

# acute aortic syndrome

- Decision making for management -

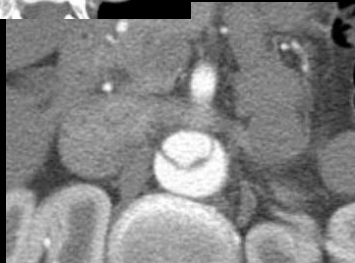
**Kay-Hyun Park**

Thoracic and Cardiovascular Surgery

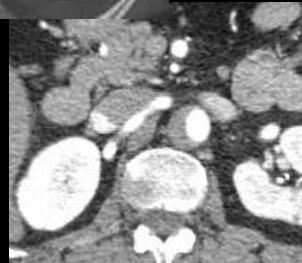
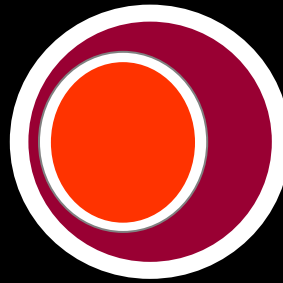
Seoul National University Bundang Hospital

# Acute aortic syndrome

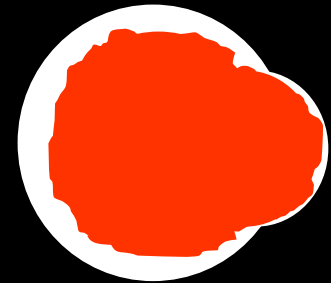
**Dissection**



**Intramural  
hematoma**



**Penetrating  
ulcer**



# *Medical Management: aims*

- prevent sudden death and later complications
  - halt distal progression of dissection
  - decrease the expanding pressure on false lumen
  - maintain vital organ perfusion
  - decrease arterial blood pressure*
  - decrease the velocity of LV contraction ( $dP/dT$ )*

# *Medical Management: BP control*

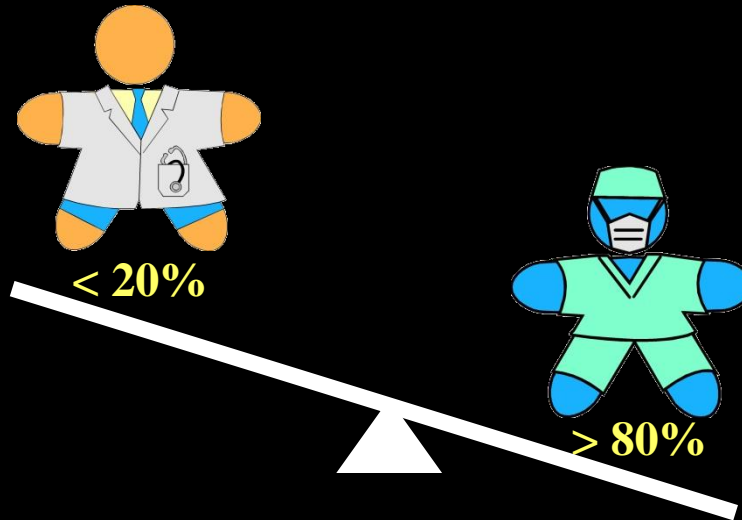
- Beta-blocker
  - atenolol, propranolol, esmolol, labetalol
  - initial target: systolic BP 90~100, HR 60~70
  - *monitoring: peripheral & vital organ perfusion*
- Vasodilator
  - sodium nitroprusside, nitroglycerine
  - *reflex positive inotropism & chronotropism*
  - *consider volume status*
- Calcium channel blocker
  - nicardipine
- Sedatives & analgesics, *prn*

# *Medical Management: others*

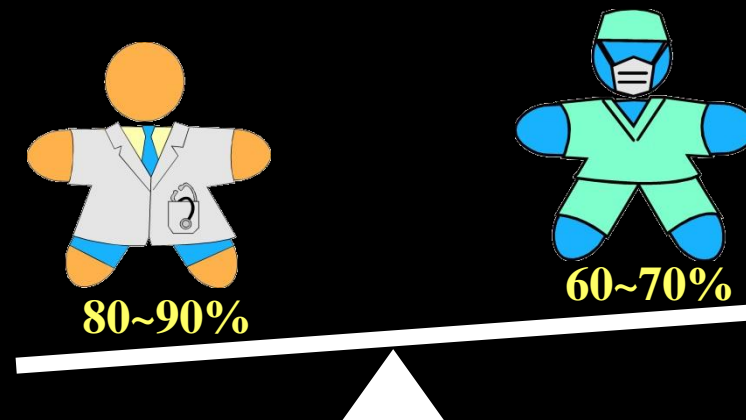
- Avoid undue irritation to the patient
- Establishment of monitoring lines
  - A-line: Right radial is preferable.
  - Central venous line: right internal jugular is preferable.
  - *must be done by an experienced member of the team.*
- Palliative pericardiocentesis for tamponade
- Management of irreversible malperfusion
  - fasciotomy, amputation, hemodialysis, bowel resection

# Survival chance in acute dissection

Type A



Type B



# Acute aortic syndrome

*- conventional principle -*

complicated

uncomplicated

- Dissection

- Type A

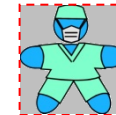


- Type B



- IMH

- Type A



- Type B



- PAU



# Endovascular repair of type A dissection



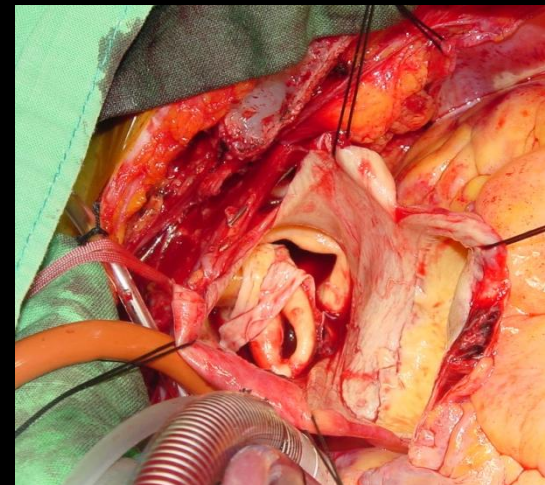
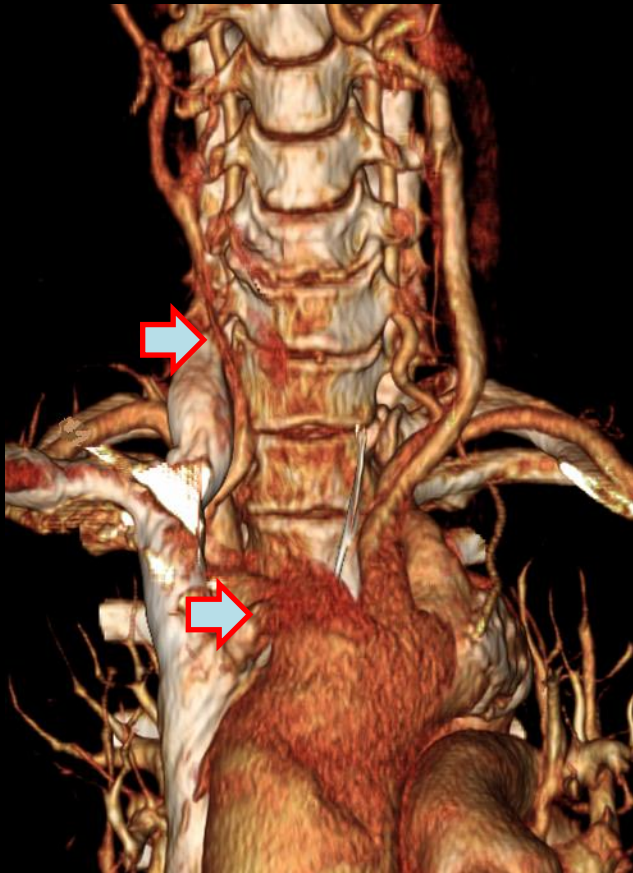


# Issues in surgery for type A dissection

---

- Indication & timing
  - preoperative cardiac arrest / CPR
  - very old age
  - severe malperfusion
  - Preoperative anticoagulation / antiplatelet / thrombolysis
- Extent of aortic replacement
  - when to replace the root and/or arch
  - Frozen elephant trunk
- Technical issues
  - cardiopulmonary bypass / brain protection
  - hemostasis

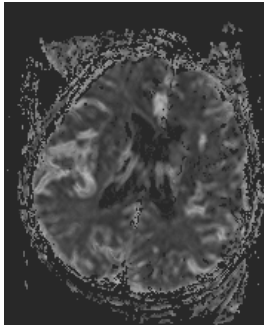
# Brain malperfusion



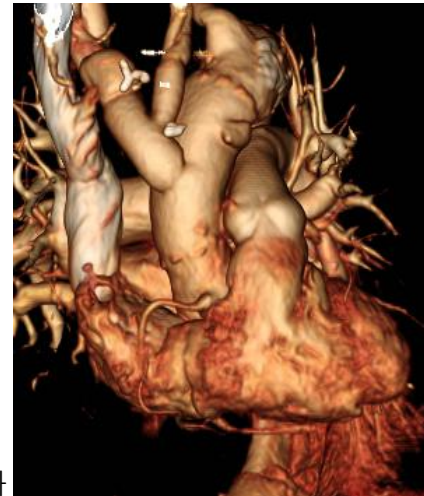
# *Aorta first or Branch first ?*



*Carotid  
first*



*Aorta  
first*



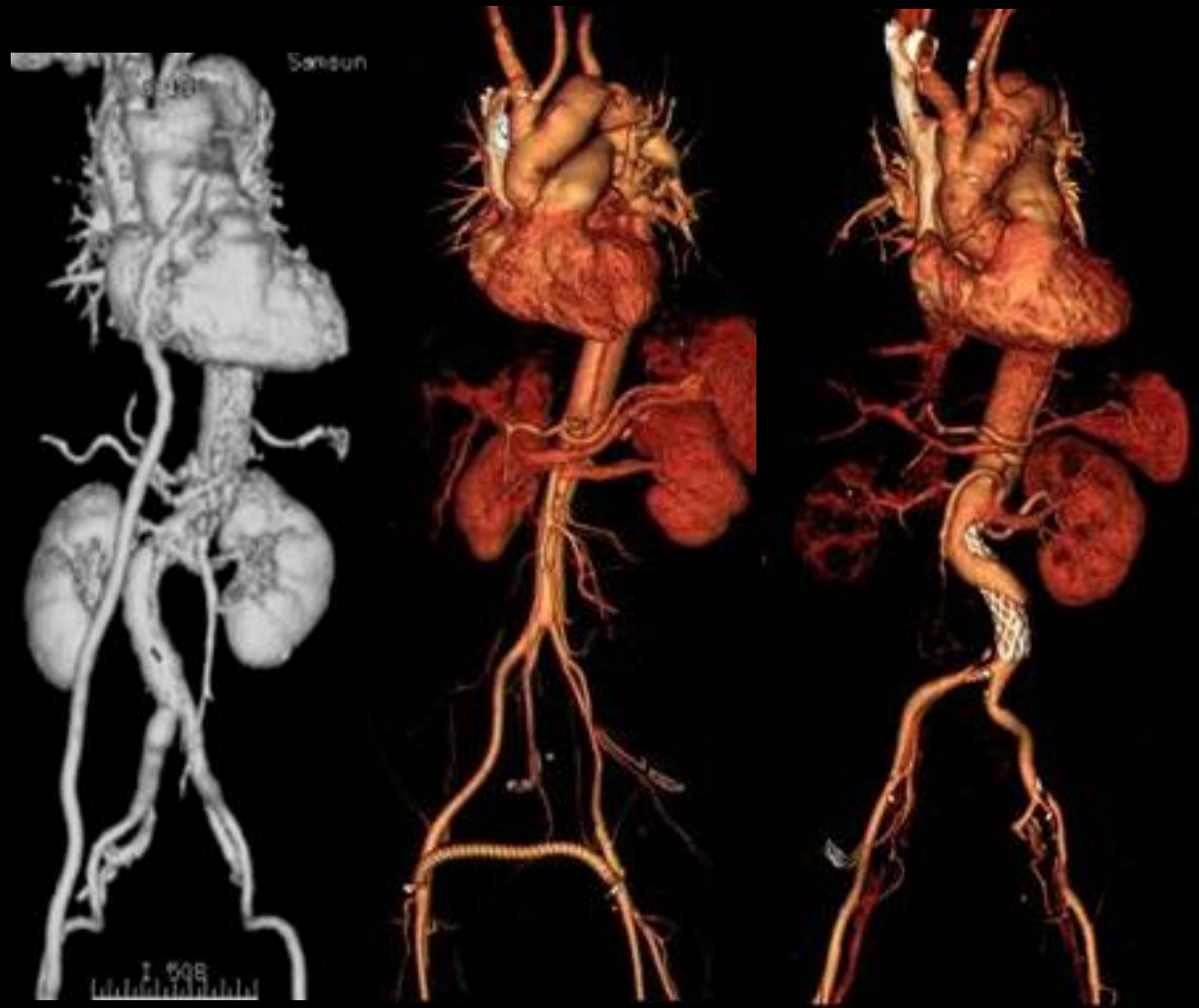
# Coronary malperfusion

---



# *Total occlusion of abdominal aorta*

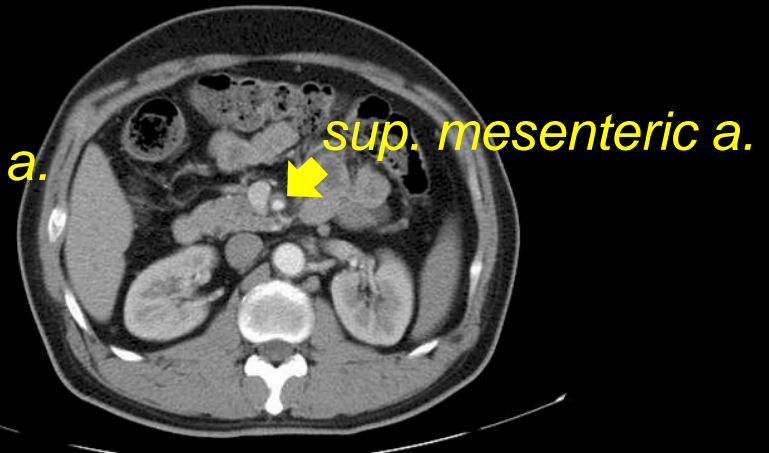
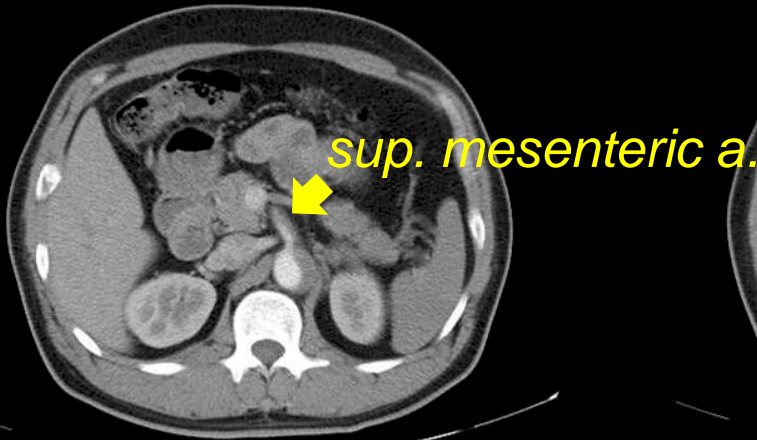
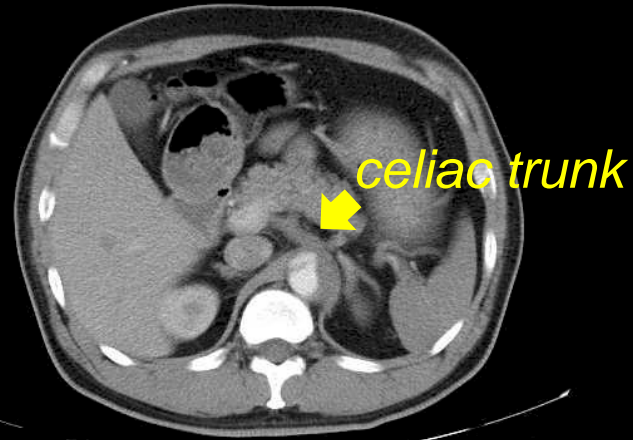




