Role of Embolic Protection Devices in TAVR: Are They Needed? Waste of Time and Money?

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REQUIRED

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I have no relevant financial relationships
Figure 1. Registry-based incidence of major and minor stroke after TAVR in current clinical practice. Abbreviations: ACC, American College of Cardiology; TAVR, transcatheter aortic valve replacement; TVT, TransValvular Therapy registry. Data are from Rodes-Cabau et al.,6 Thomas et al.,7 Elchaynoff et al.,8 Piazza et al.,9 Tamburino et al.,10 Zahn et al.,11 Bosmans et al.,12 Moat et al.,13 Gilard et al.,14 Avanzas et al.,15 Nombela-Franco et al.,16 Mack et al.,17 Popma et al.21
Stroke randomized trial TAVR/SAVR
Transcatheter Aortic Valve Replacement 2016
A Modern-Day “Through the Looking-Glass” Adventure

Torsten P. Vahl, MD, Susheel K. Kodali, MD, Martin B. Leon, MD
**SURTAVI TRIAL**

**Incidence of disabling stroke**

- **30-Day** p* = 0.057
- **2-Year** p* = 0.076

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**Graph Details:**
- **Y-axis:** Disabling stroke (%)
- **X-axis:** Months Post Procedure
- **Legend:**
  - TAVI
  - SAVR

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**No. at Risk**

<table>
<thead>
<tr>
<th></th>
<th>TAVI</th>
<th>SAVR</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>864</td>
<td>755</td>
</tr>
<tr>
<td>6</td>
<td>674</td>
<td>555</td>
</tr>
<tr>
<td>12</td>
<td>456</td>
<td>407</td>
</tr>
<tr>
<td>18</td>
<td>272</td>
<td>241</td>
</tr>
</tbody>
</table>

* log-rank
Fig 4 Forest plot for relative risk of stroke at longest follow-up for transcatheter aortic valve implantation (TAVI) compared with surgical aortic valve replacement (SAVR) for severe aortic stenosis, by valve approach.

Reed A Siemieniuk et al. BMJ 2016;354:bmj.i5130
Figure 2. Timing of stroke after TAVR with approximately 2 years follow-up. Each bar-chart represents the percentage of strokes out of the total individual study strokes. Abbreviations: PARTNER, Placement of Aortic Transcatheter Valves; TAVR, transcatheter aortic valve replacement. Data are from Amat-Santos et al,34 Nils et al,35 PARTNER A3 and B,37 and Nombela-Franco et al.36
Silent Ischemia

- >70% of TAVR have new silent cerebral lesions detected by post-procedural diffusion-weighted magnetic resonance imaging (DW-MRI)
- Has been suggested that represent silent brain infarctions that could be related to memory loss, cognitive decline, and dementia
- One study showed a >2-fold risk of dementia and decline in cognitive function.
- The same association was found after cardiac surgery within 6 weeks after the procedure
- Another study showed that in pts with silent lesions 91% of them had preserved cognitive function at 2 years follow up and only 5.4% had early cognitive decline
<table>
<thead>
<tr>
<th>Study</th>
<th>% of Patients With New Cerebral Lesions (n)</th>
<th>Mean No. of Infarcts Per Patient</th>
<th>Mean Lesion Volume and SD, cm³</th>
<th>Mean Total Volume and SD, cm³</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All</td>
<td>TF</td>
<td>TA</td>
<td>TF</td>
</tr>
<tr>
<td>Fairbairn et al, 2012²⁹</td>
<td>77% (24/31)</td>
<td>77% (24/31)</td>
<td>4.2 ± 6.5</td>
<td></td>
</tr>
<tr>
<td>Kahlert et al, 2010¹⁸</td>
<td>84.4% (27/32)</td>
<td>84.4% (27/32)</td>
<td>4.0 (2.1–6.0)</td>
<td>0.081 (0.06–0.10)</td>
</tr>
<tr>
<td>Ghanem et al, 2010⁶¹</td>
<td>72.7% (16/22)</td>
<td>72.7% (16/22)</td>
<td>3.4 ± 5.1</td>
<td></td>
</tr>
<tr>
<td>Astarci et al, 2010⁶²</td>
<td>91.5% (32/35)</td>
<td>90% (19/21)</td>
<td>93% (13/14)</td>
<td>5.9 ± 6.8</td>
</tr>
<tr>
<td>Rodès-Cabau et al, 2011⁹</td>
<td>68% (41/60)</td>
<td>66% (19/29)</td>
<td>71% (22/31)</td>
<td>3 (2–8)</td>
</tr>
<tr>
<td>Average estimate</td>
<td>78.2% (19/29)</td>
<td>4.1</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: DW-MRI, diffusion-weighted magnetic resonance imaging; SD, standard deviation; TA, transapical; TF, transfemoral.
Pathophysiology

