Dynamic right ventricular outflow tract obstruction in cardiac surgery

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Background: Right ventricular outflow tract obstruction can be a cause of hemodynamic instability but it has not been described in non-congenital cardiac surgery.

Methods: The prevalence of right ventricular outflow tract obstruction was retrospectively studied in 670 consecutive patients undergoing cardiac surgery. Significant right ventricular outflow tract obstruction was diagnosed if the right ventricular systolic to pulmonary artery peak gradient was more than 25 mm Hg. The diagnosis was based on measurement of the right ventricular and pulmonary artery systolic pressure through the paceport and distal opening of the pulmonary artery catheter. To further validate the prevalence and the importance of right ventricular outflow tract obstruction, 130 patients were prospectively studied over a 12-month period.

Results: In the retrospective cohort, 6 patients (1%) undergoing various types of cardiac surgical procedures were found to have significant dynamic right ventricular outflow tract obstruction with a mean gradient of 31 ± 4 mm Hg (26 to 35 mm Hg). In the prospective study significant dynamic right ventricular outflow tract obstruction was identified in 5 patients (4%) (average peak: 37 ± 15 mm Hg; range: 27 to 60 mm Hg). The typical transesophageal echocardiography finding was end-systolic obliteration of the right ventricular outflow tract. In patients with significant dynamic right ventricular outflow tract obstruction, hemodynamic instability was present in 10/11 patients (91%).

Conclusions: Right ventricular outflow tract obstruction is easily diagnosed using the paceport of the pulmonary artery catheter and should be considered as a potential cause of hemodynamic instability especially when transesophageal echocardiography reveals systolic right ventricular cavity obliteration.

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Hemodynamic instability after cardiac surgery has various etiologies including ventricular dysfunction, hypovolemia, tamponade, left ventricular outflow tract (LVOT) obstruction and sometimes a combination of these factors.¹ Right ventricular outflow tract (RVOT) obstruction, which can be due to extrinsic²⁻⁴ or intrinsic causes,⁵⁻⁷ can also result in hemodynamic instability. According to time-honoured hemodynamic criteria, RVOT obstruction is defined as "significant" when the peak right ventricular to pulmonary artery systolic gradient exceeds 25 mm Hg.⁸ Furthermore, on transesophageal echocardiography (TEE), significant RVOT obstruction is defined as "fixed" if there is no change in RVOT dimensions during the cardiac cycle with anatomic substrate for obstruction, and as "dynamic" if RVOT dimensions increase appreciably in diastole. Dynamic RVOT obstruction has been observed in hypertrophic cardiomyopathy⁹ and after lung transplantation^{10,11} but it has rarely been described during cardiac surgery.¹² Over a 3-year period, new-onset significant dynamic RVOT obstruction was seen in 11 patients undergoing cardiac surgery. The diagnosis of RVOT obstruction was made

Abbreviations and Acronyms

AVR	= aortic valve replacement
CPB	= cardiopulmonary bypass
HTN	= hypertension
LVOT	= left ventricular outflow tract
RVOT	= right ventricular outflow tract
TEE	= transesophageal echocardiography

using continuous right ventricular pressure and pulmonary artery pressure monitoring. After having identified and characterized this anomaly in a retrospective study, we hypothesized that this new knowledge integrated in a systemic hemodynamic and echocardiographic assessment would allow determination of a higher but correct incidence of this problem in a prospective study. Our objective was also to better understand the mechanism and risk factors involved in the appearance of RVOT obstruction.

