Dissection Aortique :
prise en charge
et devenir des patients

Sakalihasan Natzi MD,PhD

Department of Cardiovascular and Thoracic Surgery

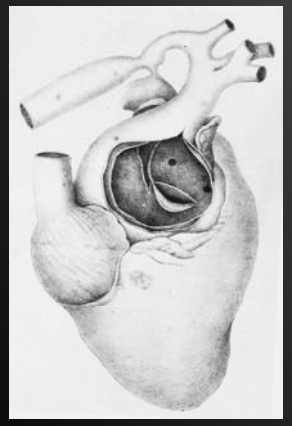
University hospital of Liège,

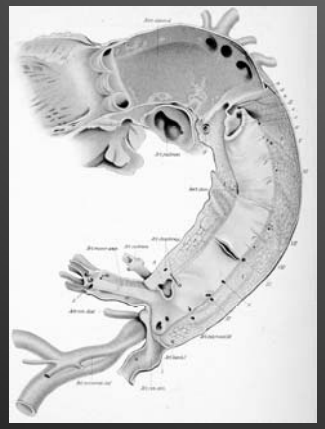
Experimental Research Center of the Cardiovascular Surgery Department, GIGA-Cardiovascular Science Unit, University of Liège,

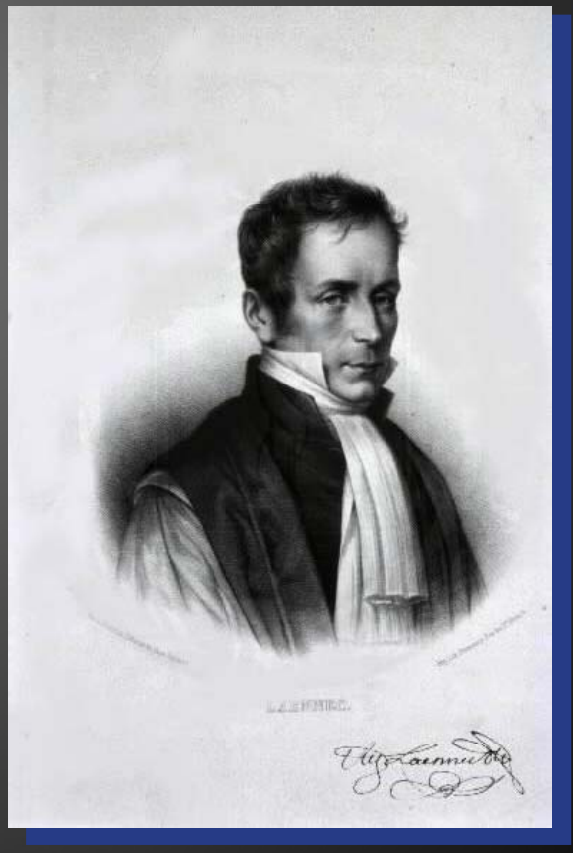
Liège, BELGIUM

DISSECTION. Sens général : action d'isoler les différents éléments constitutifs d'un organe ou d'une région pour en faciliter l'étude anatomique ou pour pratiquer un acte chirurgical.

DISSECTION AORTIQUE. ***Anévrisme disséquant de l'aorte (Laennec, 1819), hématome disséquant de l'aorte (Gonin, 1958).*** Clivage longitudinal de la paroi aortique au niveau de la média, quelle qu'en soit la cause, s'étendant sur tout ou partie de sa circonférence et de sa longueur.







l'anévrysme
est une maladie **chronique**

la dissection aortique aiguë (anév. disséquant)
est une catastrophe **aiguë** qui suppose

- un terrain favorable : la médianécrose
- une fracture pariétale : l'orifice d'entrée
- un élément moteur : la pression artérielle

(elle survient aussi bien sur une aorte de calibre normal que sur une aorte anévrysmale)

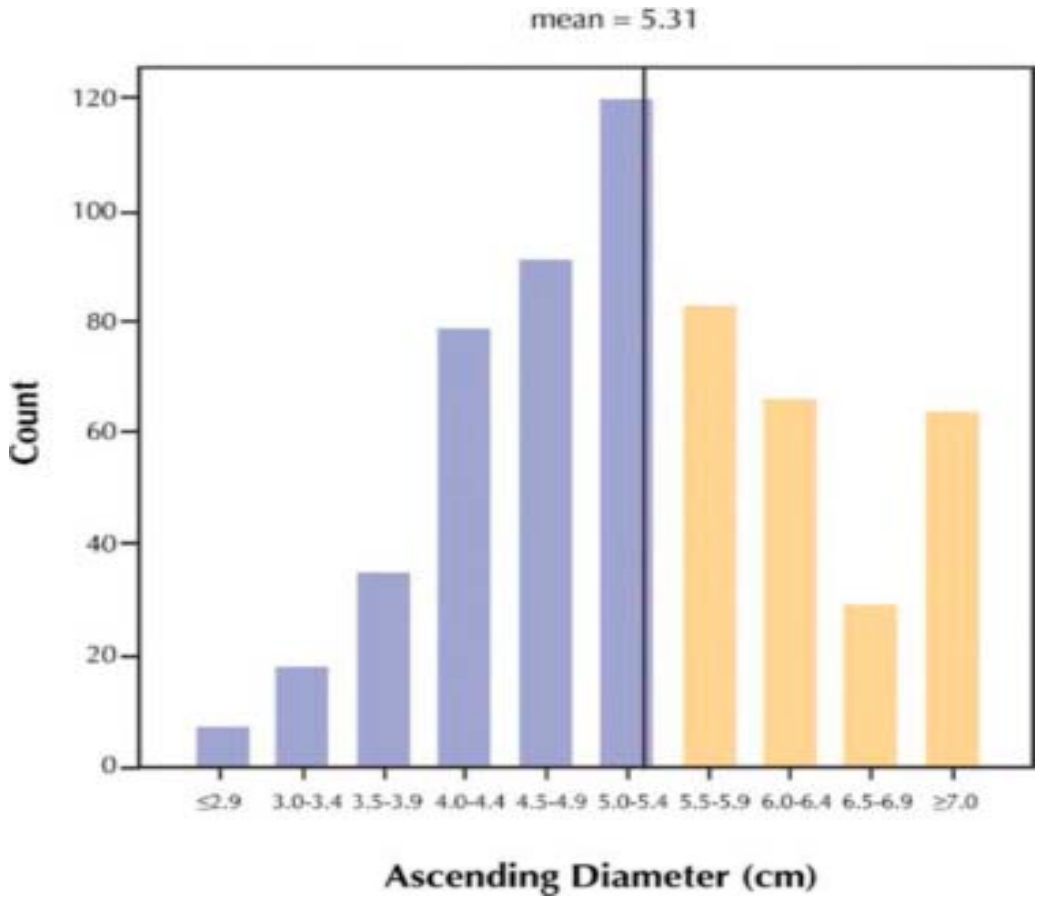


Figure 1. Distribution of aortic sizes at the time of presentation with acute Type A aortic dissection (cm). Purple bars indicate patients with diameters <5.5 cm. Data from IRAD by Pape et al.13 (Adapted with permission from Elefteriades and Farkas.12) (Col...

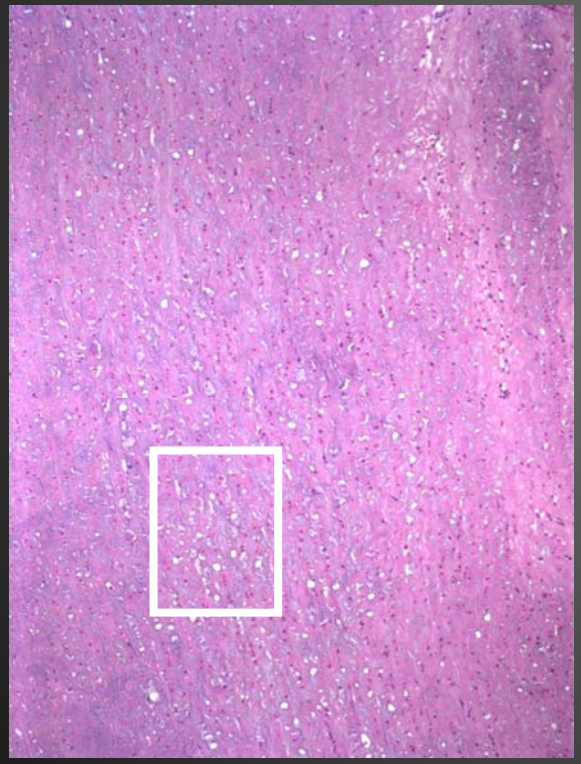
Bulat A. Ziganshin, John A. Elefteriades

