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# Embolic Cerebral Insults After Transapical Aortic Valve Implantation Detected by Magnetic Resonance Imaging

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**Objectives** This study assessed the rate of periprocedural embolic ischemic brain injury during transapical aortic valve replacement in 25 consecutive patients.

**Background** Transcatheter aortic valve implantation is rapidly being established as a new therapeutic approach for aortic valve stenosis. Although initial clinical results are promising, it is unknown whether mobilization and embolization of calcified particles may lead to cerebral ischemia.

**Methods** Twenty-five consecutive patients (10 men, 15 women, mean age: 81 ± 5 years, mean log EuroSCORE [European System for Cardiac Operative Risk Evaluation]: 32 ± 10%) scheduled for transapical aortic valve implantation were included. All patients received a baseline cerebral magnetic resonance imaging scan. The scan was repeated approximately 6 days after valve implantation. The magnetic resonance imaging studies included axial diffusion–weighted, $T_2$-weighted, fluid attenuated inversion recovery–weighted, and $T_2$ gradient echo sequences. Standardized assessment of the neurologic status was performed before aortic valve replacement and post-operatively.

**Results** Transapical aortic valve implantation was successfully performed in all patients. In 17 patients (68%), new cerebral lesions could be detected, whereas 8 patients showed no new cerebral insults. The pattern of distribution and morphology were typical of embolic origin. Despite the high incidence of morphologically detectable lesions, only 5 patients showed clinical neurologic alterations. Out of these patients, only 1 suffered from a permanent stroke.

**Conclusions** New embolic ischemic cerebral insults are detected in 68% of patients after transapical valve implantation. Clinical symptoms are rare and usually transitory. Larger trials will need to establish the clinical significance of asymptomatic ischemic lesions as well as the rate of ischemic events in patients undergoing transfemoral valve replacement. (J Am Coll Cardiol Intv 2010;3:1126–32)


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Catheter-based aortic valve implantation has been introduced as an alternative to conventional surgery for patients with high grade aortic stenosis in whom perioperative risk is assumed to be high due to comorbidities and age. Initial results are promising and an increasing number of centers are introducing this procedure into their clinical routine (1–7). Nevertheless, the clinical course of patients after catheter-based aortic valve implantation and predictors of outcome are incompletely understood.

During conventional aortic valve replacement, the calcified leaflets of the native aortic valve are meticulously excised before implantation of the valve prosthesis. The operative site is repeatedly flushed and deaired before blood flow is reinstituted. These measures are taken to avoid embolization of air or solid particles to minimize the risk of cerebral insults.

Catheter-based aortic valve replacement is performed while the heart is beating and circulation is not interrupted. Particles mobilized from the calcified native aortic valve during the procedure might therefore embolize into the cerebral circulation. Manipulations that contribute to the risk of embolization are the passage of the aortic valve with the guidewire, positioning of the valvuloplasty balloon, valvuloplasty itself, as well as positioning and implantation of the balloon-mounted valve prosthesis.

So far, neurologic sequelae of catheter-based aortic valve replacement were determined by documenting the incidence of clinically evident stroke (4–6). However, cerebral embolism may be clinically silent, but at the same time associated with impaired prognosis. Furthermore, the typical population undergoing the valve replacement procedure bears a very high risk of cerebral insults due to pre-existing comorbidities such as atherosclerotic alterations of the cerebral vasculature or atrial fibrillation. Based on the available data, the risk of cerebral embolization that is attributable to catheter-based aortic valve implantation is therefore difficult to quantify.

To detect the incidence of embolic cerebral insults related to transapical catheter-based aortic valve implantation, we performed pre- and post-procedural cranial magnetic resonance imaging (MRI) studies to detect new cerebral embolizations and correlated the findings to clinical neurological assessment.

## Methods

**Study population.** From December 2008 to November 2009, 45 consecutive patients who underwent transapical aortic valve implantation were consecutively screened for study inclusion. Eighteen patients had to be excluded (11 due to implanted permanent pacemakers or implantable cardioverter-defibrillator, 5 due to pre-operative hemodynamic instability, and 2 patients due to logistic reasons). A total of 27 patients received a baseline cerebral MRI scan before aortic valve implantation, which was repeated approximately 6 days (±2) after valve implantation. One of these patients died intraoperatively due to massive bleeding from a ruptured left ventricular apex, and another patient required implantation of a permanent pacemaker before the second MRI scan could be performed. Hence, 25 patients completed the study protocol. The mean age of the included patients was 81 ± 5 years. Ten candidates were men and 15 were women. Further patient characteristics are summarized in Table 1. Written informed consent was obtained from all patients.

