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Influence of external cardiac pacing on cerebral oxygenation measured by near infrared spectroscopy in children after cardiac surgery



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Influence of external cardiac pacing on cerebral oxygenation measured by near infrared spectroscopy in children after cardiac surgery

Running title: Cerebral oxygenation during pacing

Category: Clinical study

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ABSTRACT

Background: The brain of children in the early period after repair of congenital heart defects with cardiopulmonary bypass (CPB) may be more vulnerable to hemodynamic changes because of impaired cerebral autoregulation. During postoperative testing of the external temporary safety pacer we performed desynchronizing ventricular pacing (VVI) while monitoring cerebral oxygenation using near-infrared spectroscopy (NIRS).

Methods: We prospectively investigated 11 children (6 female, 5 male). Mean age was 6.1 months (± 3.8 months) and mean weight: 5.3 kg (± 1.5 kg). We performed measurements at four study steps: 1. baseline I, 2. VVI pacing, 3. baseline II, and 4. atrial pacing (AOO) in order to exclude effects of higher heart rate. We continuously measured the effects on hemodynamic and respiratory parameters as well as on cerebral tissue oxygenation index (TOI). Hemoglobin difference (HbD) was calculated as a parameter for cerebral blood flow.

Results: VVI pacing leads to a significant decrease in arterial blood pressure and central venous saturation accompanied by an immediate and significant decrease in TOI ($63.3\% \pm 7.6\%$ to $61.5\% \pm 8.4\%$ ($p < 0.05$)) and HbD ($0.51 \pm 1.8 \mu\text{mol/l}$ to $-2.9 \pm 4.7 \mu\text{mol/l}$ ($p < 0.05$)).

Conclusion: Cardiac desynchronization after CPB seems to reduce cerebral blood flow and cerebral oxygenation in children.

Keywords: cerebral oxygenation, near-infrared spectroscopy, children, ventricular pacing, cerebral pressure autoregulation

Abbreviations:

CBF: cerebral blood flow

CBV: cerebral blood volume

CHD: congenital heart defects

CPA: cerebral pressure autoregulation

CPB: cardiopulmonary bypass

DeoxyHb: deoxygenated hemoglobin

HbD: hemoglobin difference

NIRS: near-infrared spectroscopy

O2Hb: oxygenated hemoglobin

tHb: total hemoglobin

TOI: tissue oxygenation index

INTRODUCTION

With the advances made in pediatric cardiac surgery and critical care medicine, neurological outcome has become an important issue in children with congenital heart defects (CHD).

Hemodynamic alterations with low cardiac output may have greater impact on cerebral perfusion after cardiopulmonary bypass (CPB) as cerebral pressure autoregulation (CPA) may be impaired (1). Situations with decreased cardiac output (CO), as they can appear in postoperative arrhythmia, can be simulated by desynchronizing atrial and ventricular contraction. During ventricular pacing (VVI) cardiac output is decreased by about 10 to 20 percent due to the lack of the atrial "kick" (2). However, little is known about the impact of cardiac desynchronization on cerebral oxygenation and blood flow.

Near-infrared spectroscopy (NIRS) provides a continuous, non-invasive method to measure regional cerebral oxygenation. Previous studies have shown that the difference between the measured oxygenated (O₂Hb) and deoxygenated hemoglobin (deoxyHb) reflects cerebral blood flow (CBF), whereas total hemoglobin (tHb) seems to be sensitive to changes in cerebral blood volume (CBV) (3-5).

In order to study the effects of hemodynamic changes on cerebral oxygenation we observed the NIRS changes during testing of external pacemakers in the early phase after cardiac surgery.

The purpose of our study was to evaluate the impact of cardiac desynchronization on cerebral oxygen and blood supply.

