JACC: CARDIOVASCULAR INTERVENTIONS
VOL. 6, NO. 5, 2013

ISSN 1936-8798/$36.00
PUBLISHED BY ELSEVIER INC.
http://dx.doi.org/10.1016/j.jcin.2012.12.124

Post-Procedural Hypertension Following Transcatheter Aortic Valve Implantation

Incidence and Clinical Significance

Gidon Y. Perlman, MD, Sasa Loncar, MD, Arthur Pollak, MD, Dan Gilon, MD, Ronny Alcalai, MD, David Planer, MD, Chaim Lotan, MD, Haim D. Danenberg, MD

Jerusalem, Israel

Objectives This study sought to investigate the blood pressure (BP) response after transcatheter aortic valve implantation (TAVI) and its correlation with short- and mid-term clinical outcomes.

Background TAVI is an emerging therapy for aortic stenosis patients at high surgical risk. The acute hemodynamic sequelae of this procedure and their clinical relevance are yet unclear.

Methods Consecutive patients who underwent TAVI in a single center were prospectively monitored for BP response during 5 post-procedural days. Clinical parameters, adverse events, and medical treatment were recorded during hospitalization, at 30 days, and at 12 months after the procedure. Patients were divided according to their post-procedural BP response into 2 groups: increased BP and stable BP.

Results One hundred and five patients were analyzed. Overall, systolic BP increased immediately after TAVI in the entire cohort by an average of 15 $\pm$ 31 mm Hg. This rise was sustained and led to intensification of antihypertensive treatment in 53 patients (51%); these patients were designated as the increased BP group. The increase in systolic BP after TAVI was associated with an increase in stroke volume and cardiac output and was not related to age, baseline cardiac function, or procedural outcomes. Patients with increased BP after TAVI had a significantly better prognosis with fewer adverse events in the hospital (21% vs. 62%, $p < 0.01$), after 30 days (30% vs. 71%, $p < 0.01$), and after 12 months (53% vs. 83%, $p < 0.01$) as compared with patients with stable BP.

Conclusions After TAVI, a substantial number of patients have a significant rise in systolic BP necessitating long-term treatment. This increase in BP is associated with an increase in cardiac output and predicts a better clinical outcome. (J Am Coll Cardiol Intv 2013;6:472–8) © 2013 by the American College of Cardiology Foundation

From the Department of Cardiology, Hadassah-Hebrew University Medical Center, Jerusalem, Israel. Dr. Danenberg is a clinical proctor for Medtronic CoreValve. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

Manuscript received October 11, 2012; revised manuscript received December 14, 2012, accepted December 21, 2012.

Severe aortic stenosis (AS) often presents with normal arterial systolic blood pressure (BP) in lieu of elevated intraventricular systolic pressure and, consequently, a pressure gradient across the aortic valve. Pre-procedural hypertension (HTN) is a risk factor and a predictor of increased mortality after aortic valve replacement (AVR) (1). Remodeling and reduction of left ventricular (LV) hypertrophy after AVR appear to correlate with a favorable clinical outcome (2). The disappearance of the pressure gradient across the aortic valve following surgical AVR often leads to dramatic hemodynamic changes and increased hypertension (3,4) that can challenge the post-procedural care and may further affect these patients in the chronic phase. The use of antihypertensive medications after surgery has been shown to improve LV remodeling in these patients (5,6).

Transcatheter aortic valve implantation (TAVI) is a novel approach for the treatment of severe AS patients who are inoperable or at high surgical risk. Unlike surgical AVR, TAVI is often performed under local anesthesia (7), and the post-procedural care takes place mainly in cardiac intensive care units. Studies that assessed the post-procedural hemodynamic sequelae of TAVI have focused on complications and hypotension rather than on the hypertensive response (7,8). Gotzmann et al. (9) described a rise in systolic BP immediately after TAVI, not related to a change in ejection fraction. Nevertheless, the causes, persistence, and clinical significance of this response are yet unclear. In this study, we prospectively investigated the in-hospital hemodynamic changes post-TAVI and their clinical correlations during 12 months of follow-up.

Methods

Study population and post-procedural monitoring. Consecutive patients who underwent TAVI at our institution were prospectively analyzed. All patients had severe symptomatic AS diagnosed clinically and confirmed by Doppler echocardiography. The study subjects were declined for surgery and referred to TAVI due to an increased surgical risk as assessed by an institutional heart team.

