

Review Article

Managing Complications in Transcatheter Aortic Valve Implantation

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During the last decade, transcatheter aortic valve implantation (TAVI) has expanded the frontiers of interventional cardiology and gained a firm position in the therapeutic quiver for severe aortic stenosis. While the TAVI boom already amounts to more than 100,000 procedures worldwide,¹ its recent (2014) inclusion in the American Heart Association/American College of Cardiology guidelines for the management of valvular heart disease,² in accordance with the respective European ones (2012),³ should open new windows in the management of patients with aortic stenosis. Even though TAVI is generally a relatively safe procedure, heart teams must be prepared to tackle potential obstacles. Current experience has shown that the complications associated with TAVI include vascular complications, paravalvular regurgitation, conduction abnormalities requiring permanent pacemaker (PPM) implantation, acute kidney injury, and transient ischemic attack or stroke.⁴⁻⁸ In the present article we focus on the management of these TAVI complications.

Management of vascular complications

TAVI-associated vascular complications,

according to the Valve Academic Research Consortium - 2 (VARC-2) definitions,⁹ refer to: a) any aortic dissection, aortic rupture, annulus rupture, left ventricle perforation, or new apical aneurysm/pseudoaneurysm; b) any access-site or access-related injury (vessel dissection, perforation, rupture, stenosis, arteriovenous fistula, pseudoaneurysm, hematoma, irreversible nerve injury, compartment syndrome, percutaneous closure device failure); c) distal embolization (non-cerebral) from a vascular source requiring surgical or transcatheter intervention; and d) failure of closure of artery puncture with a percutaneous device. These complications are further classified as major or minor regarding their extent and permanent consequences.

Injury of major heart segments or the proximal aorta is sparsely reported and is managed surgically in most cases, which are related with a very poor prognosis.¹⁰ Thus, these are always classified as major complications.⁹ They are mainly associated with abrupt mechanical forces applied during the TAVI procedure. These complications may therefore occur during expansion of a balloon-expandable bioprosthesis, balloon valvuloplasty, or the appli-

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cation of off-label bailout techniques in the case of complications (see below). An aggressively oversized device may lead to rupture of the landing zone,¹¹ while undersized devices may be distally embolized.¹² Therefore, management of these complications must focus on prevention: extensive preprocedural screening with multislice computed tomography (MSCT) and meticulous individual patient assessment should always be conducted. However, in the undesirable event of such an emergency, beyond the “safe” choice of surgery, other rescue techniques may be utilized if deemed necessary. For example, Blanke et al¹¹ have described a case of effectively treating aortic rupture during TAVI with a SAPIEN prosthesis by implantation of a second bioprosthesis within the first. Additionally, aortic dissection has been reported to occur in from 0.0% up to 1.9% of transfemoral TAVI procedures.¹⁰ While such cases are largely treated with surgical graft interposition, endovascular graft placement has also been proposed as a feasible alternative.^{10,13,14} Therefore, it is easy to understand that, in order to successfully manage such complications, a collaboration of the heart team is required, ideally in a hybrid catheterization laboratory.

Closure failure at the percutaneous arterial puncture site and associated access-related complications have also been a point of discussion. The reported frequency ranges from 1.9% to 30.7%.¹⁵ A high sheath-to-vessel ratio (referring to radius or area), moderate or severe access vessel artery calcification, and access vessel tortuosity have all been identified as negative prognostic factors for vascular complications.^{10,15} Such factors should always be taken into account through comprehensive patient-centered protocols regarding the patient selection and choice of procedural access.¹⁶ Indeed, TAVI vascular access may be obtained either with surgical cut-down or with a truly percutaneous procedure. In the first case, potential complications are, as would be expected, primarily managed surgically. In the case of percutaneous access, complications that involve vascular injury are usually treated with a form of mechanical pressure, which may provide a permanent solution or serve as a bridge to surgery. This involves external manual pressure and endovascular techniques involving balloon inflation and/or (cover) stent implantation, in case of inadequate artery sealing.¹⁷⁻¹⁹

Despite the fact that interventionalists conducting TAVI are usually competent at approaching a femoral artery from the contralateral one, still in such emergencies time is the enemy. Therefore,

technique variants of balloon inflation (advanced through the contralateral femoral or radial artery) above the puncture site prior to final sheath removal and vessel suturing have been developed and effectively used by different centers, including our institution.^{15,17,20-25} These “crossover balloon” techniques, apart from offering the ability to rapidly tackle any vessel injuries that may be revealed just after sheath removal, also provide assistance to artery wall sealing, as suture stabilization is conducted under no hydrostatic tension.

