Case Report

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Monitoring of cerebral oxygen saturation with a jugular bulb catheter after near-drowning and respiratory failure

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Monitoring der zerebralen Sauerstoffsättigung nach Beinahe-Ertrinken und akuter respiratorischer Insuffizienz

Zusammenfassung. Wir berichten über die kontinuierliche Überwachung der zerebralen Sauerstoffsättigung bei einem 18 Monate alten Mädchens nach Beinahe-Ertrinken und akuter respiratorischer Insuffizienz. Die Messungen, die mit Hilfe eines retrograden fiberoptischen Jugulariskatheter durchgeführt wurden, zeigten im Gegensatz zu den zentral-venösen und arteriellen Werten eine akut bedrohliche zerebrale Sauerstoffuntersättigung an. Nachdem konventionelle Therapiemaßnahmen zur Verbesserung der zerebralen Sauerstoffsättigung fehlschlugen konnte diese erst durch den Einsatz der extrakorporalen veno-venösen Membranoxygenierung (vv ECMO) erreicht werden. Das Mädchen wurde nach 7 Tagen extubiert und ohne neurologische Defekte nach 25 Tagen nach Hause entlassen. In Fällen von Beinahe-Ertrinken ist die kontinuierliche Messung der zerebralen Sauerstoffsättigung mittels eines retrograden fiberoptischen Jugulariskatheters ein wichtiges diagnostisches Instrument, um eine kritische Sauerstoffversorgung des Gehirns frühzeitig zu erfassen.

Schlüsselwörter: Zerebrale Sauerstoffsättigung, Jugulariskatheter, Beinahe-Ertrinken, akute respiratorische Insuffizienz, zerebrale Sauerstoffuntersättigung, extrakorporale veno-venöse Membranoxygenierung.

Summary. We report on monitoring oxygen saturation with a jugular bulb fiber-optical catheter in an 18-month-old girl after fresh water near-drowning followed by acute respiratory failure. The first measured cerebral oxygen saturation was 22% despite normal values for arterial and central venous oxygen saturation. After conventional therapy had failed to improve cerebral oxygen saturation, we started veno-venous extracorporeal membrane oxygenation. Normal levels of cerebral oxygen saturation were achieved after six hours. The girl was extubated after seven days and discharged after twenty-five days in good general condition and without obvious evidence of neurological damage. We believe that in this case of near-drowning, monitoring cerebral oxygen saturation with a jugular bulb catheter was important for surveillance of cerebral hypoxia.

Key words: Cerebral oxygen saturation, jugular bulb catheter, near-drowning, acute respiratory failure, cerebral hypoxia, extracorporeal membrane oxygenation.

Introduction

The term near-drowning refers to a submersion victim who is resuscitated and then survives for at least 24 hours [1]. Unfortunately, many survivors have permanent neurological damage as a result of the initial cerebral hypoxic trauma. A patient's morbidity after near-drowning appears to depend on the degree of cerebral hypoxia at the time of submersion [2].

Since the early eighties the use of a jugular bulb catheter for monitoring cerebral oxygen saturation has been receiving increasing attention [3–8]. This method is used for adult and paediatric patients with brain injury. On-line monitoring of cerebral oxygen saturation in the jugular bulb (SjO_2) in patients after near-drowning has not been previously reported. We report on a child after near-drowning and acute respiratory failure that required extracorporeal membrane oxygenation (ECMO) in an attempt to improve cerebral oxygenation.

Case report

An 18-month-old girl experienced fresh water near-drowning with a submersion time of 15 minutes. After successful cardiopulmonary resuscitation at the site of drowning, she was brought to a local hospital for further management. On admission, body temperature was 27 °C, and initial laboratory data from venous blood were: pH 7.06, BE –21.8 mmol/L, serum sodium 127.9 mmol/L, serum potassium 2.0 mmol/L, hemoglobin 11.4 g/dL and serum glucose 400 mg/dL.

