Re: CSF and recent research -- comment from Tim Hutton

By Eric Moya and Ger Swords in Upledger Alumni (Files) · Edit Doc

A Comment on the Relationship of Recent Research on CSFProduction and Reabsorption to the Pressurestat Model

Recently, a lot of very interesting research has come out onhow cerebrospinal fluid (CSF) is produced and reabsorbed in the central nervoussystem (CNS). This research has totallytransformed our understanding of this neurological process. I wanted to comment briefly on therelationship of this recent research to the pressurestat model that is taughtin Upledger CranioSacral Therapy classes. The pressurestat model is a model that was developed by John Upledgerand Ernest Retzlaff to explain the rhythmic expansion and contraction of thecranial vault and the concurrent external and internal rotation of the tissueof the body known as the craniosacral rhythm (CSR).

The classic neurological model for CSF production andreabsorption, which has been taught for years, states that all CSF productionoccurs in the choroid plexi, particularly those in the two lateral ventricles, andall CSF reabsorption to take place in the arachnoid villi of the venous sinussystem. Recent research has demonstrated,however, that a significant portion of CSF production and reabsorption, perhapsas much as 60 to 70 percent, occurs locally at the blood brain barrier in thecapillary beds of the central nervous system (CNS). (See for example the review article, Chikly, B.,Quaghebeur, J., Reassessing cerebrospinal fluid (CSF) hydrodynamics: Aliterature review presenting a novel hypothesis for CSF physiology, Journal ofBodywork & Movement Therapies (2013), Online publication complete:12-APR-2013) Only 30 to 40 percent of the total CSF production seems tooccur in the choroid plexi. Recentresearch has also shown that a significant portion of the global CSF productionis reabsorbed directly into the lymphatic system rather than though thearachnoid villi into the venous sinuses.

Upledger and Retzlaff's pressurestat model proposed that theCSR was the result of minute rhythmic pressure changes in the cranial vaultthat occur as a consequence of the action of the body's mechanism forcontrolling baseline CSF pressure. Theypostulated that while CSF reabsorption was constant, CSF production in thechoroid plexi cycled on and off, at a rate of 6 to 12 cycles per minute, inresponse to signals from pressure and stretch receptors located primarily inthe sagittal suture. Given that we nowknow that only a portion of CSF production takes place in the choroid plexi, doesthis new research have any implications with regard to the pressurestat model?

It is my contention, speaking as a scientist, and as someonewho has been doing CranioSacral Therapy (CST) for many years, that this newresearch does not conflict in any way with the pressurestat model, and in factexplains some things that have puzzled me for a long time. The two fit together perfectly. I would, in fact, be very surprised if thebody did not have both a mechanism for local production and reabsorption ofCSF, and one for global production and reabsorption as well. These two mechanisms serve very differentpurposes in the CNS.

I have always been vaguely uneasy with the classicalneurological model, with the idea that all CSF production takes place at theplexi and all reabsorption takes place in the villi. CSF plays the roles of both interstitial fluidand lymph in the CNS. It carries awaymetabolic waste from the tissue. Thepresence of excess metabolic waste in the interstitial space is highlyinflammatory and it would seem particularly important for the properfunctioning of the body to prevent such accumulation in the tissue of the CNS. It made no sense to me that the body wouldrely on a mechanism for waste removal that was very slow, which turned thefluid volume over only a few times a day, and one that required the wasteproducts to be transported long distances through CNS tissue before they wereremoved. Local production andreabsorption of CSF would be much more efficient, and would allow for nearlyinstantaneous removal of waste products into the venous blood flow. Accomplishing this removal locally also meansthat waste products do not need to be transported through the CNS tissue to beremoved, potentially producing more inflammation along the way.

It is difficult, however, for me to see how the body wouldbe able to control baseline CSF pressure if all the fluid production was local. How would the body coordinate billionsof local CSF production centers? Thereneeds to be a mechanical sensor that provides the signal to indicate when CSFpressure is too high or too low. Distributing that signal to billions of localCSF production centers would seem to be a daunting task. The body is generally very efficient, doingthings in the easiest possible way, and Ockham's Razor would argue for a muchsimpler arrangement. Stretch andpressure receptors in the sutures seem to me to be a simple and ideal way toprovide the mechanical signal needed to control fluid pressure, and so long asa significant portion of total CSF production takes place in a centralizedlocation, passing the signal from the sutures to that location would be anelegant solution to this problem.

