Cerebral protection device in TAVR, based on pre cta brain will reduce stroke events

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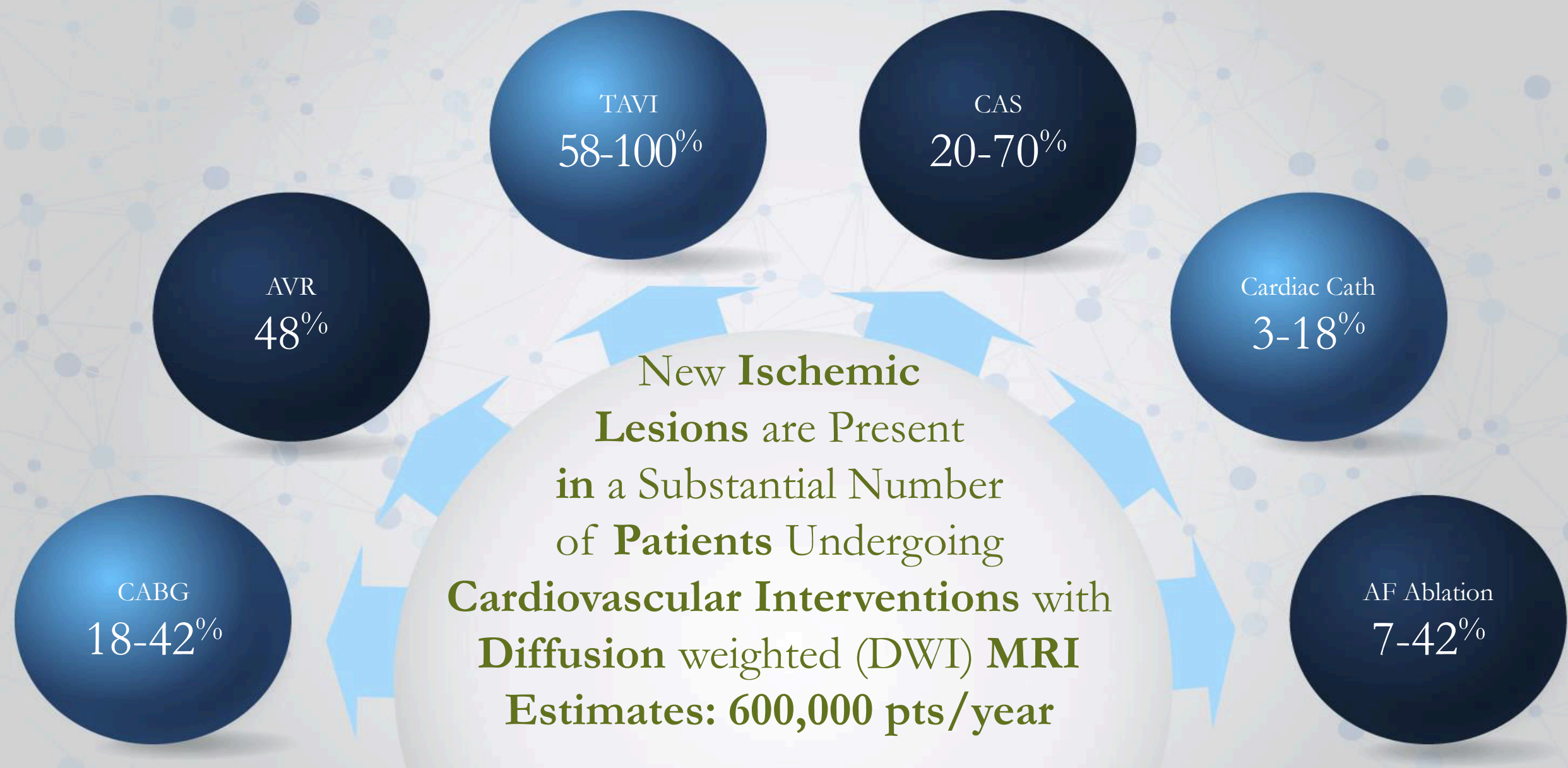


Clinical Consequences and Brain Injury

Gress D, J Am Coll Cardiol. 2012 Oct 23;60(17):1614-6

Proposed Standardized Neurologic Endpoints in Cardiovascular Clinical Trials [NeuroARC]

Framework on how to assess, measure and classify neurologic endpoints associated with cardiovascular procedures

International Multi Stakeholder Consensus. Chairs: Baumbach, Lansky, Mack, Messé

Interventional/Structural/ CT Surgery	Neurology/Neuroradiology/ Neuropsychology/NINDS	FDA/ARC/Pathology
Andreas Baumbach	Kevin Abrams	**FDA**
John Forrest	Michel Bilello	Andrew Farb
David Holmes	Adam Brickman	Nicole Ibrahim
Susheel Kodali	Jeffrey Browndyke	John Laschinger
Alexandra Lansky	Karen Furie	Carlos Pena
Axel Linke	David Greer	Bram Zuckerman
Raj Makkar	Daryl Gress	**Academic Research Consortium (ARC)**
Jeffrey Moses	Ronald Lazar	Donald Cutlip
Cody Pietras	Steven Messé	Gerrit-Anne van Es
Jeffrey Popma	Claudia Moy	Mitch Krucoff
Bernard Prendergast	Nils Petersen	Roxana Mehran
Joachim Schofer	Ola Selnes	**Pathology and Regulatory**
Arie P. Kappetein	Michael Dwyer	Semih Oktay
Michael Mack	Szilard Voros	Renu Virmani
	Bart van der Worp	

