The Problems of Posture, Pressure, and Perfusion Letter

APSF
To the Editor:

In the Summer 2007 APSF Newsletter, Cullen and Kirby cite a dramatic case of cerebral infarction during shoulder surgery in the beachchair position.1 This case was 1 of 4 apparent cerebral and/or spinal cord infarctions presented as a series by Pohl and Cullen in 2005,2 as gleaned from medico-legal reviews by one of the 2 authors (DJC).

Most anesthesia professionals would not argue against maintaining blood pressure (BP) within a reasonably close range of preoperative values during any anesthetic, in the sitting position or otherwise; nor would I. But prescriptions for acceptable BP management should acknowledge the lack of relevant human data, and should also make reference to methodologic issues in assessing cerebral perfusion pressure (CPP). As such, I would like to point out several potential areas of controversy or ambiguity that may arise from a reading of Cullen and Kirby's article:

Of relevance to the author's, and others', concern for BP management in the context of baseline values, the other 3 cases presented in the original series had no preoperative BPs reported, and one case used a BP cuff positioned on the calf while in the seated position. By "preoperative values," I mean measurements obtained outside of the stress of the operating room and in an upright position, as per usual in a preoperative clinic, holding area, or exam room setting. It is therefore impossible to know by what percentage the patients' normal baseline BP was allowed to change during the anesthetics. Consequently, that series can offer us little or no quantitative guidance, absent the extremes beyond common sense and common practice.

Discussions, including Cullen and Kirby's, of BP management in the sitting position seldom take into account that the upright position is the normal position occupied by most human beings during most waking hours, and that no numerical "compensation" is made for the upright position when measuring BP in sitting, awake outpatients. That general anesthesia decreases BP in the sitting position is irrelevant to this point. The issue here is not whether interventions should be made to restore BP to approximately normal levels (as appropriately suggested by Drummond);3 but rather, the more fundamental question of how BP should be measured in the first place—either before or after an intervention is made. If one argues that when the head is elevated above the heart, an "adjustment" should be made for a decreased CPP, then perhaps one should also explain why the same adjustment is not made for all ambulatory, upright, measurements. For example, why should we assume that a BP in the sitting position under anesthesia is any different with regard to CPP than the same BP measured in the same way in an awake patient sitting in a preoperative clinic?

Regarding the methodology of BP measurement, the practice of "compensating" for arm BP cuff readings in the sitting position extends back to 1954 when that advice was first published by Enderby,4 and it has been followed uncritically ever since. The refinement

of Enderby's advice in neurosurgical cases, where the arterial line has largely supplanted the BP cuff, applies the same assumption but by a different method. Raising an arterial line transducer to head level accomplishes by physical means the same thing as making a numerical "correction" to a BP cuff reading. Both adjustments make an intuitive assumption that the head is in a compromised position for perfusion when it is in its (normal) upright position relative to the heart. Implicit in that assumption, but rarely stated explicitly, is a correlative assumption: that the cerebral circulation is an "open" fluid path where a pump forces blood up to a higher elevation, and that it flows passively downward (like a waterfall in open air) back to the heart.

This conceptual model of the cerebral circulation is wanting for at least 5 reasons:

1. it does not match the anatomy of what we know is a closed, continuous fluid path that does not contain anywhere within it an open-air waterfall component;
2. it does not work when upside down or in weightlessness (but the actual cerebral circulation does);
3. it cannot explain the well-described phenomenon of venous air embolism (VAE) in mechanically ventilated patients;
4. it cannot explain the common observation, in sitting neurosurgical cases, of right atrial pressure (measured at heart level) being far below the expected value of the hydrostatic pressure of a 25-30 cm column of blood extending from the superior sagittal sinus down to the right atrium; and
5. it does not explain why the risk of VAE is in proportion to the degree of elevation of the perforation above the heart.

On the other hand, the conceptual model of the cerebral circulation as a "closed" circulation easily satisfies the 5 observations above. And inherent to a closed model is a very strong argument against making "compensations" for "perfusion pressure" by raising transducers or subtracting numerical adjustments from BP cuff measurements.

