Intracranial Pressure or Intracranial Venous Output Resistance. Part 2: Theory of Pathological Variations.

Iqtidar H.[1] Bhatti, Zanib Javed,[2] Muhammad Shahzad Shamim[3]

Abstract

An intracranial space occupying lesion (SOL) has only weight and volume, and does not generate a pressure directly because of its presence or growth. It evolves in the incompressible tissue environment by displacing, parallel with its growth, an equal volume of one or more intracranial contents 'extracranially', directly or indirectly, into the dural venous sinuses. The three displaceable intracranial contents are brain tissue fluid, CSF, and blood in the bridging veins and have different rates and resistances to extracranial output. Brain tissue fluid has the slowest rate but the lowest resistance to output. An SOL must grow by displacing the lowest resistance content if its rate of growth is less or equal to the rate of output of brain tissue fluid. Only a very slow growing lesion such as an osteoma or a meningioma evolves by displacing brain tissue fluid. Intracranial venous flow resistance (VFR) and intracranial venous output resistance (VOR), or dynamic ICP (DICP), remain unaffected. Faster growing SOL displaces CSF parallel with its growth. Again VFR and VOR do not rise. An acutely expanding SOL such as a parenchymal haematoma, subarachnoid haemorrhage or acute hydrocephalus can evolve only by displacing venous blood, which has the fastest output rate but the highest output resistance. Consequent narrowing of cortical veins elevates VFR and VOR (DICP). VOR elevates also if SOL obstructs dural venous sinuses. An SOL causes brain herniation by depleting one or more of displaceable intracranial contents across the midline or a foramen but elevates VOR only if there is cortical venous blood depletion with elevation of VFR. Intracranial haemorrhage displaces an equal volume of intracranial venous blood reducing intracranial arterial input, The ruptured vessel or aneurysm stops bleeding not because of a rise in ICP, which in keeping with Pascal's Law is uniform in all intracranial contents, but because of a reduction or cessation of input of blood to the arterial bed. All neurological signs are the result of regional or global ischaemia and have nothing to do with intracranial pressure.

[1]Professor Emeritus-Jinnah Postgraduate Medical Centre, [2]Chief Resident, Neuro-Surgery, Aga Khan University Hospital, [3]Muhammad Shahzad Shamim: The Abdulali D. Charolia Endowed Professor and Head of Neurosurgery, Aga Khan University Hospital, Karachi

Correspondence: M. Shahzad Shamim.**Email:** shahzad.shamim@aku.edu

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Introduction

The most common clinical application of intracranial pressure (ICP) and its effects is nowadays seen in patients suffering from traumatic brain injury (TBI) and stroke.[1-3] In high-income countries, ICP monitoring is now a standard component of TBI management guidelines and a requirement for trauma center accreditation by the American College of Surgeons.[4,5] For stroke, several randomized controlled trials have enabled us to make guidelines to control ICP, whether it is from an ischaemic or haemorrhagic stroke.6 Acute increase in ICP as a result of TBI or stroke is somewhat better understood, as we can see the effects of direct intervention in these cases almost immediately.[5,6] What has been more difficult is to understand the effect of slowly increasing ICP, and how the brain is able to adapt to it, as is seen in the cases of brain tumours or hydrocephalus.[7]

In this paper, we present to the readers interesting theoretical explanations of the pathological variations of ICP, by Professor Iqtidar H. Bhatti. Originally written nearly 40 years ago, this explanation has been recently updated with references to more recent texts. We have not altered Professor Bhatti's original text but have simplified some of it using tables and charts, with his approval. It is important to note that these theories have not been experimentally tested by the authors. While we encourage researchers to critically analyze and investigate them further, until validated through empirical studies, they should be regarded as theoretical concepts.