NeuroloArc applies to all CV trials

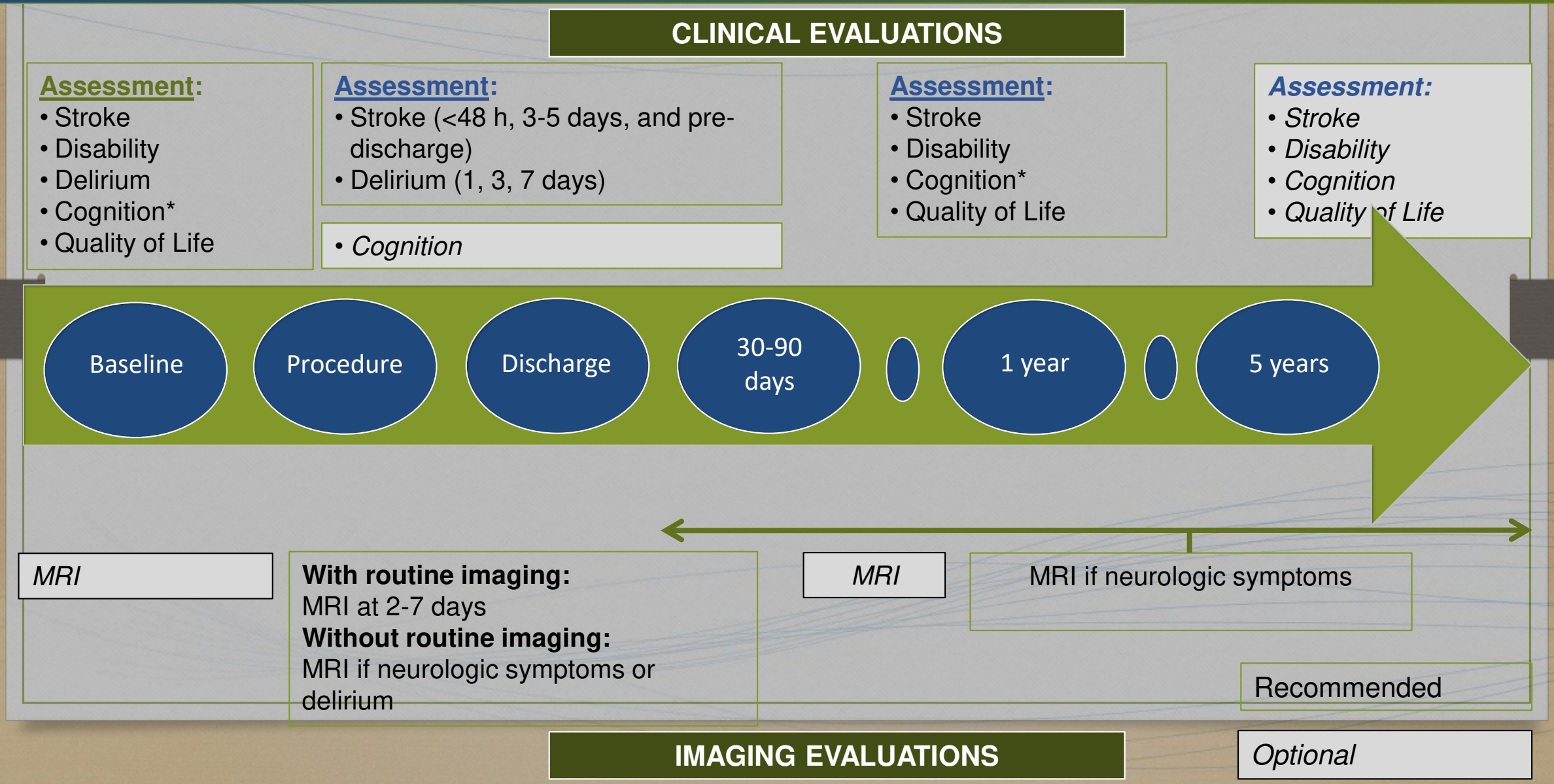
Neurologic evaluation and endpoints should be tailored to the procedure/device category

CATEGORY I **Primary** Procedural Safety Measure	CATEGORY II Primary Procedural Efficacy Measure	CATEGORY III **Primary** Procedural Safety, Long-term Efficacy Measure
Devices with inherent iatrogenic embolic risk • Surgical cardiac procedures (valve replacement, CABG, dissection, aneurysm repair) • Adjunctive pharmacology	**Devices designed to prevent iatrogenic or spontaneous acute neurologic injury** • Neuroprotection device • Cerebral temperature management devices	**Devices with inherent iatrogenic embolic risk and designed for prevention of spontaneous long-term risk** • Atrial Fibrillation Ablation • PFO or LAA closure devices

NeuroARC Definitions and Classification Relevant to Patients, Comprehensive, Practical