TABLE 1. Retrospective series

				Predisposing factor	HTN	PHT	CPB duration (min)	DSB		
Age	Sex		Procedure						Details	Outcome
1	75	m	Coronary revascularization and AVR	Inotropes and LVH	Y	N	147	Y	Return on CPB 3 times, intra-aortic balloon pump inserted. Noradrenaline 66 µg/min, adrenaline 4 µg/min, vasopressine 2 units/hour, nitric oxide 20 ppm, milrinone 50 µg/kg and dopamine 5 µg/kg/min. Milrinone and dopamine were discontinued once the diagnosis was made	Develop acute renal failure, and acute respiratory distress syndrome, vasoactive support for 24 hours, hospitalisation 20 days
2	46	f	Coronary revascularization and aortic surgery	Inotropes and LVH	N	Y	346	Y	Noradrenaline 13 µg/min, vasopressin 2 units bolus and nitroglycerine 1.3 µg/kg/min, inhaled prostacyclin 60 µg, milrinone 50 µg/kg, methylene blue 50 mg	Vasoactive support for 10 hours, hospitalisation 10 days
3	58	m	AVR, MVR, aortic surgery	Inotropes and LVH	N	Y	205	Y	Noradrenaline 2.5 µg/min, inhaled prostacyclin 60 µg and inhaled milrinone 5 mg, nitroglycerine 0.5 µg/kg/min	Vasoactive support 3 hours, hospitalisation 8 days
4	51	m	AVR, VSD closure, aortic surgery	Inotropes	N	Y	129	Y	Noradrenaline 10 µg/min, vasopressine 2 units bolus, milrinone 50 µg/kg, methylene blue 100 mg	Vasoactive support 3 hours, ventricular tachycardia requiring pace-defibrillator, hospitalisation 19 days
5	62	m	Septal myomectomy, aortic surgery	Septal hypertrophy	Y	N	86	Y	Noradrenaline 12.5 µg/min, nitroglycerine 0.7 µg/kg/min	Vasoactive support 20 hours, acute renal failure, hospitalisation 18 days
6	68	m	Coronary revascularization	Inotropes and LVH	Y	N	93	Y	Noradrenaline 7.5 µg/kg, nitroglycerine 0.7 µg/kg/min, milrinone 50 µg/kg	Vasoactive support 2 hours, hospitalisation 6 days

AVR, aortic valve replacement; CPB, cardiopulmonary bypass; DSB, difficult separation from CPB (details in Table 4); f, female; HTN, hypertension; LVH, left ventricular hypertrophy; m, male; min, minutes; MVR, mitral valve repair; PHT, pulmonary hypertension; VSD, ventricular septal defect.

Methods

After approval by the Research and Ethics Committees of our institution, the Department of Anesthesia has maintained for quality control purpose, a TEE database since 1999 to store demographic, hemodynamic and echocardiographic data. Informed consent was obtained from all patients for intraoperative use of pulmonary artery catheterization and TEE. Since August 2002, a retrospective analysis of all patients undergoing cardiac surgery was performed. Demographic data included age, gender and the presence of hypertension defined through patient's history and medications. All these patients were monitored by continuous right ventricular pressure waveform analysis through the pulmonary artery catheter paceport (Edwards Lifescience, Irvine, Ca). The paceport opening is located at 19 cm from the distal end of the catheter. Transducers extension tubing are connected to the distal port and to the paceport of the pulmonary artery catheter. The catheter is advanced until the hemodynamic curve from the paceport changes from an atrial to a ventricular pressure waveform. Then the catheter is maintained in this position throughout the procedure therefore allowing continuous monitoring of right ventricular and pulmonary artery pressure waveforms. In all patients, after the induction of anesthesia, routine hemodynamic waveforms were acquired during insertion of the pulmonary artery catheter. The normal gradient between right ventricular and pulmonary artery systolic pressure is 0 to 3 mm Hg¹³ and peak gradients between 3 and 25 mm Hg are often considered trivial based on the literature pertaining to chronic RVOT obstruction. In contrast, peak gradients above 25 mm Hg are considered significant⁸ and this cut-off value was used in this study. The gradient is calculated as the difference between the peak systolic right ventricular pressure and peak systolic pulmonary artery pressure using the hemodynamic tracing or directly calculated from the monitor. In addition to pulmonary artery catheterization, perioperative TEE monitoring (Philips, Sonos 5500, Andover, Ma) was performed by a cardiac anesthesiologist trained and board-certified in TEE in all patients undergoing cardiac surgery. The echocardiographic exam included the recommended views,¹⁴ the evaluation of diastolic function¹⁵ and the interrogation of all four valves by color Doppler. Color Doppler imaging in the mid-oesophageal four-chamber view between 0 to 70° was used to detect tricuspid regurgitation and continuous-wave Doppler was positioned to obtain the maximum regurgitant jet to measure the peak tricuspid regurgitant velocity. This peak velocity reflects the peak pressure gradient between the right ventricle and right atrium through the Bernoulli equation.¹⁶

