

Incidence, Management, and Outcomes of Cardiac Tamponade During Transcatheter Aortic Valve Implantation

A Single-Center Study

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Objectives The aim of this study was to explore the incidence, causes, and outcomes of cardiac tamponade in patients undergoing transcatheter aortic valve implantation (TAVI).

Background Use of TAVI is increasing, but the procedure is vulnerable to complications, given the cohort of patients. Cardiac tamponade is a possible complication, and there is a scarcity of data on the incidence and outcomes of cardiac tamponade during TAVI.

Methods All patients who sustained cardiac tamponade during or post-TAVI between 2007 and 2012 were included in the study.

Results Of 389 patients who underwent TAVI, 17 (4.3%) had cardiac tamponade. The mean age was 82.3 ± 3.7 years, and most were women ($n = 12$, 70.6%). Causes of cardiac tamponade were right ventricular perforation by temporary pacemaker (9 patients, 52.9%), annular rupture or aortic dissection (4 patients, 23.5%), and tear in the left ventricular free wall caused by Amplatz stiff wire or catheters (4 patients, 23.5%). Mortality occurred in 4 patients (23.5%), and all had tamponade caused by injury to the high-pressured left-sided circulation (left ventricle and aorta). Most patients ($n = 14$, 82.4%) sustained cardiac tamponade during the procedure—2 patients (11.7%) within 24 h, and 1 patient after 24 h.

Conclusions Cardiac tamponade during TAVI is not frequent but is associated with high mortality rates especially when left-sided structures are involved. Meticulous handling of the equipment and improvements in the safety of currently used devices could further reduce the occurrence of this complication. (J Am Coll Cardiol Intv 2012;5:1264–72) © 2012 by the American College of Cardiology Foundation

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Transcatheter aortic valve implantation (TAVI) is a new approach for the treatment of severe aortic stenosis (1,2). The significant clinical benefit in high-risk elderly patients, for whom few options were previously available, led to a unique dissemination of TAVI. However, enthusiasm and caution must be carefully balanced. Transcatheter aortic valve implantation remains a complex and technically demanding procedure that requires advanced skills and intensive training. Moreover, TAVI is associated with some complications that need to be further reduced to improve the procedural outcome. Cardiac tamponade is 1 such complication, which is associated with high mortality. This article reviews the presentation, frequency, timing, and outcome of cardiac tamponade during TAVI and discusses possible predisposing factors as well as our suggestions to minimize the occurrence of this serious complication.

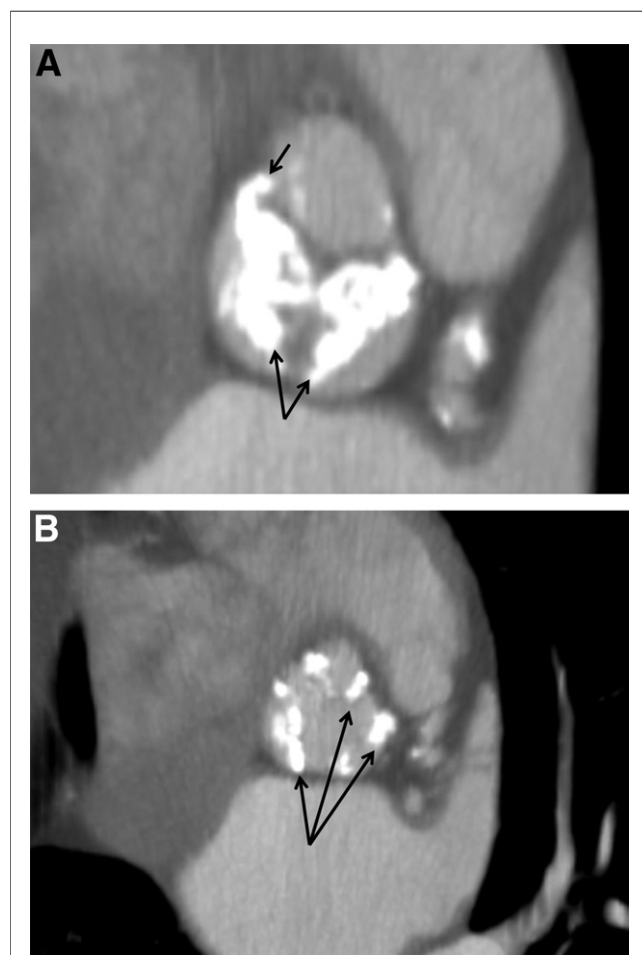


Figure 1. CT Scan Demonstrating Distribution of Calcium on the Aortic Valve

(A) Computed tomography (CT) scan exhibiting commissural calcification (arrows). (B) CT scan exhibiting annular calcification (arrows).

Methods

Study design and patient population. This is a single-center, retrospective observational cohort study of patients who developed cardiac tamponade during or after TAVI procedures. From November 2007 to January 2012, 389 patients underwent TAVI in our institution. Seventeen patients developed cardiac tamponade defined as life-threatening slow or rapid compression of the heart due to the pericardial accumulation of fluid or blood because of effusion, trauma, or rupture of the heart or surrounding structures (3,4).