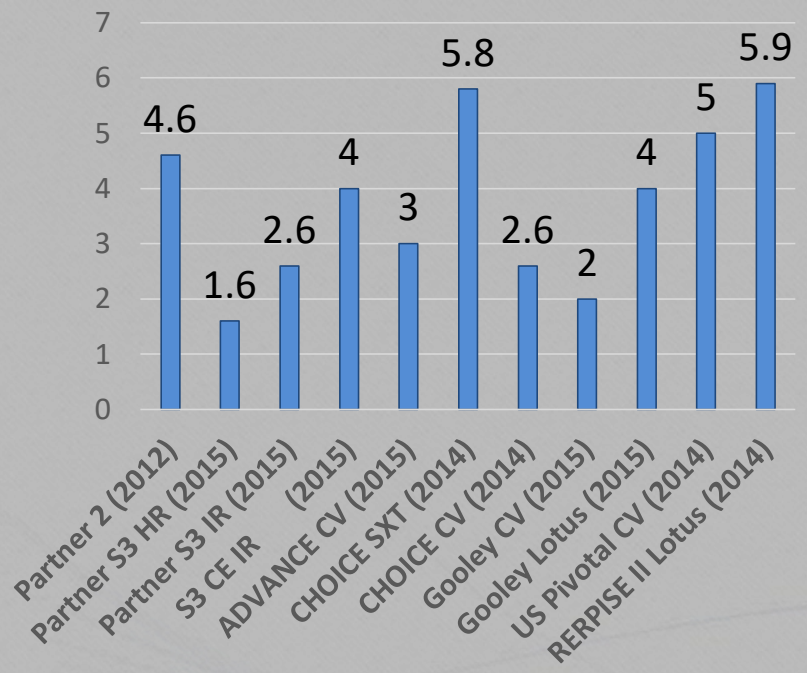
Type 1: Overt CNS Injury (Acutely Symptomatic)		
Type 1a	**Ischemic Stroke**	Focal or multi-focal vascular territory Symptoms ≥24 hours or until death or Symptoms <24 hours with neuroimaging confirmation
Subtype 1aH: Ischemic Stroke with Hemorrhagic conversion		**Class A**: Petechial Hemorrhage **Class B**: Confluent Hemorrhage (with space occupying effect)
Type 1.b	**Intracerebral Hemorrhage**	Symptoms (focal or global) caused by an intraparenchymal or intraventricular bleed
Type 1.c	**Subarachnoid Hemorrage**	Symptoms (focal or global) caused by a subarachnoid bleed
Type 1.d	**Stroke, not otherwise specified**	Symptoms ≥24 hours or until death, without imaging
Type 1.e	**Hypoxic-Ischemic Injury**	Global neurologic symptoms due to diffuse brain injury attributable to hypotension and/or hypoxia
Type 2: Covert CNS Injury (Acutely Asymptomatic brain injury detected by NeuroImaging)		
Type 2.a	**Covert CNS Infarction**	Acutely asymptomatic focal or multi-focal ischemia, based on neuroimaging
Subtype 2aH: Ischemic Stroke with Hemorrhagic conversion		**Class A**: Petechial Hemorrhage **Class B**: Confluent Hemorrhage (with space occupying effect)
Type 2.b	**Covert Cerebral Hemorrhage**	Neuroimaging or Acutely asymptomatic CNS hemorrhage on neuroimaging that is not caused by trauma
Type 3: Neurologic Dysfunction without CNS Injury (Acutely Symptomatic)		
Type 3.a	**Transient Ischemic Attack (TIA)**	Symptoms <24 hours with no evidence of acute infarction by neuroimaging
Type 3.b	**Delirium without CNS injury**	Transient non-focal (global) neurologic signs or symptoms (variable duration) without evidence of cell death by pathology or neuroimaging

NeuroARC Recommended Assessments: Clinical, Functional, Anatomic Correlations

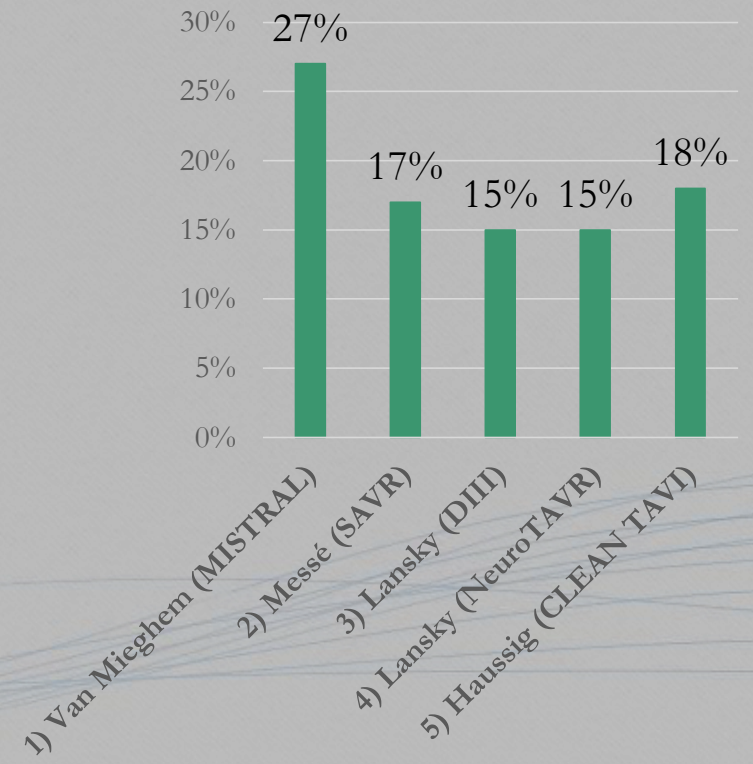


Stroke is common and underreported after TAVR

Reported stroke rates range from 1.6%-5.9% in TAVR trials



- **Stroke rate is 15-27% after TAVR by current AHA/ASA definitions (tissue-based)**