제7차 전공의 학술세미나

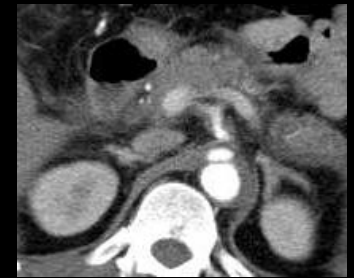
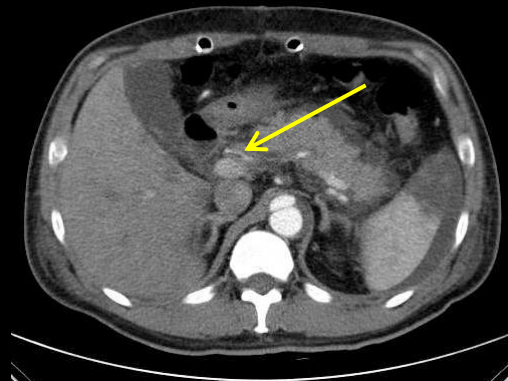
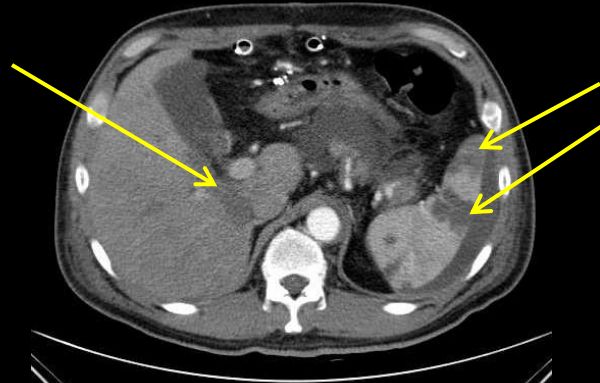
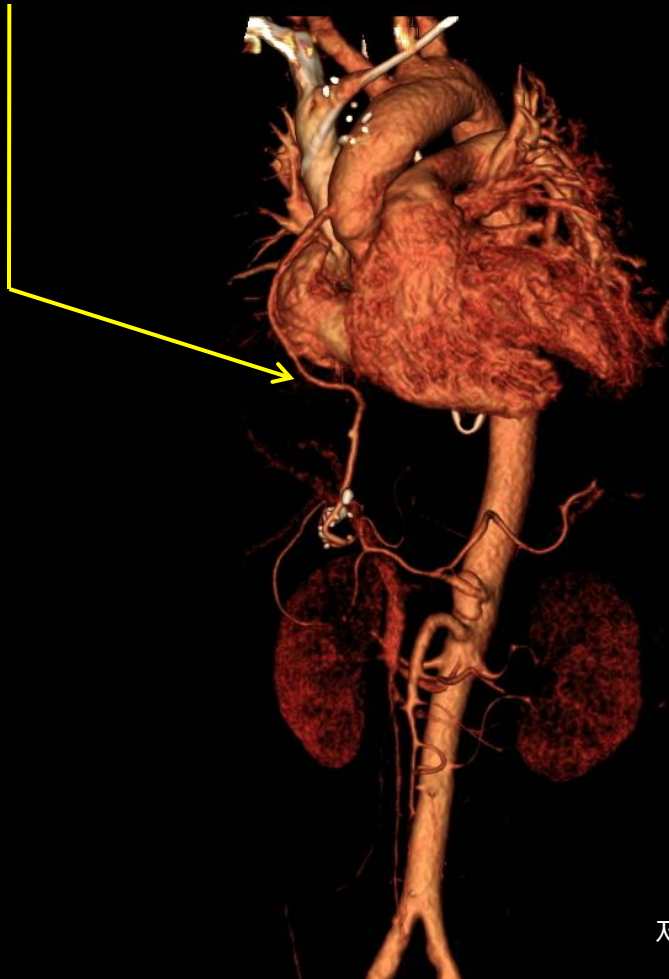
- M / 41 y

- Upper back pain & abdominal pain, nausea, vomiting



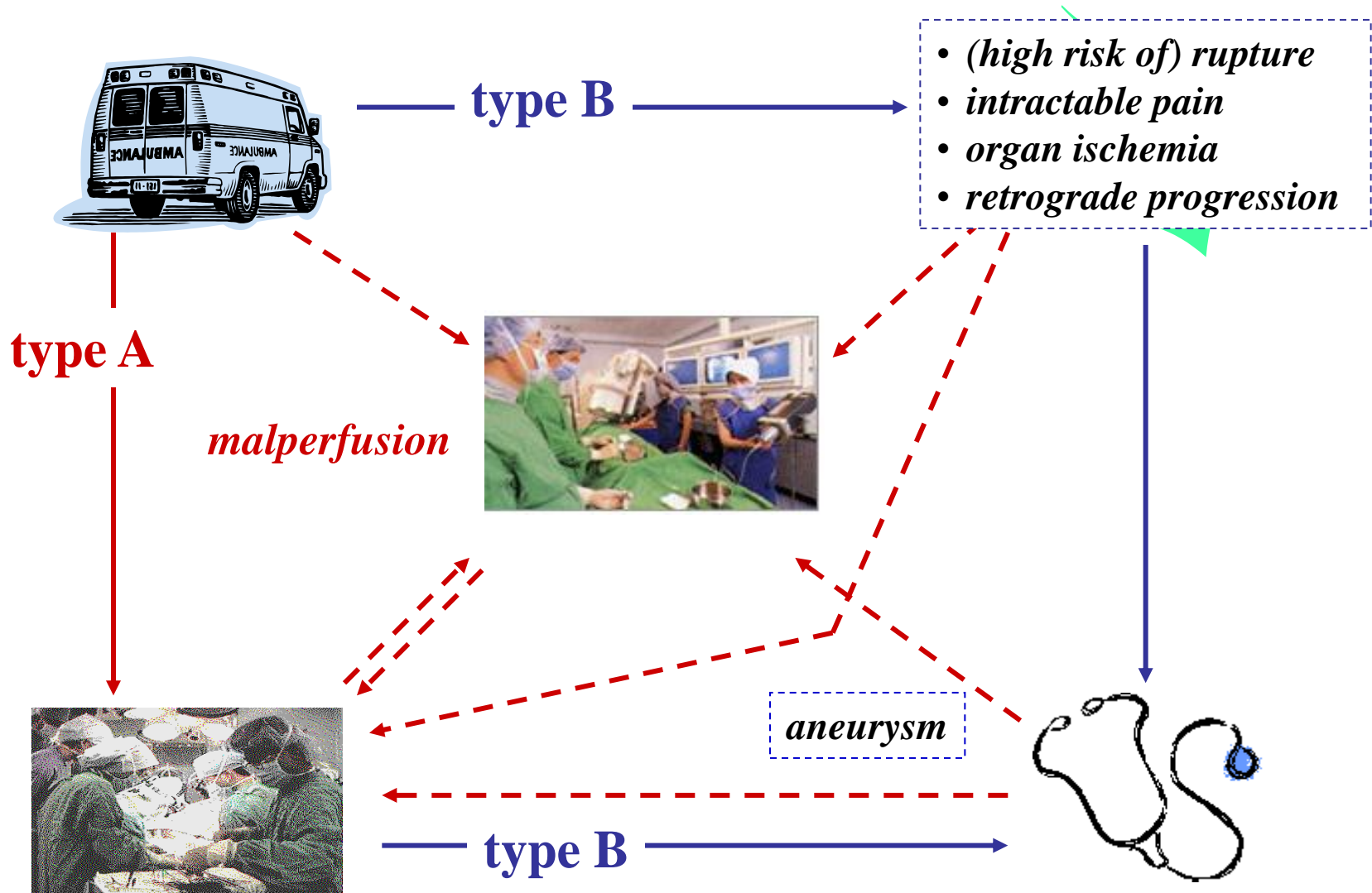
# CT angiography on POD #1

*Ascending aortic graft-to-RGEA  
saphenous vein graft*

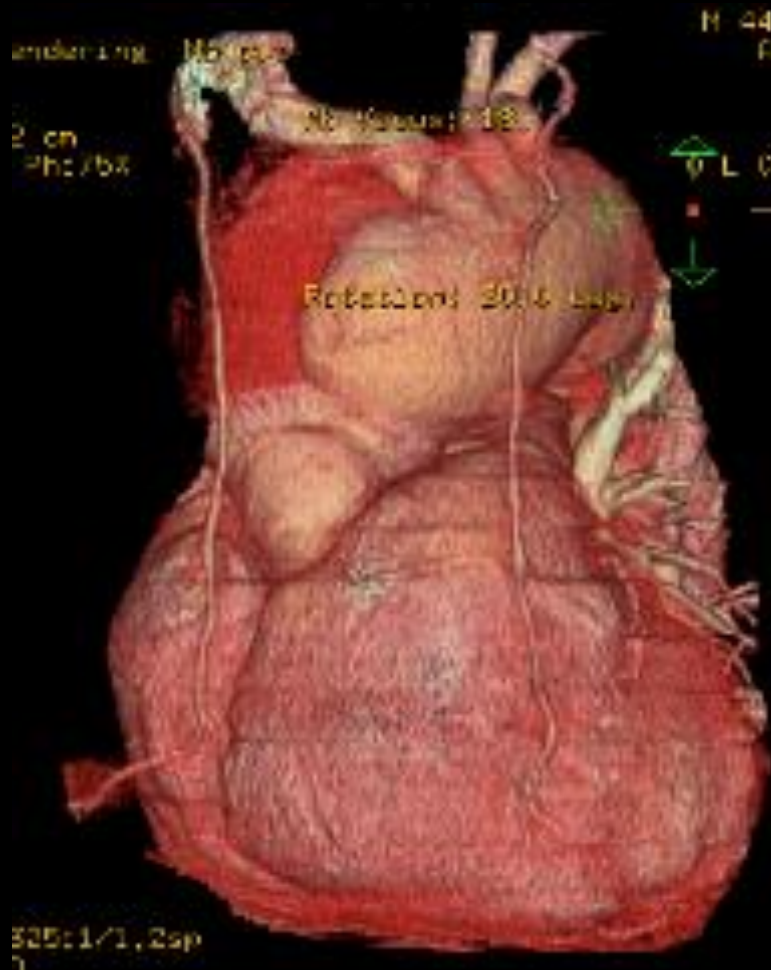




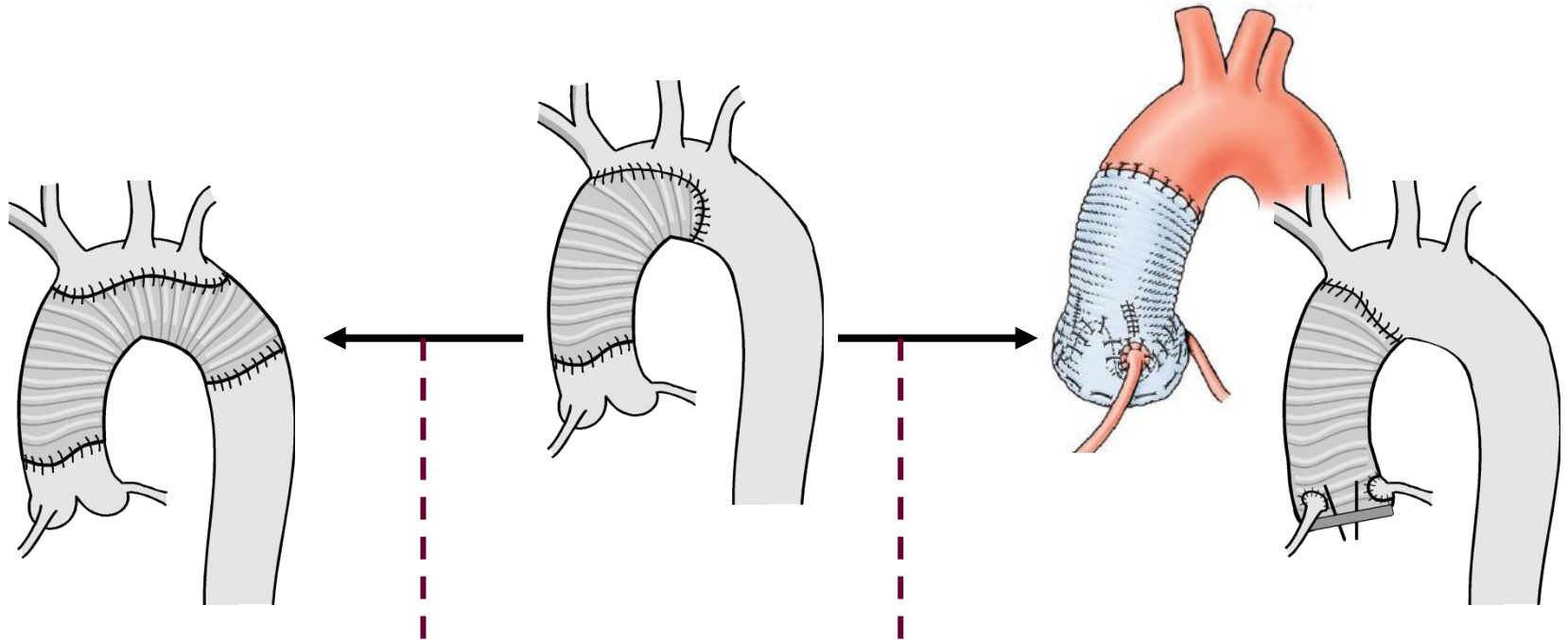
# Management scheme for aortic dissection