Echocardiographic images suggestive of the abnormal RVOT obstruction were obtained through 2D echocardiography using an inflow-outflow view of the right ventricle or through a deep transgastric view at 40° as previously described.¹⁷

Pulmonary hypertension was defined as a preoperative mean pulmonary artery pressure above 25 mm Hg obtained during preoperative catheterization or a systolic pulmonary artery pressure above 30 mm Hg¹⁸ measured directly in the operating room. Left ventricular hypertrophy was determined using either electrocardiographic findings¹⁹ or the following echocardiographic criteria: posterior wall thickness ≥ 12 mm, left ventricular mass above 131 g/m² in men or 110 g/m² in women. Difficult separation from cardiopulmonary bypass (CPB) was identified as systolic blood

Figure 1. Septal myomectomy and aortic surgery in a 68-year-old man complicated by dynamic RVOT obstruction appearing during weaning from cardiopulmonary bypass. (A) The systolic gradient between the right ventricle and the pulmonary artery was 28 mm Hg. (B,C,D) An M-mode view from a mid-oesophageal right-ventricular inflow-outflow view at 63° is illustrated with the systolic dynamic obstruction of the right ventricular outflow (LA, left atrium; Pa, arterial pressure; Ppa, pulmonary artery pressure; Ppv, right ventricular pressure; RA, right atrium; RV, right ventricle).

pressure < 80 mm Hg, confirmed by central measurement (femoral or aortic), diastolic pulmonary artery pressure or pulmonary artery capillary wedge pressure > 15 mm Hg, and inotropic or vasopressor support for more than 1 hour (norepinephrine > 4 $\mu\text{g}/\text{min}$, epinephrine > 2 $\mu\text{g}/\text{min}$, dobutamine > 2 $\mu\text{g}/\text{kg min}^{-1}$) or the use of milrinone, mechanical support or intra-aortic balloon pump (IABP)^{1,20,21} to wean from CPB until transfer to the intensive care unit. All echocardiographic recordings were stored on magnetic optical disk and the examination findings stored in the database. The hemodynamic tracings were recorded on videotape. Finally to determine the precise incidence of RVOT obstruction, in the prospective series, 130 patients operated from September 2004 to May 2005 were continuously monitored using the paceport of the pulmonary artery catheter from the beginning to the end of the procedure. Chi-square test was used to compare RVOT obstruction in patients with or without difficult separation from CPB. The data are reported as mean $\pm$ standard deviation.

Results

From August 2002 to September 2004, a total of 8 out of 670 consecutive patients undergoing TEE were found to have a RVOT gradient > 25 mm Hg. Two patients had transient mechanical RVOT obstruction during off-pump bypass surgery and were excluded from the analysis. Patient characteristics are listed in Table 1. The mean age was 60 ± 11 years, the mean duration of CPB was 168 ± 97 minutes and the average peak gradient was 31 ± 4 mm Hg (range 26 to 35 mm Hg). In all these 6 patients (1%), RVOT obstruc-