Patient selection and risk stratification. After evaluation by a team of cardiologists and cardiothoracic surgeons, patients with severe symptomatic aortic stenosis requiring aortic valve replacement were considered for TAVI if the risk for surgical aortic valve replacement was “high” according to the logistic European System for Cardiac Operative Risk Evaluation (EuroSCORE) (5) and the Society of Thoracic Surgeons (STS) score (6) or if they were considered “inoperable” (7,8). All patients had severe aortic valve stenosis with a valve area $\leq 1 \text{ cm}^2$, with or without aortic valve regurgitation and a logistic EuroScore or STS score $\geq 10\%$ (5,6) or logistic EuroScore $< 10\%$ but considered inoperable. Pre-interventional screening included transthoracic echocardiography to confirm diagnosis and to evaluate the left ventricular (LV) dimensions and volumes. Multi-slice computed tomography (MSCT) was performed to evaluate dimensions and calcification of aortic valve,

Abbreviations and Acronyms

EuroScore = European System for Cardiac Operative Risk Evaluation

LV = left ventricle/ventricular

MSCT = multi-slice computed tomography

RV = right ventricle/ventricular

STS = Society of Thoracic Surgeons

TAVI = transcatheter aortic valve implantation

TPW = temporary pacemaker wire

aorta, peripheral vessels, and coronary anatomy. All patients gave written informed consent for the procedure and data collection for research purposes. All patient, procedural, and follow-up data are prospectively entered into a dedicated database by physicians directly involved with the procedure.

Overview of procedure. Two TAVI systems have seen wide clinical application: the balloon-expandable Edwards valve (Edwards Lifesciences, Irvine, California), and the self-expandable CoreValve ReValving system (Medtronic, Minneapolis, Minnesota). Both systems have been extensively described elsewhere (9–11). Retrograde transarterial or antegrade transapical approaches are used to access the aortic valve.

Retrospective analysis of MSCT. We retrospectively analyzed annular diameters (coronal and sagittal views), the transverse view to calculate the annular eccentricity index in a multi-planar reconstruction in patients who had annular tears. The angle between the aortic root axis and the LV axis

Table 1. Patient Characteristics and Surgical Risk Score

Total Patients (n = 389)	Cardiac Tamponade (n = 17)	No Cardiac Tamponade (n = 372)	p Value
Male sex	5 (29.4)	183 (49.2)	0.06
Age, yrs	82.5 ± 3.6	79.2 ± 7.7	0.07*
Height, cm	159.2 ± 5.4	164.6 ± 8.3	0.01
Weight, kg	70.9 ± 13.7	69.3 ± 10.9	0.65*
Body mass index	27.3 ± 4.0	26.1 ± 4.7	0.21*
Body surface area, m ²	1.71 ± 0.13	1.77 ± 0.19	0.23
Current smoker	0 (0)	12 (3.2)	1.0†
Hypertension	14 (82.4)	265 (71.2)	0.77†
Hypercholesterolemia	6 (35.3)	223 (60.1)	0.04
Diabetes	3 (17.6)	108 (29.0)	0.27
Insulin therapy	2 (11.8)	36 (9.6)	0.67†
Renal insufficiency	2 (11.8)	118 (31.7)	0.07
Patients on dialysis	0 (0)	13 (3.4)	1.0†
COPD	5 (29.4)	127 (34.1)	0.69
Peripheral arterial disease	5 (29.4)	108 (29.0)	1.0†
Cerebrovascular disease	0 (0)	52 (13.9)	0.14†
Coronary artery disease	6 (35.3)	147 (39.5)	0.77
Previous myocardial infarction	1 (5.9)	78 (20.9)	0.22†
Previous PTCA/PCI	2 (11.8)	75 (20.2)	0.54†
Previous CABG	0 (0)	78 (20.9)	0.03†
Angina	2 (11.8)	107 (28.8)	0.10
Syncope	0 (0)	61 (16.4)	0.09†
Baseline creatinine	0.93 (0.84–1.28) mg/dl	1.04 (0.84–1.44) mg/dl	0.29*
Creatinine clearance at admission	46.2 ± 19.2 ml/min	52.7 ± 24.2 ml/min	0.32
Baseline Hb	12.0 ± 1.9 mg/dl	12.0 ± 1.7 mg/dl	0.64*
Porcelain aorta	5 (29.4)	62 (16.7)	0.17†
EuroScore standard, %	9.6 ± 3.0	10.6 ± 2.9	0.11*
Logistic EuroScore	13.8 (9.0–23.4)	19.7 (11.4–31.9)	0.08*
STS score	6.7 (4.5–9.3)	5.9 (3.9–10.0)	0.71*
STS morbidity and mortality	27.7 (20.9–32.4)	27.8 (21.6–40.0)	0.78*

Values are mean ± SD or median (interquartile range) n (%), unless otherwise indicated. *Wilcoxon test; †Fisher exact test.
COPD = chronic obstructive pulmonary disease; CABG = coronary artery bypass graft; EuroScore = European System for Cardiac Operative Risk Evaluation; Hb = hemoglobin; PCI = percutaneous coronary intervention; PTCA = percutaneous transluminal coronary angioplasty; STS = Society of Thoracic Surgeons.

was calculated. Annular and commissural calcifications were also evaluated to understand whether it has a relation with annular tears (Figs. 1A and 1B).

