

Cerebral Perfusion Monitors

*This handout contains a written transcription of the narration in the online presentation (video).
Please review the online presentation for additional material including interactive multimedia content, audio, and practice quizzes.*

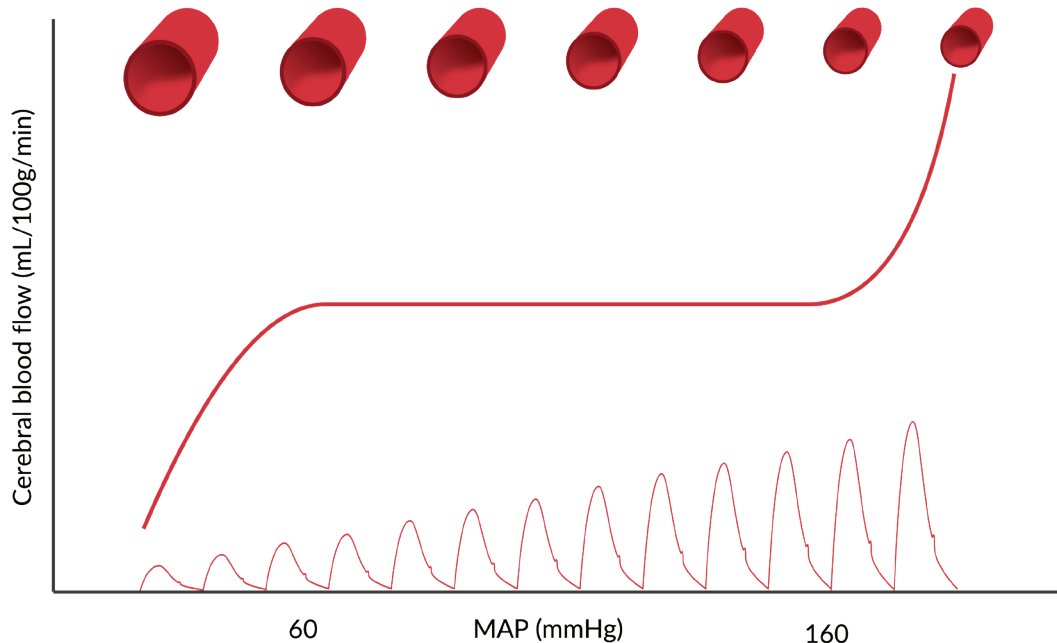
Case Study

Today, Morgan is in the ortho room where she's scheduled for several shoulder arthroscopies under general anesthesia and interscalene nerve blockade. The surgeon says the first case is complicated, and it'll take longer than usual. The patient is in his late sixties and has a history of chronic hypertension and insulin-dependent diabetes. Like always, the surgeon will use the beach chair position, and he'll frequently remind Morgan to "get the pressure down" to reduce bleeding that obscures his arthroscopic view.

Morgan has long held dear the tenets of safe anesthesia, and she's mindful that she must preserve cerebral blood flow throughout the procedure. Other than blindly assuming that the patient's cerebral autoregulation is intact, how can Morgan ensure adequate cerebral perfusion for this patient? Which technologies are available to assist her? What are the pros and cons of these monitors? In this objective, we're going to review the physiology of cerebral autoregulation and follow with a discussion of cerebral perfusion monitoring techniques and their clinical implications.

Cerebral Autoregulation: Part 1

A 1959 paper synthesized the findings of over 200 clinical investigations with the goal of detailing the complex process of cerebral autoregulation. We can define cerebral autoregulation as the brain's ability to maintain a constant cerebral blood flow over a wide range of mean arterial blood pressures. The benefit of this process is that it ensures that the brain has a steady supply of oxygen and substrates in the face of blood pressure fluctuations that accompany everyday life. Classic doctrine suggests that the brain autoregulates cerebral blood flow between a MAP of 60 – 160 mmHg (this is represented by the plateau on the curve).