METHODS

Patients. We prospectively studied 11 children (6 female, 5 male) after elective surgical repair of congenital heart defects (CHD). The characteristics of the patients and the duration of CPB are shown in table 1. The underlying diagnoses were: ventricular septal defect (VSD) (n=8), tetralogy of Fallot (TOF) (n=2) and complete atrio-ventricular septal defect (cAVSD) (n= 1). None of the patients had a significant right to left or left to right shunt that could have influenced the cerebral oxygenation. None of the patients had deep hypothermic cardiac arrest or regional head perfusion during the operation on CPB. CPB was performed in all children using the pH-stat method.

The study was approved by the local ethics committee and written informed consent was given by the parents.

Near-infrared spectroscopy (NIRS). Near-infrared spectroscopy (NIRS) provides a continuous, non-invasive method to measure regional cerebral oxygenated (O₂Hb) and deoxygenated hemoglobin (deoxyHb) concentrations based on the different light absorption spectra of the two. As the sum of both, changes in total hemoglobin (tHb) content can be calculated.

We used the NIRO 300 (Hamamatsu Phototonics, Japan) as near-infrared spectroscope. The NIRO 300 uses laser-emitting diodes to generate light at four different wavelengths (775, 810, 847 and 919 nm). It employs the technique of spatially resolved spectroscopy (SRS) and uses multiple closely spaced detectors to measure light attenuation as a function of source - detector separation. This SRS method, which is described in detail by Matcher et al. (6) and Suzuki et al. (7), allows the measurement of an absolute value for tissue oxygen saturation (TOI) (TOI =

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3 O₂Hb/ tHb x 100). TOI seems to show a good correlation with central venous
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5 saturation (CVS) in stable conditions (8).
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9 The emitter and receiver were fixed in a probe holder to ensure an interoptode
10 distance of 40 mm and shielding prevented interference by external light. The probe
11 was placed on the forehead in the supra-orbital region receiving reflected light from
12 the frontal neocortex. This position is chosen to avoid signal disturbances and
13 extracerebral contamination from the temporal muscle and the superior sagittal sinus.
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15 The NIRS probe is combined with the measuring unit, which is connected to the
16 NIRO 300 display unit. NIRS values were recorded continuously and saved to disc
17 with a sample rate of 1 second.
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30 **Cardiac pacing.** During surgical repair of congenital heart defects external pacer
31 electrodes are placed on the epimyocardium of the ventricles as well as of the atria.
32 As a result cardiac pacing can be performed with an external pacemaker in the case
33 of severe arrhythmia in the early post-operative stage. To ensure its functionality, the
34 external pacemaker is tested in the intensive care unit (ICU). During this testing atrial
35 pacing (AOO) and ventricular demand pacing (VVI) with a frequency higher than the
36 actual heart rate is performed. AOO pacing stimulates the atria and is then conducted
37 to the ventricles via the natural atrioventricular conducting system, thus leading to
38 synchronized contraction of the atria and the ventricles. VVI pacing is performed to
39 test whether effective ventricular pacing is possible in the case of atrioventricular
40 block. VVI pacing thus leads to faster contraction of the ventricles without effective
41 and synchronized atrial filling. The lack of the atrial "kick", as seen in atrial fibrillation,
42 decreases cardiac output by about 10 to 20 percent (2).
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3 **Study design.** All children were intubated and ventilated mechanically. Sedation was
4 maintained with continuous intravenous fentanyl (4-20 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{h}^{-1}$) and midazolam
5 (4-20 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$). Continuous monitoring of heart rate, systemic arterial blood
6 pressure of the radial or femoral artery, transcutaneous oxygen saturation (SaO₂)
7 and central venous pressure was performed routinely after cardiac surgery. The
8 study protocol was started 1 to 48 hours after admission to the ICU with a mean time
9 range after the end of CPB of 16.4 hours (± 10.5 hours).

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20 When children were in stable cardio-respiratory conditions, baseline values for NIRS
21 parameters were recorded for 5 minutes (baseline I). After stable baseline values
22 were reached, we performed 5 minutes of VVI pacing with a frequency 10 percent
23 higher than the baseline sinus rhythm (VVI). A second baseline (baseline II) followed
24 for 5 minutes. To evaluate whether changes in hemodynamics or cerebral
25 oxygenation are due to a higher heart rate, another step with AOO pacing with the
26 same frequency as the second step was performed. No changes in medication or
27 ventilation were made during the study period.