After TAVI, patients were transferred to the cardiac intensive care unit with continuous BP monitoring immediately after the procedure and during the following 5 days. The highest BP measurement was recorded every 8 h and used for analysis. Patients were initially kept on their pre-procedural antihypertensive therapy. Uncontrolled BP was promptly treated; treatment decisions were not restricted and were decided by the treating physicians. Baseline BP levels were measured in the pre-admission office visit.

All patients had echocardiograms performed after TAVI. Post-procedural aortic regurgitation was graded on a scale of 1 to 4 according to vena contracta width, jet diameter to left ventricular outflow tract (LVOT) diameter ratio and circumferential extent in the short-axis view. Valve areas, pressure gradients, stroke volume, and cardiac output were calculated using LVOT Doppler waveforms, LVOT diameter, heart rate, and the continuity equation as per standard techniques (10–12). Arterial compliance was calculated as stroke volume/pulse pressure (ml/mm Hg) (13). Procedural complications were recorded according to the Valve Academic Research Consortium definitions (14).

BP response after TAVI. Patients were divided into 2 groups according to the presence or absence of an increase in BP after TAVI.

Increased BP after TAVI was defined in the presence of one of the following:

1. A sustained (>48 h) systolic pressure >140 mm Hg or diastolic pressure >90 mm Hg that was not present at baseline, before TAVI.
2. A need to increase more than 2-fold the dosage of an antihypertensive drug to achieve control of systemic BP.
3. A need to add an additional antihypertensive medicine to the pre-procedural regimen in order to achieve control of systemic BP.

Endpoints. Clinical outcomes were recorded during hospitalization, at 30 days, and at 12 months after the procedure. Serious adverse events recorded during follow-up included: worsening heart failure; myocardial infarction; stroke; and recurrent hospitalization due to any cause and death.

Abbreviations and Acronyms

AS = aortic stenosis
AVR = aortic valve replacement
BP = blood pressure
HTN = hypertension
LV = left ventricle
LVOT = left ventricular outflow tract
TAVI = transcatheter aortic valve implantation

Statistical analysis. Comparison of quantitative variables was performed using an unpaired Student *t* test whereas Fisher exact test was used to compare qualitative variables. Survival analysis was obtained by Kaplan-Meier estimates. Differences among the groups were compared with the log-rank test. All data were initially entered into a commercially available statistical program SPSS (version 17.0 for Windows, SPSS Inc., Chicago, Illinois).

Results

Study population. One hundred and five consecutive patients were included in the study. Mean age was 80.7 ± 6.6 years and logistic EuroSCORE (European System for Cardiac Operative Risk Evaluation) was 23.3 ± 15.1. The baseline characteristics of the patients according to post-TAVI BP response are presented in Table 1. The 2 groups were similar in age, prevalence of HTN, logistic EuroSCORE, systolic and diastolic BP, ejection fraction, cardiac output, cardiac index, and aortic valve parameters. However,

patients with increased BP were thinner (body mass index: 26.5 vs. 28.6 kg/m^2, $p < 0.05$) and were using significantly less antihypertensive medications to achieve a similarly well-controlled BP at baseline before TAVI.

The 2 groups had similar systemic vascular resistance and arterial compliance at baseline. In accordance with the elderly age of our cohort and the high prevalence of HTN, arterial compliance was low in both groups (1.11 ± 0.47 ml/mm Hg vs. 1.26 ± 0.45 ml/mm Hg, $p = 0.11$).

There was no difference in the use of general anesthesia between the groups, nor the use of different approaches for valve delivery (transfemoral, trans-subclavian, and direct aortic). There was no difference between patients implanted with Medtronic CoreValve (Minneapolis, Minnesota) or Edwards Sapien XT valves (Edwards Lifesciences, Irvine, California) (Table 2).

Hemodynamic response. Eighty-five patients (81%) had a prior diagnosis of HTN, and 16 additional patients (15%) were treated with antihypertensive medications for other diagnoses (e.g., heart failure and ischemic heart disease).

