ORIGINAL ARTICLE

Volume change theory for syringomyelia: A new perspective

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ABSTRACT

Background: The etiopathogenesis of syringomyelia is still an enigma. The authors present a novel theory based on fluid dynamics at the craniovertebral (CV) junction to explain the genesis of syringomyelia (SM). The changes in volume of spinal canal, spinal cord, central canal and spinal subarachnoid space (SSS) in relation to the posterior fossa have been analysed, specifically during postural movements of flexion and extension. The effect of fluctuations in volume of spinal canal and its contents associated with cerebrospinal fluid (CSF) flow dynamics at the CV junction have been postulated to cause the origin and propagation of the syringomyelia. The relevant literature on the subject has been reviewed and the author's theory has been discussed.

Conclusion: Volume of spinal canal in flexion is always greater than that in extension. Flexion of spine causes narrowing of the ventral subarachnoid space (SAS) and widening of dorsal SAS while extension causes reverse changes leading to fluid movement in dorsal spinal SAS in flexion and ventral spinal SAS in extension. Cervical and lumbar spinal region with maximum bulk hence maximum area and volume undergo maximum deformation during postural changes. SSS CSF is the difference between the volume of spinal canal and spinal cord, varies in flexion and extension which is compensated by changes in posterior fossa (CSF) volume in normal circumstances. Blocked SAS at foramen magnum donot permit spinal SAS CSF exchange which during postural changes is compensated by cavitatory/cystic (syrinx) change at locations in cervical and lumbar spine with propensity for maximum deformation. Augmentation of posterior fossa volume by decompression helps by normalization of this CSF exchange dynamics but immobilizing the spinal movement theoretically will cease any dynamic volume changes thereby minimizing the destructive influence of the fluid exchange on the cord. Thus, this theory strengthens the rational of treating patients by either methodology.

Key words: Cerebrospinal Fluid, Chiari malformation, Syringomyelia, Craniovertebral junction

Introduction

Syringomyelia (SM) is a condition in which abnormal development of cyst or cavity occurs within the spinal cord commonly referred to as syrinx. The abnormal dilatation of central canal is referred to as hydromyelia.The above findings coexists simultaneously and hence are referred to as syringohydromyelia. Chiari malformation (CM) is a common cause of cervical SM. Incidence in population is around 8.4 cases per 100,000 person, presenting in third or fourth decade of life with mean age of presentation around 30 years. SM is classified into four types: Communicating, noncommunicating, atrophic cavitation (syringomyelia ex vacuo), and neoplastic cavitation. Communicating SM is due to obstruction of CSF pathways, located distal to the fourth ventricular outlets, creating a syrinx that communicates with the fourth ventricle while noncommunicating SM, the syrinx and dilated central canal do not communicate with the fourth ventricle, and the obstruction is usually located at or below the level of the foramen magnum. The latter type of SM is commonly associated with Arnold-Chiari type I malformation in nearly 50% of cases, where CSF flow from the posterior fossa to the spinal canal is obstructed.[1,2]

While other causes of syringomyelia include hindbrain anomalies, neoplastic disease, inflammatory conditions, and

Access this article online	
Quick Response Code:	Website: www.asianjns.org
	DOI: 10.4103/1793-5482.162680

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trauma.[3,4] The syrinx cavity may remain static or enlarge in size over a period of time. They may enlarge symmetrically with enlargement of central canal, paracentrally in one or more focal areas or eccentrically off the center.[5] Signs of this disorder tend to develop rather slowly, however sudden onset may occur with coughing, straining, or myelopathy.[6] This pattern of enlargement results in motor and sensory changes with severity of clinical manifestations depending upon the location and the size of the syrinx.

Review of Literature

Several theories have been put forth to explain the pathogenesis of SM, chief among them is Gardner's hydrodynamic theory which is widely accepted for more than two decades. Gardner attributed the genesis of SM to craniospinal pressure differentials in the setting of fourth ventricular outlet obstruction; these differentials favor CSF shifts from the fourth ventricle of the brain through the central canal of the spinal cord. Majority consider abnormal craniospinal hydrodynamics due to obstructed CSF flow. He proposed that syrinx communicated freely with the fourth ventricle in CM patients and its enlargement was suggested due to altered stresses in the hindbrain region. But syrinx cavity was not found often to communicate freely with the fourth ventricle.[7-9]

