.15,22,[36] The CSF maintains the electrolytic environment of the central nervous system (CNS), influences systemic acid-base balance, serves as a medium for the supply of nutrients to neuronal and glial cells, functions as a lymphatic system for the CNS by removing the waste products of cellular metabolism, and transports hormones, neurotransmitters, releasing factors and other neuropeptides throughout the CNS.[37] CSF stasis may compromise these normal physiologic functions related to CSF flow.22,34,35

CLINICAL ASSESSMENT OF CSF STASIS

CSF stasis may be associated with vertebral subluxation, skin tissue drag, and biofield disturbance.[38] Palpation may be a useful means of assessment of CSF stasis. Clinically it has been observed by Glassey that apparent changes in the coefficient of friction on the overlying skin surface create the tactile sensation of “drag” or resistance. This sensation can be detected by manual palpation with the fingertips, where Meissner's corpuscles, which detect fine touch, are concentrated in the upper layer of the dermis. The trained practitioner palpates for the sensation of resistance on the skin surface overlying the spine and cranium. Resistance is thought to be indicative of underlying CSF stasis, and following treatment, the absence of drag may indicate that the CSF stasis has been reduced.[38] Fulford described a similar sensation of drag on the skin surface, and attributed it to decreased flow of the human “life force”.[39] Toftness also described a change in tactile resistance overlying a vertebral subluxation, a phenomenon that ceased upon correction of the subluxation. He speculated that such areas of tactile resistance were associated with increased body surface electromagnetic activity.[40] Zhang et al partially validated Toftness' work with their findings that low force chiropractic adjustments can result in significant reductions in the body surface electromagnetic field, as measured by triaxial fluxgate magnetometry.[41,42]

It may be possible to clinically assess the effectiveness of therapies aimed at reducing impedance to CSF flow. CSF communicates with the inner ear through the cochlear aqueduct, and CSF pressure changes are reflected by changes in tympanic membrane tension. Impedance audiometry, which measures mechanical tension on the tympanic membrane, may offer a noninvasive means of measuring CSF pressure changes.[43] The relationship between intracranial pressure and CSF stasis is unclear. This measure has not been validated however, and tympanic membrane tension varies between individuals, so a baseline value must be established for each patient.

APPROACHES TO REDUCTION OF CSF STASIS

There is a paucity of published data on the effectiveness of therapies for reduction of CSF stasis. Therapies that are thought to affect CSF flow include osteopathic care (especially cranial manipulation), Craniosacral Therapy, chiropractic adjustments of the spine and cranium (especially Sacro-Occipital Technique and Network Care), lymphatic drainage techniques,

therapeutic breathwork, and yoga. Additionally, Cerebrospinal Fluid Technique draws from several of the above approaches with protocols specifically intended to reduce CSF stasis.[38]

The Cranial Rhythmic Impulse

Sutherland developed the first osteopathic techniques intended to enhance the flow of CSF. He was the first to describe movements of the cranial bones at the cranial sutures, primarily at the sphenobasilar junction, and he found that he was able to palpate subtle rhythmic motions at the cranium and sacrum.[15] Sutherland believed that these cranial and sacral motions were associated with his Primary Respiratory Mechanism, described above. Building upon Sutherland's observations of a craniosacral rhythm, Woods and Woods first described the cranial rhythmic impulse (CRI), an oscillation distinct from the respiratory and cardiovascular cycles, and normally occurring at a rate of 6–12 cycles per minute.[44] In 1976, Magoun named the same phenomenon “The Sutherland Wave”, after its discoverer.21 The CRI is thought to be related to CSF flow.44,45 The CRI is measurable and is palpable throughout the body. It is enhanced in health, and has been found to be depressed in numerous medical disorders.[44] John Upledger, trained as an osteopath and the founder of the Upledger Institute, taught that dural tension and decreased CSF flow corresponded with reduction in palpability of the CRI, conditions that could be corrected by gentle manipulation of the cranium and sacrum.[22]