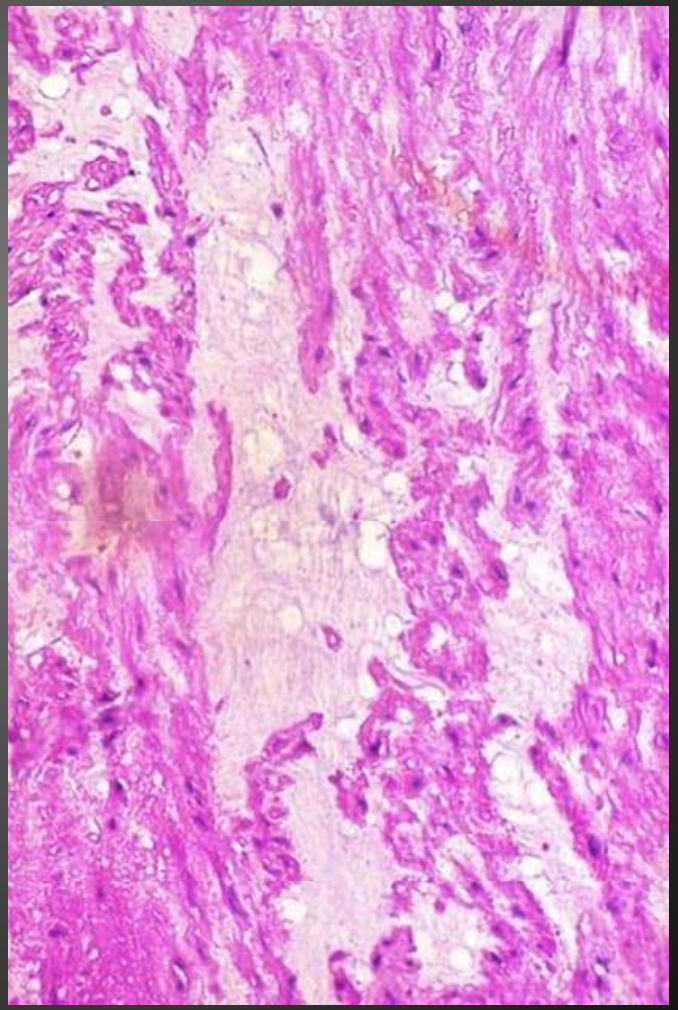
Treatment of Thoracic Aortic Aneurysm: Role of Earlier Intervention

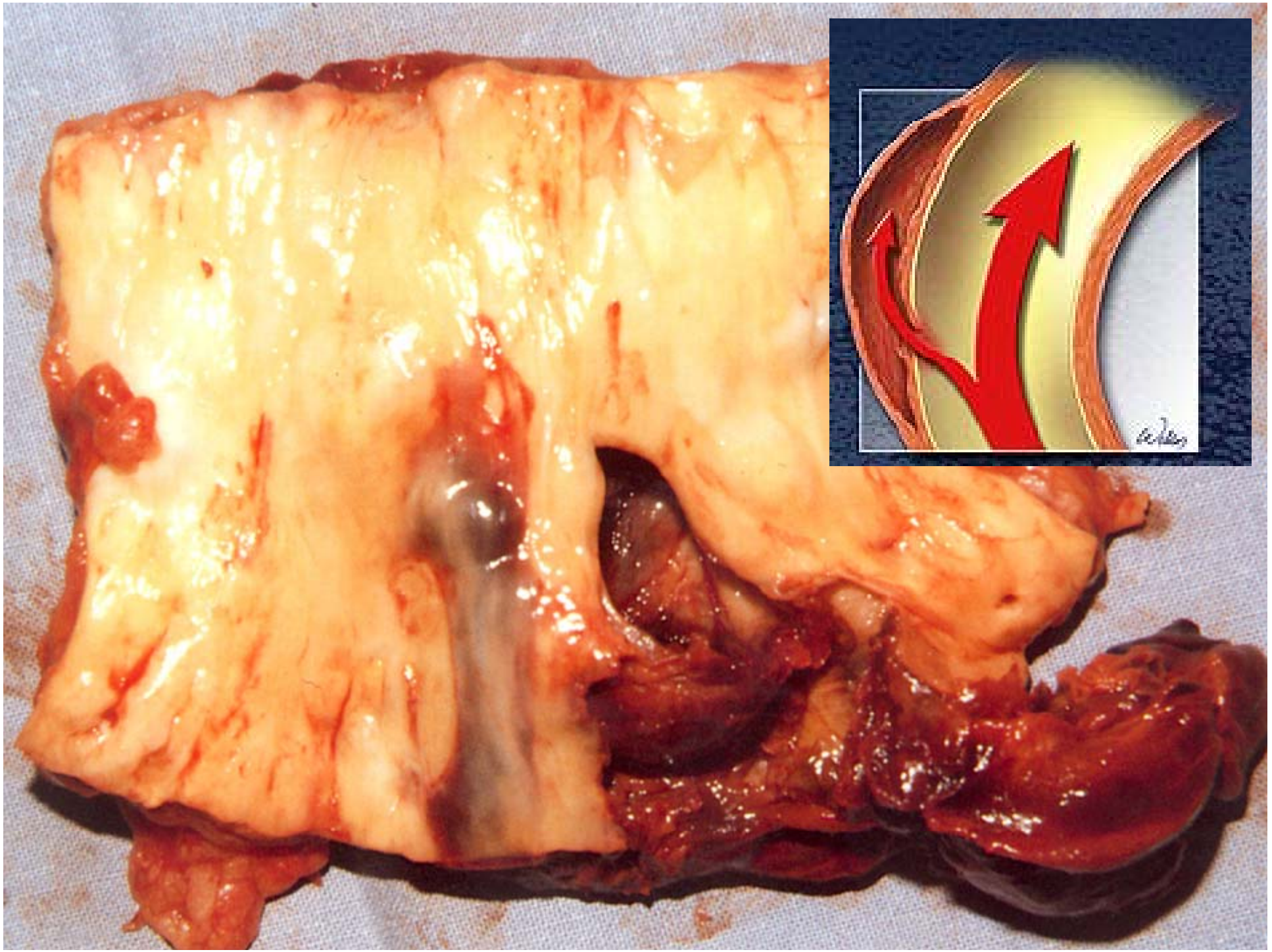
Seminars in Thoracic and Cardiovascular Surgery, Volume 27, Issue 2, 2015, 135–143