# Inadequate operation : unhappy outcome



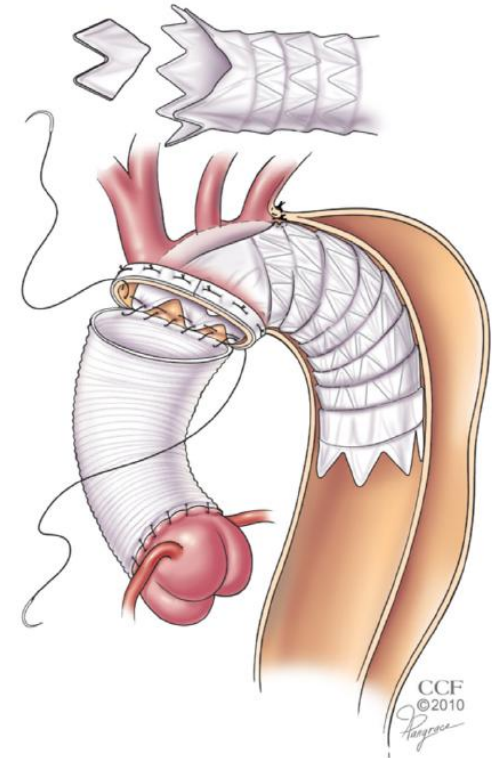
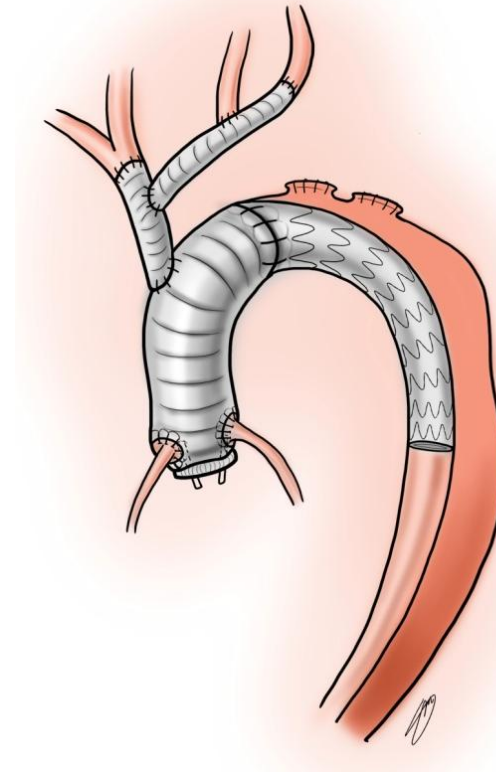
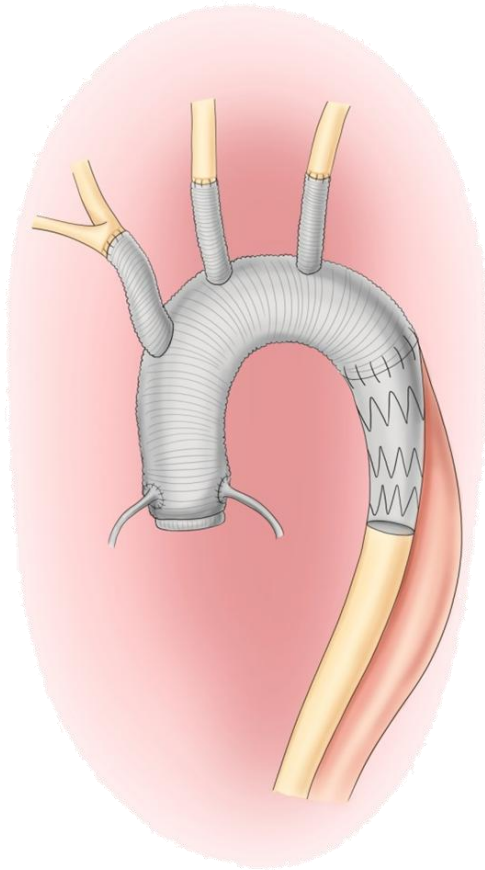
# Extent of surgery for acute type A dissection



- intimal tear in the arch
- large arch (> 4cm)
- future need of descending thoracic aorta surgery

- Marfan syndrome
- large sinus (> 3.5~4cm)
- large tear in sinus
- large false lumen in sinus

# Hybrid repair (*frozen elephant trunk*) for acute type A dissection

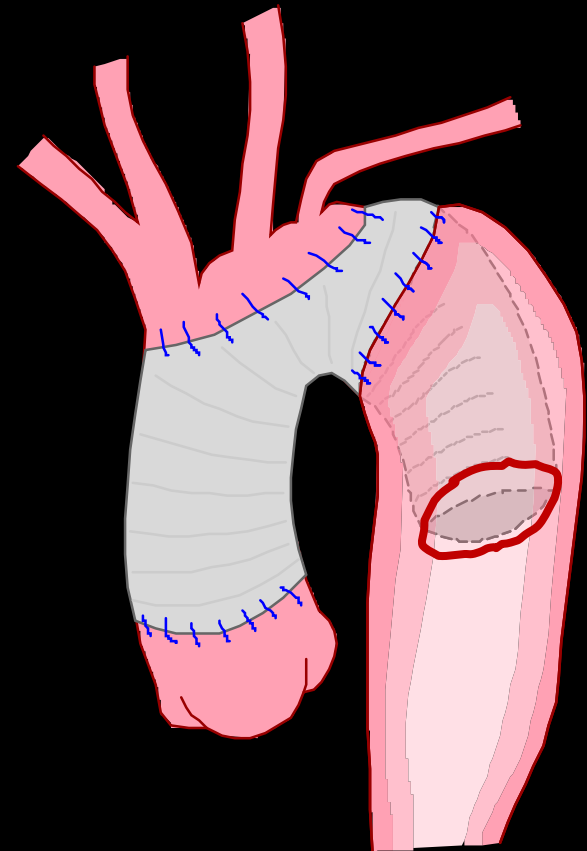
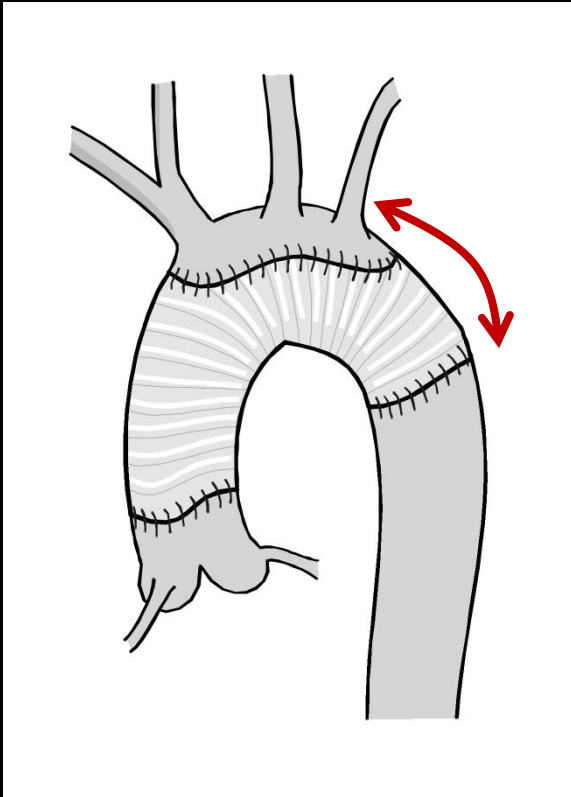


Roselli EE, et al.

*J Thorac Cardiovasc Surg* 2013;145:S197-201

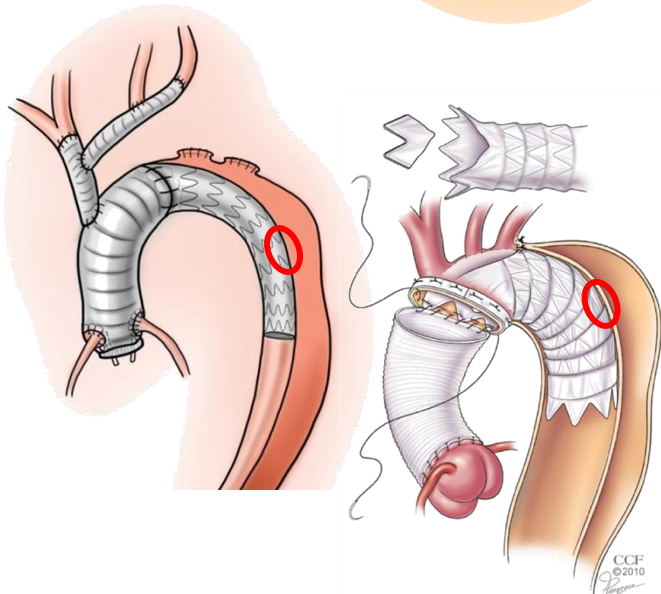
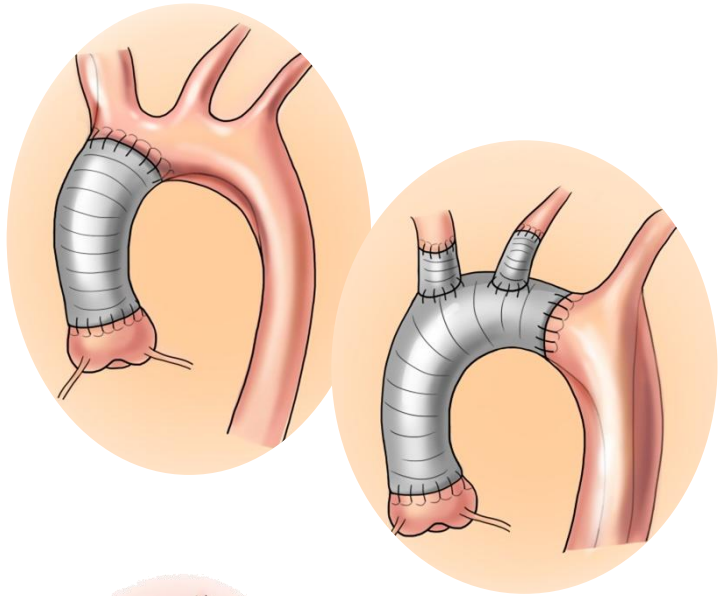
# Elephant trunk

## for aortic arch replacement



# Frozen elephant trunk (FET)

- ideas of the advocates -

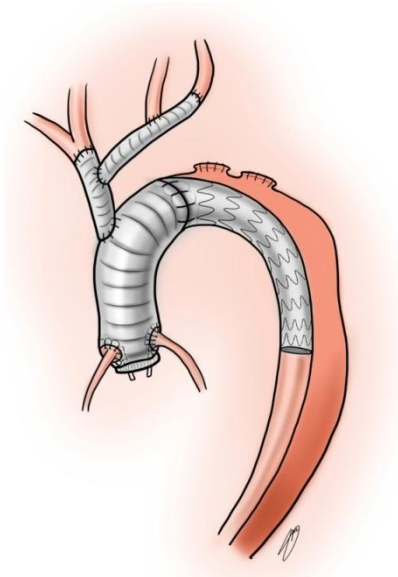


*exclusion of  
distal tear*



# FET for type A dissection: debates

- FET has some drawbacks.
  - Prolongation of *procedural time*, esp. total circulatory arrest
  - Risk of *paraplegia*
  - Uncovered aorta is still at the risk of aneurysmal dilatation.
- Even after conventional surgery without FET, improvement of residual descending false lumen does occur in some patients.

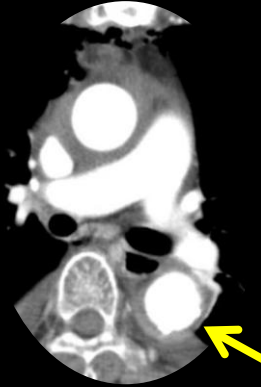


## F / 78, ascending aorta replacement

preoperative



4 days



26 months

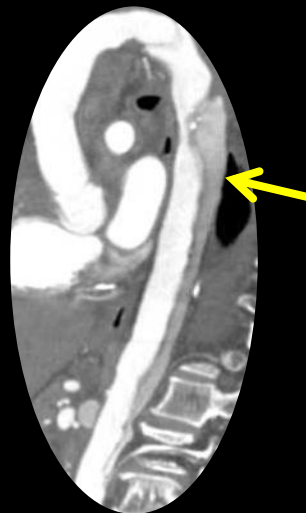


## M / 64, total arch replacement with short elephant trunk

preoperative



6 days



8 months



38 months





# Causes of persistent descending FL patency

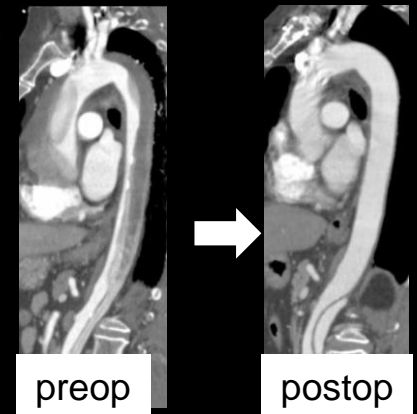
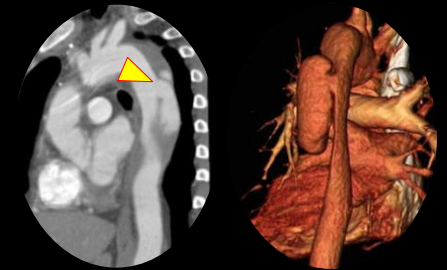
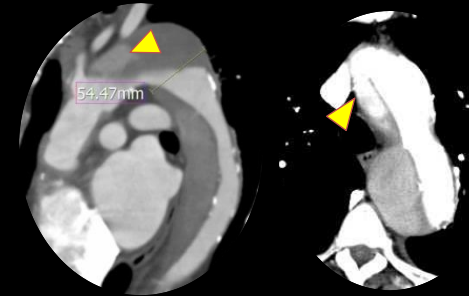
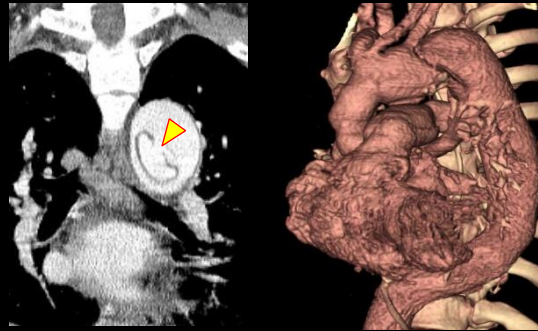
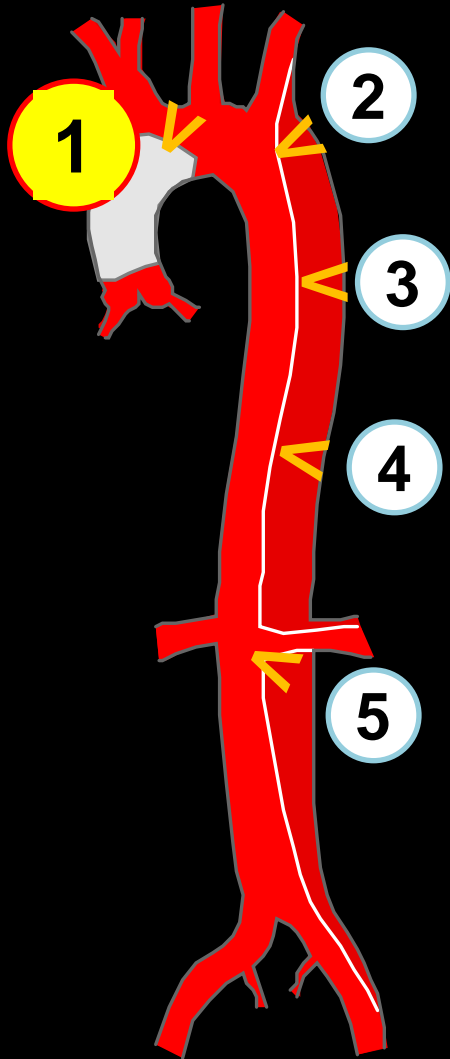
1. new, iatrogenic tear (anastomotic line)