- Stroke at 24h and 30 days strongly correlated to the procedure
- Retrograde crossing of a stenotic aortic valve results in new focal cerebral lesions in 22% of pts
- BAV could cause embolism of calcium deposits and increases the risk of thrombogenic complication
- The interaction of the stent valve with the aortic annulus over the displaced natural valve can cause additional embolic debris.
- Hypoperfusion may occur during BAV and balloon-expandable valve deployment due to repeated rapid ventricular pacing, results in transiently reduced cardiac output. This can induce ischemia in addition to impaire decreased washout of dislodged microemboli.
- prosthetic valve surface exposure, flow turbulence, blood stasis in the perivalvular space “outside” the metallic stent generate thrombi with subsequent events.

"...Improvements in the TAVR procedure may decrease risk of post-TAVR stroke. We observed that longer procedure time and more pacing runs and postdilatations were associated with a higher risk of stroke after TAVR (with variable reliability). Advances in valve and delivery-system design, along with increasing experience, may reduce procedure times and, thereby, reduce occurrence of stroke..."
Multivariable predictors
• total NIHSS score >0,
• Manifestations of prior CVA, prior TIA,
• peripheral vascular disease,
• absence of prior CABG
• presence of angina,
• low body mass index (<21 kg/m2),
• falls within the past 6 month

Multivariable procedural predictors
• total time in the cath lab or OR
• total delivery catheter time in the body
• rapid pacing during valvuloplasty
• repositioning of the CoreValve with a snare
• Number of valves implanted
Sources of Debris During TAVR

- **ASCENDING ARCH**
  - Arterial wall, calcific and atherosclerotic material
- **TRANSVERSE ARCH**
  - Arterial wall, calcific and atherosclerotic material
- **STENOTIC VALVE**
  - Leaflet tissue and calcific deposits
- **TAVR DEVICES**
  - Foreign material
- **NATIVE HEART**
  - Myocardium
Mechanism of efficient Cerebral Embolic protection

Sentinel Embolic protection (Boston Scientific)
Sentinel Filters Protection

- Fully Protected - 74% brain volume
- Partially Protected - 24% brain volume
- Unprotected - 2% brain volume

Protected blood flow to the brain

Unprotected blood flow to the brain

RVA ~10%
RCCA ~40%
LCCA ~40%
LVA ~10%

Sentinel Placement

Stroke reduction with CPF

95% of SENTINEL patients were evaluated by neurologists
Clinical Events Committee included 2 stroke neurologists

