Langfitt Curve: Importance in the Management of Patients with Neurotrauma

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Traumatic brain injury is one of the leading causes of mortality and morbidity among children and young adults in western developed countries. Subsequently to the direct effects, secondary injury is characterized by the activation of different pathophysiologic pathways; intracranial compensatory mechanisms initiate immediately as the secondary injury progresses. Furthermore, intracranial hypertension leads to the development of physiologic events that are crucial for patients' clinical outcomes.

The intracranial hypertension phenomenon is of clinical significance in patients with brain injury. Brain elastance is defined based on the intracranial pressure (ICP) per volume unit, added to the cranial compartment, whereas compliance is the inverse of the elastance, defined by the change of pressure over the change of volume. Additionally, the elastance curve can be graphically expressed as the lateral changes in the ICP compared with the volume changes within the cranial compartment.

The initial phase (also known as compensatory) is characterized by low elastance and high compliance. Posteriorly, a phase of high elastance and low compliance occurs when the compensatory reserves are depleted.

Intracranial Pressure and Hypertension

Intracranial pressure (ICP) is the pressure existing in the interior of the cranial cavity, being directly related to three components:

1. Parenchymal brain structures.
2. Cerebrospinal fluid (CSF) located in the ventricular cavities and subarachnoid space.
3. Blood circulating in the intracranial compartment at a given time, as described by the Monro-Kellie hypothesis.[1]

The Monro-Kellie hypothesis defines an equilibrium state, where the fixed volume of the cranium, and its constituents, defines the incompressibility of the compartment. Therefore, differential volume increases must be compensated by the intracranial constituents.[1]

Moreover, intracranial hypertension is the leading cause of death in patients with traumatic brain injury, and it contributes to the secondary brain injury if handled incorrectly. Any additional volume, such as bruising, swelling of the neural tissue, or hydrocephalus, will result in increased ICP when the compensatory changes of the primary volumes have been exceeded. As we have discussed elsewhere[2], derivation of, up to, 150 mm^3 of intracranial venous blood into the systemic circulation, is the first compensatory mechanism for preventing the increase of ICP; complementarily, CSF shifting-out depends on disease progression and patient's age. Elders tend to exhibit cerebral atrophy, and in this sense, rearranging high volumes results in a demanding task in the context of slow expansions. Abnormal brain volumes and blood flow self-regulation and cerebral edema persist as causes of raised ICP. It is important to highlight that the relationship between the intracranial volume and pressure is depicted as a sigmoidal function rather than linear. Nonetheless, any physiological or pathological processes, and most uncompensated variations in the volume of any component, will be evident in the values of ICP.

Clinical studies have shown that patients with cranial trauma and ICP greater than 20 mm Hg have worse clinical prognosis and are more likely to experience brain herniation

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syndrome, particularly in cases with treatment-refractory injury.[3]

Recent studies have found brain hypoxia in the settings of a cerebral perfusion pressure less than 70 mm Hg.[4] Hypoxia is as well associated with the onset of aberrant metabolic routes and an increase of mortality. Neuromonitoring guides treatment preventing this sequence by maintaining brain homeostasis.

As a mass grows inside the skull, a proportional amount of CSF is withdrawn from it. During this period, the ICP does not rise. After the extrusion of the entire CSF volume, a linear relationship is expected between the intracranial pressure and volume. However, this curve actually shows an exponential trend.[5]

Specifically, Langfitt curve (ICP vs. displaced volume) can be categorically divided into four phases (►**Fig. 1**):

Phase 1 (Initial): In this phase, intracranial expansive process promotes proportional changes in CSF input and output, without an increased ICP. There is no activation of the vasodilator cascade.

Phase 2 (Decompensation): There is virtually no more CSF in the intracranial compartment. The expansion process modifies the regional perfusion causing lactic acidosis, activation of the vasodilator cascade, and increasing the blood volume within the cranium.

Phase 3 (Exponential): At this stage, there is an exponential increase in cerebral blood volume, certainly due to the vasodilator cascade effect. The cerebral blood volume increases the ICP until it equals the mean arterial pressure (MAP), tending the cerebral blood flow to 0.

Phase 4 (Final): Phase of vasoplegia. The compensatory reserve of the brain elastance (pressure/volume) curve can be estimated using a linear correlation (LC) coefficient obtained from comparing the fundamental component of amplitude of ICP pulse wave with an average volume assessed by data mining techniques and based on 40 consecutive intervals of 6 to 10 seconds.

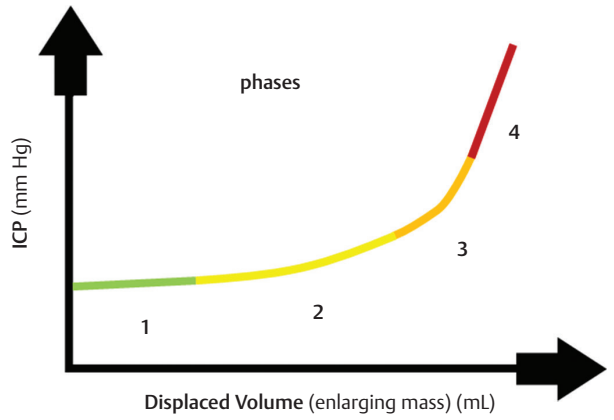

Fig. 1 Hemodynamic phases of Langfitt curve. ICP, intracranial pressure (based on concepts from Turner).[6]

An LC coefficient close to 0 indicates that there is no correlation between the fundamental component of amplitude of ICP pulse wave and the average volume. This represents the ideal compensatory damages when the values of ICP are lower; in other words, changes in the volume produce little or no variation in pressure. On the contrary, an LC coefficient close to 1 corresponds to a perfect correlation between the fundamental component of amplitude of ICP pulse wave and the mean volume. For this compensatory reserve, changes in the volume produce evident changes in pressure. If the ICP keeps increasing, the fundamental component of the pulse wave will be reduced because the capacity to direct flow from dilated capillaries into arterioles will be diminished as well. Therefore, passive collapse of vessels occurs and then the LC coefficient tends to 0.[7–10]

In the fourth phase, the end of the exponential curve, the cerebral blood volume is low, the ICP is equal to the mean arterial pressure, and perfusion pressure and blood flow to the brain are equivalent to 0. That is the brain death.

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