more potent and prolonged PWV increase (peak immediately after the end of smoking, by 0.36 m/s).

Compared with TC, EC5min smoking resulted in a less potent PWV increase throughout the study (F = 4.425, p = 0.005). On the other hand, EC30min resulted in a PWV increase similar to that of TC smoking throughout the study period (F = 0.268, p = 0.615). EC30min smoking resulted in a more potent effect on PWV compared with EC5min smoking (F = 3.167, p = 0.030).

To the best of our knowledge, this is the first study dealing with various patterns of EC smoking on aortic stiffness and BP demonstrating that it clearly exerts an unfavorable effect. EC over 30 min induces an unfavorable effect on aortic stiffness similar to TC smoking. The influence of EC smoking over 5 min on aortic stiffness is not as prompt (peak effect at 15 min) and is less potent compared with the effect of TC smoking.

Given the prognostic role of aortic stiffness and increased BP for future cardiovascular events and mortality, as well as the prolonged exposure to EC smoking throughout the day matched with the strong tendency of this form of smoking to spread worldwide, especially within younger ages, our findings have important implications that could aid recommendations regarding the use of EC smoking.

Treatment of Pure Aortic Regurgitation Using a Second-Generation Transcatheter Aortic Valve Implantation System

Transcatheter aortic valve implantation (TAVI) procedure is truly challenging in patients with pure aortic regurgitation (AR). The J-Valve TAVI device (JC Medical Inc., Burlingame, California) is characterized by a U-shaped anatomically orientating device—the “clasper” (1). This design could facilitate intuitive “self-positioning” valve implantation and provide extra-axial fixation by embracing the native valve leaflets. We report the results of TAVI in patients with pure AR using this valve.

From March 1 to December 30, 2014, 33 patients with pure AR and high surgical risk underwent TAVI using this valve including 7 women and 26 men, with mean age 74.2 ± 5.2 years. Mean logistic EuroSCORE I (European System for Cardiac Operative Risk Evaluation) was 24.4 ± 5.1%. Eighty-two percent of patients were symptomatic with New York Heart Association functional class III/IV.

The transapical-based procedure with this valve has been described previously (1). Briefly, the delivery system was inserted into the left ventricle via the apex and then advanced into the ascending aorta over a guidewire. The self-expanding clasper was released and positioned into the corresponding aortic sinus by gentle traction ventricularly. Gentle rotation or adjustment of the delivery system angulation allows the clasper to seat evenly into the aortic sinus, confirming by root angiogram and echocardiography. The valve stent was positioned in the annular plane under the guidance of the clasper and deployed without rapid ventricular pacing (Figure 1). All patients were followed for 6 months. Outcomes were analyzed according to Valve Academic Research Consortium-2 (VARC-2) criteria.
Eleven 25-mm and 22 27-mm prostheses were used with mean aortic annulus diameter of 25.2 ± 1.1 mm. VARC-defined device success was obtained in 94% of patients (31 of 33). One patient was converted to open-heart surgery due to valve embolism caused by insufficient fluoroscopic imaging guidance. One patient had moderate degree paravalvular leakage (PVL) post-implantation. No operative mortality and major complications such as third-degree atrioventricular block, myocardium infarction, cerebrovascular events, or major bleeding event were noted periprocedurally. Minor access site complications occurred in 1 patient. Planned concurrent percutaneous coronary intervention was performed in 2 patients.

All-cause mortality rate was 3% at 30 days, and the patient with moderate-degree PVL died 20 days after surgery due to congestive heart failure. Acute renal injury requiring hemodialysis occurred in 1 patient (3%). The 30-day freedom from event rate was 91% (31 of 33 patients). No patient had greater than mild PVL, whereas 72% (23 of 31 patients) had none or trivial PVL. Two patients underwent permanent pacemaker implantation due to third-degree atrioventricular block.

At 6 months, no structural valve-related dysfunction or reintervention was noted. Mean aortic valve gradient remained stable compared with the 30-day follow-up (8.4 ± 2.8 vs. 8.7 ± 2.8 mm Hg, p = 0.59 using paired t test). PVL remained low throughout follow-up with 87% (27 of 31) of the patients graded as none or trivial. These 31 patients with successful valve implantations were alive with improved exercise tolerance.