SENTINEL trial. Data presented at Sentinel FDA Advisory Panel, February 23, 2017

Kapadia SR et al. JACC 2017
# Registries

<table>
<thead>
<tr>
<th>Study Center</th>
<th>Unprotected TAVR Patients Neurological Event Rate % (n/N)</th>
<th>Sentinel TAVR Patients Neurological Event Rate % (n/N)</th>
<th>Relative Risk Reduction (RRR)</th>
<th>Number-needed-to-treat (NNT) to avoid one event</th>
<th>Specific Measures</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ulm University¹</td>
<td>4.6% (13/280)</td>
<td>1.4% (4/280)</td>
<td>70%</td>
<td>22</td>
<td>Propensity-score-matched All-stroke at 7-days</td>
</tr>
<tr>
<td>N=560</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>May 2017</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pinnacle Health²</td>
<td>10% (7/69)</td>
<td>0% (0/53)</td>
<td>100%</td>
<td>10</td>
<td>All-stroke at 7-days Length-of-stay (LOS) reduced from 3.2d without protection to 1.5d with Sentinel</td>
</tr>
<tr>
<td>N=122</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Feb 2018</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Erasmus, Rotterdam³</td>
<td>5% (23/453)</td>
<td>1% (3/294)</td>
<td>80%</td>
<td>25</td>
<td>All-stroke + TIA at 3-days</td>
</tr>
<tr>
<td>N=747</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>March 2018</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cedars Sinai⁴</td>
<td>3.8% (17/453)</td>
<td>1% (3/294)</td>
<td>74%</td>
<td>36</td>
<td>All-stroke at 3-days</td>
</tr>
<tr>
<td>N=419</td>
<td></td>
<td></td>
<td></td>
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<td>March 2018</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

2. Gada H, presented at CMS NTAP Town Hall meeting Feb 2018
3. Van Mieghem N, presented at JIM and CRT 2018, manuscript in preparation
TriGuard HDH vs TriGUARD 3

- **TriGuard HDH**
  - Nitinol frame with upper and lower stabilizers
  - Nitinol mesh (pore size 130x250 μm)
  - Filter area = 20.9 cm²
  - 9 Fr RX delivery

- **TriGUARD 3**
  - Self-positioning, nitinol frame without stabilizers
  - PEEK mesh (pore size 115x145 μm)
  - Filter area = 68.3 cm²
  - 8 Fr OTW delivery
REFLECT Trial Design (Phase I & II)

- Prospective, single-blind, randomized (2:1 device: control), multi-center safety & efficacy trial in two phases of the Keystone Heart Cerebral Embolic Protection Devices:
  - Phase 1 - TriGuard HDH
  - Phase 2 - TriGUARD 3

- Study Chairman: Jeffrey Moses
- Study PI: Taimm Nazif
- Co-PIs: Alexandra Lansky
  - Raj Makkar
  - Andreas Baumbach
  - Joachim Schofer
# Metanalysis

## Table 2 Clinical outcomes for TAVR with and without cerebral protection

<table>
<thead>
<tr>
<th>Study</th>
<th>Author</th>
<th>Year</th>
<th>30-day stroke (%)</th>
<th>30-day mortality (%)</th>
<th>Life threatening bleed (%)</th>
<th>Acute kidney injury (%)</th>
<th>Major vascular complications (%)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>TAVR + CP</td>
<td>TAVR</td>
<td>TAVR + CP</td>
<td>TAVR</td>
<td>TAVR + CP</td>
</tr>
<tr>
<td>CLEAN-TAVI</td>
<td>Haussig et al.</td>
<td>2014</td>
<td>8.0</td>
<td>8.0</td>
<td>0.0</td>
<td>2.0</td>
<td>2.0</td>
</tr>
<tr>
<td>DEFLECT-III</td>
<td>Lansky et al.</td>
<td>2015</td>
<td>4.3</td>
<td>5.1</td>
<td>2.2</td>
<td>5.1</td>
<td>2.2</td>
</tr>
<tr>
<td>EMBOL-X</td>
<td>Wednt et al.</td>
<td>2015</td>
<td>0.0</td>
<td>0.0</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
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<tr>
<td>MISTRAL-C</td>
<td>Van Mieghem et al.</td>
<td>2015</td>
<td>3.1</td>
<td>21.2</td>
<td>3.1</td>
<td>9.1</td>
<td>3.1</td>
</tr>
<tr>
<td>SENTINEL</td>
<td>Kapadia et al.</td>
<td>2017</td>
<td>5.6</td>
<td>9.1</td>
<td>1.3</td>
<td>1.8</td>
<td>NR</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td></td>
<td>5.4</td>
<td>9.3</td>
<td>1.3</td>
<td>3.2</td>
<td>2.3</td>
</tr>
</tbody>
</table>

