Popular Misunderstandings in Neuroanesthesia

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PERSPECTIVE
I have committed considerable professional time to anesthesia for neurologic surgery. That commitment has caused me to hear a great deal related to neuroanesthesia. I have been asked by faculty colleagues in anesthesia, “What do you suppose might have gone wrong during that case yesterday?” I have listened to American Board of Anesthesiology oral examination candidates misapplying or frankly misunderstanding fundamental principles. I have been involved in third-party scrutiny of procedures that were perceived to have gone astray. I acknowledge that those interactions inform the selection of topics that follows (rather than “evidence-based” material from the peer-reviewed literature). Not all of the topics will be addressed during the course of the presentation.

THE LOWER LIMIT OF CBF AUTOREGULATION
Diagrams that appear in standard texts have frequently depicted the lower limit of human cerebral blood flow (CBF) autoregulation (LLA) as being a mean arterial blood pressure (MAP) of 50 mmHg. While this number may in fact be a reasonable representation of the LLA in several animal species, it is unlikely to be an accurate value in adult humans. The first rendering of a CBF autoregulation curve was probably that drawn by Lassen.1 His diagram depicted a LLA that might be easily interpreted to be 50 mmHg. On close inspection, however, the inflection point is probably 60 mmHg. However, the inflection point on that hand-drawn curve (wherever it is) is anchored by only 2 CBF values, both of which were obtained in pregnant females at term in whom arterial blood pressure was decreased using cerebral-vasodilating drugs and in whom baseline pressures were probably well below the population average for normal adult humans. Furthermore, numerous subsequent investigations2 (Drummond3 for additional references) suggest that the LLA in nonanesthetized adult humans is nothing less than 70 mmHg. However, it should be acknowledged that the “rules” might be different during general anesthesia for at least 2 reasons. The first is the frequent inclusion of vasodilating substances in anesthetic recipes. Vasodilators might serve to shift the autoregulation curve in a leftward direction. The second resides in the observation that sympathectomy in both experimental animals and humans during hypotension increases CBF.4 This suggests that the normal autonomic response to hypotension includes some vasoconstriction of large extracranial and perhaps intracranial vessels thereby producing effective right shifting of the autoregulation curve. If a general anesthetic were to effectively prevent that autonomic response, it is possible that some resultant left shifting of the curve might occur. The reality, however, is that there has been exceptionally little systematic study of normal (noncerebrally injured) adult human cerebral autoregulation during anesthesia. The only context in which extensive study has occurred is during cardiopulmonary bypass (typically involving hypothermia, nonpulsatile flow, relative anemia and high-dose narcotic anesthesia). In those circumstances, which are very poorly representative of the physiology that prevails during the majority of general anesthetic states, the LLA is in fact about 65 mmHg (with a very large confidence interval indicating considerable interindividual heterogeneity).5 However, it seems inappropriate to extrapolate that average value (obtained in the context of nonpulsatile flow, low hematocrit and well maintained cardiac output) to all other anesthetic circumstances. In fact, it further seems likely that what pertains to any one anesthetic circumstance, e.g., spontaneous ventilation during anesthesia with a volatile drug, might not be relevant in another, e.g., total IV anesthetic with remifentanil and propofol. We know very little about the LLA during general anesthesia in humans and conservative assumptions should be made in the absence of more detailed knowledge.