http://dx.doi.org/10.1053/j.semtcvs.2015.07.006

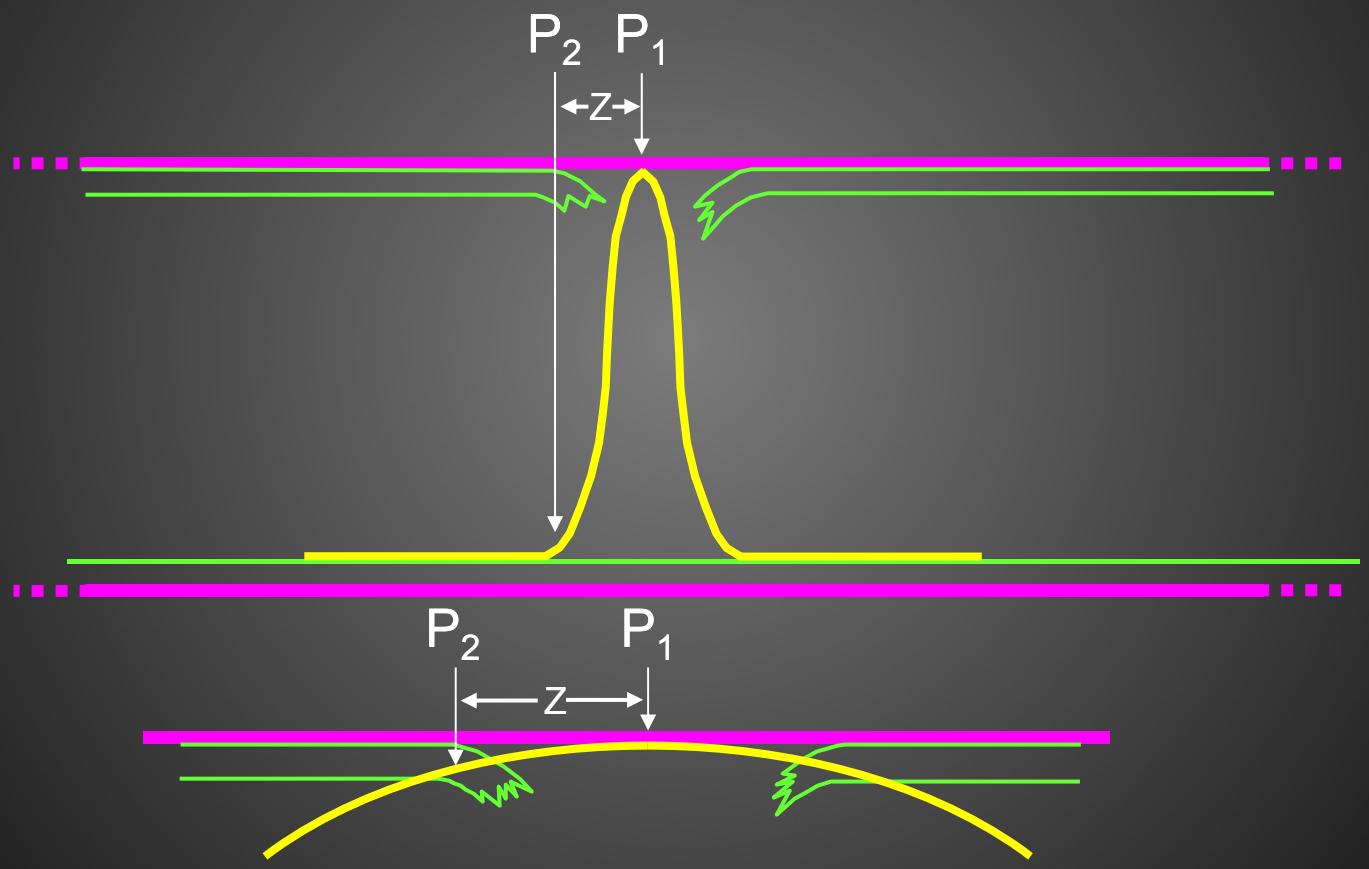
MEDIANECROSES AORTIQUES







LE MOTEUR DE LA DISSECTION
dP/dt



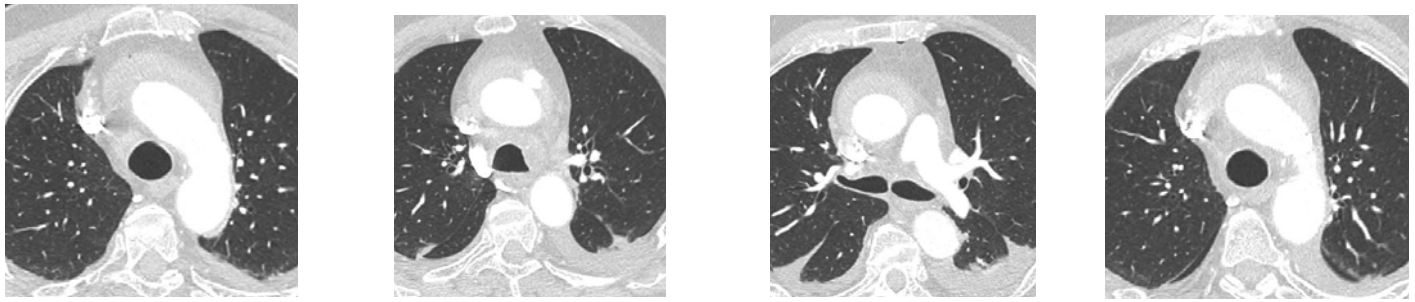
force de progression (ΔP) = P_1 - P_2

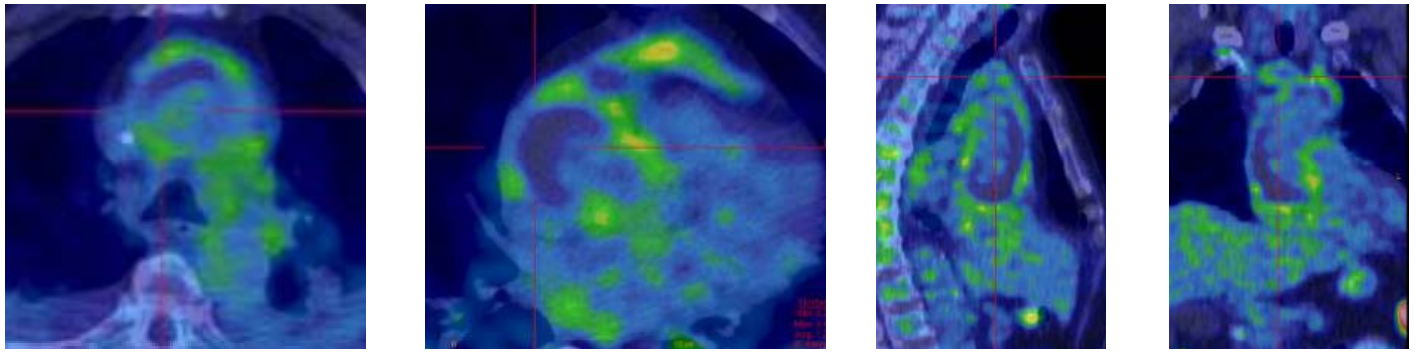
HEMATOME INTRAMURAL

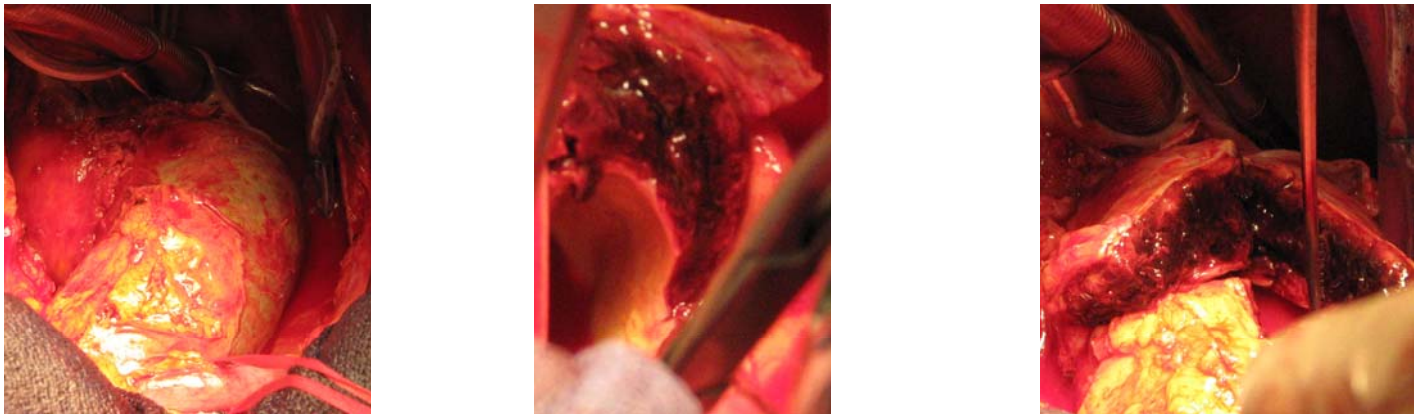
- entité identifiée récemment du fait de la généralisation des examens radiologiques
- précurseur d'une future dissection
- hématome né "in situ" (vasa vasorum)
  sans orifice d'entrée
- 1/3 des cas évolue vers la dissection aiguë

on les traite, donc, comme des dissections aiguës

From anatomical to functional imaging
Complicated Intramural hematoma of the aorta







Courtesy of Dr. Durieux R.