Systolic BP rose immediately after TAVI in the entire cohort by an average of 15 mm Hg (±31) and remained 8 mm Hg above baseline 5 days after the procedure despite medical treatment ($p < 0.01$ for both changes) (Fig. 1). Diastolic BP following TAVI was significantly reduced immediately after TAVI, and the diastolic BP was 7 mm Hg lower than baseline after 5 days ($p < 0.01$ for both changes).

Fifty-three patients (51%) had increased BP after TAVI and constituted the increased BP group. They all required intensification of antihypertensive therapy, including 27 patients in which parenteral BP lowering drugs were administered. In fact, 9 patients in this group (17%) had not been previously diagnosed as hypertensive. In the increased BP group, the number of antihypertensive drugs rose significantly by an average of 0.83 ± 1.03, whereas in the group of patients with stable BP after TAVI, the number of drugs decreased on average by 1.02 ± 1.30 ($p < 0.01$ for both changes).

Cardiac output, cardiac index, and stroke volume after TAVI were significantly increased in patients with increased BP versus patients with stable BP ($p < 0.05$). Patients with stable BP had, on average, a small reduction in stroke volumes and cardiac outputs (Table 3).

Elevated BP after TAVI was not associated with baseline arterial compliance, which was similar in both

Table 1. Baseline Characteristics According to BP Response After TAVI

Variable	Increased BP (n = 53)	Stable BP (n = 52)	p Value
Age, yrs	81 ± 6	80 ± 7	0.46
Male	25 (47)	25 (48)	1.0
BMI, kg/m^2	26.5 ± 4.2	28.8 ± 5.9	0.04
Logistic EuroSCORE	23.5 ± 15.3	23.2 ± 14.9	0.91
Ejection fraction, %	54.8 ± 11.7	56.8 ± 12.0	0.39
HTN	44 (83)	41 (79)	0.63
Number of anti-HTN drugs	2.47 ± 1.23	3.21 ± 1.67	0.01
Coronary artery disease	30 (57)	28 (54)	0.84
Previous CABG	15 (28)	11 (21)	0.49
NYHA score	3.15 ± 0.36	3.17 ± 0.51	0.81
Chronic renal failure	6 (11)	7 (13)	0.77
Diabetes	17 (32)	20 (38)	0.54

Values are mean ± SD or n (%).
BMI = body mass index; BP = blood pressure; CABG = coronary artery bypass graft; EuroSCORE = European System for Cardiac Operative Risk Evaluation; HTN = hypertension; NYHA = New York Heart Association; TAVI = transcatheter aortic valve implantation.

Table 2. Procedure Technique and Related Complications in Both Groups

Variable	Increased BP (n = 53)	Stable BP (n = 52)	p Value
Approach			
Transfemoral	46 (87)	43 (82)	0.59
Trans-subclavian	6 (11)	5 (10)	1.0
Direct aortic	1 (2)	4 (8)	0.20
Medtronic CoreValve/Edwards Sapien XT	46/7	43/9	0.59
General anesthesia	34 (64)	29 (56)	0.43
Periprocedural death	0 (0)	1 (2)	0.49
Tamponade	2 (4)	3 (6)	0.67
Major bleeding*	29 (55)	22 (42)	0.24
Major vascular complications*	5 (9)	3 (6)	0.71
Acute kidney injury, any grade*	8 (15)	9 (17)	0.79
Severe infection	6 (11)	5 (10)	1.0
Pacemaker implantation†	22 (41)	13 (25)	0.09

Values are n or n (%). *By VARC definitions. †Pacemaker implanted due to advanced conduction blocks.
BP = blood pressure; VARC = Valve Academic Research Consortium.

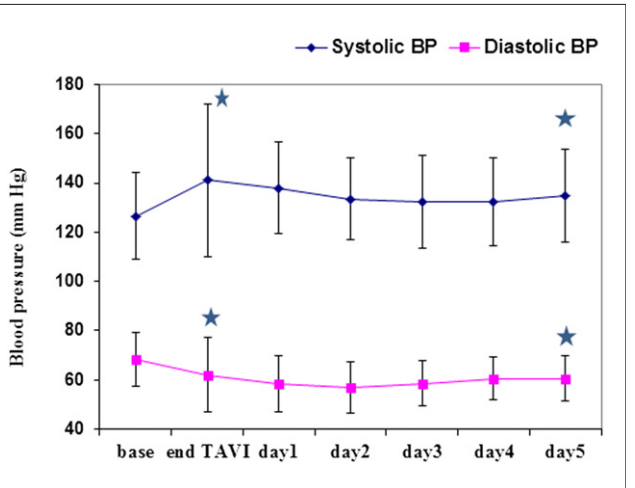

Figure 1. Average BP Response After TAVI in the Entire Cohort

Systolic blood pressure (BP) rose immediately after transcatheter aortic valve implantation (TAVI) and remained significantly higher than baseline BP despite medical treatment. Diastolic BP decreased significantly immediately after TAVI and remained lower at day 5 than at baseline. ★ = $p <$ 0.01 for difference with baseline.