THE PHYSIOLOGIC CENTRAL NERVOUS SYSTEM BLOOD FLOW RESERVE
Many clinicians may well respond to the preceding discussion of the LLA with their own observation that numerous patients in the span of their experience have tolerated MAPs in the 40s, 50s and 60s, i.e., well below the proposed LLA of 70 mmHg. That is inevitably true. Patients tolerate arterial blood pressures below the LLA because there is a substantial central nervous system (CNS) blood flow reserve. CNS flow can decrease by approximately 40% of baseline values before symptoms of ischemia begin to occur.6–8 That reserve is, in essence, a physiologic buffer that protects patients in the event of hypotension. However, it is important that clinicians recognize the situations in which that buffer may not be present, often because it has been encroached upon by some preexisting pathologic process. The most common situations in which the buffer is likely to have been attenuated occur in circumstances in which CNS tissue is under increased pressure. This may occur in the circumstances of increased intracranial pressure (ICP), increased ocular pressure or when CNS tissue is under extrinsic pressure, e.g., compressed under retractors or by a bulging disc. The significance of these situations is that the principal determinant of flow to the tissue is “transmural pressure” rather than blood pressure. Transmural pressure (which is commonly but probably erroneously referred to as “perfusion pressure” equals MAP minus local tissue pressure. Among the most commonly overlooked situations in which tissue pressure is increased (and the effective perfusing pressure is therefore less for a given value of MAP) is in the circumstances of spinal stenosis, in particular cervical spinal stenosis. In that group of patients, the normally wide latitudes for intraoperative blood pressure that anesthesiologists commonly allow should be tightly

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restricted. It is our approach at the University of California San Diego to maintain MAPs during anesthesia in these patients (at least until the decompression is complete) very close to normal waking levels. The drug we use most commonly to achieve this is phenylephrine. This introduces another common misconception, which is addressed in the second paragraph below.

THE EFFECT OF HYDROSTATIC GRADIENTS ON CEREBRAL PERFUSION PRESSURE

In patients who undergo anesthesia in horizontal positions (supine, prone, lateral), it is standard to measure arterial blood pressure with cuffs or transducers at the level of the heart. When positions are used that result in a vertical height difference between the height of the heart and the head, a pressure differential between the two that is equivalent to the weight of a column of blood of that height can be expected to occur. That gradient will be equal to approximately 2 mmHg for each one inch of height difference. The standard teaching in neuroanesthesia has long been that arterial blood pressure should be transduced at (or an arithmetic correction imposed to correct to) the level of the external auditory meatus (EAM). Clinicians who are unfamiliar with the use of the sitting position have occasionally failed to make this correction in transducer height, or have raised it only to the level of the heart with sometimes severe adverse consequences for the perfusion of the brain and/or the cervical spinal cord. This issue has been popularized recently in the context of injuries occurring in the so-called “beach-chair position.” A minority have disputed this notion, arguing that a siphon-like mechanism maintains CBF in spite of reductions in cerebral perfusion pressure calculated in the manner above. Unless and until there is wider proof of that concept, conventional hydrostatic gradient concepts should apply, and arterial transducers should be raised to the level of the EAM or arithmetic corrects should be applied to cuff pressures in order to “think” in terms of blood pressure at the EAM.

ALPHA 1 AGONISTS AND CEREBRAL VASOCONSTRICTION

It is often asserted that the various α1 agonists are significant CNS vasoconstrictors. While that may be so in canines, it is not true in humans. See Miller’s Anesthesia, 6th Ed., Ch. 21, p 818 for references. (The references were regretfully omitted in the corresponding section in the 7th edition – Ch. 13, p 311.) In human investigations done many years ago, alpha one agonists were infused directly into the cerebral circulation in concentrations sufficient to produce substantial increases in systemic arterial blood pressures; and no changes in CBF were observed. The concern that phenylephrine is a CNS vasoconstrictor has too often restricted its use in situations where there was a pressing need to augment cerebral perfusion pressure. Clinicians should “get it out of their heads.” Phenylephrine is not a significant CNS vasoconstrictor in the doses that we commonly use. When arterial blood pressure support is warranted in patients who have sustained subarachnoid hemorrhage (SAH) or traumatic brain injury or when CNS structures are under compression (spinal stenosis, retractor pressure), after assuring appropriate volume status and depth of anesthesia, phenylephrine is a reasonable choice!

