



# Multimodal monitoring in neurocritical care

MICHAEL A. DE GEORGIA, MD

For many years, monitoring in the intensive care unit was limited to clinical examination, heart rate, and blood pressure. In 1970, the Swan-Ganz pulmonary artery catheter was introduced,<sup>1</sup> and the specialty of critical care medicine was born. For the first time, clinicians could guide therapy based on physiologic parameters.

In the neurologic intensive care unit, monitoring has also been limited to the clinical exam and intracranial pressure. In the 1980s, much was learned about the importance of the cerebral perfusion pressure and cerebrovascular autoregulation. In the 1990s, the concept of “multimodal monitoring” was introduced—namely, cerebral blood flow monitoring, brain tissue oxygenation, and intracerebral microdialysis. Multimodal monitoring to assess metabolic function provides the neurointensivist with crucial information at the cellular level. This information can be used to detect potentially reversible secondary insults and to target therapy more precisely.

## ■ CEREBRAL BLOOD FLOW

Although we have been able to measure cerebral blood flow for decades, beginning in 1948 with Kety and Schmidt<sup>2</sup> and more recently with xenon-enhanced CT scanning,<sup>3</sup> these methods give only snapshots of cerebral blood flow and do not allow for continuous monitoring. Two methods are used for continuous monitoring at the bedside:

- With **laser Doppler flowmetry**, a *qualitative* estimate of regional cerebral blood flow, based on the

Doppler shift principle, is displayed (in arbitrary units).<sup>4</sup>

- With **thermal diffusion**, a *quantitative* estimate of regional cerebral blood flow, based on the tissue's ability to dissipate heat, is displayed in mL/100 g/min.<sup>5</sup>

Cerebral blood flow monitoring can be useful for determining the state of autoregulation.

## ■ BRAIN TISSUE OXYGENATION

Brain tissue oxygen tension, or tissue partial pressure of oxygen (PtiO<sub>2</sub>), can be measured using a small flexible probe that is inserted directly into the brain parenchyma, most commonly in the frontal white matter. The normal brain PtiO<sub>2</sub> varies depending on location, but it averages approximately 40 mm Hg.<sup>6</sup> Several studies have shown that cerebral oxygenation is strongly correlated with cerebral perfusion pressure.<sup>7,8</sup> Low brain PtiO<sub>2</sub> has also been associated with poor outcome.<sup>9</sup> Zauner and coworkers<sup>10</sup> showed that in patients who had a good recovery, the brain PtiO<sub>2</sub> was greater than 35 mm Hg. In patients with moderate to severe disability, the brain PtiO<sub>2</sub> was 26 to 35 mm Hg; in those with a poor outcome, it was 25 mm Hg or less.<sup>10</sup> Though intuitive, whether improving brain PtiO<sub>2</sub> improves patient outcome is inconclusive at this point. Further studies are needed.

## ■ INTRACEREBRAL MICRODIALYSIS

Introduced more than 25 years ago, microdialysis is a technique for monitoring the chemistry of the extracellular space in living tissue. The microdialysis probe is designed to mimic a “blood capillary.” When a physiological salt solution is slowly pumped through the microdialysis probe, the solution equilibrates with the surrounding extracellular tissue fluid. After a while, it will contain a representative proportion of the tissue fluid's molecules. The microdialysate can then be extracted and analyzed

From the Neurological Intensive Care Program, Department of Neurology, The Cleveland Clinic Foundation, Cleveland, Ohio.

**Address:** Michael A. De Georgia, MD, Head, Neurological Intensive Care Program, The Cleveland Clinic Foundation, 9500 Euclid Avenue, S80, Cleveland, OH 44195; e-mail: degeorm@ccf.org.

in the laboratory or by the bedside.