Williams proposed a suck and slosh mechanism in which involved longitudinal pressure dissociation between the head and spine called Craniospinal pressure dissociation which is due to partial blockage in the spine. The baseline pressures in the head and spine are usually equal in humans if measured at rest. Transient craniospinal pressure dissociation has been found by subtracting the ventricular pressure from the spinal subarachnoid pressure The pressure tracing shows upward deflection suggestive of the pressure difference available for the fluid movement upwards and vice versa. Whenever abdominothoracic pressure rises, the epidural venous plexus pressure increases causing engorgement around the dural sac leading to upward movement of CSF from the lumbar spine to the head. Coughing displaces CSF upwards with pressures beyond 100 mm/Hg lasting less than a second which is very destructive. The CSF pressure drops almost equally rapidly to its resting position. Pressure difference beyond 100 mm/Hg has been found in those with hindbrain hernia. The suck occurs during the post-Valsalva rebound. During coughing or Valsalva the CSF moves more easily towards the area of least resistance in the rostral direction as compared to caudal direction which is due to ball-valve effect at the foramen magnum.[2,10,11]

This ball-valve effect causes longitudinal pressure gradients in the spinal canal. To validate his hypothesis, Williams Obtained pressure measurements throughout the CSF system on both humans and animals.[12-17] Williams provided evidence for longitudinal pressure dissociation (LPD) due to flow obstruction which explained a mechanism causing movement of fluid within the cavity rather than bulk movement. He postulated that pressure gradients exaggerated by enlargement of the syrinx, explaining how larger diameter syrinxes progress faster than smaller ones[18,19] and suggested that the size of the syrinx may be related to the CSF pulsation.[20-23]

Ball and Dayan postulated in that CSF was forced though the Virchow-Robin spaces into the spinal cord forming a cyst.[24] Histopathological study of affected spinal cord tissue revealed dilated perivascular spaces. He thought that the syrinx formation was caused by flow obstruction leading to prolonged increased pressure in the CSF system. The communication between the CSF and extracellular fluid has been shown in a number of studies.[25,26]

Oldfield *et al*., postulated a systolic pressure theory for the progression of a syrinx in patients with CM.[27,28] He suggested that brain expansion occurs during systole leading to CSF pressure wave travelling down the SAS across the foramen magnum which was obstructed at the foramen magnum. And this led to pegging of the lower cerebellar tonsils functioning like piston leading to impaction at the foramen magnum resulting in CSF movement through the perivascular spaces or dorsal root entry zone. During intra-operative ultrasound he recorded the tonsillar movement. He was successful in throwing light on a possible mechanism for damage to the spinal cord due to the piston effect, but could not provide pressure evidence to explain CSF movement through the cord tissue and into the syrinx.[29]

Greitz suggested that increase in CSF pulse pressure was encountered in the vicinity of a SAS obstruction which is found in CM and patients of post traumatic syringomyelia leading to accumulation of fluid and distension of spinal cord (SC).[30,31] Also Venturi effect was suggested to cause outward movement of the SC leading to suction effect on the cord surface. So the combined effect of flow obstruction and localized pressure drop leads to accumulation of extracellular fluid in the cord. As per his opinion dilated caudal section of the syrinx pointed to the fact that there should be a decrease in CSF pressure in the adjacent SAS region, and he supported his argument with dilation that persisted when systolic CSF flow occurred in the nearby SAS.[27,32] Thus, Venturi effect provides reasoning for the transient pressure gradient to expand the SC tissue locally, but experimental evidence was lacking and he could not provide answer for the elevated mean syrinx pressure.[29,33]

Brodbelt *et al*., suggested that syrinx formation occurred if the normal flow of CSF through the perivascular spaces was hampered or obstructed by perturbations such as an arachnoid cyst, CM, SC tethering or loss of compliance. Stoodley *et al*., found that all cystic cavities in the SC, whether canalicular or extra canalicular, are produced by this mechanism.[34] Stoodley,

Brodbelt, Bilston, *et al*. performed many *in vivo* experiments to demonstrate the movement of extracellular fluid into the SC and the central canal, showing in one study that more than a quarter of the patients with SC injury develop SM.[10,30,34-40] The extent of oedema is related to the amount of trauma experienced by the SC.[37,38] Stoodley *et al*., proposed that the pooling of CSF in the syrinx is most likely caused by an imbalance of CSF flow moving into and out of the cyst,[41] but the passive mechanism for CSF transport could not be explained as the syrinx pressure was found to be greater than SAS pressure.[13,20,29]