groups (Table 3). Systemic vascular resistance was largely unchanged after valve implantation with no association to BP response (Table 3).

Clinical outcomes. There were 4 in-hospital deaths (3.8%), 5 deaths at 30 days (4.7%), and 13 deaths at 12 months (12.3%) (Fig. 2, Table 4). There was a trend toward more in-hospital deaths in patients with stable BP (0% vs. 8%, $p = 0.056$), but no significant differences in survival were seen after 12 months of follow-up.

The rates of in-hospital, 30 day, and 12-month serious adverse events were 21%, 30%, and 53%, respectively, for patients with increased BP as compared to 60%, 71%, and 83% for patients with stable BP ($p < 0.01$ for all differences between groups) (Fig. 3, Table 4). The majority of these events were episodes of worsening heart failure (Fig. 4), which were less frequent in the increased BP group than in the stable BP group (17% vs. 54% in-hospital, $p < 0.01$, and 23% vs. 60% at 12 months, $p < 0.01$). Adverse events possibly related to increased HTN such as stroke or transient ischemic attack, myocardial infarction, and acute kidney injury were rare and similar between the 2 groups. The adverse events reported in our cohort are detailed in Table 4 (and in Table 2 for acute kidney injury).

The rates of procedure-related complications with possible hemodynamic consequences including bleeding, vascular complications, pacemaker implantation, and post-procedural infections did not differ significantly between the 2 groups (Table 2).

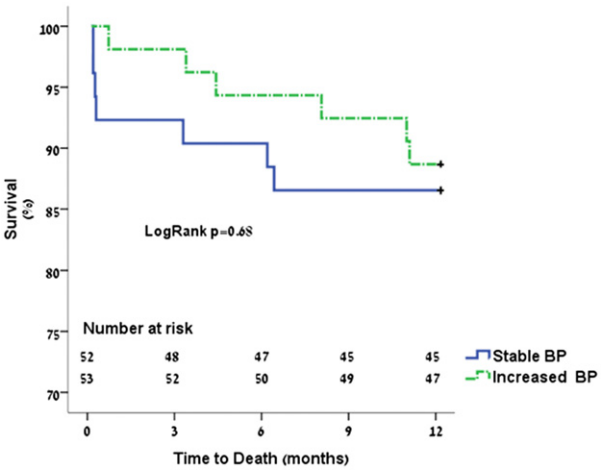

Figure 2. Kaplan-Meier 1-Year Survival According to BP Response

Cumulative survival after TAVI according to BP response. Abbreviations as in Figure 1.

Table 3. Hemodynamic and Echocardiographic Characteristics According to BP Response Before and After TAVI