Both Sutherland and Upledger believed that spontaneous cranial and sacroiliac articular motions exert a pumping action upon the CFS, and that these motions are transmitted and coordinated by tensile forces in the spinal dura;[2,3,22] this concept came to be known as the *core-link hypothesis*.[17] Mitchell and Pruzzo reported evidence of spontaneous sacral motion associated with normal respiration,[46] but there is no other data to support the notion that spontaneous motion of the sacrum upon the ilia occurs.[17] Even with gross pelvic and spinal flexion and extension, physiologic sacroiliac motion is quite limited, with rotation of 3 degrees or less and translational motion of 2 mm or less.[47] Periodic changes in dural tension alone are thus quite unlikely to transmit sufficient force to induce articular motion in the sacroiliac joints. A skilled clinician can palpate the CRI at the sacrum and coccyx, and Zanakis et al reported a 92% correlation between perception of movement at the sacrum and in the cranium.[48] However, the strongest influences on cord motion are the respiratory and cardiovascular rhythms, and by all accounts, the CRI is independent of those rhythms, so the meningeal connection between cranium and sacrum does not explain the palpability of pulsations at the sacrum. Current knowledge of spinal physiology does not support the validity of the core link hypothesis.

The relationship between the CRI and the THM oscillation serves as a more likely explanation for the perception of the CRI at the sacrum, and may also be associated with pulsatile flow of CSF in the spinal SAS. The CRI has been tied to the THM oscillation, a whole body phenomenon associated with various hemodynamic metrics, notably the THM vasomotor waves referred to above. In a double blind study, the rate of the CRI as measured by palpation was found to occur simultaneously with the THM oscillation as measured by Doppler flowmetry.[25] Given that the CRI and the THM oscillation are both whole body phenomena that occur simultaneously, it seems likely that the two are actually one and the same.[25] The THM oscillation has been linked to minor fluctuations of intracranial pressure known as *Lundberg C Waves*.[31] The propagation of the Lundberg C Wave, palpated at the sacrum as the CRI, may cause a palpable pulse of CSF flow in the distal spinal canal.

Cranial manipulation has been shown to affect the CRI/THM oscillation.[49,50] Craniosacral therapy is intended to reduce tension in the dural tube and thus free the flow of CSF. Cranial and sacral manipulative techniques that enhance the CRI/THM oscillation likely enhance the propagation of C waves in the CSF.

Autonomic Tone

"Disorder of the tone and irregularity in the rhythm are the principal causes of every illness." – *Sufi Master Hazrat Inayat Khan*[51]

Due to their close association with THM waves, we would expect that enhanced Lundberg C waves would also be a sign of autonomic balance. Balance between the sympathetic and parasympathetic nervous systems may result in frequency entrainment of multiple biological oscillators.[52] This state of optimal autonomic balance is consistent with increased heart rate variability, enhanced THM waves, and a strong palpable CRI.[53]

Osteopathic care may have profound effects upon autonomic balance. Upledger has suggested that increased sympathetic tone, facilitated segments (first described by Korr)54, dural tension and decreased CSF flow are closely related aspects of an overall pattern of structural and energetic dysfunction in the axial skeleton and central nervous system.22 Cutler et al demonstrated reduction in sympathetic nerve activity following cranial manipulation,55 In an unpublished thesis, Giles reported improvement in heart rate variability (a measure of parasympathetic activity) following manipulation of the cervical spine.56

Chiropractic care may also promote autonomic balance.[57] In a multisite clinical study, Zhang et al demonstrated significant increases in heart rate variability following a four-week course of chiropractic spinal manipulation.[58] Like Upledger, Epstein proposed that AMCT generates predictable patterns of segmental and plurisegmental facilitation with effects upon neural plasticity and perception.[36] A retrospective survey demonstrated that Epstein's Network Care was associated with numerous improvements in indicators of health-related quality of life that were suggestive of reduced sympathetic tone and enhanced autonomic balance.[59]

Adverse Mechanical Cord Tension

Excessive elongation (strain) of the spinal meninges causes adverse mechanical cord tension (AMCT). First described by Brieg,60 the phenomenon of AMCT and its clinical significance was popularized with the bodywork techniques developed by Epstein (Network Spinal Analysis)36 and Butler (Neural Stretching Technique).61,62