Because of progressive respiratory failure, she was transferred to our Paediatric Intensive Care Unit (PICU). On admission to our PICU, physical examination revealed: cold, pale and mottled skin; both pupils middle-wide in size and showing normal reaction to light stimulation. The lungs were well ventilated but breath sounds were weak; heart sounds were regular, no murmurs, and heart rate was 110/min; peripheral pulses were weak but palpable; blood pressure was 90/55 (mean 70) mmHg, central venous pressure was 7 cmH_2O. The abdomen was gas-distended with normal bowel sounds. The liver was about 1.5 cm below the costal margin, the spleen was not palpable. Body temperature was 32 °C. The first chest x-ray

showed homogenous alveolar consolidation in both lungs. Only the most apical areas of the left lung demonstrated regular ventilation, heart size was normal.

Positive-pressure ventilation (IPPV) was continued with an EVITA II (Draeger, Luebeck, Germany) ventilator. The patient required an FiO_2 of 1.0, positive end-expiratory pressure (PEEP) was set at 2 cmH_2O, peak inspiratory pressure (PIP) was 28 cmH_2O, tidal volume (V_T) was set 12 mL/kg, I:E ratio was set to 1: 2 and ventilator rate was 39 breaths per minute. Arterial gas analysis showed a pH of 7.23, $paCO_2$ was 45 mmHg, paO_2 was 171 mmHg and base excess (BE) –8.5 mmol/L. Serum lactate was 10.4 mmol/L and serum glucose was 349 mg/dL.

About two hours after admission, a jugular bulb catheter (4 Fr, Opticath Catheter, Abbott, USA) was inserted for monitoring cerebral oxygen saturation. The tip of the catheter was positioned in the jugular bulb, and this was verified by skull and neck radiography. The catheter was calibrated in vitro prior to insertion, in vivo immediately after insertion and every 12 hours thereafter. The first value of SjO_2 was 22%. At the same time arterial oxygen saturation (SaO_2) was 99% and central venous saturation (SvO_2) was 55% (Fig. 2).

To improve cerebral oxygen saturation, ventilatory settings were changed: PEEP was increased to 5 cmH_2O and the I:E ratio was set to 1: 1. PEEP was then further increased to 8 cmH_2O and tidal volume (V_T) was set 15 mL/kg. The patient received exogenous surfactant (50 mg/kg BW; Alveofact® Boehringer Ingelheim, Biberach, Germany) and inhalative nitric oxide (15 ppm). Further volume was substituted intravenously (Biseko® albumin solution, Biotest, Dreieich Germany; 10 mL/kg BW for 1 hour), and inotropic support with dobutamine (5 µ/kg/min) and epinephrine (0.2 µ/kg/min) was given to improve cardiac output; however, cerebral oxygen saturation remained low. Concerned about the risk of unchanged low SjO_2 values and possible aggravation of cerebral hypoxia, we started with preparations for a veno-venous ECMO. We used a modified type of percutaneous single needle veno-venous ECMO system, AREC (assistance respiratoire extra corporelle). This ECMO system, developed by Chevalier and Durandy [9], is driven by a non-occlusive roller pump with alternating clamps. The tubing system (Health Care Materials, Paris) was modified in our unit. We used a double raceway, a M16 Jostra membrane oxygenator (Jostra Medizin Technik GmbH Hirrlingen, Germany) and a Jostra HEC 40 heat exchanger. The initial setting was 13 rotations per minute (= tidal flow of 800 mL/min). The correct position of the cannula, inserted via the internal jugular vein and positioned in the right atrium, was verified by chest x-ray (Fig. 1). Six hours after starting with ECMO, SjO_2 increased above 70% and serum lactate decreased to 4.4 mmol/L. After 48 hours on ECMO, the chest x-ray improved significantly. The patient was weaned from ECMO after 65 hours and extubated seven days after admission. At this time chest x-ray and laboratory values were normal. The transcranial ultrasound and doppler examinations showed no signs of severe brain oedema and a good systolic and diastolic flow curve. The pulsatility index was 1.08 before starting ECMO, and on ECMO 0.85, and 0.87, measured on two consecutive days. At discharge from hospital after 25 days, the patient was in a good general condition and without obvious neurological deficit.