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39 At the end of each step a blood sample was taken from a central venous catheter
40 which was placed in the right atrium. Blood gas analysis with measurement of central
41 venous saturation (CVS) was performed using co-oxymetry (OSM3-Hemoxymeter,
42 Radiometer Copenhagen, Denmark). All hemodynamic parameters together with
43 blood gas analysis and cerebral TOI were documented simultaneously.

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53 **Statistics.** Mean values of the above parameters over the 5 minutes of each step of
54 the study were calculated. All data were analyzed using SPSS 10.0 for Windows. As
55 data were not normally distributed, the Wilcoxon signed rank test was used to
56 compare the deviations of the mean values of each step from its baseline during
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3 cardiac pacing. The data are presented as mean \pm SD. P values <0.05 were
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5 considered to be significant.
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8 9 **RESULTS**

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12 ***Effects of cardiac pacing on cardio-respiratory parameters.*** During VVI pacing a
13 significant decrease in systolic, diastolic and mean arterial pressure (MAP: $p= 0.00$)
14 from baseline was observed (table 2); however there was no significant change in
15 central venous blood pressure (CVP: $P= 0.47$) or transcutaneous arterial oxygen
16 saturation (SaO₂: $P= 0.75$). Central venous saturation decreased significantly after
17 VVI pacing (CVS: $p= 0.03$), but remained stable during AOO pacing ($p= 1.0$). There
18 were no significant changes in blood gas analysis parameters such as pH_v, pO_{2v} or
19 pCO_{2v} taken from the central venous line during the whole study. The changes in
20 arterial blood pressure and all the other hemodynamic parameters from baseline II
21 caused by AOO pacing were not significant (table 2).
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39 ***Effects of cardiac pacing on NIRS parameters.*** A characteristic example of the
40 original NIRS data collected during the study is shown in figure 1.
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43 O₂Hb and tHb showed a relative decline, whereas deoxyHb increased during VVI
44 pacing. As a result a significant decrease in HbD ($p = 0.02$) was calculated (table 2).
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47 When VVI pacing was ended the values tended to return to baseline values and no
48 further changes were observed during AOO pacing.
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52 Cerebral TOI was significantly diminished during VVI pacing ($p= 0.03$) (table 2). AOO
53 pacing, however, did not cause any significant changes in cerebral TOI ($p= 0.76$).
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DISCUSSION

During our study, we observed a statistically significant fall in arterial blood pressure during ventricular pacing because of cardiac desynchronization, which however was not considered to be clinically relevant. NIRS monitoring showed a simultaneous decrease in cerebral TOI even if SaO₂ and CVP did not change significantly from baseline. We also observed a significant decrease in central venous saturation measured after 5 minutes of VVI pacing.

To demonstrate that the observed changes are not caused by a higher heart rate during pacing, another step of atrial pacing was carried out, without any significant changes in hemodynamic or respiratory parameters or in cerebral oxygenation.

Oxygen supply and consumption in sedated and mechanically ventilated children should be stable, which is also reflected by the stable blood gas parameters and unchanged transcutaneous SaO₂ in our children. Therefore we concluded that the decreased central venous saturation is mainly caused by reduced cardiac output due to cardiac desynchronization. Reduced cerebral blood flow is indicated by a significant increase in deoxygenated hemoglobin and a decrease in oxygenated hemoglobin concentration (figure 1). As a result the hemoglobin difference HbD decreased significantly. In a neonatal piglet study it could be demonstrated that a decrease in HbD reflects a decrease in cerebral blood flow (3-5).

