muscle tone has returned, and even at this stage muscle weakness may recur. In future, we would avoid the use of magnesium sulphate for at least 30 min after reversal of residual neuromuscular block, to minimize the risk of this recurring.

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Cerebral oxygenation measured by near-infrared spectroscopy during circulatory arrest and cardiopulmonary resuscitation

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We measured cerebral oxygenation by near-infrared spectroscopy (NIRS) during an unexpected cardiac arrest and successful cardiopulmonary resuscitation (CPR). A 4-yr-old girl with cyanotic congenital heart disease developed an arrhythmia during cardiac catheterization with subsequent circulatory arrest. Continuous monitoring of cerebral oxygenation showed marked changes in oxygen status immediately after the beginning of the tachyarrhythmia. After 1 min of circulatory arrest, a decrease in oxygenated haemoglobin concentration and cytochrome oxidase signal indicated a critical reduction of oxygen tension. With the beginning of CPR, a rapid increase in cytochrome oxidase oxygenation was observed. Previous values, however, were only restored when sinus rhythm was obtained after successful cardiac defibrillation. Our observations suggest that non-invasive cerebral NIRS measurement gives useful additional realtime information on cerebral oxygenation during cardiac arrest and CPR.

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Near-infrared spectroscopy (NIRS) is a non-invasive method to measure oxygenation in a localized tissue field and measures the transmission of infrared light through biological tissue. This indicates changes in oxygenation and the concentration of tissue chromophores such as total haemoglobin concentration (tHb) with its constituent oxygenated haemoglobin (HbO₂) and deoxygenated haemoglobin (HHb) and cytochrome oxidase (CytOx), which is the terminal enzyme in the mitochondrial respiratory chain. Changes in chromophore concentrations are quantified by using the modified Lambert–Beer law. NIRS is becoming used in paediatric intensive care medicine to obtain real-time information on tissue oxygenation, particularly in pre-term and term neonates to investigate brain, liver and splanchnic tissue oxygenation.2–4

The NIRO 300 (Hamamatsu Phototonics, Japan), which uses spatially resolved spectroscopy (SRS) as an algorithm,5,6 measures a tissue oxygenation index (TOI) and a tissue haemoglobin index (THI) as absolute values without the need to know a path-length factor. TOI reflects the ratio of \( k \times HbO_2 / (k \times HbO_2 + k \times HHb) \) and THI reflects the denominator \( k \times HbO_2 + k \times HHb \), where \( k \) is an unknown but constant tissue parameter.5 TOI represents the tissue saturation and is measured in percent, whereas THI is an absolute figure of the total haemoglobin but, due to the factor \( k \), measured in arbitrary units. As it is an absolute value, its changes from one measuring point to another can be measured as a percentage. We describe our observations on cerebral NIRS monitoring in a patient who needed CPR during cardiac catheterization.

Case report
A 4-yr-old girl (18 kg) with an Ebstein malformation was to have diagnostic cardiac catheterization. The child suffered from increasing exertional dyspnoea and cyanosis with a transthecanal peripheral oxygen saturation of <90%. An episode of supraventricular tachycardia was suggestive of a pre-excitation syndrome. The initial aim of NIRS recording was to investigate the effect of deep conscious sedation on cerebral oxygenation. The study was approved by the Ethics Committee of the Medical Faculty, and written informed consent was given by the parents.

The NIRS probe was placed on the forehead in the supraorbital region receiving reflected light from the frontal neocortex. The patient was given midazolam and ketamine i.v. Monitoring was by ECG and pulse oximetry. Blood pressure was measured non-invasively at intervals of 5 min. During the examination a catheter was introduced into the right atrium and through the atrial septal defect into the left atrium. This triggered a supraventricular tachycardia (heart rate=230 beats min\(^{-1}\)). A sudden decrease in HbO₂, tHb, CytOx, TOI and THI was observed. Figure 1 shows how the NIRS values changed.

The NIRS output showed desaturation of the TOI from 65% to 13%, and the THI decreased from 82 to 55 arbitrary units, indicating a decrease of 33% in the total haemoglobin content in the measured area. HbO₂ decreased by 37 \( \mu \)mol litre\(^{-1}\) and the HHb signal increased by 24 \( \mu \)mol litre\(^{-1}\). CytOx signal showed a rapid decrease of 1 \( \mu \)mol litre\(^{-1}\) from baseline (Fig. 1, event 2). Mean arterial pressure decrease to <20 mm Hg and peripheral pulse oximetry did not show any valid signals because of the low pulse pressure. After two unsuccessful attempts at cardioversion, ventilation and cardiac compressions were started and with an increase in HbO₂, tHb, THI and CytOx, TOI values started to increase with a short latency (Fig. 1, event 3). During this time ventricular fibrillation started (Fig. 1, event 4). After 24 min of CPR, the ventricular fibrillation was stopped by defibrillation and followed by a supraventricular tachycardia (heart rate=190 beats min\(^{-1}\)) with a mean arterial pressure of 60 mm Hg. Values for HbO₂, tHb and TOI started to decline again with the beginning of tachycardia, while HHb values increased once again (Figure, event 5). The CytOx signal demonstrated slight fluctuations at a greater value compared with the baseline values before the resuscitation (A0.8 \( \mu \)mol litre\(^{-1}\)). Cardioversion led to sinus rhythm and blood pressure increased to 90 mm Hg. A slight increase in HbO₂ and TOI and a decline in HHb were seen (Fig. 1, event 6). CytOx signal started to decline, but NIRS monitoring was interrupted because the patient was transferred to the intensive care unit. Under therapy with propafenon no further cardiac tachyarrhythmia occurred. Corrective surgery of the congenital heart defect was carried out and the child was discharged from intensive care with no neurological deficits.

