Cerebral Embolization During Transcatheter Aortic Valve Implantation
A Transcranial Doppler Study

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Background—Transcatheter aortic valve implantation (TAVI) is associated with a higher risk of neurological events for both the transfemoral and transapical approach than surgical valve replacement. Cerebral magnetic resonance imaging has revealed more new, albeit clinically silent lesions from procedural embolization, yet the main source and predominant procedural step of emboli remain unclear.

Methods and Results—Eighty-three patients underwent transfemoral (Medtronic CoreValve [MCVTF], n = 32; Edwards Sapien [ESTF], n = 26) and transapical (ESTA, n = 25) TAVI. Serial transcranial Doppler examinations before, during, and after TAVI were used to identify high-intensity transient signals (HITS) as a surrogate for microembolization. Procedural HITS were detected in all patients, predominantly during manipulation of the calcified aortic valve while stent valves were being positioned and implanted. The balloon-expandable ES prosthesis caused significantly more HITS (mean [95% CI]) during positioning (ESTF, 259.9 [184.8–334.9]; ESTA, 206.1 [162.5–249.7]; MCVTF, 78.5 [25.3–131.6]; P < 0.001) and the self-expandable MCV prosthesis during implantation (MCVTF, 397.1 [302.1–492.2]; ESTF, 88.2 [70.2–106.3]; ESTA, 110.7 [82.0–139.3]; P < 0.001). Overall, there were no significant differences between transfemoral and transapical TAVI or between the MCV and ES prostheses. No HITS were detected at baseline or 3-month follow-up. There was a major procedural stroke that resulted in death and 1 minor procedural stroke with full recovery at 3-month follow-up in the MCV group.

Conclusions—Procedural HITS were detected by transcranial Doppler in all patients. Although no difference was observed between the transfemoral and the transapical approach with the balloon-expandable ES stent valve, transfemoral TAVI with the self-expandable MCV prosthesis resulted in the greatest number of HITS, predominantly during implantation. (Circulation. 2012;126:1245-1255.)

Key Words: aortic valve stenosis cerebral ischemia transcatheter aortic valve implantation valves transcranial Doppler sonography

Transcatheter aortic valve implantation (TAVI) has evolved as the new standard of care for inoperable patients with severe, symptomatic aortic valve stenosis and as a viable treatment option for high-risk yet operable patients, as demonstrated recently in the Placement of Aortic Transcatheter Valves (PARTNER) trial. However, the increased risk of stroke associated with TAVI compared with surgical aortic valve replacement remains a concern. In contrast to surgical aortic valve replacement, strokes and transient ischemic attacks were more frequent after TAVI than after surgical aortic valve replacement, with 30-day event rates of 5.5% versus 2.4%, respectively. Apart from clinically apparent neurological events, TAVI is associated with frequent new, clinically silent lesions on postprocedural cerebral magnetic resonance imaging (MRI), most likely caused by procedural release of atherosclerotic or calcific debris from the aorta or the

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calcified aortic valve. The TAVI entails several procedural steps that can cause cerebral embolization, notably wire passage of the aortic valve, balloon valvuloplasty, advancement of the semi-rigid large-bore device through the aortic arch, positioning of the metallic stent valve within the annulus, and crushing of the stenotic native leaflets during implantation. The precise source and procedural step of embolization, however, remain unclear.

Clinical Perspective on p 1255

The present study reports the results of serial transcranial Doppler (TCD) before, during, and after transfemoral (TF) or transapical (TA) TAVI with either CE-marked device. Microemboli were quantified during each procedural step.

Methods

Patient Population
Between August 2009 and May 2011, 204 consecutive high-risk patients with severe symptomatic aortic valve stenosis underwent TAVI with 1 of the currently CE-approved bioprostheses (Edwards SAPIEN/Edwards SAPIEN XT [ES], Edwards Lifesciences Inc, n = 140 [69%]; Medtronic CoreValve [MCV], Medtronic Inc, n = 64 [31%]) at our institution. Of these 204 patients, 83 (41%) were ultimately included in the present study: 32 (39%) underwent TF-TAVI with the self-expandable MCV prosthesis (group 1); 26 (31%) underwent TF-TAVI (group 2) and 25 (30%) underwent TA-TAVI (group 3) with the balloon-expandable ES prosthesis. One hundred twenty-two patients were excluded from the study: 107 patients had poor acoustic windows and were unsuitable for TCD examination, and 14 refused to participate.

Patients with symptomatic severe aortic valve stenosis were considered for TAVI if they had a logistic European System for Cardiac Operative Risk Evaluation score (EuroSCORE) >20% or surgery was considered an excessive risk because of comorbidities and other risk factors not reflected by EuroSCORE (e.g., porcelain aorta or prior chest radiation). The indication for TAVI in the individual patient was discussed by consensus of cardiologists (P.K., T.K., R.E., H.E.), cardiac surgeons (D.W., M.T., H.G.J.), and cardiac anesthetists (L.B., E.K.), and patient’s preference alone was not considered sufficient. TAVI in these patients was approved by the local authorities, and patients gave informed consent. Patients were excluded from TAVI in the presence of any of the following conditions: Bicuspid aortic valve, aortic annulus diameter <18 or >27 mm, severe iliofemoral artery disease (TF-TAVI only), unprotected left main coronary disease, recent myocardial infarction or cerebrovascular event, sepsis or active endocarditis, severe aortic atheroma (TF-TAVI only), left ventricular (LV) or atrial thrombus, active peptic ulcer, bleeding diathesis, or hypersensitivity to antiplatelet therapy.

TAVI Procedure
TAVI was performed either transfemorally or transapically in a hybrid operating room by standard techniques. The balloon-expandable, trileaflet bovine ES prosthesis was used for both TF-TAVI and TA-TAVI, whereas the self-expandable, trileaflet porcine MCV stent valve was only used for retrograde transfemoral access.