2. arch tear left alone

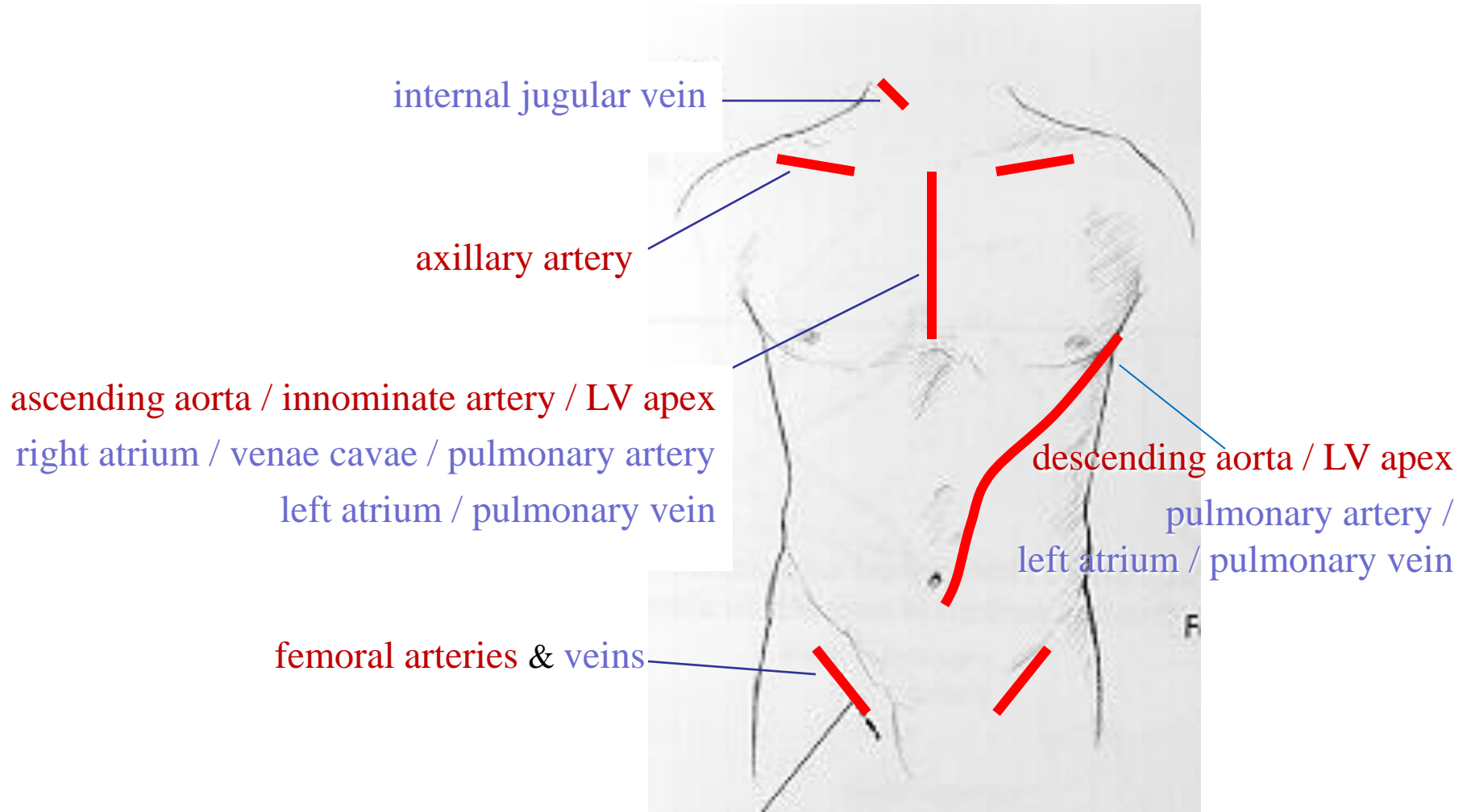
3. tear at descending thoracic aorta

4. primary tear at far distal aorta  
(retrograde type A dissection)

5. re-entry tear at far distal aorta

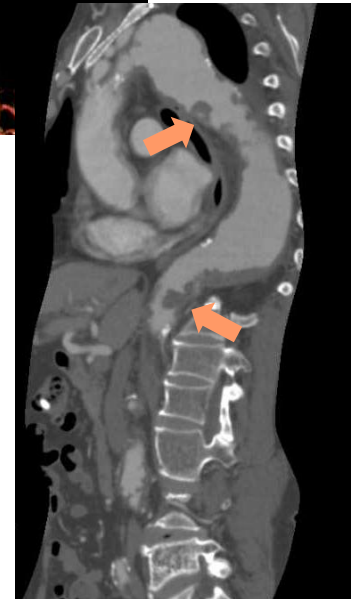
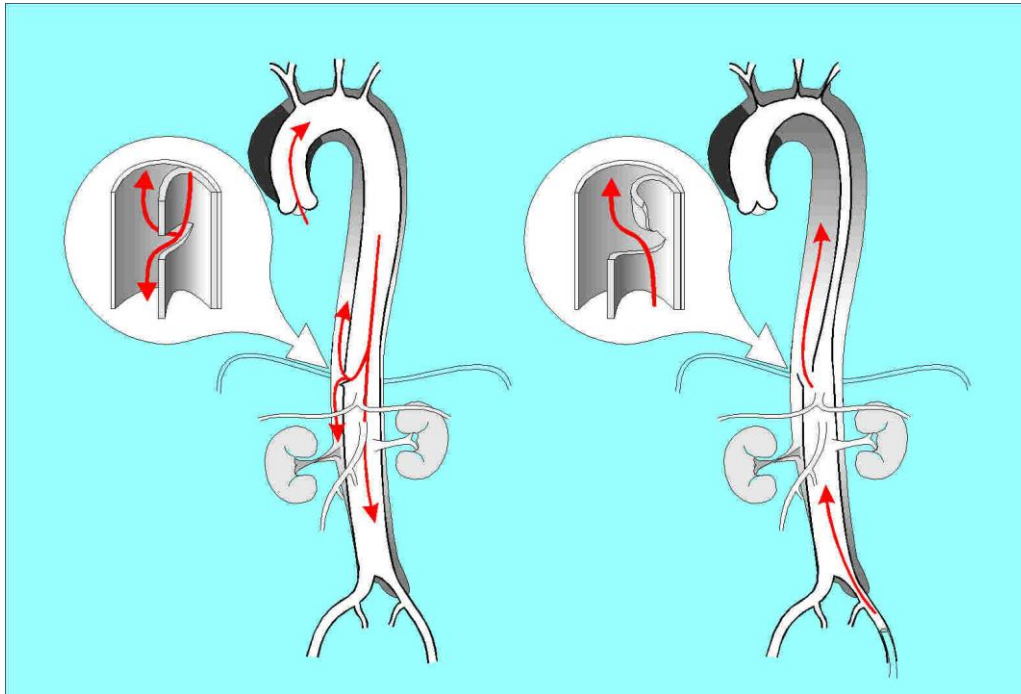


# Access for cannulation

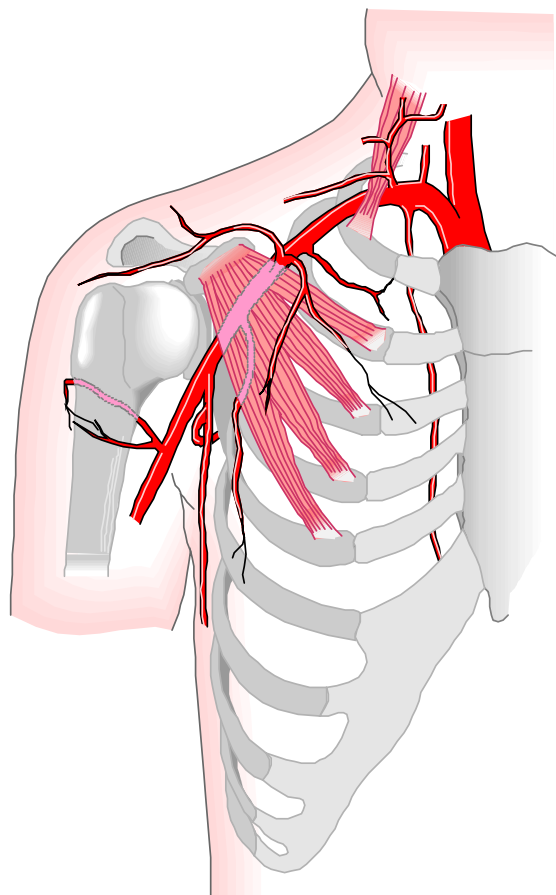


# Problems of femoral artery cannulation

- Malperfusion in dissection
- Embolism of atheroma from the aorta
- Impossible, if iliofemoral occlusion/stenosis is present.

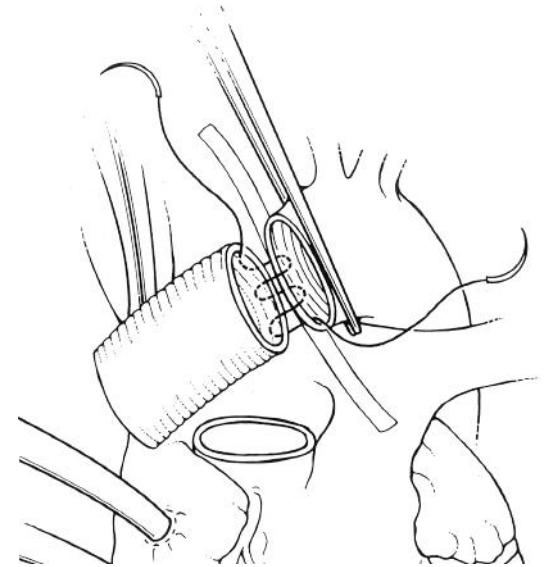
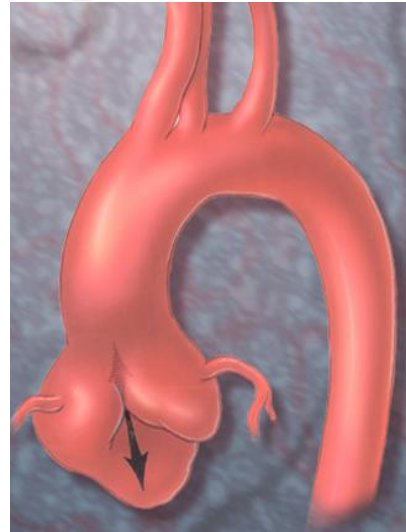


# Right axillary artery cannulation

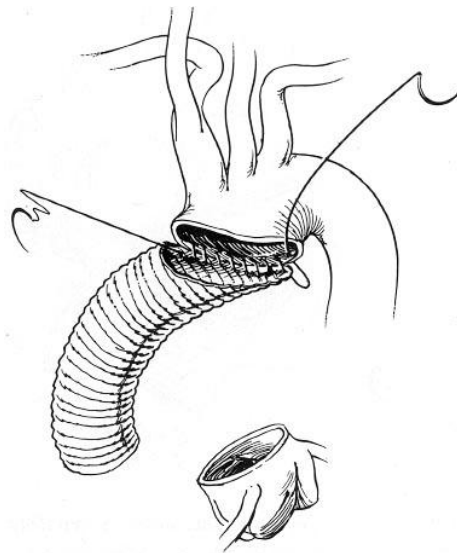
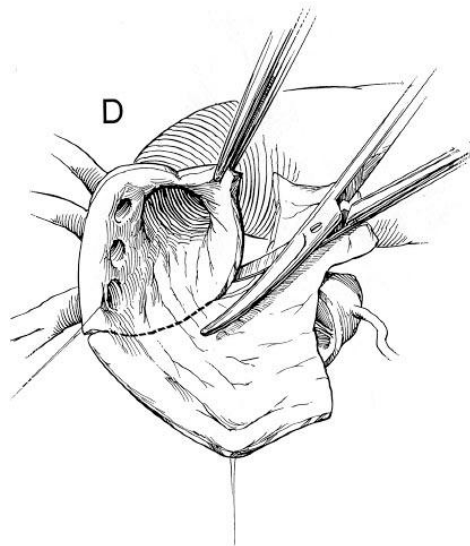


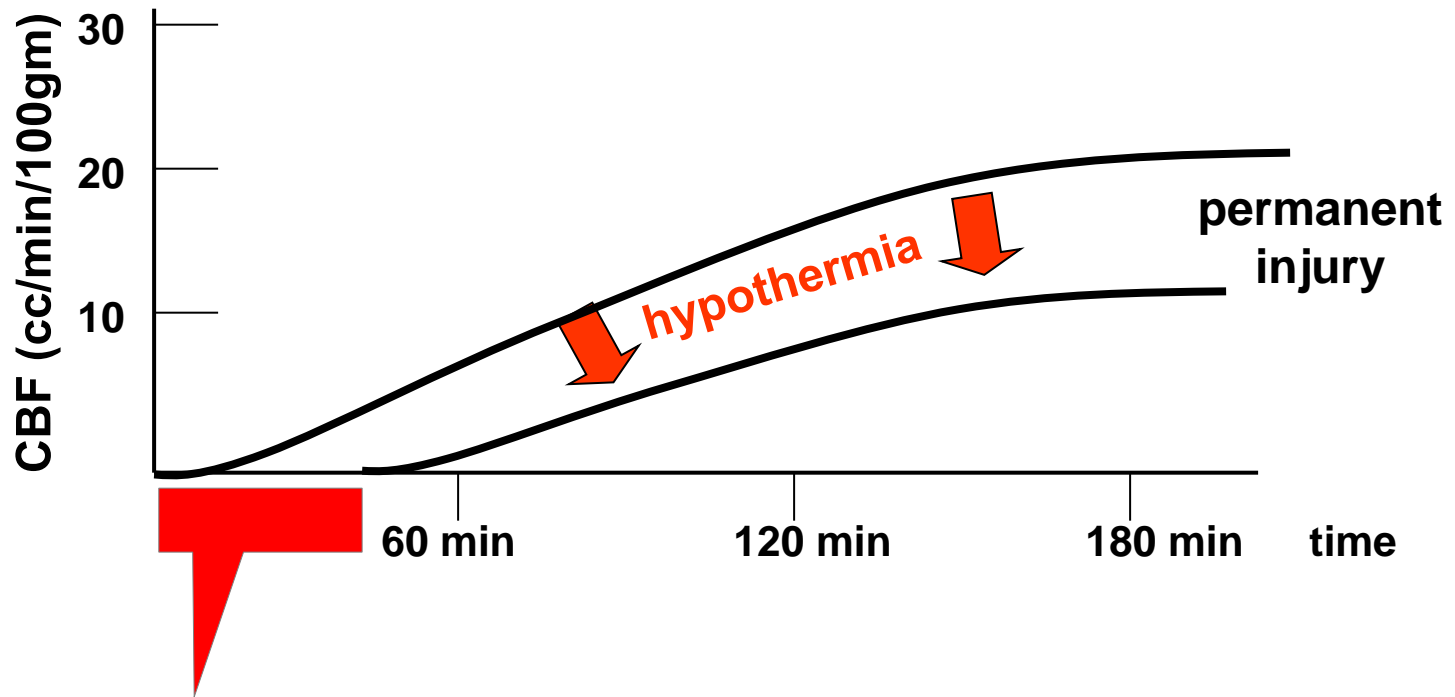
- Rarely involved by atherosclerosis / dissection
- Detour aortic atherosclerosis  
→ much less risk of cerebral embolism
- Enables & simplifies selective cerebral perfusion
  
- Time for exposure
- Anatomic variation
- Nerve injury – brachial plexus
- Lower body perfusion ↓ / cerebral blood flow ↑ (?)  
- in case of small axillary/subclavian/innominate a.

