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Neurophysiological Effects of Pediatric Balloon Dilatation Procedures

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Abstract. Balloon dilatation of valvar and vascular stenoses has become routine therapy in pediatric cardiology. Repeated balloon inflations cause many episodes of low cerebral oxygen delivery. This study is a prospective study to assess the effects of balloon dilatation on cerebral perfusion and oxygenation. The study included 11 patients scheduled for elective catheterization and balloon dilatation at a university pediatric hospital. Blood flow velocity in the middle cerebral artery (V_{mca}) and regional cerebral oxygen saturation (rSO_2) were monitored by means of transcranial Doppler sonography and near infrared spectroscopy, respectively. In group 1, consisting of 6 patients without an intracardiac shunt, inflation of the balloon resulted in a decrease in V_{mca} followed by a minor decrease in rSO_2. In group 2, consisting of 5 patients with an interatrial communication, inflation resulted in an increase in right-to-left shunt fraction, arterial desaturation, and a major decrease in rSO_2 with minor changes in V_{mca}. Balloon dilatation causes an important decrease in cerebral oxygen delivery by different mechanisms. This may lead to serious morbidity and even mortality. Neuromonitoring is a useful tool in assessing the cerebral effects of balloon dilatation and brain recovery.

Key words: Balloon dilatation — Cerebral oxygen delivery

Balloon valvulo- and angioplasty has replaced or postponed surgical interventions in children [1, 5]. It is minimally invasive compared to surgery and has a good efficacy and safety record [7]. Balloon inflation causes a temporary decrease in oxygen delivery under normothermic conditions either by a critical reduction in cardiac output (e.g., by fully obstructing the aortic outflow during aortic valve dilatation) or by enhancing right-to-left shunting, as occurs while inflating the balloon in the pulmonary valve or artery in the presence of an interatrial communication. Since the brain is extremely sensitive to hypoxia, this may lead to a rapid impairment of cerebral function, and if longer lasting it may lead to permanent damage and dysfunction. This has been demonstrated in adults after comparable repetitive short episodes of low cerebral oxygen delivery during defibrillation threshold (DFT) testing following implantation of cardioverter defibrillators [8]. To date, two patients with stroke and two cases of brain death have been reported after balloon valvuloplasty in children [5, 9, 12]. Although these numbers are small compared to the number of interventions that are carried out annually, the seriousness of these complications and the fact that neurological complications are probably underdiagnosed and underreported warrant further investigation. The aim of the current study was to assess the changes in cerebral hemodynamics and oxygen metabolism during balloon dilatation in children.

Patients and Methods

The study was approved by the Institutional Research Board. Eleven consecutive patients scheduled for elective balloon dilatation of an aortic valve (AS), pulmonary valve (PS), or pulmonary artery stenosis (PAS) were enrolled in the study after parental informed consent. The demographic data are listed in Table 1. Patients were divided into two groups according to the presence or absence of an intracardiac shunt; group 1 consisted of patients with stenosis of the pulmonary trunk after a previous arterial switch procedure for transposition of the great arteries ($n = 4$) or congenital AS ($n = 2$). The five patients in group two had congenital PS and an associated interatrial communication with the potential for a right-to-left shunt. All procedures were performed under general anesthesia. The blood flow velocity in the middle cerebral artery (V_{mca}) was monitored by means of transcranial Doppler (TCD) sonography. The right middle cerebral artery was interrogated via a transtemporal window using a MultiDop X (DWL, Sibblingen, Germany). Power and depth were adapted to the size of the patient and the quality of the signal. The cerebral regional oxygen saturation (rSO_2) was monitored by means of near-infrared spectroscopy (NIRS) using the Invos 3100A (Somanetics, Troy, MI). A patch containing a light emitter and two receivers was placed over the right side of the forehead and secured with a stretch bandage. Further monitoring consisted of electrocardiograph, noninvasive blood pressure, pulse oximetry (SpO_2), capnography, and nasopharyngeal temperature measurement.

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Table 1. Demographic data

	Group 1	Group 2
No. of patients	6	5
Age median	7 years	0.5 years
Range	2 days–14.7 years	2 days–11.7 years
Lesion	PAS: $n = 4$ AS: $n = 2$	PS: $n = 5$
Number of inflations	34	23
Inflation time (seconds)	13.4 ± 6.8	20.3 ± 10.4
Recovery time (seconds)	30.8 ± 12.5	44.0 ± 12.9

AS, aortic valve; PAS, pulmonary artery stenosis; PS, pulmonary valve.