TF-TAVI was performed preferably under anesthetist-controlled conscious sedation with percutaneous right- or left-sided femoral artery access and closure (Perclose ProGlide, Abbott Vascular Inc). After insertion of the large-bore delivery sheath into the access vessel, the native valve was crossed with a left Amplatz diagnostic 6F coronary catheter and a straight-tip 0.035-in guidewire (both from Cordis Corp). A stiff 0.035-in guidewire (Amplatz Super Stiff, 260 cm, 3 mm J-tip, Boston Scientific Corp) with a manually bent curve at the guidewire tip was then placed deep into the LV apex over a standard 6F pigtail catheter. Balloon aortic valvuloplasty was subsequently performed with a 20- or 23-mm balloon catheter under rapid right ventricular pacing to facilitate later passage of the stent valve. Finally, the delivery system that contained the manually crimped and loaded prosthesis was introduced into the LV with retrograde passage of the aortic valve. After it was positioned with fluoroscopic, angiographic, and eventually echocardiographic guidance, the balloon-expandable stent valve was deployed rapidly by balloon inflation under rapid right ventricular pacing at 160 to 220 bpm, whereas the self-expandable prosthesis was deployed stepwise with or without accelerated pacing (100–120 bpm) to prevent dislocation into the ascending aorta during premature ventricular beats.

TA-TAVI was performed under general anesthesia with a left anterolateral minithoracotomy. After placement of purse-string sutures, the LV apex was punctured, and a standard 0.035-in J-tip guidewire advanced antegrade across the aortic valve and directed through the aortic arch down to the descending aorta with a right Judkins 6F diagnostic coronary catheter. After exchange to a stiff guidewire and exchange to the large-bore delivery sheath, balloon aortic valvuloplasty was performed. The crimped ES bioprosthesis was then introduced and positioned within the aortic annulus under fluoroscopic, angiographic, and echocardiographic guidance and finally implanted with rapid right ventricular pacing at 160 to 220 bpm.

Before the procedure, all patients received acetylsalicylic acid (100 mg/d), clopidogrel (75 mg/d after a loading dose of 300 mg/d), and ceftriaxon (2 g) as single-shot antibiotic prophylaxis. During the procedure, intravenous heparin was administered to achieve an activated clotting time >250 seconds for the entire procedure; activated clotting time was measured every 30 minutes. Catheters were flushed carefully with saline to avoid air embolism, and guidewires were cleaned thoroughly before catheter insertion to avoid formation of thrombi on their surface. Hemodynamic stability during the entire procedure was ensured by the attending cardiac anesthetist using a pulmonary artery catheter for invasive hemodynamic monitoring. After the procedure, acetylsalicylic acid was continued indefinitely, whereas clopidogrel was discontinued after 6 months. In patients with atrial fibrillation, phenprocoumon and clopidogrel were administered for 6 months and clopidogrel then exchanged for acetylsalicylic acid.

Assessment of Neurological Status and Cognitive Function
All patients underwent clinical and neurological examination at baseline, after the procedure (when anesthetist-controlled conscious sedation or general anesthesia was reversed), and at 3 months. Neurological status was assessed with the National Institute of Health Stroke Scale (NIHSS) rating.11 The Mini Mental State Examination12 and the Montreal Cognitive Assessment test13 were used to evaluate global cognitive function based on the brief neuropsychological test protocol proposed by the National Institute of Neurological Disorders and Stroke and the Canadian Stroke Network.14 At 3 months, the modified Rankin Scale14 was assessed to characterize the patient’s neurological impairment during daily activities with reference to pre-TAVI activities by grading as no (0), no significant (1), slight (2), moderate (3), moderately severe (4), and severe (5) disability and death (6). Postprocedural neurological events were assessed according the standardized end-point definitions for TAVI trials proposed by the Valve Academic Research Consortium.16

Preoperative Assessment of Potential Sources of Embolism
Before the procedure, all patients were examined for possible sources of embolism by use of electrocardiography, echocardiography, and carotid artery ultrasonography. The history of any previous embolism was recorded.

Transesophageal echocardiography was mandatory as part of the preinterventional TAVI evaluation and was used to detect spontaneous echo contrast, intracardiac thrombi, low left atrial appendage peak velocities of <55 cm/s by pulsed-wave Doppler,17 patent foramen ovale, or other intracardiac shunts, as well as aortic atheroma. Presence, thickness, and characteristics of the atheroma (mobile/protruding/sessile) in the ascending aorta, aortic arch, and descending
Thoracic aorta were graded as absent, mild (<4 mm without complex features), moderate (>4 mm without complex features), or severe (any size with protruding or mobile components). Carotid Duplex ultrasound was used to detect plaque burden and carotid stenoses. Stenoses of the common, internal, and external carotid arteries were measured as reduced diameter and were graded with consideration of all information from B-mode, pulsed-wave, and color flow Doppler. Carotid stenoses were considered significant if there was >70% diameter reduction.

Transcranial Doppler Examination
Simultaneous TCD of both middle cerebral arteries was performed with the subject in a supine position from a transtemporal window with a multigated Multi-Dop T Digital system (DWL Compumedics Germany GmbH) with software for automated HITS detection (QL, version 2.5) and 256-point fast Fourier transformation. Two pulsed-wave 2-MHz Doppler probes were fixed to the patient’s head with a size-adjustable head-mounting system (Dia Mon, DWL Compumedics Germany GmbH) to prevent movement during recording and were used to insonate the middle cerebral arteries at a depth of 50 to 56 mm with a sample volume of 9 to 13 mm. The pulse repetition frequency was set to 7 kHz and the detection threshold for HITS adjusted to 9 dB to reduce artifacts. A fast sweep speed of 4 seconds’ display duration was chosen, and the Doppler velocity range spectrum was adjusted to the expected maximum velocity. Doppler velocity and power M-mode spectrograms were monitored simultaneously (Figure 1).

At baseline and 3 months’ follow-up, TCD was performed for 30 minutes. During TAVI, TCD was performed from femoral or left apical puncture until valve implantation or completion of postimplantation maneuvers, such as postdilation and snaring.

HITS detection and artifact rejection were based on automated software analysis in conjunction with online human observation with use of standard criteria. Two experienced observers (P.D., K.M.) recorded all procedural details to recognize artificial false-positive signals and to attribute signals to the procedural steps. Time-stamped signal recording on a hard drive was used for further offline analysis and regression analysis with the dependent variable HITS (P<0.20) and deemed of clinical importance were included in the multivariable model. All probability values are 2-sided, and a probability value <0.05 was considered significant. All analyses were performed with SPSS (version 19.0, IBM SPSS). The authors had full access to the data and take full responsibility for their integrity. All authors have read and agree to the manuscript as written.

