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Case Report

$PbtO_2$ monitoring in normobaric hyperoxia targeted therapy in acute subarachnoidal hemorrhage

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Abstract

Background: Low brain tissue oxygen tension ($PbtO_2$), or brain hypoxia, is an independent predictor of poor outcome. Increasing inspirational fraction of oxygen could have a significant influence on treating lower $PbtO_2$. Combined $PbtO_2$ therapy, compared to the approach that focus only on regulation of cerebral perfusion pressure and intracranial pressure, shows better patient outcomes. Monitoring of $PbtO_2$ could be helpful in individualizing treatment, preventing or limiting secondary brain injury, and maintaining better patient outcome.

Case Description: We present a case of a patient with subarachnoidal hemorrhage to whom $PbtO_2$ monitor was implanted, and normobaric hyperoxia treatment was adjusted according to $PbtO_2$ measurement. The patient progressively recovered and was dismissed with Glasgow Coma Score 4/5/6.

Conclusion: The use of $PbtO_2$ monitoring may be useful for monitoring the local tissue values that are useful for induction of normobaric hyperoxia and optimizing the therapy toward more target-defined values. It is an important part of multimodal neuromonitoring, and is the gold standard for brain oxygenation monitoring that can lead to better patient outcome.

Key Words: Anesthesia, Licox, neuromonitoring, normobaric hyperoxia, $PbtO_2$ monitoring, subarachnoidal hemorrhage

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INTRODUCTION

The main goals in neuroanesthesia are maintaining intracranial pressure (ICP), cerebral perfusion pressure (CPP), and aerobic brain metabolism, together with diminishing secondary brain injury.[1] Low brain tissue oxygen tension ($PbtO_2$), or brain hypoxia, is an independent predictor of poor outcome.[7] The $PbtO_2$ monitor can be useful in evaluating cerebral ischemia after traumatic brain injury (TBI), aneurysmal subarachnoidal hemorrhage (SAH), intracerebral hemorrhage, and stroke.[7]

Normobaric hyperoxia enhances aerobic metabolism in brain and therefore may be neuroprotective. It is achieved

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by increasing inspirational fraction of oxygen (FiO_2) resulting in supraphysiological arterial oxygen tension. Optimal arterial oxygen tension (PaO_2) should be 13.3 kPa (100 mm Hg), although higher values of PaO_2 in the first 24 h do not increase predicted 6 months mortality.[10] Increasing FiO_2 could have a significant influence on treating lower $PbtO_2$.[9]

Combined $PbtO_2$ therapy, compared to the approach that focus only on regulation of CPP and ICP, shows better patient outcomes.[9]

The Licox is triple-lumen catheter that is inserted through intracranial bolt and measures ICP, $PbtO_2$, and brain tissue temperature. It may detect poor $PbtO_2$ before ICP rise. Likewise, it may help in defining optimal CPP values. Therefore, by individualizing the treatment of patients, monitoring of $PbtO_2$ could be helpful in preventing or limiting secondary brain injury and maintaining better patient outcome.[8] To prevent hypooxygenation, $PbtO_2$ should be maintained above 20 mm Hg.[2] Ericsson *et al.*[4] showed that even $PbtO_2 < 29$ mm Hg in the first 72 h increases mortality.

CASE DESCRIPTION

We present a case report of a patient with SAH, to whom Licox monitor was implanted. The patient, aged 35, was admitted to hospital after rupture of right medial cerebral artery aneurysm, scored 3 on the Hunt and Hess Scale. After neurosurgical procedure of aneurysmal clipping, a Licox monitor was implanted in her penumbra region, as well as the **CODMAN®MICROSENSOR®** ICP monitoring. She was transferred to the neuroanesthesia intensive care unit (ICU), intubated, and mechanically ventilated. During the first 24 h in the ICU, normobaric hyperoxia was applied (FiO_2 80%) and the $PbtO_2$ was kept between 20 and 35 mm Hg. On the second day, FiO_2 was set down to 40% and a decline in $PbtO_2$ from 20 to 10 mm Hg was noticed, so FiO_2 was set to 50% with rise in $PbtO_2$ again. During that time peripheral capillary oxygen saturation (SpO_2) was 97–100%.

Invasive arterial pressure was monitored and maintained in adequate range and other means of brain relaxation and possible neuroprotection were already met by meticulous neurointensive treatment (normocapnia, hypertonic saline and high normal blood sodium, analgosedation and relaxation, addition of magnesium). By introducing Licox system of PbO_2, we were able to monitor closely PbO_2 of the affected brain that was reacting. After aforementioned neurointensive treatment was not giving any further result by not elevating further PbO_2, we decided to elevate FiO_2, which produced instantaneously positive results visible on the Licox monitor and hence positive clinical outcome on patient morbidity [Figure 1]. During that period measured ICP was below 20 mm Hg.