Further, the role of optimal arterial access during TAVI must be underlined. Arterial puncture during transfemoral TAVI can be conducted under angiographic guidance using special landmarks, apart from the bony (puncture below the centerline of the femoral head) and radiopaque (i.e. a pigtail catheter advanced in the femoral artery from the contralateral one) indicators. In particular, the inferior epigastric artery may be visualized with angiography and utilized accordingly. Indeed, the classic knowledge that femoral puncture below the level of the most inferior border of the inferior epigastric artery is associated with fewer procedural complications has been confirmed in TAVI treated patients in a retrospective study conducted in our institution.²⁶

Management of paravalvular regurgitation

Paravalvular regurgitation after TAVI is a common procedural complication that has been identified as a negative prognostic factor.²⁷ Indeed, moderate to severe post-TAVI paravalvular regurgitation is reported to occur at rates of 6.0-13.9% and 9.0-21.0% for the SAPIEN and CoreValve devices, respectively.²⁷ Prosthesis undersizing, prosthesis underexpansion, too high or low implantation depth, and native aortic annulus or left ventricular outflow tract calcification may potentially be implicated in paravalvular regurgitation after TAVI.^{28,29}

In the case of prosthesis undersizing the only treatment is prevention. This means extensive preprocedural imaging. In the initial TAVI experience, two-dimensional (2D) echocardiography and contrast aortography served as main imaging modalities. Today, there are data suggesting that MSCT aortic root evaluation may be associated with less paravalvular regurgitation after TAVI, as the true (greater compared to 2D-echocardiography) dimensions of the aortic annulus are revealed.³⁰ This may be attributed to the oval shape of the aortic annulus.³¹ In addition,

three-dimensional echocardiography has emerged as an alternative to MSCT and is likely to play a role in the near future.³²

Extensive calcification of the aortic annulus and left ventricular outflow tract has also been identified as a predisposing factor for paravalvular regurgitation after TAVI, with either a self- or a balloon-expandable bioprosthesis.³³⁻³⁵ In comparison with surgical aortic valve replacement, where native structures are completely removed, in TAVI the native aortic valve is “sandwiched” between the bioprosthesis and the walls of the aortic root (and ascending aorta), thus trapping the debris of the native valve and calcifications, which form an irregular surface. Therefore, sealing of the bioprosthesis to the adjacent walls may not always be efficient. The most likely mechanism for this is valve underexpansion due to the external forces from the hard calcified compartments of the landing zone; indeed, in extreme cases, this may be observed fluoroscopically during TAVI as a characteristic “string-sign”.³⁶ Additionally, another complementary mechanism to explain the relation between extensive annulus calcification and paravalvular regurgitation may be the formation of fissures within calcium plaques during aortic balloon valvuloplasty. Moreover, extensive calcification could theoretically hamper optimal valve positioning, thus contributing to paravalvular regurgitation via a different mechanism. With the exception of suboptimal valve positioning, post-TAVI balloon dilatation(s) may initially overcome the forces that did not allow the device to reach its default dimensions and treat paravalvular regurgitation.³⁷ However, if this strategy is to be followed, the balloon diameter should not exceed the annular dimensions, as there is always a risk of rupture.¹¹ Moreover, embolic neurological events and conduction abnormalities have been associated with post-TAVI balloon dilatation.³⁸ Additionally, there is a theoretical risk of injury to the prosthesis leaflets, although such a complication is rare and there is only one report of late failure of the valvular mechanism of the prosthesis after balloon dilatation.³⁹ Finally, it should be mentioned that further limited improvement may also be expected with time, if a self-expandable prosthesis has been implanted, as the radial force of the self-expandable stent frame is constant even after deployment.⁴⁰ Nevertheless, there are no firm data regarding the regression of untreated paravalvular regurgitation over time.²⁷