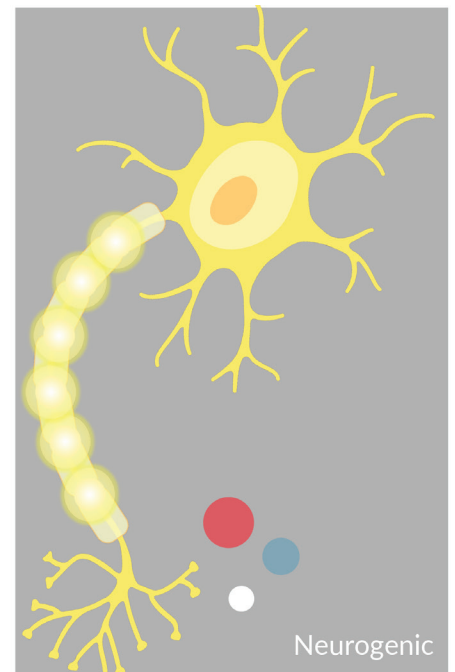
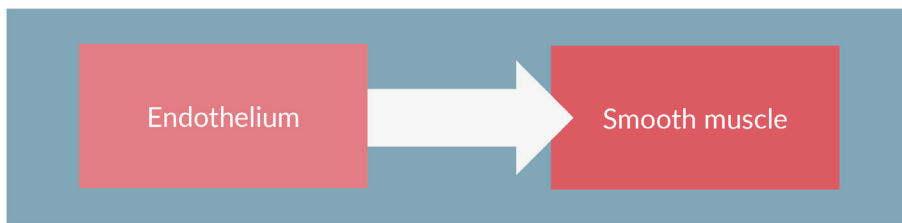
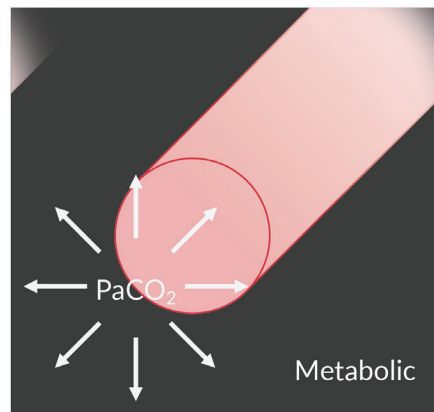
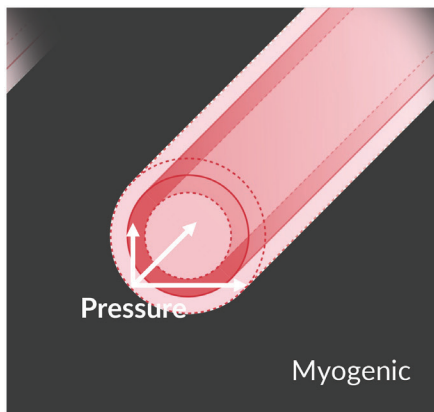


Below the lower limit of autoregulation, the cerebral vessels are maximally dilated. Cerebral perfusion becomes dependent on mean arterial pressure, and the brain is at risk of hypoperfusion and ischemia. Above the upper limit of autoregulation, the cerebral vessels are maximally constricted. Again, cerebral perfusion becomes dependent on mean arterial pressure, but this time the brain is at risk for cerebral edema and hemorrhage.

Newer evidence reveals that the limits of autoregulation vary from patient to patient. For instance, in cardiac surgery patients, investigators measured the lower limit of autoregulation (LLA) in the range of a mean arterial pressure from 43 – 90 mmHg. For any given patient, the range of cerebral autoregulation can be affected by the aging process, medical therapy, pathophysiology, and anesthetic technique. For instance, chronic hypertension shifts the curve to the right. The benefit is that this protects the brain from a higher mean arterial pressure, however this comes at the expense of being less tolerant of hypotension. As an aside, it's important to realize that some sources list MAP on the x-axis while others list cerebral perfusion pressure (CPP). As a reminder, cerebral perfusion pressure is the difference between MAP and ICP or CVP (whichever is higher), so keep this in mind as you progress through this objective.