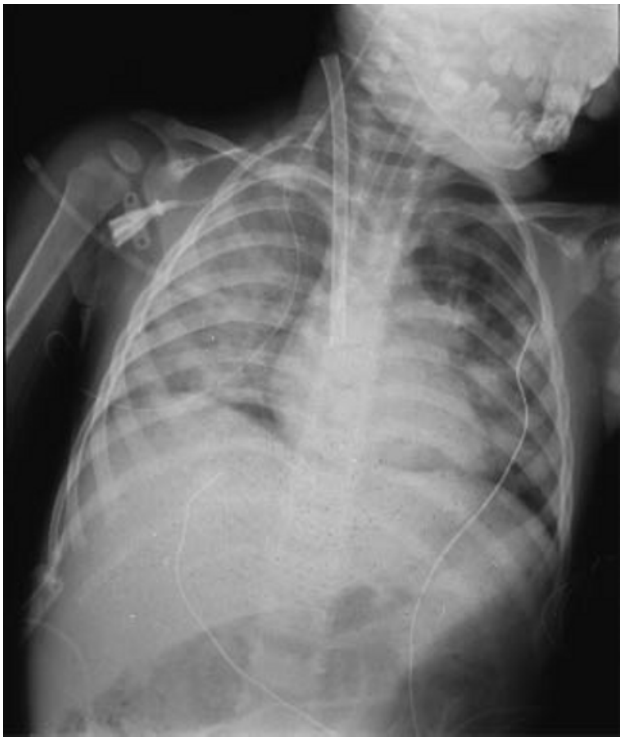

Fig. 1. Chest x-ray (a.p) one hour after starting single needle v-v ECMO. Note the homogenous alveolar consolidation in both lungs. The veno-venous ECMO cannula is positioned in the right artrium. A jugular bulb catheter with a fiber-optical device is in the right internal jugular vein. The central venous catheter is placed in the right subclavian vein

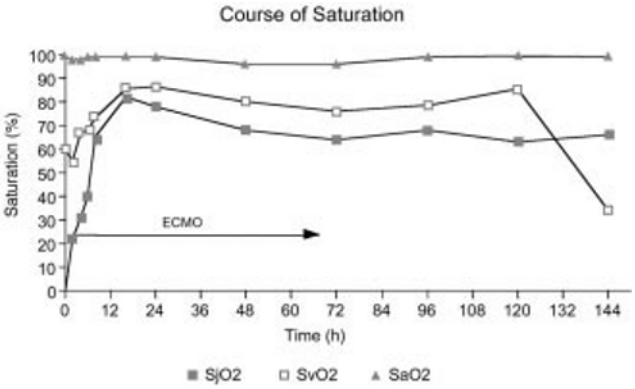

Fig. 2. Oxygen saturation during the clinical course: Solid triangles represent SaO_2, open squares SvO_2 and solid squares SjO_2. SjO_2 was first measured two hours after admission using a jugular bulb catheter. Note the difference between SjO_2 and SvO_2 or SaO_2. Six hours after initiating ECMO, SjO_2 levels became normal. ECMO, marked with an arrow, lasted 65 hours