CP, cerebral protection; NR, not reported; TAVR, transcatheter aortic valve replacement.
Cerebral protection devices in transcatheter aortic valve replacement: a clinical meta-analysis of randomized controlled trials

Nelson Wang, Kevin Phan

## Combined stroke & mortality @ 30 days

<table>
<thead>
<tr>
<th>Study or subgroup</th>
<th>CP Events</th>
<th>CP Total</th>
<th>No CP Events</th>
<th>No CP Total</th>
<th>Weight</th>
<th>Odds Ratio M-H, Fixed, 95% CI</th>
<th>Odds Ratio M-H, Fixed, 95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>CLEAN-TAVI</td>
<td>4</td>
<td>50</td>
<td>5</td>
<td>50</td>
<td>15.6%</td>
<td>0.78 [0.20, 3.10]</td>
<td></td>
</tr>
<tr>
<td>DEFLECT-III</td>
<td>3</td>
<td>46</td>
<td>4</td>
<td>39</td>
<td>13.7%</td>
<td>0.61 [0.13, 2.91]</td>
<td></td>
</tr>
<tr>
<td>EMBOL-X</td>
<td>0</td>
<td>14</td>
<td>0</td>
<td>16</td>
<td>Not estimable</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MISTRAL-C</td>
<td>1</td>
<td>32</td>
<td>6</td>
<td>33</td>
<td>19.4%</td>
<td>0.15 [0.02, 1.28]</td>
<td></td>
</tr>
<tr>
<td>SENTINEL</td>
<td>16</td>
<td>234</td>
<td>12</td>
<td>111</td>
<td>51.3%</td>
<td>0.61 [0.28, 1.33]</td>
<td></td>
</tr>
<tr>
<td>Total (95% CI)</td>
<td>376</td>
<td>249</td>
<td></td>
<td></td>
<td>100.0%</td>
<td>0.54 [0.30, 0.98]</td>
<td></td>
</tr>
<tr>
<td>Total events</td>
<td>24</td>
<td></td>
<td>27</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Heterogeneity: $\chi^2=1.77$, df=3 (P=0.62); $I^2=0\%$
Test for overall effect: $Z=2.03$ (P=0.04)
### Stroke @ 30 days

<table>
<thead>
<tr>
<th>Study or subgroup</th>
<th>Events</th>
<th>Total</th>
<th>Events</th>
<th>Total</th>
<th>Weight M-H, Fixed, 95% CI</th>
<th>Odds Ratio M-H, Fixed, 95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>CLEAN-TAVI</td>
<td>0</td>
<td>50</td>
<td>1</td>
<td>50</td>
<td>16.2%</td>
<td>0.33 [0.01, 8.21]</td>
</tr>
<tr>
<td>DEFLECT-III</td>
<td>1</td>
<td>46</td>
<td>2</td>
<td>39</td>
<td>23.2%</td>
<td>0.41 [0.04, 4.71]</td>
</tr>
<tr>
<td>EMBOL-X</td>
<td>0</td>
<td>14</td>
<td>0</td>
<td>16</td>
<td>Not estimable</td>
<td></td>
</tr>
<tr>
<td>MISTRAL-C</td>
<td>1</td>
<td>32</td>
<td>3</td>
<td>33</td>
<td>31.3%</td>
<td>0.32 [0.03, 3.28]</td>
</tr>
<tr>
<td>SENTINEL</td>
<td>3</td>
<td>234</td>
<td>2</td>
<td>111</td>
<td>29.3%</td>
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</tr>
<tr>
<td><strong>Total (95% CI)</strong></td>
<td>376</td>
<td>249</td>
<td>100.0%</td>
<td>0.46 [0.15, 1.40]</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Total events</strong></td>
<td>5</td>
<td>8</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Heterogeneity: $\chi^2=0.36$, df=3 ($P=0.95$); $I^2=0\%$