Results

Patient Characteristics
Patients who were excluded from the study had a slightly higher Society of Thoracic Surgeons’ risk score than included patients, were more often female, and more often had diabetes mellitus, whereas a history of smoking and prior cardiac surgery was less frequent. Despite this potential selection bias, the present patient cohort nevertheless reflects typical TAVI patients who are elderly; have severe, symptomatic aortic stenosis; and are at increased surgical risk because of age and comorbidities. The characteristics of the 3 patient groups (group 1: TF-TAVI with MCV prosthesis; group 2: TF-TAVI with ES valve; group 3: TA-TAVI) are summarized in Table 1. Patients undergoing TA-TAVI had a higher logistic EuroSCORE than TF-TAVI patients (29.3 [23.8–34.7] versus 17.7 [13.1–22.4] and 16.1 [12.6–19.6] percent, P<0.001), and diabetes mellitus was more frequent in patients undergoing TF-TAVI with the ES valve (50% versus 21.9% and 20%, P=0.028).

Neurological Status and Cognitive Function Before TAVI
At baseline, focal neurological deficits were observed in 4 patients. One patient of group 1 was blind, and 1 had impaired motor function of the left leg caused by multiple sclerosis, which accounted for NIHSS ratings of 3 and 2, respectively. One patient in group 2 and 1 in group 3 had an NIHSS rating of 1 because of minor facial palsy from a prior stroke. For the other patients, the NIHSS rating was 0. Cognitive function according to the Mini Mental State Examination and Montreal Cognitive

![Figure 1. Transcranial Doppler (TCD) of a patient at rest (A) and during implantation of a Medtronic CoreValve prosthesis (B). No high-intensity transient signals (HITS) were observed at rest, whereas several signals were monitored during valve deployment.](image-url)
Assessment test at baseline revealed no major impairments, with scores of 27.9 (27.5–28.3) and 23.4 (22.7–24.0), respectively, and no differences between groups (online-only Data Supplement I).

Preinterventional Screening for Sources of Embolism
Aortic atheromata were present in all TAVI patients (Table 2) and were graded as mild in 72 patients (87%) and moderate in the remaining 11 (13%). Severe atheroma was considered an exclusion criterion for TF-TAVI in groups 1 and 2 but also was not found in the TA-TAVI group. A single patient in group 2 had a porcelain aorta. Twenty patients (24%) were in permanent atrial fibrillation, and 61 (73%) had reduced left atrial appendage peak velocities, which were found significantly more frequently in TF-TAVI patients treated with the MCV prosthesis than in the other 2 patient groups. Spontaneous echo contrast was found in 14 patients (17%), but no intracardiac thrombi were seen. A patent foramen ovale was present in a single patient each in groups 2 and 3, and 9 patients (11%) had a prior cerebral ischemic event. Carotid artery stenosis with 70% diameter reduction was seen in 2 patients (2%), luminal narrowing between 30% and 70% in 27 (33%), and 30% in the remaining 54 patients (65%). Sixty-three patients (76%) received statins. Overall, variables that indicated potential sources for embolism were not significantly different among groups except for left atrial appendage velocities. Of note, there were also no differences in these variables between patients included in and excluded from the study.

Procedural Results
TAVI was technically successful in all patients; mortality at 30 days and at 3 months was 8.4% and 12.0%, respectively (Table 3). Implantation of the stent-valve prostheses resulted
in a postinterventional mean transaortic gradient of 9.9 (8.8–10.9) mm Hg and an aortic valve area of 1.70 (1.61–1.80) cm² (P<0.001 versus baseline). Overall, procedural data were not different among groups except for prosthesis size, procedure time, and fluoroscopy time, which remained statistically significant variables after adjustment for age, sex, logistic EuroSCORE, and diabetes mellitus. Procedure time was lowest in patients undergoing TF-TAVI with the MCV prosthesis and highest in TA-TAVI patients, whereas fluoroscopy time was lowest in patients undergoing TF-TAVI with the MCV prosthesis and highest in TA-TAVI patients, though this did not change rating of significance.

Nonneurological procedural complications related to the vascular access site occurred in 5 TF-TAVI patients (group 1, n=2; group 2, n=3) and required endovascular (group 1, n=2; group 2, n=2) or surgical (group 1, n=1; group 2, n=0) repair. With the MCV prosthesis, dislocation of the stent valve was observed in 4 patients during the first implantation attempt, which resulted in subsequent valve retrieval, re-cramping, and reimplantation. Three patients became hemodynamically unstable during the implantation, 1 in group 1 because of volume depletion and 2 in group 2 because of new-onset tachycardic atrial fibrillation that required cardioversion and low-output failure that required short-term cardiopulmonary resuscitation, catecholaminergic support >0.1 µg·kg⁻¹·min⁻¹, and switch from anesthetist-controlled conscious sedation to general anesthesia.

Postdeployment valvuloplasty within the same session was performed in 11 patients (group 1, n=8; group 2, n=1; group 3, n=2; P=0.04) and snaring in 2 patients (both in group 1; P=0.195) to diminish residual paravalvular regurgitation from incomplete stent-frame expansion or too low initial implantation, respectively.

### Transcranial Doppler Examination

Baseline TCD revealed no HITS in any patient. During TAVI, however, HITS were observed in every patient in each group. A similar amount of HITS were observed for all 3 approaches (Figure 2; Table 4) during antegrade (TA-TAVI)
and retrograde (TF-TAVI) wire passage of the aortic valve and introduction and propagation of the stiff guidewire into the LV apex (TF-TAVI) or the descending aorta (TA-TAVI). Introduction and placement of the balloon for preparatory balloon valvuloplasty was associated with slightly fewer HITS, especially for the transfemoral approach. During subsequent balloon valvuloplasty, the amount of HITS was again similar to the previous steps and not different between groups. Introduction and propagation of the loaded delivery devices toward the aortic annulus resulted in a similar HITS frequency as during introduction of the valvuloplasty balloon. During positioning of the stent valve within the native aortic annulus, a considerable increase in HITS was noted in all groups. This increase in HITS was 3 times more pronounced with the transfemoral and transapical ES valve than the MCV prosthesis. Conversely, significantly more HITS were observed during stepwise implantation of the MCV prosthesis than during rapid ES valve implantation via the transfemoral or transapical approach.