Cerebral Autoregulation: Part 2

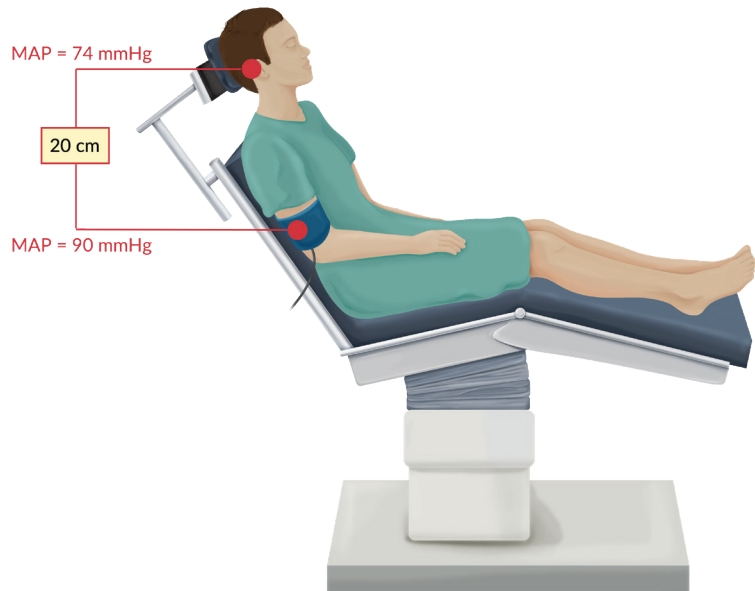
How does the cerebral vasculature autoregulate? While an in-depth look at this physiology is beyond the scope of this objective, we can broadly describe it as the summative effect of four mechanisms. There's a myogenic mechanism where the forces exerted by pulsatile blood flow cause cerebral arterial vessel diameter to increase or decrease. There's a metabolic mechanism that responds to PaCO_2 , PaO_2 , neural substrate concentration, and accumulation of metabolic byproducts. An endothelial mechanism involves secretion of nitric oxide, endothelin-1, and thromboxane A2. Finally, the neurogenic mechanism controls moderate- and smaller-diameter vessels by releasing vasoactive neurotransmitters.



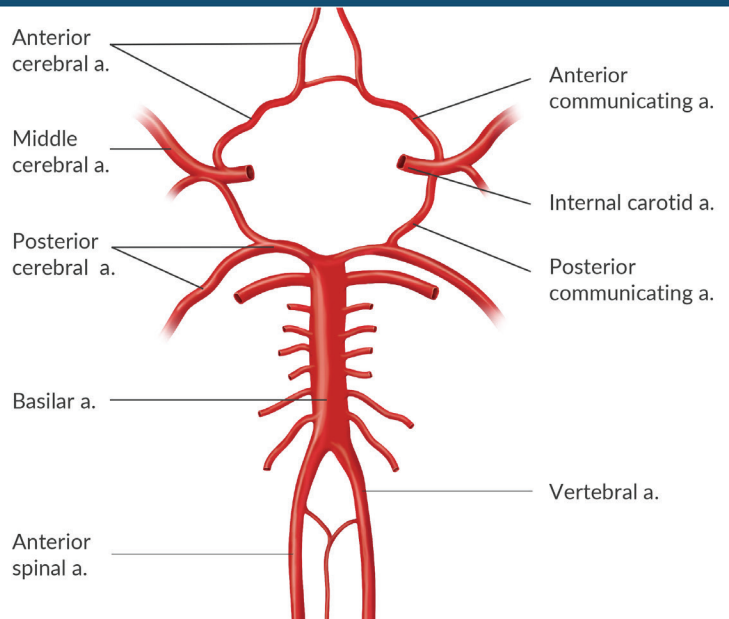
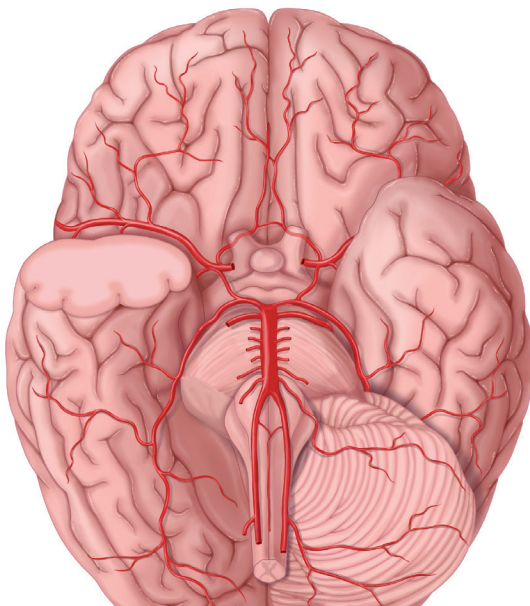
The Problem With The Beach Chair Position

What is it about the beach chair position that puts the patient at risk for cerebral ischemia? When the patient is in the supine position, the mean arterial blood pressure at the level of the heart and the level of the brain is the same. In the beach chair position, however, vertical blood flow (blood traveling from the heart to the brain) must overcome the influence of gravity. Indeed, the mean arterial blood pressure decreases proportionately to the weight of the vertical column of blood. For every 1.25 cm difference in height, we can estimate a 1 mmHg reduction in mean arterial pressure.