Variable	Increased BP (n = 53)	Stable BP (n = 52)	p Value
Baseline data			
Systolic BP, mm Hg	128.6 ± 18.0	124.6 ± 17.1	0.25
Diastolic BP, mm Hg	68.0 ± 11.6	68.6 ± 10.0	0.78
Pulse pressure, mm Hg	60.6 ± 14.8	56.0 ± 17.2	0.15
Aortic valve area, cm^2	0.65 ± 0.18	0.66 ± 0.17	0.77
Maximal gradient, mm Hg	74.8 ± 19.8	69.2 ± 22.2	0.18
Mean gradient, mm Hg	46.2 ± 13.4	43.3 ± 14.7	0.29
Arterial compliance, ml/mm Hg	1.11 ± 0.47	1.26 ± 0.45	0.11
Systemic vascular resistance, dyne/cm	1,702 ± 606	1,562 ± 460	0.2
Cardiac output, l/min	4.3 ± 1.2	4.5 ± 1.4	0.43
Cardiac index, l/min	2.4 ± 0.7	2.5 ± 0.8	0.56
Stroke volume, ml	64 ± 19	68 ± 21	0.33
Post-procedural data			
Systolic BP immediately after TAVI, mm Hg	154.9 ± 29.9	126.1 ± 24.4	<0.01
Diastolic BP immediately after TAVI, mm Hg	64.3 ± 14.1	59.6 ± 16.3	0.17
Pulse pressure immediately after TAVI, mm Hg	90.6 ± 24.7	66.6 ± 19.9	<0.01
Cardiac output increase, l/min	0.26 ± 1.3	−0.44 ± 1.5	<0.05
Cardiac index increase, l/min/m^2	0.14 ± 0.7	−0.27 ± 0.9	<0.05
Stroke volume increase, ml	0.32 ± 22.1	−11.07 ± 23.31	<0.05
Systemic vascular resistance, dyne/cm	1,642 ± 524	1,581 ± 564	0.61
Aortic valve area, cm^2	1.72 ± 0.59	1.74 ± 0.51	0.85
Maximal gradient, mm Hg	15.4 ± 5.8	15.3 ± 7.5	0.93
Mean gradient, mm Hg	8.4 ± 3.6	8.2 ± 4.3	0.79

Values are mean ± SD.
Abbreviations as in Table 1.

Table 4. SAE in Both Groups

Adverse Event	Increased BP (n = 53)	Stable BP (n = 52)	p Value
In-hospital			
Death*	0 (0)	4 (8)	0.056
Myocardial infarction	0 (0)	0 (0)	1.0
Stroke or TIA	2 (4)	5 (10)	0.27
Worsening heart failure	9 (17)	27 (52)	<0.01
Any SAE	11 (21)	31 (60)	<0.01
30 days			
Death	1 (2)	4 (8)	0.20
Myocardial infarction	0 (0)	0 (0)	1.0
Stroke or TIA	2 (4)	5 (10)	0.27
Worsening heart failure	10 (19)	28 (54)	<0.01
Readmission for other causes	4 (8)	9 (17)	0.14
Any SAE	16 (30)	37 (71)	<0.01
12 months			
Death	6 (11)	7 (13)	0.74
Myocardial infarction	0 (0)	0 (0)	1.0
Stroke or TIA	3 (6)	5 (10)	0.48
Worsening heart failure	12 (23)	31 (60)	<0.01
Readmission for other causes	17 (32)	21 (40)	0.42
Any SAE	28 (53)	43 (83)	<0.01

Values are n (%). *In-hospital deaths were due to: annular rupture in 1 patient; stroke in 1 patient; and pneumonia in 2 patients.
BP = blood pressure; SAE = serious adverse event(s); TIA = transient ischemic attack.

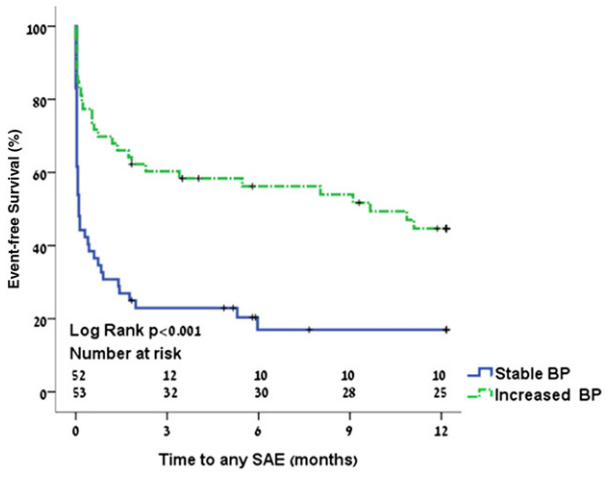

Figure 3. Kaplan-Meier Event-Free Survival According to BP Response

Total serious adverse events (SAE) after 12 months of follow-up according to the BP response after TAVI. Abbreviations as in Figure 1.

The severity of post-TAVI aortic regurgitation was likewise similar in both groups. We observed low rates of moderate or severe aortic regurgitation as shown in Table 5.