### Table 3. Procedural Data

<table>
<thead>
<tr>
<th></th>
<th>Overall TAVI (n = 83)</th>
<th>Group 1: TF-TAVI With MCV (n = 32)</th>
<th>Group 2: TF-TAVI With ES (n = 26)</th>
<th>Group 3: TA-TAVI With ES (n = 25)</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>General anesthesia, n (%)</td>
<td>33 (39.8)</td>
<td>2 (6.2)</td>
<td>6 (23.1)</td>
<td>25 (100)</td>
<td>&lt;0.001†</td>
</tr>
<tr>
<td>Conscious sedation, n (%)</td>
<td>50 (60.2)</td>
<td>30 (93.8)</td>
<td>20 (76.9)</td>
<td>0 (0)</td>
<td>&lt;0.001‡</td>
</tr>
<tr>
<td>Switch from conscious sedation to general anesthesia, n (%)</td>
<td>1 (1.2)</td>
<td>0 (0)</td>
<td>1 (3.8)</td>
<td>0 (0)</td>
<td>0.614</td>
</tr>
<tr>
<td>Hemodynamic instability, n (%)</td>
<td>3 (3.6)</td>
<td>1 (3.1)</td>
<td>2 (7.7)</td>
<td>0 (0)</td>
<td>0.498</td>
</tr>
<tr>
<td>Vascular complications, n (%)</td>
<td>5 (6.0)</td>
<td>3 (9.4)</td>
<td>2 (7.7)</td>
<td>0 (0)</td>
<td>0.372</td>
</tr>
<tr>
<td>Prosthesis size, mm, n (%)</td>
<td>23 (18 [22]</td>
<td>-</td>
<td>14 (54)</td>
<td>4 (16)</td>
<td>&lt;0.001§</td>
</tr>
<tr>
<td>Procedure time, min Mean (95% CI)</td>
<td>68.0 (60.0–75.5)</td>
<td>48.5 (42.5–54.4)</td>
<td>59.3 (50.4–68.2)</td>
<td>101.2 (83.2–119.3)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Fluoroscopy time, min Mean (95% CI)</td>
<td>12.0 (10.3–13.3)</td>
<td>12.4 (10.5–14.3)</td>
<td>16.3 (13.4–19.2)</td>
<td>6.3 (4.3–8.3)</td>
<td>&lt;0.001#</td>
</tr>
<tr>
<td>Contrast volume, mL Mean (95% CI)</td>
<td>169.4 (153.1–185.6)</td>
<td>170.5 (145.4–195.5)</td>
<td>199.7 (165.1–234.2)</td>
<td>136.6 (114.0–159.1)</td>
<td>0.009**</td>
</tr>
<tr>
<td>Administered heparin, IU Mean (95% CI)</td>
<td>6963.9 (6412.7–7515.0)</td>
<td>6828.1 (6072.1–7584.1)</td>
<td>6327.0 (5556.4–7097.4)</td>
<td>7800.0 (6424.5–9175.5)</td>
<td>0.209</td>
</tr>
<tr>
<td>Aortic valve area after TAVI, cm² Mean (95% CI)</td>
<td>1.70 (1.61–1.80)</td>
<td>1.68 (1.57–1.79)</td>
<td>1.68 (1.46–1.91)</td>
<td>1.76 (1.54–1.98)</td>
<td>0.764</td>
</tr>
<tr>
<td>Mean transaortic gradient after TAVI, mm Hg Mean (95% CI)</td>
<td>9.9 (8.8–10.9)</td>
<td>9.5 (8.5–10.6)</td>
<td>9.3 (6.6–12.0)</td>
<td>10.9 (8.6–13.2)</td>
<td>0.476</td>
</tr>
<tr>
<td>30-d Mortality, n (%) Mean (95% CI)</td>
<td>7 (8.4)</td>
<td>3 (9.4)</td>
<td>2 (7.7)</td>
<td>2 (8)</td>
<td>1.000</td>
</tr>
<tr>
<td>3-mo Mortality, n (%) Mean (95% CI)</td>
<td>10 (12.0)</td>
<td>4 (12.5)</td>
<td>3 (11.5)</td>
<td>3 (12)</td>
<td>1.000</td>
</tr>
</tbody>
</table>

TAVI indicates transcatheter aortic valve implantation; TF, transfemoral; MCV, Medtronic CoreValve; ES, Edwards Sapien valve; CI, confidence interval; Q1, quartile 1; and Q3, quartile 3.

Continuous variables are presented as mean (95% CI) and median (Q1; Q3); other values are n (%).

*After adjustment for age, sex, and the 2 significantly different variables in baseline patient characteristics (logistic EuroSCORE and diabetes mellitus), differences in the use of general anesthesia and conscious sedation and in the amount of contrast volume lost significance.

Pairwise comparisons with Bonferroni correction were performed for variables for which there was a significant difference between groups:

†Group 1 vs group 2: P=0.192; group 1 vs group 3: P<0.001; group 2 vs group 3: P<0.001.
‡Group 1 vs group 2: P=0.369; group 1 vs group 3: P<0.001; group 2 vs group 3: P<0.001.
§Group 1 vs group 2: P<0.001; group 1 vs group 3: P=0.045; group 2 vs group 3: P<0.001.
∥Group 1 vs group 2: P=0.012; group 1 vs group 3: P<0.001; group 2 vs group 3: P<0.001.
#Group 1 vs group 2: P=0.054; group 1 vs group 3: P<0.001; group 2 vs group 3: P<0.001.
**Group 1 vs group 2: P=0.474; group 1 vs group 3: P=0.150; group 2 vs group 3: P=0.009.
There was no difference in overall periprocedural HITS between transfemoral and transapical ES valve implantation and only a trend toward more HITS during transfemoral MCV implantation. There was also no difference between the number of HITS in the right and left middle cerebral arteries (group 1, 330.2 [260.4–400.1] versus 281.5 [227.3–335.6], P=0.109; group 2, 243.8 [197.3–290.1] versus 238.4 [185.1–291.7], P=0.594; group 3, 217.4 [169.7–265.1] versus 266.9 [210.6–323.3], P=0.108).

The 4 patients in group 2 who had the MCV prosthesis dislocated during the first implantation attempt had an additional 136.8 (27.5–304.8) HITS during initial positioning, dislocation, and the subsequent retrieval process. Without a counterpart in both ES groups, this additional step was not included in the comparison but contributed to the higher HITS rate in the MCV group.

Postdeployment valvuloplasty resulted in an additional 21.6 (16.1–27.1) HITS (group 1, 19.5 [12.5–26.5]; group 2, 23; group 3, 27 and 31; P=0.331), which was less than the HITS during preparatory valvuloplasty before valve implantation in these patients (21.6 [16.1–27.1] versus 58 [23.0–93.0], P=0.049). The 2 patients with snaring of the MCV prosthesis had 9 and 14 additional HITS, respectively.