Let's examine this in context. Assume the patient is in the beach chair position and the distance between the site of blood pressure measurement (let's say the upper arm) is 20 cm below the external auditory meatus. We choose this structure because it corresponds to the level of the circle of Willis. If the MAP on the monitor is 90 mmHg, then the blood pressure at the circle of Willis is ~ 74 mmHg. And to that end, if the MAP is below the lower limit of cerebral autoregulation, then the pressure in the higher cortical structures will be even less. On top of this, you must factor in the influence of ICP on the cerebral perfusion pressure.



Circle of Willis



How do we monitor cerebral perfusion in the operating room? What technologies are available, or in development, to assist us with clinical decision making?

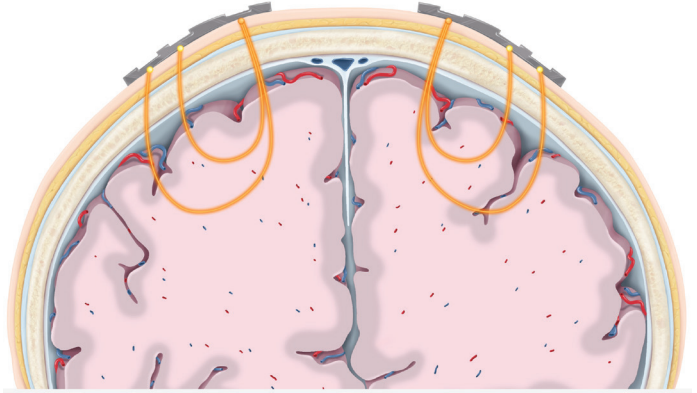
There are two approaches that we can use to monitor cerebral perfusion in the operating room. First, we can look at oxygen delivery and utilization with near-infrared spectroscopy or tissue oxygen partial pressure monitors. These methods are predicated on the assumption that a “normal” value at the site of measurement can be generalized to other regions of the brain. Second, we can measure blood velocity with transcranial Doppler. This technique is grounded in the notion that a “normal” value suggests there’s enough blood flow to satisfy the brain’s metabolic needs. Let’s take a few moments to explore each of these technologies.

Cerebral Oximetry

How Does It Work?

Near-infrared spectroscopy (NIRS) is a noninvasive technique that calculates regional cerebral oxygen saturation (rScO₂). How does it work? Adhesive pads are placed on the scalp over the frontal lobes. Similar to the pulse oximeter, NIRS is based on the Beer-Lambert law where it compares the optical characteristics (i.e., absorption of infrared light) of arterial hemoglobin, venous hemoglobin, and tissue cytochromes. Unlike the pulse oximeter, cerebral oximetry relies on the fact that ~70% of the blood in the brain resides in the venous

circulation. Since NIRS does not detect pulsatile blood flow, it’s primarily a measure of relative changes in venous oxyhemoglobin saturation as a function of oxygen delivery and consumption in the sampled area. If cerebral oxygen delivery decreases, the brain will try to compensate by extracting more oxygen, and we’ll observe a decreased cerebral oximetry value on the monitor.



How Do You Use It?

Ideally, a baseline rScO₂ value is obtained before the patient receives any anesthetic agents, but certainly a value should be obtained prior to induction of general anesthesia. A normal rScO₂ value is in the range of 60 – 80%, however in some patients (such as those undergoing cardiac surgery), a range of 55 – 60% is generally accepted as within normal limits. It’s best not to interpret rScO₂ in isolation, but rather to examine the trend relative to the baseline value over time. Furthermore, rScO₂ is best interpreted in the context of other clinical data. There is ample evidence that in select cases cerebral oximetry has the potential to be an important safeguard of cerebral function.