Discussion

Hypertension following TAVI is frequent. In the study cohort, 53 of 105 patients (51%) had increased BP that led to intensification of antihypertensive therapy. Post-TAVI hypertension was associated with improved LV function, and a favorable in-hospital clinical outcome as well as at 30 days and at 12 months of follow-up. Mortality at 30 days in the entire cohort was relatively low or similar to other reported series (15–17). TAVI is a novel therapeutic option for patients with severe and symptomatic AS that are declined for surgery due to high surgical risk. These patients are often old with a multitude of major comorbidities. In our study, the average logistic EuroSCORE was 23.3 ± 15.1% and the prevalence of HTN was very high, approaching 81%, which is similar to other reports (17,18).

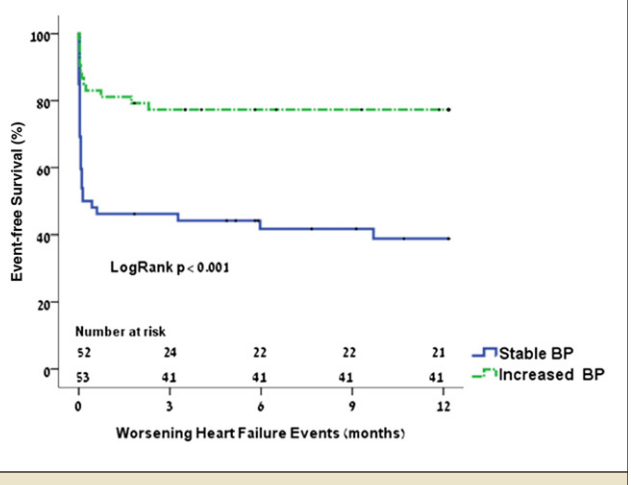

Figure 4. Kaplan-Meier Survival Free of Worsening Heart Failure According to BP Response

Total heart failure events after TAVI according to the BP response after TAVI. Abbreviations as in Figure 1.

Table 5. Post-Procedural Aortic Regurgitation in Both Groups

Aortic Regurgitation Grade	Increased BP (n = 53)	Stable BP (n = 52)	p Value
None or trace	8 (15)	6 (12)	0.77
Mild or mild-moderate	38 (72)	38 (73)	1.0
Moderate	7 (13)	7 (13)	1.0
Severe	0 (0)	1 (2)	0.49

Values are n (%).
BP = blood pressure.

Interestingly, the increase in BP after TAVI predicted a favorable short- and mid-term clinical outcome independent of known risk factors for surgical AVR or TAVI such as age, LV dysfunction, ischemic heart disease, previous HTN, and chronic renal failure, which were common in our patients (1,15,16).

Increased BP after TAVI correlated with an increase in cardiac output, cardiac index, and stroke volume. The relative improvement of cardiac function in patients with increased BP may be the underlying mechanism for the BP response as the rise in BP was not associated with better baseline cardiac function parameters or procedural complications. Patients with increased BP were taking fewer antihypertensive drugs at baseline, but the level of BP control was similar before TAVI to that observed in patients in which BP was not elevated (Table 1); thus, the difference in the number of antihypertensives is probably not responsible for the different post-procedural response.

The association between good prognosis and increased BP, a potentially harmful response, is unexpected and its underlying mechanism is yet to be defined. The rise in BP may represent myocardial contractile reserve even in patients with reduced LV function at baseline. This possibility is supported by the fact that patients with increased BP had a significantly greater increase in both stroke volume and cardiac output than did stable BP response patients. A Pearson test for bivariate correlation between increased BP and an increase in cardiac output showed a significant ($p = 0.015$), though moderate, correlation ($R^2 = 0.28$) with the combined outcome of death or worsening heart failure. The rise in BP may, in addition, represent a lack of adverse events that are associated with hypotension such as vascular injury and bleeding. However, there were no significant differences in the rates of procedural complications between the 2 groups in our study. Of note, 2 of our patients

developed increased BP only after pericardiocentesis for tamponade, exemplifying that the absence of increased BP after TAVI might be a sign of a severe complication.

HTN is generally considered a marker of poor prognosis. Chronic HTN and accelerated HTN are well-described contributors to cardiovascular morbidity and mortality, especially in elderly patients (19–22). Baseline HTN has been found to be associated with worse outcomes after TAVI (18). The better prognosis associated with an increased BP response after TAVI persisted over 12 months of follow-up. Longer follow-up of these patients is needed to ascertain if increased HTN might also have negative effects on the very elderly patients undergoing TAVI.