CLASSIFICATION

CLASSIFICATIONS TOPOGRAPHIQUES : ESSENTIELLES POUR LE CHOIX THERAPEUTIQUE

1966 De Bakey

Type I : aorte ascendante
→ aorte sous-rénale

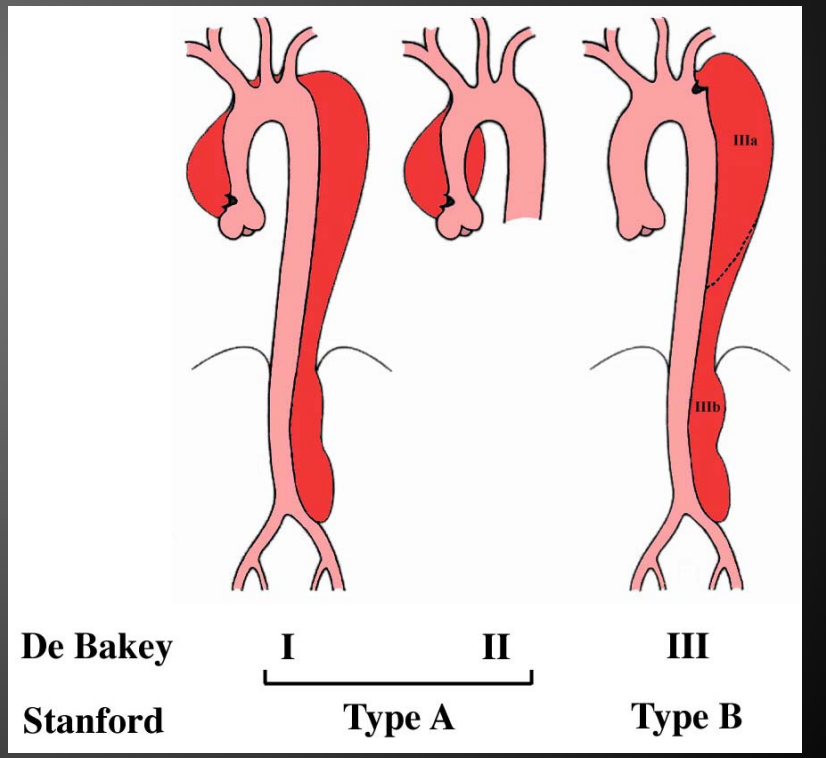
Type II : aorte ascendante

Type III : aorte descendante
IIIa > diaphragme
IIIb < diaphragme

1970 Standford

Type A : toute dissection intéressant l'aorte ascendante

Type B : toute dissection ne concernant pas l'aorte ascendante



EPIDEMIOLOGIE

Incidence : 3-6 / 100.000 / année

2000 cas /année aux USA
3000 cas /année en Europe
1-3% de toutes les autopsies (USA)

Sex ratio : 5 hommes - 1 femme

Hôpital Universitaire de Liège (1985-2005) : 439 cas (302 A - 137 B)

FACTEURS ETIOLOGIQUES DES DISSECTIONS AORTIQUES

Hypertension chronique

- tabagisme, dyslipidémie, drogues (cocaïne)

Maladie des tissus conjonctifs

- Marfan, Ehlers-Danlos,Turner,Loeys-Dietz

Anomalies cardio-vasculaires congénitales

- bicuspidie
- coarctation

Maladie vasculaire inflammatoire ou auto-immune

- "giant cell arteritis", Takayasu, Behcet

Trauma

- accident de roulage
- cathétérisme interventionnel
- chirurgie valvulaire/aortique

Enfin, la **grossesse** peut représenter une période dangereuse chez les personnes à risque.

Cocaine & Aortic Dissection

Cocaine induces apoptosis in primary cultured rat aortic vascular smooth muscle cells: possible relationship to aortic dissection, atherosclerosis, and hypertension

Cocaine serves as both a predisposing factor to aortic dissection due to its effect on aortic connective tissue and as a precipitating factor due to its propensity to produce abrupt and severe hypertension

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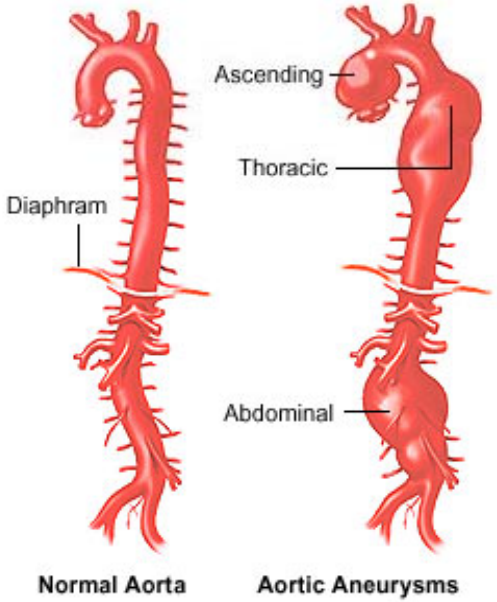


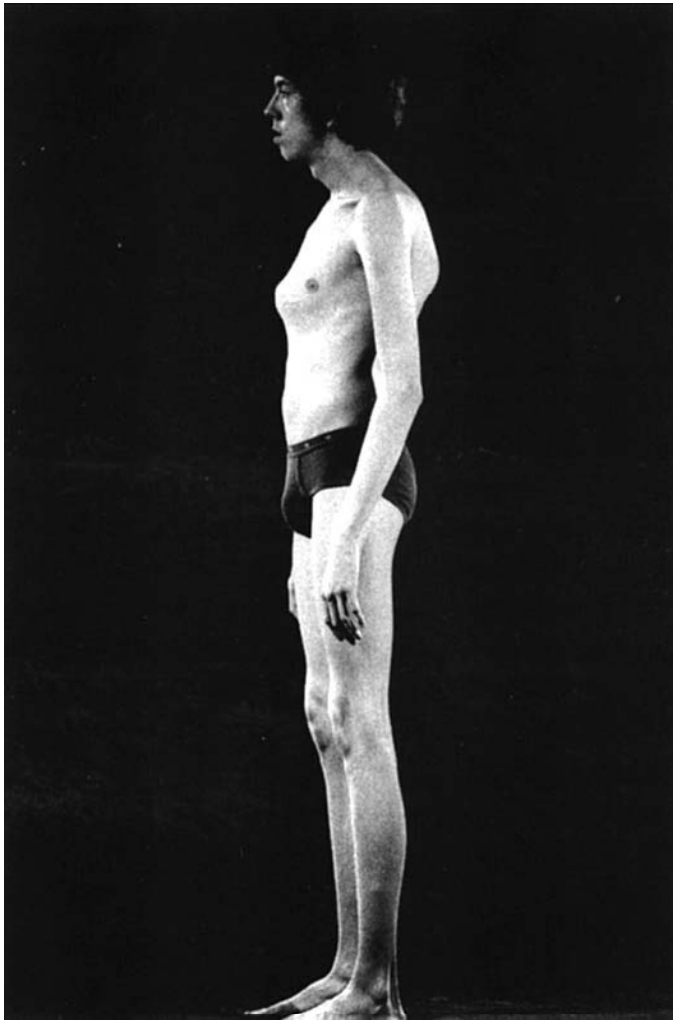
Genes Causative of Thoracic Aortic Disease:

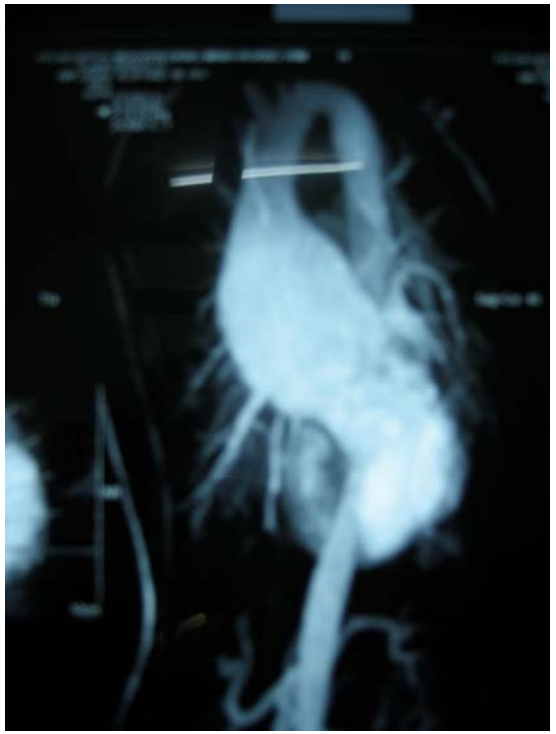
Syndromic Thoracic Aortic Aneurysm and Dissection:

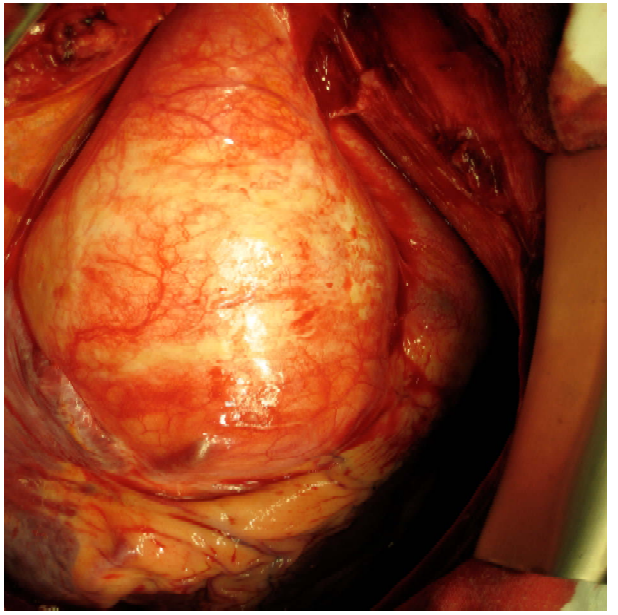
- 1. FBN1 → Marfan Syndrome
- 2. COL1A1
- 3. COL1A2
- 4. COL3A1 → Ehlers-Danlos Syndrome
- 5. COL5A1
- 6. COL5A2
- 7. TGFBR1
- 8. TGFBR2 → Loeys-Dietz Syndrome
- 9. TGFβ2 → TGFβ-related vasculopathy
- 10. SMAD3 → Aneurysm-Osteoarthritis Syndrome
- 11. FBLN4
- 12. ELN → Cutis Laxa Syndrome
- 13. SLC2A10 → Arterial Tortuosity Syndrome
- 14. FLNA → Periventricular Heterotopia Syndrome
- 15. ADAMTS10 → Weill-Marchesani Syndrome
- 16. FBN2 → Contractural arachnodactyly

Non-Syndromic Thoracic Aortic Aneurysm and Dissection:

- 1. TGFBR1
- 2. TGFBR2
- 3. ACTA2
- 4. MYLK → Familial Thoracic Aortic Aneurysm
- 5. SMAD3
- 6. TGFβ2
- 7. PRKG1
- 8. MYH11 → Familial Thoracic Aortic Aneurysm with Patent Ductus Arteriosus
- 9. NOTCH1 → Familial Thoracic Aortic Aneurysm with Bicuspid Aortic Valve







MARFAN

autosomale dominante

mutation du gène de fibrilline

HUMAN MUTATION 28(4), 387–395, 2007

RESEARCH ARTICLE

Three Arginine to Cysteine Substitutions in the Pro-Alpha (I)-Collagen Chain Cause Ehlers-Danlos Syndrome With a Propensity to Arterial Rupture in Early Adulthood

Fransiska Malfait,[1†] Sofie Symoens,[1†] Julie De Backer,[1] Trinh Hermanns-Lê,[2] Natzi Sakalihasan,[3] Charles M. Lapière,[4] Paul Coucke,[1] and Anne De Paepe[1*]

[1]Center for Medical Genetics, Ghent University Hospital, Ghent, Belgium; [2]Department of Dermatopathology, University Hospital of Sart Tilman, Liège, Belgium; [3]Department of Cardiovascular Surgery, University Hospital of Liège, Liège, Belgium; [4]Laboratory of Connective Tissues Biology, University of Liège, Liège, Belgium

EHLERS-DANLOS
autosomale dominante
mutation du gène du
procollagène de type III

Contents lists available at ScienceDirect

European Journal of Medical Genetics

journal homepage: http://www.elsevier.com/locate/ejmg

Cervical artery dissections and type A aortic dissection in a family with a novel missense COL3A1 mutation of vascular type Ehlers–Danlos syndrome

Georgios Makrygiannis [a, b, *], Bart Loeys [c], Jean-Olivier Defraigne [a, b], Natzi Sakalihasan [a, b]

[a] Department of Cardiovascular Surgery, University Hospital of Liège, Liège, Belgium
[b] Department of Surgery, Surgical Research Center (CREDEC), GIGA Cardiovascular Sciences, University of Liège, Liège, Belgium
[c] Center for Medical Genetics, Faculty of Medicine and Health Sciences, University of Antwerp and Antwerp University Hospital, Antwerp, Belgium

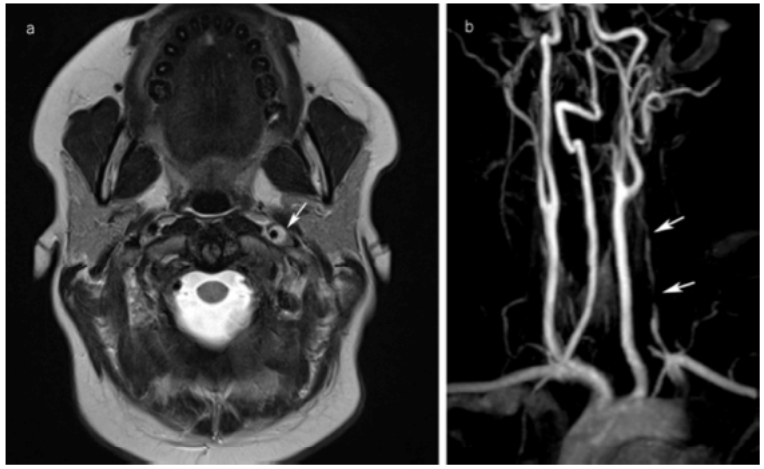


Fig. 1. a; MRI of the head, axial T2-weighted Turbo Spin Echo sequence shows the presence of the dissection of the left internal carotid artery (arrows), b; MRA of the neck, coronal view, shows the dissection of the vertical part of the left vertebral artery (arrows).

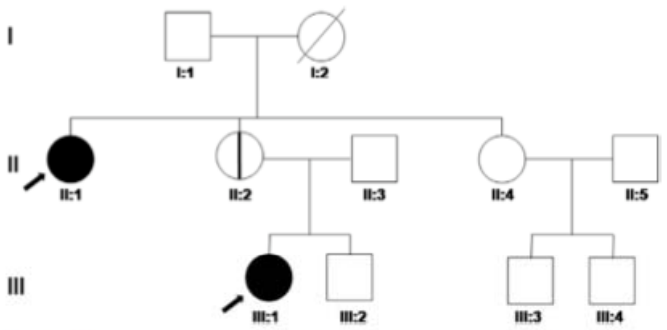
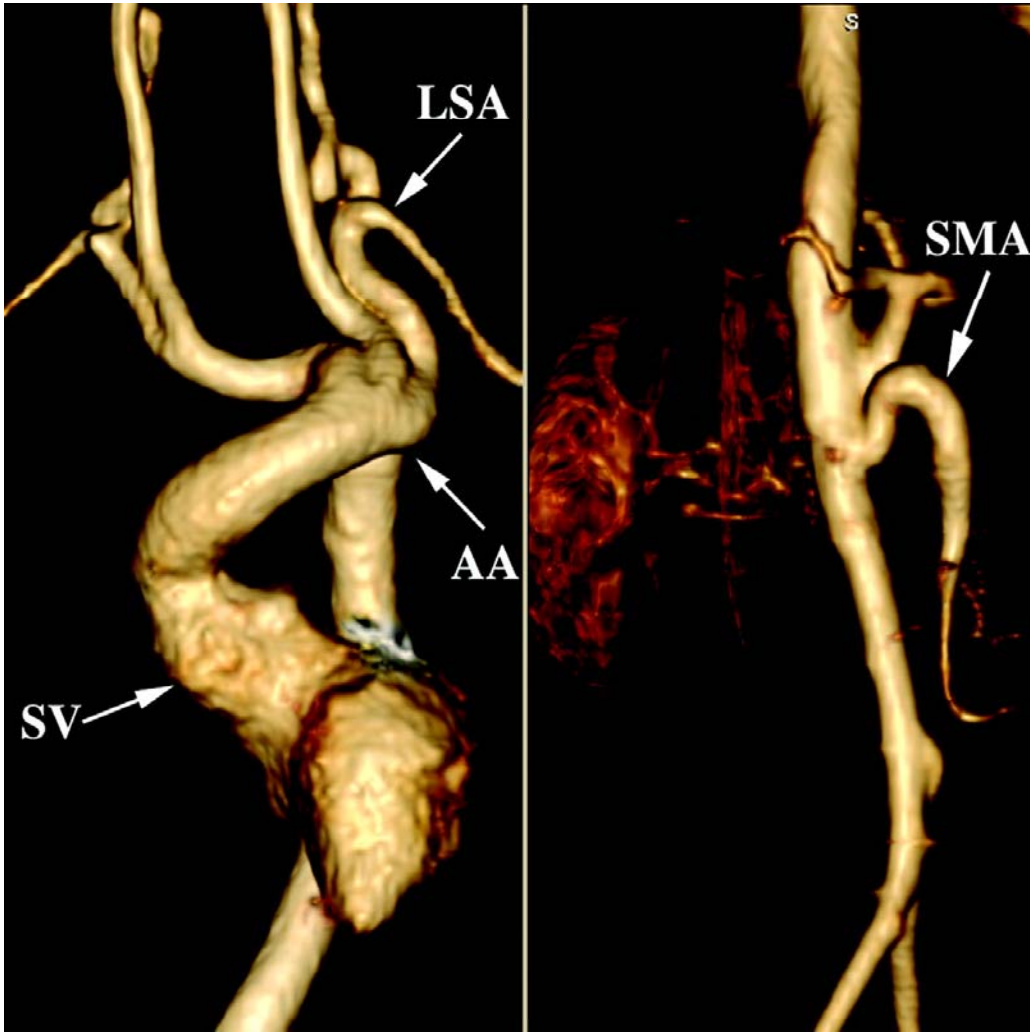