We postulate, in our children, a constant rate of cerebral oxygen consumption and supply, and therefore a reduced cerebral blood flow might result in a relatively higher extraction of oxygen from the blood during its passage through the brain. As a result, TOI as an integral measurement of arterio- capillary- venous oxygen saturation in the frontal neocortex decreased significantly.

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3 CBF, however, should be maintained constant by cerebral pressure autoregulation
4 (CPA) over a range of cerebral perfusion pressures despite an alteration in systemic
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6 blood pressure (9).
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10 Within the range of the autoregulatory plateau, rises in blood pressure induce
11 cerebral vasoconstriction, whereas a decrease in blood pressure will cause dilatation
12 of the cerebral arteriolar vessels leading to diminished cerebrovascular resistance to
13 keep the CBF constant (9). A recently published study evaluated the integrity of CPA
14 in children after open-heart surgery by measuring NIRS-derived HbD and middle
15 cerebral artery flow velocity by transcranial Doppler. In this study the two methods
16 showed a high correlation (1). Bassan et al. suggested that concordance between
17 the waveforms of mean arterial pressure and hemoglobin difference, as we observed
18 in our children, is indicative of disturbed CPA. Also Morren et al. examined the
19 cerebrovascular autoregulation by NIRS in premature infants and concluded that
20 concordance between HbD and mean arterial pressure reflects impaired cerebral
21 autoregulation (10).
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38 Bassan et al., however, found that hypercarbia and fluctuating MAP might predispose
39 a patient to a disruption of CPA (1). As hypercarbia leads to cerebral vasodilatation of
40 the resistance arterioles, CPA might become less efficient because of a reduced
41 residual capacity for vasodilatation.
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48 In our study there were no significant changes in carbon dioxide partial pressure
49 which could explain the pressure passive changes of CBF. The changes in MAP we
50 observed were statistically significant but still within clinical limits which can be
51 considered hemodynamically irrelevant. Therefore the fall in MAP should not exceed
52 the autoregulatory plateau.
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59 However our study is limited by the fact that we did not measure cerebral blood flow
60 directly. We studied cerebral tissue oxygenation with NIRS, which gives information

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3 about the perfusion and oxygenation status of the smallest vessels and of the
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5 capillary bed in a small, defined part of the frontal neocortex. The cortical blood flow
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7 seems to be more vulnerable to changes in CBF so that HbD might be sensitive also
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9 for small changes in CBF. NIRS might reflect best the impact of changes in CBF on
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11 the smallest vessels, where minimal changes in diameter have the largest effect. In
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13 addition we saw a decrease in tHb which indicates a reduction not only in cerebral
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15 blood flow but also in cerebral blood volume. We therefore believe that CPA in our
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17 children after cardiac surgery was affected.
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22 We cannot differentiate whether the postulated alteration in CPA is caused by the
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24 CPB during cardiac surgery or is a result of the clinical conditions prior to cardiac
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26 surgery. All children were in clinically stable condition when admitted and the
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28 correction of CHD was elective. Therefore we believe the impact of presurgery
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30 conditions on CPA not to be significant.
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34 It should be mentioned that there was a rather large range of bypass times (34 mins.
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36 - 308 mins.) in our study. It is possible that the longer the bypass runs the greater
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38 could be the inflammatory response and hence the greater the sequelae for cerebral
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40 vasculature and cerebral autoregulation. Unfortunately splitting the group into long
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42 and short bypass times would not be statistically feasible because of the small
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44 sample size and the unequal distribution of the groups.
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48 However, to evaluate the impact of prior CPB on impaired CPA a control group which
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50 was not operated on with CPB would be necessary, but such a group would be
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52 difficult to investigate because of the rarity of children with an internal pacemaker and
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54 the difficulty of examining awake children with a changing degree of cerebral
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56 activation.
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60 A further limitation of our data must be mentioned: CVS declines significantly during
VVI pacing but does not recover completely during the second baseline. This might

