Cerebral oxygenation normalization is delayed following balloon atrial septostomy in neonates with transposition of the great arteries compared to preductal peripheral saturation.

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Introduction

Fetal hypoxia has been implicated in the abnormal brain development seen in newborns with cyanotic congenital heart disease. After birth, neonates with transposition of the great arteries (TGA) are at risk of hypoxic-ischemic brain-injury. Balloon atrial septostomy (BAS) is an important intervention in neonates with TGA that increases systemic oxygenation. Following BAS in patients with TGA cerebral oxygenation increases rapidly but very little is known about the direct effect of BAS on the neonatal brain and on cerebral oxygenation. It is yet unclear how BAS affects cerebral oxygenation and does rapid increase of oxygen delivery results in brain reperfusion injury in neonates with TGA.

Materials and Methods

We performed a prospective single center study to evaluate changes in the cerebral oxygenation before and for a period of 48 hours following BAS in 12 neonates with TGA. Data collected included peripheral oxygen saturation, mixed venous saturation, regional cerebral tissue oxygen saturation (rcSO2) measured by using near infrared spectroscopy (NIRS), heart rate, blood pressure and hemoglobin prior and following BAS. Differences in the course of rcSO2, preductal peripheral oxygen saturation, cerebral tissue oxygen extraction (FTOE) and cerebral oxygen extraction (CEO2) were analyzed prior and following BAS. Cerebral oxygen extraction was estimated from the difference of SaO2 and ScO2 as ScO2 is close to venous SO2. FTOE was calculated FTOE=CEO2/SaO2. We also analyzed metabolic status of the patients including acidosis, lactate, and ischemia markers e.g. S100 before and following BAS.

Subject Demographics	n (%)
Patients	12 (100 %)
Male	10 (83 %)
Balloon septostomia	9 (75 %)
Expired	1 (8 %)
Prenatal diagnosis	6 (50 %)
	Median (range)
Gestational age (weeks)	39.5 (37.0 – 41.3)
Birth weight (kg)	3.4 (2.0 – 4.0)
Postnatal age (hours)	
At the time of the first measured peripheral saturation	1.6 (0.5 – 13.2)
At the time of septostomy	3.2 (2.0 – 9.0)
First peripheral saturation	78.7 (56.4 – 91.0)
First $rcSO_2$	58.4 (37.0 – 78.8)
Lactate	
Before septostomy	3.2 (1.3 – 4.9)
5 minutes after septostomy	2.7 (1.4 – 5.0)

Table 1.
Subjects demographics.

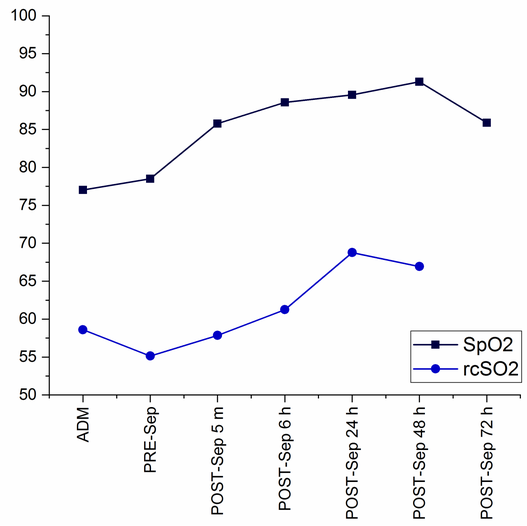

Figure 1.
SpO2 and rcSO2 in TGA patients.
ADM, admission.

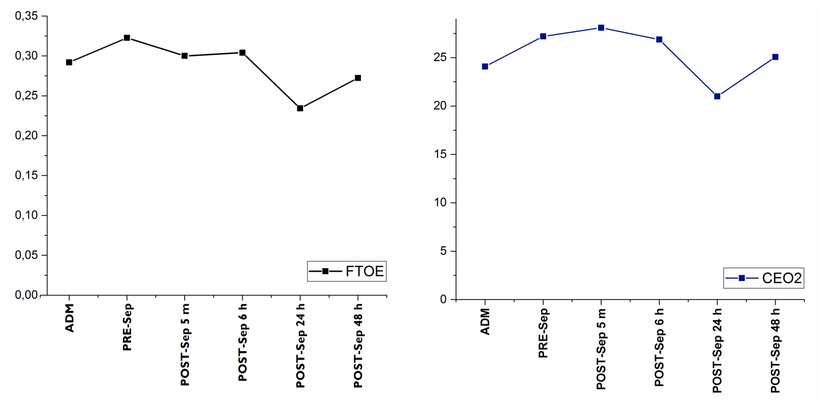

Figure 2.
FTOE and CEO2 in TGA patients.

Results

9 (75%) patients underwent BAS at the age 4.6 (±2.7) hours. Six (50 %) patients had prenatal diagnosis. There were 1 (1/12) early death prior to the septostomy. Lowest preductal peripheral oxygen saturation at admission was median of 64.5 % (range 39.0 - 92.0). Preductal SpO2 increased from a median of 85.6 % (range 62.0 – 90.6) before BAS to 89.1 % (range 81.8 – 93.5) 6 hours following BAS and 90.0 % (range 85.2 93.6) 24 h following BAS. rcSO2 at the same time points were 50.0 % (range 35.0 – 70.0), 52.8 % (range 36.4 – 72.5), 63.0 % (range 48.2 – 74.1) and 69.2 (range 58.8 – 80.8), respectively. rcSO2 correlated strongly with simultaneously measured spO2 (Spearman's r=0.89, P < 0.001). CEO2 increased after BAS (27.2 – 28.1) but both CEO2 and FTOE decreased 24 hours following BAS (Figure 2). There was no difference between the patients with prenatal diagnosis versus the patients without prenatal diagnosis.

Conclusions

Improvement of cerebral tissue oxygenation in neonates with TGA is delayed compared to peripheral oxygen saturation increase. Stabilization of main ScO2 was seen 24 hours following BAS. rcSO2 increased and FTOE and CEO2 decreased 24 h following BAS compared to SpO2 which increased immediately following BAS. This suggests delay in improvement of cerebral oxygenation following BAS. This may be due to brain reperfusion injury following BAS.

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