The Pelvic Compartment as Modulator of Intracranial Pressure: The Moscote–Janjua–Agrawal Hypothesis

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Introduction

High intracranial pressure (ICP) is the leading cause of death in the patients with traumatic brain injury and contributes to the secondary brain injury if not managed correctly.[1] The Monroe–Kelly doctrine proposes that the rigid skull contains three components: blood, brain tissue, and cerebrospinal fluid (CSF). Any additional component, such as hematomas, cerebral edema, or hydrocephalus, will increase ICP once compensatory shifts in the primary components have been exceeded.[2] The ability to store up to 150 cc of new intracranial volume without a significant increase in ICP occurs through displacement of venous blood into the general circulation, and CSF displacement is time- and age-dependent.[3] Clinical studies have demonstrated that the patients with traumatic brain injury with ICP greater than 20 mm Hg, particularly when refractory to treatment, have a worse clinical prognosis and are more likely to develop cerebral herniation syndromes. Clinical studies have demonstrated that the patients with traumatic brain injury with ICP greater than 20 mm Hg, particularly when refractory to treatment, have a worse clinical prognosis and are more likely to develop cerebral herniation syndromes. There is also recent evidence that cerebral perfusion pressure below 60 to 70 mm Hg is associated with decreased brain parenchymal oxygenation, altered metabolism, and poor prognosis.[4] The goal of neuromonitoring and treatment is to maintain adequate cerebral perfusion, oxygenation, and metabolism while limiting the progression of elevated ICPs, desaturation phenomena, and edema.

Understanding Components

Venous Return and Blood Volume Regulation: The pelvic veins, particularly during postural changes or increased intra-abdominal pressure (IAP), can impact venous return to the heart. This alteration in venous return may influence cerebral blood flow and ICP.

CSF Dynamics: Changes in IAP, modulated by the pelvic compartment, might affect CSF pressure and circulation. This can occur through modifications in the spinal CSF absorption or adjustments in the spinal venous pressure.

Neuroendocrine Interactions: Hormonal changes due to pelvic organs—such as variations in cortisol, aldosterone, and renin–angiotensin system activity—could indirectly impact ICP regulation by affecting the fluid balance and vascular tone.

Autonomic Nervous System Modulation: The autonomic innervation of pelvic viscera may influence systemic vascular resistance and cardiac output. These alterations could affect cerebral perfusion pressure, thereby impacting ICP. Understanding the potential interactions between the pelvic compartment and ICP could have significant clinical implications. This knowledge may inform strategies for managing ICP in conditions such as traumatic brain injury, hydrocephalus, or during neurosurgical procedures. Future research should focus on empirical studies exploring these interactions in clinical settings, possibly through controlled experiments and longitudinal studies.

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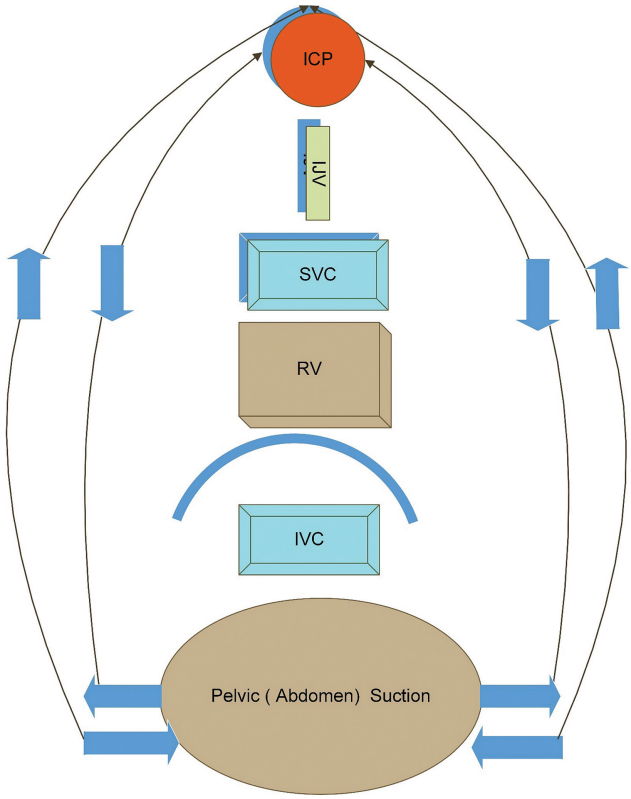

Fig. 1 Pelvic suction leading to a reduction in ICP with interaction with IVC and SVC. ICP, High intracranial pressure; IJV, internal jugular veins; IVC, inferior vena cava; RV, right ventricle; SVC, superior vena cava.