Fig. 2. Pedigree; In grey member of the family heterozygous of the c.953G > A (p.Gly318Asp) mutation in the exon 14 of *COL3A1* gene, II:1; Type A aortic dissection, III:1; Cervical artery dissections, II:2; heterozygous asymptomatic mutation carrier, I:1; very old person to whom we did not perform genetic analysis.

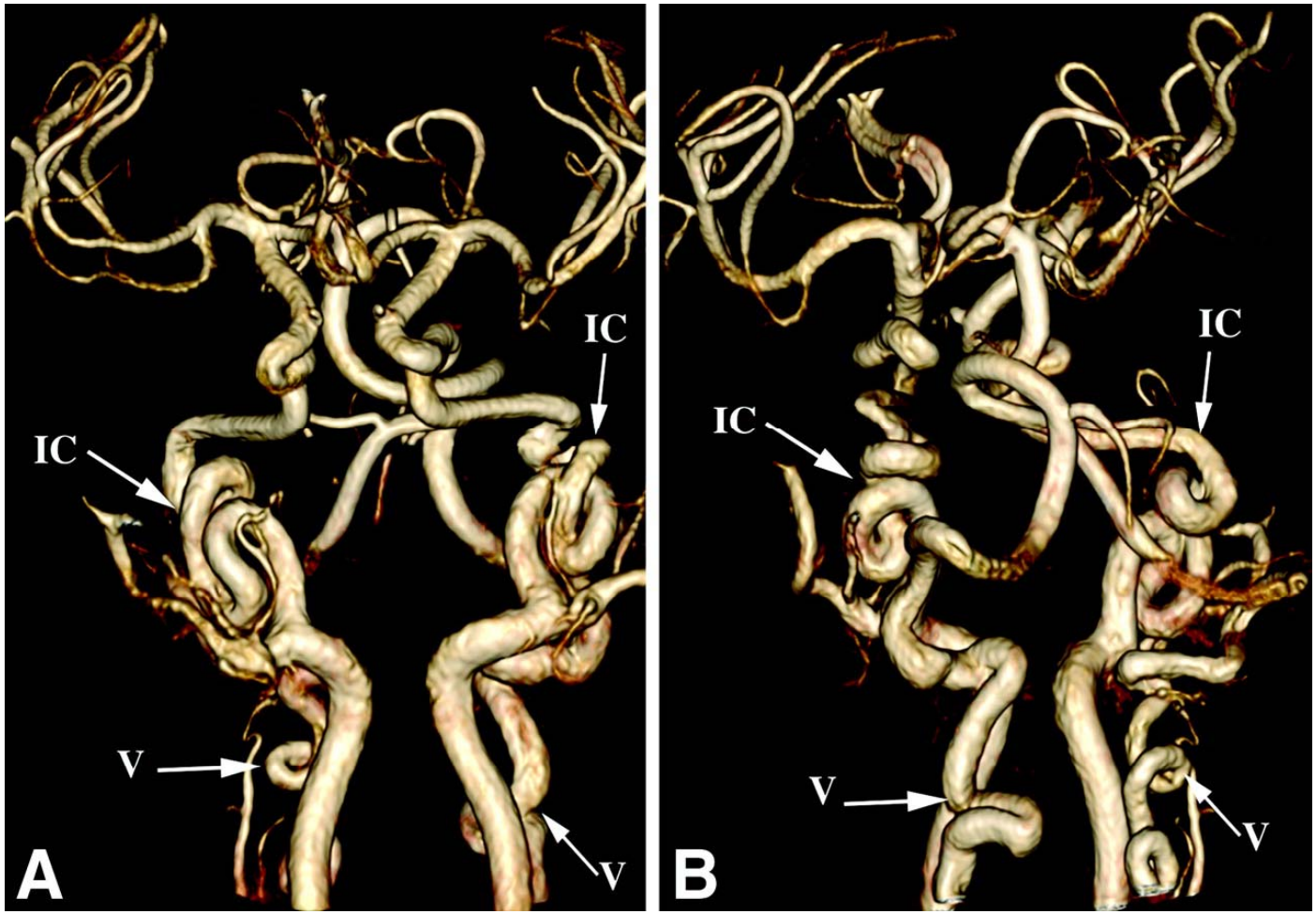
Imaging findings in a child with Loeys-Dietz syndrome.



Amira Dhouib et al. Circulation. 2012;126:507-508

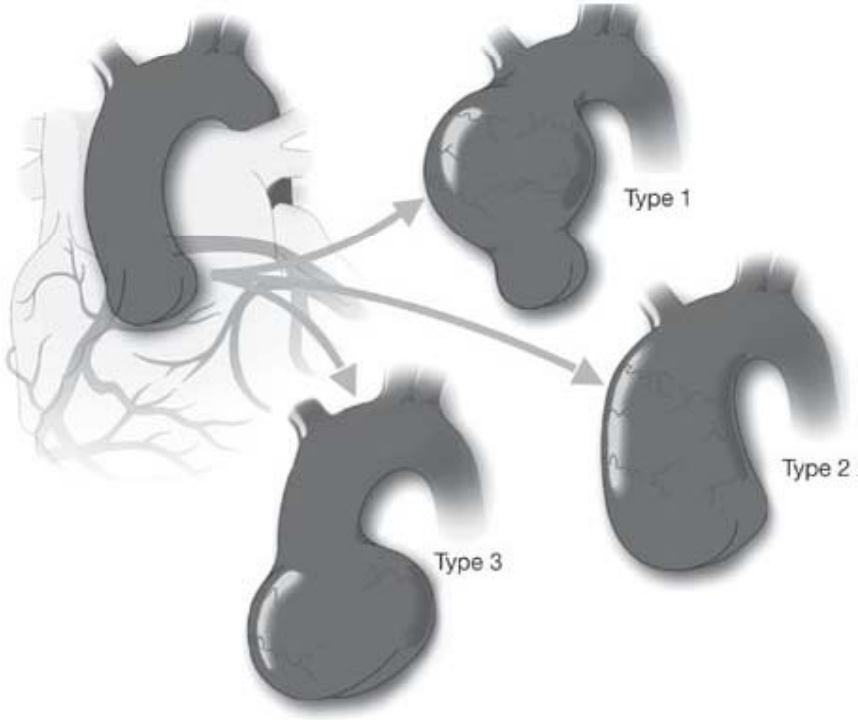
American Heart Association

Time-of-flight sequence at the level of neck vessels and the brain, anteroposterior projection (A) and left oblique posterior view (B), shows major tortuosities of the internal carotid arteries (IC) and vertebral arteries (V).



