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Intracranial Pressure and Cerebrovascular Autoregulation in Pediatric Critical Illness

Robert C. Tasker, MBBS, MD, FRCP[*,†,‡]

Protecting the brain in vulnerable infants and children with critical illness involving the brain is a central aspect of pediatric intensive care and neurocritical care. Collectively, illness-induced derangements in intracranial pressure, circulatory homeostasis, and pressure autoregulation are all fundamental in informing bedside management. Therefore, this review provides an understanding of these entities and a physiological approach to bedside care and monitoring.
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Introduction

Central to our practice of supporting and treating critically ill children with acute brain injury is the recognition and prevention of impaired cerebral perfusion and the consequences of intracranial hypertension. In general, in patients affected with coma, clinical attendants have little to inform them of what might be occurring in their patients except data from a thorough clinical examination and bedside physiological monitoring of heart rate, blood pressure (BP), and sometimes, intracranial pressure (ICP). The purpose of this article is to review physiological interactions between these variables in the context of present day intensive care. These relationships may also emphasize which principles derived from developmental physiology or adult data are relevant to pediatric neurocritical care patients. This review therefore focuses on 3 key physiologies: (1) what we mean by the term ICP, (2) what is the role of the baroreceptors and chemoreceptors reflexes in defending brain perfusion, and (3) what is the cerebrovascular pressure autoregulation.

From the *Departments of Neurology and Anaesthesia (Pediatrics), Harvard Medical School, Boston, MA.
†Department of Anesthesiology, Perioperative and Pain Medicine, Boston Children's Hospital, Boston, MA.
‡Division of Critical Care Medicine, Boston Children's Hospital, Boston, MA.
Address reprint requests to Robert C. Tasker, MBBS, MD, FRCP, Department of Anesthesiology, Perioperative and Pain Medicine, Boston Children's Hospital 300 Longwood Ave, Bader 627, Boston, MA 02115. E-mail: robert.tasker@childrens.harvard.edu

Intracranial Pressure

Normal cerebrospinal fluid (CSF) pressure in children at the time of lumbar puncture is positive in relation to atmospheric pressure, with 10th-90th percentile of 11.5-28 cm of H_2O or 9-21 mm Hg, respectively.[1] Normal values of CSF pressure in newborns are usually less than these values, at 3-8 cm of H_2O or 2-6 mm Hg. ICP is the pressure of CSF inside the cerebral ventricles, which is determined by cerebral blood flow (CBF) and CSF circulation. The Davson equation describes this interrelationship and states that ICP is the sum of sagittal sinus pressure and the product of CSF formation rate and resistance to CSF outflow.[2] Normal values for sagittal sinus pressure, CSF formation rate, and resistance to CSF outflow are 5-8 mm Hg, 0.15-0.30 mL/min, and 6-10 mm Hg/mL/min, respectively. Measured ICP is often greater than the calculated value because of a vascular component, which is probably a result of pulsation in the arterial bed and the interaction between pulsatile arterial inflow and venous outflow curves, cardiac function, and cerebral vasomotor tone.[3]

Intracranial Content, Volume, and ICP in Health and Illness

In health, ICP also reflects the volume of 3 compartments; in the adult human, we have brain parenchyma (1200-1600 mL), extracellular or CSF (100-150 mL), and cerebral blood volume ([CBV], 100-150 mL). Because the intracranial vault is fixed in volume in the developed cranium, increases in the size of one component of the intracranial contents must be compensated by removal of an equivalent amount of another intracranial component or ICP will increase. The point at which perfusion-compromising ICP elevation occurs is dependent on brain elastance and potential displacement of intracranial contents.

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As the infant's cranium has the potential for growth with open fontanels and sutures, there is greater total compliance of the system. For example, a slow-growing central nervous system (CNS) tumor will not have an acute mass effect as compensatory increase in intracranial volume occurs with expansion (widening of sutures and fontanels) and growth. As a result, infants and young children may have advanced intracranial pathology at the time of presentation, with little reserve.