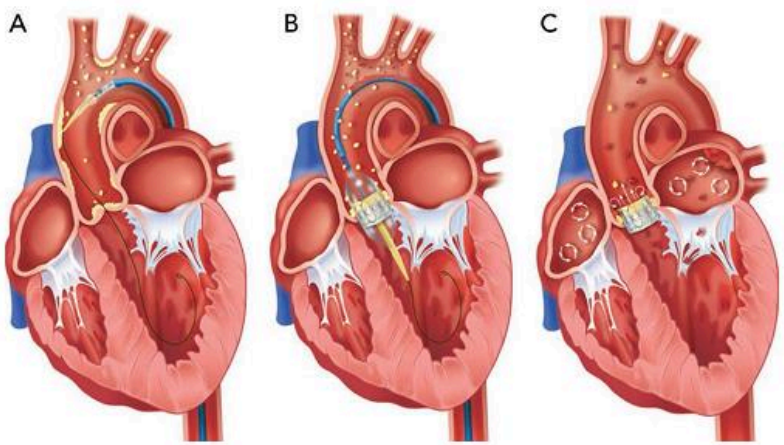
[1]Van Mieghem NM, EuroIntervention. 2016;12:499. [2]Messe S, Circulation. 2014;129:2253. [3]Lansky AJ, Eur Heart J. 2015; 36:2070.
[4]Lansky AJ, PCR London Valves 2015, AJC 2016 (in press). [5]Haussig S, JAMA. 2016;316:592.

Complications

- Migration of Valve
- Access site
- AV block
- Shock
- Bleeding
- Urgent SAVR
- Coronary artery closure

Disruption of calcium, plaque and thrombus

Figure 1: Aetiology of Embolisation Secondary to Transcatheter Aortic Valve Implantation



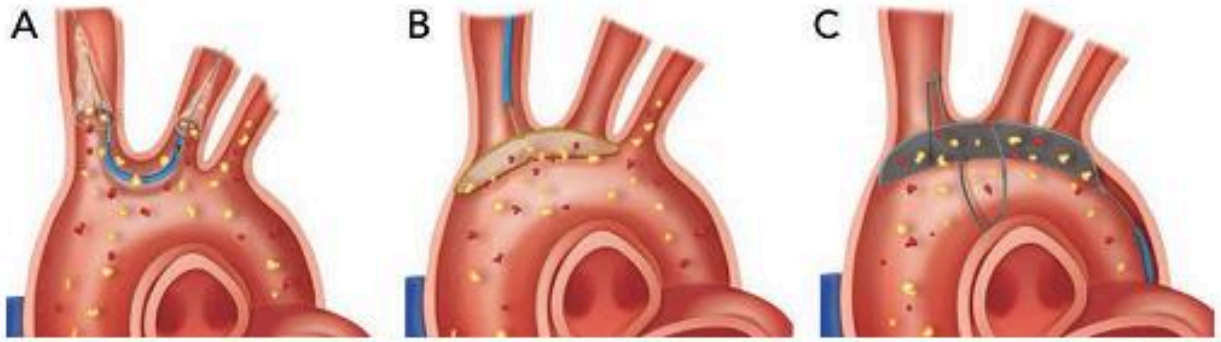
(A) and (B) Acute embolisation. Disruption of calcific/atheromatous plaques and valve/vascular tissue associated with: (A) the passage of guide wires and large-bore catheters and during valvuloplasty and device delivery; and (B) device positioning and implantation with thrombus forming on the catheter. (C) Subacute embolisation. Persistent nidus of calcium on the native valve leaflets provides a source for further calcific embolisation: thrombus formation secondary to structural changes to native leaflets, the presence of a prosthetic device and altered rheology attributable to both the apposition of native leaflets to the aortic wall and atrial fibrillation. Adapted from Fanning et al., 2014[25] with permission from Wolters Kluwer Health, Inc, © 2014.

