Parenchymal Brain Oxygen Monitoring in the Neurocritical Care Unit

Peter D. Le Roux, MD[a,*], Mauro Oddo, MD[b]

KEYWORDS

• Brain monitoring • Brain oxygen • Clark electrode • Hypoxia • Neurocritical care
• Optical fluorescence • Traumatic brain injury • Subarachnoid hemorrhage

KEY POINTS

- Parenchymal brain tissue oxygen ($PbtO_2$) monitoring is a safe and reliable technique for continuous bedside evaluation of patients with severe brain injury.
- Two techniques, a modified Clark electrode that uses the electrochemical properties of noble metals or optical fluorescence technology, can be used to measure $PbtO_2$.
- $PbtO_2$ indicates the balance between regional oxygen supply and cellular oxygen consumption and may be described by the equation $PbtO_2 = CBF \times AVTo_2$, where CBF is cerebral blood flow, Pvo_2 is partial oxygen pressure in mixed venous blood, and $AVTo_2$ is $Pao_2 - Pvo_2$.
- $PbtO_2$ values less than 20 mm Hg are considered worth treating and values less than 15 mm Hg are consistent with brain hypoxia or ischemia.
- Reduced $PbtO_2$ is associated with worse outcome in acute brain injury in adults and children, although the strength of this relationship may depend on probe location.
- When severe traumatic brain injury care is based on data from both a $PbtO_2$ and intracranial pressure (ICP) monitor, some (but not all) observational series suggest that outcome is better than when just ICP-based care is provided.

INTRODUCTION

Patients with a variety of severe, acute neurologic disorders such as traumatic brain injury (TBI), subarachnoid hemorrhage (SAH), acute ischemic stroke, and intracerebral hemorrhage (ICH) often are admitted to the neurocritical care unit (NCCU). Despite much research and success in animal models, effective drug therapies for these disorders have not been identified in clinical trials.[1] Instead, much of patient management in the NCCU is centered on the early identification and removal of mass lesions and on the detection, prevention, and management of secondary brain insults that exacerbate outcome (eg, hypotension, hypoxia, seizures, increased intracranial pressure [ICP]). Careful and repeated assessment and monitoring of clinical and laboratory findings, imaging studies, and bedside physiologic data are consequently what drive modern neurocritical care.

A variety of monitors are currently in clinical use (**Box 1**), although the ideal monitor to assess neurologic function in the NCCU does not yet exist. These monitors may be classified into 2 broad categories: (1) radiographic or tomographic techniques that provide information at a single point in time, and (2) bedside monitors that may

[a] The Brain and Spine Center, Lankenau Medical Center, 100 E. Lancaster Ave, Wynnewood, PA 19096, USA;
[b] Service de Médecine Intensive Adulte, Medico-Surgical ICU, Centre Hospitalier Universitaire Vaudois - CHUV, Rue du Bugnon 46, Lausanne 1011, Switzerland
* Corresponding author.
E-mail address: lerouxp@mlhs.org

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Box 1
Examples of monitors in clinical use in the NCCU

- Clinical evaluation, serial assessment
- Laboratory analysis
- Systemic: electrocardiogram, heart rate, blood pressure, O_2 saturation, end tidal CO_2 (Et_{CO_2}), temperature
- Hydraulic: ICP/cerebral perfusion pressure (CPP)
- Electrophysiology: electroencephalogram, somatosensory evoked potentials, brain stem auditory evoked response
- Radiographic/tomographic: positron emission tomography (PET), single-photon emission computed tomography, CT-P, stable Xe-CT (^{133}XE), magnetic resonance imaging (MRI)
- Cerebral blood flow (CBF): transcranial Doppler, laser Doppler, thermal diffusion probe, transcranial cerebral oximetry
- Metabolic: microdialysis, jugular venous oximetry, direct brain oxygen, near infrared spectroscopy
- Biosamples (cerebrospinal fluid or serum): eg, S100B, GFAP, NSE

Abbreviations: CT-P, CT – perfusion scan (computed tomography); GFAP, Glial fibrillary acidic protein; NSE, neuron specific enolase.

be subdivided into monitors that are (1) invasive or noninvasive, or (2) continuous or noncontinuous. More than 1 monitor is ideally used because the brain is a complex organ and no single method can provide complete information about its health. Furthermore, monitoring by itself does not alter outcome. Instead, it is how the information provided by the monitor is used that contributes to patient wellbeing, particularly when targeted to patient-specific pathophysiology. This article reviews one type of continuous physiologic monitor: direct measurement of parenchymal brain oxygen.

THE IMPORTANCE OF BRAIN OXYGEN

Maintenance of adequate tissue oxygenation is a fundamental objective in critical care medicine in general, and the assessment of tissue oxygenation is indispensable to care of the critically ill patient. The adult brain represents about 2% of body weight, but consumes about 20% of the oxygen consumed by the body. Greater than 90% of this oxygen is used by the mitochondria to produce ATP, which is integral to cell function.[2] For this energy metabolism, brain cells must be supplied with oxygen and glucose, the primary fuel for the brain (although, in some circumstances, lactate also may be used).[3] Only then, and with normal mitochondrial function, can sufficient energy (ATP) be produced to maintain neuronal integrity and function. The brain lacks fuel stores and requires a continuous supply of glucose and oxygen. Therefore, continuous CBF, cerebral oxygen tension and delivery, and normal mitochondrial function are of vital importance to maintain brain function and tissue viability.

DEFINITION

There are 4 basic methods to measure brain oxygen: jugular venous bulb oximetry, direct brain tissue (parenchymal) oxygen tension measurement, near infrared spectroscopy, and oxygen-15 PET. This article discusses parenchymal brain oxygen measurement, the commonest technique currently used in the NCCU to assess cerebral oxygenation. Brain tissue oxygen, or parenchymal brain oxygen, is defined as the partial pressure of oxygen in the brain interstitial space and reflects the availability of oxygen for oxidative energy production. There has been debate about whether the technique measures tissue oxygen pressure or tension, and, accordingly, several abbreviations have been used for brain tissue oxygen. A consensus conference at the 13th International Symposium on Intracranial Pressure and Brain Monitoring held in July 2007 in San Francisco, California, proposed that $PbtO_2$ be used as the standard abbreviation. Hence, this article uses $PbtO_2$ when referring to brain tissue oxygen or parenchymal brain oxygen. Consistent with this abbreviation, recent clinical studies suggest that $PbtO_2$ may be best defined by the equation: $PbtO_2 = CBF \times AVT_{O_2}$, where AVT_{O_2} is $Pa_{O_2} - Pv_{O_2}$ (ie, $PbtO_2$ represents the interaction between plasma oxygen tension and CBF).[4]

TECHNOLOGY

Brain oxygen ($PbtO_2$) monitors were first used in the clinical environment in 1993 and included in the treatment guidelines for severe TBI in 2007.[5] Two techniques are available: (1) a modified Clark electrode that uses the electrochemical properties of noble metals (eg, Licox, Integra Lifesciences, Plainsboro, NJ; or Neurovent-P Temp, Raumedic AG, Munchberg, Germany), and (2) optical fluorescence technology (eg, Neurotrend, Diametrics Medical, St Paul, MN, and Codman, Johnson & Johnson, Raynham, MA; and OxyLab P_{O_2}, Oxford Optronix Ltd, Oxford, UK).