

CONGENITAL HEART DISEASE

Rapid right ventricular pacing is an alternative to adenosine in catheter interventional procedures for congenital heart disease

I Daehnert, C Rotzsch, M Wiener, P Schneider

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See end of article for authors' affiliations

Correspondence to:
Dr med I Daehnert, Klinik fuer Kinderkardiologie, Herzzentrum, Universitaet Leipzig, Struempellstrasse 39, 04289 Leipzig, Germany; ingodaehnert@yahoo.de

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Objective: To describe the use of rapid right ventricular pacing to facilitate balloon stability during balloon dilatation procedures for congenital heart disease.

Setting: Tertiary paediatric cardiac centre.

Design and patients: This was a prospective pilot study of 37 consecutive patients with congenital aortic stenosis undergoing elective balloon dilatation. If the first dilatation manoeuvre failed due to balloon displacement, rapid right ventricular pacing at a rate of 220 beats/min was performed during repeat balloon inflation.

Interventions: Balloon aortic valvotomy and rapid right ventricular pacing.

Main outcome measures: Balloon stability versus displacement during balloon dilatation and procedure related complications.

Results: Initial balloon displacement occurred and rapid right ventricular pacing was performed in 14 patients. The balloon remained in stable position in 11 patients. In three patients the balloon was displaced. In two of them an increase of the pacing rate to 240 beats/min provided balloon stability. In one patient stability was obtained at an unchanged pacing rate after correction of a suboptimal balloon position. No sustained arrhythmias occurred. There were no other procedure related complications.

Conclusions: Rapid right ventricular pacing is a safe and effective method to provide balloon stability during balloon dilatation of the aortic valve. It may be applied in other fields of catheter intervention where it is desirable to maintain stable device positions during the critical phase of the procedure.

Catheter interventions have found widespread use in the treatment of congenital heart disease either as an alternative to or in combination with surgery. While surgeons use cardioplegia, fibrillators, or clamps to stop blood flow in their working field, most catheter interventions are performed on normal circulation. During catheterisation, cardiac contraction and pulsatile blood flow can cause movements of devices and inflated balloons leading to failure or suboptimal results of the procedures or causing damage to vessels and intraluminal structures. Only a few methods are known to avoid these problems. Extra stiff wires, long balloons, and long sheaths give additional support reducing the risk of displacement.¹ Temporary proximal balloon occlusion has found only limited use for some procedures such as coil occlusion of large ducts and collaterals.² Adenosine has been shown to be safe and effective in creating transient cardiac standstill and is used during balloon dilatation procedures³ and implantation of stents and grafts.⁴⁻⁵ However, there is a wide range of effective doses and significant variation in the time intervals from adenosine injection to asystole and duration of asystole.⁶

We describe a different approach to prevent unwanted device movement by rapid right ventricular pacing. Balloon dilatation of valvar aortic stenosis is an established procedure⁷⁻⁹ and was chosen as a model intervention because of its high rate of balloon displacement.

METHODS

This was a prospective pilot study of patients with congenital aortic stenosis undergoing elective aortic valve balloon dilatation. Exclusion criteria were the impossibility of performing the intervention and a stable balloon position during the first balloon dilatation. The study group was

composed of patients in whom the first dilatation manoeuvre failed due to displacement of the balloon from the valve before complete inflation.

Patients

During a two year period between September 2001 and August 2003, 37 patients underwent elective aortic valve balloon dilatation. There were 11 newborns (0-30 days), 11 infants (1-12 months), and 15 children and adolescents (1-21 years). The procedure was feasible in all. The first dilatation manoeuvre was technically correct without balloon displacement in 23 patients (all of the neonates, 10 of 11 infants, and 2 of 15 children). The remaining 14 patients (one infant and 13 children) formed the study group. The patients of the study group (nine boys, five girls) had a median weight of 48.5 kg (range 7-79 kg) and a median length of 158 cm (range 65-180 cm). They were older (median 13.4 years, range 0.3-20.2 years) than patients who did not require repeated dilatation (median 29 days, range one day to 8.3 years). Three patients of the study group had aortic restenosis following surgical commissurotomy in two and balloon dilatation in one.