In four patients (two from each group), invasive arterial blood pressure was also monitored. V_{mca} and rSO_2 values (a) at baseline, (b) maximal inflation of balloon, (c) immediately after deflation of balloon, (d) when hemodynamic parameters had returned to baseline, and (e) before next intervention were expressed as deviations from baseline. The recovery time (RT) was defined as the interval from the moment of balloon deflation to recovery of all cerebral parameters to baseline values.

Data were analyzed using Spearman's rank correlation, Kruskal–Wallis test, and Wilcoxon test where appropriate. A p value <0.05 was considered significant.

Results

Fifty-seven balloon inflations were performed—34 in group 1 and 23 in group 2. In group 1, inflation of the balloon caused an instantaneous decrease in V_{mca} followed by a decrease in rSO_2. After deflation of the balloon the V_{mca} showed a mild, statistically insignificant overshoot before returning to baseline. The rSO_2 had returned to baseline once the systemic hemodynamic parameters had also normalized (Fig. 1). In group 2 inflation of the balloon caused only a modest decrease in V_{mca} but a massive decrease in rSO_2. After deflation of the balloon both systemic and cerebral oxygen saturations returned to baseline levels. At time b (Fig. 1), during maximal balloon inflation, the difference in V_{mca} between the groups was statistically significant ($p < 0.001$). At this time the V_{mca} in group 1 had diminished significantly when compared with baseline measurement ($p < 0.001$). However, for group 2 there was no significant change in this parameter at any time during the balloon inflation–deflation cycle. At times b and c, during full inflation and immediately after balloon deflation, the differences in rSO_2 between the groups were statistically significant ($p < 0.001$), with a dramatic decrease in group 2. At these times the rSO_2 in group 2 also differed significantly from baseline ($p < 0.001$). Noninvasive blood pressure measurement and SpO_2 measurements were unreliable during and immediately after all balloon inflations in both groups. In the two patients without right-to-left shunt in whom systemic arterial pressure was invasively recorded, the mean arterial blood pressure decreased to 20 mmHg. In the two patients with right-to-left shunt, the decrease in invasive mean arterial pressure was less than 10 mmHg compared to baseline. In contrast, in the latter group the arterial oxygen saturation fell to values as low as 36%. There was a positive correlation between inflation time and the time for all parameters to recover to baseline (RT) ($r^2 = 0.62$, $p < 0.001$). The RT was approximately twice as long as the inflation time. There was no difference in this relationship between the two groups.

Balloon dilatation was successful in nine patients; two patients later underwent successful surgical treatment. Postprocedure recovery was uneventful in all cases.

Discussion

To our knowledge this is the first study to describe the changes in cerebral hemodynamics and oxygen metabolism during balloon dilatation procedures in children. Four cases of gross neurological sequelae associated with balloon dilatation have been reported. Two patients recovered without significant residual damage after suffering from stroke possibly caused by emboli [12]. Two patients suffered severe irreversible brain damage and died [7, 9]. In one of them, cerebral ischemia was caused by prolonged balloon inflation [9].

We used TCD monitoring of the V_{mca}, a reliable trend monitor of cerebral blood flow, and the rSO_2 by means of NIRS [6, 14]. This latter parameter reflects mainly the venous oxygen saturation, and for a smaller proportion the capillary and arterial oxygen saturation [11]. Balloon dilatation affected cerebral oxygen delivery either by a decrease in blood flow velocity or a decrease in arterial oxygen content. In group 1 the precipitous decrease in V_{mca} induced a compensatory increase in oxygen extraction causing a secondary decrease in rSO_2. In group 2 massive shunting at atrial level during balloon inflation led to a gross decrease in arterial oxygen content and subsequently in rSO_2. The mild decrease in V_{mca} in these cases was probably due to restricted flow through the interatrial defect. After deflation, the time for all parameters to recover to baseline was approximately twice the duration of balloon inflation in both groups.

There is a striking similarity between the neurophysiological effects of short episodes of circulatory arrest due to ventricular fibrillation and balloon dilatation in patients without right-to-left shunt [2–4, 10, 11, 13]. From studies of DFT testing it is known that episodes of normothermic circulatory arrest for as little as 15 seconds give rise to ischemic changes in the electroencephalogram (EEG) ranging from slowing to isoelectricity