Bilston, Brodbelt *et al*., conducted their reaserch to find the influence of relative phase of arterial and CSF pulsations and its effect in the perivascular spaces.[34] Arterial pulsations resulted in an active mechanism causing perivascular flow into the SC even with very low pressure gradients.[7,11,34] *In vivo* and vitro studies suggested possible fluid conduit and identified a valve-like transport mechanism.[32,34,36,39,40,41]

Greitz was of opinion that the dye tracer injection studies of Stoodley *et al*., did not prove bulk fluid motion, instead this was due to diffusion of fluid into the syrinx cavity without a preferred direction.[30] Venous congestion theory of Levine[33] proposed that CM causes obstruction of foramen magnum leading to higher CSF pulsations above the blockage. The CSF pressure in the SAS is dependent upon the venous pressure during diastole and the arterial pressure during systole and the CSF moving into the extra cellular spaces is dependent on the CSF flow waveform. If flow is obstructed at the foramen magnum the veins collapse above the blockage and increase in size below the blockage this mechanism results in venous congestion and ultimately to the formation of a syrinx but there was no experimental proof.[31,42-44]

Iskandar *et al*., reported five cases of SM due to Chiari-like pathological conditions in which hindbrain herniation was lacking and Inspite of this his patients responded to traditional posterior fossa decompression surgery.[28] Carpenter *et al*., and Berkouk *et al*. Bertram *et al*. and Cirovic *et al*., developed computational models of fluid-filled coaxial elastic tube systems similar to that found in SM.Carpenter was of opinion that elastic jump may result in transient rises in SAS pressure near a spinal stenosis site.[10,18,19,45,46]

Chang *et al*., developed an electrical circuit model.[47] Bilston *et al*., developed a computational model of arachnoiditis in PTS modelled as a porous obstruction in the SAS, and found that peak pressures in the SAS were strongly dependent on obstruction permeability.[35,48]

Hydrodynamic modelling of the pulsatile CSF flow in the spinal SAS was done by Loth *et al*. The computational model can be used to detail the spatial pressure environment, but have their own limitation due to assumptions such as axisymmetric motion and generalization of tissue properties.[49] However paper by Elliot *et al*. do not support the elastic jump hypothesis which rely on impulse generated due to cough as a mechanism for the pathogenesis of SM.[50]

Discussion

First of all we must acknowledge the fact that all most all humans are born normal except those born with dysraphism/congenital anomalies. Since we do not find congenital syrinx. We must admit the fact that these syrinx develop over period of time and to begin with all are normal. So there must be some chronic recurrent event occurring over period of time to cause syrinx. Hence the pathophysiology must be linked with some normal activities which when present in some special situation will lead to syrinx. Authors have tried to link the phenomenon of syrinx to the most commonly performed action right from birth till death that is flexion extension, lateral bending and rotation.

Whatever may be the movement of spine in whichever position there is a definite change in the volume of the spinal cord and the spinal canal formed by the bony elements and the movement of the CSF in the above situation. Indeed this a very dynamic process which starts right from the intrauterine fetal movements till the last breath of life. Hence authors would try to throw light on the normal changes occurring in spinal canal volume, spinal cord volume, central canal volume and subarachnoid space volume in both the places one in spinal canal and another in posterior fossa. And their impact over period of time, I would like to first discuss about the various volumes of spinal cord, spinal canal and central canal. Dynamic changes occurring in spinal canal formed by the bony elements viz. let volume in neutral position be V. As per Breig A *et. al*. on hyperextension there is narrowing of the spinal canal by shingling the laminae and buckling of the ligamentum flavum so volume Ve in extension will be less than that in flexion i.e. Vf.[51,52] During flexion cord stretches and it shortens and thickens with extension. There is change in both length and circumference of the cervical spinal cord between extended and flexed positions.