Test for overall effect: Z=1.37 ($P=0.17$)

---

### Mortality @ 30 days

<table>
<thead>
<tr>
<th>Study or subgroup</th>
<th>Events</th>
<th>Total</th>
<th>Events</th>
<th>Total</th>
<th>Weight M-H, Fixed, 95% CI</th>
<th>Odds Ratio M-H, Fixed, 95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>CLEAN-TAVI</td>
<td>0</td>
<td>50</td>
<td>1</td>
<td>50</td>
<td>16.2%</td>
<td>0.33 [0.01, 8.21]</td>
</tr>
<tr>
<td>DEFLECT-III</td>
<td>1</td>
<td>46</td>
<td>2</td>
<td>39</td>
<td>23.2%</td>
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<td>0</td>
<td>14</td>
<td>0</td>
<td>16</td>
<td>Not estimable</td>
<td></td>
</tr>
<tr>
<td>MISTRAL-C</td>
<td>1</td>
<td>32</td>
<td>3</td>
<td>33</td>
<td>31.3%</td>
<td>0.32 [0.03, 3.28]</td>
</tr>
<tr>
<td>SENTINEL</td>
<td>3</td>
<td>234</td>
<td>2</td>
<td>111</td>
<td>29.3%</td>
<td>0.71 [0.12, 4.30]</td>
</tr>
<tr>
<td><strong>Total (95% CI)</strong></td>
<td>376</td>
<td>249</td>
<td>100.0%</td>
<td>0.46 [0.15, 1.40]</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Total events</strong></td>
<td>5</td>
<td>8</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Heterogeneity: $\chi^2=0.36$, df=3 ($P=0.95$); $I^2=0\%$

Test for overall effect: Z=1.37 ($P=0.17$)
New total volume lesions @ 30 days

<table>
<thead>
<tr>
<th>Study or subgroup</th>
<th>CP Mean</th>
<th>SD</th>
<th>Total</th>
<th>Mean</th>
<th>SD</th>
<th>Total</th>
<th>Weight</th>
<th>Std. Mean difference IV, Random, 95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>CLEAN-TAVI</td>
<td>219.3</td>
<td>170.2</td>
<td>45</td>
<td>588.7</td>
<td>400</td>
<td>43</td>
<td>21.5%</td>
<td>-1.20 [-1.66, -0.75]</td>
</tr>
<tr>
<td>DEFLECT-III</td>
<td>58.5</td>
<td>52.5</td>
<td>33</td>
<td>68.3</td>
<td>43.8</td>
<td>26</td>
<td>20.3%</td>
<td>-0.20 [-0.71, 0.32]</td>
</tr>
<tr>
<td>EMBOL-X</td>
<td>88.0</td>
<td>60.0</td>
<td>14</td>
<td>168.0</td>
<td>217</td>
<td>16</td>
<td>16.3%</td>
<td>-0.47 [-1.20, 0.25]</td>
</tr>
<tr>
<td>MISTRAL-C</td>
<td>120.7</td>
<td>191.7</td>
<td>22</td>
<td>272.3</td>
<td>333.2</td>
<td>15</td>
<td>17.3%</td>
<td>-0.58 [-1.25, 0.10]</td>
</tr>
<tr>
<td>SENTINEL</td>
<td>383.2</td>
<td>538.2</td>
<td>91</td>
<td>425.0</td>
<td>562.4</td>
<td>98</td>
<td>24.6%</td>
<td>-0.08 [-0.36, 0.21]</td>
</tr>
</tbody>
</table>

Total (95% CI) 205 198 100.0% -0.49 [-0.96, -0.03]