Statistical analysis. Continuous variables are presented as mean ± SD and range. Continuous variables were compared by Student *t* test (for the skewed data, Mann-Whitney test was used), whereas categorical variables were compared by chi-square tests or Fisher exact tests as appropriate. Statistical analysis was performed with SPSS (version 18 for Windows; SPSS, Inc., Chicago, Illinois).

Results

Of 384 patients, 17 (4.3%) developed cardiac tamponade either during or post procedure. Patients who developed tamponade were older than rest of the patients, with a mean age of 82.5 ± 3.6 years versus 79.2 ± 7.7 years, respectively

(*p* value = 0.07), and most were women (*n* = 12, 70.5%). Tamponade occurred in patients who were relatively shorter compared with the rest of the patients who did not develop tamponade (159.2 ± 5.4 cm vs. 164.6 ± 8.3 cm; *p* = 0.01). Mean standard EuroSCORE, Logistic EuroSCORE, and STS score were 9.6 ± 3.0%, 13.8% (9.0% to 23.4%), 6.7% (4.5% to 9.3%), respectively. The STS score for morbidity and mortality was 27.7% (20.9% to 32.4%). The rest of the demographic data are described in detail in Table 1. Details of the aortic valve and LV measurements are tabulated in Online Table 1. No significant differences were detected between patients who developed tamponade versus those who did not in terms of demographic and echocardiographic data.

Procedural data. In the 17 patients who developed tamponade, Edwards valve was implanted in 9 patients (52.9%)—7

Table 2. Procedural Data			
Total Patients (n = 389)	Cardiac Tamponade (n = 17)	No Cardiac Tamponade (n = 372)	p Value
Type of anesthesia			1.0*
Sedation	14 (82.4)	301 (80.9)	
General anesthesia	3 (17.6)	71 (19.1)	
Type of temporary pacemaker wire			
Screw-in lead	4 (23.5)	13 (3.5)	0.005*
Passive leads with a balloon	11 (64.7)	336 (90.3)	0.006*
Epicardial lead	2 (11.7)	23 (6.2)	0.43*
Prosthesis size, mm	26.0 ± 2.0	26.1 ± 2.2	0.45
23-mm valve	3 (17.6)	89 (23.9)	0.57*
26-mm valve	9 (52.9)	183 (49.2)	0.8
29-mm valve	4 (23.5)	94 (25.3)	1.0*
31-mm valve	1 (5.8)	6 (1.6)	0.35*
Sheath size (F)	18.9 ± 1.9	19.5 ± 2.2	0.77†
CoreValve	8 (47.1)	152 (40.9)	0.38
Edwards valve	9 (52.9)	220 (59.1)	0.38
Edwards Sapien XT	7 (41.2)		
Edwards Sapien	2 (11.7)		
Femoral approach	14 (82.3)	309 (83.1)	0.7*
Transapical	2 (11.7)	25 (6.7)	0.3*
Trans axillary	1 (5.8)	38 (10.2)	1.0*
Guidewires			
Amplatz Extra stiff 0.35"	9 (52.9)	220 (59.1)	0.38
Amplatz Superstiff 0.35"	8 (47.1)	152 (40.9)	0.38
TAVI in bioprosthesis, mm	1 (5.8) (23, pericarbon)	14 (3.8)	0.46*
Pre-dilation	11 (64.7)	307 (82.5)	0.10*
Pre-dilation balloon diameter, mm	22.8 ± 1.5	21.9 ± 1.8	0.05†
Post-dilation	5 (29.4)	86 (23.1)	0.56*
Post-dilation balloon diameter, mm	24.4 ± 3.8	Pre-dilation balloon diameter, mm 25.3 ± 2.5	0.52†
Elective percutaneous closure	14 (82.3)	301 (80.9)	1.0*
Elective surgical closure	3 (17.6)	71 (19.1)	1.0*
Device success VARC	15 (88.2)	333 (89.5)	0.34*

Values are mean ± SD or median (interquartile range) n (%), unless otherwise indicated. *Fisher exact test; †Wilcoxon test.
 TAVI = transcatheter aortic valve implantation; VARC = Valve Academic Research Consortium.

(41.2%) Edwards Sapien XT through transfemoral approach, and 2 (11.7%) Edwards Sapien through transapical approach (Table 2). Amplatz 0.35-inch Extrastiff guidewire (Cook Medical, Inc., Bloomington, Indiana) was used for delivery of all Edwards bioprosthetic valves. Corevalve (Medtronic) was implanted in 8 patients (47.1%)—1 (5.8%) through transaxillary approach, and the rest through transfemoral approach. Amplatz 0.35-inch Superstiff guidewire (Boston Scientific, Natick, Massachusetts) was used to deliver all Corevalves.

Pre-dilation was performed in 10 patients (58.8%) with a mean balloon size of 22.5 ± 1.1 mm, whereas post-dilation was done in 5 patients (29.4%) with a mean balloon size of 25.33 ± 4.04 mm.