Professor Iqtidar H. Bhatti is a revered pioneer in the field of neurosurgery and a prominent figure in Pakistan's medical community. Widely celebrated for his contributions, Professor Bhatti played a vital role in establishing neurosurgery departments at several leading tertiary care centres in Karachi, greatly enhancing access to specialized care. His interest in Cerebrospinal Fluid (CSF) dynamics lead him to his most remarkable

innovation, the Bhatti Shunt and Bhatti CSF Access Chamber, which was the first implantable neurosurgical device developed in Pakistan. This low-cost system not only ensures continuous CSF drainage but also allows direct ventricular access for antibiotic administration. As a testament to his services, Professor Bhatti was honoured as the first neurosurgeon in Pakistan to receive the prestigious Tamgha-e-Imtiaz (TI).

Theory of Pathological Variations

The term SOL (space occupying lesion) in this paper refers to all mass lesions and includes brain oedema, intracranial haemorrhage and hydrocephalus. An SOL grows by displacing one or more of displaceable intracranial contents 'extracranially' into dural venous sinuses directly or indirectly parallel with its growth. The three displaceable intracranial contents are brain tissue fluid, CSF and cortical venous blood. These contents exit their respective compartments against different resistances and at different rates. Brain tissue fluid (including pathological displaceable contents of brain tissue compartment such as products of lysis of brain and blood cells) has the lowest output resistance for it leaves its compartment directly via venous capillaries. However, its output rate, defined as the maximum volume displaceable from a unit area in a unit time, is the slowest of the three contents because physiological output of tissue fluid from arterial capillaries and its input to venous capillaries, in tissue perfusion, are balanced and the capacity of the latter vessels to take additional fluid is limited. CSF, the second displaceable content, leaves its pathways to enter the venous system indirectly via arachnoid villi and other routes of exit. It encounters greater resistance passing through these routes than brain tissue fluid does in entering venous capillaries. The output rate of CSF, however, is faster than that of brain tissue fluid because it has several exit routes and the capacity of these routes to transfer it 'extracranially' is about five times that of choroid plexuses to form the fluid.8 Venous blood, the third displaceable intracranial content, encounters the highest output resistance, which is the sum of intracranial venous flow resistance (VFR) and dural venous sinus pressure (DVSP). Its output rate via bridging cortical veins into the 'extracranial' dural sinuses for anatomical reasons, however, is the fastest of the three displaceable contents.

Consonant with physical laws, a space occupying lesion grows in an adult rigid cranium by displacing the content that offers the least resistance to extracranial output provided that the content has the capacity to exit its compartment at a rate less than, or equal to, the rate of growth of the lesion. Osteomas of the inner table of the skull, and very slowly enlarging tumours, cysts and hydrocephalus evolve by displacing the contents of brain tissue fluid compartment. Such lesions do not reduce the volume of blood in the bridging veins to raise VFR. Intracranial venous output resistance (VOR) which is the sum of VFR and DVSP, therefore, remains unchanged. A tumour that grows slowly but at a rate faster than the output rate of brain tissue fluid displaces 'extracranially' an equal volume of CSF that has a higher output resistance than brain tissue fluid but a faster output rate. Such a lesion narrows or obliterates CSF pathways but also does not raise VFR. Increase in VFR occurs when an SOL enlarges so rapidly that only an equal volume of venous blood, the content with the fastest output rate, can move 'extracranially' to accommodate the increase in the volume of the lesion. Thus, acute hydrocephalus, haemorrhage and oedema develop in the incompressible intracranial environment solely by displacing an equal volume of blood from the bridging veins to the dural venous sinuses.[9] Consequent narrowing of bridging veins, consonant with Poiseulles law, not only elevates VFR but also increases its pulse synchronous variation (ICP pulse).

An SOL does not usually increase in size at a constant rate or grow at the expense of a single displaceable intracranial content. A slowly enlarging intracranial tumour that grows by displacing brain tissue fluid initially may deplete the capacity of brain tissue compartment to accommodate its growth at a later stage. It then starts obliterating the CSF pathways parallel with its growth. During these phases of tumour enlargement VFR is unaffected. When the CSF can deplete no more or the tumour develops acute increase in volume because of oedema, necrosis or haemorrhage, the blood in bridging veins exits 'extracranially' to accommodate the rapid change in volume. VFR elevates and the resultant reduction in intracranial systolic input of blood heralds the stage of 'decompensation' in the evolution of the tumour.[10]