Arterial stiffness and the resulting loss of blood pressure autoregulation are prevalent in elderly patients (23) and even more common in patients with cardiovascular disease (24,25). Reduced arterial compliance was present in our cohort, but arterial stiffness and peripheral vascular resistance cannot explain the rise in BP observed after TAVI, as these were similar in the 2 groups. However, reduced arterial compliance may explain the sustained nature of the elevated BP response recorded after TAVI due to the patients' inability to perform adequate arterial vasodilation after the relief of the aortic valve gradient. Of note, both groups had well-controlled BP before TAVI. Simultaneously with the increase in BP after TAVI we observed a slight reduction in diastolic pressure. This might represent mild aortic regurgitation after valve implantation. Diastolic BP was further decreased at day 5, probably by antihypertensive treatment administered for systolic HTN.

TAVI is often performed under local anesthesia and conscious sedation that do not afford the tight hemodynamic control that can be achieved under general anesthesia (26,27). We observed a BP surge immediately after valve implantation that required prompt treatment. Treating physicians should be aware of and alert to this response that is more challenging when patients are only mildly sedated.

The rapid and sustained BP changes after TAVI reported in this study for the first time are common and should be identified and treated throughout the procedure and the post-procedural phase. This is exemplified by a patient who had an abrupt rise of systolic BP to 220 mm Hg immediately after valve deployment and consequently developed severe pulmonary edema that necessitated mechanical ventilation.

TAVI is a novel therapy for a challenging population of patients. Treatment of these complex patients requires a dedicated multidisciplinary team of interventional cardiologists, intensive care specialists, and anesthesiologists to monitor their responses and promptly treat adverse events. The hemodynamic responses described herein may also bring insight to the recuperative capabilities of the myocardium in patients with AS.

Study limitations. The data we used for this study were prospectively collected but the cohort size is not large and a relatively short follow-up was used. As a result, we might have inherent biases between the 2 groups. In addition, the method we used for calculating cardiac output is indirect and could possibly have led to overestimation of the differences we observed. Nevertheless, the hemodynamic changes we noted were very significant, and we believe that these changes and the associated clinical outcomes are valid.

Conclusions

After TAVI, about one-half of the patients have a significant rise in systolic BP, necessitating immediate and long-term treatment. To our knowledge, this is the first study showing that an increase in BP after TAVI predicts a better recovery of cardiac function and favorable short- and mid-term clinical outcome.

Reprint requests and correspondence: Dr. Haim D. Danenberg, Interventional Cardiology Unit, Hadassah Hebrew University Medical Center, Jerusalem 91120, Israel. E-mail: haimd@ekmd.huji.ac.il.

REFERENCES

1. Tjang YS, van Hees Y, Körfer R, Grobbee DE, van der Heijden GJ. Predictors of mortality after aortic valve replacement. Eur J Cardiothorac Surg 2007:32:469–74.
2. Ali A, Patel A, Ali Z, et al. Enhanced left ventricular mass regression after aortic valve replacement in patients with aortic stenosis is associated with improved long-term survival. J Thorac Cardiovasc Surg. 2011;142:285–91.
3. Estafanous GF, Tarazi RC, Buckley S, Taylor PC. Arterial hypertension in immediate postoperative period after valve replacement. Br Heart J 1978;40:718–24.
4. Layton C, Monro J, Brigden W, McDonald A, McDonald L, Weaver J. Systemic hypertension after homograft aortic valvar replacement: a cause of late homograft failure. Lancet 1973;2:1343–7.
5. Dahl JS, Videbaek L, Poulsen MK, et al. Effect of candesartan treatment on left ventricular remodeling after aortic valve replacement for aortic stenosis. Am J Cardiol 2010;106:713–9.
6. Imanaka K, Kohmoto O, Nishimura S, Yokote Y, Kyo S. Impact of postoperative blood pressure control on regression of left ventricular mass following valve replacement for aortic stenosis. Eur J Cardiothorac Surg 2005;27:994–9.
7. Covello RD, Ruggeri L, Landoni G, et al. Transcatheter implantation of an aortic valve: anesthesiological management. Minerva Anestesiol 2010;76:100–8.
8. Bagur R, Webb JG, Nietlispach F, et al. Acute kidney injury following transcatheter aortic valve implantation: predictive factors, prognostic value, and comparison with surgical aortic valve replacement. Eur Heart J 2010;31:865–74.
9. Gotzmann M, Lindstaedt M, Bojara W, Mügge A, Germing A. Hemodynamic results and changes in myocardial function after transcatheter aortic valve implantation. Am Heart J 2010;159:926–32.
10. Moss RR, Ivens E, Pasupati S, et al. Role of echocardiography in percutaneous aortic valve implantation. J Am Coll Cardiol Img 2008;1:15–24.
11. Zoghbi WA, Chambers JB, Dumesnil JG, et al. Recommendations for evaluation of prosthetic valves with echocardiography and Doppler ultrasound: a report from the American Society of Echocardiography's