Types, presentation, and outcome. The causes of tamponade were classified according to the site of origin.

ANNULAR TEAR. Of the 17 patients with tamponade, 3 (17.6%) had annular tears (Fig. 2). In the first case, pre-dilation was performed with a 23-mm balloon, followed by implantation of a 26-mm Edwards valve. Angiographic control revealed annular tear in relation to the left coronary cusp, and echocardiography showed signs of cardiac tamponade. Pericardiocentesis was done after, in which a drainage tube was inserted with auto-transfusion in the femoral vein. A trial to seal that annular tear with another valve was done but was unsuccessful. The patient sustained cardiac arrest and could not be resuscitated.

In the second case, the annular rupture occurred after post-dilation of a 29-mm Core valve with a 26-mm balloon. Percutaneous pericardiocentesis was performed, which stabilized the condition of the patient. However, the patient sustained cardiac arrest 7 days later and could not be

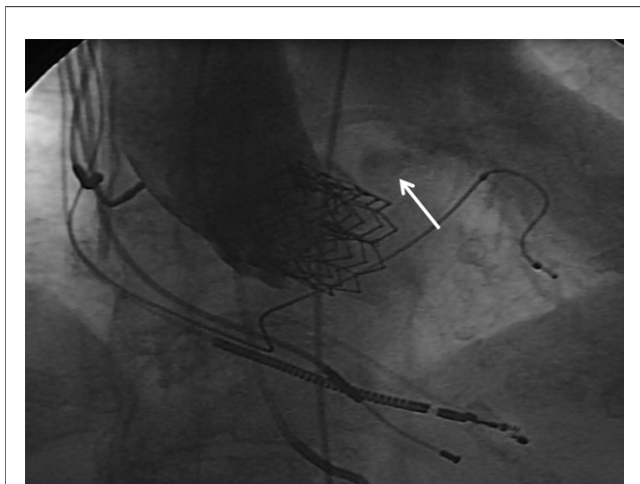


Figure 2. Aortography Demonstrating Annular Tear

Extravasation of contrast around the aortic annulus during aortography, indicative of annular tear (arrow).

resuscitated. The cause of death was unclear, but electrolyte disturbance was suspected.

In the third case, annular rupture was encountered after deployment of a 26-mm Edwards Sapien XT valve, which was immediately recognized, and prompt intervention (in the form of percutaneous pericardiocentesis) successfully stabilized the patient.

LV TEARS. Four (23.5%) patients had LV tear during TAVI (Fig. 3). In 2 patients, the LV tear was diagnosed on ventriculography, which exhibited extravasation of contrast into the pericardium. In the other 2 patients, the diagnosis was confirmed on surgical exploration, performed for ongoing bleeding despite pericardiocentesis.

The first patient developed tamponade after implantation of a 29-mm Corevalve, and the LV tear was caused by the Amplatz Superstiff wire. Pericardiocentesis was not sufficient, and hence surgical closure was necessary.

The second patient developed tamponade after implantation of a 23-mm Edwards valve. The tear was probably caused by the nose cone of the delivery system, which was advanced too distally with poor wire support. Pericardiocentesis was not sufficient, as with the first patient, and hence surgical repair was necessary.

The third patient had an LV tear caused by the Amplatz Extrastiff wire. The perforation occurred after implantation of a 23-mm Edwards valve. Surgical exploration revealed a rather large tear in the LV free wall, requiring surgical stitch closure. The patient had an unstable post-operative course: developed sepsis and multi-organ failure and died 10 days later. The fourth patient developed cardiac tamponade during wire exchange from the floppy to Amplatz Super stiff wire. The perforation was successfully repaired surgically.

RIGHT VENTRICULAR PERFORATIONS. Nine patients (52.9%) had tamponade due to right ventricular (RV) perforation, and all were caused by temporary pacemaker wires: 4 (23.5%) with a screw-in-lead, 3 (17.6%) with passive leads, and 2 (11.7%) with epicardial leads.

Tamponade after suspected aortic dissection. One patient had tamponade 24 h after the procedure. A 26-mm Edwards valve was implanted, after pre-dilation with a 23-mm balloon, in a patient with a bicuspid aortic valve. There were no immediate post-procedural complications; however, the patient developed hemodynamic instability on the following day. Bedside echocardiography revealed massive pericardial effusion, and aortic dissection was suspected. The patient sustained cardiac arrest before an effective treatment was established, and prolonged resuscitation was unsuccessful.

Timing. Most patients (n = 14, 82.3%) developed cardiac tamponade during the procedure: 3 (17.6%) due to annular tear, 4 (23.5%) due to LV tear, and 7 (41.2%) due to RV perforations. Two patients had tamponade within 24 h. Both were due to RV perforations and promptly responded to pericardiocentesis. One patient developed tamponade 1 day after the procedure, probably due to aortic dissection; the details have been presented in the previous section (Table 3).

Post-dilation. Post-dilation was done in 5 (29.4%) of 17 patients with tamponade, 2 of which resulted in annular rupture/aortic dissection, and 3 had RV perforations, which were probably not related to post-dilation (Online Table 2).

