

# Shivering during induced hypothermia after cardiac arrest results in significant decrease in non-invasive cerebral oxygen saturation, as measured by near infrared spectroscopy

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## Introduction

Despite improvements in prehospital and hospital care, the prognosis for cardiac arrest (CA) survivors remains extremely poor. **Moderate hypothermia** (32°-34°C) is the only form of therapy that improves both survival and neurological outcome after cardiac arrest.

Using near-infrared spectroscopy (NIRS), the FORE-SIGHT<sup>®</sup> technology (CAS Medical systems) makes it possible to continuously monitor the **absolute cerebral tissue oxygen saturation (SctO<sub>2</sub>)**. Currently, no data are available on cerebral oxygenation during therapeutic hypothermia (TH) after CA.

In this study, we wanted to monitor the changes in cerebral tissue oxygenation during induced hypothermia after CA.

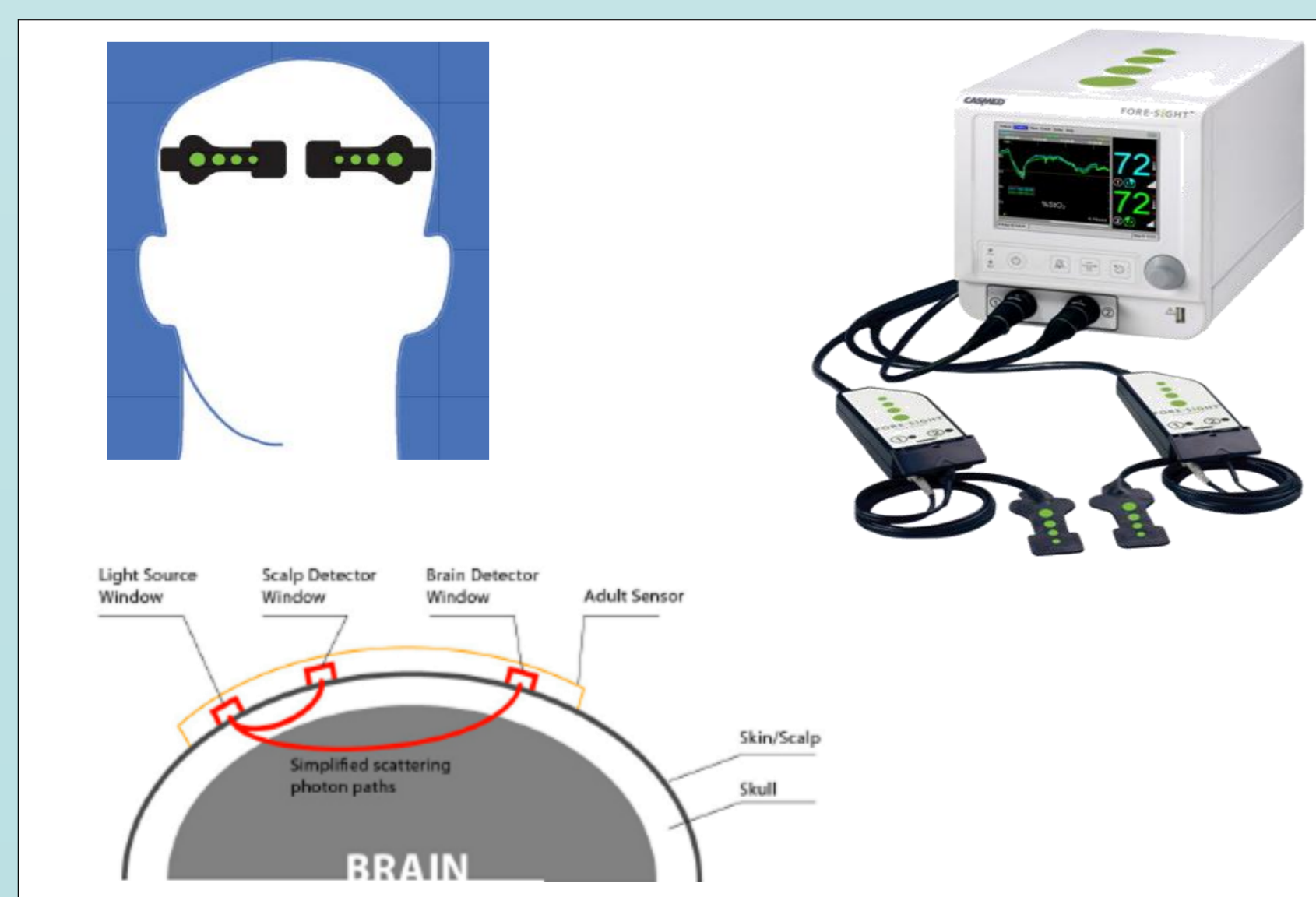
## Patients and methods

After IRB approval, patients undergoing therapeutic hypothermia after cardiac arrest were included. Bilateral SctO<sub>2</sub> monitoring was started when patients arrived at CCU. Cold saline (30-40 ml/kg) was administered as soon as possible. Patients were sedated (propofol/remifentanyl) for the duration of TH.