How Do You Treat A Low Value?

Currently there is no consensus driven approach to guide therapy in the setting of a desaturation event. Having said this, we’ll cite an example of an approach used in a recent study involving high-risk cardiac surgery patients. The following treatment algorithm was initiated when the rScO₂ fell 10% (or more) from baseline:

- Verify head position and equipment-related factors that may impair blood flow to the head.
- Is the patient hypotensive? Treat the blood pressure and find the etiology.
- Is the SaO₂ low? Increase the FiO₂ and find the etiology.
- Is the PaCO₂ < 35 mmHg? Correct hyperventilation.
- Is the patient anemic (Hgb < 7 – 8 g/dL)? Consider erythrocyte transfusion.
- Is there a problem with ventricular function or SvO₂? Optimize cardiac function and venous return.
- Is there an increased cerebral oxygen consumption? Rule out seizures, hyperthermia, and increased intracranial pressure. Treat as needed.

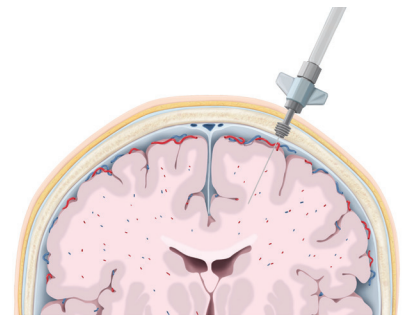
What Are The Limitations?

Cerebral oximetry is not a perfect tool at present and the technology has well defined, and clinically significant, limitations:

- Current oximeters only measure regional brain oxygenation in the area below the sensors. Thus, the best we can do is extrapolate $rScO_2$ to the rest of the brain.
- Current monitors cannot identify a cause for a desaturation event.
- Unlike pulse oximetry, where there's a consensus on abnormal values and how to treat them, treatment guidelines are not established for cerebral oximetry.
- Extracranial blood contamination (e.g., generalized hypoxemia) will contaminate measures made by the cerebral oximeter, thus current devices may not be measuring oxygenation of brain tissue alone.
- Current oximeters are sensitive to a variety of electrosurgical interferences.
- Devices from different manufactures use different algorithms for their calculations, so reported values from different devices are not necessarily equivalent.

Tissue Oxygen Partial Pressure Monitors

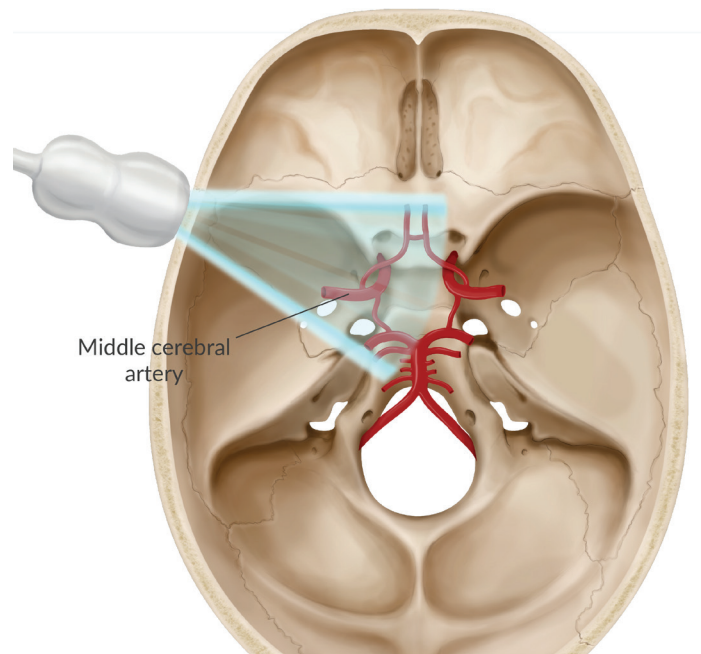
In contrast to cerebral oximetry, brain tissue oximetry is an invasive technique that monitors oxygen tension in the brain. A burr hole is created, and a catheter is placed in the subcortical white matter. Local oxygen tension is monitored with either a Clark electrode (like the oxygen analyzer in the breathing circuit) or an infrared sensor. Its invasive nature introduces several risks including infection (such as meningitis or ventriculitis), bleeding, cerebrospinal fluid leak, technical difficulty with placement, and patient discomfort. Since it's a monitor of regional oxygen tension, we can only use this data to infer conditions in the rest of the brain.