Neurological symptoms and signs associated with an SOL are due to ischaemia or infarction and not to pressure or brain herniation.[11] The pressure within the dural compartment is merely the unspent fraction of intracranial arterial blood pressure (ICABP), equal to VOR, that fails to effect arterial input of blood to the brain and, in keeping with Pascall's law, is transmitted uniformly through all intracranial contents where it is called dynamic intracranial pressure (DICP). A deep-seated or infiltrating SOL may grow by displacing blood out of the veins in its vicinity without affecting the volume of venous blood in the bridging veins. When this occurs, VFR

does not rise and VOR (DICP) is unaffected. Although the total arterial input of blood to the brain does not reduce, depletion of venous bed near the SOL reduces systolic arterial input to the region so depleted. Focal neurological symptoms and signs appear because of regional ischaemia. Periventricular ischaemia in normal pressure hydrocephalus and ischaemia around infiltrating tumours with symptoms and signs of neurological dysfunction, without a rise in VOR (DICP), are examples of such focal or regional vascular effects.[12]

Brain herniations indicate that the volume of displaceable intracranial contents has depleted across midline, partitions or foramens to accommodate the growth of an SOL. These are not due to any differentials in intracranial pressure because pressure is uniform throughout the intradural compartment. A slowly growing supratentorial SOL located away from the midline increases in volume by displacing 'extracranially' an equal volume of brain tissue fluid and CSF of the ipsilateral hemisphere without affecting VFR. After attaining a certain volume, it starts growing by displacing the same contents of the contralateral hemisphere. As a result, midline shift or herniation occurs without a rise in VFR. When the SOL starts reducing the volume of venous bed, however, the midline shift becomes associated with a rise in VFR and a fall in the input of blood to the brain. If it continues to grow at the expense of venous blood, it finally depletes the cranium of all displaceable blood in the bridging veins and VFR and VOR become absolute. In the incompressible tissue environment, intracranial arterial bed stops expanding in systole and input of blood to the brain ceases altogether. The entire magnitude of IABP remains unspent, equals VOR (DICP) and is uniform throughout the intradural compartment. Absence of input of blood to the brain leads to brain death. In slowly evolving non-communicating hydrocephalus with obstruction at or proximal to the fourth ventricle exit foramina, the ventricular system proximal to the obstruction may attain a large size by displacing fluid out of the brain tissue compartment, and CSF out of the extraventricular CSF pathways distal to the obstruction. On imaging, the brain appears 'tight' because the expanding ventricles obliterate the subarachnoid spaces flattening the sulci.[13,14]

VFR is not affected and total systolic input of blood to the brain is unchanged. However, regional periventricular ischaemia producing symptoms of generalized motor slowness may occur. When CSF starts accumulating in the ventricles by displacing venous blood from cortical veins 'extracranially', VFR elevates, reducing input of blood to the brain. If the process is untreated or does not become 'arrested', it progresses ultimately to global ischaemia and widespread infarction. Slowly evolving communicating hydrocephalus begins to deplete cortical venous bed earlier than does an equally slowly evolving non-communicating hydrocephalus.[15] The reason is that the CSF pathways distal to the ventricular system, being part of the hydrocephalic process in the communicating variety, cannot reduce in volume to allow excess CSF to accumulate in the ventricles as occurs in the non-communicating variety. Thus, symptoms and signs of reduced input of blood to the brain usually appear earlier in communicating hydrocephalus, with smaller ventricular size, than in the non-communicating variety. In acute hydrocephalus, CSF accumulates rapidly in the ventricular system by displacing an equal volume of blood in the bridging veins 'extracranially'. VFR elevates quickly leading to rapidly progressive global ischaemia.