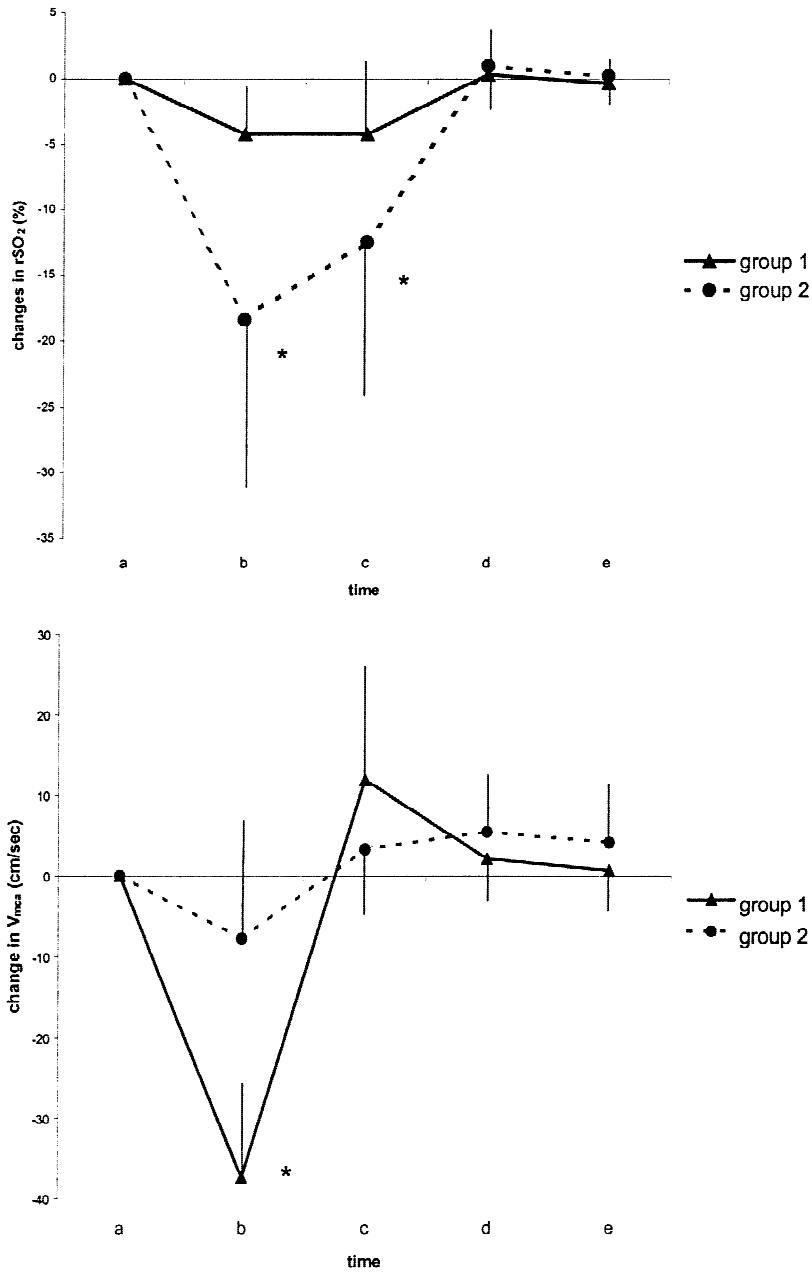

Fig. 1. Changes in rSO_2 and V_{mca} from baseline (mean ± SD) at various times during and after the balloon inflation/deflation cycle. The asterisks indicate measurements that were statistically significant ($p < 0.001$) between the two groups and compared with baseline.

[2–4, 10, 13]. Electroencephalography was not applied in this study. However, it may be assumed that if cerebral oxygen delivery is as seriously affected by balloon dilatation as observed in this study, regardless of whether these changes are induced by low flow or low oxygen content, this must have effects on the EEG comparable to the effects observed during DFT testing. Murkin et al. [6] demonstrated the importance of a sufficiently long interval between two consecutive insults during DFT testing because short interval testing was associated with significant new neuropsychological deficits. It is likely that this also applies to balloon dilatations to allow parameters of cerebral blood flow and oxygen saturation to recover to baseline, prior to the next balloon inflation. The recovery times of rSO_2 and V_{mca} were approximately twice the duration of balloon inflation. In the absence of these neurophysiological monitors, systemic parameters such as arterial blood pressure, pulse oximetry, and capnometry should be used to assess recovery.

This study has several limitations, one of which is the small number of patients. Also, monitoring of the hemodynamic parameters in the majority of the patients depended on slowly responding techniques with poor performance during inflation. However, in the four pa-

tients in whom invasive measurements of arterial pressure and saturation were performed during balloon dilatation, the changes in these parameters were consistent with the postulated mechanisms for decreased cerebral oxygen delivery in each patient group. Additional studies including larger numbers of patients and using invasive blood pressure measurement are required. EEG monitoring may provide evidence of more subtle neurologic insult during repeated or prolonged balloon inflation.

In conclusion, balloon valvuloplasty and angioplasty affect cerebral oxygen delivery. Transcranial Doppler sonography and NIRS can reliably be used for the diagnosis of low cerebral oxygen delivery and the assessment of cerebral recovery during pediatric balloon dilatation procedures.

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