Prevent Stroke by Filter device

- Claret device
- Embrella
- Trigaurd

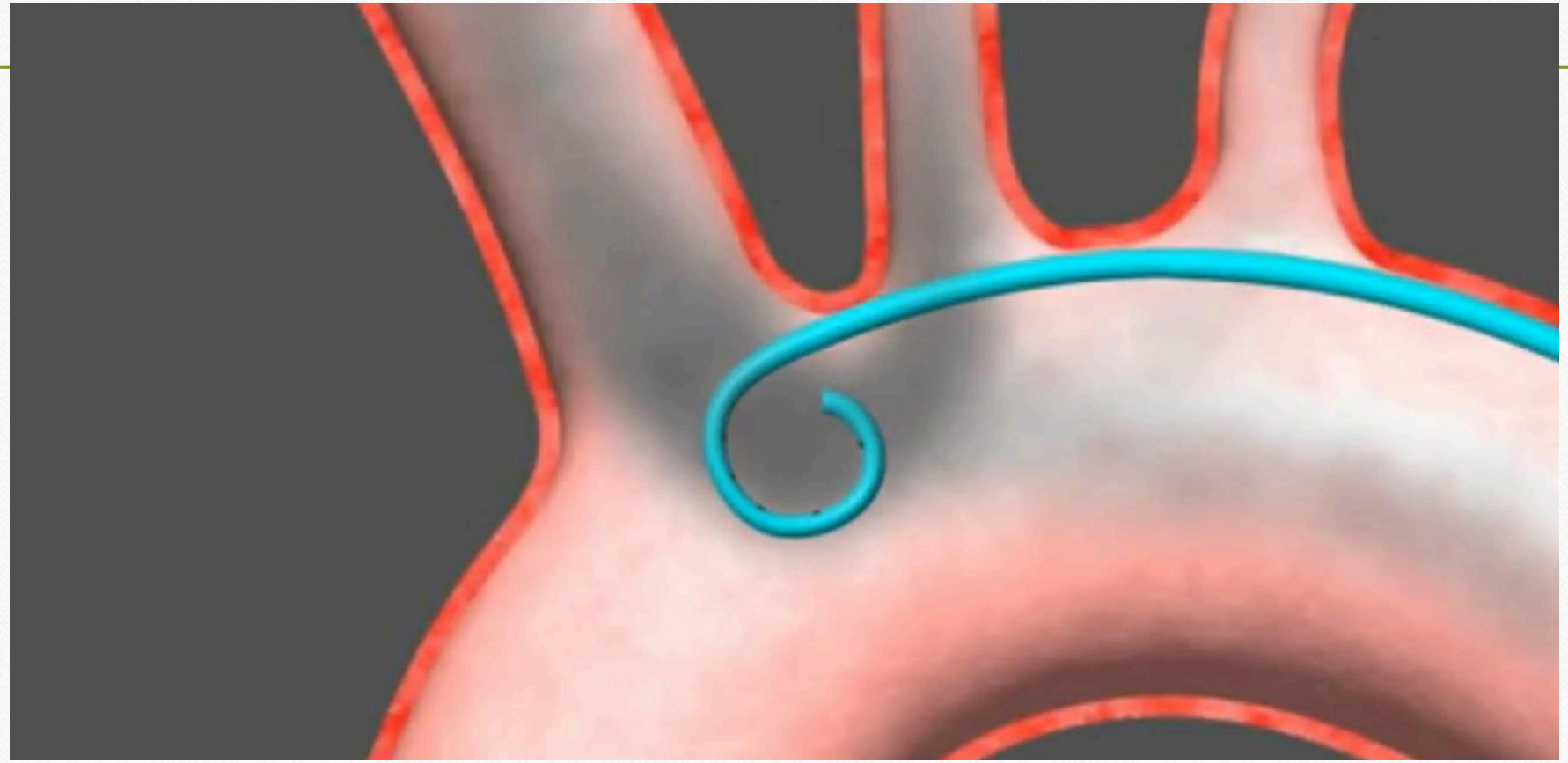
Various embolic protection devices

Figure 3: Embolic Protection Devices



(A) Montage™ 2 Capture Device (Claret); (B) Embrella Embolic Deflector System (Edwards Lifesciences); and (C) TriGuard™ Cerebral Protection Device (Keystone Heart). Adapted from Fanning et al., 2014[35] with permission from Wolters Kluwer Health, Inc, © 2014.

