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Clinical Paper

Hemodynamic targets during therapeutic hypothermia after cardiac arrest: A prospective observational study☆

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ABSTRACT

Aim: In analogy with sepsis, current post-cardiac arrest (CA) guidelines recommend to target mean arterial pressure (MAP) above 65 mmHg and SVO_2 above 70%. This is unsupported by mortality or cerebral perfusion data. The aim of this study was to explore the associations between MAP, SVO_2, cerebral oxygenation and survival.
Methods: Prospective, observational study during therapeutic hypothermia (24 h – 33 °C) in 82 post-CA patients monitored with near-infrared spectroscopy.
Results: Forty-three patients (52%) survived in CPC 1–2 until 180 days post-CA. The mean MAP range associated with maximal survival was 76–86 mmHg (OR 2.63, 95%CI [1.01; 6.88], $p = 0.04$). The mean SVO_2 range associated with maximal survival was 67–72% (OR 8.23, 95%CI [2.07; 32.68], $p = 0.001$). In two separate multivariate models, a mean MAP (OR 3.72, 95% CI [1.11; 12.50], $p = 0.03$) and a mean SVO_2 (OR 10.32, 95% CI [2.03; 52.60], $p = 0.001$) in the optimal range persisted as independently associated with increased survival. Based on more than 1 625 000 data points, we found a strong linear relation between SVO_2 (range 40–90%) and average cerebral saturation (R^2 0.86) and between MAP and average cerebral saturation for MAP's between 45 and 101 mmHg (R^2 0.83). Based on our hemodynamic model, the MAP and SVO_2 ranges associated with optimal cerebral oxygenation were determined to be 87–101 mmHg and 70–75%.
Conclusion: we showed that a MAP range between 76–86 mmHg and SVO_2 range between 67% and 72% were associated with maximal survival. Optimal cerebral saturation was achieved with a MAP between 87–101 mmHg and a SVO_2 between 70% and 75%. Prospective interventional studies are needed to investigate whether forcing MAP and SVO_2 in the suggested range with additional pharmacological support would improve outcome.

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1. Introduction

The relationship between global hemodynamics, cerebral oxygenation and survival has not been investigated in post-cardiac arrest patients.[1] In the absence of strong evidence, current guidelines are based on the assumption that the post-cardiac arrest syndrome is a sepsis-like syndrome. Therefore, it is recommended to target mean arterial pressure (MAP) above 65 mmHg, systolic blood pressure above 90 mmHg and mixed venous oxygen saturation (SVO_2) above 70%.[2] However, the post-cardiac arrest syndrome is clearly a distinct and more complex entity than a sepsis-like syndrome alone, and aiming for the same hemodynamic goals is probably oversimplification. First, in critically ill patients without primary brain injury (e.g. sepsis), cerebral perfusion is kept stable in a broad range of blood pressures by cerebral autoregulation. In contrast, in a subset of post-cardiac arrest patients the lower threshold of cerebral autoregulation is shifted rightward and these patients might benefit from resuscitation to higher MAP's.[3] Second, patients with a reduced left ventricular function might benefit from afterload reduction to maintain stroke volume and cerebral perfusion.[4] The optimal MAP should maintain cerebral perfusion without exposing the damaged myocardium to excessive

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afterload.[5] Therefore, the aims of this prospective observational study were to explore the relationships between global hemodynamics, cerebral oxygenation and outcome in post-cardiac arrest patients during therapeutic hypothermia in the first 24 h after ICU admission.

2. Methods

2.1. Study population

All comatose survivors after non-traumatic cardiac arrest treated in our tertiary care hospital (Ziekenhuis Oost-Limburg, Genk, Belgium) were prospectively enrolled in this study. Patients could have been resuscitated in-hospital, referred by another hospital or admitted by our emergency ward. All patients were treated uniformly according to the institutional post-cardiac arrest protocol. Patients were routinely monitored by cerebral saturation and invasive arterial blood pressure monitoring on hospital arrival and by pulmonary artery catheter (PAC) shortly after admission on the cardiac intensive care unit unless PAC placement was contra-indicated or considered inappropriate by treating physicians. Written informed consent was obtained from a next of kin. Five patients with refractory shock who died during the first 24 h were excluded from analysis. The study protocol was approved by the local medical ethics committee.