Discussion
In our patient, a decrease of the NIRS values (HbO₂, CxtOx, THI, TOI) was noted before the circulatory situation became apparently severe. The pulse oximeter failed to provide data on arterial saturation during the resuscitation as it would be expected because of the poor signal whereas the NIRS signal changes were immediately visible. Changes in CytOx redox state are only seen when the availability of oxygen at the cellular intramitochondrial level has decreased to critical values.7–8 A decrease of CytOx correlated with a decreased brain energy state and may indicate impending cellular injury.9 The immediate decrease of intravascular cerebral oxygen differs clearly from the slow changes following circulatory arrest in deep hypothermia during corrective cardiac surgery.2

The reason for an increase in CytOx to values greater than baseline after restoration of spontaneous circulation remains unclear. A possible explanation could be the compensatory reoxygenation following an ischaemic period or the influence of mechanical ventilation with oxygen 100%.

NIRS has been studied in cardiac arrest in 18 patients arriving in an emergency department with circulatory arrest or shortly after restoration of spontaneous circulation.10 The
Fig 1 Measurements of cerebral oxygenation data. Oxygenated (HbO₂), deoxygenated (HHb) and total haemoglobin (tHb), cytochrome oxidase (CytOx) and tissue oxygenation index (TOI). Display of the full measurement (numbered dotted lines show the different events): 1=baseline with sinus rhythm (SR); 2=beginning of supraventricular tachycardia (SVT) and cardioversion (CV); 3=beginning of cardiopulmonary resuscitation (CPR); 4=ventricular fibrillation (VF) and CPR; 5=defibrillation (SVT, end of CPR); 6=cardioversion (SVT was cardioverted into SR). When low cardiac output occurred (event 2) an immediate change of intravascular oxygen was shown by a decrease in tHb, HbO₂, CytOx, TOI and THI. A decrease in CytOx indicated reduced oxygen in the cell, which was reversed by CPR (event 3). All values started to normalize when spontaneous circulation was restored.
authors reported that if regional cerebral oxygen saturation ($S_{rO_2}$) is small after cardiac arrest, there is a greater mortality. Recently published data\textsuperscript{11} describe cerebral oximetry in a patient with cerebral infarction and circulatory arrest, with a difference between the NIRS signals obtained from the centre of the stroke compared with the non-stroke areas. The saturation in an ischaemic area that is metabolically inactive can be normal because oxygen extraction does not occur from cerebral venous blood, which can also be from other adjacent brain regions that are perfused. Thus, the interpretation of NIRS data may be difficult in patients with cerebral stroke without the use of other imaging methods.\textsuperscript{11} Our data support observations of another report where $S_{rO_2}$ decreased from 60% to 41% in a patient with circulatory arrest.\textsuperscript{12} Systemic perfusion was restored with cardiopulmonary bypass and the changes during hypoxaemia were reversed. The authors inferred that NIRS might be useful to determine the effectiveness of CPR. None of the reports\textsuperscript{10,12} that measured cerebral oxygenation during cardiac arrest commented on CytOx changes because they used a different transcranial NIRS monitoring system (INVOS 3100, Somanetics Inc., Troy, MI, USA). A recent study compared two different near-infrared spectrophotometers (INVOS 4100 and the NIRO 300) measuring cerebral oxygen saturation during changes of cerebral blood flow induced by carbon dioxide challenge.\textsuperscript{13} The INVOS 4100 and the NIRO 300 showed a significant linear correlation for values of $S_{rO_2}$ and TOI during carbon dioxide alteration, but Bland–Altman analysis\textsuperscript{14} showed that the individual values and the relative changes displayed by the two devices were not equivalent.

Discrepancies between persisting bowel desaturation measured with NIRS and short episodes of systemic desaturation were reported in neonates.\textsuperscript{15} We could not measure the circulation and systemic saturation continuously to detect differences between cerebral and systemic saturations.

Studies of the relationship between cerebral measurements with other devices and regional cerebral oxygenation measured by spatially resolved NIRS give different results.\textsuperscript{16,17} Measurements of jugular bulb oxygen saturation ($S_{jO_2}$) measured by co-oximetry and TOI measured by NIRO 300, do not agree well despite a statistically significant correlation.\textsuperscript{16} The NIRS cerebral monitoring measures TOI in a small region of the cerebral microvasculature, whereas $S_{jO_2}$ reflects a more global measurement, so inhomogeneous distribution of blood or metabolic activity will reduce the agreement between the two methods.\textsuperscript{18} In addition, the TOI signal reflects an average of arterial (25%), capillary (5%) and venous (70%) blood. Cerebral cortex haemoglobin saturation, measured directly by the spatially resolved method, reflects predominantly the saturation of the intracranial venous compartment of circulation.\textsuperscript{17} Unfortunately, we did not have other measurements of brain oxygenation in our patient to verify that the decrease in TOI represented a change in cerebral oxygen saturation. $S_{jO_2}$ and TOI may measure different entities, so that although there is a significant correlation between $S_{jO_2}$ and TOI, the two measures are not interchangeable.\textsuperscript{16}

Our observations show that non-invasive cerebral NIRS measurement gives useful real-time information on cerebral oxygenation during cardiac arrest and CPR and this deserves further investigation.

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