Optimal prosthesis positioning is the target of all interventionalists. However, this is not always

achieved and the prosthesis may be placed too high or too low, resulting in paravalvular aortic regurgitation. Our heart team has described the technical details of the available strategies for bailout correction of initial prosthesis malpositioning.⁴¹⁻⁴³ A too-low bioprosthesis implantation may be partially corrected using two techniques. The most commonly used approach is the “Snare” technique, where special loop-equipped catheters are employed to capture and pull the valve slightly towards the ascending aorta (Figure 1).^{41,42} Additionally, the “Balloon Withdrawal” technique has been tried. This consists of dilating a balloon within the expanded bioprosthesis and simultaneously applying a gentle withdrawal force during peak balloon inflation (Figure 2).⁴³ If a too-high bioprosthesis placement is looming, the operator may use the “Remove-and-Reinsert” technique, in which the CoreValve bioprosthesis is retracted in the sheath, removed from the body, and reinserted (Figure 3).^{41,42} However, this strategy is technically feasible only if the prosthesis is still semi-deployed, and it should be noted that the CoreValve prosthesis is not primarily designed to be retractable. Implantation of the same bioprosthesis after its removal should only be attempted after thorough inspection of the device, as distortion of the stent of the bioprosthesis after such a procedure has been described.⁴⁴ However, a range of fully retrievable and retractable devices are likely to be available in the near future.⁴⁵⁻⁴⁷

In the case of incorrect prosthesis placement, the most widely used technique is the implantation of a second bioprosthesis, either within the first (Figure 4) or through the first if the latter has been distally repositioned using one of the aforementioned techniques. In such cases, special care should be taken not to occlude coronary ostia by the continuity of the prosthesis frames. Recently, Witkowski et al, in a literature review,⁴⁸ illustrated that this strategy may be conducted with safety (despite the theoretical risks) and may have favourable short- and mid-term outcomes. This approach has been labelled as “valve-in-valve” by Ruiz et al⁴⁹ and as the “Russian doll concept” by Piazza et al,⁵⁰ while Witkowski et al⁴⁸ called it “TAV-in-TAV”. Currently, the term “valve-in-valve” is widely used in the literature to describe the performance of TAVI through a surgically implanted bioprosthesis.

Finally, if all the above strategies fail, another possible approach – if the anatomy is suitable – is the closure of the paravalvular leak by implantation of an Amplatzer vascular plug (St. Jude Medical, Inc.). Indeed, successful procedures have been described with

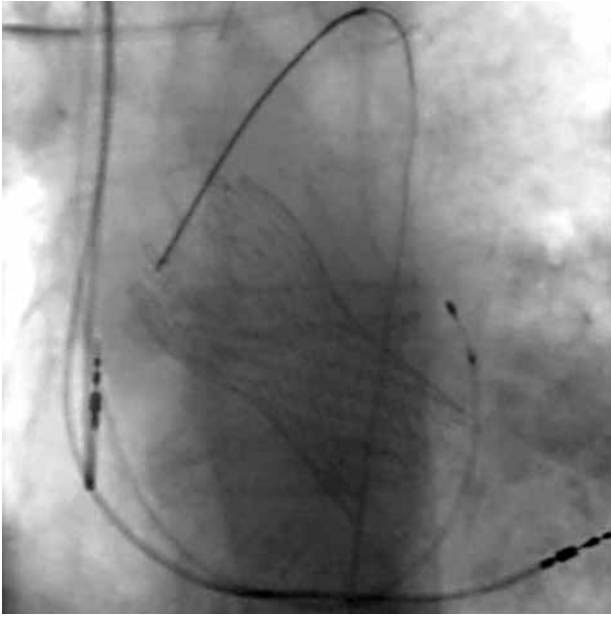


Figure 1. Snare technique: the valve is captured with special loop-equipped catheters and a gentle withdrawal force is applied.

both available aortic bioprostheses (SAPIEN; CoreValve) but most experience is derived from procedures with a SAPIEN device.⁵¹⁻⁵⁶ Of course, this strategy bears a theoretical risk of entrapment of the wires within bioprosthesis struts, closure device embolisation, stroke, valve dislodgement during device delivery, interference with the prosthetic leaflets, coronary occlusion, or persistent haemolysis (after implantation).^{51,57,58} Furthermore, it is clear that such an approach through an implanted CoreValve device, as opposed to a SAPIEN device, may confront extra difficulties, as the nitinol stent of the bioprosthesis extends from the annulus to the ascending aorta.⁵⁸ Recently, however, Gafoor et al described a case series involving successful closure of a CoreValve device paravalvular leak, without facing any major complications.⁵⁶