2.2. General management

Our post-cardiac arrest protocol has been described previously.[7] Shortly, all patients were intubated, mechanically ventilated and sedated with propofol and remifentanil if hemodynamically tolerated. Cisatracurium was administrated in case of shivering during hypothermia. Unless an obvious non-cardiac cause could be identified, all patients were referred for urgent coronary angiography. Therapeutic hypothermia was induced in all patients shortly after admission by cold saline (4 °C – 30 ml/kg) and further mechanically maintained in the ICU by endovascular (Icy-catheter, CoolGard® 3000, Alsius, Irvine, CA, USA) or surface (ArcticGelTM pads, Arctic Sun® 5000, Medivance, Louisville, Colorado, USA) cooling systems at 33 °C for 24-h. After rewarming (0.3 °C/h) sedation was titrated toward patient's comfort. Patients were extubated after sufficient recovery.

2.3. Cerebral saturation monitoring

Cerebral tissue oxygen saturation was continuously measured with near infrared spectroscopy (NIRS), using the FORE-SIGHT™ technology (CAS Medical Systems, Branford, CT, USA). Sensors were bilaterally applied to each frontotemporal area before the start of mechanically induced hypothermia. Sensors were covered to prevent ambient light interference. Cerebral saturation data were transmitted electronically to a personal computer with a 2 s time interval. Cerebral saturation data were not used to guide any form of hemodynamic management.

2.4. Hemodynamic monitoring and management

Patients were treated according to the recommended guidelines.[2] If signs of inadequate circulation persisted despite correct fluid resuscitation (wedge pressure > 18 mmHg), norepinephrine was infused with a target MAP > 65 mmHg and subsequently dobutamine to target a cardiac index > 2.2 L/min/m^2. An IABP was installed as deemed necessary by treating physicians. A transthoracic echocardiography was performed in the first 24 h after admission. Continuous thermodilution cardiac output and SVO_2 were measured continuously by a new generation PAC (CCOmbo PAC®, Edwards Life Science, Irvine, CA, USA) connected to the appropriate monitor (Vigilance II®, Edwards Life Science, Irvine, CA, USA). The continuous SVO_2 monitoring system was calibrated as prescribed by the company. These data were transmitted electronically to a personal computer with a 2 s time interval, together with information on blood temperature, oxygen saturation, MAP obtained from a radial artery line and cerebral saturation data. In the first 29 patients enrolled in the database, the hemodynamic data were written down manually every 15 min.

2.5. Statistics

Results are expressed as mean (±SD, standard deviation) unless otherwise stated. Survival was defined as survival in CPC 1–2 until 180 days post-CA. First, for each patient, the mean SVO_2 and MAP were calculated by averaging all obtained values during the first 24 h after the first SVO_2 value (or MAP value in patients not monitored with a PAC). To determine the MAP and SVO_2 associated with maximal survival, odds ratios (and 95% confidence intervals) were calculated per 5% SVO_2 and per 10 mmHg MAP intervals. To test for significance, a Chi-square test was performed for the interval with the highest odd's ratio (all expected frequencies were more than 5). Additionally, the percentage of time in the obtained optimal SVO_2 and MAP range was calculated for each patient. Univariate logistic regression was used to test for significance (assuming that a higher percentage of time spent in the suggested SVO_2 or MAP range would result in better survival). Similarly, other candidate binary variables were evaluated by a Chi-square test and univariate logistic regression was used for continuous candidate variables. A multivariate model was constructed using backward multivariate logistic regression with all candidate variables (age, bystander CPR < 10 min, shockable rhythm, initial lactate, mean lactate, left ventricular ejection fraction). A separate multivariate model was constructed for MAP and SVO_2.

Second, patients were stratified to be in the low/optimal/high MAP and SVO_2 subgroups according to their average values during the first 24 h after admission. These subgroups were compared using one-way ANOVA.

Third, to construct a hemodynamic model, the average cerebral saturation was calculated per mmHg MAP (range 45–110 mmHg) and per % SVO_2 (range 40–90%) and the average SVO_2 was calculated per mmHg MAP. Univariate linear regression with calculation of Pearson's correlation coefficient was used to describe the relationships between MAP, SVO_2 and cerebral saturation. Assuming that the optimal MAP range based on this hemodynamic model has to maintain cerebral oxygenation without exposing the damaged myocardium to excessive afterload, the lowest optimal MAP was defined as the lowest MAP with a corresponding cerebral saturation equal to the average of all cerebral saturations associated with all higher MAP's than the taken MAP value. The highest optimal MAP was defined as the MAP associated with maximal cerebral saturation. The optimal hemodynamic SVO_2 range was considered to be corresponding with this optimal MAP range. Statistical analysis was performed using Matlab software (version R2010b, Mathworks, USA). A p-value <0.05 was considered significant.

3. Results

3.1. Patients

Eighty-two patients were included in the study. Baseline characteristics are summarized in Table 1. Sixty-three patients were monitored by a PAC (63/82, 77%). In 41 of these patients, data were

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