Transcranial Doppler

Instead of monitoring oxygen delivery and consumption, we can use transcranial Doppler to evaluate cerebral perfusion in real time. A common technique is to monitor the middle cerebral artery by placing an ultrasound probe on the temporal bone above the zygomatic arch just anterior to the tragus of the ear. This technique assumes that middle cerebral artery diameter stays constant even if there are fluctuations in MAP or arterial blood gas tensions. Thus, a reduction in cerebral blood velocity is assumed to be the result of decreased cerebral perfusion pressure and not an increased cerebral vascular resistance. Having said this, there is literature that calls this assumption into question.

Transcranial Doppler can also be used to monitor blood velocity in the other cerebral vessels as well as to diagnose cerebral vasospasm, stenosis, hemorrhage, and emboli. The major disadvantage of transcranial Doppler is the measurement angle of the Doppler probe must stay in a fixed position, which can be difficult during surgery. Additionally, a trained technician is required to obtain reliable data. An inadequate acoustic window in the cranium (because the temporal bone is too thick) is encountered in approximately 20% of patients.

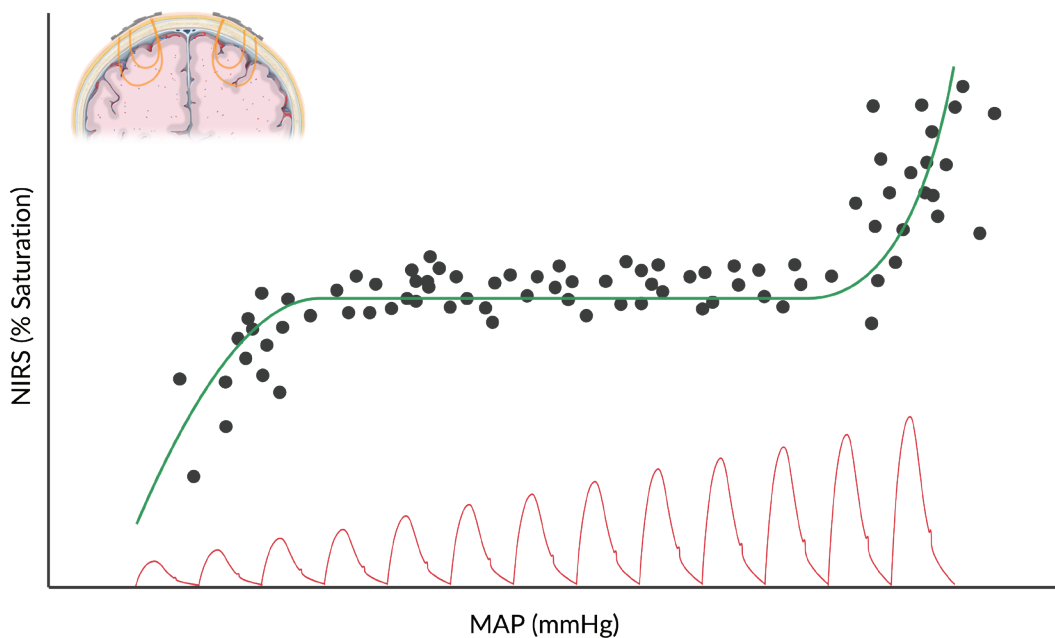


Autoregulation Index

Let's revert our attention to Morgan's patient who's undergoing shoulder surgery in the beach chair position. The problem is that Morgan doesn't truly know her patient's lower limit of autoregulation, so using a deliberate hypotensive technique to satisfy the surgeon's request for a bloodless field is a risky proposition.

Up to this point, we've explored methods of assessing cerebral blood flow, but is there a convenient and widely available way for Morgan to monitor the patient's unique cerebral autoregulation curve (and thus establish the patient's unique LLA) in the operating room? The short answer is not yet, but here's a look at the technology that may soon make this a reality.