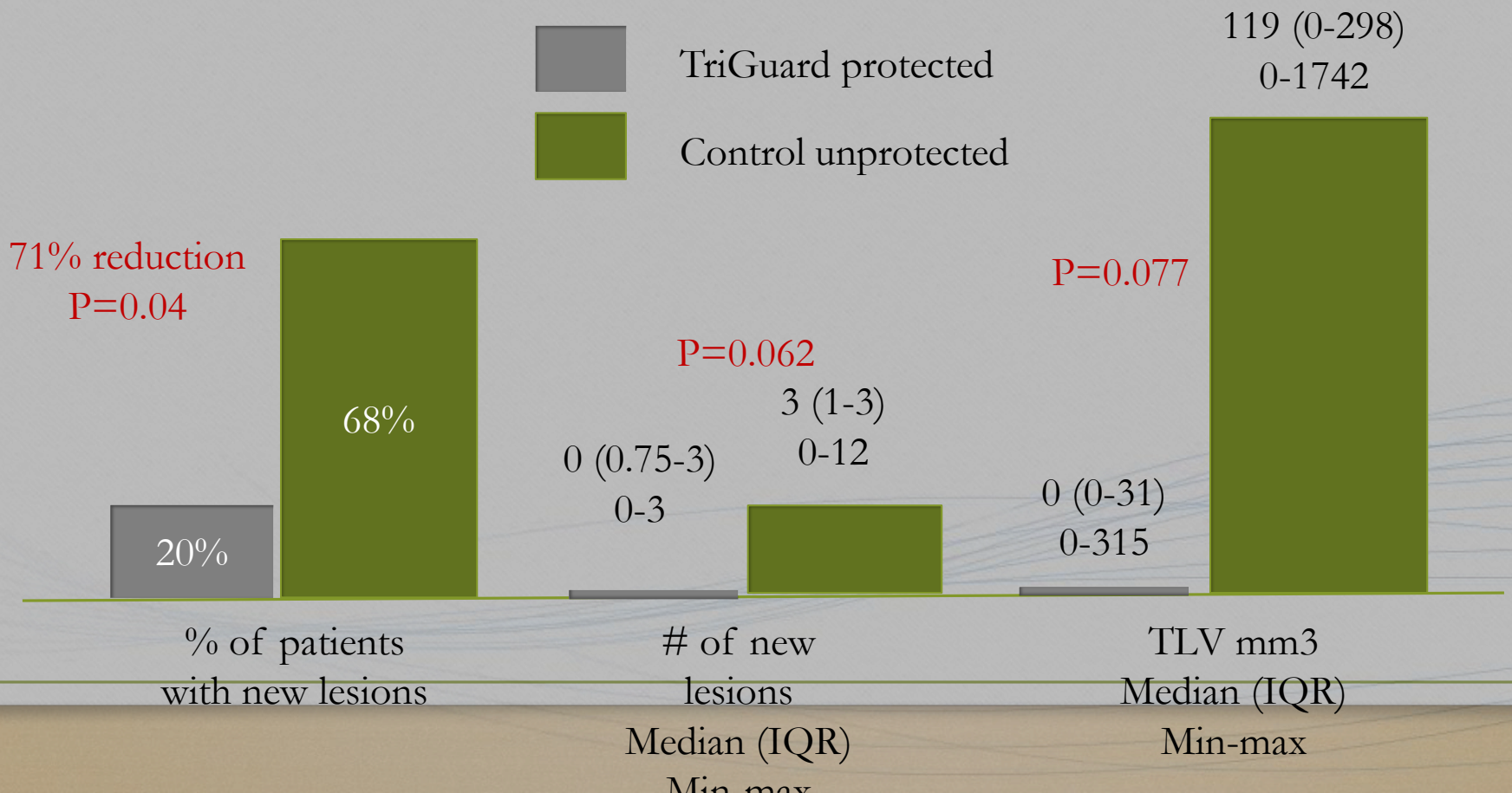
Claret filter



TriGuard Device for Cerebral Embolic Protection During Trans catheter Aortic Valve Replacement: A Multicenter Real-World Experience

Masieh Abawi, Ermela Yzeiraj; Adriaan Kraaijeveld,; Michiel Voskuil,; Pieter A. Doevendans; Joachim Schofer; Pieter R. Stella

51pts with TG vs 150 controls, Logistic EuroSCORE 12.6±8.3, Stroke rate was 0% in both groups