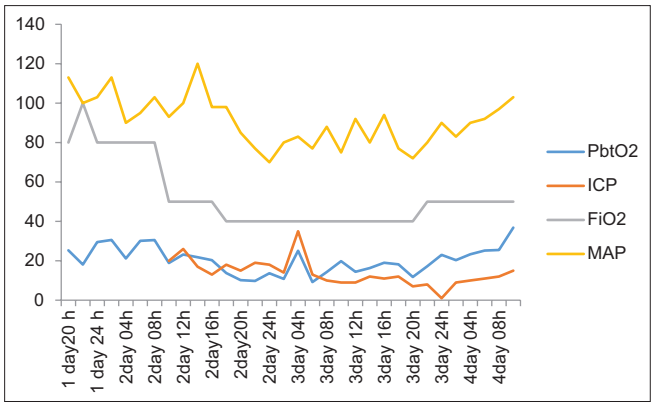

Figure 1:Values of $PbtO_2$, ICP, FiO_2, and MAP over time

On the fourth day, the Licox was removed, according to the guidelines for Licox monitoring use.

Progressive recovery was obvious, and the patient was dismissed with the Glasgow Coma Score (GCS) of 4/5/6. Glasgow Outcome Score after rehabilitation was 5, and Glasgow Outcome Score Extended was 8; no neurologic sequelae were present.

DISCUSSION

Evaluating brain aerobic metabolism and oxygenation based only on PaO_2 or SpO_2 values may be inadequate and may mislead to the conclusion based on the insufficient data. Also cerebral hypoxia and ischemia can develop in the normal range of CPP and ICP. Additional measurement of $PbtO_2$ in penumbra region is therefore needed, and according to the results, it is possible to adjust the patient therapy toward better cerebral oxygen delivery and utilization ratio.

There is still much controversies about the clinical effect of normobaric hyperoxia in trauma brain injuries (lesser) and/or ischemic brain lesions (greater). Our goal was to circumvent the possible further brain injury and prevent any secondary complications by simply elevating PbO_2 in the given delicate moment of the patient treatment, because at that point current standard neurointensive treatment was not met by the criteria given by the Licox system probe measurement of the PbO_2.

Normobaric hyperoxia can be beneficial to specific group of patients, and the efficiency of its administration may be monitored with Licox. Therefore, the use of Licox may be useful for monitoring the local tissue values which are useful for induction of normobaric hyperoxia and optimizing the therapy toward more target defined values. $PbtO_2$ monitoring is an important part of multimodal neuromonitoring in the perioperative and ICU settings, and is the gold standard for brain oxygenation monitoring.[7]

Most studies using tissue oxygen monitors treat initial desaturation episodes with 100% inspired oxygen rather

than a transfusion of red blood cells or vasopressor administration to improve CPP.[11] Moreover, it is considered that aggressive attempts to maintain CPP in the presence of cerebral ischemia with fluids and vasopressors should be avoided due to acute lung injury.[1,3]

Both normobaric hyperoxia and hyperbaric hyperoxia may improve cellular redox state and cerebral metabolic rate for oxygen after TBI in the presence of $PbtO_2$ values that are within or above the normal physiological range.[6]

In our experience, we have noticed the decrease of $PbtO_2$ values during transport of the patient to the operating room, or to the computed tomography (CT) scan. We have also noticed the decrease of $PbtO_2$ values during mechanical ventilation with PEEP. PEEP is known to obstruct cerebral venous return, which may cause higher ICP,[5] resulting in lower brain tissue oxygenation.

For reliable results, proper positioning of $PbtO_2$ monitoring system should always be confirmed with the CT brain scan, which is usually performed within 8 h from the placement of the probe, as well as performing the oxygen test.

CONCLUSION

In our experience with Licox, there was no complications in 5-year period in sense of intracerebral bleeding. In that time we had 20 patients to whom Licox was implanted. Only two of them had lethal outcome, but they were already admitted to the ICU with GCS 1/1/1 and dilated and fixed pupils. The management differs in patients with Licox compared to those who had only ICP monitoring. This case shows how $PbtO_2$ values were low in spite of adequate PaO_2 and SpO_2 values. With individualizing treatment and increasing FiO_2 according to $PbtO_2$ values, subsequently those values have risen. Enhancing cerebral oxygenation may lead to better patient outcome.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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