The recent success of transcatheter aortic valve implanta-
tion (TAVI) has been associated with a heightened
awareness of the potential risks, particularly stroke. Recent
meta-analyses report 30-day stroke rates of 3% to 4%,1,2 and
diffusion-weighted MRI studies have revealed new, clinically
silent, cerebral lesions in 68% to 84% of patients undergoing
TAVI.3–5 Although the majority of patients undergoing TAVI
benefit greatly in terms of quality of life and functional status,
concerns about neurological disability remain.

Transcranial Doppler (TCD) has been extremely helpful in
clarifying the central role of cerebral embolism as the major
cause of intra procedural stroke. As Kahlert et al4 report,
cerebral microembolism occurs in essentially all patients
undergoing TAVI. High-intensity transient signals, largely
reflecting particulate emboli, are routinely detected during
many invasive cardiac procedures. Nevertheless, it has been
difficult to demonstrate that high-intensity transient signals
correlate directly with stroke, and attempts to correlate
high-intensity transient signals with biomarkers of neuronal
injury have been inconsistent.3,6

Importantly, TCD has provided considerable information
about the relative contributions of the various elements of
the TAVI procedure to the risk of cerebral embolization.

1. Initial passage of wires and catheters into the ascending
aorta. This seems relatively benign in the great majority
of patients.
2. Crossing of the diseased native valve with a wire and
diagnostic catheter carries a significant but modest risk of
microembolic TCD signals and new MRI lesions.7 Unless
necessary, crossing a diseased native valve should be
avoided, and when necessary, crossing should be accom-
plished with as much care and skill as possible.4,7
3. Balloon valvuloplasty disrupts both the endothelial
covering and underlying friable calcific material within
the diseased valve. Disruption of the valve is progres-
sive; least with balloon dilation, greater yet with repeated
or more aggressive dilation, even greater with expansion of the stent frame, and greatest when post-
implant balloon dilation is used.4
4. Passing large-diameter, relatively stiff prosthesis delivery
catheters around the aortic arch is associated with a
relatively small atheroembolic risk.
5. Inserting and positioning the prosthesis within the
native valve. TCD suggests that the greatest risk with
prosthesis delivery is when the prosthesis is actually
forced across the diseased, calcified native valve.4
Factors involved include the diameter, stiffness, track-
ability, and crossing profile of the prosthesis delivery
catheter.
6. Prosthesis expansion. As the stent frame of the prosthesis expands it displaces and crushes the diseased native
valve. This is a major source of microemboli.8
7. Prosthesis redilatation, recapture, repositioning, or re-
moval. TCD routinely demonstrates microemboli.

It seems that the more stenotic and calcified the diseased
aortic valve, the more likely it is that manipulating the valve
will be associated with TCD microembolic events and with
neurological events.4,9 One pattern seems clear, that the
highest risk of microemboli is during manipulation of the
aortic valve itself, particularly when this involves the bulky
prosthesis: crossing, positioning, expansion, and postimplant
manipulation.4,8,10

Does Access Affect Stroke Risk?
Kahlert et al9 found no overall difference in the rates of
cerebral microemboli with the transfemoral and transapical
approaches to TAVI. It has been suggested that an antegrade
approach to the aortic valve (transvenous or transapical) with
easier entry into the inflow of the diseased native valve and
minimal manipulation of the aortic arch might reduce the risk
of stroke. Although a few small series supported this hypoth-
thesis, larger series generally did not.9,11 Rodés-Cabau found no
difference with cerebral MRI when transapical and trans-
arterial approaches were compared.4,8,12 However, 2 recent
meta-analyses did suggest a lower stroke risk with transapical
TAVI. Comparing transapical and transarterial access with
the balloon expandable SAPIEN valve Eggebrecht found a
neurological event rate (stroke/transient ischemic event) of
2.7% versus 4.2%.1 Similarly, Jilaihawi found a stroke rate of
2.2% versus 3.4%.13 In neither analysis was the lower stroke
rate with the transapical approach statistically significant,
although a higher risk of mortality with the transapical approach
was. There may be a small advantage to transapical antegrade
access to the aortic valve in terms of neurological events,
however any such benefit is small and overshadowed by other
concerns.11 We know little about embolic risk with the transax-
illary or transaortic approaches, although it seems unlikely that
these offer a particular advantage.