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3 indicate that the periods of 5 minutes were not long enough for the patients'
4 physiology to have returned to equilibrium. The increase of TOI above CVS during
5 AOO pacing (table 2), with unchanged blood pressure, might suggest some degree
6 of cerebral autoregulation.
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12 In small children invasive measurement of cardiac output is more complicated than in
13 adults and highly invasive; therefore cardiac output was estimated only indirectly by
14 CVS, which of course implies a lot of pitfalls.
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20 Our results are limited by a rather small sample size and a quite broad age range;
21 however we did not include any premature or newborn children as the maturation and
22 vulnerability of the neonatal brain might have an impact.
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27 Based on our observations we believe that cerebral oxygenation and cerebral blood
28 flow may be sensitive to cardiac desynchronization in children shortly after repair of
29 CHD with cardiopulmonary bypass. Near-infrared spectroscopy might be useful in the
30 early detection of cerebral desaturation and impaired cerebral blood flow.
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36 [On the basis of our study](#) and observations we would recommend strict
37 synchronization of the atria and ventricles in children after CPB e.g. in the case of
38 temporary or permanent atrioventricular block, junctional ectopic tachycardia (JET) or
39 AV tachycardia. Effective resynchronization using external pacing or medical
40 treatment improves not only cardiac output but also cerebral oxygenation, even if the
41 blood pressure is within normal ranges.
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51 However, further studies with larger sample sizes are necessary to evaluate the
52 correlation of low TOI and adverse neurological outcome in the postoperative course
53 after repair of CHD.
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58
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Table 1: Patients' characteristics

	age (month)	weight (kg)	height (cm)	minimal temperature on CPB (°C)	time on CPB (min)
Mean ± SD (range)	6.1± 3.8 (2.1-15.0)	5.3± 1.5 (3.5- 7.8)	61.7± 8.1 (52.0- 73.0)	30.5± 2.5 (24.0- 32.0)	124.8± 97.9 (34.0- 308.0)

For Peer Review

Table 2: Hemodynamics, **venous blood gas analysis** and NIRS values presented as mean \pm standard deviation for all four steps.

* Deviation from baseline is significant for $P < 0.05$

	Baseline I	VVI Pacing	Baseline II	AOO Pacing
HR (bpm)	138.4 \pm 14.3	154.4 \pm 12.4 *	139.0 \pm 15.6	153.4 \pm 11.4*
MAP (mmHg)	55.1 \pm 10.6	50.1 \pm 10.5 *	57.7 \pm 10.7	54.5 \pm 8.6
CVP (mmHg)	11.7 \pm 2.4	11.8 \pm 1.9	11.9 \pm 2.2	11.8 \pm 2.4
SaO2 (%)	98.2 \pm 2.7	98.1 \pm 2.7	98.8 \pm 1.6	99.0 \pm 1.4
pH ven.	7.37 \pm 0.1	7.38 \pm 0.1	7.38 \pm 0.1	7.38 \pm 0.1
pO2 ven. (mmHg)	37.2 \pm 6.7	36.2 \pm 7.3	34.9 \pm 6.6	35.1 \pm 7.8
pCO2 ven. (mmHg)	47.9 \pm 7.4	47.7 \pm 7.3	47.3 \pm 6.5	46.5 \pm 7.5
CVS (%)	65.6 \pm 12.2	61.6 \pm 9.8*	61.8 \pm 11.1	61.7 \pm 12.4
TOI (%)	63.3 \pm 7.6	61.5 \pm 8.4*	63.6 \pm 7.4	63.6 \pm 7.6
HbD (μ mol/l)	0.51 \pm 1.8	-2.90 \pm 4.7*	0.17 \pm 2.1	0.14 \pm 2.6

Figure 1 Original NIRS data of a 3.9 month old male baby after VSD patch closure:

The measurement shows the changes in total hemoglobin (cHb), deoxygenated hemoglobin (HHb), oxygenated hemoglobin (O2Hb) and tissue oxygenation index (TOI) during the different steps of cardiac pacing: baseline values (BASE I + II) ventricular pacing (VVI), atrial pacing (AOO).