Ko *et al*., showed that the transverse (TS) diameter is largest at segment C5 and it decreases progressively to segment T8. However sagittal diameter of each segment does not change significantly.TS diameters of the segments determines the cervical and lumbar enlargements. The Variability of the TS diameter is more prominent than that of the sagittal diameter at each segment level. So transverse diameter is a more significant measurement for the cross-sectional area.The cervical and lumbar enlargements are more dependent on the TS diameter than that on the sagittal diameter. Segment C5 has the largest cross-sectional area of 75.0 mm^2. Segment T6 is the longest, averaging 22.4mm in length. The longest segment of cervical spinal cord is segment C5 which is 15.5 mm and

segment L1 in the lumbar spinal cord. The volume is largest at segment C5 with a value of 1173.9 mm^3.[53]

Sherman *et al*., reported that cervical enlargement is usually not visualized on sagittal images because it is present mainly in the axial plane, and it maybe seen on coronal images.[54] There is clear evidence provided by MRI study done by Kuwazawa *et al*., for the fact that significant differences occur in anterior cervical spinal cord circumference (ACSCC) in extension, neutral and flexion. In the recumbent and erect series, ACSCC is larger in extension than that in neutral and flexion at all levels. ACSCC was measured at following levels C2/3, C3/4, C4/5, C5/6, and C6/7 disc levels.[55,56]

Chu *et al*. described a similar changes in spinal cord shape between thoracic adolescent idiopathic thoracic scoliosis and normal subjects measured as the ratio of antero-posterior to TS diameter of the cord.[57,58]

Breig *et al*., showed through cadaveric studies transverse sections of the spinal cords at T3 level in forward flexion and extension showing the rounder shape of the cord in extension. Normal adult spinal canal is elongated during forward flexion and the neuraxis is axially stretched due to its cranial and caudal points of fixation and, when extended backward it is slackened. *In vivo* human cervical spinal cord is subjected to deformation and displacement in flexion.[51]

Yuan, Dougherty, Margulies has concluded in his paper that between a neutral posture and full flexion, the entire cord (C2-C7) elongated linearly with head flexion, increasing 10% and 6% of its original length along the posterior and anterior surfaces.[59]

Smith CG *et al*. pointed in monkey that during flexion cervical cord stretches most (24%) and least in the caudal cord (4%). During flexion below the mid cervical level movements in flexion is towards the head and above this level is away from it. At each segment of cord, the stretch of the cord is proportional to the amount of flexion at the joint immediately ventral to it, which was found to be greatest in the lower cervical and upper thoracic segments, and up to 24% in amount and an average value of 10 movements of the head and spine. Movement was found to be greatest between roots C8 to T5 which was ranging up to 1.8 cm and this was associated with increase in the length of the spinal canal during flexion thereby stretching of the cord and dura, predominantly between levels of roots C2 to T1. Greatest stretch was found in approximately in the same region with a maximum of 17–6% with an average value of 10%.[60]

Holmes A *et al*., has also concluded that changes in cervical canal spinal volume during *in vitro* flexion-extension. The average change in volume of the spinal canal with flexion-extension motion was found to be 1.9 ml. A linear correlation with regard to dynamic canal width and also with the total angle of flexion or extension.[61] The above fact has been verified by axial computed tomography scans which shows significantly decreased canal area during extension, midsagittal diameter, and sub articular sagittal diameter, while flexion has the opposite changes.[62-64] Similarly foraminal dimension undergoes similar changes with the cross-sectional area 12% greater for the flexion group and for the extension group 15% smaller than the cross-sectional area of the neutral group. Impact of these changes happening during flexion and extension was 11 degrees at C2-C3, 12 degrees at C3-C4, 15 degrees at C4-C5, 19 degrees at C5-C6, and 20 degrees at C6-C7. During flexion the ventral SAS is narrowed up to 43% and dorsal SAS is widened up to 89% if it was compared with the neutral position, 0 degrees. During extension, the ventral SAS is increased in the diameter up to 9% while the dorsal SAS is reduced to 17%. During flexion, sagittal diameter is reduced in the cervical cord by 14%, and on the extension it increases up to 15% compared to the neutral position (0 degrees). Authors would like to draw attention on the naturally occurring disease process Hirayama flexion myelopathy in which there occurs loss of attachment from posterior dural sac and subjacent lamina shows the above-discussed changes which attests, natures confirmation of the above phenomenon.