Rapid right ventricular pacing

Rapid right ventricular pacing enforces ventricular tachycardia. Ventricular filling is compromised because of the high rate and absent atrioventricular synchrony. Ventricular contractility is reduced because of the dyskinetic ventricular contraction caused by apical stimulation. Thus, reduced stroke volume and cardiac output lead to decreased blood pressure and decreased blood pressure amplitude. Figures 1 and 2 show the effect of rapid right ventricular pacing on aortic and left ventricular pressures in patients with valvar

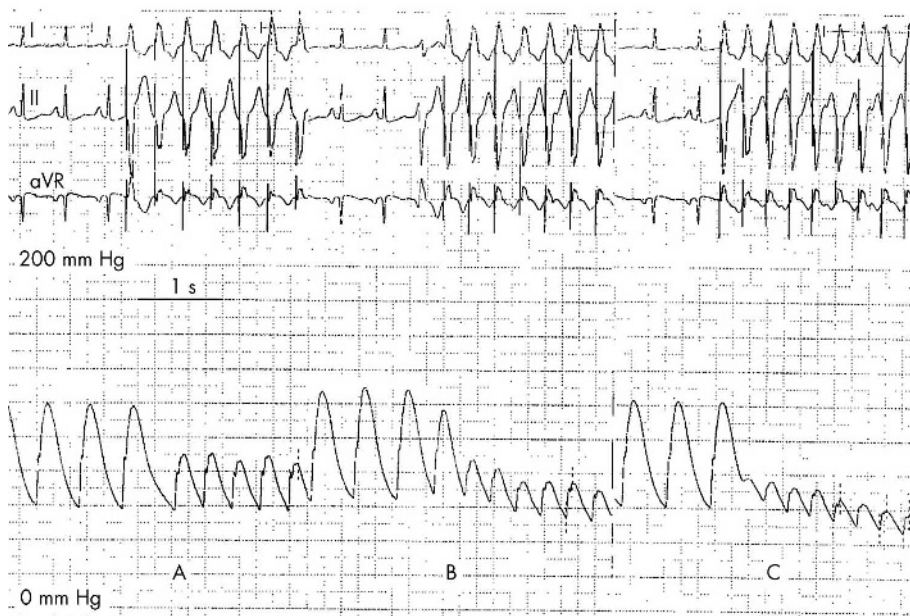


Figure 1 Aortic pressure before and during rapid right ventricular pacing at different rates (A, 180 beats/min; B, 200 beats/min; C, 220 beats/min) in a patient with valvar aortic stenosis before balloon dilatation (patient 11).

aortic stenosis. Another favourable mechanism contributing to device stability in rapid right ventricular pacing is a frequency dependent effect of the physical inertia of the interventional system.

Protocol

The procedure was performed under analgesic sedation (midazolam 0.1 mg/kg, ketamine 1 mg/kg) and local anaesthesia of the groin. A defibrillator set to about 3 J/kg body weight was on standby. Arterial and venous femoral access was obtained. The aortic valve was crossed retrogradely after pressure recording and aortic root angiogram. The left ventricular pressure was recorded and a left ventricular angiogram was taken. The diameter of the dilatation balloon was chosen to be not larger than the aortic valve diameter. The balloon length was chosen to be not smaller than two

times and not larger than three times the balloon diameter. It was introduced with a long extra stiff guidewire. The first balloon dilatation was performed in the usual manner. When the balloon stayed correctly in place in the aortic valve until the balloon was fully inflated, the ventricle was not stimulated. When the balloon slipped through the valve, a 4 French bipolar pacing catheter was introduced from the venous access to the right ventricular apex. A single chamber pacemaker capable of rapid stimulation was connected, the VVI mode was chosen, and effective sensing and stimulation were confirmed. Balloon dilatation was then repeated during rapid right ventricular pacing at a rate of 220 beats/min. The sequence was as follows: start of cine sequence, start of rapid pacing, balloon inflation, balloon deflation, end of rapid pacing, end of cine angiogram. If the balloon continued to move, the manoeuvre was repeated at 240 beats/min.