TABLE 2. Prospective series

							CPB			
Age	Sex	Procedure	Predisposing factor	HTN	PHT	duration (min)	DSB	Details	Outcome	
1	26	F	AVR and aortic pseudoaneurysm repair	Inotropes	N	N	138	Y	Noradrenaline 4 μg/min, vasopressine 2 units/h, nitroglycerine 0.14 μg/kg/min, inhaled milrinone 5 mg	Vasoactive support for 40 hours, hospitalisation 21 days
2	61	M	Coronary revascularization	None	Y	N	69	No	Noradrenaline 2.5 μg/kg/min, vasopressine 3 units bolus	Vasoactive support 3 hours, hospitalisation 5 days
3	24	M	Repeated aortic surgery (Cabrol)	Inotropes	N	N	208	Y	Noradrenaline 9 ug/min, nitroglycerine 0.3 μg/kg/min, vasopressine bolus 2 units, calcium chloride1 gm	Vasoactive support for 20 hours, death from severe heart failure from myocardial infarction 6 days later despite left ventricular assist device
4	46	M	Heart transplantation	Inotropes	N	Y	121	Y	Noradrenaline 25 μg/min, nitroglycerine 0.7 μg/kg/min, milrinone 50 μg/kg, inhaled prostacyclin 60 μg, adrenalin 2 μg/min, vasopressine 2 units/hour, isoproterenol 3 μg/min	Vasoactive support for 27 days, hospitalisation 119 days
5	68	M	AVR	LVH, inotropes	Y	N	168	Y	Noradrenaline 30 μg/min, nitroglycerine 0.2 μg/kg/min, inhaled milrinone 5 mg, ephedrine 5 mg, vasopressine 1 unit bolus	Had unexplained hypotension requiring vasoactive support for 3 days, hospitalisation 9 days

AVR, aortic valve replacement; CPB, cardiopulmonary bypass; DSB, difficult separation from CPB; HTN, hypertension; LVH, left ventricular hypertrophy; min, minutes; PHT, pulmonary hypertension.

TABLE 3. Hemodynamic conditions during RVOT obstruction

Retrospective series	HR (beats/min)	Pa (mm Hg)	Ppa (mm Hg)	Systolic Prv (mm Hg)	RVOT Gradient (mm Hg)	Pra (mm Hg)
Patient 1	91	86/44 (68)	34/22 (24)	69	35	20
Patient 2	95	118/42 (59)	21/11 (15)	47	26	11
Patient 3	100	113/56 (76)	29/17 (22)	59	30	13
Patient 4	71	85/55 (65)	55/27 (38)	90	35	15
Patient 5	80 paced	159/84 (109)	32/22 (26)	60	28	6
Patient 6	125	116/48 (70)	18/11 (14)	48	30	8
Prospective series						
Patient 1	93	108/74 (88)	25/18 (20)	52	27	18
Patient 2	90	123/57 (79)	27/14 (20)	54	27	12
Patient 3	100	112/40 (55)	18/11 (15)	62	44	8
Patient 4	94	117/69 (86)	39/21 (28)	67	28	8
Patient 5	103	123/51 (70)	18/9 (14)	78	60	4

RVOT, right ventricular outflow tract; HR, heart rate; Pa, arterial pressure; Ppa, pulmonary artery pressure; Prv, right ventricular pressure; Pra, right atrial pressure.

Figure 2. A 68-year-old man underwent aortic valve replacement. He became hemodynamically unstable with right ventricular dysfunction and went back on cardiopulmonary bypass. Inotropes were started. On the second weaning attempt, he developed severe right ventricular outflow tract obstruction confirmed with the paceport of the pulmonary artery catheter and through continuous-wave Doppler interrogation of the tricuspid regurgitant flow in a mid-oesophageal right ventricular inflow-outflow view at 61°. The measured pressure gradient of the tricuspid regurgitant flow was 75 mm Hg (with a right ventricular systolic pressure of 80 mm Hg) and the pulmonary artery pressure (Ppa) was 30/16 mm Hg during the echocardiographic measurement. (EKG, electrocardiogram; Pa, arterial pressure; Prv, right ventricular pressure).

tion occurred after CPB and was associated with systolic obliteration in the RVOT (Figure 1). In most of these patients, the gradient improved or normalized as the right ventricular preload was increased and the inotropic agents were decreased or discontinued.