Management of need for permanent pacemaker

According to a recent review of the available TAVI registries,⁵⁹ PPM implantation rates ranged from 1.8% to 7.1% and from 9.3% to 26.2% after implantation of a SAPIEN or CoreValve aortic bioprosthesis, respectively. These findings were in line with most of the available studies, with the exception of the German TAVI registry (84.4% CoreValve; 15.6% SAPIEN), in which PPM implantation after TAVI reached 39.3%.⁶⁰ Further, a meta-analysis of the lit-

erature in 2002 reported PPM rates of 6.5% vs. 25.8% (i.e. more than threefold higher risk) for TAVI with SAPIEN vs. CoreValve bioprostheses ($p < 0.001$).⁶¹ Finally, in the CHOICE study (an investigator-initiated randomized study including 241 patients: 121 SAPIEN; 120 CoreValve), significantly different rates of new PPM implantation at 30 days post-TAVI were observed for the two devices (17.3% vs. 37.6% for SAPIEN vs. CoreValve; $p = 0.001$).⁶²

The pathophysiological pathway causing conduction abnormalities after TAVI could theoretically involve: (i) the mechanical stress applied to the subvalvular region (which houses critical parts of the conduction system); or (ii) the induced local inflammation and associated oedema caused by pre- or post-TAVI balloon dilatation or directly by the bioprosthesis frame; (iii) “subclinical” microembolisms of the coronary arteries during TAVI; and finally (iv) a combination of the above. Still, it should be highlighted that the validity of the above mechanisms is theoretical, and more clinical, experimental, or post-mortem data are needed to verify their contributions.^{5,63}

Various clinical studies have identified a series of predictors for the development of conduction abnormalities requiring implantation of a PPM. In the meta-analysis by Erkapic et al,⁶¹ pre-existing right bundle branch block, bioprosthesis implantation depth (especially for the CoreValve device), aortic calcification (especially in the non-coronary cusp area and in the area adjacent to the left ventricular outflow tract), and bioprosthesis type (CoreValve) were identified as predictive factors for PPM implantation. Accordingly, prosthesis implantation in a relatively higher positioning, pre-TAVI balloon valvuloplasty with a relatively undersized balloon, or even TAVI without prior valvuloplasty, could possibly reduce the occurrence of conduction abnormalities requiring PPM implantation. However, this needs to be confirmed by randomized studies.⁶⁴

A significant dilemma arises regarding the timing of PPM implantation. The wait-and-see approach involves the additive risks of prolonged temporary pacing (e.g. hospital infections, ventricular perforation, issues related to patient immobilisation, prolongation of hospitalisation duration), in contrast to a more proactive attitude, which is associated with extra financial costs.⁶⁵ Additionally, there is controversy about the permanent or temporary character of the conduction abnormalities that serve as indications for PPM implantation. Though data from Pereira et al⁶⁶ indicate that 66.7% of the patients receiving a PPM

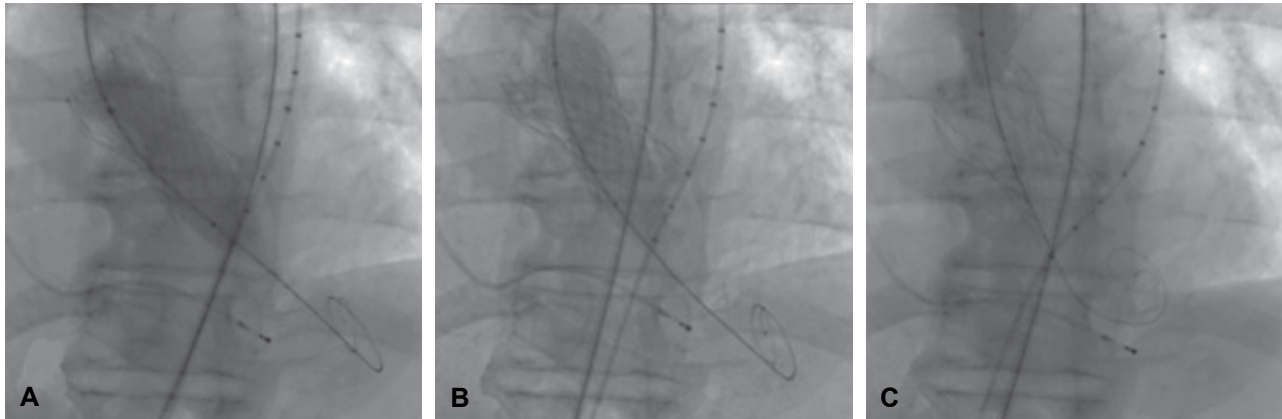


Figure 2. Balloon Withdrawal technique: a balloon is dilated within the deployed bioprosthesis (A). At peak balloon inflation a gentle withdrawal force is applied (B). The bioprosthesis is finally minimally repositioned upward (C).

were pacemaker dependent, this may not always be a criterion for justification of the real need for a PPM.

