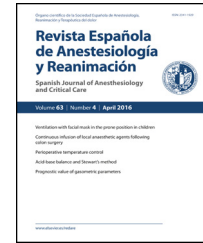




Revista Española de Anestesiología y Reanimación

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CASE REPORT

Cerebral oximetry monitoring in the management of severe hypoxaemia associated with transposition of the great arteries with balloon atrial septostomy[☆]

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Received 15 May 2017; accepted 12 December 2017

KEYWORDS

Cerebral perfusion;
Cerebral oxygen saturation;
Near infrared spectroscopy;
Balloon atrial septostomy;
Transposition of the great arteries

Abstract Transposition of the great arteries (D-TGA) is one of the most common congenital heart diseases requiring neonatal surgical intervention. In the desperately ill neonate with TGA and the resultant hypoxaemia, acidemia, and congestive heart failure, improvement is often obtained with balloon atrial septostomy (BAS). Current methods employed to evaluate oxygen delivery and tissue consumption are frequently nonspecific. Near infrared spectroscopy (NIRS) allows a continuous non-invasive measurement of tissue oxygenation which reflects perfusion status in real time. Because little is known about the direct effect of BAS on the neonatal brain and on cerebral oxygenation, we measured the effectiveness of BAS in two patients with D-TGA using NIRS before and after BAS. We concluded BAS improves cerebral oxygen saturation in neonates with D-TGA.

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[☆] Please cite this article as: Pérez Moreno JC, Nájera Losada DC, Sanabria Carretero P, Paredes Lacave Á, Benito Bartolomé F. Monitorización de la oximetría cerebral en el manejo de la hipoxemia severa asociada a la transposición de grandes arterias mediante atrioseptostomía con balón. Rev Esp Anestesiol Reanim. 2018. <https://doi.org/10.1016/j.redar.2017.12.008>

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PALABRAS CLAVE

Perfusión cerebral;
Saturación cerebral
de oxígeno;
Espectroscopia
cercana al infrarrojo;
Atrioseptostomía con
balón;
Transposición de
grandes arterias

Monitorización de la oximetría cerebral en el manejo de la hipoxemia severa asociada a la transposición de grandes arterias mediante atrioseptostomía con balón

Resumen La transposición de las grandes arterias (D-TGA) es una de las cardiopatías congénitas más comunes que requieren una intervención quirúrgica en la etapa neonatal. En neonatos muy afectados por una D-TGA, la hipoxemia, la acidemia y la insuficiencia cardiaca congestiva secundaria se mejora a menudo con una atrioseptostomía con balón (ASB). Los métodos actuales empleados para evaluar el aporte y el consumo de oxígeno tisular, con frecuencia no son específicos. La espectroscopia cercana al infrarrojo o near infrared spectroscopy (NIRS) permite una medición continua no invasiva de la oxigenación tisular, reflejando el estado de la perfusión tisular en tiempo real. Debido a que se sabe poco sobre el efecto directo de la ASB en el cerebro neonatal y en la oxigenación cerebral de los mismos, nosotros medimos la eficacia de la ASB en 2 pacientes con D-TGA utilizando el NIRS antes y después de la ASB. Concluimos que la ASB mejora la saturación cerebral de oxígeno en neonatos con D-TGA.

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Introduction

The transposition of the great arteries or D-TGA (D refers to the dextroposition of the bulboventricular loop) is one of the most common neonatal congenital heart diseases that require surgical intervention, and accounts for 7%–8% of all congenital heart diseases. D-TGA is characterised by an atrioventricular concordance and ventriculoarterial discordance, in such a way that the aorta arises from the right ventricle and the pulmonary artery arises from the left ventricle. Because the systemic and pulmonary circulations run in parallel, to be compatible with life they must communicate in some way, either by means of an atrial or ventricular septal defect, or at the arterial level in the form of patent ductus arteriosus.¹

The gold standard surgical treatment is anatomical correction or arterial switch. Before this can be performed, however, steps must be taken to improve circulatory mixing, decrease oxygen consumption and optimise flow-volume rate with the administration of, for example, prostaglandin E1, colloids, inotropics, sedation and respiratory assistance.² If prostaglandin infusion is not accompanied by a frank increase in oxygen saturation (SpO₂) and the foramen ovale is restrictive or the neonate is unstable, the atrial septal defect must be enlarged. This should be done by interventional catheterisation through the femoral vein using a balloon catheter (described by Rashkind and Miller in 1966), a new method that has replaced the surgical opening of the interatrial septum (Blalock–Hanlon method).^{2,3} Rashkind and Miller observed that the intra-atrial pressure gradient decreased and arterial oxygen saturation increased after balloon atrioseptostomy (BAS), and concluded that BAS is an effective procedure in D-TGA associated with severe hypoxaemia, acidosis and congestive heart failure.³

We present 2 cases of neonates with D-TGA that were treated with a non-invasive method known as near infrared spectroscopy (NIRS), in which a cranial sensor was placed in the frontal position to measure regional oxygen saturation

(rSO₂), thus showing brain oxygenation and the balance between oxygen delivery and consumption before and after performing BAS. This allowed us to evaluate the efficacy of the therapy, defined as an improvement cerebral oxygenation or the balance between cerebral oxygen delivery and consumption. Most studies performed with NIRS involve repair surgery (arterial switch), and very little is known about the effects of presurgical interventions on cerebral oxygenation and the effectiveness of BAS with non-invasive NIRS technology in neonates with D-TGA.

Case report 1

A full-term male neonate with a gestational age of 37.5 weeks, weight 3500 g, delivered by caesarean section due to prolonged labour in a mother with high-risk pregnancy due to type 1 diabetes, with no prenatal diagnosis of heart disease. At birth, the infant was hypotonic, with central cyanosis that required resuscitation with intermittent positive pressure, with poor response to oxygen therapy (pre- and postductal SpO₂ 50%). Ultrasound examination led to a diagnosis of D-TGA with intact ventricular septum, severely restrictive foramen ovale and 2 mm ductus. The infant was intubated and treatment was started with prostaglandin E1 0.1 g/kg/min, which increased ductus size to 4 mm and SpO₂ to 65%. Inotropic therapy was started with dobutamine 10 µg/kg/min, and the patient was referred to our hospital. The infant arrived at our unit at 6 h of life, unstable, with BP 43/23 mmHg, HR 150 bpm, RR 45 rpm, preductal SpO₂ 36% and postductal 45%, with FiO₂ 100% and hyperlactacidaemia of 21 mmol/l. BAS was performed, leaving an 8 mm wall defect with unrestricted flow between the left and right atrium. This normalised rSO₂ (Fig. 1), which increased from 23% to 68%, improved hypoxaemia in the first hour with SpO₂ of 83%, and reduced lactate levels to 9 mmol/h in the first the 24. In the ICU, he continued with inotropic support and nitric oxide was started (16 ppm) due to persistent pulmonary hypertension. At 11 days of life, an anatomical

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