Mortality. Four patients died after tamponade (23.5%); causes were LV tear, annular tear, and suspected aortic dissection. All mortality occurred in patients who developed bleeding from the arterial side of the circulation, and none occurred from RV perforations.

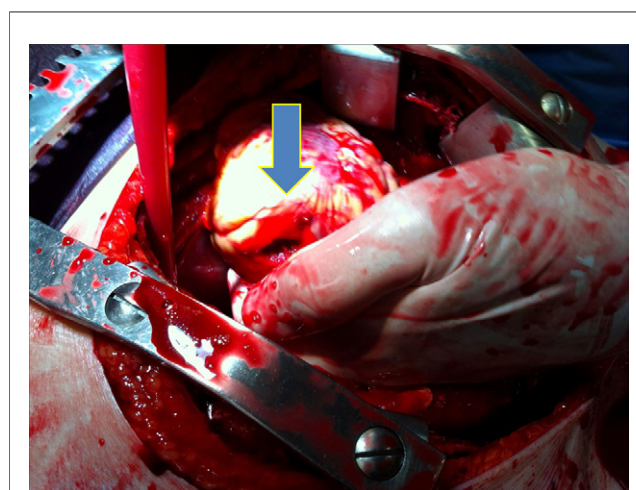


Figure 3. Left Ventricular Free-Wall Tear

Left ventricular free-wall tear noted during surgical exploration after cardiac tamponade during transcatheter aortic valve implantation.

Table 3. Cardiac Tamponade, Timing, Presentation, and Follow-Up

	Total Patients (n = 17)
Timing of presentation	
During/immediately at end of procedure	14 (82.4)
20 min after procedure	1 (5.8)
1 h after procedure	1 (5.8)
1 day after procedure (query aortic dissection)	1 (5.8)
Type of rupture	
Annular rupture	3 (17.6)
Probable aortic dissection	1 (5.8)
LV free wall rupture	4 (17.6)
RV perforation	9 (52.9)
Percutaneous pericardiocentesis only	
	12 (70.6)
Pericardiocentesis + surgical intervention	
LV tears	3 (17.6)
RV tears (mini-sternotomy)	1 (5.8)
Average amount of effusion drained, ml	362.5 ± 179.7
Mortality	
	4 (23.5)
Time of mortality	
1 day post-procedural	2 (11.7)
7 days post-procedural	1 (5.8)
10 days post-procedural	1 (5.8)
Values are n (%), unless otherwise indicated. LV = left ventricular; RV = right ventricular.	

MSCT analysis. Retrospective analysis of the computed tomography scans of patients with annular rupture revealed presence of both commissural (of at least 2 commissures) and annular calcification (Online Table 2). Interestingly, patients who had annular rupture had both commissural (of at least 2 commissures) and annular calcification.

Discussion

The main findings of this study are: 1) the incidence of cardiac tamponade was 4.3%; 2) the most common cause was RV perforation due to pacemaker leads; 3) other causes of tamponade were perforations in the arterial side almost equally divided between annular/supra-annular ruptures and LV perforations; 4) all perforations occurring in the RV could be treated with pericardiocentesis, whereas open surgery was needed in the arterial perforations; and 5) no death occurred after RV perforation, whereas the mortality after arterial perforation was 50%.

The 4.3% incidence of cardiac tamponade reported in our experience is not higher than in other cardiac interventions, such as pacemaker insertion, atrial fibrillation ablation, and so forth (12). However, mortality after tamponade was high with a rate of 23.5% in our cohort, which reflects the vulnerability of the patient cohort, because they are usually elderly and frail with multiple comorbidities. In particular, the severe consequences of arterial perforation with an almost universal need for surgical intervention and a mor-

tality of 50% impose the need to eliminate this kind of complication.

There are many predisposing factors for the development of tamponade during TAVI: calcified aortic annulus, and fragile and scarred myocardium due to likely co-existence of ischemic heart disease with previous infarcts. It is also related to the devices, technique, and experience of the operator. However, some of these factors are avoidable. Technically, there are 5 steps in the procedure.

Step “1,” introduction of the temporary pacemaker wire into the RV. A significant proportion of our cardiac tamponade occurred due to temporary pacemaker, especially with the screw-in lead. In our study, 9 patients (52.9%) had RV perforations, of which 4 had screw-in leads. Mahapatra et al. (13) demonstrated that use of screw-in leads was an independent risk factor for having RV perforation. However, the rationale behind using screw-in lead pacemaker was to have a stable position and threshold during the time of balloon inflation and valve deployment. In addition, even with passive wire we were probably more aggressive in positioning to achieve best threshold. It is also well-documented that elderly patients are more prone for complications such as RV rupture after temporary pacemaker wire (TPW) insertion than younger patients due to thinned RV. After significant cases of tamponade from TPW, we introduced changes to our practice. Only passive TPW were used, and we were less aggressive in manipulating the wire, which resulted in significant reduction in occurrence of tamponade in the recent cohort (2 patients between 2011 and 2012 vs. 7 patients between 2007 and 2010). Hence, this could be attributable to our learning curve. Passive TPW should be first choice, and screw-in lead should only be considered if there are issues with stable positioning of the lead, threshold, and sensitivity.

