Intracranial Pressure or Intracranial Venous Output Resistance Part I: Theory of origin and physiological variation

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Abstract

Intracranial Pressure (ICP) has two components, static or gravitational and dynamic or vascular. Dynamic intracranial pressure (DICP) is the unspent fraction (or entirety) of the intracranial arterial blood pressure and equals intracranial venous output resistance (ICVOR), which is the sum of intracranial venous flow resistance (ICVFR) in the bridging veins and the dural venous sinus pressure (DVSP). Intracranial contents possess only volume and weight, and cannot directly create or vary ICP. Contraction of voluntary, cardiac or smooth muscle is the sole mechanism for generating a pressure in the living body. All perpetually dynamic pressures in the living body, such as ICP, derive from cardiac systole, the only muscular contraction that recurs unceasingly during life, and disappear at asystole. Intracranial contents possess physical properties of fluids, are constant in volume in physiological state and are incompressible at biological pressures. Input to an intracranial content equals and parallels output from it, directly or indirectly, to 'extracranial' dural venous sinuses. The total volume of these contents has no relationship with ICP and the so-called elastance and compliance are not known physical properties of matter in the fluid state and do not, therefore, merit a role in the origin or variation of ICP. DICP varies because of changes in ICVFR, DVSP or both. ICVFR varies, in accordance with Poisieulle's equation, with passive changes in the lumens of bridging veins that occur due to opposite and parallel active changes in the lumens of intracranial arteries and arterioles. Changes in DVSP are secondary to changes in CVP or obstruction in dural sinuses. ICVOR or DICP is uniform throughout the intradural compartment and dynamic pressure gradients do not exist in intracranial contents. In the incompressible intracranial environment, blood input, flow and tissue perfusion occur because the arterial bed, from the large arteries to the ends of arterial capillaries, expands and contracts during the cardiac cycle creating equal, parallel and opposite volume changes in the venous bed, and balanced inputs to and outputs from arteries, veins and capillaries.

Keywords: Arterial blood pressure, Bridging cortical veins, Central venous pressure, Compliance, Dural venous sinuses, Elastance, Intracranial pressure, Retinal pulse

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Introduction

The concept of increasing pressure within the cranial cavity necessitating relief through trephination or skull removal has been recognized since ancient Egyptian civilization, and documented by Greek physicians such as Hippocrates and Galen. The most well-known modern explanation stems from the work of two Scotsmen: Alexander Monro in the late 17th century and George Kellie in the early 18th century, culminating in the Monro-Kellie doctrine. However, it was not until 1891 that the first method for measuring intracranial pressure (ICP) was published by the German physician Heinrich Quincke.[1-3] More than a hundred years later, despite significant research; many aspects of ICP still remain poorly understood. There are ongoing efforts to find out the most effective method of measuring ICP, both through invasive and non-invasive techniques.[4,5] At the same time, numerous studies have explored methods to control increasing ICP through medical management and surgical interventions.[6,7]

In this paper, we present to the readers interesting theoretical explanations of the origin and physiological 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

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tertiary care centers in Karachi, greatly enhancing access to specialized care. His interest in Cerebrospinal Fluid (CSF) dynamics led 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 honored as the first neurosurgeon in Pakistan to receive the prestigious Tamgha-e-Imtiaz (TI).

Theory of origin and physiological variation

During the past hundred years, many investigators have studied intracranial pressure (ICP) extensively. Yet the mechanisms of this dynamic pressure remain poorly understood. Present views attribute the origin and variation of ICP to certain intracranial volume-pressure relationships, dynamics of intracranial blood and CSF and physical properties of intracranial contents called elastance and compliance.[8-27] These views lack a rational basis and are in conflict with physical laws. Intracranial contents, physiological and pathological, possess only volume and weight, and cannot generate a dynamic pressure. The sole mechanism for generating a dynamic pressure in the living body is contraction of smooth, cardiac or voluntary muscle, and no such primary mechanism exists intracranially. ICP is the unspent fraction of intracranial arterial blood pressure transmitted, across the wall of the intracranial arterial bed, uniformly throughout the cranial intradural compartment. It is equal to, and determined by, cranial venous output resistance, which is the sum of resistance to flow of venous blood through the bridging veins and pressure in the dural venous sinuses.[28-32] While the dural sinuses are anatomically intracranial, the pressure within these rigid preformed structures is independent of ICP and reflects central venous pressure (CVP).[33] These sinuses are therefore 'extracranial' for the purpose of this paper.