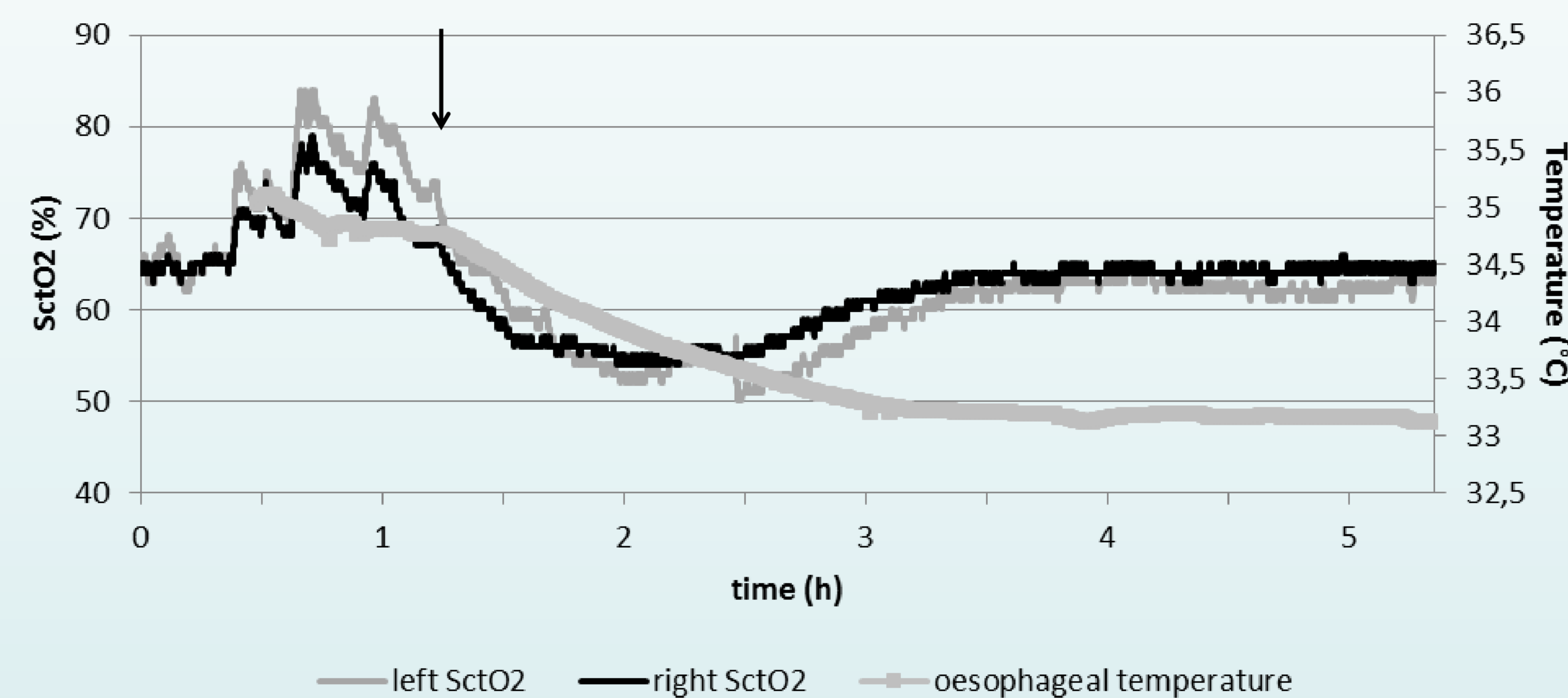
Hypothermia (33°C) was applied for 24 hours using endovascular cooling (n=13; Coolgard system, Alsius) or surface cooling (n=3; Arctic Sun, Medivance). Patients were rewarmed at 0.3°/h.

Sixteen patients (♂/♀=13/3, mean age=67 years) were included in this study. Percutaneous coronary intervention was performed in nine patients, five patients received an intra-aortic balloon pump.