Step “2,” introduction of the stiff wire in the LV cavity; and step “3,” introduction of the delivery system of the valve along the stiff wire. Amplatz stiff wire provides excellent support for delivery of the valve and is a vital component of the procedure (Fig. 4). However, due to the stiffness of the wire, it has the potential of causing significant trauma to the LV myocardium. Even if the operators perform an appropriate curve on the distal stiff end of the wire, the curve itself can cause a linear perforation of the LV, when the wire supported by the delivery system advances too distally in a small LV cavity. In our study, 4 patients (23.5%) had LV tears, mostly attributed to stiffness of the wire curvature. It is not uncommon that the operators focus exclusively on the area of valve deployment, especially during positioning and deployment phase. This might briefly divert the attention from the stiff wire in the LV that could migrate and cause catastrophe. Hence great caution is necessary during introduction of Amplatz stiff wire in the LV.

In an aim to provide a cushioning effect to the stiff wire in the LV, we have adopted a new trick: “cushioned stiff

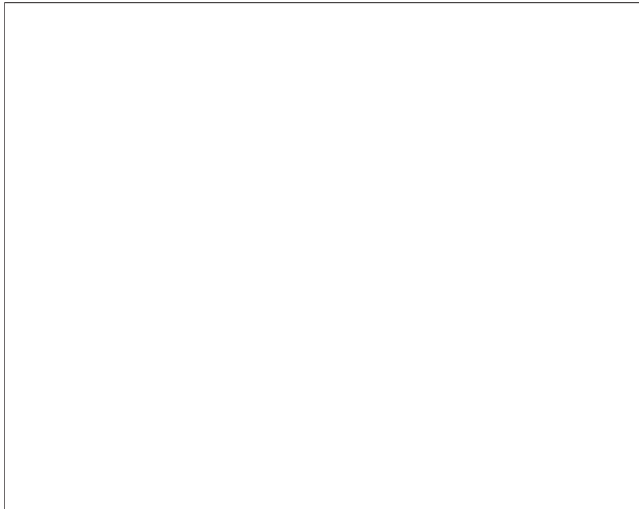


Figure 4. Positioning of Valve

Fluoroscopic image demonstrating nose cone of the Edwards' delivery system and narrow-curved Amplatz stiff wire (potential sources for left ventricular [LV] tear).

wire” technique. The idea is based on the fact that the curve of the Amplatz stiff wire acts as a sharp blade, and accordingly, we intended to cover the distal part of wire with a pig-tail catheter to make it less aggressive, especially when it comes in contact with the wall of LV. Details of how to

make this wire are given (Fig. 5): 1) cut the distal segment of a 6-F pigtail catheter (approximately 3 cm); 2) insert and position the cut segment of the pigtail catheter on the distal part of the Amplatz stiff wire just overlapping onto the soft portion of the wire; and 3) the pigtail catheter is then splinted by 2.0 silk sutures, which are tied on the wire, proximal and distal to the pigtail.

The end result is a smooth and soft curved wire, which is relatively less traumatic to the LV. In addition, the proximal knot on the wire also acts as a barrier against the forward advancement of the delivery system, preventing nose cone injury. The wire is then delivered into the LV with a 90-cm 6-F sheath. This new technique has now been regularly used at our center with over 50 cases to date, and we have not had any cases of tamponade from LV tear. This unsophisticated technique is an interim measure while we wait for the availability of a dedicated modified wire.

Step “4,” deployment of the valve. This specifically applies to the balloon expandable-valve and to post-dilation of any valve. The retrospective analysis of the computed tomography scan in patients with annular tear demonstrated significant annular and commissural calcifications. Although we could not correlate calcification with annular tear, due to small number of patients, nevertheless the degree of annular and commissural calcification might be the predictor for