Management of transient ischemic attacks and stroke

Stroke and/or transient ischemic attacks (TIA) should theoretically stand out as the foremost TAVI complication, given that: (i) a series of manipulations (involving wires, catheters and the prosthesis delivery system) are conducted within the aorta; (ii) a significant burden of calcified tissues (of the native valve leaflets) is crushed between the implanted bioprosthesis and the aortic annulus (or during native aortic valve pre-treatment with balloon valvuloplasty); and (iii) (bio-) prosthetic materials are permanently implanted in the human body. Fortunately, real life does not conform to this pessimistic scenario and disabling strokes are infrequent. Nevertheless, TAVI-associated stroke and/or TIA remain an important issue.

The true extent of the problem is not known, as the available data regarding stroke definitions are not consistent. Indeed even the VARC definitions, which aimed at uniform result reporting, have recently (VARC-2) updated their suggested stroke classification from “TIA, minor stroke, and major stroke” to “TIA, non-disabling stroke, and disabling stroke”.⁹ Furthermore, cerebral injury is not always clinically evident; the impact of “silent” events (identified only by transcranial Doppler during TAVI or by diffusion-weighted magnetic resonance imaging), whilst not included in the VARC-2 definitions, needs further study.^{67,68}

According to data from a recent (2014) meta-analysis by Athanapap et al,⁶⁹ which included 25 mul-

ti-centre and 33 single-centre studies, 30-day post-TAVI stroke ranged from 2.8% to 3.4%. These findings were in line with an earlier (2012) meta-analysis by Eggebrecht et al,⁷⁰ which included 32 studies and reported a 3.3% incidence of 30-day post-TAVI stroke. Moreover, in the same meta-analysis, post-TAVI stroke occurred in 1.5% of the patients in the first 24 h and 5.2% in the first 12 months post-procedure, implying different pathogenetic mechanisms. Additionally, in the randomised “US CoreValve high-risk” trial, the rates of “any stroke” (TAVI vs. surgical AVR) were 4.9% vs. 6.2% at 30 days and 8.8% vs. 12.6% at 12 months (p:NS for both).⁷¹ In contrast, higher stroke frequencies were observed in the PARTNER Cohort A study for TAVI compared to surgical AVR (all stroke & TIA at 30 days: 5.5% vs. 2.4%; p = 0.04, 1 year: 8.3% vs. 4.3%; p = 0.04; 2 years: 11.2% vs. 6.5%; p=0.05; for TAVI vs. surgical AVR, respectively).

As far as pathogenesis is concerned, TAVI-related cerebrovascular events may be classified as being of embolic or non-embolic origin. A further classification may be conducted in relation to the timing of the event: pre-, intra-, or post-procedural. A comprehensive review of these mechanisms was conducted by Ghanem et al in 2013.⁶⁷

Therefore, in an attempt to treat each and every potentially culprit “stage”, the following approaches have been suggested on a theoretical basis:⁶⁷ (i) pre-treatment with antiplatelet (and/or antithrombotic) agents, pre-treatment with lipid-lowering agents, imaging and screening and revascularization of cerebral arteries; (ii) use of embolic protection devices, special care to limit or avoid intra-procedural (iatrogenic or