**Clinical assessment.** Standardized clinical assessment of the neurologic status was performed before aortic valve replacement and daily during the post-operative period. The assessments were done by intensive care physicians. A neurologic complication was defined as any new cranial-nerve, motor, or sensory deficiency; reflex change; pyramidal sign; or mental alteration that arose within 48 h of aortic valve implantation.

**Transapical aortic valve implantation.** We performed transapical aortic valve implantation with the Edwards-SAPIEN aortic valve prosthesis (Edwards Lifesciences, Inc., Irvine, California). Via a transfemoral approach, a 6-F pigtail was placed in the ascending aorta. Access to the left ventricular apex was established via a left anterolateral mini-thoracotomy in the fourth to sixth intercostal spaces. The pericardium was incised and the apex was exposed by stay sutures. Two purse-string sutures were placed around the intended puncture site and an epicardial pacemaker lead adjacent to it. The left ventricle was then punctured with an 18-G needle (Intradyn 1.3 × 70 mm, Braun AG, Melsungen, Germany), and a guidewire (Angiodyn J-tip 0.035-inch, Braun AG, Melsungen, Germany) was advanced through the aortic valve into the ascending aorta. A 26-F sheath (Edwards Lifesciences, Inc.) was inserted into the left ventricle. A right Judkins angiographic catheter (JR4 6-F 100 cm 0.038-inch, Cordis Corp., Waterloo, Belgium) was used to guide the wire into the descending aorta. The guidewire was then changed to an Amplatz Extra Stiff guidewire (Cook Ltd., Limerick, Ireland) through the Judkins catheter to provide better support. Over the Amplatz wire, a 20-mm balloon (Edwards Lifesciences, Inc.) was placed in the aortic valve under fluoroscopic control and valvuloplasty of the aortic valve was performed under rapid ventricular pacing at a rate of 180 to 220 beats/min. The balloon was removed and the valve delivery system (Ascendra, Edwards Lifesciences, Inc.) with the balloon-mounted prosthesis (Edwards-SAPIEN, Edwards Lifesciences, Inc.) was introduced over the guidewire and the in-place 26-F sheath. The

**Abbreviations and Acronyms**

ARWMC = age-related white matter changes
FLAIR = fluid attenuated inversion recovery
FOV = field of view
MRI = magnetic resonance imaging
TE = echo time
TR = recovery time

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**Table 1. Pre- and Perioperative Patient Data**

| | |
|---|---|
| Study population, n | 25 |
| Women/men | 15/10 |
| Age, yrs | 81 ± 5 |
| Log EuroSCORE, % | 32.3 ± 10 |
| LVEF, % | 51 ± 14 |
| AVA, $cm^2$ | 0.8 ± 0.2 |
| History of atrial fibrillation, n | 15 |
| History of stroke, n | 6 |
| ACI stenosis >50%, n | 9 |
| Pre-operative serum creatinine, mg/dl | 1.25 ± 0.6 |
| Pre-operative NT-proBNP, U/l | 6,354 ± 9,571 |
| NYHA functional class, median (min; max) | III (I; IV) |
| Procedural time, min | 111 ± 30 |
| Cardiopulmonary bypass time, min | 0 |
| Post-operative ventilation, n | 2 |
| Size of implanted prosthesis, n | |
| 23 mm | 7 |
| 26 mm | 18 |

ACI = internal carotid artery; AVA = aortic valve area; LVEF = left ventricular ejection fraction; NT-proBNP = N-terminal pro–B-type natriuretic peptide; NYHA = New York Heart Association.

prosthesis was positioned under fluoroscopic guidance. During another period of rapid ventricular pacing, the optimal position of the valve prosthesis was confirmed by angiography and the valve was implanted by expanding the balloon. The final position and degree of paravalvular leakage were assessed by another angiography and by transesophageal echocardiography. Post-dilation of the valve was performed if necessary. The valve delivery system, the guidewire as well as the sheath, was removed from the heart during a final phase of rapid ventricular pacing. The puncture site and mini-thoracotomy were closed surgically, and the epicardial pacemaker wire as well as a pleural drain were left in place. The first 2 patients were transferred to the intensive care unit on ventilatory support. After the first 2 patients, the periprocedural protocol was changed, and all patients were extubated in the operating room directly after the procedure.