In the prospective study (Table 2), a total of 6 patients out of 130 had significant RVOT obstruction. One patient had RVOT obstruction during ventricular mapping secondary to cardiac manipulation and was excluded. Five patients (4%) were therefore found to have new-onset significant dynamic RVOT obstruction after CPB. The mean age of the patients was 45 ± 20 years, the mean

duration of CPB was 141 ± 52 minutes and the average peak gradient was 37 ± 15 mm Hg (range 27 to 60 mm Hg). The hemodynamic conditions of both groups of patients at the time that the RVOT obstruction was observed are described in Table 3. The average heart rate was 96 ± 13 beats/min, systolic, diastolic and mean systemic arterial pressures were 114 ± 20 , 56 ± 14 and 75 ± 15 mm Hg, systolic, diastolic and mean pulmonary artery diastolic pressures were 29 ± 11 , 17 ± 6 and 21 ± 7 mm Hg. The average systolic right ventricular and right atrial pressures were 63 ± 13 mm Hg and 11 ± 5 mm Hg. The mean gradient was 34 ± 10 mm Hg (range 26 to 60 mm Hg). Difficult separation from CPB was observed in 41% of the 670 patients in the retrospective group, in 46% of the 130 patients of the prospective group and in 91% of patients with dynamic RVOT obstruction ($P = .0014$). The drugs required for weaning from CPB, the duration of vasoactive support and the outcome are described in Table 1 & 2. In 3 patients in whom a Doppler tricuspid regurgitant jet could be measured, the pressure gradient estimated with the simplified Bernoulli equation was superior to the pulmonary artery pressure (Figure 2).

Discussion

The observed prevalence of significant dynamic RVOT obstruction in the study was 1% in the retrospective cohort, while it was 4% in the prospective cohort. In 10/11 patients (91%) RVOT obstruction was associated with difficult separation from CPB from hemodynamic instability. The diagnostic criteria of RVOT obstruction and the associated echocardiographic findings observed in the study are summarized in Table 4.

Right ventricular outflow tract obstruction was documented in all patients through right ventricular pressure monitoring, with direct pressure measurements in the

TABLE 4. Criteria for significant right ventricular outflow tract (RVOT) obstruction

Hemodynamic findings

- Systolic right ventricular to pulmonary artery gradient >25 mm Hg measured through
 - combined pulmonary artery and right ventricular pressure monitoring
 - pulmonary artery catheter pullback in the right ventricle if paceport unavailable.

Echocardiographic findings consistent with RVOT obstruction

*First exclude other mechanisms of hemodynamic instability

M-mode: Mid-oesophageal inflow-outflow view showing obliteration of the RVOT

Two-dimensional:

Mid-oesophageal inflow-outflow view showing obliteration of the RVOT

Deep transgastric view with right-sided rotation 40° showing RVOT obliteration

Color Doppler:

Aliasing in the RVOT in the mid-oesophageal right ventricular inflow-outflow view with/without M-mode

Continuous-wave Doppler:

Transtricuspid pressure gradient $>$ systolic pulmonary artery pressure

Pulsed-wave Doppler:

Atrial-reversal velocity more than 50% of the systolic velocity of the hepatic venous flow suggestive of right ventricular impairment.

pulmonary artery and right ventricle. Systolic obliteration of the RVOT was seen with TEE in the mid-oesophageal right ventricular inflow-outflow tract view. Right ventricular systolic pressure derived from continuous Doppler interrogation of the tricuspid valve and central venous pressure also revealed that the right ventricular maximal systolic pressure was higher than the pulmonary artery systolic pressure.