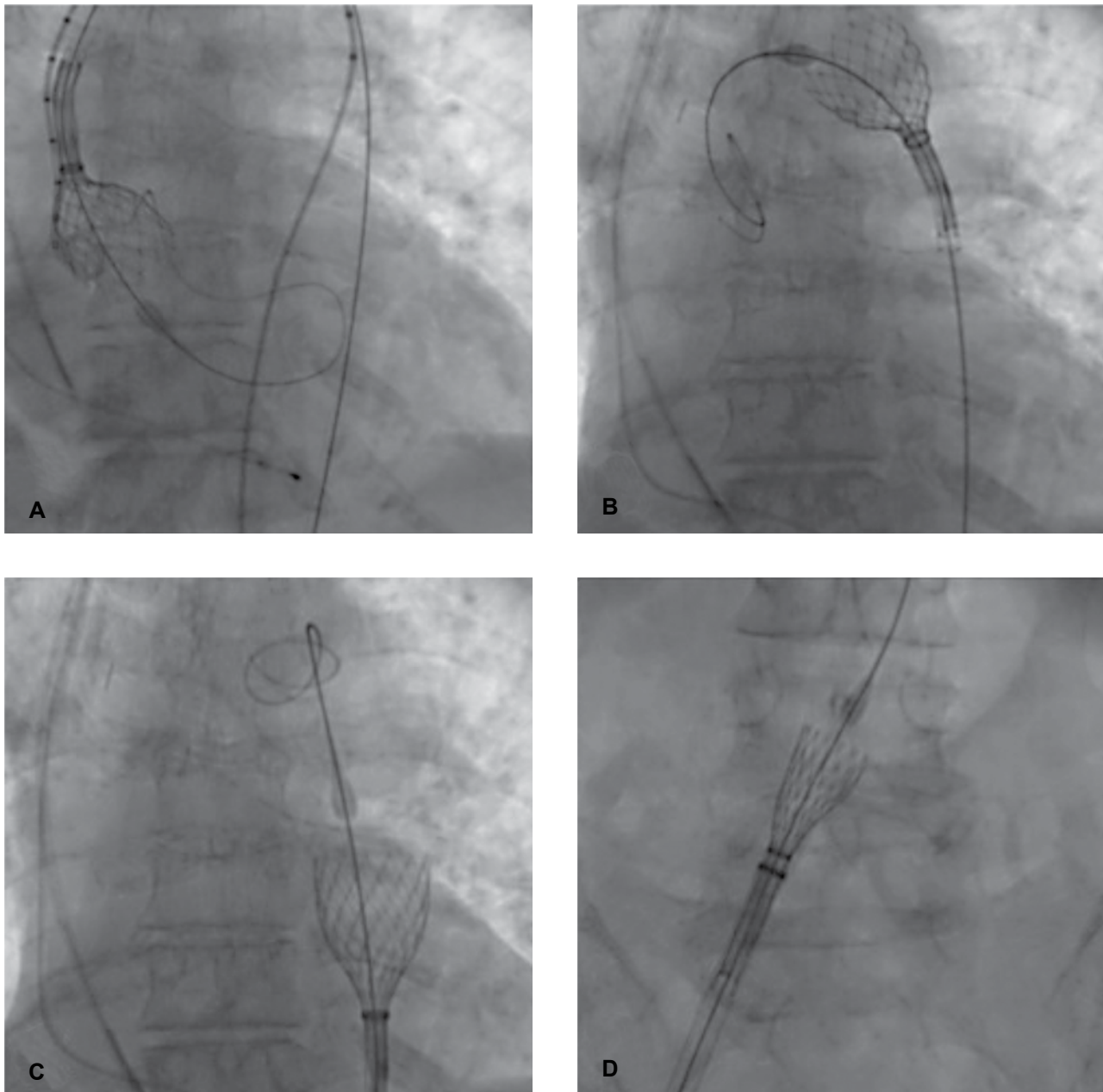


Figure 3. Remove and Reinsert technique: the valve is retracted from the aortic root (A-B) and gradually reinserted into the sheath during continuous flushing with cold normal saline (4°C) (C-D).

complication-related) hypoperfusion, special care to limit or avoid manipulations that may produce emboli; and (iii) post-treatment with antiplatelet (and/or antithrombotic) agents. It should be underlined that little evidence is available and the use of the aforementioned strategies is mainly empirical.

Regarding pre-treatment with antiplatelet, antithrombotic, and/or lipid-lowering agents no data are available. The same is true for the prophylactic revascularization of carotid arteries. However, knowledge

derived from carotid artery stenting studies, where such agents have been used, could possibly be extended to TAVI if confirmed by investigational data.^{67,72}

The use of embolic protection devices during TAVI has not been justified by randomised trials. Experience with such devices is mainly derived from transluminal carotid artery interventions. However, there are reports of feasible and safe utilisation of available devices for emboli deflection (Triguard cerebral protection device [also known as SMT]; Keystone Heart