Total volume of the cranial cavity viz. supratentorial and infratentorial/posterior fossa remains constant however as per the literature reviewed above there is change in spinal canal volume during flexion, extension and in neutral position. For the purpose of simplified understanding lets assume that volume of spinal cord is Vsc and volume of spinal canal V in neutral position, volume of central canal Vcc, volume of spinal subarchnoid space Vsas, volume of posterior fossa Vpf and the volume of spinal canal in flexion Vf and volume in extension be Ve. Now, based on these abbreviations we shall try to establish the relationship between these volumes changes during various positions of neck viz. flexion and extension.

$$V = Vcc + Vsc + Vsas \qquad \text{(Eq 1) neutral position}$$

As per paper published by various authors, we have come to conclusion that the spinal canal volume in flexion, that is, Vf is greater than volume in extension Ve. Hence,

$$Vf > Ve \qquad \text{(Eq 2)}$$

$$Vf > V > Ve \qquad \text{(Eq 3)}$$

Now, we also know that the CSF SAS is blocked by various causes listed by various authors. Hence, the spinal subarchnoid space that is, Vsas becomes isolated from the general circulation of the CSF. Hence, we come to conclusion that during flexion and extension, the equations will be as under:

$$Vf = Vccf + Vscf + Vsasf \qquad \text{(Eq 4) volume in flexion}$$

$$Ve = Vcce + Vsce + Vsase \qquad \text{(Eq 5) volume in extension}$$

But, Vf is always greater than Ve. Hence, once the SSS is blocked due to any cause, then Vsas will always remain constant in any neck position. Viz flexion, extension and neutral position. So, considering this fact, we arrive at a conclusion that

$$Vsasf = Vsase \quad \text{(Eq 6)}$$

Hence, considering equation 4, 5 and 6, we arrive at conclusion that in case of any postural change in volume Vsas is not available for any volume change in case of SM where CSF SAS s are occluded. So, the only way available for the volume change in spinal canal contents will be by the spinal cord volume Vsc and volume of central canal Vcc. However, we understand by the common logical thinking that the Vcc change is more rapidly possible than the change in spinal cord volume *per se*. Hence its quite natural to conclude that central canal cavitation is the best possible mechanism to compensate for the rapid changes in volume disturbance occuring due to spinal movement while hernation of the tonsils into the spinal canal is compensatory mechanism/physiological mechanism considering the volume compensation required from the posterior fossa.

Now consider that ΔVfe be the volume change happening during flexion and extension.

Hence

$$Vf - Ve = \Delta Vfe \quad \text{(Eq 7)}$$

ΔVfe is the volume change that is happening since birth during any movement of flexion and extension. This has to happen over either small period of time or shall be sustained over a prolonged period of time depending upon the posture taken by the individual while carrying out any activities.

Very important thing to notice is that volume change during either flexion or extension is not uniform throughout the spinal cord. It is maximum at the lower part of the cervical region of the and the lumbar spinal cord.

So ΔVfe is volume of CSF exchange required during various movements viz. flexion and extension. So extra ΔVfe extra volume has to be compensated from anywhere. But, the only way for the exchange to happen is by exchange of CSF but rapid exchange of volume by the solid spinal cord is not possible. If the CSF exchange is to happen, it has to happen by open CSF SAS and open central canal.

So the amount of CSF in posterior fossa SAS has to be equal to or more than the exchange volume ΔVfe if normal dynamics has to take place.

Always in normal condition

$$\Delta Vfe \leq \Delta Vpf \quad \text{(Eq 8)}$$

Now consider that CSF SAS is blocked at the foramen magnum level then we will be in a situation where the free CSF exchange is not possible then in that situation ΔVfe is isolated. However, if there is increase in spinal canal volume by ΔVfe but posterior fossa CSF SAS volume will not be available for the exchange due obstruction hence the only way available for filling up of the increased space would be by the increase in central canal volume by movement of CSF through the central canal. But this volume change will occur maximum at the site where there is maximum volume change in cervical and lumbar spinal cord. So the CSF will accumulate maximum in the cervical and the lumbar regions as by law vacuum cannot be there even if there is an increase in spinal canal volume with regard to the spinal cord volume. The only matter available for the rapid change in volume would be CSF. Hence in this situation spinal cord will try to maintain constant volume by cavitation and accumulating CSF in those regions. This is the reason why syrinx starts in the cervical region and lumbar region in case of tethering of spinal cord.