**MRI.** Magnetic resonance imaging was performed with 1.5-T systems (Magnetom Espree, Magnetom Sonata, Siemens Healthcare Sector, Erlangen, Germany) at a medium interval of 6 ± 12 days before and repeated 6 ± 2 days after aortic valve replacement. The imaging protocol included a diffusion-weighted single-shot spin-echo planar sequence with apparent diffusion coefficient map (diffusion gradient b values: 0, 500, and 1,000 $s/mm^2$; recovery time [TR]: 3,800 ms; echo time [TE]: 84 ms; slice thickness: 5 mm; matrix: 128 × 128; field of view [FOV]: 230 × 230), a fluid attenuated inversion recovery (FLAIR) sequence (TR: 8,430 ms, TE: 109 ms, inversion time: 2,500, slice thickness: 5 mm, matrix: 332 × 512, FOV: 173 × 240), a $T_2$-weighted sequence (TR: 5,520 ms, TE: 97 ms, slice thickness: 5 mm, matrix: 195 × 384, FOV: 180 × 230), and a $T_2$-weighted gradient echo flash sequence (TR: 858 ms, TE: 26 ms, slice thickness: 5 mm, matrix: 336 × 512, FOV: 201 × 230).

Diffusion-weighted MRI was performed to identify even small acute cerebral ischemias. Diffusion-weighted MRI is sensitive to changes in the mobility of water molecules. Lowered water mobility due to a shift of water from the extracellular to the intracellular compartment is an early event in the cascade of ischemic tissue changes. The regional decrease of diffusion is visible as hyperintensity on diffusion-weighted MRI images and as hypointensity on quantitative maps of the apparent diffusion coefficient. It occurs before $T_2$-weighted MRI shows any abnormality related to an increase in total water content in the infarction (8). The FLAIR- and $T_2$-weighted images were performed to show the infarct demarcation, the $T_2$-weighted flash sequence to differentiate possible hemorrhages, cavernomas or microbleedings (9). Image analysis was performed jointly by 2 experienced neuroradiologists who were blinded to all clinical data. Classification of pre-existing white matter lesions in the initial MRI, based on the $T_2$- and FLAIR-weighted images, was independently performed, according to the rating scale of Wahlund et al. (10). For analysis of diffusion-weighted MRI, the neuroradiologists were asked to determine the presence of any focal diffusion abnormalities in a pattern consistent with embolic lesions.

## Results

Of the 25 patients with catheter-based aortic valve implantation who were included in our investigation, none had focal diffusion abnormalities before aortic valve implantation. After the procedure, new focal diffusion abnormalities with a correlating signal reduction in the apparent diffusion coefficient map consistent with new embolic lesions were documented in 17 patients (68%). All lesions were also visible in the FLAIR- or $T_2$-weighted sequence, which is consistent with infarct demarcation. The maximum size of the lesions was up to 5 mm in 4 patients, up to 10 mm in 11 patients, and >10 mm in 2 patients. One of the latter patients had symptomatic major acute cerebellar and occipital infarcts. Thirteen patients had more than 1 lesion; 6 patients had more than 5 lesions in up to 5 vascular territories (see Fig. 1 for the involved vascular territories). More than 50% of lesions were localized in the posterior circulation involving the cerebellum, brain stem, and the vascular distribution of the posterior cerebral artery.

White matter signal abnormalities (age-related white matter changes [ARWMC]) are common alterations in asymptomatic elderly individuals, and typically appear as periventricular focal or confluent signal hyperintensity on $T_2$-weighted images on MRI. Strongest risk factors are

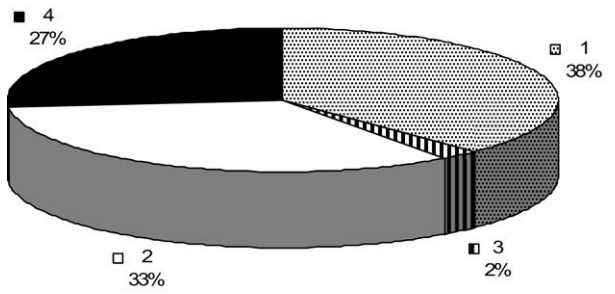


**Figure 1. Distribution of Embolic Cerebral Insults in the Vascular Territories**

Chart 1 shows the distribution of the acute embolic cerebral insults of the 17 affected patients in the vascular territories: 1 = middle cerebral artery, 2 = posterior cerebral artery, 3 = anterior cerebral artery, 4 = cerebellum/brainstem. If both territories, for example, of the middle cerebral artery in 1 patient were affected, 2 vascular territories of the middle cerebral artery were counted.