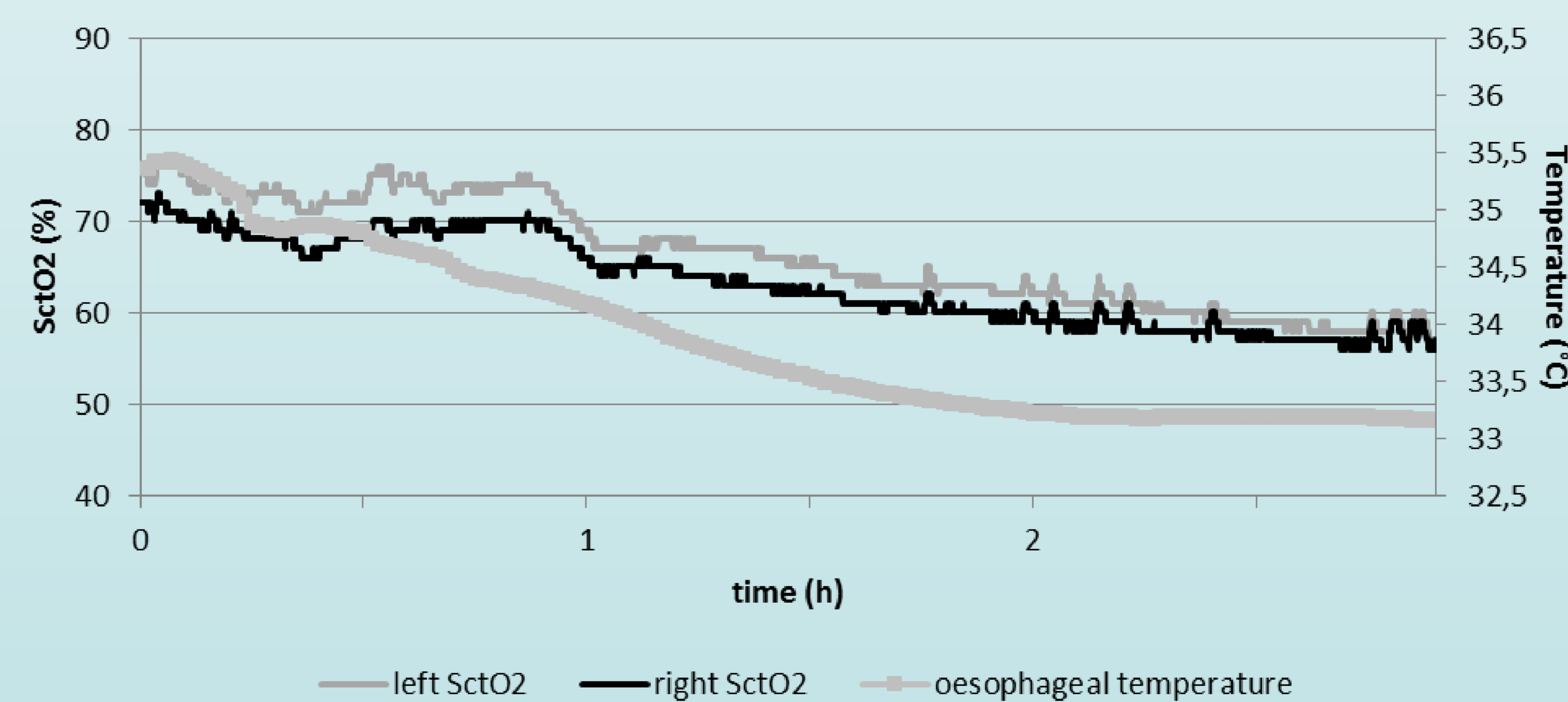
Eight patients survived with good neurological outcome. Six patients died in the hospital. In two patients, TH was stopped because of poor prognosis and patients died a few hours later.



## Results



- Initial SctO<sub>2</sub> values: 65% (left and right)
  - Start TH: **decrease** in SctO<sub>2</sub> below 55% due to shivering (↓)
  - Increase** in SctO<sub>2</sub> before reaching target temperature of 33°C
- Patient was discharged without neurological deficit



- Initial SctO<sub>2</sub> values: 70% (left) and 75% (right)
  - Start TH: gradual **decrease** in SctO<sub>2</sub>
  - No increase in SctO<sub>2</sub> during induction TH
- Patient died 2 weeks after CA due to post anoxic brain damage

## Conclusion

Non-invasive monitoring of cerebral oxygenation could indicate any imbalance between oxygen supply and oxygen demand of the brain during induced hypothermia.

The ability to monitor cerebral processes will allow greater insight into the protection mechanisms of therapeutic hypothermia and can lead to better titration of treatment.