The first human studies of microdialysis in the brain were published in the 1990s.<sup>11</sup> Subsequent studies have shown the potential of intracerebral microdialysis for monitoring in the neurologic intensive care unit.<sup>12</sup> This technique has been used to show early impaired cerebral blood flow after severe head injury, which was found to result in poor brain tissue oxygen delivery, lactate accumulation, and massive release of glutamate.<sup>13</sup> Unterberg and coworkers<sup>14</sup> have demonstrated changes in microdialysis in patients who develop vasospasm following subarachnoid hemorrhage.

## ■ CONCLUSION

Multimodal monitoring that includes cerebral blood flow monitoring, brain tissue oxygenation, and intracerebral microdialysis appears to have a unique potential for providing critical basic physiologic information about brain function in patients with brain ischemia. This approach, along with a new system for integrating these parameters in the neurologic intensive care unit, may revolutionize the way in which patients with brain injury are monitored and treated.

## ■ REFERENCES

1. Swan HJC, Ganz W, Forrester JS, et al. Catheterization of the heart in man with use of a flow-directed balloon-tipped catheter. *N Engl J Med* 1970; 283:447–451.
2. Kety SS, Schmidt CF. The nitrous oxide method for the quantitative determination of cerebral blood flow in man: theory, procedure, and normal values. *J Clinical Invest* 1948; 27:476–483.
3. Yonas H, Johnson DW, Pindzola RR. Xenon-enhanced CT of cerebral blood flow. *Scientific American: Science and Medicine* 1995; 2:58–67.
4. Bolognese P, Miller JI, Heger IM, Milhorat TH. Laser-Doppler flowmetry in neurosurgery. *J Neurosurg Anesthesiol* 1993; 5:151–158.
5. Carter LP, Weinand ME, Oommen KJ. Cerebral blood flow (CBF) monitoring in intensive care by thermal diffusion. *Acta Neurochir Suppl (Wien)* 1993; 59:43–46.
6. Meixensberger J, Dings J, Kuhnigk H, Roosen K. Studies of tissue PO<sub>2</sub> in normal and pathological human brain cortex. *Acta Neurochir Suppl (Wien)* 1993; 59:58–63.
7. Stocchetti N, Chierregato A, De Marchi M, et al. High cerebral perfusion pressure improves low values of local brain tissue O<sub>2</sub> tension (PtiO<sub>2</sub>) in focal lesions. *Acta Neurochir Suppl (Wien)* 1998; 71:162–165.
8. Unterberg AW, Kiening KL, Hartl R, et al. Multimodal monitoring in patients with head injury: evaluation of the effects of treatment on cerebral oxygenation. *J Trauma* 1997; 42(5 suppl):S32–S37.
9. van den Brink WA, van Santbrink H, Avezaat CJ, et al. Monitoring brain oxygen tension in severe head injury: the Rotterdam experience. *Acta Neurochir Suppl (Wien)* 1998; 71:190–194.
10. Zauner A, Doppenberg EMR, Woodward JJ, Choi SC, Young HF, Bullock R. Continuous monitoring of cerebral substrate delivery and clearance: initial experience in 24 patients with severe acute brain injuries. *Neurosurgery* 1974; 1:1082–1093.
11. Hillered L, Persson L, Ponten U, Ungerstedt U. Neurometabolic monitoring of the ischaemic human brain using microdialysis. *Acta Neurochir (Wien)* 1990; 102:91–97.
12. Persson L, Hillered L. Chemical monitoring of neurosurgical intensive care patients using intracerebral microdialysis. *J Neurosurg* 1992; 76:72–80.
13. Goodman JC, Valadka AB, Gopinath SP, Uzura M, Robertson CS. Extracellular lactate and glucose alterations in the brain after head injury measured by microdialysis. *Crit Care Med* 1999; 27:1965–1973.
14. Unterberg AW, Sakowitz OW, Sarrafzadeh AS, Benndorf G, Lanksch WR. Role of bedside microdialysis in the diagnosis of cerebral vasospasm following aneurysmal subarachnoid hemorrhage. *J Neurosurg* 2001; 49:740–749.