# *Anastomosis under clamp*



# *Open technique*





**40 ~ 50 minutes at brain temperature of 10 ~ 15 °C**

**20 ~ 30 minutes at 20 °C**

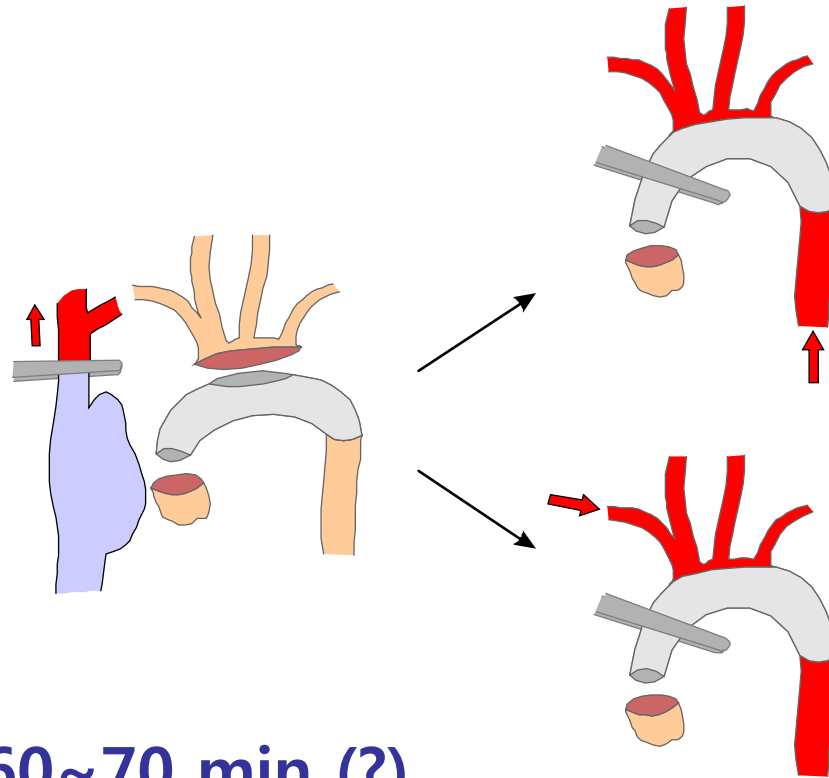
**10 ~ 20 minutes at 25°C**

**5 ~ 10 minutes at 28°C**

# Conventional technique for cerebral protection

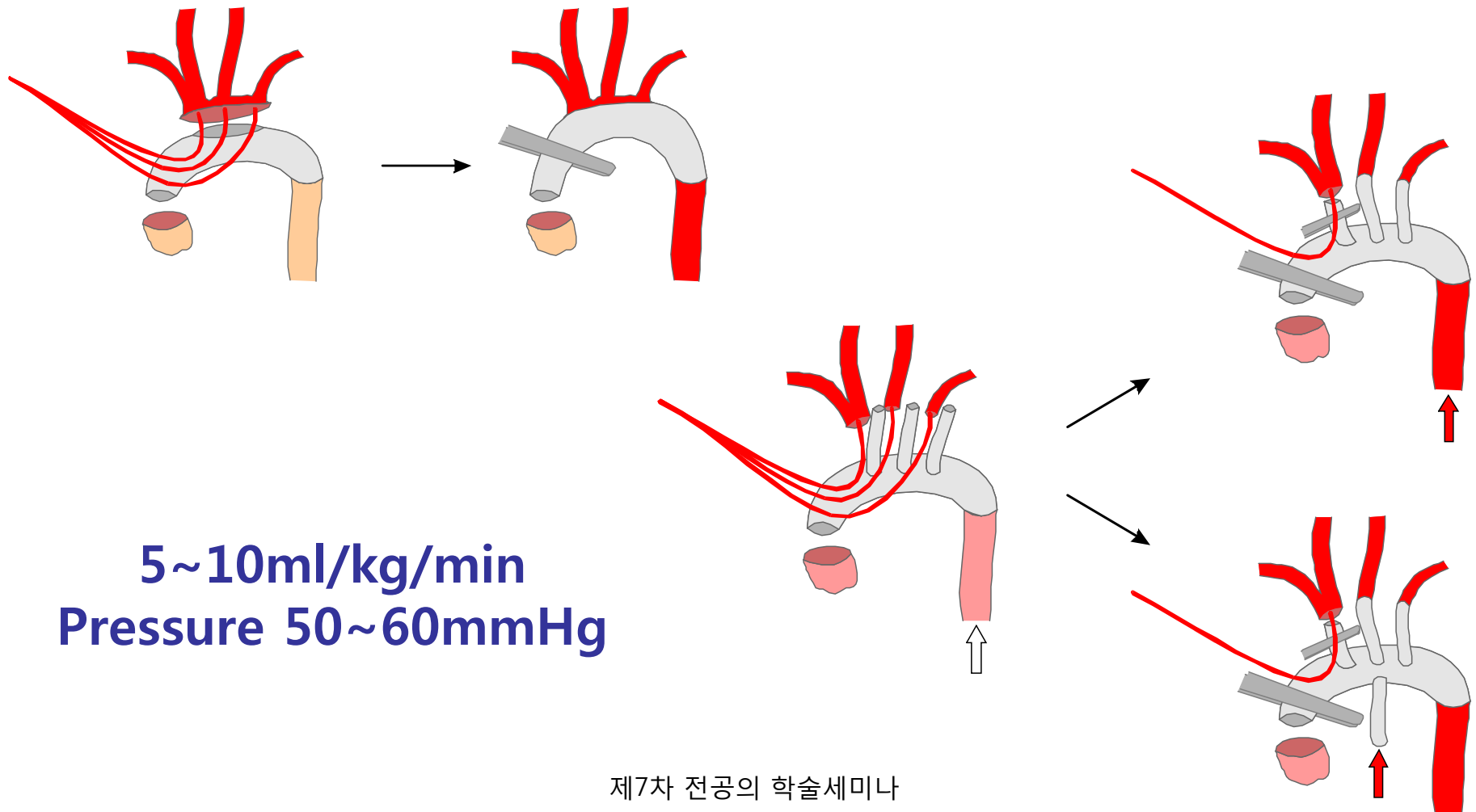
- *hypothermic circulatory arrest ± retrograde cerebral perfusion (RCP)* -

100~300ml/min  
CVP ≤ 20~25mmHg



- 40~50 min →→ 60~70 min (?)
- Back-flushing of air, debris, harmful metabolites
- **Brain edema**
- **Fluid overload**

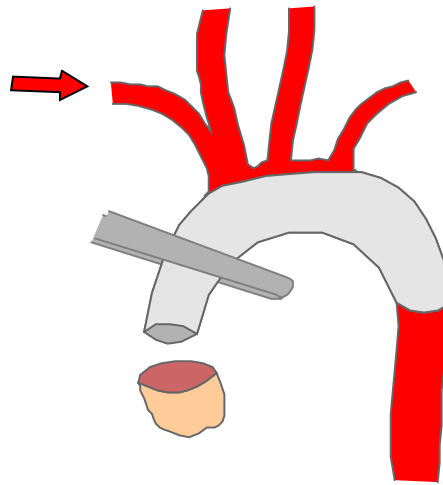
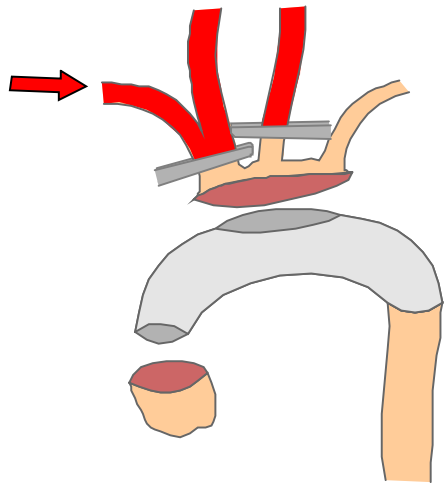
# Selective antegrade cerebral perfusion (SACP, ACP)



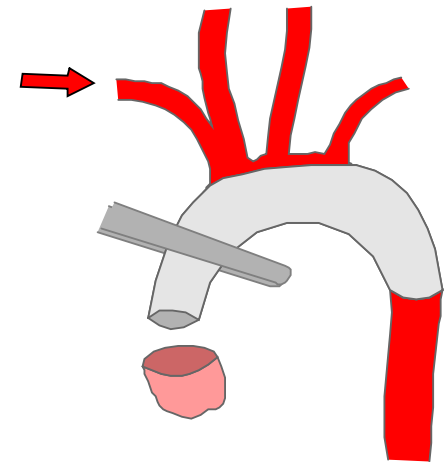
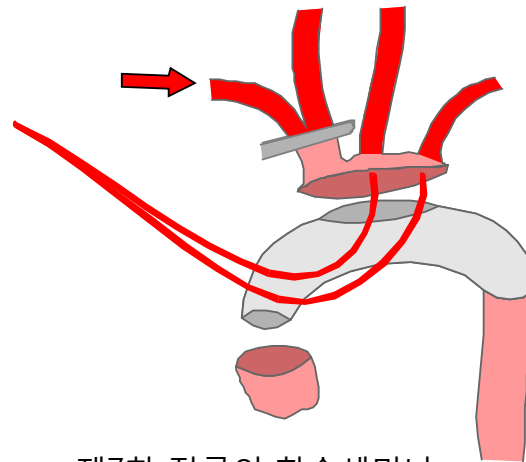
5~10ml/kg/min  
Pressure 50~60mmHg



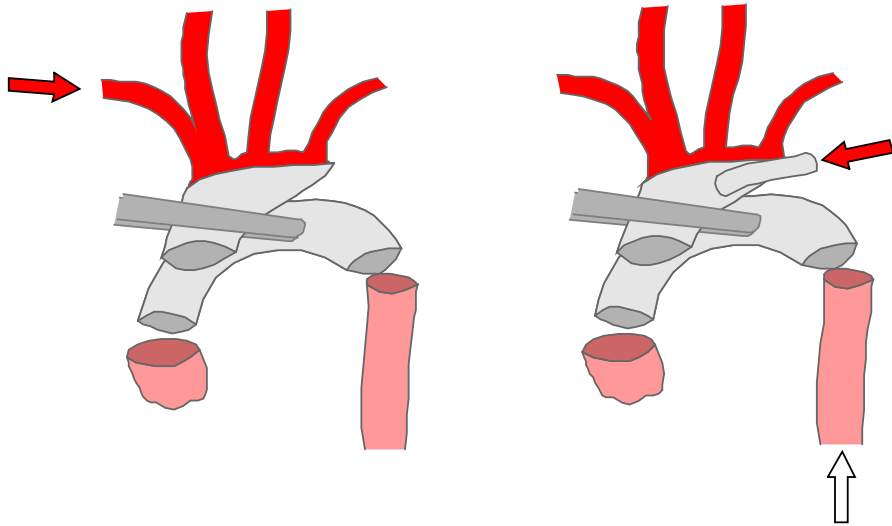
# ACP using right axillary artery



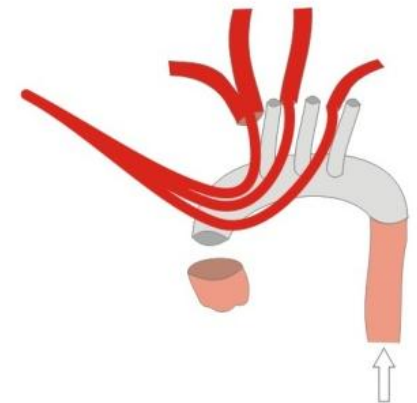
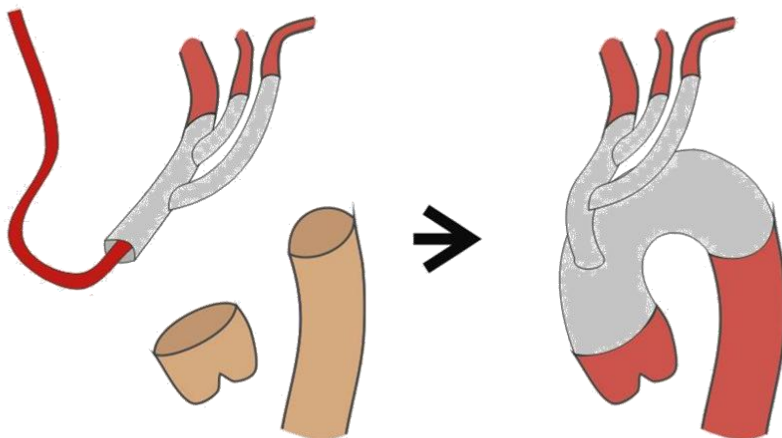
Anatomy  
Back-flow  
Brain oximetry



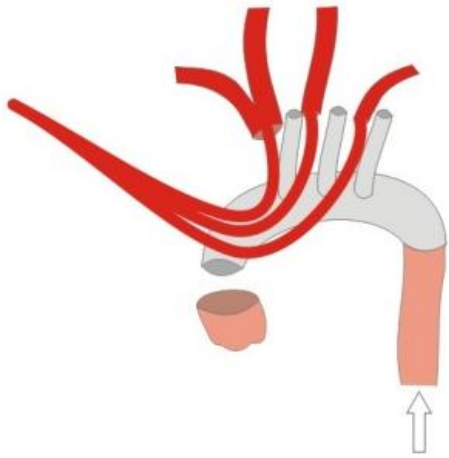
# Shortening of brain ischemia - arch-first technique -



- less crowded field during distal anastomosis
- no need of clamping or ballooning of arch branches
- Need of additional anastomosis (graft-to-graft)

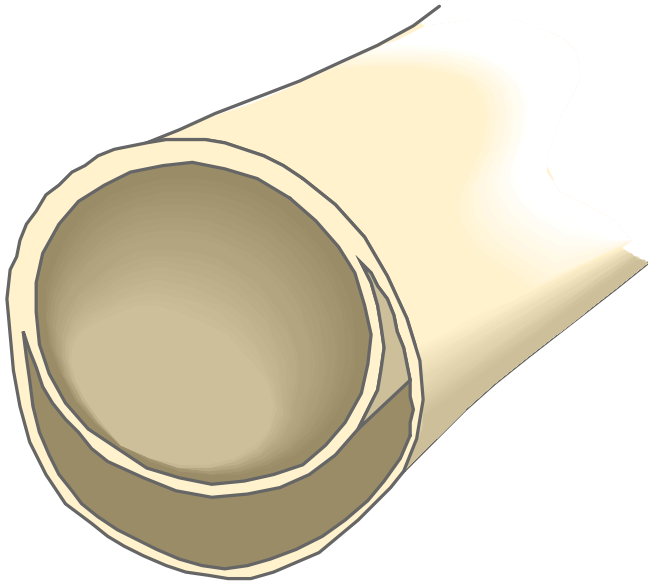


# Options in selective cerebral perfusion



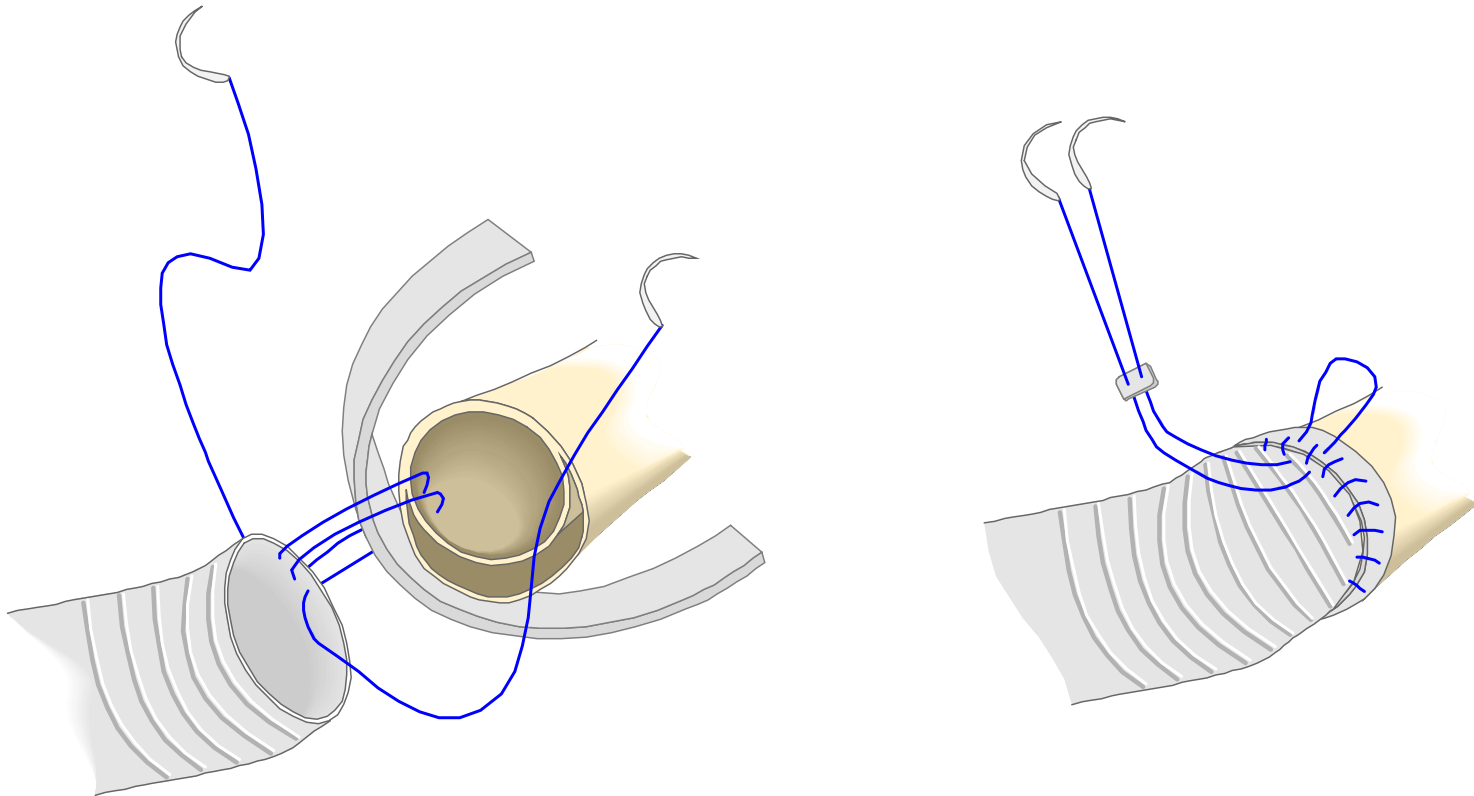
- Routine for ascending / hemiarch replacement vs. only for total arch replacement
- Deep vs. moderate / mild hypothermia (25~30°C)
- Unilateral vs. bilateral perfusion
- Temporary vs. continuous selective perfusion

