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## Cerebral embolism in transcatheter aortic valve implantation (TAVI): Mechanisms and strategies for prevention

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Transcatheter aortic valve implantation (TAVI) has now widely been accepted as a treatment option for high-risk patients with severe symptomatic aortic stenosis. Recent clinical trials and current registries indicate, at follow-up of 24 - 30 months, excellent outcome and low mortality rates, which are similar to surgical aortic valve replacement in comparable patient cohorts. (1,2) Nevertheless, TAVI may be associated with severe adverse cerebrovascular events which may massively impair patients' post-procedural quality of life.

In a recent prospective registry series of 400 patients undergoing transapical or transfemoral TAVI, periprocedural stroke occurred in 2.8%. (3) Periprocedural stroke may be due to prevalent cerebro-vascular occlusive disease, intraprocedural embolic or hemodynamic ischemia, or to peri- and post-procedural cardioembolic events due to atrial fibrillation. (4) In a recent risk analysis of the Bern TAVI registry, abnormally low body mass index, prior stroke, and presence of atrial fibrillation at baseline contribute to the risk of postprocedural stroke. (5) Hence, TAVI patients are exposed to an increased risk of all-cause and cardiovascular mortality. (6) Apart from stroke, new but clinically silent ischemic lesions have been reported in up to 84% of TAVI recipients in postinterventional diffusion-weighted magnetic resonance imaging (DW-MRI) studies. (7) When investigating TAVI procedural phases separately by using transcranial Doppler (TCD)

ultrasonography, the incidence of solid or gaseous embolic events was higher with the transfemoral access route and the deployment of self-expandable prostheses. (8,9) In this regard, manipulations on the calcified aortic valve (e.g. dense catheter traffic, prosthesis deployment, postimplantation balloon dilatation) and prolonged friction between the rigid hardware and the calcified aortic root due to gradual prosthesis release without rapid pacing, i.e., with ejection maintained, appear to be associated with more high-intensity transient signals (HITS), reflecting embolic load to the brain. (8-12)

The clinical significance of HITS has not yet been unequivocally resolved. Obviously, the majority of HITS has no clinically apparent sequelae, but neither do so many lesions on DW-MRI. This appears to be more a sensitivity problem of neurological and neurocognitive testing than of clinical relevance. Nevertheless, in view of current indications for TAVI and the respective patient population, it is highly questionable whether a completely HITS-free TAVI procedure can ever be designed. However, there is increasing evidence for postprocedural cognitive decline with higher HITS frequency (13,14) and associated with it, substantial costs in health care. Therefore all efforts should be made to reduce HITS during TAVI.

Currently, several embolic protection devices are in clinical evaluation. Future prevention strategies also include less traumatic delivery systems, as well as new prostheses

with better sealing and less need for redilatation and repositioning. (10) Likewise a heart-team based careful patient and device selection, and close intraprocedural collaboration within the treatment team appear to be an effective approach to optimize outcome. (15)

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