Brain oedema of gradual onset that accompanies many slowly growing benign and malignant tumours evolves usually by displacing an equal volume of brain tissue fluid and CSF from its periphery and does not raise VFR.[16] If it does not produce regional ischaemia, neurological symptoms and signs do not appear. Brain oedema of acute onset on the other hand progresses rapidly by displacing regional and cortical venous blood. It produces neurological symptoms and signs because of regional and global ischaemia. Elevation of VOR (DICP) secondary to elevation of DVSP as occurs in pseudotumour cerebri does not produce regional cerebral ischaemia and is therefore not associated with focal neurological signs. In dural sinus thrombosis, superior vena cava obstruction or chronically increased intra-abdominal pressure, such as occurs in morbidly obese, the cortical veins engorge because of increased 'extracranial' venous pressure giving rise to sustained elevation in VOR (DICP). The increase in the volume of the venous bed causes the CSF pathways and brain tissue fluid to reduce by an equal volume. The ventricles do not enlarge but become slit-like, and the subarachnoid spaces narrow. All patients with pseudotumour cerebri have an obstructive 'extracranial' venous etiology.[17]

An acute intracerebral haematoma forms by reducing the volume of venous blood that has the fastest output rate. VFR rises rapidly. When such a haematoma stabilizes in volume, the depleted venous bed, being a higher resistance system, reopens by reducing the volume of lower resistance compartments.[18] Although the haematoma is a potentially displaceable content of the lowest-resistance brain tissue compartment, it is not displaceable initially because, for a few days after forming, it comprises clots and unlysed cells that resist

displacement through the capillaries. The venous bed 'squeezed' by the haematoma, therefore, begins to reopen by first reducing the volume of CSF pathways. After the damaged blood and brain cells have undergone lysis, the squeezed CSF pathways, being part of a higher resistance system than the contents of brain tissue compartment, reopen and may enlarge by 'pushing' the products of lysis into the venous capillaries. Thus, simple mechanical processes governed by differences in the output rates and resistances of the three displaceable intracranial contents determine the fate of the haematoma. While the venous bed is compromised, VFR remains elevated and blood input to the brain reduced. As the haematoma stabilizes in volume and the venous bed reopens at the expense of CSF pathways, VFR begins to fall and input of blood to the brain starts to increase. This is associated with neurological improvement although the size of the lesion is unchanged at this stage. Similar events occur repeatedly during the evolution of a tumour, which changes in volume at varying rates because of episodic intrinsic and extrinsic events such as necrosis, oedema and haemorrhage.

As an intracranial aneurysm ruptures in the incompressible tissue environment and blood enters the subarachnoid space or brain tissue, an equal volume of venous blood is pushed out of the bridging veins into the 'extracranial' dural sinuses.[19] Depending upon the volume of blood entering the subarachnoid space and consequent parallel reduction in the volume of intracranial displaceable venous blood. Systolic input of arterial blood to the brain reduces or ceases altogether depending upon the volume of blood entering the subarachnoid space. The IABP that does not expand the intracranial arterial bed because of raised VFR remains unspent. It is transmitted to all intracranial contents. The aneurysm stops bleeding only because of reduced or absent input of blood to the intracranial arterial bed and not because of any extravascular pressure. In all intracranial contents, brain tissue, CSF, arteries and veins pressure is uniform and equal to VOR. If brain death does not occur immediately because of global ischaemia, arterial vasoconstriction follows and leads to parallel reopening of the venous bed and fall in VFR, which reflects increased input of blood to the brain and is associated with neurological improvement. (Table 1)

Conclusion

An SOL has weight but does not generate pressure. It grows in the incompressible environment by displacing extracranially an equal volume of one or more of three displaceable intracranial contents that have different output resistances and rates. ICVOR elevates when an SOL grows by displacing venous blood extracranially to raise VFR or by obstructing dural venous sinuses to raise DVSP. It is unaffected when the lesion grows at the expense of the other two displaceable contents. A slowly enlarging SOL may grow to a large volume by displacing brain tissue fluid, CSF or both that have lower output resistances but slower output rates than blood in the bridging veins. Acutely evolving SOL, such as intracranial haemorrhage, develops by displacing venous blood extracranially and elevates VFR. Brain herniation is associated with rise in VOR (DICP) only when the SOL depletes venous blood to raise VFR or obstructs venous sinuses to elevate DVSP. All movements of intracranial fluids and tissues in physiological and pathological states occur because of differences in their resistance to flow or movement needs further work.

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