
ICU Volume 13 - Issue 3 - Autumn 2013 - Matrix Features

Ventilation After Cardiac Arrest

Authors

Yuda Sutherasan, MD

IRCCS AOU San Martino-IST

Department of Surgical Sciences and Integrated Diagnostics

University of Genoa, Genoa, Italy

Ramathibodi Hospital, Mahidol University

Bangkok, Thailand

sutherasan_yuda@yahoo.com

Iole Brunetti, MD

IRCCS AOU San Martino-IST

Department of Surgical, Sciences and Integrated

Diagnostics University of Genoa, Genoa, Italy

ibrunetti@tin.it

Paolo Pelosi, MD

Professor in Anaesthesiology

IRCCS AOU San Martino-IST

Department of Surgical, Sciences and Integrated Diagnostics University of Genoa

Editorial Board Member, ICU Management ppelosi@hotmail.com

Post-cardiac arrest syndrome is associated with high morbidity and mortality not only from poor neurological outcome but also from respiratory dysfunction. Optimising ventilation and gas exchange by protective ventilation, keeping normoxia and avoiding hypocapnia may play a relevant role in improving outcome. Furthermore, transcranial Doppler (TCD) can be considered as a new non-invasive monitoring tool.

Introduction

For decades, physicians have focused on how to improve outcome in post-cardiac arrest patients. They have used several methods to optimise neurological recovery such as therapeutic hypothermia, control of seizure and blood sugar during the 'golden period' after return of spontaneous circulation (ROSC). However, non-neurological

post-cardiac arrest syndrome is another issue that should be of concern.

Not only neurological dysfunction but also cardiovascular dysfunction and respiratory impairment have an impact on mortality. However, there are few data in terms of ventilator strategy in patients after cardiac arrest. In this review, the authors aim to describe the outcome of organ failure after cardiac arrest, the effect of protective ventilation, the goal of gas exchange and a non-invasive method for cerebral blood flow monitoring.

Outcome and Organ Failure After Cardiac Arrest

After cardiac arrest, mortality and bad neurologic consequences become the major concern. Therapeutic hypothermia improves neurological outcome and decreases the mortality rate (Nolan et al. 2010). Several studies have demonstrated that neurological abnormalities mainly impact on worse clinical outcome. Glasgow-Pittsburgh Cerebral Performance Categories and prognostic biomarkers such as serum neuron-specific enolase (NSE) and S100B are known as predictors for neurological outcome (Rana et al. 2012; Rana et al. 2011).

In their recent large prospective cohort study for evaluating outcome of patients with mechanical ventilation, in 1998, 2004 and 2010, Esteban et al. demonstrated that intensive care unit and in-hospital mortality are decreased over time. However, the incidence of cardiac arrest in this group rose from 2% to 5% despite a decrease in the incidence of congestive heart failure (CHF) from 10% to 6-8%. The mortality in patients with cardiac arrest was still between 44-48% (Esteban et al. 2013).

Roberts et al. (2013a) reported that the highest cardiovascular and respiratory specific Sequential Organ Failure Assessment (SOFA) scores were associated with in-hospital mortality in 203 post-cardiac arrest patients, in which the majority of causes of cardiac arrest were pulseless electrical activity and asystole initial rhythm. The respiratory SOFA score is the degree of oxygen impairment. In an animal experimental model, Wang et al. (2013) have demonstrated that dead space, airway resistance and lung elastance are increased after ROSC. The previously mentioned findings represent the value of haemodynamic and respiratory optimisation during the post-cardiac arrest period.

Protective Mechanical Ventilation

Inappropriate mechanical ventilator settings can be harmful to the lungs even in non-pre-existing lung injury. The mechanisms of ventilator-induced lung injury are as follows:

1) overstretching from high tidal volume (VT); 2) repeated recruitment and de-recruitment of unstable lung units (Whitehead and Slutsky 2002) and; 3) peripheral airway collapse at low end expiratory lung volume from cyclic open and closing of peripheral airway (Pelosi and Rocco 2007). In an animal experimental model low VT ventilation with positive end expiratory pressure (PEEP) showed a favourable outcome in terms of decreased lung inflammation compared to high VT without PEEP (Curley et al. 2011; Pelosi and Rocco 2011). In clinical investigations, a recent meta-analysis has demonstrated that low VT ventilation in patients without preexisting lung injury is associated with lower incidence of acute respiratory distress syndrome, overall mortality and pulmonary infection. The authors suggest avoiding VT more than 10 ml/kg even with no lung injury (Serpa Neto et al. 2012).

Although current evidence suggests protective ventilation in patients without pre-existing lung injury, there is no sufficient data in terms of optimal tidal volume to decrease lung injury and mortality in post-cardiac arrest after ROSC. Further study should be warranted in the future.