We don't, of course, monitor hemodynamics in a conceptual vacuum. Instead, we interpret the numbers we measure in the context of our best mental model of the circulation. One consequence of rejecting the "open" model is that we now have to distinguish carefully, when we talk about "pressure," between true perfusion pressure and transmural pressure. The practice of raising transducers to head level or making numerical adjustments to BP cuff readings in a closed circulation model actually "adjusts" for something very different from perfusion pressure—it adjusts for transmural pressure.

Why does this matter? Because only perfusion pressure, not transmural pressure, is associated with flow. And flow is what we are interested in. An arterial line measurement can be used to estimate perfusion pressure only if both inlet and outlet pressures on either side of the organ of interest are measured, and only if both pressures are referenced to the same level. By conventional definition, "perfusion pressure" is a pressure gradient, not a single point measurement at only one place in a circuit. Making inferences about perfusion based on a transmural pressure reading at only one point in the circuit can be

misleading in certain circumstances. The sitting position is one of them. While it may seem intuitive that the "real" perfusion pressure to the brain is a single-point transmural pressure reading referenced to brain level (i.e., the transducer is elevated to the level of the head), this fails to take into account that the outlet (venous) pressure of the brain should also be considered in similar fashion.

Not only does elevating the head (to its normal day-to-day position) reduce cerebral arterial transmural pressure relative to the heart; so too, does elevating the head reduce the sinus and venous outlet transmural pressures relative to the heart, and by the same amount. For that reason, elevating the head does not, by itself, decrease cerebral blood flow so long as mean arterial pressure (MAP) at the level of the heart is not allowed to change. A change in transmural pressure at one point in the circuit—which is what a numerical "adjustment" of a BP cuff reading, or raising an arterial line transducer to head level tells us—does not imply a change in flow.5,6

A simple illustration may help to clarify this point: the flow rate of fluid through IV tubing is proportional to the relative height of the IV bag and the patient. The path that the IV tubing takes between the IV bag and the patient does not affect flow rate. The tubing can be looped down to the floor and then back up to the patient, or even looped up over the top of the IV pole and back down to the patient, and the flow will be the same in either case. If you make a mark at one point on the tubing and measure the transmural pressure (again, inside minus outside pressure) at that one point, it will be dramatically different depending on its position relative to the patient. The transmural pressure at your mark in the tubing may be negative (subatmospheric) if it is elevated above the IV pole; or it may be markedly positive if that point is dropped down to the floor below the patient. But in either case, flow through the tubing remains unchanged because perfusion pressure (inlet minus outlet pressure) is unchanged. Local transmural pressure at just one point cannot be substituted for perfusion pressure. They are completely different concepts, and should not be used interchangeably.

Returning to the cerebral circulation, if we say that "perfusion pressure" at the elevated level of the upright brain is lower, we are in fact referring not to perfusion pressure, but to a local transmural pressure. Perfusion pressure remains inlet (aorta) minus outlet (right atrium) pressure. If we insist on "compensating" for a fall in local (transmural) arterial pressure at the inlet of the brain (either by moving the transducer above the heart to head level; or by a numerical adjustment to a BP cuff reading), then to be consistent, we should also "compensate" for the corresponding fall in the transmural pressure of the brain's sinuses and veins when measured at the same level in the sitting position. That could be accomplished by also raising the CVP transducer to head level. If we do so, we will see that both inlet and outlet pressures have fallen, and by the same amount. Cerebral perfusion pressure remains unchanged and there is, in fact, no point in making the 2 self-cancelling "compensations." The standard definitions of CPP (CPP = MAP - CVP when CVP > ICP; CPP = MAP - ICP when ICP > CVP) remain unchanged, and there is no rationale for leveling MAP and CVP transducers at different heights when measuring CPP.