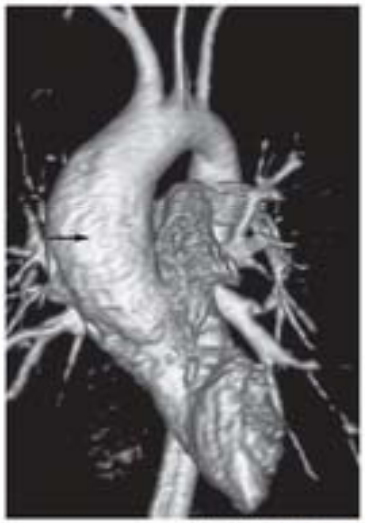
Amira Dhouib et al. Circulation. 2012;126:507-508

Figure 4 Aneurysms associated with bicuspid aortic valve can be of three general phenotypes



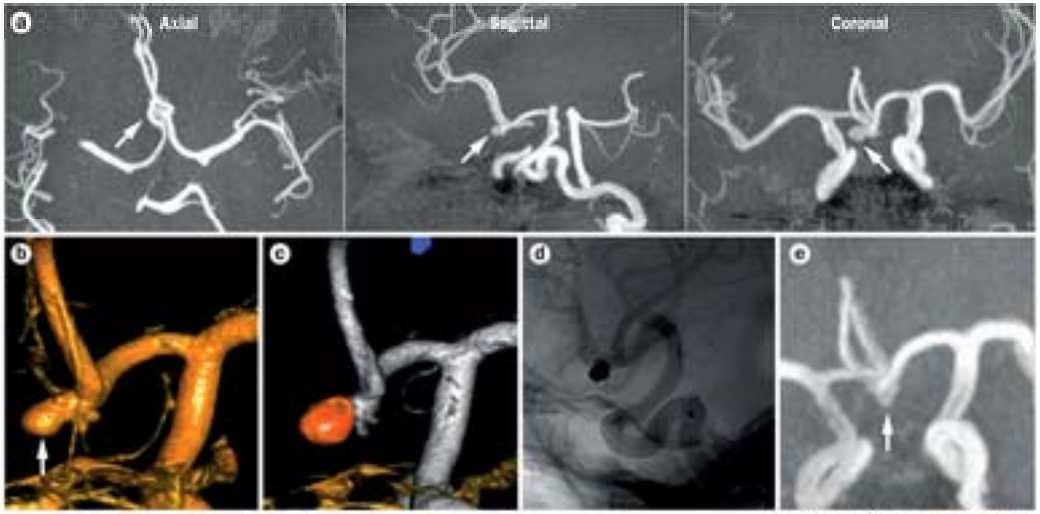
Davies JE and Sundt TM (2007) Surgery Insight: the dilated ascending aorta—indications for surgical intervention *Nat Clin Pract Cardiovasc Med* **4:** 330–339 doi:10.1038/ncpcardio0885

nature CLINICAL PRACTICE
CARDIOVASCULAR
MEDICINE



Nature Reviews | Nephrology

3D magnetic resonance angiogram showir the ascending aortic aneurysm (arrow). Abbreviation: ADPKD, autosomal dominar polycystic kidney disease. Permission obtained from the American Society of Nephrology © Liu, D. *et al. J. Am. Soc.*...



Nature Reviews | Nephrology

Case Report

AORTA

Aorta, July 2013, Volume 1, Issue 2: 123–125
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Dissection of Iliac Artery in a Patient With Autosomal Dominant Polycystic Kidney Disease

A Case Report

Audrey Courtois, PhD[1]*, Betty V. Nusgens, PhD[1], Philippe Delvenne, MD, PhD[2], Michel Meurisse, MD, PhD[3], Jean-Olivier Defraigne, MD, PhD[4], Alain C. Colige, PhD[1], Natzi Sakalihasan, MD, PhD[4]

[1]Laboratory of Connective Tissues Biology, GIGA, University of Liège, Sart-Tilman, Belgium; [2]Department of Anatomopathology, CHU Liège, University of Liège, Liège, Belgium; [3]Department of Abdominal, Endocrine and Transplantation Surgery, CHU Liège, University of Liège, Liège, Belgium; [4]Department of Cardiovascular and Thoracic Surgery, CHU Liège, University of Liège, Liège, Belgium

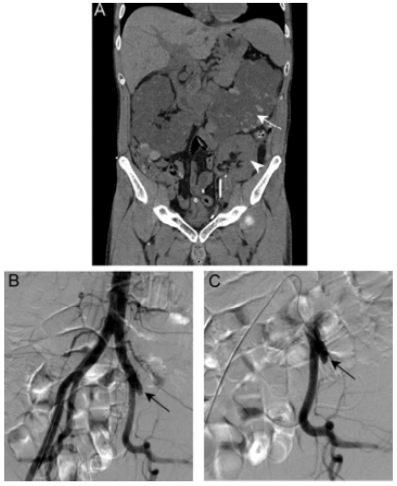


Figure 1. Imaging of the ADPKD patient. (A) CT-scan showing the polycystic kidney (**arrow**) and the transplanted kidney (arrowhead). (B) and (C) Aorto-femorography showing the occlusion of the left external iliac artery (**arrow**).

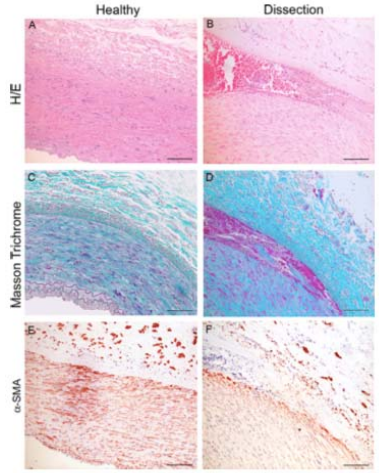


Figure 2. General organization of the wall of the dissected iliac artery. (A) and (B): hematoxylin/eosin staining. (C) and (D): Masson trichrome staining. (E) and (F): α-smooth muscle actin immunohistochemistry performed on a healthy iliac artery (A, C, E) and the dissected segment of the iliac artery of the ADPKD patient (B, D, F). Bar = 100 μm.

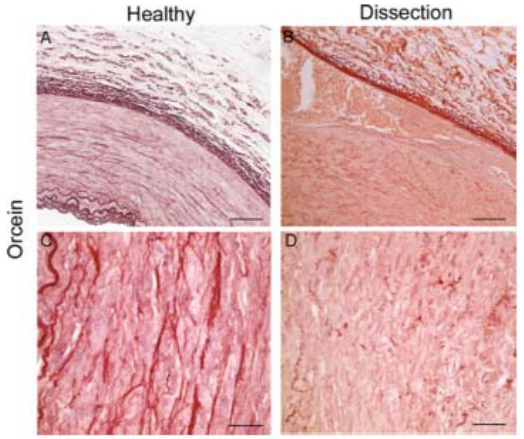
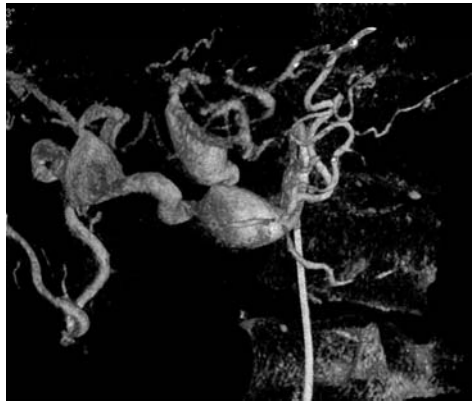
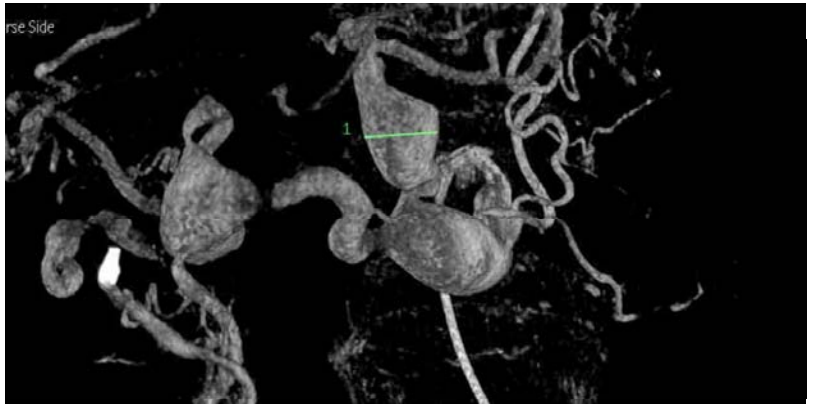


Figure 3. Visualization of elastic fibers by orcein staining of a healthy iliac artery (A) and (C) and the dissected segment of the iliac artery of the ADPKD patient (B) and (D). Bar = 100 μm in (A) and (B) and 25 μm in (C) and (D).