Guidelines and Standards Committee and the Task Force on Prosthetic Valves. J Am Soc Echocardiogr 2009;22:975–1014.
12. Lancellotti P, Tribouilloy C, Hagendorff A, et al. European Association of Echocardiography recommendations for the assessment of valvular regurgitation. Part 1: aortic and pulmonary regurgitation (native valve disease). Eur J Echocardiogr 2010;11:223–44.
13. Chemla D, Hébert J, Coirault C, et al. Total arterial compliance estimated by stroke volume-to-aortic pulse pressure ratio in humans. Am J Physiol 1998;274:H500–5.
14. Leon B, Piazza N, Nikolsky E, et al. Standardized endpoint definitions for transcatheter aortic valve implantation clinical trials: a consensus report from the Valve Academic Research Consortium. Eur Heart J 2011;32:205–17.
15. Webb JG, Altwegg L, Boone RH, et al. Transcatheter aortic valve implantation: impact on clinical and valve-related outcomes. Circulation 2009;119:3009–16.
16. Thomas M, Schymik G, Walther T, et al. Thirty-day results of the SAPIEN Aortic Bioprosthesis European Outcome (SOURCE) registry: a European registry of transcatheter aortic valve implantation using the Edwards SAPIEN valve. Circulation 2010;122:62–9.
17. Lefèvre T, Kappetein AP, Wolner E, et al., for the PARTNER EU Investigator Group. One year follow-up of the multi-centre European PARTNER transcatheter heart valve study. Eur Heart J 2011;32:148–57.
18. Thomas M, Schymik G, Walther T, et al. One-year outcomes of cohort 1 in the Edwards SAPIEN Aortic Bioprosthesis European Outcome (SOURCE) registry: the European registry of transcatheter aortic valve implantation using the Edwards SAPIEN valve. Circulation 2011;124:425–33.
19. Chobanian AV, Bakris GL, Black HR, et al. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Hypertension 2003;42;1206–52.
20. Aronow WS, Fleg JL, Pepine CJ, et al. ACCF/AHA 2011 expert consensus document on hypertension in the elderly: a report of the American College of Cardiology Task Force on Clinical Expert Consensus documents. J Am Coll Cardiol 2011;57:2037–114.
21. Varon J, Marik PE. The diagnosis and management of hypertensive crises. Chest 2000;118:214–27.
22. Vaughan CJ, Delanty N. Hypertensive emergencies. Lancet 2000;356:411–7.
23. Vaitkevicius PV, Fleg JL, Engel JH, et al. Effects of age and aerobic capacity on arterial stiffness in healthy adults. Circulation 1993;88:1456–62.
24. Najjar SS, Scuteri A, Lakatta EG. Arterial aging: is it an immutable cardiovascular risk factor? Hypertension 2005;46:454–62.
25. Brunner EJ, Shipley MJ, Witte DR, et al. Arterial stiffness, physical function, and functional limitation: the Whitehall II study. Hypertension 2011;57:1003–9.
26. Guarracino F, Covello RD, Landoni G, et al. Anesthetic management of transcatheter aortic valve implantation with transaxillary approach. J Cardiothorac Vasc Anesth 2011;25:437–43.
27. Vavuranakis M, Voudris V, Vrachatis DA, et al. Transcatheter aortic valve implantation, patient selection process and procedure: two centres' experience of the intervention without general anaesthesia. Hellenic J Cardiol 2010;51:492–500.

Key Words: aortic valve stenosis ■ blood pressure ■ prognosis ■ transcatheter aortic valve implantation.