Debris during TAVR

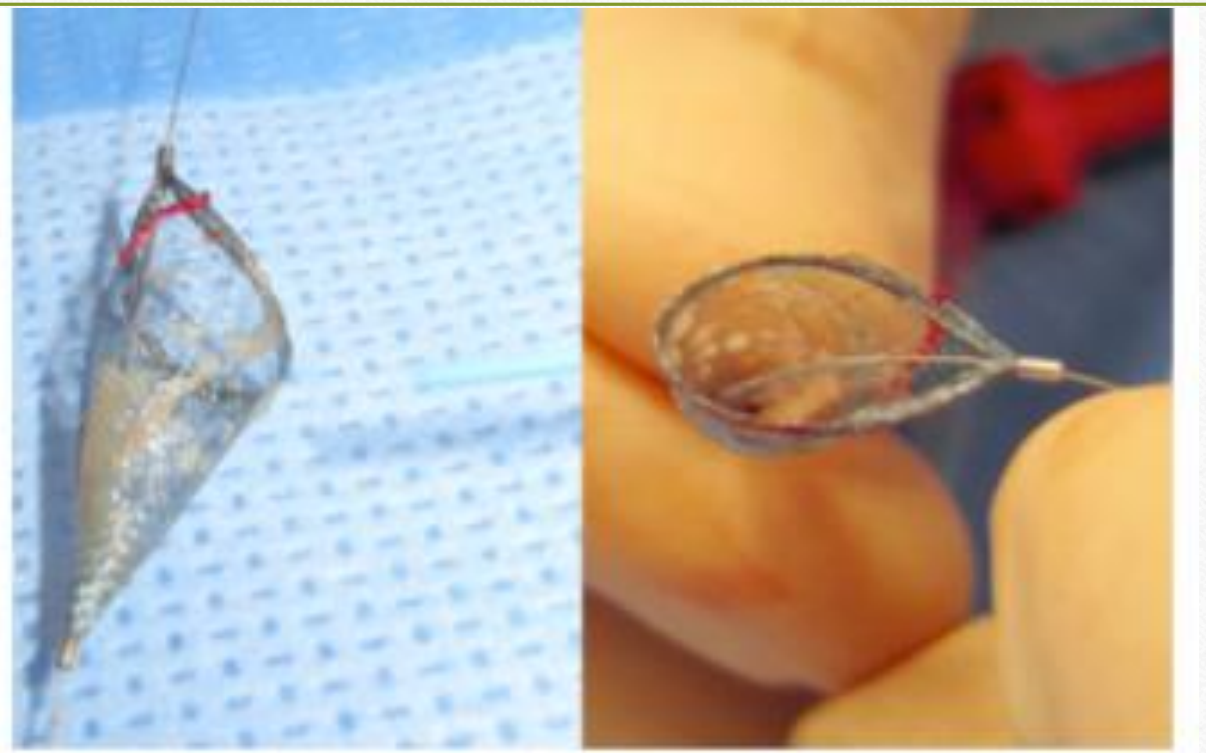
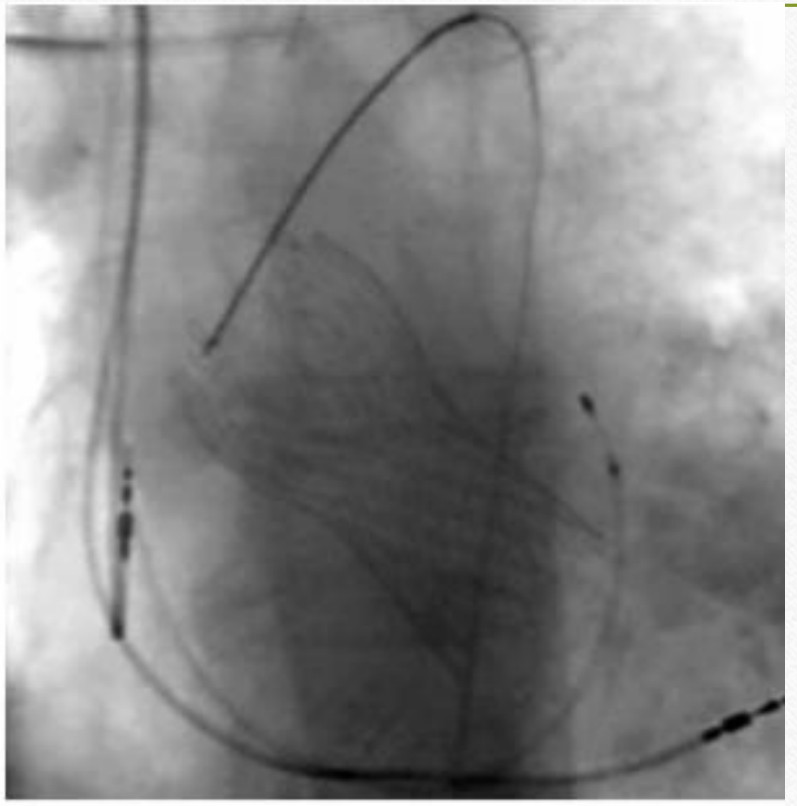