On a physiologic basis, the application of PEEP might worsen haemodynamic status in patients with either acute myocardial infarction or CHF. However, some evidence suggests that PEEP can decrease oxygen demand, increase oxygen delivery and decrease intracardiac lactate production (Wiesen et al. 2013). According to the principle of heartlung interaction, positive pressure ventilation (PPV) has a positive effect on haemodynamic parameters, namely decreased left ventricular (LV) preload and LV afterload from decreased intra-thoracic transmural pressure (Pinsky, 2005). The PPV can reduce metabolic demand and decrease hypoxaemic-induced pulmonary vasoconstriction. Furthermore, PEEP aggravates the shifting of fluid from alveoli and interstitial space back to the circulation (Fernandez Mondejar et al. 1996). Several prospective studies have demonstrated that PEEP can improve pulmonary capillary wedge pressure, cardiac index, stroke index and ability to wean from mechanical ventilation (Jubran et al. 1998; Lenique et al. 1997). Furthermore, application of PEEP at level 5-8 cmH₂O in non-hypoxaemic patients can reduce the incidence of ventilator-associated pneumonia (Manzano et al. 2008). The optimal level of PEEP in post-cardiac arrest patients with cardiogenic shock is still debated, depending on the volume status. Cardiac output monitoring should be warranted during initiation of PEEP (Wiesen et al. 2013).

The Role of O₂ and CO₂

In a multicentre cohort study of 120 hospitals in the United States, Kilgannon et al. (2011) demonstrated that in non-traumatic cardiac arrest adult patients with hyperoxia defined by arterial oxygen tension (PaO₂) more than 300 mmHg was associated with decrease in survival to hospital discharge compared with either normoxia or hypoxia. In addition, exposure to hyperoxia increased the risk of death (OR 1.8 95% confidence interval 1.2–2.2) after adjusting the model for propensity score. Likewise, hypoxia less than 60 mmHg is associated with mortality. However, the authors did not mention the optimal level of PaO₂ and ventilator setting (Kilgannon et al. 2010). The same group demonstrated that each 100 mmHg increase of PaO₂ was associated with a 24% increase in risk of death (Kilgannon et al. 2011). The 100% oxygen supplement after ROSC caused the increase of NSE level compared to the 30% oxygen supplement in the patients who were not treated with therapeutic hypothermia (Kuisma et al. 2006).

The increase of oxidative stress activity by oxidative impairment of mitochondrial respiration is proposed as the main mechanism that causes worsening of brain injury by neuronal damage, particularly during one hour after ROSC (Pilcher et al. 2012; Neumar 2011). We recommend titrating oxygen and keeping pulse oximetry between 94% and 98% (Nolan et al. 2010) or PaO₂ between 60-100 mmHg instantly after ROSC. During CPR, there are insufficient data about the optimised level of oxygen (Nolan et al. 2010).

Mild hypercarbia can improve cerebral perfusion by cerebral vasodilatation concomitant with decrease of cerebral lactate. It has a protective effect to neurons in terms of seizure threshold and oxidative stress. However, arterial carbon dioxide tension (PaCO₂) more than 100 mmHg causes further brain injury (Zhou et al. 2010). Hypocarbica is associated with decrease of cerebral perfusion and neuronal injury (Pynnonen et al.

2011; Tolner et al. 2011). In a recent cohort study, hypocapnia ($\text{PaCO}_2 < 35 \text{ mmHg}$) was associated with worse clinical outcome compared to normocapnia (PaCO_2 between 35 and 45 mmHg) and hypercapnia ($\text{PaCO}_2 > 45$). Additionally Schneider et al. (2013) have reported that hypercapnia is associated with the greater chance of discharge home. However, $\text{PaCO}_2 \geq 50 \text{ mmHg}$ is associated with poor neurological outcome defined by cerebral performance category ≥ 3 at discharge. Therefore, we recommend keeping PaCO_2 between 35 and 45 mmHg in adults after cardiac arrest (Roberts et al. 2013b). However, only 55% of cardiac arrest patients after successful onsite resuscitation achieve PaCO_2 of 35-45 mmHg during mild therapeutic hypothermia (Falkenbach et al. 2009).

In paediatric cardiac arrest, a retrospective cohort study demonstrated that neither $\text{PaO}_2 > 200 \text{ mmHg}$ nor $< 50 \text{ mmHg}$ are associated with worse outcome (Bennett et al. 2013). The level of $\text{PaCO}_2 < 30 \text{ mmHg}$ and $> 50 \text{ mmHg}$ are associated with poor outcome with OR of 2.71 (95% CI 1.04-7.05) and 3.27 (95% CI 1.62-6.61) respectively (Del Castillo et al. 2012).

Doppler as an Adjunctive Tool For Non-Invasive Cerebral Blood Flow Monitoring

Since in patients with brain injury and post anoxic-ischaemic encephalopathy PEEP can increase intracranial pressure (Videtta et al., 2002), and therapeutic hypothermia can decrease CO_2 production (Polderman, 2004), that may have the consequence of worsening cerebral blood flow. Ventilator settings will need to be changed during induced hypothermia and during the rewarming phase. Physicians need a specific guided tool for monitoring cerebral blood flow (CBF).