hypertension and age, both of which are common in our study population. Mechanisms for development of such white matter lesions are not yet fully understood. They may include degeneration of myelin and axons or infarctions of perforating small arteries (even though embolic infarcts more commonly involve the cortex). According to the rating scale of Wahlund et al. (10), we classified pre-existing ARWMC of the investigated patients in the FLAIR- and $T_2$-weighted sequences to detect a possible correlation of the observed acute infarctions and pre-existing white matter changes.

All 25 patients had focal white matter lesions that corresponded at least to a score of ARWMC 1, and no patient showed distinct, confluent white matter lesions that would correspond to a score of ARWMC 3. However, we could not observe significant differences in the presence of pre-existing white matter changes between patients with and without acute cerebral insults after aortic valve implantation. Of the 17 patients with acute infarcts, 6 had a score ARWMC 2 (35%), corresponding to beginning confluence of white matter lesions. Of 8 patients without acute lesions, 3 had a score of ARWMC 2 (38%).

In 5 patients, neurologic alterations were detected by clinical assessment in the post-operative course. One patient suffered from symptomatic major acute cerebellar and occipital infarcts resulting in a persistently reduced awareness. This was caused by more than 5 new lesions with a maximum size of 41 mm (Fig. 2). Another patient developed transitory diplopia. A corresponding single lesion of 6-mm diameter was detected in the brain stem. Three patients experienced transitoric psychotic syndromes. Although 2 of them had more than 5 new cerebral lesions of at least 7-mm diameter (Fig. 3), in 1 case, no new embolic lesions were detected by MRI.

The number and size of new affections detected in MRI was not directly related to clinical presentation. Most patients even with multiple lesions did not show any clinical signs of neurologic impairment (Fig. 4).

