A case report: use of cerebral oximetry in the early detection of cerebral hypoperfusion in a post-cardiac arrest patient during targeted temperature management

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Background

We present a patient who received cerebral oximetry monitoring during targeted temperature management (TTM) post-cardiac arrest and discuss its potential in the early detection of cerebral hypoperfusion and implications on haemodynamics and ventilatory management.

Case summary

A 60-year-old Chinese male was admitted for acute pulmonary oedema with Type 2 respiratory failure. He failed an initial trial of non-invasive ventilation and was planned for intubation and mechanical ventilation. However, the patient suffered a pulseless electrical activity cardiac arrest peri-intubation. He was started on our institution’s protocolized post-cardiac arrest care bundle, which included cerebral regional oxygen saturation (rSO2) monitoring and TTM. Initial arterial blood gas (ABG) post-return of spontaneous circulation showed severe respiratory acidosis, and the patient was sedated, paralyzed, and ventilator settings optimized. Repeat ABG showed resolution of respiratory acidosis. However, a drop in rSO2 to 35% was subsequently noted. Ventilator settings were quickly adjusted, and dobutamine was started to improve global and cerebral perfusion. These measures improved cerebral rSO2 to more than 50%. Patient was cooled for 24 h and gradually rewarmed. He was later extubated with a cerebral performance category of 1 and is now on outpatient follow-up.

Discussion

During post-cardiac arrest care, there are many factors which can contribute to a decrease in cerebral blood flow. Therapeutic hypothermia and ventilation strategies, including the use of neuromuscular blocking agents, can both reduce pCO2 which is a major regulator of cerebrovascular tone. Accidental hypocapnia can lead to adverse cerebral vasoconstriction and hypoperfusion. Without cerebral oximetry, cerebral ischaemia may not be detected early and can potentially result in secondary brain injury.

Keywords
Case report • Cerebral oximetry • Hypocapnia • Post-cardiac arrest care • Targeted temperature management
Learning points
- During post-cardiac arrest care, haemodynamic changes, ventilation strategies, and therapeutic hypothermia may lead to a decrease in cerebral blood flow.
- Cerebral oximetry offers real-time monitoring of brain tissue oxygenation and serves as a safety measure during targeted temperature management and haemodynamic and ventilator titration.
- Cerebral oximetry can help in the early detection of cerebral hypoperfusion and prevent secondary brain injury.

Introduction
Cerebral oxygenation can be monitored non-invasively with the use of near-infrared spectroscopy (NIRS) to determine brain regional oxygen saturation (rSO2). In post-cardiac arrest patients, there is often global cerebral ischaemia contributing to neurological dysfunction. Targeted temperature management (TTM) is often implemented to limit neurological injury caused by the hypoxia during cardiac arrest and help improve survival, as shown in the Hypothermia after Cardiac Arrest Study Group trial.1 During TTM post-cardiac arrest, it is crucial to optimize cerebral perfusion to achieve good neurological recovery. We present a case where the use of cerebral oximetry monitoring post-cardiac arrest led to the early detection of cerebral hypoperfusion and discuss its implications on management and outcome.

Timeline

| Clinical events                      | Patient admitted for acute pulmonary oedema with Type 2 respiratory failure and started on trial of non-invasive ventilation |
|-------------------------------------|-------------------------------------------------------------------------------------------------------------------------|
| Admission                           | Intubated in view of worsening hypoxaemia                                                                              |
| PEA collapse (PEA) collapse          | Total downtime 20 min                                                                                                  |
| Return of spontaneous circulation (ROSC) | Initial arterial blood gas (ABG) post-ROSC: pH 7.14, pCO2 54, pO2 110, HCO3 18, and SaO2 97%                         |
| Measures                             | Started on regional cerebral oxygen saturation (rSO2) monitoring: 66% (left) and 67% (right)                           |
| Drop in rSO2                         | rSO2 dropped to 33% (left) and 35% (right)                                                                             |
| Repeat ABG: pH 7.3, pCO2 33, pO2 138, HCO3 16, and SaO2 99%                                                           |
| Central venous oxygen saturation (ScvO2) gap 14 mmHg suggesting a low flow state                                     |
| Measures                             | Ventilation rate decreased to target CO2 50–55 mmHg                                                                    |
| TTM complete                         | Cooled for total 24 h, then gradually rewarmed                                                                          |
| Outcome                             | Extubated successfully with good neurological recovery                                                                  |

Case presentation
A 60-year-old Chinese male presented to the hospital with acute shortness of breath and lower limb swelling. He has a past medical history of stroke disease, chronic kidney disease, and ischaemic cardiomyopathy with a mildly reduced left ventricular ejection fraction of 45%. His cardiovascular risk factors include diabetes mellitus, hypertension, and hyperlipidaemia.