The Association Between RVOT Obstruction and Hemodynamic Instability

Extrinsic RVOT obstruction has been recognized as a possible cause of hemodynamic instability¹⁷ after cardiac surgery. Extrinsic compression can occur from an aortic² or pulmonary artery aneurysm,³ mediastinal hematoma⁴ or direct surgical compression during off-pump surgery. In contrast, intrinsic obstruction has been described with congenital heart disease,^{5,6,7} hypertrophic cardiomyopathy⁹ and after lung transplantation.^{10,11}

New onset dynamic subvalvular RVOT obstruction, as was observed in this study, usually occurs in the setting of reduced preload, hypertrophied right ventricle and in patients on inotropic therapy.¹⁰ In our series inotropic agents and left ventricular hypertrophy were present in 10 patients (91%). The occurrence of dynamic RVOT obstruction in a patient on inotropic therapy could contribute to hemodynamic instability. This conclusion is based on the fact that further deterioration of the hemodynamic status of the patients in the study was often associated with the development of significant RVOT obstruction. In this circumstance, optimisation of right ventricular preload instead of increasing inotropic therapy, improved the clinical status of the patients.

Although uncommon, the presence of significant dynamic RVOT obstruction should be suspected in any hemodynamically unstable patients receiving inotropes. In our patients, none had a significant right ventricular to pulmonary artery systolic gradient before CPB and inotropes were used only after CPB for weaning. The average CPB time was more than 2 hours which increases the risk of cardiac function impairment and may explain why the anesthesiologist considered using inotropic agents. These agents were stopped once the diagnosis of RVOT obstruction was made. The administration of inotropic agents may have promoted RVOT obstruction in our study. It should be emphasized that significant RVOT obstruction can also occur without any preoperative risk factor as we observed in one patient. In the operating room, continuous right ventricular pressure monitoring through the pacemaker port represents an easier method to rapidly diagnose RVOT obstruction compared to pulling back the pulmonary artery catheter in the right ventricle. In the latter situation, ventricular arrhythmias can occur, which we did not observe using the paceport catheter as a continuous pressure monitor.

Pathophysiologically, dynamic RVOT obstruction can be explained by the increased susceptibility of the infundibulum to inotropic agents. In fact, the inotropic response of the infundibulum is greater than that of the inflow tract, perhaps as a mechanism to protect the pulmonary vasculature from high pressure.^{22,23} This differential response could explain the presence of dynamic RVOT obstruction in patients on inotropes or during cardiac surgery. Moreover, the coordinated sequence of activation of the right ventricle can also be reversed by the post-operative hyperadrenergic states.²⁴ In normal conditions, right ventricular contraction is sequential starting with the contraction of the trabeculated myocardium and ending with that of the infundibulum (approximately 25 to 50 milliseconds later). In the presence of sympathetic stimulation or norepinephrine infusion the normal sequence of activation may be abolished and RV inflow contraction can even occur after infundibular contraction.

There are several limitations from this case series. The diagnosis of RVOT obstruction was unexpected and most of the time was associated with hemodynamic instability. Therefore we did not have the opportunity to obtain a detailed hemodynamic profile. This is why we only reported the maximal gradient that occurred at that time. The number of patients is also too small to make generalisation about the best therapeutic approach but in all our patients, except the one undergoing heart transplantation, the inotropes were stopped and avoided in the post-operative period. However, the interest of our findings lies at this point in the knowledge of this entity when faced with patients with hemodynamic instability after CPB and the potential correct therapy.

In summary, dynamic RVOT obstruction can occur in several types of cardiac surgery. The diagnosis is easily obtained with continuous right ventricular and pulmonary artery pressure monitoring, and can also be suspected with TEE. In cardiac surgical patients experiencing hemodynamic instability, dynamic RVOT obstruction should be considered especially when TEE reveals right ventricular cavity obliteration or turbulent flow patterns. The presence of dynamic RVOT obstruction can contribute to hemodynamic instability, and in that situation inotropic agents could be responsible for or could exacerbate this condition. In our experience, preload optimization and inotropic agents withdrawal were successful in the management of this condition.

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