Image 1: Debris liberated during TAVI which has been captured by an

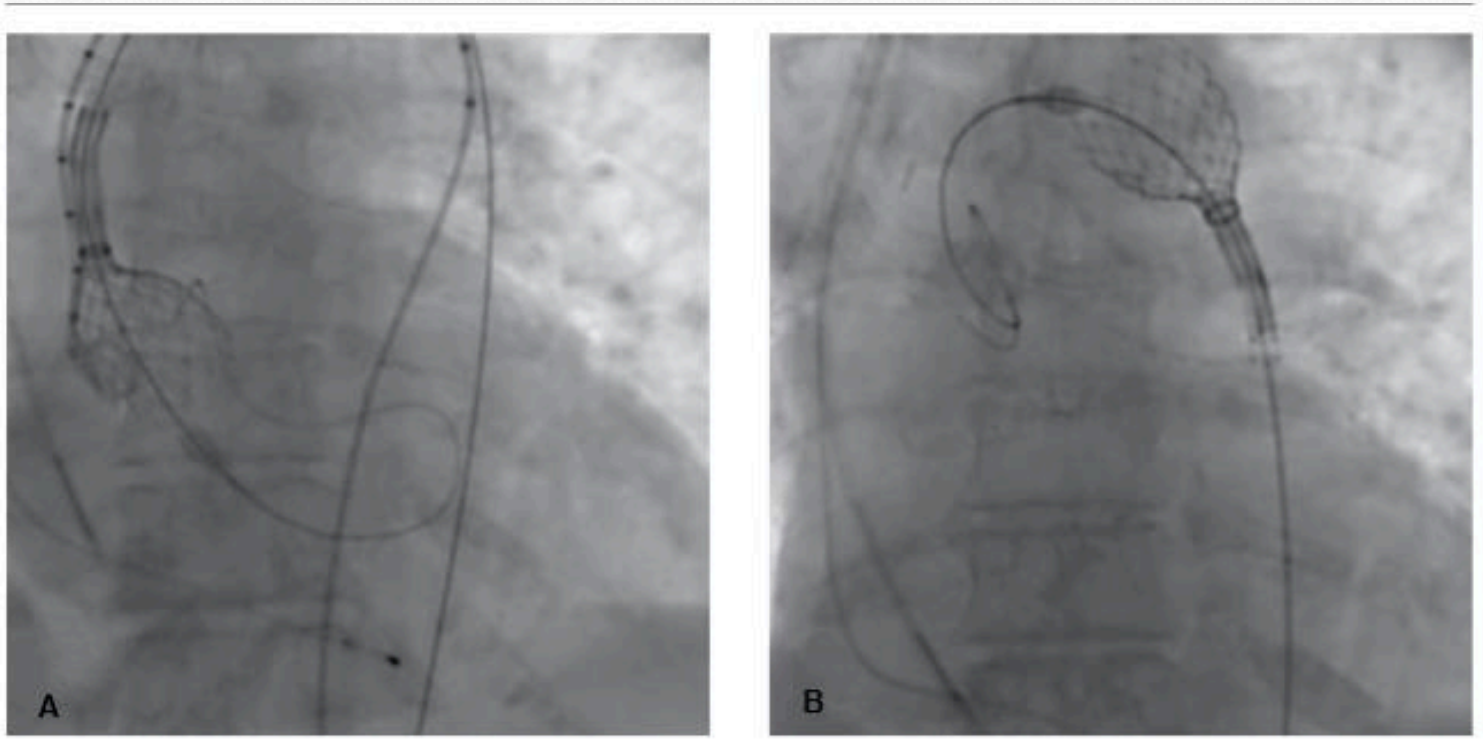
Missing of target area or Migration

- It will be major setback which can cause acute stroke.
- Shock.
- Unnecessary bleeding.
- Escalate the treatment cost.
- Increases hospital stay.

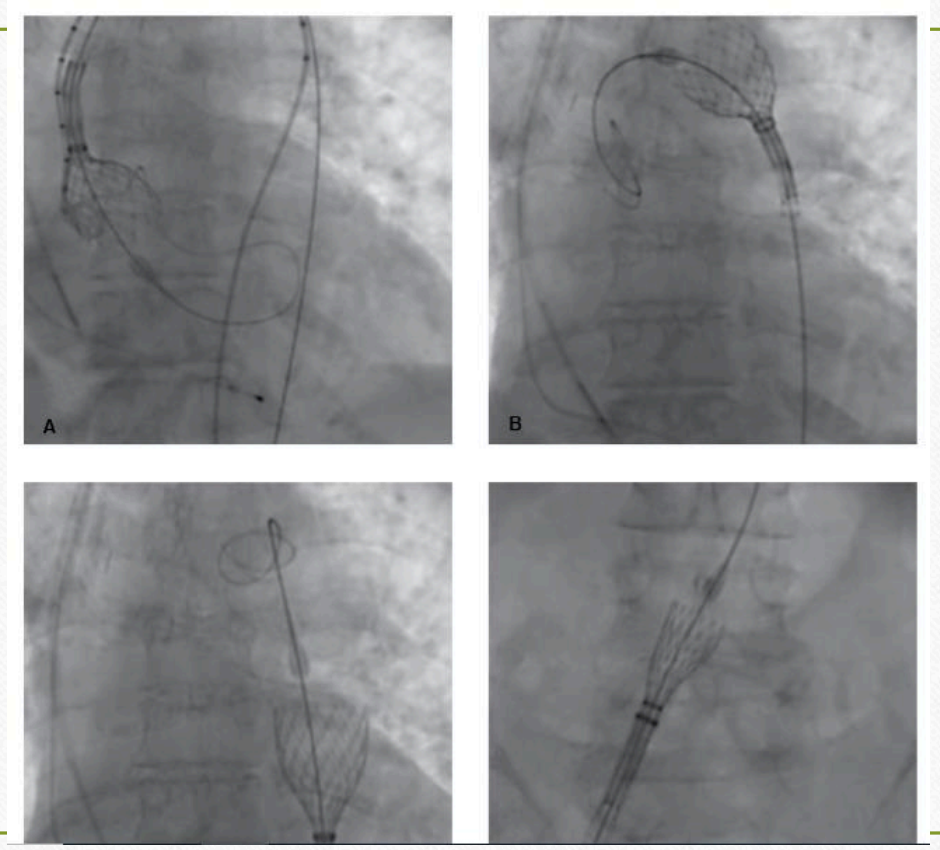
Migration of Valve into LV after deployment



Valve jumped back to descending Aorta



Gradually retrieved into working sheath



Small numbers study-27 pts

Inclusion criteria:

Normal renal function.

No previous history of stroke

EF->45%

Diabetes, Hypertension, Dyslipidemia

Only Aortic stenosis not on AR

Ages-56-90 years

Moderate to severe symptoms

Exclusion criteria

- CKD with <40ml GFR
- EF-<40%
- History of hemorrhagic stroke.
- Takayasu Arteritis.
- Severely immune compromised patients

Compared 1:1

- 14 patients were implanted TAVR under protection with pre CTA of brain and 13 patients were implanted with routine evaluation.

Primary Endpoints

- All cause mortality and stroke at 6 months and 1 year

Secondary endpoints

- Safety and acute and late stroke and neurologic deficits.

Conclusion/ Results

- CEPD significantly reduces acute and late stroke in TAVR patients at 6 months and 12 months periods.
- Devices designs and size factors also plays important role in neurological events in TAVR procedures.