THE EFFECT OF VOLATILE ANESTHETICS ON CBF

Figures that appear widely in standard texts indicate that the common volatile anesthetics (isoﬂurane, sevoﬂurane and desﬂurane) cause little or no increase in CBF at sub-minimum alveolar concentrations (MAC). In the majority of elective neurosurgical patients that is almost certainly true. In fact, in subjects with generally normal cerebral physiology, CBF actually decreases from the awake state to reach a nadir in the vicinity of 0.75–1.0 MAC. Thereafter, CBF increases in parallel with increasing end-tidal concentrations of volatile anesthetics. This superficially unusual biphasic pattern is almost certainly the product of the very substantial suppression of cerebral metabolic rate (CMR) that occurs with the initial exposure to volatile anesthetics. The reduction in CBF is probably largely a “coupled” reduction in the CBF reverting as a consequence of the reduction of CMR. The important issue for clinicians is that in patients in whom CMR has already been depressed by either pathologic processes or CMR-suppressing drugs (benzodiazepines, narcotics, propofol) or who have sufﬁciently disordered physiology that the coupling mechanism may not be functional, volatile anesthetics may act as potent vasodilators even at the sub-MAC concentrations that are normally associated with a reduction in CBF. The consequence for the clinician is that in patients with badly impaired intracranial compliance (or, as some would say, “elastance”) especially in whom those in whom CMR is already depressed, volatile agents should be introduced very cautiously. Ideally, in those extreme circumstances, they should probably not be introduced unless ICP is being monitored or until the cranium is open and the brain can be observed directly.

OBSTRUCTION OF VENOUS DRAINAGE

The venous side of the cerebral circulation is a passive, but relatively large intracerebral compartment. It is quite commonly the cause of increased ICP or “tightness” in the surgical field and is relatively under-recognized. The cerebral venous drainage is easily obstructed by anything that puts pressure on the underlying jugular veins including circumferential ties and cervical collars. Extremes of head position can also obstruct venous drainage. Any rotation of the head that is sufficient to put tension on the sternocleido-mastoid muscles is sufficient to compress the underlying jugular vein. In addition, the jugular veins drain downstream into the chest. Accordingly, anything that increases intrathoracic pressure can impair cerebral venous drainage. This includes a medley of common entities including coughing against an endotracheal tube, kinking of the endotracheal tube, bronchospasm, and pneumothorax. Confirmation of the patency of the jugular venous system and verification of normal airway pressures are accomplished easily and should be the first things that the clinician does when evaluating increased ICP or a tight surgical field.

HYPERVERVENTILATION

It has become a well-established concept that the vasoconstriction associated with hyperventilation has the potential
to cause sufficient cerebral vasoconstriction to result in ischemia when imposed on the low-flow circumstances that can prevail after acute cerebral injuries, in particular head injury\textsuperscript{12} and SAH. Routine hyperventilation in neuroanesthesia and neurosurgical critical care has ceased. While prophylactic hyperventilation is never inappropriate, hyperventilation is by no means totally “verboten.” Hyperventilation remains an adjunct in the management of patients with critically increased ICP at risk of herniation in whom other measures, short of barbiturate coma, have proven inadequate. Its use should be as brief as allowed for by patient circumstances.