Trans-Cranial Doppler (TCD) ultrasonography is a bedside and non-invasive technique that allows repeated or continuous monitoring of blood flow velocity in the major intracranial arteries. Mean flow velocity cannot be directly interpreted as volume blood flow due to the unknown diameter of the insonated vessel, but it is possible to derive interesting additional information about cerebral haemodynamics from the TCD waveform analysis (Moppett and Mahajan 2004).

Since its introduction by Aaslid and colleagues (1982) TCD has rapidly evolved, and its application in critical care and research has expanded. The most widespread application of TCD is for the detection of vasospasm in patients with subarachnoid haemorrhage. TCD is also being studied as a noninvasive estimator of intracranial pressure (ICP) in patients with severe traumatic brain injury and in the setting of clinical brain death (Rasulo et al. 2008). In post-cardiac arrest syndrome TCD has been proposed to investigate CBF modifications during the first 72 hours in patients treated with therapeutic hypothermia, and their correlation with neurological outcome (Lemiale et al. 2008).

TCD is performed using a low-frequency probe (usually 2 MHz) to penetrate thin areas of the skull with an ultrasonic beam emitted in a range-gated, pulsed-wave manner. Three main ultrasonic windows are temporal, orbital and foramen magnum. The most popular window is the temporal window, which is located between the tragus and the ipsilateral eye. This window allows insonation of the anterior, middle, and posterior cerebral arteries, the terminal segment of the internal carotid artery, and the anterior and posterior communicating arteries. The patient should be in the supine position, and the probe faces perpendicular to the temporal bone to insonate. The middle cerebral artery is usually insonated at a depth of 35 to 55 mms. The anterior cerebral artery and posterior cerebral artery are insonated at 60 to 70 mms. The best quality signal can be obtained by making small adjustments in probe position and angle. It is possible to conduct the exam with a 'blind' probe to identify vessels from insonation depth and waveform, but it is also possible to use an echo colour- Doppler probe to visualise the main intracranial arteries and position the pulse wave Doppler-sample on the identified vessels. When flow velocity is displayed, the peak systolic velocity (PSV) and the end diastolic velocity (EDV) can be recorded and the mean velocity (MV) calculated. Dedicated TCD devices also calculate automatically derived parameters such as resistance index (RI) and pulsatility index (PI).

Different information can be obtained from TCD, depending on the clinical setting. Elevated flow velocities are associated with increased CBF, anaemia and cerebral vascular abnormalities (arterial stenosis, vasospasm or arterio-venous malformations), while low flow velocities may indicate a proximal flow-reducing lesion, a state of decreased cerebral metabolic rate (for example, during coma) or a low CBF such as in poor cardiac output conditions. The Gosling PI (peak systolic flow velocity – end diastolic flow velocity / mean flow velocity) is an index proposed to quantify waveform, and is not affected by the angle of insonation. Normal range is from 0.6 to 1.1 and PI variations reflect changes in resistance to flow in specific areas of the cerebral circulation (Czosnyka et al. 1996). A higher than expected PI might result from a distal occlusion, raised intracranial pressure (ICP) or hypocarbia. A PI below 0.5 suggests a proximal flow-reducing lesion, such as an extracranial stenotic lesion, or a low intracranial resistance (arterio-venous malformation) (see Figure 1). So PI was proposed for non-invasive monitoring of ICP in traumatic brain injury, but its accuracy and reliability remain controversial (Bellner et al. 2004; Behrens et al. 2010; Zweifel et al. 2012). Recent literature underlines that it is possible to describe PI as a complex mathematical function not dependent solely on CVR: it is a product of the interplay between CPP, pulse amplitude of arterial pressure, cerebrovascular resistance (carbon dioxide reactivity) and compliance of the cerebral arterial bed as well as the heart rate (De Riva et al. 2012). Particularly in post-cardiac arrest comatose patients, a better knowledge of cerebral haemodynamic changes during the post-resuscitation period is essential to treat patients correctly. As with other techniques routinely used in neuro intensive care monitoring, TCD could add complementary information, but more studies are warranted to define its role in clinical practice.

Key Messages

- In adult patients after cardiac arrest, non-neurological organ failures affecting mortality are respiratory and cardiovascular failure.
- Protective ventilation by low tidal volume ventilation may decrease pulmonary dysfunction and improve the outcome. Low level of PEEP can be initiated and titrated with careful cardiac output monitoring.
- In paediatric patients after cardiac arrest, the optimal PaO_2 level is between 60 -100 mmHg and pulse oximetry between 94- 96 (98) %.

Hypocapnia ($\text{PaCO}_2 < 35 \text{ mmHg}$) and hypercapnia ($\text{PaCO}_2 > 45 \text{ mmHg}$) are detrimental.

- In adults, hyperoxia and hypoxia are detrimental. We recommend titrating oxygen and keeping pulse oximetry between 94% and 98 % or PaO_2 between 60-100 mmHg. We should avoid hypocapnia ($\text{PaCO}_2 < 35 \text{ mmHg}$).
- TCD is the visualised non-invasive tool that may play an important guided tool during ventilator setting.

Acknowledgements

We would like to thank Dr. Pezzato Stefano for providing figure material.

For full references, please send a request to editorial@icu-management.org

Published on : Wed, 2 Oct 2013