AMCT may be a principal cause of CSF stasis in the spinal SAS. The spinal cord is suspended in the dural sac bilaterally by the denticulate ligaments. The dura is in turn anchored superiorly and inferiorly to the interior bony surfaces of the spinal canal, and bilaterally at multiple levels to connective tissues in the intervertebral foramina. Strain of the spinal dura between points of attachment may result in strain of the spinal cord, expressed as increase in length over original length.[65] Cord strain occurs with normal physiologic spinal flexion; the greatest strain is sustained by the posterior aspect of the cord. In flexion the spinal cord and medulla normally stretch a total of approximately 2 to 3 cm, and at the level of the cervicothoracic junction, strain = 0.24 in flexion.[65]

Koschorek found that in flexion, the cord elongates an average of 12 mm, while the spinal canal lengthens 28 mm on average.[66] Cord strain exaggerated beyond normal physiologic limits becomes AMCT and may lead to development of CSF stasis. Deformation of the cord and meninges associated with AMCT reduces patency of the SAS,[65] and may cause CSF stasis. Abnormal dural fixation results in transmission of tension to the cord via the dentate ligaments, especially with spinal flexion, which adds an axial component to lateral tensile forces on the cord.[67]

Dentate ligament strain and associated AMCT have been described in the context of cervical spondylotic myelopathy (CSM).[65] As the cord is displaced posteriorly by an osteophytic deformity, the dentate ligaments are stretched between their pial attachments and the dura. The

resultant lateral shear stresses cause distortion and deformation of the viscoelastic tissues of the cord, accompanied by a corresponding reduction in width of the SAS, both anterior and posterior to the cord.65 Because CSF follows the path of least resistance in the SAS,14 flattening of the SAS is likely to reduce CSF flow, especially low pressure cephalad flow. Anteroposterior flattening of the cord may also account for occlusion of the central canal, which is common in adults,4 but is probably suboptimal. Central canal occlusion may be an early sign of adverse cord tension, and patency of the central canal may be a sign of a biomechanically healthy cord. AMCT may also contribute to development of CSF stasis by means other than flattening of the SAS, including loss of capacitance due to cord tethering[18] or chronic dural tension[18,68]. Because dural tension reduces compliance and motility,[18,68] adverse tension may reduce meningeal capacity for stretching in response to the cranial systolic pressure wave, with a corresponding reduction in the rebound pressure wave that is caused by shortening of the dura in the diastolic phase.[18] AMCT may also occur in association with vertebral subluxation, the manipulable lesion ostensibly treated by chiropractors.[36] Sacro-occipital Technique, Network Spinal Analysis and Cerebrospinal Fluid Technique all incorporate chiropractic clinical protocols intended to reduce AMCT.[36,38,69]

Lymphatic Drainage

Perrin advocates treatment of chronic fatigue syndrome by reducing CRI abnormalities due to neurolymphatic blockage. He has suggested that impaired CRI is associated with CSF stasis and a build-up of cerebral toxins, and restrictions in CSF circulation can be reduced with manual therapies intended to enhance neurolymphatic drainage. Perrin's approach consists of multiple components, including effleurage techniques directed to the cervical and thoracic lymphatics, manipulation of the spine, sacrum and ribs, massage of the levator scapulae, paravertebral muscles, accessory respiratory muscles, rhomboids, and trapezii, and an osteopathic technique of fourth ventricle compression to stimulate the CRI.[35]

Therapeutic Breathwork

Therapeutic breathwork techniques that are thought to enhance CSF fluctuation include Rebirthing, Transformational Breathwork and Holotropic Breathwork, as well as yogic breathing techniques such as alternate nostril breathing (as taught in Kundalini Yoga), Shushumna Breath, and Kriya Yoga Pranayama. Expiration propels CSF caudad, while inspiration favors cephalad flow.[30] Due to the normal fluctuations in intrathoracic pressure associated with respiration that favor filling and emptying of cardiopulmonary vessels, entrainment of inspiration to diastole and expiration to systole would have the effect of enhancing cerebrovascular circulation, and increasing CSF fluctuation.70 Chronic stress and anxiety lead to restriction of the respiration, and associated muscle and connective tissue tension may contribute to the development of AMCT by way of direct and indirect connections with the spinal dura. Therapeutic breathwork is intended to reduce chronic restrictions in the breath. Modified respiratory effort associated with certain body/mind practices such as breathwork, meditation, yoga, and relaxation therapies tend to induce deep relaxation, increased parasympathetic tone and an enhanced CRI, and may have the effect of enhancing Lundberg C Waves.