The Monroe–Kelly Hypothesis

The Monroe–Kelly hypothesis postulates that the skull is a rigid structure, a nonexpansible box, and its contents primarily consist of three components: brain tissue, CSF, and blood. To maintain stable blood pressure, the volumes within the cranial vault must remain stable. Any additional volume increases due to hyperemia or the presence of hematomas will reduce the volume of other components and lead to increased ICP (►**Fig. 1**). Approximately, one-third of patients with diffuse brain injury and half of those with intracranial masses exhibit elevated intracranial pressure. Typical adult intracranial pressure ranges from 5 to 15 mm Hg, while pediatric values range from 0 to 10 mm Hg. Persistent elevation of intracranial pressure has been associated with a poor prognosis, and mortality is directly related to the degree and duration of elevated intracranial pressure. As intracranial pressure increases, cerebral perfusion pressure generally decreases, contributing to reduced cerebral blood flow, ischemia, and neuronal death. Many clinical goals have focused on limiting the increase in intracranial pressure, maintaining adequate perfusion pressure, and aggressively avoiding ischemic situations closely related to ischemia and hypoxia. Other modalities include evacuating intracranial lesions and halting the progression of cerebral edema.

The Pelvic Compartment: An Overlooked Influence

The pelvic compartment encompasses organs and structures involved in reproductive, urinary, and digestive functions. It includes the bladder, uterus (in females), rectum, and associated vasculature and connective tissues. Traditionally, its influence on systemic physiology has been well-documented, particularly in relation to blood flow dynamics, hormone regulation, and homeostasis.[5–9]

The Moscote–Janjua–Agrawal Hypothesis

We propose (the Moscote–Janjua–Agrawal hypothesis), based on the available evidence, that the pelvic compartment represents a new link in the pathophysiological compression phenomenon of intracranial hypertension. Our hypothesis is based on physiological substrates. We believe that modulation through various strategies that reduce the volume of the pelvic compartment at the expense of its visceral content will allow the modulation of intracranial pressure in acute neurological pathologies associated with acute intracranial hypertension. It has been reported that an acute increase in IAP leads to two significant pathophysiological phenomena: an increase in intracranial pressure and a reduction in cerebral perfusion pressure. The involvement of the jugular venous system is likely linked to this alteration, possibly due to increased pleural pressure and changes in intrathoracic pressures.[10] It is interesting that in morbidly obese patients, who have an increase in IAP, a higher rate of intracranial hypertension is observed in these populations.[11] Evidence has also suggested a relationship between BMI and intraocular pressure, demonstrating the role of obesity as a modulating factor of intracranial pressure and, consequently, of intracranial pressure.[12] One key aspect of general surgery knowledge is that abdominal decompression is the standard treatment for abdominal compartment syndrome. On the other hand, acutely elevated IAP significantly increases intracranial pressure (ICP) and impairs cardiovascular and pulmonary function.[13] Evidence showed that continuous negative abdominal pressure can offer useful benefits for patients with abdominal acute pathologies. Sugerman et al[14] proposed utilizing an externally applied negative abdominal pressure device designed to lower the effects of IAP. The use of the device demonstrated promising clinical improvement.[14]

Future Considerations

Older individuals tend to have more brain atrophy and can accommodate larger volumes that expand slowly. Younger individuals with acute processes become symptomatic more quickly due to the same pathophysiological processes. Space-occupying lesions will be discussed in the subsequent section, assuming these lesions have been surgically evacuated. Abnormal cerebral autoregulation, blood flow, and cerebral edema persist as causes of elevated intracranial pressure.

Conclusion

In conclusion, while traditionally viewed as separate physiological entities, the pelvic compartment may exert modulatory effects on intracranial pressure through various mechanisms. Further exploration of these interactions could pave the way for novel therapeutic approaches and improve outcomes in patients at risk of elevated ICP. This narrative review proposes a framework for understanding these potential connections and calls for future research to elucidate their clinical relevance.

Conflict of Interest
None declared.

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