Cerebral Edema and Intracranial Dynamics

Monitoring and Management of Intracranial Pressure

Matthew Eccher and Jose I. Suarez

INTRODUCTION

Elevated intracranial pressure (ICP) is a relatively common clinical problem, potentially encountered daily in any neurocritical care unit. Intracranial hypertension can be a hyperacute emergency that must be reversed if profound morbidity or death are to be avoided. The astute clinician can improve patients’ outcomes if judicious steps are taken at the right time (1). There have been many advances in our understanding of the physiology of intracranial dynamics. Although our armamentarium remains fairly limited, we may begin to envision its use on a rational, pathophysiologically grounded basis. Unfortunately, too little is yet known to predict exactly which interventions will be effective in exactly which disease states exactly when. Owing in part to the limits of our current technology, but also to a regrettable dearth of clinical trials in the field, current clinical practice is based on a conceptual understanding of underlying pathophysiology but backed by insufficient systematic research with patients. Practice is inevitably the product of the idiosyncratic experience of each individual intensivist. There are insufficient hard data to guide those in the process of gaining that experience.

The goal of this chapter is to first provide an overview of our models of pathophysiology, then to highlight their application in the management of deranged intracranial dynamics. Those few circumstances in which we have objective clinical trial data to guide patient management will be highlighted.

PATHOPHYSIOLOGY

Intracranial Elastance

In all normal humans whose cranial fontanelles have closed, the intracranial contents—brain, blood, and cerebrospinal fluid (CSF)—are encased in a rigid skull; in infants and others with incomplete closure of the calvaria, cerebral expansion is limited by the fibrous dura. In the average adult male, the skull encloses a volume of approx 1450 mL: 1300 mL of brain, 65 mL of CSF, and 110 mL of blood (2).

The Monro-Kellie doctrine dictates these anatomists’ observation that the volume of the cranial vault is unchangeable—any process which adds volume to this system must therefore displace volume from elsewhere in the system. Which of the above components is displaced to accommodate extra volume will be considered below. Initially, there is minimal resistance to this displacement. When the limits of displaceability are reached, however, further addition encounters resistance, and this addition must be “squeezed” into the rigid container. This quickly results in an increase in the
pressure within the system, i.e., raised ICP, which normally ranges between 5 and 15 mmHg (7.5 and 20 cm H₂O). This relationship of ICP to increasing volume of intracranial contents (in one experimental paradigm, an expanding subdural balloon) (3) can be expressed as a graph (Fig. 1). This model is just a model, and the pressure-volume curve it generates differs in some respects from that produced by other pathophysiological processes (4), but the basic shape of the curve assumes an increasingly upsloping form in all cases. The slope represents the change in pressure produced by a given change in volume: ΔP/ΔV, termed “elastance.” Initially, with low added volumes, CSF and venous blood are highly displaceable, and pressure rises little; elastance is low. With sufficient added volume, however, compensatory fluid shifts meet with increasing resistance, and pressure rises more and more precipitously—elastance rises. A simple analogy can be made to an elastic band: initial stretch on the band elicits little tensile resistance, but with increasing displacement (stretch), the elastic exerts greater and greater resistance. Likewise, with increasing addition of intracranial displacement (volume), the intracranial contents exert greater force (pressure), resisting further addition.

For semantic and historical reasons, most clinicians describe the status of the intracranial system in terms of ΔV/ΔP, “compliance,” the inverse of elastance: a system which will accommodate significant changes in volume with little increase in pressure has high compliance (because it exerts little elastic resistance, i.e., has low elastance), whereas a system which has exhausted its compensating mechanisms can accommodate little additional volume without large changes in pressure has low compliance (increased elastance).

The Brain

The brain is a viscoelastic solid. It can be displaced to moderate degrees to accommodate an expanding mass. Slowly expanding masses can reach substantial sizes before becoming symptomatic, provided they are not primarily destructive of brain parenchyma, even if they encroach on structurally susceptible locations, such as the tentorial incisura or the foramen magnum (5). The brain’s inherent elastic properties generate pressure gradients in such situations (6,7)—gradients of up to 20 mmHg across as little as 2 cm of white matter have been reported (8), and ICP is therefore not always uniform (9). Brain is thus one source of intracranial elastance. Its inherent elastic properties can be modulated by changes in brain composition (see Brain Water/Brain Edema section) or by the addition of mass effect to the brain parenchyma (e.g., tumor, abscess).

While the glycoproteolipid matrix of the brain produces its structural integrity and elastic properties, the brain remains approx 80% water (10,11), in two compartments. The extracellular compartment represents approx 15% of brain water (10,11), and is in communication with the CSF space (as evidenced by edema bulk flow); the intracellular space comprises the other 85%. It is commonly held that neither of these spaces is appreciably compressible, with moderate direct evidence at best (11). It is clear that either or both these spaces can expand in different disease states (see Brain Water/Brain Edema section). Such expansion leads, in effect, to an expansion of the volume of the brain. If such expansion overwhelms volume-compensatory mechanisms, ICP rises.

Brain, then, is minimally compressible, minimally displaceable, and can in some circumstances expand. Venous blood and CSF, by contrast, are much more displaceable, and represent the compensatory mechanisms for increased intracranial volume.

CSF

CSF buoys the brain and cushions it. It is produced as a modulated ultrafiltrate of plasma, with tight control of electrolyte and protein content. Most (80–90%) of its production is at the choroid plexus, with the remainder at the brain capillaries as brain interstitial fluid (10–12). Roughly 500 cc is produced and resorbed each day (13). Resorption occurs at the arachnoid villi into the cerebral venous sinuses (superior sagittal and transverse) by a mechanism that remains poorly understood (14). Produc-