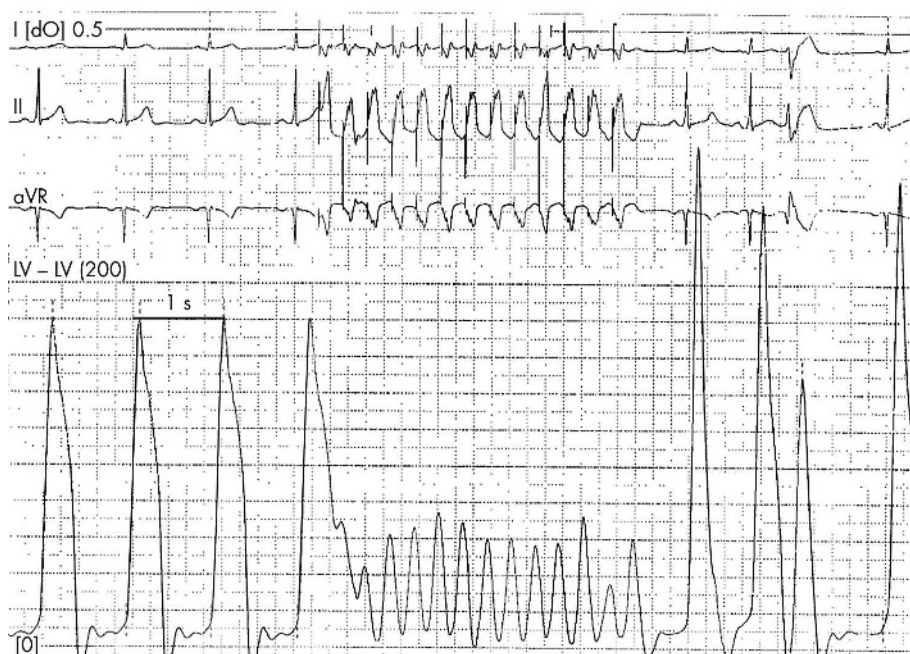


Figure 2 Ventricular pressure before and during right ventricular pacing at 220 beats/min in a patient with valvar aortic stenosis before balloon dilatation (patient 14).

Finally, the balloon catheter was exchanged with a pigtail catheter. Pressure recordings and angiograms of the left ventricle and aorta were repeated.

RESULTS

Fourteen patients required rapid pacing. Table 1 shows the stimulation response and haemodynamic results. The balloon remained in a stable position during the first procedure in 11 patients. In three patients the balloon was displaced. In the two patients with the largest valvar annulus (patients 3 and 12) the balloon moved into the aorta. After the pacing rate was increased to 240 beats/min the balloon stayed in position. In one patient (patient 10) the balloon was placed too proximally and moved backwards into the ventricle during inflation. Correct positioning during the next attempt provided a stable balloon at the same pacing rate. The duration of each manoeuvre was less than 16 seconds in all patients. There were no sustained arrhythmias after cessation of ventricular stimulation. There was haemodynamic improvement in all patients with a fall in peak gradient from a mean of 82.5 mm Hg to 28.6 mm Hg. No patient developed severe aortic regurgitation. Aortic regurgitation increased by one degree in 11 patients and remained unchanged in three patients. No patient developed mitral regurgitation. Post-interventional echocardiographic and Doppler studies were consistent with the results recorded at the time of catheterisation in all patients. No patient developed neurological symptoms or signs at the time of or after the procedure. There were no other procedure related complications.

DISCUSSION

Balloon dilatation is an established method of treating congenital aortic stenosis.⁷⁻⁹ Balloon stability during the procedure is crucial for a successful procedure and likely to reduce potential complications. However, cardiac contraction tends to push the balloon across the stenosed valve into the aorta. Movement of the balloon by cardiac contraction during inflation can be prevented by manoeuvres such as the use of extra stiff wires¹ and double balloons.¹⁰ A special coaxial double balloon was developed for stent delivery.¹¹ In our experience, stiff guidewires are sufficient to prevent balloon movement in neonates and most infants. Balloon

displacement is common in older children and adolescents, as stroke volume has increased and heart rate decreased.

Adenosine is a powerful drug that creates arterial hypotension and leads to transient cardiac standstill after bolus injection. In 1996 Dorros and Cohn⁴ presented their experience with adenosine for the precise deployment of aortic stent grafts. In 1998 De Giovanni and colleagues³ described the use of adenosine to create transient cardiac standstill during balloon dilatation of congenital aortic valve stenosis, pulmonary valve stenosis, coarctation, and conduit stenosis. However, the dose-response to adenosine varies widely and has to be tested individually.⁶ A standard adenosine dose may induce a wide range of onset and duration of asystole.^{3 5 6} In addition, adenosine does not prevent ventricular extrasystoles, which may occur spontaneously or can be triggered by the intraventricular part of the guidewire or the balloon itself during inflation. Therefore, adenosine has not become a standard tool for transcatheter interventions depending on balloon stability. Kahn and colleagues¹² described cardiac standstill by induction of ventricular fibrillation to facilitate endovascular stent graft deployment in 1998. The authors claim that this technique has the advantage over high dose adenosine of being able to predict cardiac arrest time. However, defibrillation was necessary to restore synchronised cardiac activity after the procedure.