Discussion

In the present case of near-drowning and acute respiratory failure we observed a critical decrease in cerebral oxygen saturation, with levels below 30% in the first hours after drowning. The normal value for SjO_2 in the adult population is ± 65% [10]. The normal arterial-jugular venous oxygen difference ($AVDO_2$) in children has been reported to be lower than in adults [11]. Therefore, we can assume that the normal range for SjO_2 in children

is higher than in adults. This emphasizes the critical low level of cerebral oxygen saturation in our patient. SjO_2 levels below 55% are usually regarded as critical for cerebral oxygen supply and may be due to a reduction in blood flow, haemoglobin level or arterial oxygenation [7]. But in our patient there was no evidence of decrease in cerebral blood flow or a low haemoglobin level. Our patient obviously had a low cardiac output as a result of hypoxic injury caused by drowning. This low cardiac output may explain the elevated cerebral extraction of oxygen in the brain tissue and therefore the discrepancy between low cerebral oxygen saturation and normal values of arterial and venous oxygen saturation. Cerebral oxygen saturation remained low even after changing ventilator settings and starting with inotropic support to improve the hemodynamic situation. Therefore, after conventional therapy had failed to improve cerebral oxygen saturation, we decided to start veno-venous ECMO to increase cardiac output and thereby improve cerebral oxygen saturation. On ECMO we achieved normal levels of SjO_2 within six hours.

The human brain is highly dependent on adequate aerobic metabolism. In the case of brain injury, it is necessary to optimise oxygenation to reduce further tissue damage. This can be achieved by maintaining adequate arterial oxygenation and blood flow relative to tissue oxygen consumption [6]. However, in the normal clinical setting, adequate cerebral oxygenation is difficult to evaluate. Cerebral hypoxia is one of the main problems in the treatment of children after near-drowning. The extent of hypoxic injury depends on submersion time, the level of hypothermia and the effectiveness of primary resuscitation before the patient reaches an intensive care unit. Monitoring cerebral oxygen saturation with a jugular bulb fiber-optical catheter makes it possible to follow the response to brain-specific therapy in patients with cerebral hypoxia, such as near-drowning patients.

We performed catheterisation of the right internal jugular vein, as described by Goetting and Preston [4]. We did not use the jugular bulb catheter for administration of routine fluid and medication, although some authors describe this usage. The rate of complications of jugular bulb catheters is relatively low, inadvertent carotid puncture occurred in 3% of cases [4, 12].

The use of a single needle cannula for veno-venous ECMO via the right internal jugular vein does not influence the measurements of SjO_2 from the jugular bulb catheter, even though they are inserted in the same vessel. The orifice of the ECMO cannula, where oxygenated blood is coming out, lies in the right atrium and therefore a retrograde flow of oxygenated blood to the jugular bulb region is impossible.

Morton et al. [13] reviewed neurologic morbidity (i.e., intracranial infarction or hemorrhage, brain death) in patients undergoing ECMO, but such patients seem to have a different etiology from those treated with conventional therapy. In patients treated conventionally, neurologic insult is caused by either the initial event (i.e., near-drowning or strangulation) or hypoxic events related to pulmonary insufficiency. Moreover, it remains unclear which patient is at greatest risk of neurologic insult. Selection criteria are needed in order to determine when the risk of death or hypoxic-ischemic insults outweighs the risk of ECMO-related neurologic complications.

During the recovery period after a near-drowning almost every child shows some abnormalities of behaviour or intellect. This often appears more disturbing to the child or to the family than any evident physical handicap [14, 15]. Deficits in verbal and visual recognition memory are particularly seen in younger children and are proportional to the duration of impaired consciousness [16] and the extent of the resulting hypoxic brain damage. Our patient fully recovered and left hospital without any obvious neurological deficit. Nevertheless, we agree with van der Hoeven [17] that still further studies, with large case numbers and extended periods of monitoring oxygen saturation with a jugular bulb catheter, are necessary for differentiation between cerebral-injured patients with good or poor cerebral outcome.

We conclude that the use of a jugular bulb catheter for monitoring oxygen saturation is an effective, feasible and safe procedure for use in patients after near-drowning and cerebral hypoxia. However, still more outcome studies are needed in order to prove the benefits of this cerebral monitoring procedure.

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