**TENSION PNEUMOCEPHALUS**

Tension pneumocephalus is a phenomenon that can occur when gas is trapped within the intracranial space with no communication to the outside atmosphere. The phenomenon is widely associated with the sitting position and many clinicians associate its occurrence with the use of N\textsubscript{2}O. It most certainly is a phenomenon that can be both caused or exaggerated by N\textsubscript{2}O and many clinicians will have decided to omit N\textsubscript{2}O from the anesthetics used for posterior fossa procedures done in the sitting position as a result. However, the assumption that one no longer needs to be concerned about tension pneumocephalus if one has made the decision to omit N\textsubscript{2}O is an erroneous one. Clinically significant, and even life-threatening, tension pneumocephalus can occur in the absence of the use of N\textsubscript{2}O. Imagine a situation in which a craniotomy has been performed in a head-up posture with the craniotomy located such that a significant portion of the cranium is above the surgical site. With optimal venous drainage, mannitol administration, hyperventilation, the use of anesthetic drugs that reduce brain bulk and opening of the arachnoid membrane resulting in drainage of cerebrospinal fluid, a substantial potential space can occur between the surface of brain parenchyma and the highest point of the skull. That space will fill with air. When the procedure is concluded and the patient is restored to a near supine position, venous blood, arterial blood, cerebrospinal fluid and extracellular fluid all begin to return. Albeit that the oxygen is absorbed quickly from the air within the cranium, the remaining nitrogen can be a substantial and unyielding “mass” that will diffuse away only very slowly (over a period of days).\textsuperscript{13} Frontal craniotomies performed in a brow-up position in which the frontal bone is removed and replaced, are particularly prone to the development of tension pneumocephalus in the immediate postoperative period. Tension pneumocephalus is an under-appreciated and under-recognized cause of postoperative delayed awakening, delirium and nonawakening. When it is suspected, the diagnosis can be made by a cross table lateral radiograph or, more commonly in these days, readily available computed tomography. The treatment entails a twist drill hole and dural perforation, ideally performed by the surgeon.

**THE BRAIN PARENCHYMA IS INSENSATE BUT THE CRANIAL NERVES ARE NOT**

When Wilder Penfield performed the brain surface stimulation surveys that lead to the development of the now familiar homunculus diagrams, the craniotomies were performed under local anesthesia. While the meninges have some innervation and required gentle handling and some local anesthesia at the skull base, brain stimulation and/or resection of brain parenchyma required no anesthesia whatsoever. The brain parenchyma is insensate. Accordingly, when a general anesthetic is used for intracranial neurosurgery, the intracranial portion requires only “light” anesthesia. An error occasionally made by clinicians is the failure to anticipate stimulation of the extra-axial but intracranial portion of cranial nerves. The issue arises most often in the context of procedures formed in the vicinity of the fifth cranial nerve. The fifth cranial nerve subserves the sensation from the entire face and mouth. Stimulation of the extra-axial portion of the fifth cranial nerve can result in very sudden arousal. In circumstances in which this has occurred in nonparalyzed patients, the arousal has resulted in sudden straining against the endotracheal tube with herniation of brain around retractors and around the edges of the bony craniotomy. Substantial injury to brain parenchyma and adverse neurologic events have occurred. Where feasible, patients should be maintained paralyzed during surgery in this vicinity of Cranial Nerve V. When patients are not paralyzed, clinicians should be very attentive to the possibility of arousal and should be ready to deepen anesthesia at a second’s notice, e.g., a syringe of induction drug should be maintained in line at all times.

**STRIDOR/NARROW AIRWAY**

Swelling and expanding hematomas occurring after carotid endarterectomy or anterior cervical discectomy/fusion procedures have the potential to encroach upon the extra-thoracic airway. The clinician should keep several principles in mind. 1) The airway will always look much worse on the inside than it does on the outside. Swelling of periglottic structures is a larger component of airway compromise than is mechanical encroachment. I suspect that the enlarging mass impairs lymmphatic and venous drainage. When one visualizes in the airway, there is often remarkable swelling of periglottic structures. 2) Stridor is a late (and ominous sign). Delaying in the face of progressive enlargement of the neck until stridor occurs increases the likelihood of extreme difficulty in securing the airway. 3) Racemic epinephrine (or Heliox) may “buy you time” in the event of respiratory compromise, but they should not be viewed as cures. When an airway has become sufficiently narrow to produce stridor and labored respiration (and concomitant turbulent flow), very small increases in airway diameter will reduce airway resistance enough to mitigate symptoms. But the clinician should assume that progressive swelling will occur if the mass lesion is not relieved and distress will recur.

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