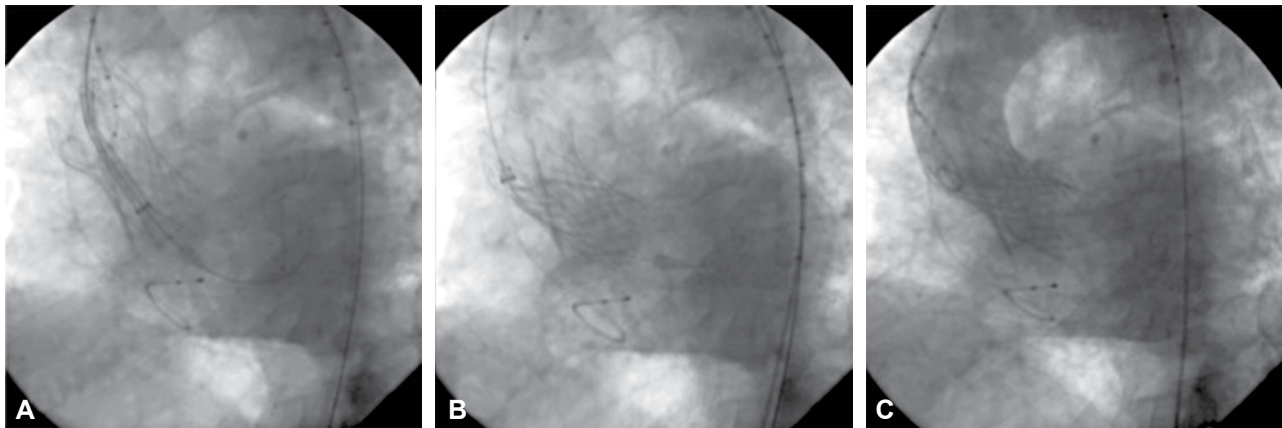


Figure 4. TAV-in-TAV technique: a second bioprosthesis is implanted within the first. A, B: valve deployment, C: final angiography.

Ltd., Embrella device; Edwards Inc.), or emboli capture (Montage device; Claret Medical Inc.) during TAVI.⁷³⁻⁷⁵ Until, more evidence is available, the use of such devices during TAVI should only be considered in selected patients who are at high risk for stroke.^{67,76}

During the procedure, administration of unfractionated heparin with a target activated clotting time of 250-300 s is a common practice. However, the use of bivalirudin instead of heparin is currently being studied in the randomized BRAVO trial (ClinicalTrials.gov Identifier: NCT01651780; estimated primary completion date October 2015). The occurrence of neurological events is a secondary endpoint of this trial.

Regarding antiplatelet and antithrombotic therapy after TAVI, no evidence from randomised trials is available. Knowledge derived from surgical AVR with bioprostheses and observational studies guides treatment strategies. Currently, the 2012 European Society of Cardiology guidelines³ suggest that a 3-month low-dose acetylsalicylic acid regimen should be considered after surgical aortic replacement with a bioprosthesis (recommendation class IIa, level of evidence C), while oral anticoagulation with vitamin K antagonists may also be considered (recommendation class IIb, level of evidence C). However, the authors admit that, in real life, there is off-label administration of double antiplatelet therapy with low-dose aspirin and thienopyridine in the “early” post-TAVI period; when there is a need for oral anticoagulation, vitamin K antagonists with either acetylsalicylic acid or thienopyridine are generally administered. The recent (2014) American College of Cardiology/American Heart Association guidelines² are generally in

line with the European ones regarding recommendations for antiplatelet and antithrombotic therapy after surgical AVR with a bioprosthesis. However, they suggest that 75 mg/day clopidogrel may be considered for the first 6 months after TAVI, together with a lifelong 100 mg/day acetylsalicylic acid regimen (recommendation class IIb, level of evidence C).

In our institution,⁷⁷ we pre-treat patients with acetylsalicylic acid (100 mg/day) for 5 days and with a loading dose of thienopyridine (300 mg clopidogrel). During TAVI, unfractionated heparin is administered with a target activated clotting time of 250-300 s. Post-procedurally, 75 mg/day clopidogrel is prescribed for the first 6 months and 100 mg/day acetylsalicylic acid lifelong. If oral anticoagulation is needed, a vitamin K antagonist is administered along with 75 mg/day clopidogrel for the first 6 months, followed by lifelong 100 mg/day acetylsalicylic acid.