# Anastomosis & stump re-enforcement

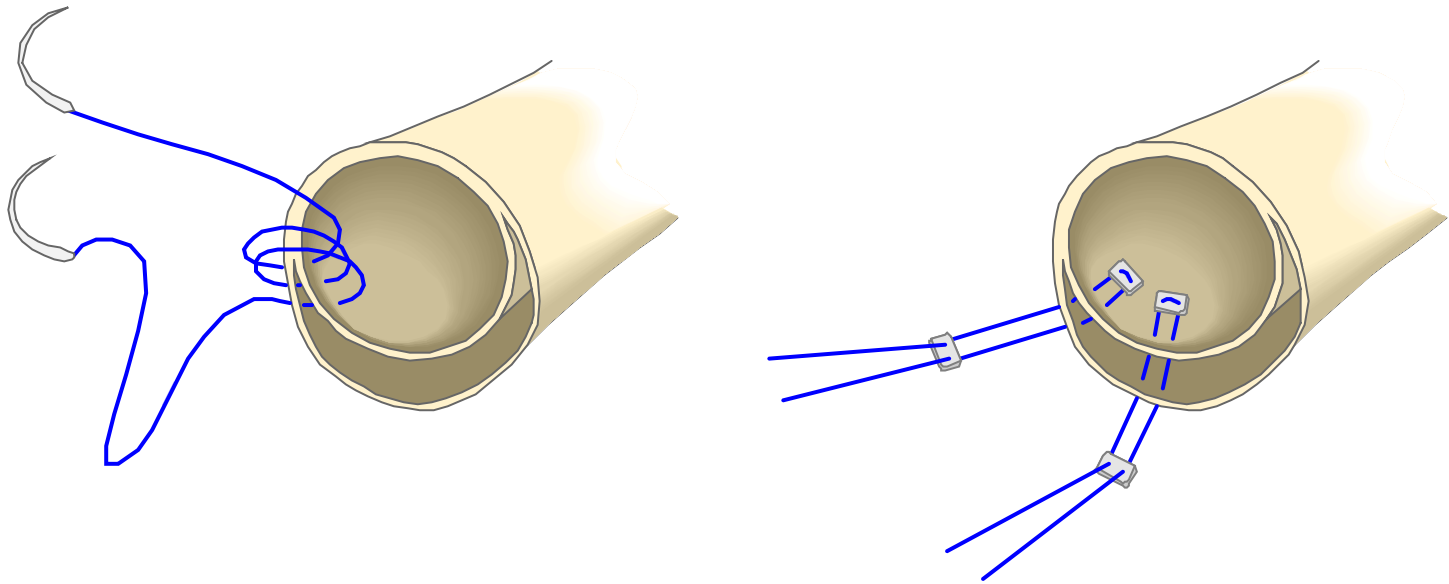


- Hemostasis
- Prevention of pseudoaneurysm
- Obliteration of adjacent false lumen

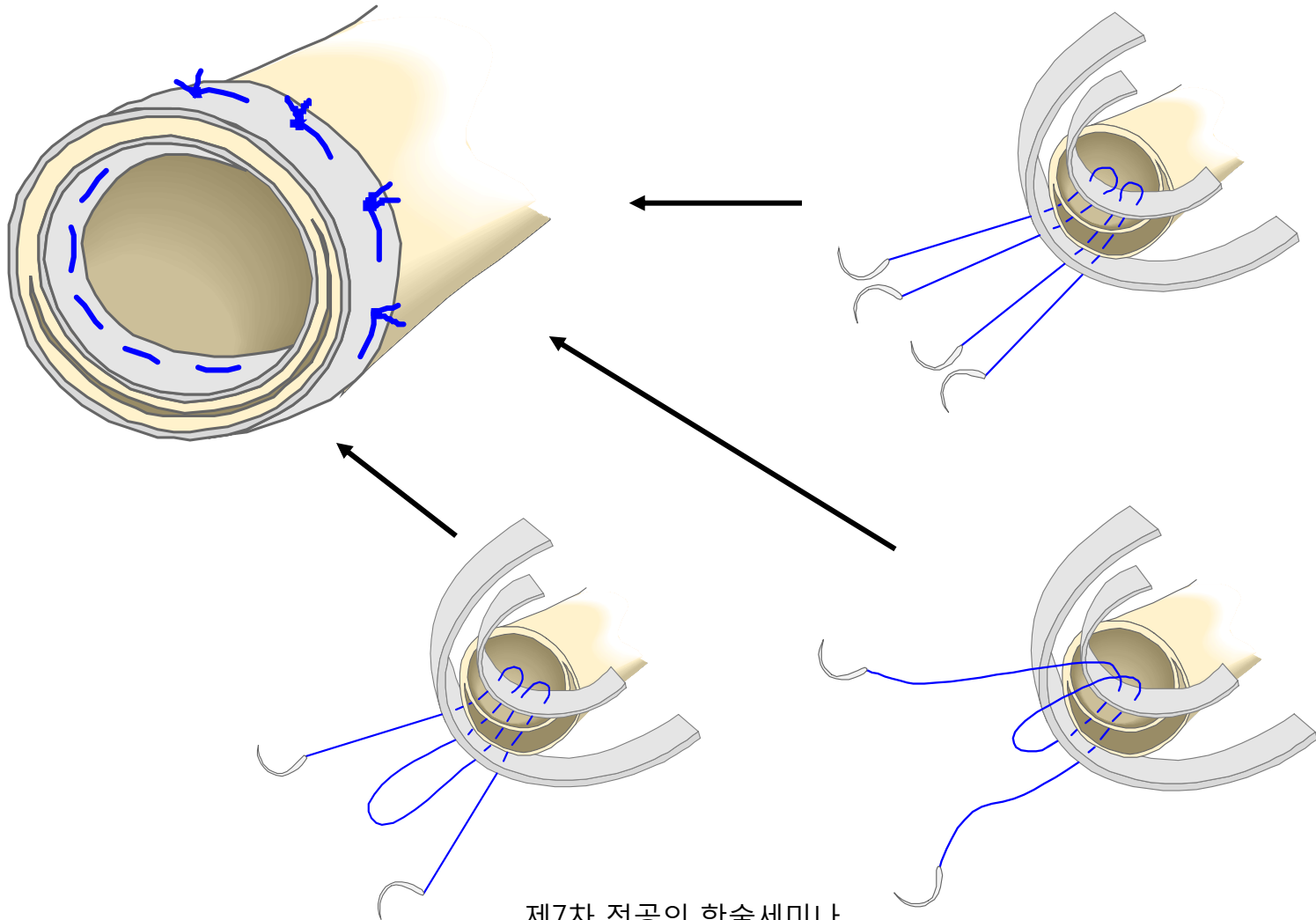
**nothing or outside felt only**  
**→ multiple re-enforcement**



# Simple re-attachment with suture

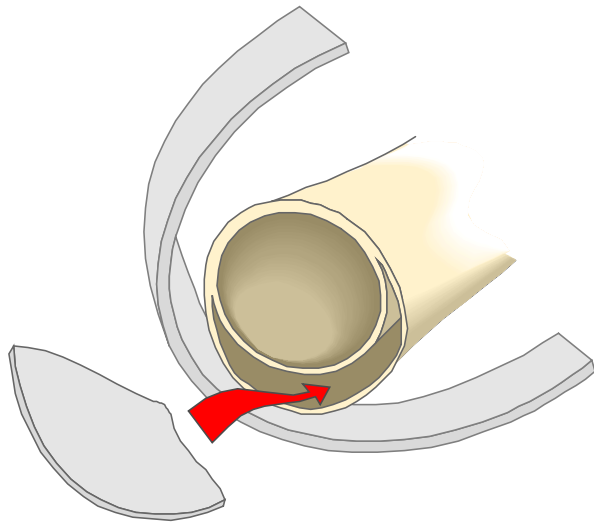


# Sandwich

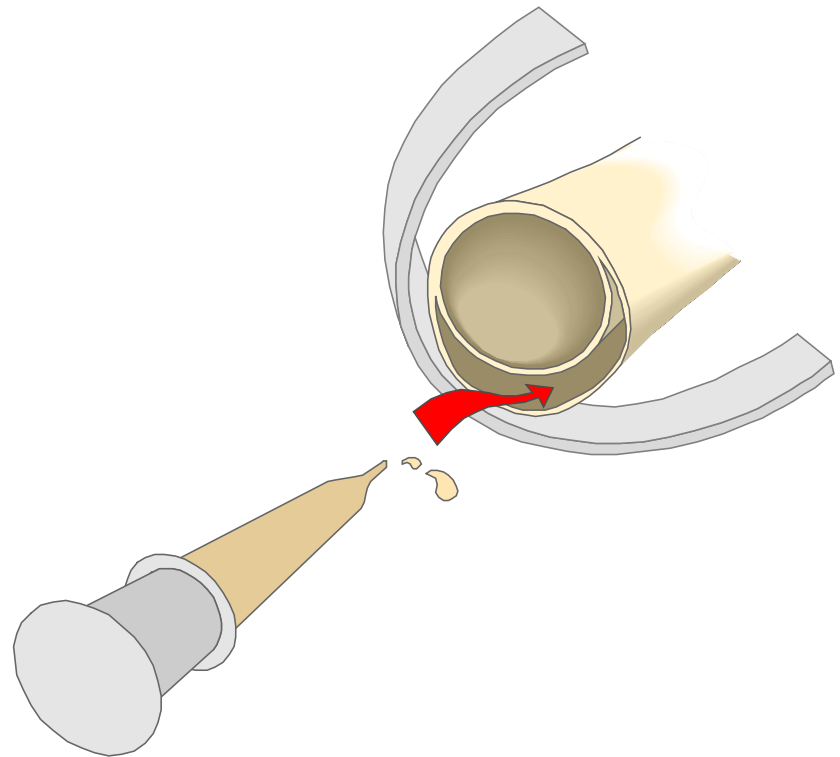


# Neo-media

**Teflon felt patch**

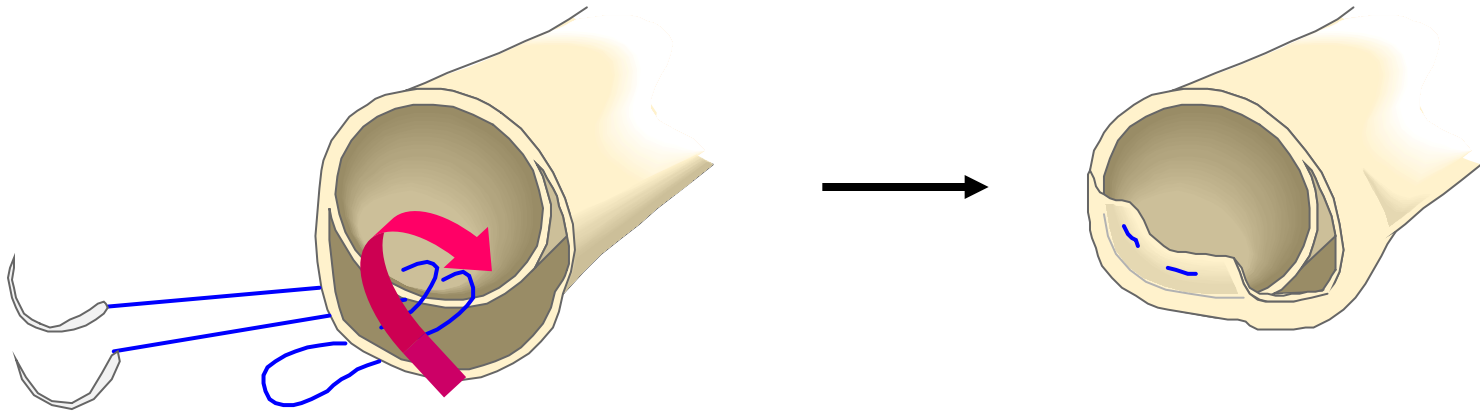


**glue**





# Adventitial inversion



# Controversies regarding IMH

- Is the prognosis as bad as that of the overt dissection?
- Is it really a “dissection without intimal tear”  
caused by rupture vasa vasorum,  
or a “thrombosed dissection without a visible tear”?

# Different Clinical Features of Aortic Intramural Hematoma Versus Dissection Involving the Ascending Aorta

Jae-Kwan Song, MD, FACC,\* Hyun-Sook Kim, MD,\* Duk-Hyun Kang, MD,\* Tae-Hwan Lim, MD,†

the mortality rate with medical treatment was much lower in patients with AIH than it was in patients with AD (6% vs. 58%,  $p = 0.003$ ). In follow-up imaging studies of 13 patients who survived AIH without surgical repair, seven patients showed complete resolution. Typical AD developed in three patients, and the other three patients showed focal AD only in the descending aorta. The two-year survival rate did not show significant difference ( $84\% \pm 6\%$  in AIH vs.  $76\% \pm 17\%$  in AD,  $p = 0.47$ ).

## CONCLUSIONS

Absence of continuous flow communication can explain a more favorable clinical course of AIH than for AD, and medical treatment with frequent imaging follow-up and timed elective surgery in cases with complications can be a rational option for patients with proximal AIH. (J Am Coll Cardiol 2001;37:1604–10) © 2001 by the American College of Cardiology

---

## Acute Aortic Intramural Hematoma

### An Analysis From the International Registry of Acute Aortic Dissection

Kevin M. Harris, MD; Alan C. Braverman, MD; Kim A. Eagle, MD; Elise M. Woznicki, BS;

in-hospital mortality was not statistically different for type A IMH compared to AD (26.6% versus 26.5%;  $P=0.998$ ); type A IMH managed medically had significant mortality (40.0%), although less than classic AD (61.8%;  $P=0.195$ ). Patients with type B IMH had a hospital mortality that was less but did not differ significantly (4.4% versus 11.1%;  $P=0.062$ ) from classic AD. One-year mortality was not significantly different between AD and IMH.