All perpetual dynamic tissue pressures in the living body, such as ICP, arise from the arterial blood pressure (ABP) and disappear with cardiac asystole. ABP expands the arterial bed as it fills it with blood. This systolic arterial expansion, extending from the aorta to the end of the arterial capillary, is the most vital and perhaps the most ignored event in peripheral circulatory physiology. In its absence, input of blood to organs and tissues does not occur. When arterial expansion is unrestricted, the entire magnitude of ABP is spent within the arterial bed. If the tissue environment resists arterial expansion partially (or totally), a fraction (or entirety) of ABP equal to the resistance remains unspent within the bed and transmits across its wall into the surrounding tissues. Thus, the environment of the arterial bed finally controls the input of blood to organs and tissues, and determines the magnitude of unspent ABP transmitted to it. If we disregard gravitational effects, the pressure in the vascular bed from the large arteries to the capillaries and veins in each organ of the body is uniform and equals the resistance offered by the tissue environment to arterial expansion in that organ.

ICP has static or gravitational, and dynamic or vascular components. Static ICP (SICP) is the weight of intracranial contents and persists uniformly during life and after death. It adds to the dynamic component a magnitude that varies with the weight of intracranial and spinal contents at the site where the pressure is measured. Dynamic ICP (DICP) arises from the intracranial arterial blood pressure (IABP), varies continuously during life and disappears with cardiac asystole. The fact that DICP disappears with the cessation of cardiac activity is proof of its origin from the intracranial arterial blood pressure.

Water occupies 90 percent of the volume of craniospinal axis. The brain parenchyma alone, excluding CSF and blood, contains about 80 percent water.[34] Intracranial contents therefore have the physical properties of fluids and are incompressible at biological pressures. Kellie, in 1821, confirmed the thesis of James Munro that intracranial contents were incompressible, volume of intracranial blood and other contents was constant and input to the rigid cranium equaled output.[35] His observations became the Munro-Kellie law. Subsequently, the law came in conflict with the prevalent views that intracranial contents were compressible and changes in the volume and so-called elastance and compliance of these contents varied ICP.[36-40] It therefore became necessary to modify the law and relegate it to the status of a hypothesis or doctrine. Laws of physics, however, fully support the Monro-Kellie Law in its original form and confirm that all intracranial contents are incompressible at biological pressures, and that their volumes are constant in physiological states. Thus, biological input to the rigid cranial cavity equals and parallels output from the cavity to the 'extracranial' dural venous sinuses.[41] In physiological states, the volume of each intracranial content remains constant at all times over short and long periods, barring the process of ageing that leads to brain atrophy. This indicates that the vascular bed, CSF pathways and brain tissue compartment have independent balanced circulations. In other words, systolic input of blood to the arterial bed parallels an equal output of venous blood from the bridging veins to the 'extracranial' dural sinuses. The volume of CSF that enters the ventricular system, 'forces' an equal volume of fluid to leave the CSF pathways via the arachnoid villi and other routes of CSF exit.[42,43] Similarly, the volume of fluid that leaves the arterial capillary during the cardiac cycle equals the volume that