Clinical examination revealed bibasal crepitations with bilateral pitting oedema. The blood pressure on admission was 150/87 mmHg with sinus tachycardia with a heart rate of 129 beats per minute.

Initial arterial blood gas (ABG) on admission (Table 1) revealed Type 2 respiratory failure, while chest X-ray done showed bilateral pulmonary congestion.

He was initially started on non-invasive ventilation and intravenous frusemide and glyceryl trinitrate infusion. Despite initial clinical improvement, the patient became increasingly restless with...
worsening hypoxaemia and decision was made for intubation and mechanical ventilation. However, he suffered a pulseless electrical activity cardiac arrest peri-intubation with a low-flow time of 20 min before return of spontaneous circulation (ROSC).

He was started on our institution’s protocolized post-cardiac arrest care bundle (Figure 1) which included continuous rSO2 monitoring and TTM with mild induced hypothermia at 33°C. An oesophageal probe was inserted for core body temperature monitoring. Initial ABG post-ROSC revealed a Type 2 respiratory failure with pH 7.14, pCO2 54, pO2 110, HCO3 18, and SaO2 97%. Patient was sedated and paralyzed and ventilator settings were optimized to correct the respiratory acidosis. Patient was ventilated with a tidal volume of 7 mL/kg predicted body weight and at a ventilator rate of 28 breaths per minute. Initial cerebral oximetry revealed an rSO2 66% (left) and 67% (right).

Post-resuscitation SpO2 was maintained above 94% and mean arterial pressure (MAP) was constantly above 80 mmHg without the need for inotropes or vasopressors. However, it was subsequently noted that the cerebral rSO2 had decreased to 33% (left) and 35% (right) and a repeat ABG showed resolution of the respiratory acidosis with pH 7.3, pCO2 33, pO2 138, HCO3 16, and SaO2 99%.

Ventilator settings were immediately adjusted and the minute ventilation reduced by lowering the ventilator rate to aim for mild therapeutic hypercapnia with a target pCO2 of 50–55 mmHg. PEEP was reduced from 12 cmH2O to avoid an excessively high intrathoracic pressure which may impede cerebral venous drainage. Patient was also started on dobutamine infusion to improve the global and cerebral perfusion as the central venous oxygen saturation (SvO2) of 62% reflected a systemic oxygen delivery/consumption mismatch, while a Pcv-aCO2 gap of 14 mmHg suggested a low-flow state. The above interventions resulted in an improvement in cerebral rSO2 to 55% (left) and 51% (right) (Table 2).

Patient was cooled for 24 h and then gradually rewarmed. A repeat transthoracic echocardiogram revealed severe global hypokinesia with a left ventricular ejection fraction of 26%.

He was subsequently extubated after 6 days in the cardiac intensive care unit with improvement in Glasgow coma scale score and underwent rehabilitation in the general ward. On discharge, he had a cerebral performance category of 1 and is now on follow-up in the outpatient clinic.

### Table 1 Investigations on admission

| ABG                           | Troponin | Creatinine | Haemoglobin |
|-------------------------------|----------|------------|-------------|
| pH 7.12                       | 47 ng/L  | 206 μmol/L | 8.9 g/dL    |
| pCO2 51 mmHg                  |          |            |             |
| pO2 45 mmHg                   |          |            |             |
| HCO3 17 mmol/L                |          |            |             |
| SaO2 64%                      |          |            |             |
| Troponin 47 ng/L              |          |            |             |
| Creatinine 206 μmol/L         |          |            |             |
| Haemoglobin 8.9 g/dL          |          |            |             |