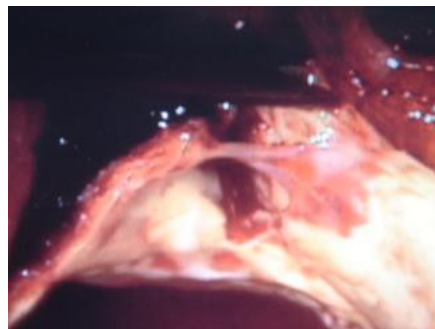
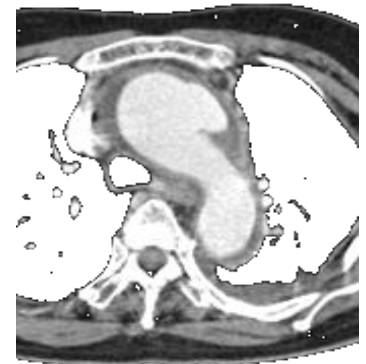
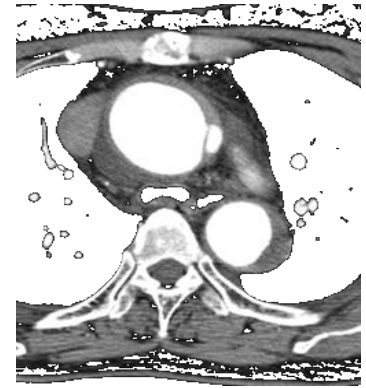
**Conclusions**—Acute IMH has similar presentation to classic AD but is more frequently complicated with pericardial effusions and periaortic hematoma. Patients with IMH have a mortality that does not differ statistically from those with classic AD. A small subgroup of type A IMH patients are managed medically and have a significant in-hospital mortality. (Circulation. 2012;126[suppl 1]:S91–S96.)

# Prevalence of Aortic Intimal Defect in Surgically Treated Acute Type A Intramural Hematoma

Kay-Hyun Park, MD, PhD, Cheong Lim, MD, PhD, Jin Ho Choi, MD, Kiick Sung, MD, Kwhanmien Kim, MD, PhD, Young Tak Lee, MD, PhD, and Pyo Won Park, MD, PhD

Department of Thoracic and Cardiovascular Surgery, Seoul National University Bundang Hospital, Gyeonggi-do, and Department of Thoracic and Cardiovascular Surgery, Sungkyunkwan University School of Medicine, Samsung Medical Center, Seoul, Korea

**Results.** In 18 patients (48.6%), intimal defects were suggested in preoperative computed tomography (CT). During surgery, 27 patients (73.0%) had small intimal defects in the ascending aorta or arch, while 14 of them (51.9%) did not have preoperative CT findings suggestive



# Acute aortic syndrome

## - *conventional principle* -

complicated

uncomplicated

### ● Dissection

■ Type A

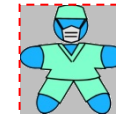


■ Type B



### ● IMH

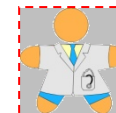
■ Type A



■ Type B



### ● PAU

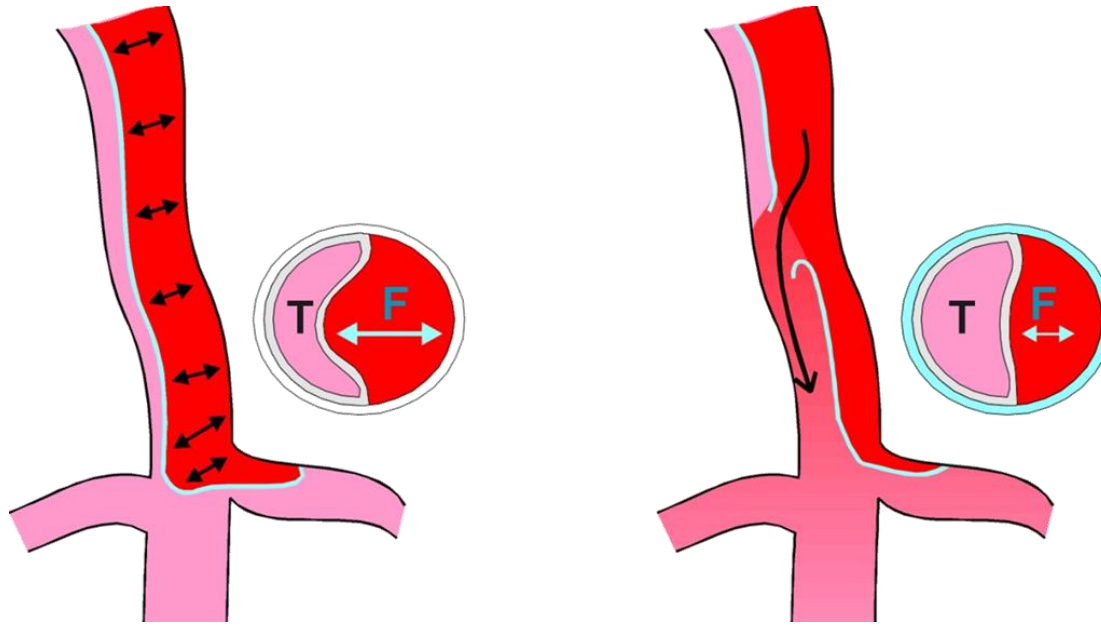


# Acute aortic syndrome

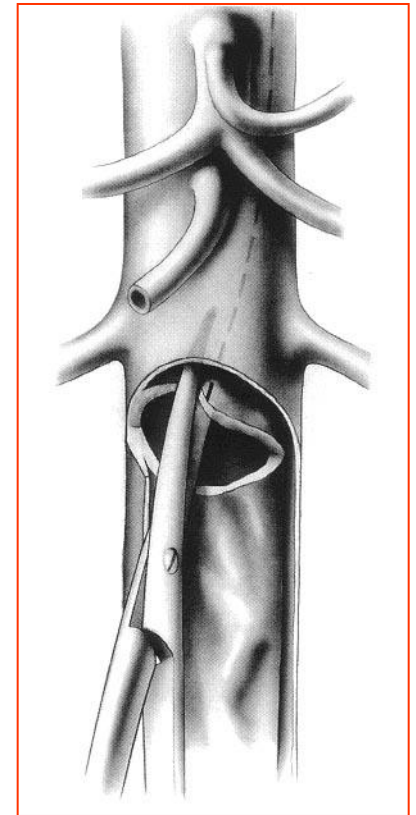
## - *“complicated”* -

- **(Impending) rupture**
  - **periaortic hematoma / sanguineous effusion**
- **Branch vessel obstruction/compromise**
- Resistant hypertension
- Persistent pain
- Aortic growth  $\geq 5\text{mm}$  within 3 months
- Total aortic diameter  $\geq 40\text{mm}$

# Fenestration

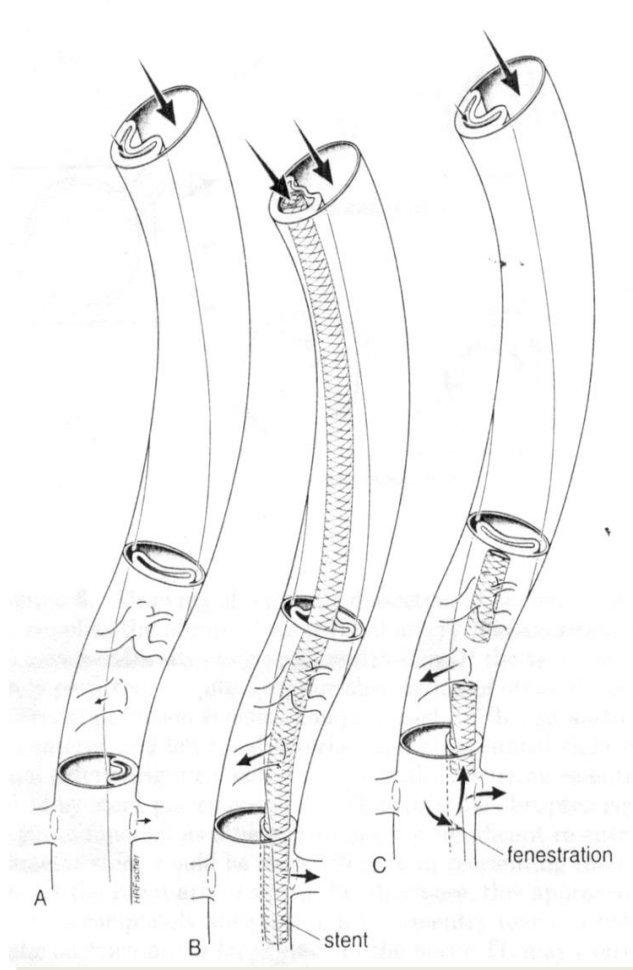
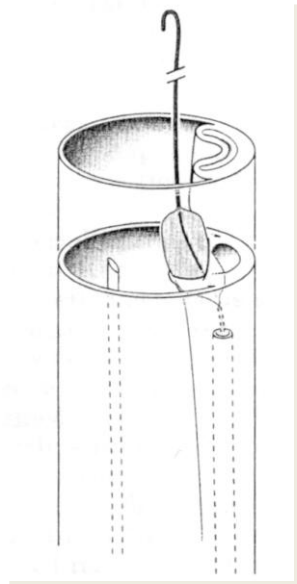
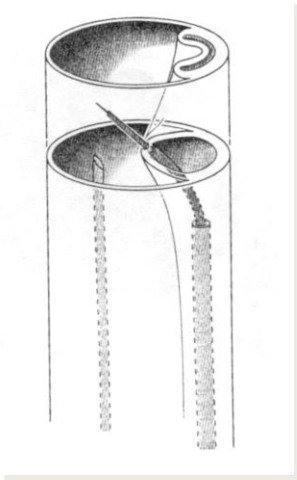


- Decompression of false lumen
  - halt distal progression of dissection
  - prevent rupture
- Re-establishment of flow to distal aorta & branches



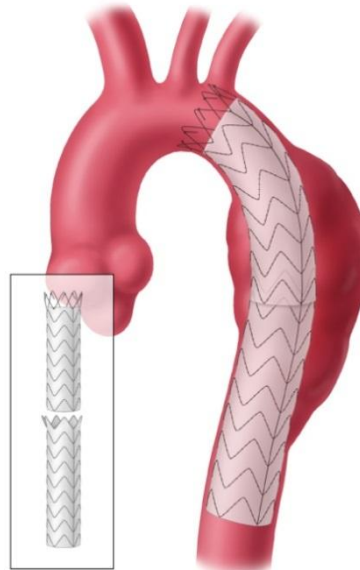
# Stenting of true lumen

## Endovascular fenestration

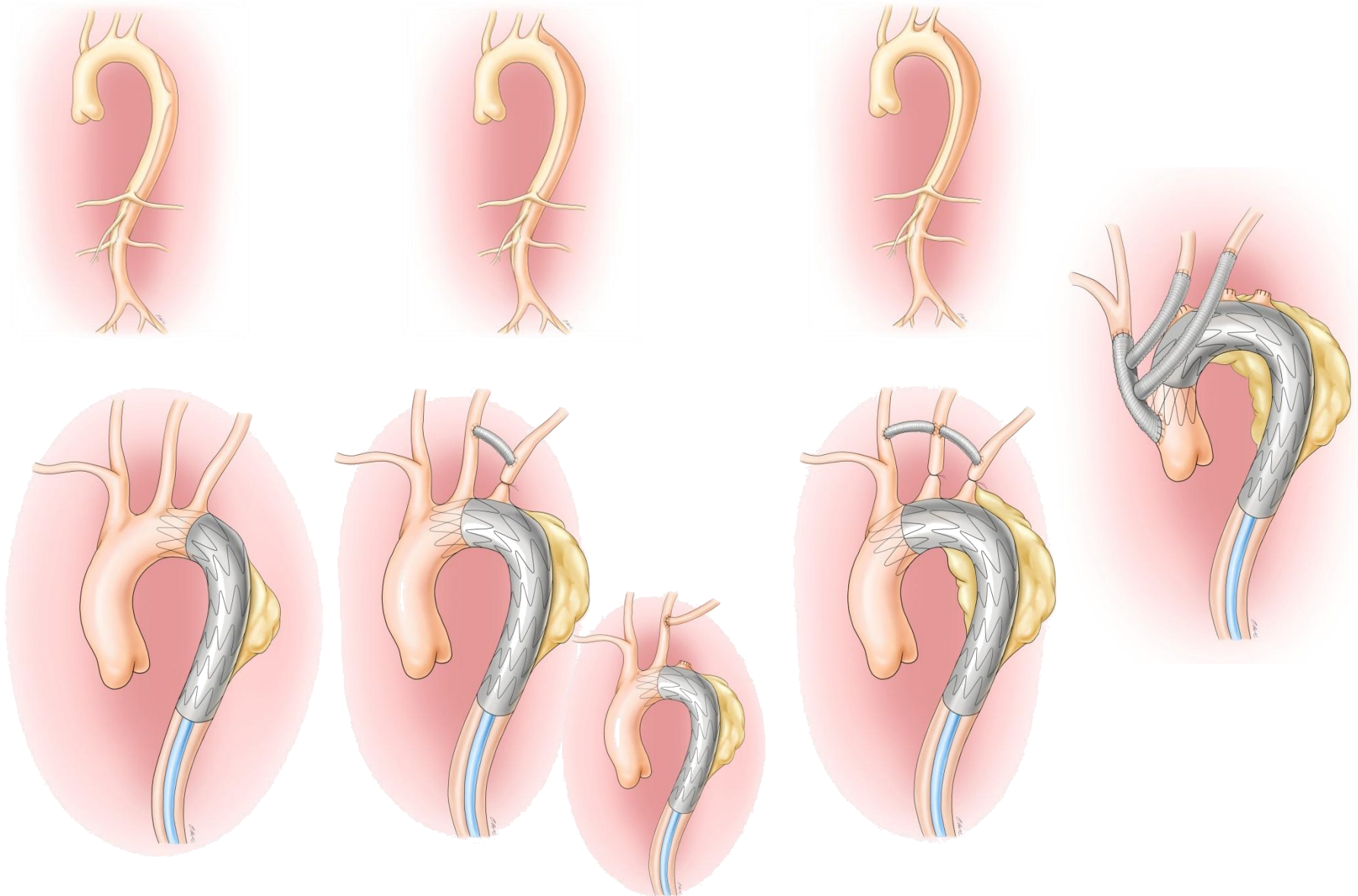




# Thoracic EndoVascular Aortic Repair



# Hybrid TEVAR with arch debranching



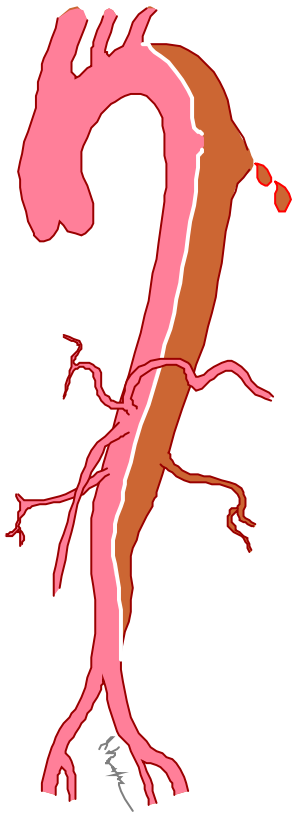
# TEVAR in type B dissection

**complicated** acute dissection

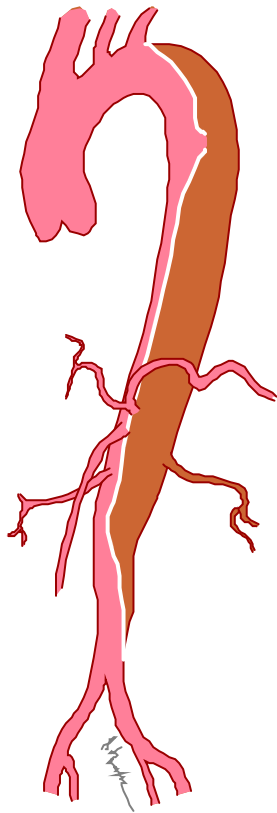
exclusion of  
intimal tear



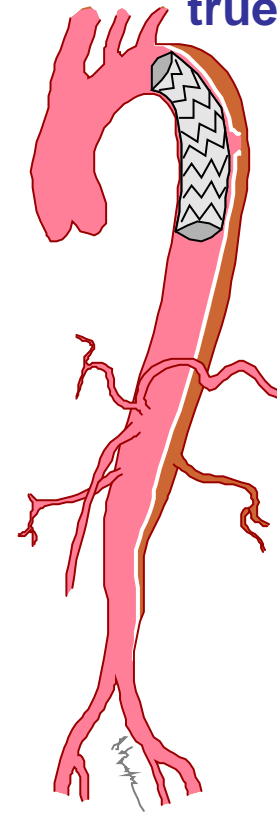
prevention  
of rupture &  
true lumen expansion