**Acute Neurological Outcome**

We observed 2 procedural strokes, both in group 2. The patient requiring cardiopulmonary resuscitation and vasoactive support experienced a major stroke and died 3 days later (modified Rankin scale score=6). Another patient experienced a stroke that resulted in right-sided hemiparesis and accounted for an immediate postinterventional NIHSS rating of 8. Three points were scored for impairment of motor leg function, 2 points for impairment of motor arm function, 1 point for limb ataxia, and 2 points for severe aphasia. The neurological symptoms regressed continuously, which resulted in an NIHSS rating of 3 (2 points for impairment of motor leg function and 1 point for limb ataxia) after 4 days and at discharge. Because of full recovery without sequelae at 3-month follow-up (NIHSS rating 0, modified Rankin scale score=0), this stroke was graded as minor.

In the other patients, there were no neurological complications with changes in NIHSS scoring, and a postprocedural Mini Mental State Examination score of 27.7 (27.3–28.2) indicated no decline in cognitive function compared with baseline (P=0.521). A postprocedural Montreal Cognitive Assessment score of 24.0 (23.3–24.6) showed a slight but significant decline (P=0.001), probably caused by a learning effect (online-only Data Supplement Table I).

**Follow-Up**

During 3-month follow-up, there were no late neurological events, and TCD at 3 months showed no HITS. Clinical examination showed no sequelae in the patient with the periprocedural stroke in group 2, which accounts for the NIHSS rating of 0 and a modified Rankin scale score of 0. In all 3 patient groups, neither a new neurological deficit nor a new disability or any progressive deficit in cognitive function were observed. Accordingly, the NIHSS rating remained unchanged compared with baseline, and there was no decline (and even a minimal increase) in the Mini Mental State Examination (28.3 [28.0–28.7]) and Montreal Cognitive Assessment (24.3 [23.6–24.9]) scores (P versus baseline <0.001; online-only Data Supplement Table I).

**Potential Determinants of Procedural HITS**

Univariate linear regression analysis identified logistic EuroSCORE, Society of Thoracic Surgeons’ risk score, mean transaortic gradient at baseline, presence of coronary artery disease, obesity, diabetes mellitus, aortic atheroma thickness, carotid artery plaque burden, left atrial appendage velocity, a prior cerebral ischemic event, amount of administered heparin during the procedure, and valve size to be associated with an increased frequency of HITS during TAVI. Eight of these parameters (logistic EuroSCORE, mean transaortic gradient at baseline, aortic atheroma thickness, carotid artery plaque burden, left atrial appendage velocity, a prior cerebral ischemic event, amount of administered heparin, and valve size) were entered into the multivariable model, and mean transaortic gradient at baseline was confirmed as an independent predictor for the frequency of HITS, although the relatively small number of patients in our single-center study and the relatively high number of independent variables eligible for the multivariate model might have affected the precision of this analysis (online-only Data Supplement Table II).

**Discussion**

The present prospective study characterized the origins of periprocedural embolization in 83 patients undergoing transfemoral or transapical TAVI with either of the balloon-expandable ES or the self-expandable MCV prosthesis. TCD of both middle cerebral arteries revealed cerebral microembolization as reflected by HITS in all patients during TAVI, notably during manipulation of the calcified aortic valve while the stent prostheses were positioned and implanted, reminiscent of what is seen during stent implantation in the coronary circulation.22 Despite HITS in all patients, only 2...
neurological events occurred periprocedurally, and neurocognitive function during the 3-month follow-up was not impaired.

Stroke and neurological events are major complications of both cardiovascular interventions and surgery. During isolated surgical aortic valve replacement, stroke is rare, with an occurrence rate of 1.5% in normal-risk and up to 4.5% in elderly high-risk patients. For TAVI, however, an increased rate of neurological events has raised safety concerns. In both the inoperable cohort B and the operable cohort A of the PARTNER trial, neurological events within the first 30 days were observed in 6.7% and 5.5% of patients, respectively, and occurred more frequently than in patients treated with optimal medical therapy (1.7%) or surgical aortic valve replacement (2.4%). In the high-risk patients eligible for either TAVI or surgical aortic valve replacement, the major stroke rate within 30 days was 3.8% and was higher than that after surgical aortic valve replacement (2.1%). Seven (58%) of the 12 strokes observed within 30 days after TAVI occurred within the first 2 days. The assignment to TAVI versus surgical aortic valve replacement was identified as an independent risk factor for such early strokes.

Manipulation of a calcified stenotic aortic valve during diagnostic retrograde catheterization has been associated with an increased rate of neurological events, and an even higher rate of cerebral embolization was detected in 22% of cases by cerebral diffusion-weighted MRI. TAVI exerts an even greater trauma on the calcified valve during preparatory valvuloplasty, valve passage with the semirigid large-bore delivery catheters that contain the crimped stent valve, valve positioning, and crushing of the native leaflets to the aortic wall during final implantation. New but clinically silent lesions were found on cerebral diffusion-weighted MRI in 68% to 91% of patients after TAVI.

<table>
<thead>
<tr>
<th>Table 4. Procedural High-Intensity Transient Signals</th>
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</thead>
<tbody>
<tr>
<td>Overall TAVI (n = 83)</td>
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<tr>
<td>-----------------------</td>
</tr>
<tr>
<td>Aortic valve passage</td>
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<tr>
<td></td>
</tr>
<tr>
<td>Stiff guidewire</td>
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<tr>
<td></td>
</tr>
<tr>
<td>Propagation and placement of the valvuloplasty balloon</td>
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<tr>
<td></td>
</tr>
<tr>
<td>Balloon aortic valvuloplasty</td>
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<tr>
<td></td>
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<tr>
<td>Delivery device</td>
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<tr>
<td></td>
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<tr>
<td>Valve positioning</td>
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<tr>
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<tr>
<td>Valve implantation</td>
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<tr>
<td></td>
</tr>
<tr>
<td>Sum of all procedural steps</td>
</tr>
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<td></td>
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</tbody>
</table>

TAVI indicates transcatheter aortic valve implantation; TF, transfemoral; MCV, Medtronic CoreValve; ES, Edwards Sapien valve; CI, confidence interval; Q1, quartile 1; and Q3, quartile 3.

Variables are presented as mean (95% CI) and median (Q1; Q3).

*Adjustment for age, sex, and the 2 significantly different variables in baseline patient characteristics (logistic EuroSCORE and diabetes mellitus) did not change rating of significance.

Pairwise comparisons with Bonferroni correction were performed for variables where there was a significant difference between groups:

†Group 1 vs group 2: P=1.0; group 1 vs group 3: P=0.012; group 2 vs group 3: P=0.066.

‡Group 1 vs group 2: P<0.001; group 1 vs group 3: P<0.001; group 2 vs group 3: P=1.0.