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**Figure 1** Cardiac intensive care unit management of post-cardiac arrest syndrome (the ‘A to I’ approach).
| Date            | 19 April 2018 | 19 April 2018 | 20 April 2018 | 20 April 2018 | 20 April 2018 | 20 April 2018 | 20 April 2018 | 20 April 2018 | 20 April 2018 | 20 April 2018 |
|-----------------|---------------|---------------|---------------|---------------|---------------|---------------|---------------|---------------|---------------|---------------|
| Time            | 2000          | 2300          | 0000          | 0300          | 0600          | 0800          | 0900          | 1100          | 1300          | 1500          |
| Prior to collapse |               |               |               |               |               |               |               |               |               |               |
| Post-intubation  |               |               |               |               |               |               |               |               |               |               |
| After correction of respiratory acidosis |               |               |               |               |               |               |               |               |               |               |
| After allowing for permissive hypercarbia and increasing perfusion |               |               |               |               |               |               |               |               |               |               |
| Temperature (°C) | 37.1          | 36.9          | 33.0          | 33.0          | 33.0          | 33.0          | 33.0          | 33.0          | 33.0          | 33.0          |
| Tympanic        |               |               |               |               |               |               |               |               |               |               |
| Oesophageal     |               |               |               |               |               |               |               |               |               |               |
| Heart rate (b.p.m.) | 128          | 161          | 130          | 106          | 96           | 88           | 86           | 88           | 89           | 92           |
| Blood pressure (mmHg) | 152/78 (99)  | 204/145 (169) | 162/78 (102) | 127/66 (84)  | 112/68 (81)  | 125/76 (93)  | 113/98 (105) | 128/72 (90)  | 134/70 (91)  | 168/87 (116) |
| NIBP ABP        |               |               |               |               |               |               |               |               |               |               |
| ABP             |               |               |               |               |               |               |               |               |               |               |
| ABP             |               |               |               |               |               |               |               |               |               |               |
| Respiratory rate | 31            | 38            | 24            | 28            | 28            | 28            | 28            | 22            | 20            | 20            |
| Oxygen saturation (%) | 88            | 80            | 98            | 100           | 98            | 98            | 99            | 97            | 97            | 98            |
| End-tidal CO₂ (mmHg) | 31            | 30            | 23            | 24            | 25            | 30            | 34            | 34            | 34            | 25            |
| ScvO₂ (%)       | 65            | 40            | 30            | 61            | 65            | 74            | 81            |               |               |               |
| rSO₂ (%)        | Left 66       | Left 67       | Left 53       | Left 41       | Left 33       | Left 40       | Left 66       | Left 66       | Left 67       | Left 67       |
|                 | Right 67      | Right 67      | Right 45      | Right 40      | Right 36      | Right 41      | Right 67      | Right 67      | Right 67      | Right 67      |

ABP, arterial blood pressure; NIBP, non-invasive blood pressure.
Early detection of cerebral hypoperfusion in a post-cardiac arrest patient during targeted temperature management

Discussion

It is important to optimize cerebral perfusion after ROSC to improve the number of neurologically intact survivors. There have been studies looking at the correlation of cerebral rSO2 during resuscitation and successful ROSC and it has also been shown that a high rSO2 on arrival at the hospital predicted good 90-day neurologic outcomes.1,3,4 However, equally important to a successful ROSC is the care the patient receives after admission to the intensive care unit. There have been limited tools to guide optimization of cerebral oxygenation during post-cardiac arrest care.

Targeted temperature management is an essential part of the post-ROSC care in cardiac arrest patients and it has been shown to increase the chance of survival with favourable neurological outcomes.5 However, during post-cardiac arrest care, there may be a drop in mean arterial pressure and cardiac output leading to a drop in cerebral blood flow.5 Ventilation strategies including the use of neuromuscular blocking agents together with the reduction of metabolism during therapeutic hypothermia6 also reduce pCO2 which is a major regulator of cerebrovascular tone. Accidental hypocapnia can occur and result in adverse cerebral vasoconstriction and cerebral ischaemia.7,8 This may not be detected early as neurological function is difficult to assess clinically when the patient is sedated and paralyzed during TTM.

Conclusion

Cerebral oximetry offers real-time and dynamic monitoring of brain tissue oxygenation and can potentially serve as a safety measure during the hemodynamic and ventilatory management of patients post-cardiac arrest.9 Cerebral oximetry may be the answer to assessing cerebral perfusion during the crucial period of TTM and aid in preventing secondary iatrogenic brain injury. More studies can be done to assess the morbidity and mortality benefit of cerebral rSO2 monitoring during post-cardiac arrest care, its accuracy in assessing cerebral ischaemia and the cut-off values which would signify sufficient cerebral perfusion to impact good neurological recovery.

Supplementary material

Supplementary material is available at European Journal - Case Reports online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

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Lead author biography

Shonda Ng graduated from the Yong Loo Lin School of Medicine, National University of Singapore (NUS) in 2014. She obtained her Master of Medicine and MRCP (UK) in 2016. She completed her internal medicine residency training in 2017 and is currently a third year senior resident in the National Healthcare Group Cardiology residency programme. She is also an active clinical tutor for medical students from Yong Loo Lin School of Medicine and Lee Kong Chian School of Medicine.