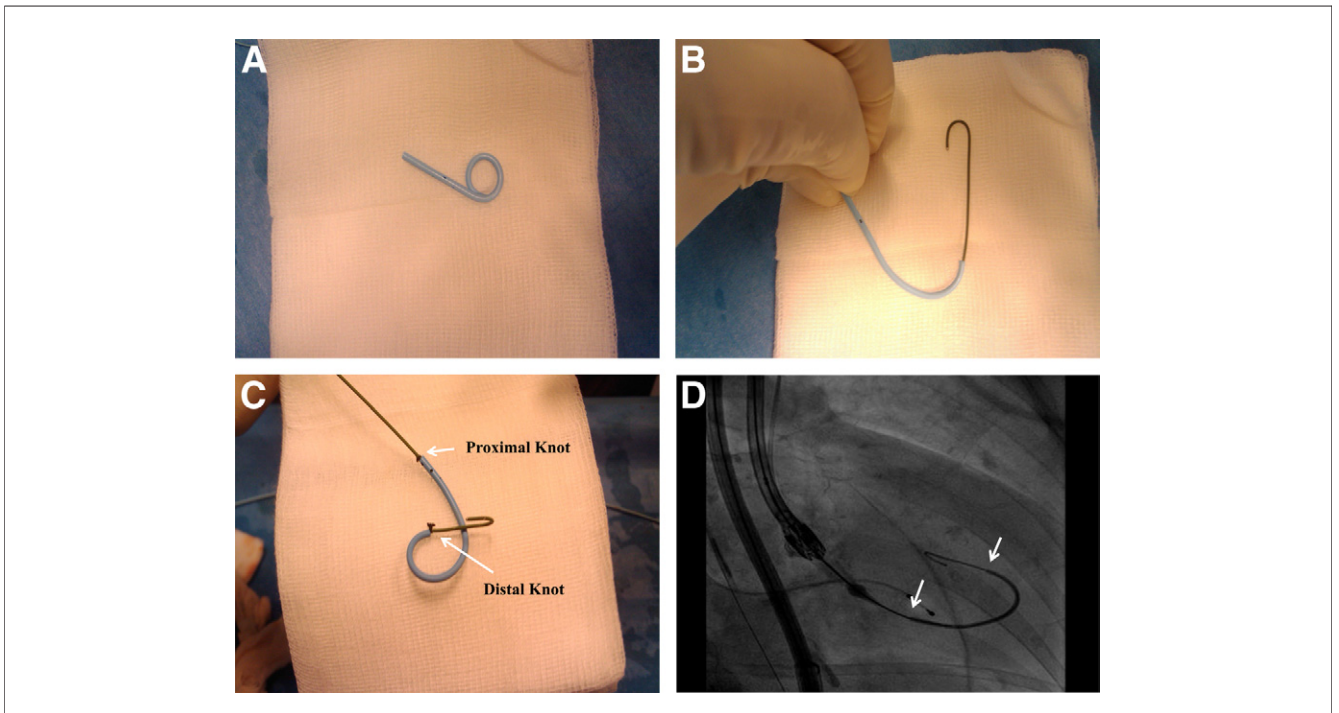


Figure 5. Steps Involved in Making “Cushioned Stiff Wire”

(A) Distal edge of 6-F pigtail catheter. (B) Stiff wire inserted through distal pigtail catheter. (C) Distal and proximal knots with silk sutures to secure the pigtail catheter. (D) Fluoroscopic image showing “Cushioned Stiff Wire” inside the left ventricular cavity.

Table 4. Summary Table for All Cases

Patient No.	Log. EuroSCORE %	Valve Type	Valve Size (mm)	Stiff Wire	TPW	Post-Dilation	Post-Dilation Balloon Size (mm)	Type of Tear	Time of Presentation	Management	Mortality
1	7.94	Corevalve	26	Amplatz Superstiff	Screw-in lead	Yes	23	RV	On table	Percutaneous pericardiocentesis	No
2	22.52	Corevalve	26	Amplatz Superstiff	Screw-in lead	Yes	23	RV	On table	Percutaneous pericardiocentesis	No
3	16.81	Corevalve	26	Amplatz Superstiff	Screw-in lead	No		RV	1 h later	Percutaneous pericardiocentesis	No
4	10.76	Corevalve	29	Amplatz Superstiff	Passive lead	No		RV	20 min later	Percutaneous pericardiocentesis+ Mini-sternotomy	No
5	3.26	Corevalve	29	Amplatz Superstiff	Passive lead	No		LV	On table	Percutaneous pericardiocentesis+ surgical closure	No
6	21.20	Corevalve	29	Amplatz Superstiff	Passive lead	No		RV	On table	Percutaneous pericardiocentesis	No
7	36.33	Corevalve	29	Amplatz Superstiff	Passive lead	Yes	26	Annular tear	On table	Percutaneous pericardiocentesis	Yes
8	7.03	Corevalve	31	Amplatz Superstiff	Passive lead	No		LV	On table	Percutaneous pericardiocentesis+ surgical closure	No
9	8.97	Edwards	23	Amplatz Extrastiff	Passive lead	No		LV	On table	Percutaneous pericardiocentesis+ surgical closure	No
10	80.36	Edwards	23	Amplatz Extrastiff	Passive lead	No		RV	On table	Percutaneous pericardiocentesis	No
11	24.18	Edwards	23	Amplatz Extrastiff	Passive lead	No		LV	On table	Percutaneous pericardiocentesis+ surgical closure	Yes
12	25.13	Edwards	26	Amplatz Extrastiff	Passive lead	Yes	26	Annular tear	On table	Percutaneous pericardiocentesis	Yes
13	8.97	Edwards	26	Amplatz Extrastiff	Epicardial lead (Transapical)	No		RV	On table	Percutaneous pericardiocentesis	No
14	14.15	Edwards	26	Amplatz Extrastiff	Passive lead	No		Annular tear	On table	Percutaneous pericardiocentesis	No
15	9.76	Edwards	26	Amplatz Extrastiff	Screw-in lead	No		RV	On table	Percutaneous pericardiocentesis	No
16	13.8	Edwards	26	Amplatz Extrastiff	Passive lead	No		Probable aortic dissection	1 day later	Percutaneous pericardiocentesis	Yes
17	11.29	Edwards	26	Amplatz Extrastiff	Passive lead	No		RV	On table	Percutaneous pericardiocentesis	No

EuroScore = European System for Cardiac Operative Risk Evaluation; LV = left ventricular; RV = right ventricular; TPW = temporary pacemaker wire.

annular tear. Caution should be taken in choosing the valve size especially in patients with heavy annular calcification; it might be that choosing a smaller valve in such patients would be safer. In addition the angle between aortic root axis and LV axis should be considered. Increase in the angulation tends to direct the delivery system on to the wall of the LV rather than into the cavity, which can force the stiff wire or the nose-cone to dissect through the LV free wall.