Management of acute kidney injury

The impact of TAVI on kidney function is indisputably a very important issue. In patients with aortic stenosis, kidney function should improve after resolution of the stenosis with TAVI, as the low output state is resolved and the need for kidney-unfriendly medications is lessened.⁷⁸ On the other hand, the required contrast media injections, potential temporary renal hypoperfusion or atheroembolisms during the intervention could have a transient (or not) negative effect.⁷⁸ Additionally, it has been shown that acute kidney injury (AKI) post-TAVI is an independent predictor of death at 30 days, 1 and 2 years.⁷⁹ However, the true extent of this multifactorial phenomenon has not yet been well defined. This may be attributed

to the lack of uniformity in the classification systems used in the available literature. Moreover, in the most recent VARC definitions update, the timing of the diagnosis of AKI has been extended from 3 (VARC-1) to 7 (VARC-2) days, while diagnosis may also be made on the basis of the produced urine output.^{9,80}

Studies comparing TAVI with surgical AVR evaluated post-procedural kidney function and provided such data. In the “U.S. CoreValve High Risk” study, a lower prevalence of AKI at 30 days was observed after TAVI compared to AVR (6.0% vs. 15.1%, respectively, $p < 0.001$).⁷¹ Moreover, in Cohort A of the PARTNER trial, the frequency of patients with serum creatinine levels above 3 mg/dL or in need of renal replacement therapy was reported to be comparable at 30 days and at 1-year post procedure.⁸¹ Finally, in a meta-analysis by Cao et al in 2013,⁸² which included 32 studies (3465 patients; 1688 TAVI) and used the VARC-1 definitions,⁸⁰ the rates of AKI were comparable for TAVI and surgical AVR (6.5% vs. 5.3%, $p = 0.66$).

A very detailed approach regarding the absolute observed post-TAVI frequency of AKI has been provided by Tagaki et al.⁷⁹ The authors summarised all the available published studies (18 studies; 4583 patients), which reported AKI according to the VARC-1 definitions.⁸⁰ The incidence of AKI in the included studies was as follows (cumulative mean; pooled estimate confidence intervals): all AKI (22.0%; 16.3-28.9%), stage 1 (14.3%; 8.4-21.6%), stage 2 (3.5%; 1.5-6.1%), stage 3 (5.0%; 3.5-7.0%), and stages 2 or 3 (10.2%; 4.7-14.6%). While no meta-analysis was conducted in the Tagaki study,⁷⁹ the authors mentioned that blood transfusions, logistic euroSCORE, transapical access, life-threatening bleeding, peripheral vascular disease, and post-procedural leukocyte count had been identified (in some of the included studies) as independent prognostic factors for post-TAVI AKI. Moreover, AKI (in most of the studies regarding advanced stages) was also found to be an independent prognostic factor for death at 30 days, 1 and 2 years.⁷⁹

Unfortunately, there is no way of primarily modifying any of the aforementioned prognostic factors. Therefore, intensive prevention strategies are needed in high-risk patients. First, any potentially nephrotoxic agents (e.g. metformin) should be discontinued. Second, maintenance of a proper balance of hydration with haemodynamic stability and close urine output monitoring before, during, and after intervention is crucial. However, it should be underlined that ex-

cessive intravenous hydration may not always be well tolerated in the fragile TAVI subset of patients (especially pre-procedurally); therefore, it should be administered cautiously.

Regarding contrast media volume, interestingly it has not been reported as a prognostic factor for AKI in a series of TAVI studies.⁷⁹ However, its negative effect on kidney function has been well established. Indeed, Yamamoto et al,⁸³ employing a simple formula (contrast media volume \times serum creatinine \div body weight), showed that increments in contrast media volume and AKI are well correlated.

To conclude, it is beyond dispute that interventionalists should limit contrast media use as far as possible. In this direction, newer devices, like the Direct Flow Medical valve (Direct Flow Medical Inc., Santa Rosa, CA), may afford the capability of implantation guided by transoesophageal echocardiography, using only a few cubic centimetres of contrast media during the entire TAVI procedure.⁸⁴

Finally, the use of protective agents against contrast-induced nephropathy, such as N-acetylcysteine and/or sodium bicarbonate, has been controversial.⁸⁵ The lack of data from randomised studies is to be covered by the results of the PRESERVE study (Prevention of Serious Adverse Events Following Angiography; ClinicalTrials.gov Identifier: NCT01467466; results completion in 2016). In the meantime, however, there are expert opinion publications suggesting thoughtful off-label use of such agents in high-risk patients, despite the current low level of evidence.⁸⁵

Disclosures

Manolis Vavuranakis is a proctor for CoreValve (Medtronic Inc.) Maria Kariori has received a Scholarship from the State Scholarships Foundation of Greece.

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