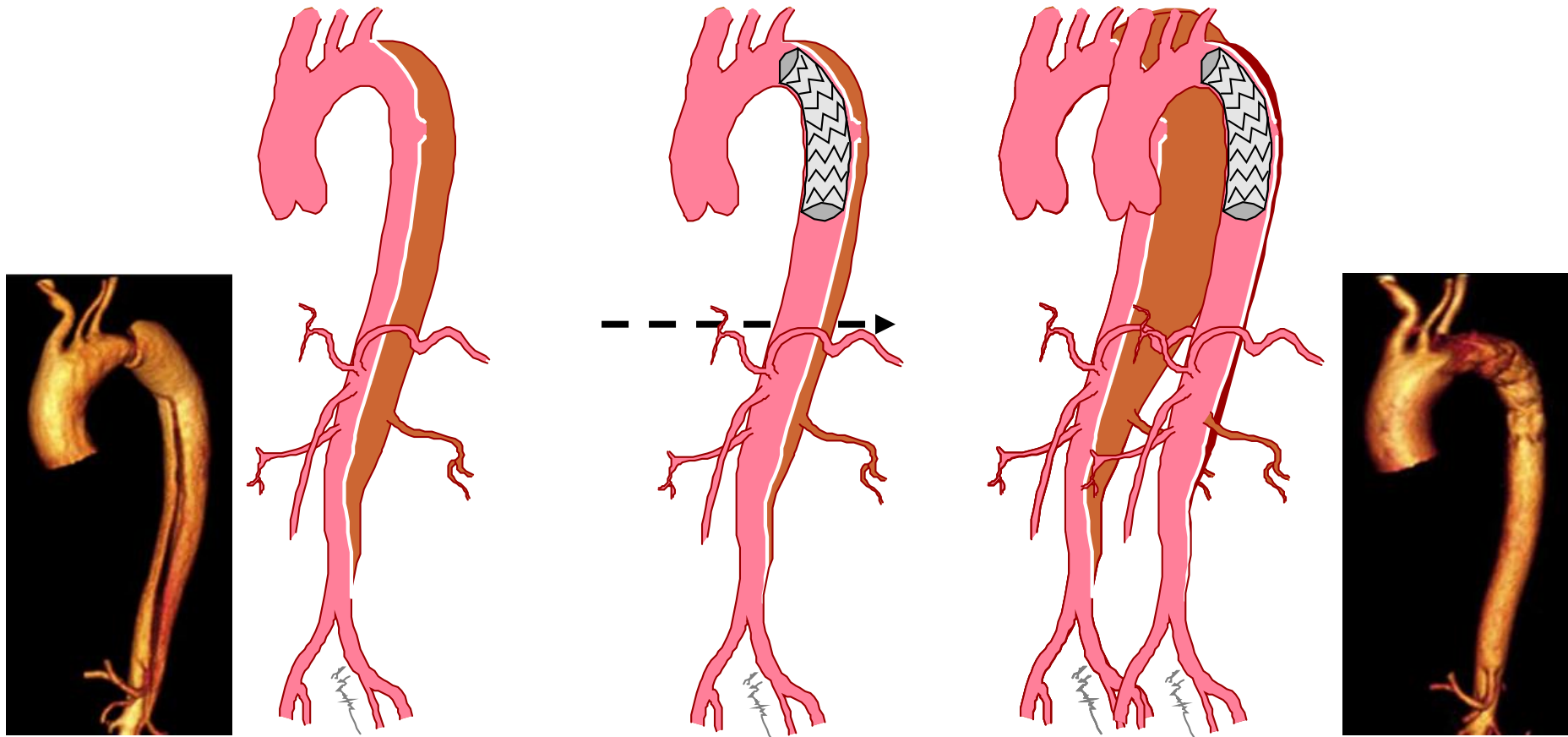
*rupture*



*malperfusion*



# Rationale of TEVAR in **uncomplicated** dissection



# However, the reality is that

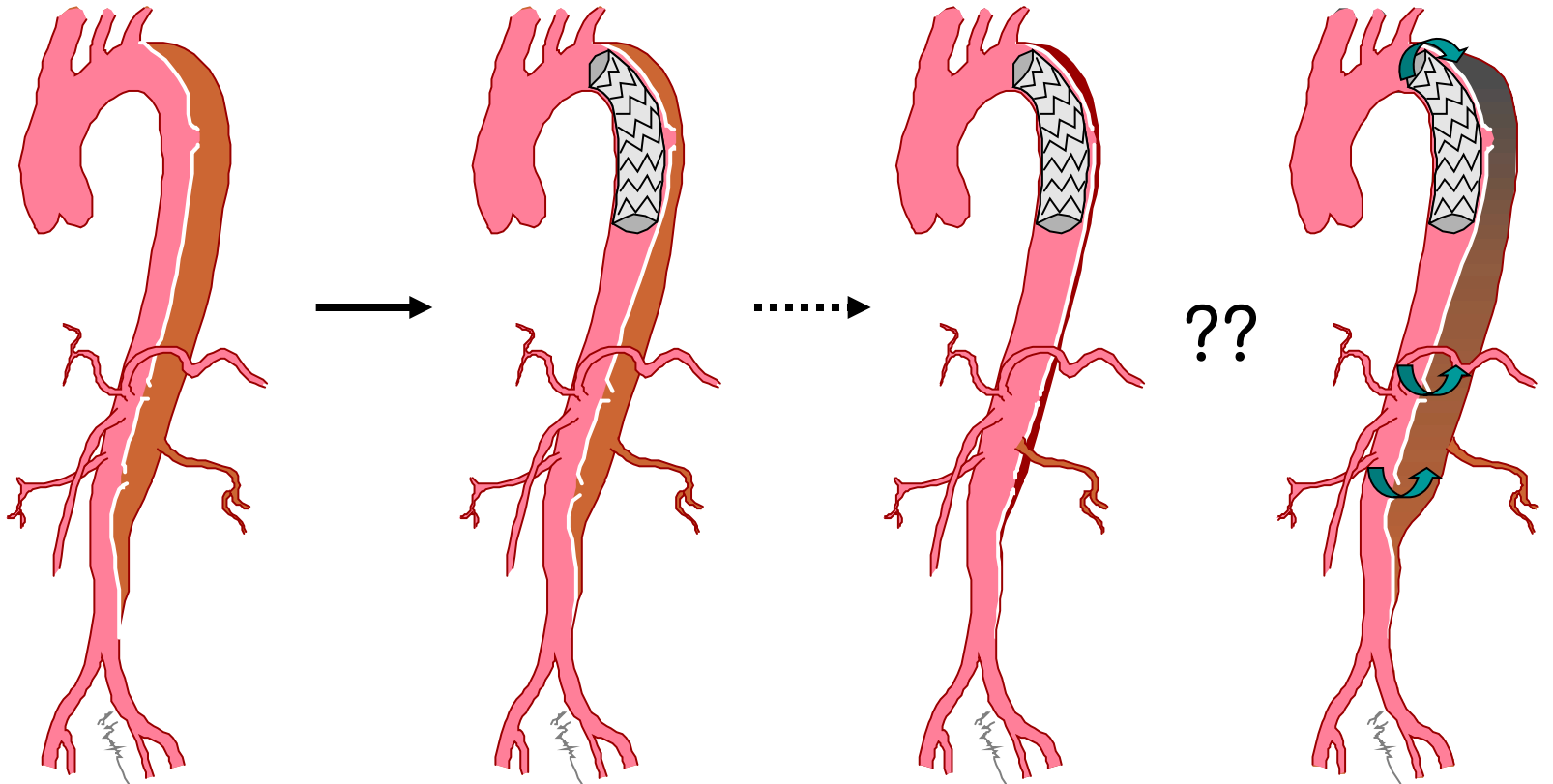
**TEVAR**

≤ 80% in acute

≤ 50% in chronic

*Type I endoleak*

*Distal re-entry tears*



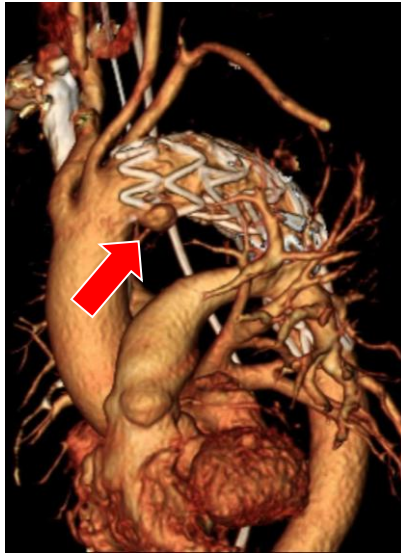
# Problems/complications of TEVAR

---

- Mortality 1.5~6.5%
- Procedural failure 1~5%
- Conversion to open surgery up to 5%
- Retrograde type A dissection 1.9~2.5%
- Endoleak 0~45%
- Late mechanical failure (fracture/breakage/wear) up to 9%
- Renal failure 5.2~13%
- Vascular access problems
  - need of iliac conduit up to 40%
  - serious injury to iliofemoral arteries 1.4~14%
- Neurological complications
  - stroke 2.9~11%
  - paraplegia 2~5%

# Post-TEVAR surgery

*retrograde dissection*



*pseudoaneurysm*

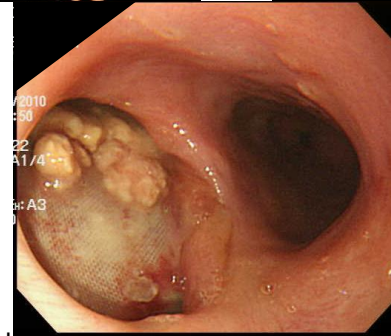
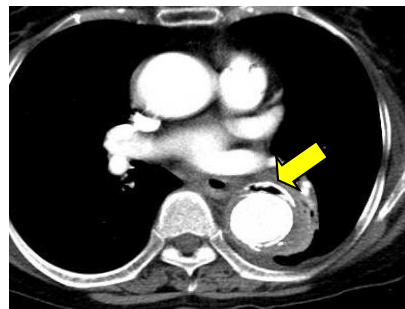
*distal erosion – aneurysm / rupture*



*residual aneurysm*



*aortoesophageal fistula*



# Secondary surgical procedures after TEVAR

---

- Talent registry (*J Thorac Cardiovasc Surg* 2006;132:332-9)
  - 457 patients, freedom from 2<sup>nd</sup> procedure = 81% / 3 years, 70% / 5 years
- Talent registry (*J Thorac Cardiovasc Surg* 2008;135:1322-6)
  - 422 patients, median F/U 17 months
  - Conversion to surgery in 3.8%, hospital mortality 6.2%
- Heidelberg (*J Vasc Surg* 2011)
  - 47 patients of **hybrid TEVAR** (1997~2009)
  - 19% in-hospital mortality, 27.6% 2<sup>nd</sup> procedure, 6.3% open conversion
- U Penn (*J Thorac Cardiovasc Surg* 2013;145:S165-70)
  - 680 TEVAR (2000~2011) → 60 2<sup>nd</sup> TEVAR + 20 surgery
  - 8.7% 30-day mortality
- Kobe (*Ann Thorac Surg* 2013;95:1584-90)
  - 147 TEVAR (2000~2011) → 10 2<sup>nd</sup> TEVAR + 9 surgery
  - 11.5% in-hospital mortality



# Surgery after (T)EVAR

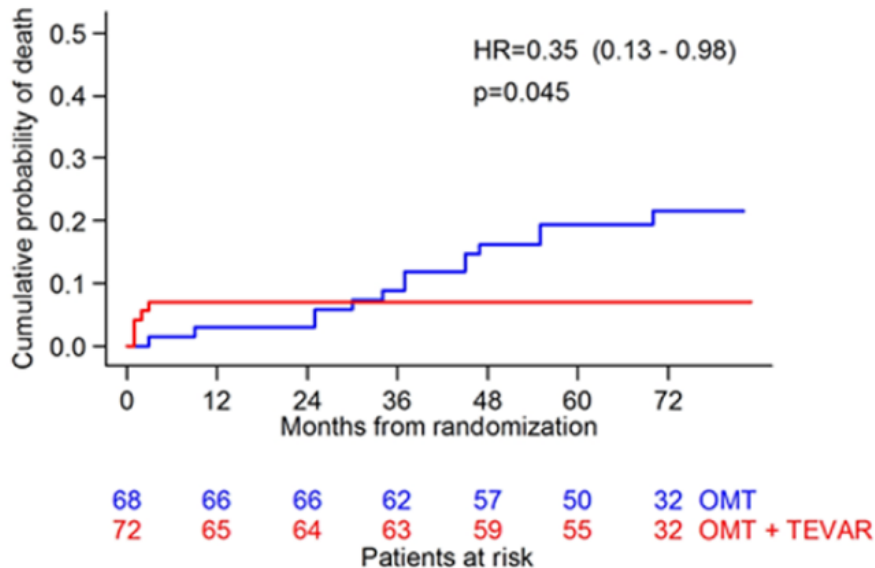
- 2<sup>nd</sup> procedure is not rare after TEVAR,  $\geq 20\%$  of them should be open surgery. In most cases, they are more challenging than primary surgery.
- Causative factors
  - *Heroic TEVAR for marginal anatomy and/or debatable indication*
  - *Procedural / early success  $\neq$  clinical / long-lasting stabilization*  
*e.g., late endoleak, adjacent aneurysm, infection, erosion (fistula)*



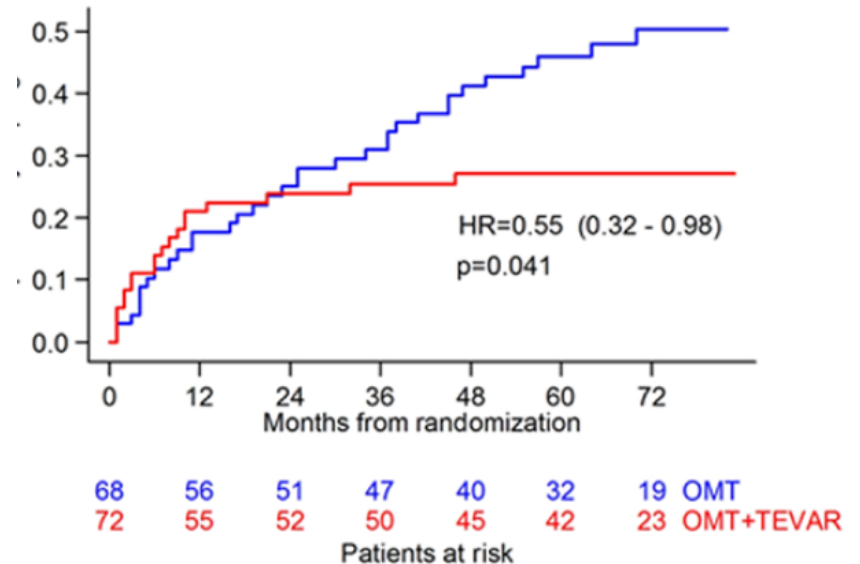
# INSTEAD trial

## Endovascular Repair of Type B Aortic Dissection Long-term Results of the Randomized Investigation of Stent Grafts in Aortic Dissection Trial

### Aorta-specific mortality



### Adverse events



# Acute aortic syndrome

*- standard in endovascular era -*

complicated

uncomplicated

## ● Dissection

■ Type A

■ Type B

## ● IMH

■ Type A

■ Type B

## ● PAU