In infants and children with neurologic illness (eg, traumatic brain injury [TBI], CNS tumors, hydrocephalus, acute toxic encephalopathy, or CNS infection), cerebral hemodynamic and hydrodynamic interrelationships may be altered. For example, the presence of focal brain contusion, cerebral edema, increased CBV, and variable levels of CBF and cerebrovascular carbon dioxide (CO_2) reactivity has potential consequences on sagittal sinus pressure, CSF formation rate, and resistance to CSF outflow (mentioned previously). The net result is raised ICP along with significant risk of brain tissue herniation and ischemic syndromes, and death.[4]

Clinical Target: Level for ICP

In adults with severe TBI undergoing invasive ICP monitoring, the threshold for initiating treatment of elevated ICP is taken as 20-25 mm Hg. It is likely that the ICP threshold for poorer outcome is similar across all ages, and the recent *Second Edition of the Brain Trauma Foundation Guidelines for the Acute Medical Management of Severe TBI in Infants, Children, and Adolescents*[5] concludes that the treatment of raised ICP at a threshold of 20 mm Hg may be considered *Level III* evidence. Brief increases in ICP that return to normal in <5 minutes may be insignificant; however, sustained increases of ≥ 20 mm Hg for ≥ 5 minutes likely warrant treatment. Despite this recommendation, it should be noted that the optimal ICP target or targets for children remain to be defined, although the 90th percentile of the normal range (21 mm Hg) appears to be a good starting point.[1]

Cardiovascular Reflexes Defending Brain Perfusion

The first line of defense in preserving blood flow to the brain is the global baroreflex systems, which attempt to maintain and stabilize BP on a time scale of seconds to minutes by dynamically controlling heart rate, ventricular contractility, vascular tone and total peripheral resistance, and systemic venous unstressed volume through autonomic efferent pathways.[6,7] The heart rate baroreflex is the most extensively studied and understood of these 4 pathways because of the ease of noninvasive monitoring. The afferent inputs from cardiovascular receptors relay in the brainstem and modify the autonomic outflow to the heart and vessels.

Arterial Blood Vessel Receptors

Baroreceptors

Arterial baroreceptors in the carotid sinus and aortic arch are stimulated by the magnitude of the BP (static sensitivity) as well as the rate of increase in BP (dynamic sensitivity). Carotid baroreceptors signal the size of the pulse, that is, the pulse pressure as well as mean BP. The greater the oscillation in pressure about a given mean, the greater the aggregate activity in the nerve. As a result, a pulsating pressure elicits a greater depressor reflex than a steady pressure. The responsiveness of the reflex to pulsation is partly due to the dynamic sensitivity of baroreceptors, partly due to recruitment with each systole, and partly due to the adaptation of brainstem neurons to a sustained signal.[8]

The signaling of pulse pressure is important during orthostasis and moderate hemorrhage, when reduced stroke volume is often associated with a reduced pulse pressure but little or no decrease in mean BP. During hypovolemia, baroreceptor unloading elicits tachycardia, increased contractility, vasoconstriction and venoconstriction, interstitial fluid reabsorption, and renal fluid retention (via renin-angiotensin-aldosterone stimulation and antidiuretic hormone suppression of diuresis). These responses help to maintain BP and cerebral perfusion. The optimal sensitivity or *gain* of the reflex is the maximum slope of the response curve (ie, heart rate vs effective carotid sinus pressure). In humans, the gain is reduced by ageing and chronic hypertension because the elasticity of the artery wall declines. The *set point* is the pressure that the reflex strives to maintain. This can be altered by neural interactions with the CNS (central resetting) or by physical changes in the receptor region (peripheral resetting). *Resetting* enables the baroreflex to buffer pressure around different steady-state pressure levels, which may occur during exercise (ie, central resetting) or during pathologic levels in BP such as clinical hypertension (ie, peripheral resetting). Baroreceptor sensitivity (BRS) is determined by the slope of the regression of simultaneous changes in BP and heart rate (Fig. 1).

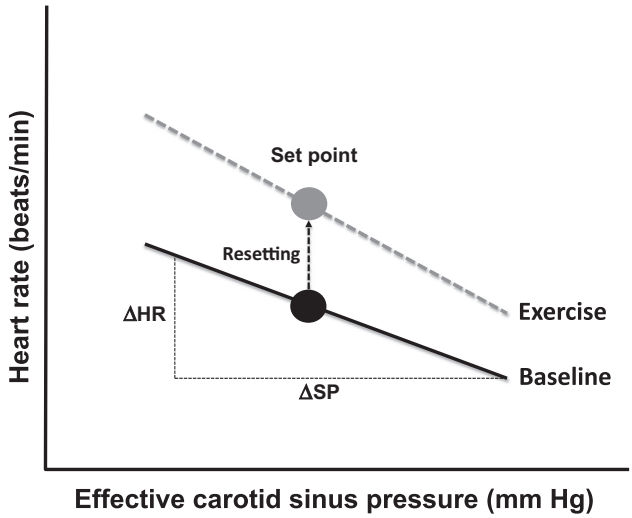

Figure 1 Resetting of the baroreflex with change in set point. Baroreceptor sensitivity is defined by the slope for change in heart rate (HR) vs change in pressure (P), $\Delta HR/\Delta P$.