Rapid right ventricular pacing decreases stroke volume and blood pressure without causing cardiac standstill. Onset and duration can be individually adapted to the needs of the intervention. Spontaneous or intervention triggered extrasystoles are irrelevant. These advantages seem to justify the hypothetical increase in the risk of sustained ventricular arrhythmias, which must be anticipated in all cardiac catheterisation procedures. In our series, no sustained arrhythmias were induced. In the study setting, rapid right ventricular pacing was used only after the balloon was displaced. However, in older children and adolescents it may be advisable to use rapid pacing without a previous attempt at balloon inflation, as displaced balloons can still damage the aortic valve.

Thus, the method of rapid ventricular pacing provides balloon stability facilitating successful procedures and possibly reducing the risk of complications.

Table 1 Stimulation frequency, balloon response, pre- and post-interventional peak systolic gradients and pre- and post-interventional aortic regurgitation in patients undergoing aortic valve balloon dilatation with rapid right ventricular stimulation

Patient	Pacing		Balloon size			Gradient		Regurgitation	
	Rate (beats/min)	Time (s)	Length (mm)	Diameter (mm)	Balloon displacement	Before dilatation (mm Hg)	After dilatation (mm Hg)	Before dilatation	After dilatation
1	220	12	60	20	None	75	10	None	Mild
2	220	14	60	20	None	110	50	Trivial	Mild
3a	220	10	60	25	Aorta	110	NA	Mild	NA
3b	240	15	60	25	None	NA	50	NA	Mild
4	220	13	40	20	None	100	20	None	Mild
5	220	14	40	18	None	100	40	Mild	Moderate
6	220	12	60	18	None	60	20	None	Mild
7	220	11	60	18	None	110	20	Trivial	Mild
8	220	10	30	10	None	75	30	None	None
9	220	15	60	22	None	60	15	None	Mild
10a	220	07	40	16	Ventricle	60	NA	None	NA
10b	220	14	40	16	None	NA	35	NA	Mild
11	220	15	40	18	None	70	20	Mild	Moderate
12a	220	12	60	25	Aorta	80	NA	Mild	NA
12b	240	14	60	25	None	NA	25	NA	Mild
13	220	12	60	22	None	70	30	Trivial	Mild
14	220	16	60	22	None	75	35	None	Mild

NA, not applicable.

Study limitations

The number of patients is too small to compare results and complications with similar groups that were treated with adenosine or without adjuvants. The group of neonates and infants is not appropriate for comparison because of different demographic data. The frequency of 220 beats/min was chosen randomly. We did not address frequency titration; however, this may be advisable to find an optimal pacing rate.

Conclusion

Rapid ventricular pacing is a safe and effective method to provide transient reduced cardiac output during balloon dilatation of the aortic valve. This achieves stability of the dilatation balloon and may improve results and reduce complications. Rapid pacing may be applied in other fields of catheter intervention where low cardiac output is desirable to maintain stable device positions during the critical phase of the procedure.

Authors' affiliations

I Daehnert, C Rotzsch, M Wiener, P Schneider, Klinik fuer Kinderkardiologie, Herzzentrum, Universitaet Leipzig, Leipzig, Germany

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Immunosuppressive therapy in acute myocarditis: an 18 year systematic review

C P P Hia, W C L Yip, B C Tai, S C Quek

Please visit the Heart website [www.heartjnl.com] for a link to the full text of this article.

Background: Immunosuppressive therapy is reportedly ineffective in adults with acute myocarditis.

Aims: To systematically review the impact of immunosuppressive therapy on the outcome of acute myocarditis in children.

Methods: A literature search for articles published from 1984 to 2003 was conducted with the following keywords: myocarditis, dilated cardiomyopathy, and immunosuppression. The relevant studies were systematically reviewed and comparison of treatment effect was made by calculating the odds ratio (OR) and confidence interval (CI) using the exact method based on the exact discrete reference distribution.

Results: Of the 1470 articles found, only nine studies were eligible. The odds for improvement with immunosuppression was between 4.33 (95% CI 0.52 to 52.23) and 2.7 (95% CI 0.59 to 14.21). Addition of a second immunosuppressive agent to prednisolone only proved effective in one randomised controlled trial (OR 0.09, 95% CI 0.01 to 0.52). Heterogeneity of these studies precluded pooled odds ratio.

Conclusion: Current data suggest that immunosuppressive therapy does not significantly improve outcomes in children with acute myocarditis and there is insufficient evidence for its routine use. However, statistical power to detect a significant difference in the treatment effect may be limited because of the small number of subjects. This, together with problems of diagnosis, varying treatment practices, and a relative lack of evidence based guidelines would support efforts for a large multicentre, randomised controlled trial to better define the role of immunosuppression in acute myocarditis.

▲ *Archives of Disease in Childhood* 2004;**89**:580–584.