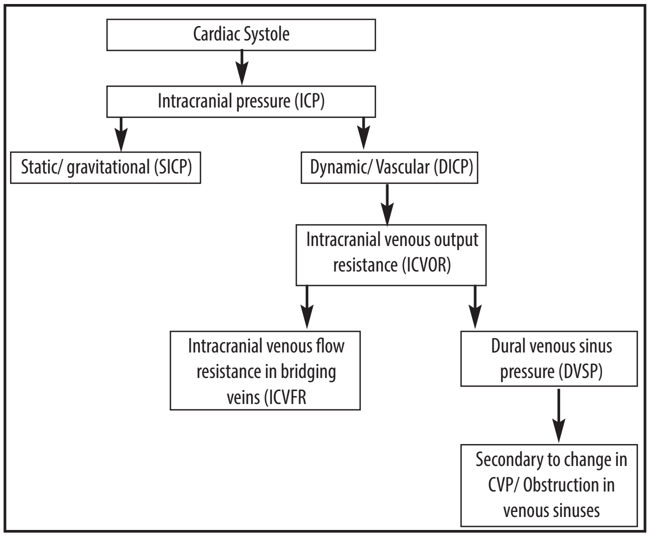

Figure-1: Theory of origin and physiological variation in Intracranial Pressure.

enters the venous capillary. The above considerations apply to the spine as well because spinal intradural contents form a continuum with the intracranial contents and are equally incompressible at biological pressures (Figure).

Both intraocular and intracranial vascular beds have incompressible tissue environments; have similar pressure ranges and responses to changes in central venous pressure.[44] Mechanisms of arterial input and venous output in the eye and the brain are therefore similar. In these organs, as indeed in many other organs confined within rigid capsules or 'containers' with incompressible contents, the arterial bed can expand during cardiac systole only by 'squeezing' the venous bed parallel with its expansion thereby creating blood flow and venous return. Evidence of the existence of this arterial-venous relationship is visible in the fundus of the eye. In physiological states, the retinal veins exiting at the optic disc pulsate spontaneously.[45,46] The veins narrow as the intraocular arteries expand with systolic input of blood. Similarly, in the cranium, bridging veins narrow during systole to accommodate intracranial arterial expansion and blood input.[47] Narrowing begins at the optic disc ends of the retinal veins and at the dural venous sinus ends of the bridging veins. It progresses proximally for a variable distance. Distal to proximal narrowing occurs because the venous intraluminal resistance to flow of blood is lowest distally and as arterial expansion proceeds during systole, consistent with physical laws, parallel venous 'squeezing' begins at the lowest resistance ends of veins and progresses proximally. In diastole, the elastic arteries and arterioles narrow progressively. The contracting arterial bed pulls open the venous bed, which fills with blood. Reopening of the narrowed bridging veins during diastole also follows physical laws. It begins at the proximal lowest resistance ends of the narrowed venous segments and progresses distally. This distal to proximal emptying and reverse filling of veins, synchronous with changes in cardiac cycle, is usually visible in the retinal veins of the normal eye on fundoscopy.

Resistance to flow of blood through bridging veins rises progressively during systole as venous blood passes through increasingly narrowed venous segments and falls in diastole as venous lumens enlarge to fill with blood. (Pouisielle's Law). This intracranial venous flow resistance (ICVFR) together with the 'extracranial' dural venous sinus pressure (DVSP) constitutes intracranial venous output resistance (ICVOR) in this paper. A fraction (or entirety) of IABP equal to ICVOR fails to expand the arterial bed and remains unspent. This unspent fraction (or entirety) of IABP transmits across the wall of the arterial bed into the surrounding brain and CSF where it is called ICP.[48] ICVOR or DICP varies continuously. Most constant variation occurs synchronously with changes in cardiac cycle.[49] Rise in ICVFR during systole and fall in diastole constitutes the so-called ICP pulse. Other variations in ICVOR occur spontaneously and with changes in respiratory cycle. These variations are secondary to changes in arterial vasomotor tone and in DVSP. Arterial dilatation leads to parallel venous contraction and increase in ICVFR. Thus, ICVOR and the amplitude of pulse synchronous variation in ICVFR (ICP pulse) increase. Rise in CVP (and DVSP) during expiration and Valsalva maneuvers increases the resistance to 'extracranial' output of cortical venous blood and also increases ICVOR (DICP) but the pulse synchronous variation in ICVFR (ICP pulse) diminishes in amplitude because rise in DVSP distends cortical veins and prevents or reduces systolic cortical venous narrowing. Continuous simultaneous graphic records of ICVOR (DICP) and CVP confirm the occurrence of these intracranial pressure variations.