If one doubts that cerebral veins and sinuses have lower, even negative, transmural pressures in the upright position, then consider the well-described phenomenon of venous air embolism (VAE). In a mechanically ventilated patient who is making no inspiratory efforts, the same "siphon" effect that is inherent to a closed model of the circulation causes subatmospheric pressure in the IV tubing example also causes subatmospheric pressure in the elevated sinuses and veins of the head. This is how VAE occurs even in mechanically ventilated patients when the operative site is elevated above the heart, and it is also why the tendency for VAE is proportional to the degree of elevation of the operative site above the heart.

An open model of the circulation provides no explanatory power in this domain, and this limitation of the open model should be addressed in any discussion of the mechanism of VAE specifically; and in any discussion of hemodynamic monitoring in the sitting position generally. Among circulatory physiologists, the controversy between adopting an open versus a closed model of the cerebral circulation is just that: a controversy.6 I am not advocating an uncritical acceptance of the closed model, along with its implications for hemodynamic monitoring. But I am advocating that the anesthesia and monitoring communities acknowledge and address, on its merits, arguments for and against both models. In this domain, where the "right" answer may very well be counterintuitive, it is especially important to allow physiology to lead the discussion.

Every day in almost every anesthetic, we make BP cuff measurements and infer something about whole body perfusion. That is a time-tested empiric relationship for which we have much experience and much data. I am not, of course, suggesting that we discount BP cuff readings in general just because they measure a local transmural pressure in the arm beneath the cuff. Nor am I suggesting that we allow blood pressure, properly measured and interpreted, to fall significantly below the patient's preoperative baseline. Instead, I am suggesting that we not make an unnecessary numerical adjustment for the use of BP cuffs in the sitting position. Such an adjustment is predicated on a false assumption made a half century ago about the physics and the physiology of CPP; and a confusion of transmural for perfusion pressure.

There is a great need to revisit the important question of "what is a safe blood pressure?" The cases referred to by Cullen and Kirby can offer a general wake-up call that even modest hypotension may be dangerous; and that we should be circumspect in agreeing to a surgeon's request for deliberate hypotension. But absent a case population denominator, or even sufficient documentation of baseline and equally-measured intraoperative BPs in the 4 cases presented, they can offer very little quantitative guidance to help explore the question. Most practitioners would not run their patients' BPs as low as those presented; regardless of where or how they were measured.

As a specialty, we may very well reexamine what we accept as best practice for BP management so that we are not losing patients on one tail of the susceptibility curve to bad outcomes. By all means, we should run the BP, measured normally in what is a normal human upright position, higher than in the cases presented until we know the answers. Most of us would anyway. But let's not add to our current ignorance of what a

safe BP is, in general, by making an adjustment that may not make physiological sense, however timeworn it is. That is simply using a physiologically suspect means to achieve a laudable end. We don't need to do that. We can have our laudable end while still respecting, or at least acknowledging, that the underlying physiology is not as straightforward or as intuitive as many of us were taught. Adding an extra level of complexity through BP "adjustments" that fail to acknowledge or even take into account the basic physiological principles above will only obscure, not clarify, the eventual answer.

James Munis, MD, PhD
Chair, Division of Neuroanesthesia
Assistant Professor of Anesthesiology, Physiology, and Biomedical Engineering
Mayo Clinic College of Medicine
Rochester, MN
References

1. Cullen DJ, Kirby RR. Beach chair position may decrease cerebral perfusion: catastrophic outcomes have occurred. APSF Newsletter 2007;22(2):25,27.
2. Pohl A, Cullen DJ. Cerebral ischemia during shoulder surgery in the upright position: a case series. J Clin Anesth 2005;17:463-469.
3. Drummond JC. The lower limit of autoregulation: time to revise our thinking? Anesthesiology 1997;86:1431-1433.
4. Enderby GEH. Postural ischaemia and blood pressure. Lancet 1954;1:185-187.
5. Munis JR, Lozada LJ. Giraffes, siphons, and Starling resistors: cerebral perfusion pressure revisited. J Neurosurg Anesthesiol 2000;12:290-296.
6. Hicks JW, Munis JR. The siphon controversy counterpoint: the brain need not be "baffling." Am J Physiol 2005;289:R629-632.