Does Transcatheter Valve Type Alter
Stroke Risk?
It seems reasonable to question whether there are differences
in cerebral embolic risk with the 2 most commonly used
transcatheter valves: CoreValve (Medtronic Inc., Minneapolis, MN) and the Edwards SAPIEN valve (Edwards Lifesciences Inc., Irvine, CA). The manufacturers of both devices have steadily reduced the diameter of their delivery catheter systems. Early catheters were 24 French (≈8 mm), whereas current systems are routinely 18 French (≈6 mm), and even smaller systems are becoming available. Both manufacturers have also improved the crossing profile of their devices by incorporating smooth tapered nosecones. The CoreValve system has incorporated a sheathed delivery system that covers the stent frame during delivery, whereas the SAPIEN system has incorporated an atraumatic and smooth catheter profile with the ability to actively steer through the arch and native valve. Newer entries to the field can be expected to meet these standards.

Kahlert et al found similar rates of TCD-detected embolic events with implantation of the CoreValve and SAPIEN prosthesis. Importantly, however, the timing of embolic events differed. The higher profile unsheathed SAPIEN prosthesis caused more events during crossing the stenotic native valve and positioning than the lower profile sheathed CoreValve prosthesis. In contrast, CoreValve caused more events during deployment, possibly as a result of the unsheathed stent frame acting, in the words of the authors, “in a grater-like fashion scraping calcific debris from the native valve.” Additional embolic events were routinely observed with repositioning. Also using TCD, Erdoes et al found significantly more embolic events with CoreValve than with SAPIEN during deployment and postimplant interventions such as postdilation and repositioning.

Evaluating stroke risk is limited by variable ascertainment and nonstandardized definitions. In meta-analyses Eggebrecht compared neurological events (stroke/transient ischemic event) and Jilaihawi compared stroke rates after transarterial TAVI with CoreValve or SAPIEN prostheses. Thirty-day event rates were 3.1% versus 4.2% and 1.8% versus 3.4%, respectively (differences not significant). Importantly, the SAPIEN valve used incorporated the older, high-profile 22–24 French delivery system, not the lower profile SAPIEN XT or even lower profile SAPIEN 3 system.

**Postimplant Interventions**

TCD studies have shown that cerebral microemboli are most common at the time of interaction of the prosthesis with the native valve, specifically during positioning and expansion. However, we have less information about postimplant interventions. Balloon dilation after prosthesis implantation is commonly associated with embolic events as detected by TCD, although this has not been well quantified. The Quebec city group reported an increase in neurological events with postdilation from 11.9% to 2.0% (P = 0.006). Transient expansion and recoil of a metallic stent frame within a partially disrupted native valve might be a particularly efficient way to generate embolic particles, although the prosthesis itself may provide some embolic protection. Implant strategies that reduce the need for postdilation may be helpful to minimize stroke.

A unique characteristic of self-expanding valves is the potential to recapture, reposition, or remove a poorly positioned prosthesis. However, forcefully withdrawing or resheathing a prosthesis is, not surprisingly, routinely associated with TCD embolic events. It seems reasonable to suggest that an optimal strategy to minimize stroke would incorporate ideal initial prosthesis positioning with minimal requirements for repeat manipulation.

**Embolic Protection**

Three devices designed to protect the cerebral circulation from embolic particles are currently undergoing clinical evaluation, with many more anticipated. The transradial Embrella device (Edwards Lifesciences Inc., Irvine, CA) and the transfemoral Keystone device (Keystone Heart Inc., Herzliya, Israel) deflect emboli away from the cerebral and toward the peripheral circulation. The transradial Claret CE Pro device (Claret Medical Inc., Santa Rosa, CA) filters and captures embolic material, with >one half of cases in a small series having evidence of particulate on filter removal. Although embolic protection devices may be feasible, benefit has not been established.

**Future Directions**

Stroke will always be a concern with invasive aortic valve procedures. This should not overshadow the potential for improved duration and quality of life that can be achieved. Still, the risk of cerebral embolism and stroke must be addressed. There is much room for improvement in delivery catheters and prostheses that allow for more atraumatic, controlled, and accurate implantation, better sealing, and less need for repeat manipulation. Increased experience and better technique will help. Embolic protection devices are appealing but as yet unproven.

**Disclosures**

Dr Webb is a consultant to Edwards Lifesciences.

**References**


**Key Words:** Editorials / stroke
Cerebral Embolization During Transcatheter Aortic Valve Implantation
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Circulation. 2012;126:1567-1569
doi: 10.1161/CIRCULATIONAHA.112.136796
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2012 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/126/13/1567

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