In order to define a patient's unique cerebral autoregulation curve, we can calculate a cerebral autoregulation index. One way of doing this is by plotting changes in NIRS saturation against a range of arterial blood pressures. This means we need two things. First, we need a continuous assessment of arterial blood pressure. Second, we need a cerebral oximeter which provides surrogate data to measure cerebral blood flow in real-time. These data can be fed into an algorithm that calculates a cerebral oximetry index. In turn, this can tell us when MAP is outside the boundaries of a patient's unique autoregulation curve providing us the clinical data we need to safely manage his blood pressure.



Literature

Let's finish with an examination of some of the key literature in the domain of cerebral autoregulation in the beach chair position. A chilling case report detailed in the Anesthesia Patient Safety Foundation newsletter described a healthy 47-year-old female who underwent shoulder arthroscopy in the beach chair position. Because the patient was hypertensive just before induction, the anesthesia provider administered labetalol 50 mg IV. The patient's systolic blood pressure settled in the 80-90 range for most of the case. This was likely below the patient's lower limit of autoregulation, and the patient suffered brain infarction and remained in a persistent vegetative state as a result. This paper described another patient (also in the beach chair position) who suffered brain death. In this case, a deliberate hypotensive technique was used, and the blood pressure was measured on the calf. Despite systolic blood pressures in the 70 - 90 range (which are arguably too low), you can imagine how poorly perfused the brain was during this case.

A paper in *Anesthesia & Analgesia* compared the effect of surgical positioning (beach chair vs. lateral decubitus) for patients undergoing shoulder surgery. Despite similar anesthetic management in both groups, NIRS derived cerebral oxygenation was lower in the beach chair patients. Furthermore, this group suffered a higher frequency of cerebral desaturation events, raising concerns about failed autoregulation.

Mounting concerns prompted the Anesthesia Patient Safety Foundation to commission its Beach Chair Study. This study compared patients in the beach chair and lateral decubitus positions and measured cerebral oxygenation with NIRS along with a direct assessment of mean arterial blood pressure. Each patient's cerebral oximetry index was calculated using statistical procedures that generated a coefficient between cerebral oxygen saturation and MAP. Although patients in the beach chair position experienced diminished autoregulation, there were no differences in cognitive outcome or biomarkers between the two groups.

If the beach chair position is used, then we can apply what we know about manipulating cerebral blood flow to optimize cerebral perfusion. A prospective study examined 56 patients randomized to receive either desflurane-based general anesthesia or propofol-based total intravenous anesthesia. The study revealed that increasing both $F_{I}O_2$ and end-tidal CO_2 resulted in a reliable and clinically significant increase in $rScO_2$ that overcame desaturation events occurring in the beach chair position. This held true for both types of anesthesia.

Where does this leave us? While we can employ clinical maneuvers (such as avoiding hyperventilation) to support cerebral blood flow, the technology that can define a patient-specific autoregulatory curve is expensive, time consuming, and not widely available. Having said this, think back just a few decades when pulse oximetry, capnography, and potent inhaled agent monitors were expensive, imprecise, and lacked controlled trials. Over a period of rapid, punctuated evolution, these technologies not only proved their worth for enhancing patient safety, but they also became cheaper, smaller, and far more efficient.

Before we end, we'd be remiss not to consider how monitoring cerebral autoregulation can affect organ function aside from the brain. In a study of cardiac surgical patients undergoing cardiopulmonary bypass, investigators examined the relationship between systemic blood pressure below the lower limit of cerebral autoregulation and kidney function. The researchers concluded that blood pressure reductions below the lower limit of cerebral autoregulation was an independent predictor of acute kidney injury. This finding suggests that using an index of cerebral oximetry may provide useful data to help protect organ systems other than the brain during cardiopulmonary bypass.

Key Points

Here are some key points for your practice:

- The lower limit of cerebral autoregulation in each patient is unknown unless it's specifically determined by the technologies described in this objective.
- We must consider the effects of head elevation on blood pressure measurements obtained in the arm or the calf, as unintentional cerebral hypoperfusion may result.
- Blood pressure below the LLA does not necessarily predispose the patient to cerebral ischemia if compensatory mechanisms (such as increased tissue oxygen extraction or preferential shunting of blood) occur.
- Patients with impaired cerebral autoregulation are at greatest risk, including those with cardiovascular or cerebrovascular disease, autonomic nervous system dysfunction, and old age.