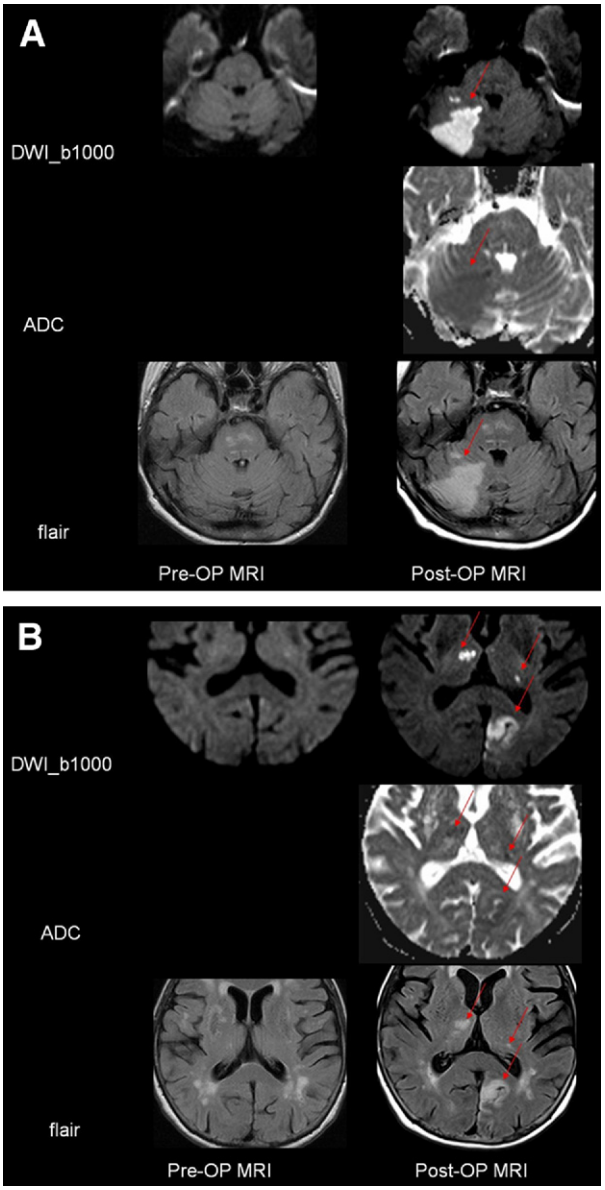


**Figure 2. MR Images of a Patient Who Suffered From a Permanent Stroke**

Pre- and post-operative magnetic resonance images of corresponding planes of a 79-year-old patient who suffered from a permanent stroke after transapical aortic valve implantation. **(A)** There is a 4.1 × 2.7-cm lesion in the right cerebellum **(red arrows)**. **(B)** There are additional lesions in the perfusion territories of the medial cerebral arteries bilaterally and left posterior cerebral artery **(red arrows)**. ADC = apparent diffusion coefficient; b1000 = low T2 weighting; DWI = diffusion-weighted magnetic resonance imaging; FLAIR = fluid attenuated inversion recovery; MR = magnetic resonance; MRI = magnetic resonance imaging; OP = operative.

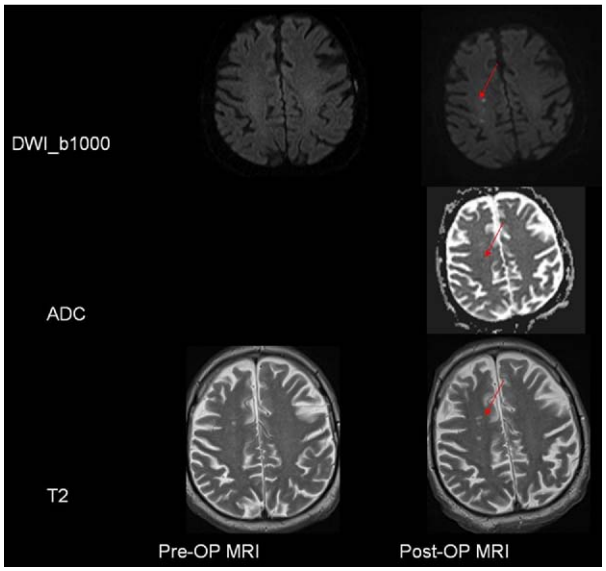


**Figure 3. MR Images of a Patient With a Transitoric Psychotic Syndrome**

Pre- and post-operative magnetic resonance images of corresponding planes of a 76-year-old patient who presented with a transitoric psychotic syndrome after transapical aortic valve implantation. There is a 4.1 × 2.7-cm lesion in the right cerebellum **(red arrows)** and an example of an embolic lesion in the perfusion territory of the right medial cerebral artery **(red arrow)**. Five lesions were detected in this patient T2 = $T_2$-weighted imaging; other abbreviations as in Figure 2.

## Discussion

With MRI, we analyzed the incidence of cerebral insults related to transapical aortic valve implantation. In a group of 25 patients, 17 showed new focal lesions. Two potential mechanisms of these cerebral alterations have to be considered: 1) general cerebral hypoperfusion; and 2) embolization into the cerebral circulation.

We did not see any infarctions affecting the border zones of the major cerebral arteries that would indicate relevant hypotensive periods. Even in patients with a stenosis of the internal carotid artery of more than 50% such alterations were not detected. Therefore, hypotension due to repeated rapid ventricular pacing did not lead to detectable morphologic changes of the brain in our study population.

Most patients with new focal lesions had multiple defects. Furthermore, the lesions were almost equally distributed in all major cerebral vascular territories except for the anterior cerebral artery. The pattern of distribution and the morphology thus prove that the new lesions were most likely of embolic origin.

It has been reported that repeated manipulations at the aortic valve, the aortic root and the ascending aorta lead to embolization of particles into the cerebral perfusion. In a previous study using MRI, the passage of a high grade aortic stenosis with a guidewire alone during diagnostic catheterization resulted in a cerebral embolization rate of 22% (11). No clinical neurologic alterations were reported from these patient cohorts.

Clinical neurologic symptoms after catheter-based aortic valve replacement are described at a rate from 0% to 10% (4,6,12–14). In our series, 5 of 25 patients showed clinical signs of a cerebral event. Except for 1 case, the symptoms resolved completely.

Clinically relevant focal neurologic events were rather rare compared with the overall number of cerebral insults documented by MRI. The incidence of clinically relevant neurological events seems not to be directly related to the detection of cerebral lesions. In our investigation, 1 patient presented with a transient ischemic attack. In the MRI scan, a single lesion was documented. In contrast, several patients had multiple lesions but did not show symptoms.