Yoga

Yoga practice may enhance CSF flow. CSF flow appears to be related to postural changes in pressure measurements, which correspond closely with the degree of inclination of the body. In the prone position, cranial and lumbar CSF pressures are approximately equal. Standing posture reduces cephalad flow of CFS, lowers cervical SAS pressure, and increases lumbar pressure.[71] With cervical spine flexion, the ventral SAS narrows up to 43%, and the dorsal SAS widens up to 89%. In extension, the ventral cervical SAS increase up to 9%, and the dorsal

SAS is reduced up to 17%, as compared with the neutral position. These changes are associated with changes in the sagittal diameter of the cervical cord, which is decreased 14% in flexion and increased 15% in extension, compared with neutral.[72] Because CSF flow appears to follow the path of least resistance,[14] yoga postures of spinal flexion, extension and inversion, as well as patient positioning for structural and energetic therapies, can be expected to affect CFS flow. Alternating spinal flexion and extension may create a pumping effect that enhances CSF fluctuation. In a study involving seventy-one human subjects, Byrne et al demonstrated that oscillatory head tilt significantly increased the amplitude of THM variance, and the changes were apparently moderated by increased parasympathetic tone.[73] Yogic practices that induce what some sources refer to as *kriyas* (involuntary oscillations of the spine and or extremities) may enhance CSF flow as well. (In Sanskrit the word "*kriya*" denotes action or a process that can produce a desired effect.) The somatopsychic wave phenomenon reported in recipients of Network Care may have a similar effect.[36]

Cerebrospinal Fluid Technique

Cerebrospinal Fluid Technique (CSFT) developed and described by Glassey, employs a multidisciplinary approach to treatment of CSF stasis. The CSFT clinical procedure employs both low force chiropractic techniques and massage strokes at multiple points along the cerebrospinal axis. The CSFT protocol is intended to free impedance to CSF flow around the brain and spinal cord. CSFT procedures address all the bones of the spinal column, from the first coccygeal segment up to and including the atlas. Also included are the cranial bones which can be accessed externally. Other osseous structures which directly or indirectly attach to the spine such as the pelvis and sternum may also be included in the clinical protocol.[38]

CSF and the Life Force

Early investigators ascribed powerful vitalistic properties to the cerebrospinal fluid. Sutherland referred to the CSF as "the breath of life", and "liquid light",[3] and Randolph Stone, the founder of Polarity Therapy, called CSF "the liquid medium for the Breath of Life".[74] In yoga and meditation practice, the experience of awakening the dormant power potential of the *Kundalini* has been described as a powerful flow of energy from the base of the spine to the crown of the head. On a subtle energetic level, *Kundalini* energy is said to flow up the spinal pathway (in Sanskrit the *shushumna nadi* or central pathway) in a manner similar to some aspects of the physiologic movement of CSF.[38] This and other peak experiences often associated with yoga, meditation, breathwork and various structural and energetic therapies may result from enhanced CSF flow.

CONCLUSION

There is evidence to suggest that CSF stasis may occur commonly in the absence of pathology or symptomatology, and may have adverse systemic health effects. CSF stasis may be associated with adverse mechanical cord tension, vertebral subluxation syndrome, reduced cranial rhythmic impulse and restricted respiratory function. Various structural and energetic therapies may have the effect of enhancing CSF flow, but little is known about their mechanism of action and effectiveness in this regard. Further investigation into the nature and causation of CSF stasis, its effects upon human health, and effective therapies for its correction, is warranted.

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