As mentioned above, the essence of the pressurestat model isthat, while the fluid reabsorption is constant, the fluid production cycles onand off. Where the fluid is reabsorbedis irrelevant as far as this model is concerned, so it makes no difference tothe pressurestat if the globally produced CSF is reabsorbed into the lymph,locally in the region of the choroid plexi where it is produced, or into thevenous sinuses via the arachnoid villi, or any combination thereof. The pressurestat model also does not requirethat **all**the fluid production be turned on and off in this way. So long as a large enough percentage of totalproduction cycles on and off, it would be possible to control baseline CSF pressureeffectively. The recent research stillpostulates 30 to 40 percent of total CSF production taking place in the choroidplexi, presumably more than enough to run the pressurestat.

As evidence that the mechanical signal needed to controlbaseline CSF pressure does indeed come from the sagittal suture, I offer thefollowing case report. Several years agoI treated a young man, age 14, who a few years previously had taken a fall onhis bicycle and landed on his head, crushing the helmet he was wearing over theleft parietal. Sometime after theaccident he developed a severe constant category 9 - 10 headache. Medical examination revealed that he hadseverely elevated CSF pressure and he was given a

diagnosis of pseudotumorcerebri. When I initially examined him, he exhibited no CSR anywhere on his body. He was not in a stillpoint or a significancedetector stop. It felt more like the CSsystem simply did not exist, as if the master switch had been turned off. Examination also revealed a severelycompressed left parietal, totally jamming the sagittal suture. Releasing the left parietal immediatelyrestored good CSR throughout his tissue, and two weeks after the treatment, once the accumulated metabolic waste had been carted off and the resulting inflammationhad died down, his headache went away.

This one treatment convinced me of the basic validity of thepressurestat model. With the sagittalsuture jammed, stretch sensors in the suture never fired, and were unable tosend a signal to the choroid plexi instructing them to turn off fluidproduction. In addition, pressuresensors in the suture were constantly being stimulated, in effect telling thebody to produce more fluid. The plexicontinued to produce fluid until the baseline CSF pressure matched baselinearterial pressure, at which point it was no longer possible to filter CSF fromthe arterial blood. The entire systemshut down and the result was a massive accumulation of waste products in theCNS and a horrendous headache. Releasingthe suture allowed the system to begin functioning again and start carting offthe metabolic waste.

All scientific models explain certain behaviors of thesystem they are meant to describe, and do not explain others. Thus, all scientific models have limitedregions of validity. Another way to saythis is to note that all scientific models are to some extentoversimplifications. Hopefully they are usefuloversimplifications, but they are oversimplifications nonetheless. I am certain the pressurestat model is anoversimplification of what happens in the tissue, but I am also convinced thatit offers a good explanation of the basic physiological basis for the CSR.

In what areas is it likely that the pressurestat model isvalid, and in what areas is it likely not valid? The pressurestat model explains the physiologicalbasis for the CSR quite well, and is consistent with the fact that the body cango into stillpoint. Presumablystillpoint is a process mediated by the CNS to release tensions in the autonomicnervous system, and in the body as a whole. I see no theoretical conflicts between thisbehavior and the pressurestat model. Thepressurestat model does not, however, explain such behavior as the significancedetector. In particular, it does notexplain the fact that one can dialogue, silently or out loud, with the CSR, andit will turn on and off locally in response to yes/no questions from thetherapist. These behaviors are muchbetter explained using a model that considers the CSR to be a vehicle forcommunication between the conscious mind of the therapist and the nonconsciousof the patient.

It is interesting to note that **any** rhythm in the bodycan be used to dialogue with the nonconscious of the patient in this way. Thisincludes visceral motility, any of the lymphatic rhythms, or indeed any of themany other rhythms of the body. In myexperience, this seems to be a universal property of the body/psyche, not aproperty of any individual rhythm. Askingyes/no questions of any of these rhythms will cause the rhythm to turn on andoff locally, provided the therapist has set that intention, in effect providedthe therapist has made that agreement with the Inner Physician of the patient.

In summary, I see no conflict whatsoever between this latestresearch and the pressurestat model. This research, in fact, clears up sometroubling aspects of the classical neurological model. Now that we understand how it all works, Iwould be surprised if the body functioned any other way.

Tim Hutton, Ph.D., CST-D