Age and extent of atherosclerotic disease were described as risk factors for perioperative strokes in cardiac and endovascular aortic surgery (15,16). Our patients were older (81 years vs. 65 years) and presented with more severe comorbidities (log EuroSCORE [European System for Cardiac Operative Risk Evaluation]: 32% vs. 5%) than those in the previously mentioned studies. This might explain the higher rate of cerebral lesions irrespective of the procedure performed.

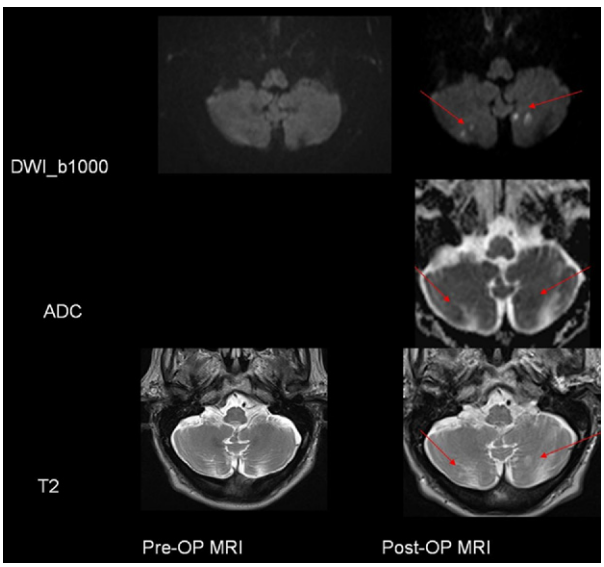


**Figure 4. MR Images of a Patient Without Clinical Neurological Symptoms**

Pre- and post-operative magnetic resonance images of corresponding planes of an 87-year-old patient who did not show clinical neurological symptoms after transapical aortic valve implantation. The figure shows an exemplary plane with bilateral cerebellar lesions **(red arrows)**. More than 5 new lesions with a maximum size of 12 × 7 mm were detected by magnetic resonance imaging. Abbreviations as in Figures 2 and 3.

In 2 studies evaluating the frequency of small brain lesions after conventional valve surgery by diffusion-weighted MRI, 43% and 47% of the patients showed alterations post-operatively (17,18). One study revealed only transitory psychotic syndromes in 13% of the patients; in the other study, permanent strokes occurred in 5% of the study population. Patients with a history of stroke or stenosis >70% of the carotid artery were excluded. The average age was 65 years and the mean estimated log EuroSCORE was 5%. The overall rate of neurological alterations in a population of octogenarians (median additive EuroSCORE: 6) who underwent conventional cardiac surgery (not differentiating valve surgery and coronary bypass surgery) reported by Ngaage et al. (19) was 13%.

Direct comparison with data from patients after conventional aortic valve replacement is difficult as patient populations are likely to strongly differ from each other. Specific data on high-risk candidates for aortic valve replacement is lacking. Several patients who are now candidates for catheter-based procedures probably were formerly not referred to surgery (20).

In our study, all patients uniformly underwent transapical aortic valve implantation. In contrast to transfemoral procedures where the valve delivery system is advanced through the aorta, manipulations in the ascending aorta and aortic arch are limited to the placement of guidewires and the angiographic catheter during transapical aortic valve implantation. This setting provides the unique opportunity to specifically study the risk of embolization caused by manipulations located in the aortic valve. In a study by Ghanem et al. (21), in almost 73% (compared with 68% in our population) of the patients, new cerebral lesions were detected after transfemoral aortic valve implantation. Similar to our results, permanent neurologic impairment was relatively rare (3.6%). Although one may tend to attribute the increased rate of cerebral embolization to more intense manipulations in the ascending aorta and aortic arch during the transfemoral approach, the difference may simply be caused by statistical variations due to the relatively small sizes of the patient groups in both studies. Moreover, 2 different types of valve prostheses were implanted in the 2 studies.

Our MRI protocol permitted the detection of embolic lesions with a high sensitivity. Although it seems probable that they were caused by calcified particles that were mobilized from the aortic valve during the procedure, the morphology of the lesions would not differ if they were caused, for example, by air or thrombotic material. Therefore, we cannot finally reveal the mechanism that results in cerebral lesions. As mentioned before, a variety of risk factors attributed to the incidence of stroke are described.

Therefore, larger trials will have to establish the clinical significance of our findings, especially with regard to a comparison of antegrade and retrograde approach of catheter-based aortic valve implantation.

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**Key Words:** cerebral embolism ■ MRI ■ TAVI ■ transapical aortic valve implantation ■ transcatheter aortic valve implantation.