The present study demonstrates a high successful implantation rate with low complication rate and excellent hemodynamic performance of this valve in patients with pure AR. The design of this valve relies on the U-shaped anatomical orientating clasper and 2-stage deployment design. The clasper facilitates self-positioning valve implantation, simplifying the implantation procedure, increasing procedural success, and minimizing the risk of coronary ostial obstruction. The clip-fixation between the valve stent and the clasper provides extra-axial fixation force, which makes this device uniquely suitable for AR patient. In contrast to the commercially available JenaValve (JenaValve Technology, Munich, Germany) (2), the anatomically orientating device of the J-Valve is not directly attached to the prosthesis and is flexibly connected to the valve stent by Darcon cords. It has a 2-stage releasing design concept: stage I: the clasper is completely released and accurately positioned; stage II: the valve stent is then deployed with the guidance of the clasper. This unique feature makes it possible for the axial alignment between the valve stent and the clasper to be adjusted separately as needed in order to achieve optimal alignment and positioning of the valve stent.

Our results demonstrated that this new valve could become a potentially feasible treatment option in AR patients who are at high risk for open-heart surgery.
A Tale of 3 Diseases

I have read the paper by Havakuk and Viskin (1) with great interest, and congratulate the authors on their excellent work. As the authors correctly state, both long-QT syndrome and Brugada syndrome may lead to polymorphic ventricular tachyarrhythmias. However, I would like to call attention to another disease: catecholaminergic polymorphic ventricular tachycardia (CPVT). CPVT is characterized by exercise- or stress-induced ventricular tachyarrhythmias, leading to syncope or sudden cardiac death, and it also shares important characteristics.

First, it presents as a congenital or acquired (mainly drug-induced) arrhythmogenic disorder. Disease-causing mutations in ryanodine receptor (RyR2) in autosomal dominant form or calsequestrin 2 genes (CASQ2) in recessive form have been identified in most of affected patients (2). An inducing role of β-adrenergic agonists has been demonstrated. Notably, in CASQ2 CPVT2 patients, a higher risk for cocaine cardiotoxicity has been shown as well (3).

Second, the electrocardiogram at rest is normal (2) but a pathologic early repolarization pattern is present in an unexpected large proportion (45%) of patients and it is associated with an increased frequency of syncope. In patients with unexplained syncope, an exercise testing should be performed to detect CPVT (4).

Third, an evolution of therapeutic approaches has been shown.

Initially, beta-blockers were considered the mainstay therapy for CPVT (2). Recently, flecainide appears to be effective have a role in modulation of intracellular calcium in all CPVT patients. For nonresponders to drug therapy or in patients after a life-threatening arrhythmia, even an implantable cardioverter-defibrillator is needed. Left cardiac sympathetic denervation was reported to be effective in patients with intractable RyR2 mutation-associated CPVT (2).

The history of CPVT (5) retraced the same steps of the 2 arrhythmias described in the article of Havakuk and Viskin (1) and I would have expected that the authors had also shown the tale of CPVT in addition to the tale of Brugada syndrome and long-QT syndrome.

Long-QT Syndrome, Brugada Syndrome, and Catecholaminergic Polymorphic Ventricular Tachycardia

A Tale of 3 Diseases

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http://dx.doi.org/10.1016/j.jacc.2016.03.568

Please note: This study was supported, in part, by grant no. 81200144 from the National Research Foundation of Nature Science, People’s Republic of China and grant no. 1 from JC Medical Inc. Dr. Zhang is the designer of the J-Valve system and the shareholder of JC Medical Inc., Burlingame, California. Dr. Wei and Zhu contributed equally to this work. (Evaluation of the Performance, Efficacy, and Safety of Transapical Aortic Valve Implantation of the J-Valve System in Patients With Severe Symptomatic Native Aortic Valve Stenosis and/or Regurgitation With Elevated Surgical Risk; ChiCTR-OPC-150083540).

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JUNE 14, 2016:2802–8

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Please note: Dr. Patanè has reported that he has no relationships relevant to the contents of this paper to disclose.

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