§Group 1 vs group 2: P<0.001; group 1 vs group 3: P<0.001; group 2 vs group 3: P=1.0.
after surgical aortic valve replacement. Collectively, these findings support the notion that the predominant cause of periprocedural strokes during TAVI is embolic and that emboli consist of debris from the calcified native aortic valve or from aortic arch atheromata, which are common in elderly patients undergoing TAVI.

Indeed, TCD identified cerebral microembolization as a common event in each of the procedural TAVI steps, for both the balloon-expandable ES and the self-expandable MCV and for the transfemoral and the transapical approach, and the calcified aortic valve as the main source of emboli. Most HITS were recorded during valve manipulation, and mean transaortic gradient at baseline (reflecting stenosis severity) was the main determinant of HITS by multivariate analysis. Apparently, the aortic arch plays only a minor role in periprocedural stroke, and the transapical approach may therefore not be superior to the transfemoral approach.

The lower amount of HITS during MCV than ES prostheses positioning was expected because initial positioning of the MCV prosthesis is rather quick, and continuous adjustments are performed subsequently during stepwise release of the self-expandable stent frame, whereas precise positioning before implantation is more time-consuming for correct placement of the ES valve. Conversely, more HITS were recorded during the relatively slow, stepwise release of the MCV prosthesis than during rapid, single-shot implantation of the ES valve, which supports the idea that the metallic stent frame acts in a grater-like fashion, scraping calcific debris from the native valve. The duration of aortic valve manipulation apparently also determines cerebral embolization. In line with the time is brain concept, additional HITS were recorded in 4 patients with MCV dislocation during the initial implantation attempt, which required additional time and caused additional stress to the aortic valve. High numbers of new cerebral lesions on diffusion-weighted MRI in 2 cases of MCV dislocation have been reported previously (7 and 26, respectively). These caveats must be considered for next-generation TAVI devices that offer repositionability and retrievability and require more extensive valve manipulation.

Surprisingly, the amount of HITS during preparatory balloon aortic valvuloplasty was relatively low. Possibly, endothelial coverage prevents calcific debris from release and embolization at this stage of the procedure. Balloon valvuloplasty may only disrupt the protective endothelial layer and expose friable debris that is subsequently liberated during valve positioning and implantation. We can currently only speculate whether or not omission of the preparatory valvuloplasty reduces the risk of embolic stroke. Grube et al have recently shown that TAVI with the MCV prosthesis is safe without balloon predilation and is associated with a 5% risk of stroke. Although this rate was lower than in their nonrandomized historical control group (11.9%), it is not lower compared with the stroke rates currently reported for the MCV prosthesis in large multicenter registries, and postdilation was necessary in 16.7% of cases. Of note in this context, postdeployment valvuloplasty results in fewer HITS than preparatory valvuloplasty. Although cerebral microembolization during TAVI occurred in all patients, neurological events were rare, with only 1 major and 1 minor stroke, in agreement with the disparity between frequent cerebral lesions and the few neurological events in the above-mentioned neuroimaging studies. The clinical relevance of silent neuroimaging lesions and the high number of periprocedural microemboli remain unclear but must be resolved when the indication for TAVI is broadened to younger, lower-risk patients, because silent emboli have been associated with declining neurocognitive function and deterioration of dementia. In the present analysis, we did not observe changes in neurocognitive function; however, subtle behavioral and cognitive impairments may not have been detected by the crude Mini Mental State Examination and the Montreal Cognitive Assessment test.

The high frequency of lesions on neuroimaging and HITS in TCD calls for procedural and technical developments to reduce the risk of periprocedural embolization: Less traumatic devices, avoidance of extensive manipulation of the calcified aortic valve (“time is brain”), carotid artery compression during valve positioning and deployment, omission of preparatory balloon valvuloplasty, and protection devices are currently under consideration. Omission of preparatory balloon valvuloplasty did not reduce strokes below currently reported rates in a pilot study, and only 1 small feasibility study suggested placement of an embolic deflector device over the brachiocephalic trunk and the left carotid artery as a safe and promising approach for active cerebral protection.

Study Strengths and Limitations

Serial real-time TCD is a standard surveillance technique during neurological interventions such as carotid endarterectomy, and it identified the source and procedural steps of cerebral embolization in patients in the present study undergoing TAVI. Our single-center study was descriptive, without prespecified power and sample size and without randomization between balloon and self-expandable prostheses or between transfemoral and transapical access, but nevertheless, it reflects a typical cohort of patients currently undergoing TAVI. Surgical aortic valve replacement encompasses entirely different procedural steps and was therefore not used for comparison.

Unfortunately, characterization of individual emboli by their size and composition is currently impossible by TCD. Specifically, solid and gaseous emboli are not distinguished by conventional systems, and even with more sophisticated equipment that uses dual-frequency transducer technology, such distinction is not sufficiently accurate, especially during procedures in which both occur. Because of this methodology-inherent limitation of TCD, it remains impossible to clearly attribute HITS to embolic valve material, trapped air, or microbubbles released during catheter flushing and contrast injections. Obviously, the neurological consequences of a few large, solid emboli are greater than those from multiple gaseous microbubbles. In the present study, however, most emboli were recorded during phases with a high probability of solid emboli, namely, valve positioning and implantation, and phases of catheter flushing and contrast injections were excluded from the analysis. If one assumes
that solid rather than gaseous emboli are associated with new neuroimaging lesions, correlation of HITS with new lesions on postinterventional MRI might have been interesting, but this was not feasible in all patients, and our previous neuroimaging studies had already demonstrated a high rate of new cerebral lesions after TAVI.

Conclusions
Cerebral microembolization, reflected by HITS on TCD, is inherent to TAVI and occurs in each procedural phase, predominantly during positioning and implantation of the stent prostheses, rendering the calcified aortic valve as the main source of emboli. HITS, however, were not associated with an increased rate of neurological deficits or acutely impaired neurocognitive function. The real neurological risk of the TAVI procedure and the feasibility, safety, and efficacy of upcoming strategies to reduce the risk of cerebral embolization require further study.

Acknowledgments
We thank Nils Lehmahn from the Institute of Medical Informatics, Biometry and Epidemiology of the University Hospital Essen for his advice.

Disclosures
Drs Kahlert and Thielmann are clinical proctors for Edwards Lifesciences Inc and have received honoraria payments. Dr Eggbrecht is a clinical proctor for Medtronic Inc and has received honoraria payment. The other authors report no conflicts.