Step "5," post-dilation after valve deployment. Two-thirds of our patients who had annular rupture underwent post-dilation with relatively larger balloons (Online Table 2). It might be safer to initiate with a smaller balloon and proceed further after thorough evaluation at every step.

To summarize, there are 2 main determinants of cardiac tamponade in patients undergoing TAVI: 1) equipment and materials used, and 2) patient risk factors. With regard to the equipment, it seems that there is room to improve the ancillary equipment. We can minimize trauma by selecting less aggressive devices, such as passive temporary pacemaker wires over screw-in leads, and make stiff wire more user-friendly. With regard to patient risk factors (most of which are unavoidable), annular size, annular calcification, commissural calcification, LV size, LV hypertrophy, and myocardial ischemia/scar must all be thoroughly evaluated before the procedure. Finally, it is crucial to emphasize the experience of the operator, and learning curve is extremely important to reduce the complication rate.

Study limitations. This study is a single-center retrospective analysis. Although we have speculated possible predictors for tamponade, we could not analyze or correlate the predictors, due to the small number of patients with tamponade. Nevertheless, this study might lead future projects of combining data from various centers (Table 4).

Conclusions

Cardiac tamponade, although not commonly encountered, remains 1 of the serious complications of TAVI with a relatively high mortality. The most common cause of tamponade is from RV rupture related to transient pacemaker wire followed by LV and annular ruptures, which are attributable to Amplatz stiff wire and delivery device. Meticulous selection and handling of currently available devices should minimize occurrence of cardiac tamponade.

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REFERENCES

1. Ross J Jr., Braunwald E. Aortic stenosis. *Circulation* 1968;38:61-7.
2. Bonow RO, Carabello BA, Chatterjee K, et al. 2008 focused update incorporated into the ACC/AHA 2006 guidelines for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to revise the 1998 guidelines for the management of patients with valvular heart disease). Endorsed by the Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons. *J Am Coll Cardiol* 2008;52:e1-142.
3. Spodick DH. Pericardial diseases. In: Braunwald E, Zipes DP, Libby P, editors. *Heart Disease: A Textbook of Cardiovascular Medicine*. 6th edition, Vol. 2. Philadelphia: W. B. Saunders, 2001:1823-76.
4. Shabetai R. Diseases of the pericardium. In: Schlant RC, Alexander RW, editors. *Hurst's the Heart: Arteries and Veins*. 6th edition, Vol. 1. New York: McGraw-Hill, 1994:1647-1674.
5. Roques F, Michel P, Goldstone AR, Nashef SAM. The logistic EuroSCORE. *Eur Heart J* 2003;24:881-2.
6. Edwards FH, Grover FL, Shroyer AL, Schwartz M, Bero J. The Society of Thoracic Surgeons national cardiac surgery Database: Current risk assessment. *Ann Thorac Surg* 1997;63:903-8.
7. Chiam PT, Ruiz CE. Percutaneous transcatheter aortic valve implantation: Evolution of the technology. *Am Heart J* 2009;157:229-42.
8. Reynolds MR, Magnuson EA, Wang K, et al. Cost effectiveness of transcatheter aortic valve replacement compared with standard care among inoperable patients with severe aortic stenosis: Results from PARTNER trial (Cohort B). *Circulation* 2012;125:1102-9.
9. Grube E, Laborde JC, Gerckens U, et al. Percutaneous implantation of the CoreValve self-expanding valve prosthesis in high-risk patients with aortic valve disease: The Siegburg first-in-man study. *Circulation* 2006;114:1616-24.
10. Webb JG, Chandavimol M, Thompson CR, et al. Percutaneous aortic valve implantation retrograde from the femoral artery. *Circulation* 2006;113:842-50.
11. Ye J, Cheung A, Lichtenstein SV, et al. Transapical aortic valve implantation in humans. *J Thorac Cardiovasc Surg* 2006;131:1194-6.
12. David R, Holmes JR, Nishimura R, Fountain R, Turi ZG. Iatrogenic pericardial effusion and tamponade in the percutaneous intracardiac intervention era. *J Am Coll Cardiol Intv* 2009;2:705-17.
13. Mahapatra S, Bybee KA, Bunch TJ, et al. Incidence and predictors of cardiac perforation after permanent pacemaker placement. *Heart Rhythm* 2005;2:907-11.

Key Words: tamponade in TAVI ■ TAVI
■ TAVI complications.

APPENDIX

For supplementary tables, please see the online version of this article.