Dynamic Cerebral Autoregulation and Monitoring Cerebral Perfusion

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Cerebral autoregulation (CA) describes the capability of the brain to maintain its flow as relatively stable over a wide range of mean arterial pressures (MAPs), for example, from 60 to 150 mm Hg. Because the brain is encompassed in the skull, CA is vital for preventing cerebral edema and hemorrhage, as illustrated in some patients experiencing acute liver failure. Also, CA counteracts the effect on the skull, CA is vital for preventing cerebral edema and hemorrhage, as illustrated in some patients experiencing acute liver failure.1 Also, CA counteracts the effect on cerebral blood flow (CBF) of a reduction in MAP in response to, for example, hemorrhage or a change in body position. Yet, when rising from a supine or seated position, one sometimes experiences symptoms indicative of a reduced CBF, like blurred vision.2 Thus, CA is of general clinical interest. For example, diabetic patients have increased risk of developing ischemic or hemorrhagic stroke, and in some diabetic patients CA is affected.

Evidence for CA was provided in the 1930s when Fog,3 through a cranial window, observed the pial artery reaction to manipulation of MAP. For humans, CBF was measured with introduction of the method of Kety-Schmidt in 1948,4 and it was used in 1954 by Finnerty et al5 to determine cerebral hemodynamics of the ischemic brain after an acute reduction in MAP in attempt to define what now would be considered the lower limit of CA. However, it was the review by Lassen6 that introduced the concept of CA by plotting CBF against MAP to illustrate the relative stability of CBF over a wide range of blood pressure. Also, the lower and upper limits of CA were identified by a decrease in CBF at a low MAP and an increase in CBF when MAP was at a high level.

A different approach to CA became available when Aaslid et al7 introduced the transcranial Doppler method for determination of mean flow velocity (Vmean) in basal cerebral arteries. Because transcranial Doppler reports a continuous (middle cerebral artery) Vmean, cerebral perfusion could be followed during the drop in MAP that follows release of thigh cuffs. It took ≈3 seconds for Vmean to recover from the sudden drop in MAP that followed the release of the thigh cuffs, and, accordingly, the acute response of cerebral perfusion to a change in MAP was referred to as the dynamic CA. Dynamic CA, or the instability of CBF during a sudden change in MAP, is important because it explains blurred vision sometimes experienced in response to a change in body position.2 The study by Tzeng et al8 takes advantage of the drop in blood pressure that manifests from a seated to an upright position to characterize dynamic CA. Conversely, the marked increase in MAP that develops in response to a Valsalva maneuver may be used to characterize CA during a sudden increase in MAP (Figure).9

Some scepticism may remain as to whether the classic (static) and dynamic CA describe the same physiological phenomenon, but supporting evidence was provided by Tiecks et al.10 These authors determined static and dynamic CA from monitoring Vmean during elective surgical procedures using propofol and high-dose isoflurane anesthesia. During propofol anesthesia, both static and dynamic CA (the Vmean response to administration of phenylephrine versus thigh-cuff release) remained intact, whereas the 2 expressions of CA became similarly affected when general anesthesia was shifted to isoflurane. Taken together, such observations indicate that CA can be described in terms of a high-pass filter for the transfer function from MAP to Vmean. Thus, CA is specified with regard to its gain and the phase angle for which Vmean leads blood pressure.

In this issue of Hypertension, Tzeng et al8 renew the evaluation of dynamic CA. The Vmean response to a lowering of MAP, either by rising from a seated position or in response to the administration of nitroprusside, is supplemented by an evaluation of the Vmean response to an elevation of MAP by phenylephrine in volunteers. Surprisingly, they found that the ability of CA to respond (damp) to a rise in MAP is approximately twice as well developed as the ability to withstand a reduction in MAP, as suggested previously by Ogoh et al11 during exercise.

If CA is much more capable of damping an increase than a decrease in blood pressure, it implicates that a simple correlation analysis is not ideal for characterization of the transfer function from blood pressure to Vmean. On the other hand, the pharmacological approach to CA introduced by Tzeng et al8 is both clinical relevant and feasible and definitely much easier to apply to patients than the invasive procedures needed for the determination of static CA based on CBF. We consider also that, in a clinical setting, it would be more straightforward to administer 2 drugs after each other than to use the thigh-cuff release method of Aaslid et al7 for the evaluation of (dynamic) CA.

The question, however, remains regarding whether an evaluation of CA provides enough information to verify that CBF remains stable. We think not. We agree that it is relevant...
and may be vital to know whether a given patient’s CBF is under CA control, for example, in regard to how aggressive treatment of hypertension should be.

However, it remains a problem that the margins for (static) CA are highly individual and also dependent on the circumstances under which the CA is evaluated. Notably, an elevation of the arterial carbon dioxide tension (Paco2) impairs CA, whereas a lowering of Paco2 has the opposite effect. Also, the lower level of CA may be as low as 35 mm Hg, provided that the central blood volume and, thereby, cardiac output are maintained, whereas subjects tolerate only a reduction in MAP to ≈ 80 mm Hg if the central blood volume and cardiac output are compromised as during hemorrhage, head-up tilt, or lower body negative pressure.12

Similarly, with the lower limit of CA ranging from 17 to 54 mm Hg among healthy people,3 it is a problem to know whether the individual patient’s CBF is maintained at a given MAP. More predictable is that the lower limit of CA is elevated in hypertensive patients and that it is further elevated in patients with malignant hypertension. Yet, again, the individual differences were astounding, that is, between 23 and 65 mm Hg, respectively, between 84 and 114 mm Hg in the 2 groups of hypertensive patients.

A further complication for evaluation of CBF is that there are substantial interindividual differences. Therefore, changes in CBF in response to treatment are of more interest that the absolute value, and in response to the marked changes in MAP associated with anesthesia and intensive care medicine, CBF needs to be monitored to secure the well being of the patients, considering that it may be impossible to communicate with the patient.

As illustrated by the study of Tzeng et al,8 transcranial Doppler allows for continuous evaluation of cerebral perfusion, but it is, according to our experience, too complicated to apply transcranial Doppler to allow for routine monitoring of CBF in a large group of patients. In contrast, near infrared spectroscopy is available and operating by the same principle as pulse oximetry, and it is as simple to apply. Furthermore, a near infrared spectroscopy-based evaluation of (frontal lobe) oxygenation allows for evaluation of dynamic CA, as illustrated during a Valsalva maneuver (Figure).9 Accordingly, we congratulate Tzeng et al8 for providing potentially new insight to CA and for illustrating a clinically feasible method for CA evaluation. At the same time, we would like to see a similar evaluation based on the recording of cerebral oxygenation by near infrared spectroscopy and maybe by inclusion of a Valsalva maneuver so that there is a parallel physiological and pharmacological evaluation of dynamic CA.

Disclosures

None.

References

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