References


Neurological events are currently considered one of the most pressing concerns with transcatheter aortic valve implantation (TAVI). A nearly 4-fold risk of such events within 30 days after the procedure was observed for nonoperative patients undergoing TAVI compared with patients treated with optimal medical therapy in cohort B of the Placement of AoRtic TraNscathetER valves (PARTNER) trial, and similar results were found when TAVI was compared with surgical aortic valve replacement in the high-risk population of cohort A. Moreover, a high load of clinically silent embolic lesions was documented on postprocedural cerebral diffusion-weighted magnetic resonance imaging, which has raised additional safety concerns. In the present study, serial transcranial Doppler monitoring was performed to elucidate the main source of procedural emboli. During TAVI, high-intensity transient signals were detected in all patients as a surrogate for microembolization. They predominantly occurred during manipulation of the calcified native valve while positioning and implanting the stent valves, with no differences between the transfemoral and the transapical approach and only a trend toward a higher amount of high-intensity transient signals for the self-expandable prosthesis. Despite the omnipresence of high-intensity transient signals, however, only 2 (2.4%) neurological complications occurred within 30 days, and there were no late neurological events. However, these findings corroborate the importance of periprocedural embolization during TAVI and reinforce current calls for an increased focus on this issue, especially when TAVI indications are broadened toward younger, lower-risk patients. Future research is essential to better determine the “real” neurological risk of the TAVI procedure and to thoroughly investigate the feasibility, safety, and efficacy of upcoming strategies to reduce the risk for cerebral embolization, eg, use of less traumatic devices, omission of preparatory valvuloplasty, carotid compression during valve manipulation, or use of active cerebral protection devices.
Cerebral Embolization During Transcatheter Aortic Valve Implantation: A Transcranial Doppler Study
Philipp Kahlert, Fadi Al-Rashid, Philipp Döttger, Kathrine Mori, Björn Plicht, Daniel Wendt, Lars Bergmann, Eva Kottenberg, Marc Schlaman, Petra Mummel, Dagny Holle, Matthias Thielmann, Heinz G. Jakob, Thomas Konorza, Gerd Heusch, Raimund Erbel and Holger Eggebrecht

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**SUPPLEMENTAL MATERIAL**

**SUPPLEMENTAL TABLES**

Table 1. Neurocognitive Testing.

<table>
<thead>
<tr>
<th></th>
<th>Overall TAVI (n=83)</th>
<th>Group 1 TF-TAVI with MCV (n=32)</th>
<th>Group 2 TF-TAVI with ES (n=26)</th>
<th>Group 3 TA-TAVI with ES (n=25)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>MMSE</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>baseline</td>
<td>27.9 (27.5-28.3)</td>
<td>28.0 (27.5-28.5)</td>
<td>27.3 (26.1-28.6)</td>
<td>28.3 (27.8-28.8)</td>
<td>0.625</td>
</tr>
<tr>
<td></td>
<td>28 (27; 29)</td>
<td>28 (27; 29)</td>
<td>28 (27; 29)</td>
<td>28 (27; 29)</td>
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</tr>
<tr>
<td>post TAVI</td>
<td>27.7 (27.3-28.2)</td>
<td>27.7 (26.9-28.4)</td>
<td>27.6 (26.4-28.7)</td>
<td>28.0 (27.4-28.5)</td>
<td>0.911</td>
</tr>
<tr>
<td></td>
<td>28 (27; 29)</td>
<td>28 (27; 29)</td>
<td>28 (27; 29)</td>
<td>28 (27; 29)</td>
<td></td>
</tr>
<tr>
<td>3-months follow-up</td>
<td>28.3 (28.0-28.7)</td>
<td>28.4 (27.9-28.9)</td>
<td>28.1 (27.0-29.1)</td>
<td>28.6 (28.1-29.0)</td>
<td>0.840</td>
</tr>
<tr>
<td></td>
<td>29 (28; 29)</td>
<td>28 (28; 29)</td>
<td>29 (27; 29)</td>
<td>29 (28; 29)</td>
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</tbody>
</table>

*Pairwise comparison*

- baseline vs. post TAVI: p=0.521, p=0.488, p=0.400, p=0.256
- baseline vs. 3 months: p<0.001, p=0.083, p=0.003, p=0.096
- post TAVI vs. 3 months: p=0.001, p=0.048, p=0.079, p=0.023
<table>
<thead>
<tr>
<th></th>
<th>baseline</th>
<th>post TAVI</th>
<th>3-months follow-up</th>
<th>Pairwise comparison</th>
</tr>
</thead>
<tbody>
<tr>
<td>MoCA</td>
<td>23.4 (22.7-24.0)</td>
<td>24.0 (23.3-24.6)</td>
<td>24.3 (23.6-24.9)</td>
<td>Variables are presented as mean (95% CI) (<em>first row</em>) and median (Q1; Q3) (<em>second row</em>).</td>
</tr>
<tr>
<td></td>
<td>24 (21; 25)</td>
<td>25 (22; 26)</td>
<td>25 (23; 26)</td>
<td></td>
</tr>
<tr>
<td>MoCA</td>
<td>23.9 (22.9-24.9)</td>
<td>24.2 (23.1-25.3)</td>
<td>24.4 (23.3-25.5)</td>
<td></td>
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<td></td>
<td>24 (22; 26)</td>
<td>25 (22; 26)</td>
<td>25 (23; 26)</td>
<td></td>
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<tr>
<td>MoCA</td>
<td>23.4 (22.0-24.8)</td>
<td>23.9 (22.4-25.4)</td>
<td>24.2 (22.7-25.7)</td>
<td></td>
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<td></td>
<td>25 (20; 26)</td>
<td>25 (22; 26)</td>
<td>25 (22; 26)</td>
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<tr>
<td>MoCA</td>
<td>22.6 (21.4-23.8)</td>
<td>23.7 (22.7-24.7)</td>
<td>24.1 (23.0-25.3)</td>
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<td></td>
<td>23(21; 24)</td>
<td>24 (21; 25)</td>
<td>25 (23; 25)</td>
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<tr>
<td>Variables are presented as mean (95% CI) (<em>first row</em>) and median (Q1; Q3) (<em>second row</em>).</td>
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</tbody>
</table>

Pairwise comparison

- baseline vs. post TAVI: *p*=0.001 vs. *p*=0.288 vs. *p*=0.029 vs. *p*=0.009
- baseline vs. 3 months: *p*<0.001 vs. *p*=0.192 vs. *p*=0.001 vs. *p*<0.001
- post TAVI vs. 3 months: *p*=0.270 vs. *p*=0.620 vs. *p*=0.381 vs. *p*=0.559
Table 2. Multivariate Regression Analysis.