In physiological states, the volume of intracranial blood remains constant. Changes in the volume of arterial bed induced by variations in motor tone create parallel, equal and opposite passive changes in the volume of venous bed. The arterial changes occur spontaneously and in response to local, humoral and neural stimuli. Arterial constriction causes parallel venous dilatation with fall in ICVOR because ICVFR reduces for two reasons. Firstly, arterial constriction dilates the venous bed and thereby reduces systolic narrowing of cortical veins. Secondly, a given IABP creates less unit arterial expansion in a bed that is constricted or 'tight' than when it is dilated, and so the parallel systolic narrowing of bridging veins is reduced. Thus, vasoconstriction not only reduces ICVOR and pulse synchronous variation of ICVFR (ICP pulse) but it also reduces input of blood to the arterial bed unless there is a rise in IABP to increase the systolic expansion of the 'tight' arterial bed.[50-52] Arterial dilatation on the other hand forces an equal volume of cortical venous blood into the dural sinuses and narrows the lumens of bridging veins. ICVOR (DICP) increases because of a rise in ICVFR. Arterial dilatation therefore increases the unspent fraction of IABP and the pulse synchronous variation of ICVFR (ICP pulse). Although the fraction of IABP available to expand the arterial bed reduces with arterial dilatation, systolic blood input may not reduce because a smaller magnitude of pressure creates unit expansion in a 'relaxed' or dilated arterial bed than in a 'tight' or constricted one.[53]

Variations in ICVFR secondary to spontaneous rhythmic changes in arterial vasomotor tone give rise to the physiological intracranial pressure oscillations or the so-called B waves of Lundberg.[54-58]

ICVOR varies in physiological and pathological states because of changes in ICVFR, DVSP, or both, affecting volume of intracranial arterial input and magnitude of unspent fraction of IABP. Its variation is unrelated to a primary volume-pressure effect that implies compressibility of intracranial contents. These contents have constant volumes, cannot be compressed biologically and lack a primary mechanism for generating pressure. Furthermore, intracranial contents possess the physical properties of fluids and neither elastance nor compliance is a valid physical property of matter in the fluid state.

In physiological states, greater magnitude of IABP dissipates in expanding the arterial bed. The fraction that remains unspent is present uniformly throughout the intracranial contents including the entire vascular bed. During the cardiac cycle, fluid moves out of the arterial capillary because some components of blood encounter less resistance in exiting the capillary than in travelling linearly through it. Fluid moves into the venous capillary parallel with the output from the arterial capillary because of the incompressible pericapillary environment and the low resistance to flow of fluid into the venous capillary. In a closed rigid cavity filled with fluid, changes in pressure at any point in the fluid transmit equally throughout the cavity (Pascall's Law). As intracranial contents have physical properties of fluids, only the unspent fraction of IABP, equal to ICVOR, is present not only in the entire vascular bed but also in all the other contents of the cranium. Therefore, dynamic pressure gradients do not form or exist in the vascular bed or anywhere else intracranially.[59] Blood flow and tissue perfusion are generated through simple mechanical action of the expanding and contracting arterial bed squeezing the venous bed in systole and pulling it open in diastole causing blood components to flow along the paths of least resistance.

Conclusion

Intracranial pressure (ICP) is fundamentally determined by the unspent fraction of intracranial arterial blood pressure (IABP) that transmits uniformly throughout the cranial compartment. This unspent pressure is directly related to intracranial venous output resistance (ICVOR), influenced by venous flow resistance and dural venous sinus pressure. The Monro-Kellie Law, in its original form, remains valid, reinforcing that intracranial contents are incompressible at biological pressures. Consequently, ICP dynamics are driven by arterial expansion and venous contraction without reliance on elastance, compliance, or primary volume-pressure effects.

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