We need to understand that if the SSS is blocked then in that situation with increase in spinal canal volume there is decrease in SSS pressure rather than increase in CSF SAS pressure as postulated by other authors explaining the fact that there is very good reason why the there should be dilatation of the central canal. So if pressure inside the central canal is Pcc and pressure inside spinal subarachnoid space be Psas then we have

$$Pcc > Psas \quad \text{(Eq 9)}$$

But if CSF SAS is getting obstructed intermittently in that situation there would time when CSF SAS would try to reach equilibrium hence these situation the dilatation of the central canal would reach the maximum size of the cord but not beyond the diameter of spinal cord.

The fluid accumulated at any place would also have its own weight. Hence, this weight would increase if the column of syrinx increases in length. But in this situation, the syrinx should be bulging in the lower part rather than being of uniform length. But the bulging can be due to the weight of the fluid more than the CSF subarachnoid pressure or the pressure in the SSS is less than that of the syrinx fluid column. Now if in some way the SSS is able to communicate with the cranial SAS then the Pascals law would be obeyed and the undue bulging of the spinal cord due to the syrinx would not be found as the pressure equilibrium will be achieved both inside and outside.

SM is surgical condition with treatment directed towards by normalizing the CSF pathways rather than drainage of the cavities. Drainage operations involves myelotomy and has high failure and complications rates. Best treatment strategy is re-establishment of a cisterna magna and normalisation of craniospinal pressure dissociation. This is achieved surgically by artificially creating a large cisterna magna even if it means resecting part of the herniated cerebellar tissue and utilising dural grafts which means making provision for the

space which in turn will lead to effective volume changes. Surgery for SM is not easy, there are risks associated with the procedure however if hydrocephalus exists its treatment is always priority.[65]

This brings us to conclusion that if the above hypothesis be true then it opens another door/avenue for the management of patient with syringomyelia that is immobilisation. Since the volume changes are related to the spinal movement, immobilisation would result in complete cessation or at least minimising the volume changes there by minimising neural tissue damage resulting from the syrinx. Goel *et al*., has published his revolutionary procedure with the hypothesis that Chiari Malformation patient with or without basilar invagination and or syringomyelia is primarily related to atlantoaxial instability and such cases should be treated with atlantoaxial stabilisation and segmental arthrodesis.[66,67] His postoperative outcome with no neurological deterioration and clinical improvement attest to the above fact that the immobilisation may be another solution to this problem and another evidence in favour of this hypothesis.

Conclusion

Spinal canal is most dynamic part with volume changes starting right from intrauterine part till the end. Although volume of the cranial cavity remains constant but the volume of spinal cord varies. Volume of spinal cord increases during flexion and it decreases during extension. This difference in volume between flexion and extension is always compensated by posterior fossa volume. In cases of small posterior fossa or any obstruction at foramen magnum level which hampers this volume exchange required during various postural changes. This is compensated readily by cavitatory/cystic (syrinx) changes within the spinal cord at locations which are more prone to rapid volume changes in cervical and lumbar spine. This also points to the beginning of these changes i.e., syrinx formation in cervical region area with maximal diameters. As rapid compensation can be only accomplished by means of syrinx formation. However in long run tonsillar herniation in to the spinal canal is the long-term solution by nature for the uncompensated state of volume exchange which appears like pathological in nature but in reality serving great physiological function by making itself available the for the volume exchange depending on the spinal volume changes. During flexion the ventral SAS is narrow and dorsal SAS is widened as compared with the neutral position however during extension, the ventral SAS is increased while the dorsal SAS is reduced.

These volume changes explain the fluid movement in spinal canal towards the dorsal SSS during flexion and later to the ventral subarachnoid space during extension. These volume changes relate to the fact that treatment may be directed towards normalizing the CSF pathways either by restoring the normal volume by means of posterior fossa decompression or else immobilisation should also help as per this hypothesis as it causes cessation of volume exchange leading to cessation of neural tissue damage due syrinx which is supported by immobilisation surgery for SM in the literature. Thus, the role spinal canal volume changes in the pathogenesis of syringomyelia has been discussed here and it appears to be one of the contributing factors in the pathogenesis of syringomyelia along with other factors.

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How to cite this article: Rai SR, Rai PS. Volume change theory for syringomyelia: A new perspective. Asian J Neurosurg 2015;10:245-51.
Source of Support: Nil, **Conflict of Interest:** None declared.