<table>
<thead>
<tr>
<th></th>
<th>estimator b</th>
<th>95% confidence interval</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Logistic EuroSCORE, %</td>
<td>-1.106</td>
<td>-6.667 – 4.455</td>
<td>0.692</td>
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<tr>
<td>Mean transaortic gradient at baseline, mm Hg</td>
<td>3.979</td>
<td>0.197 – 7.760</td>
<td>0.040</td>
</tr>
<tr>
<td>Aortic atheroma thickness, µm</td>
<td>-0.395</td>
<td>-0.943 – 0.153</td>
<td>0.154</td>
</tr>
<tr>
<td>Carotid artery plaque burden, %</td>
<td>-0.887</td>
<td>-4.510 – 2.737</td>
<td>0.626</td>
</tr>
<tr>
<td>LAA velocity, cm/s</td>
<td>-1.171</td>
<td>-4.684 – 2.342</td>
<td>0.507</td>
</tr>
<tr>
<td>Prior cerebral ischemic event, n</td>
<td>-104.940</td>
<td>-321.794 – 111.915</td>
<td>0.336</td>
</tr>
<tr>
<td>Prosthesis’ size, 23 / 26 / 29 mm</td>
<td>18.396</td>
<td>-10.616 – 47.407</td>
<td>0.209</td>
</tr>
<tr>
<td>Administered heparin, IU</td>
<td>0.005</td>
<td>-0.023 – 0.034</td>
<td>0.709</td>
</tr>
</tbody>
</table>
patients porteurs de stimulateurs cardiaques et essai d’abolition de la fibrillation atriale par électrostimulation des oreillettes) a porté sur 2 580 patients âgés de plus de 65 ans qui étaient porteurs d’un stimulateur cardiaque bicavitaire et atteints d’hypertension artérielle, mais n’avaient aucun antécédent de FA. Des études électrophysiologiques non invasives ont été régulièrement pratiquées sur une période de deux ans dans un sous-groupe de 485 patients. Il n’a pas été relevé de différences entre les caractéristiques cliniques des patients chez lesquels des tachyarythmies atriales avaient été décelées par le stimulateur au cours de la première année et de ceux qui étaient demeurés indemnes de tels troubles. Chez les premiers, il a été noté un allongement des durées des ondes P déclenchées (153 ± 29 ms versus 145 ± 28 ms ; p = 0,046) et détectées (128 ± 46 ms versus 118 ± 25 ms ; p = 0,06) et une plus forte propension au développement de FA induites à l’occasion des études électrophysiologiques (23,5 % versus 13,6 % ; p = 0,03). Aucun différence n’a, en revanche, été objectivée entre les deux groupes de patients en ce qui concerne leurs temps de récupération sinusale corrigés à 90 battements/min (388 ± 554 ms versus 376 ± 466 ms ; p = 0,86), la durée de leur période réfractaire atriale efficace à 90 battements/min (250 ± 32 ms versus 248 ± 36 ms ; p = 0,70) et le raccourcissement de cette dernière en adaptation à la fréquence (14 ± 13 ms versus 12 ± 14 ms ; p = 0,11). Il n’a pas été identifié de différence significative entre les modifications du substrat électrophysiologique enregistrées au cours des deux ans chez les patients ayant présenté des tachyarythmies atriales et chez ceux en qui ont été demeurés indemnes.


Mots clés : fibrillation atriale ■ électrophysiologie ■ hypertension artérielle ■ stimulateurs cardiaques ■ remodelage

Les embolies cérébrales lors de remplacements valvulaires aortiques percutanés
Une étude par Doppler transcrânien
Phili"p Kahlert, MD ; Fadi Al-Rashid, MD ; Philipp Döttger, MS ; Kathrine Mori, MS ; Björn Plicht, MD ; Daniel Wendt, MD ; Lars Bergmann, MD, DESA ; Eva Kottenberg, MD ; Marc Schlamann, MD ; Petra Mummel, MD ; Dagny Holle, MD ; Matthias Thielmann, MD ; Heinz G. Jakob, MD ; Thomas Konorza, MD ; Gerd Heusch, MD ; Raimund Erbel, MD ; Holger Eggebrecht, MD

Contexte—Qu’il soit pratiqué par voie transfémorale ou transapicale, le remplacement valvulaire aortique percutané (RVAP) augmente le risque d’accident neurologique comparativement à l’approche chirurgicale. L’imagerie par résonance magnétique cérébrale a montré que la fréquence des lésions nouvelles, cliniquement silencieuses, secondaires à la migration de microemboles est plus élevée lors de l’emploi de cette technique ; pour autant, on ignore quelle est l’origine principale de ces microemboles et à quel temps de l’intervention ils sont le plus susceptibles d’être libérés.

Méthodes et résultats—Des RVAP ont été réalisées chez 83 patients par abord transfémoral (Medtronic CoreValve [MCVTF], n = 32 ; Edwards Sapien [ESTF], n = 26) ou par voie transapicale (ESTA, n = 25). Des explorations par écho-Doppler transcrânien ont été pratiquées avant, pendant et 3 mois après les RVAP en vue de rechercher les éventuels hypersignaux transitoires (HST) tenant lieu de marqueurs de la présence de microemboles. De tels HST ont été mis en évidence chez tous les patients pendant la période opératoire, principalement lors de la manipulation de la valve aortique calcifiée alors que le stent-valve était positionné et implanté. L’emploi de la prothèse ES expansible sur ballonnet a donné lieu à un nombre d’HST (moyenne 106,3 ± 131,6 ; p <0,001) et celui de la prothèse MCV auto-expansible sur ballonnet, les abords transfémorale et transapical ni entre les implants MCV et ES. Aucun HST n’a été observé lors de l’implantation dudit implant. En termes de fréquence de ces embolies, la migration de microemboles. De tels HST ont été observés par le stimulateur au cours de l’intervention, ainsi que lors du suivi à 3 mois. Dans le groupe traité par prothèses MCV, un accident vasculaire cérébral peropératoire majeur, ayant entraîné le décès du patient, et un autre de type mineur, qui avait totalement régressé au 3ème mois, ont été enregistrés.


Mots clés : rétrécissement valvulaire aortique ■ ischémie cérébrale ■ remplacement valvulaire aortique percutané ■ valves ■ écho-Doppler transcrânien