Heterogeneity: Tau²=0.21; Χ²=17.71, df=2 (P=0.94); I²=0%
Test for overall effect: Z=1.52 (P=0.13)

Pts with new brain lesions @ 30 days

<table>
<thead>
<tr>
<th>Study or subgroup</th>
<th>CP Events</th>
<th>Total</th>
<th>No CP Events</th>
<th>Total</th>
<th>Weight</th>
<th>Odds Ratio M-H, Fixed, 95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>DEFLECT-III</td>
<td>16</td>
<td>22</td>
<td>13</td>
<td>15</td>
<td>30.0%</td>
<td>0.41 [0.07, 2.38]</td>
</tr>
<tr>
<td>EMBOL-X</td>
<td>8</td>
<td>14</td>
<td>11</td>
<td>16</td>
<td>31.3%</td>
<td>0.61 [0.14, 2.71]</td>
</tr>
<tr>
<td>MISTRAL-C</td>
<td>26</td>
<td>33</td>
<td>23</td>
<td>26</td>
<td>38.8%</td>
<td>0.48 [0.11, 2.10]</td>
</tr>
</tbody>
</table>

Total (95% CI) 69 57 100.0% 0.50 [0.20, 1.23]

Total events 50 47

Heterogeneity: Χ²=0.11, df=2 (P=0.94); I²=0%
Test for overall effect: Z=1.52 (P=0.13)
Considerations

- Metanalysis failed to detect a significant reduction in clinically overt stroke and all-cause mortality. However, both endpoints were numerically lower in the EP group.

- The effect of EP on neurological imaging endpoints appeared to be uniform between SE and BE valve types, which differ significantly in terms of design and implantation technique.

- Because a substantial number of emboli during TAVR are of thrombotic origin, complementary antithrombotic strategies to EP are warranted.
Technical and procedural factors favouring CVA

• Thrombus formation in large diameter sheath despite optimal anticoagulation
• Wire and catheter manipulation in aortic arch,
• Aggressive retrograde aortic valve crossing
• Suboptimal preparatory BAV (ineffective RVP and excessive balloon movement)
• DCS navigation
• ↓ cardiac output reduce cerebral perfusion which can results in diffuse silent ischemia
  • RV pacing during BAV pre and post implant or during valve implant
  • Hemodynamic instability

Device malpositioning, dislodgment, or embolization;
• Consensus to better characterize, track, and report CVEs in TAVR and SAVR
  • accuracy
  • etiology (stroke because of atrial fibrillation versus device thrombosis)
  • Easy of use (ex noncomplex tools in diagnosis)
• The importance of antithrombotic drugs in mitigating stroke risk after TAVR,
• Characterize modifiable factors for periprocedural stroke to identify patients who may benefit of intraprocedural embolic protection devices
  • complex anatomic characteristics (eg, highly calcified native valves, large aortic arc atheromas, or angulated aorta)
  • Aortic valve mobile vegetations
  • expected challenging-longer procedures.
Role of Embolic Protection Devices in TAVR: Are They Needed? Waste of Time and Money?

• Yes if is used extensively
• No if we are able to Identify patient at risk of intra-periprocedurale CVA:
  • Optimize procedure technique
    • Be Precise, follow rigorously all the procedural steps, don’t waste time be fast but not in hurry
    • Reduce unuseful manipulation
      • aggressive approach for crossing the valve
      • mantain the wire in the ventricle,
      • reduce RV Pacing for BAV pre and post
      • during deployement mantain the valve position
• Use CEP in patient considered at high risk for CVA
• Avoid CEP if:
  • Unfavourable vascular anatomy
  • Potential device related complication of the procedure
Discussion

1. Who is the patient at higher risk of periprocedural stroke?
2. Do the younger or lower risk patient benefit more of the cerebral embolic protection?
3. Technical advise for reducing periprocedural stroke.
4. Concern about potential complication during CEP device manipulation/postioning?
5. Importance of full protection of epiaortic vessels.
Thank You