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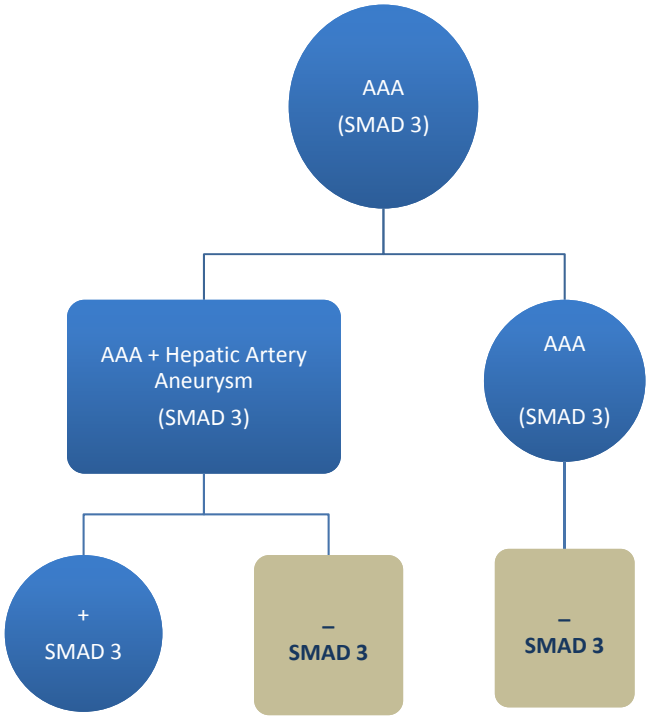
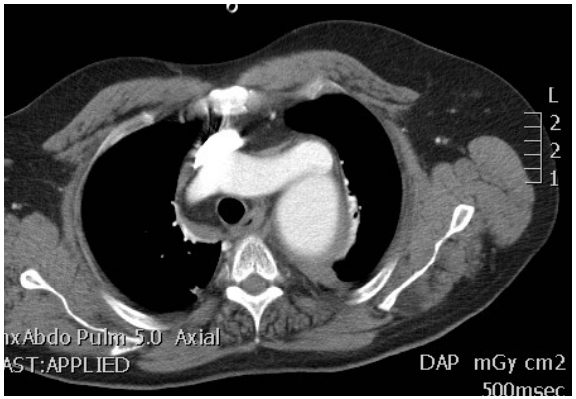
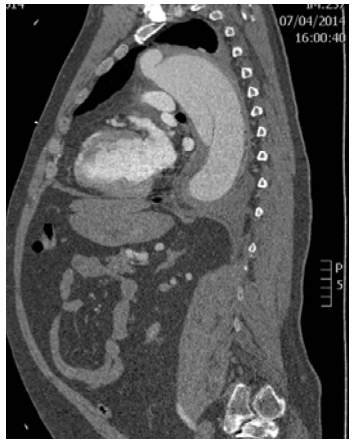
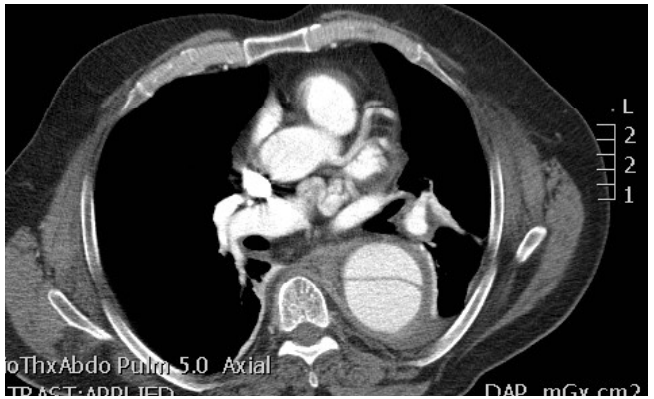
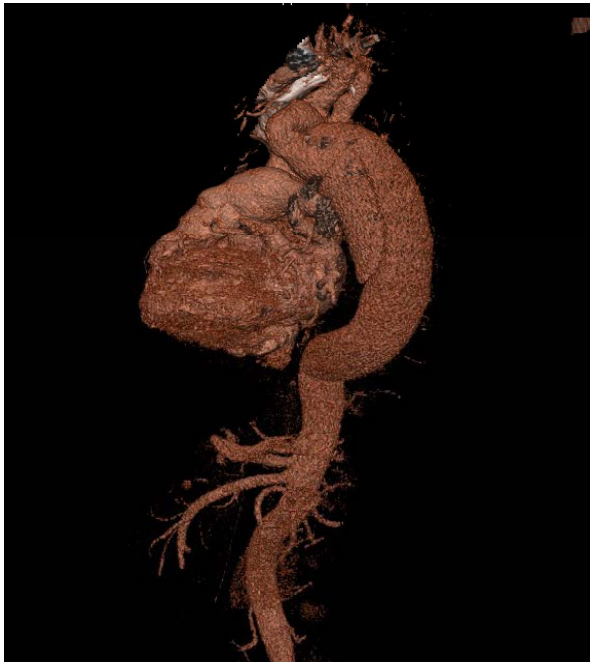

AAA
(SMAD 3)
AAA + Hepatic Artery Aneurysm
(SMAD 3)
AAA
(SMAD 3)
+
SMAD 3
–
SMAD 3
–
SMAD 3

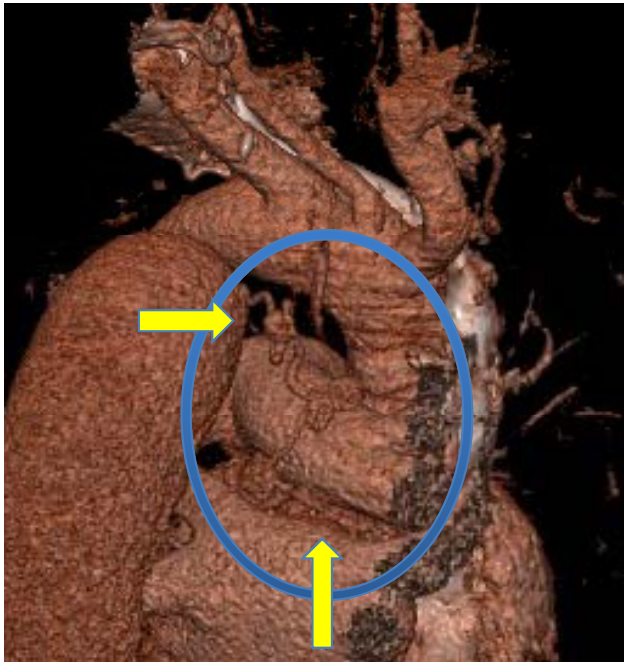
Outcome of the type B dissection
(with increased multifocal 18F-FDG uptake)



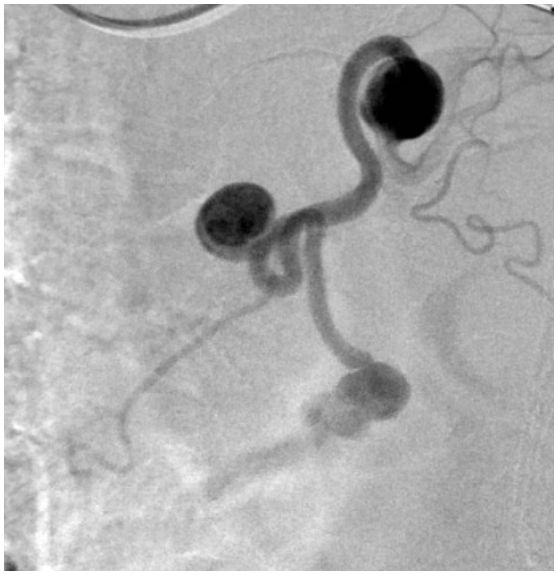


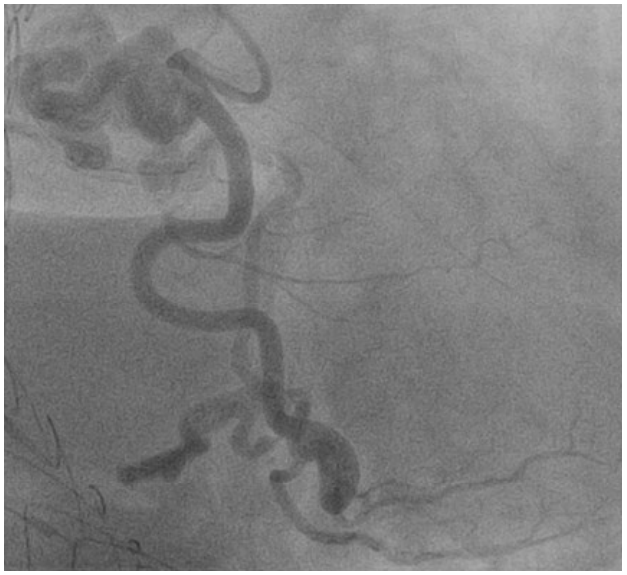


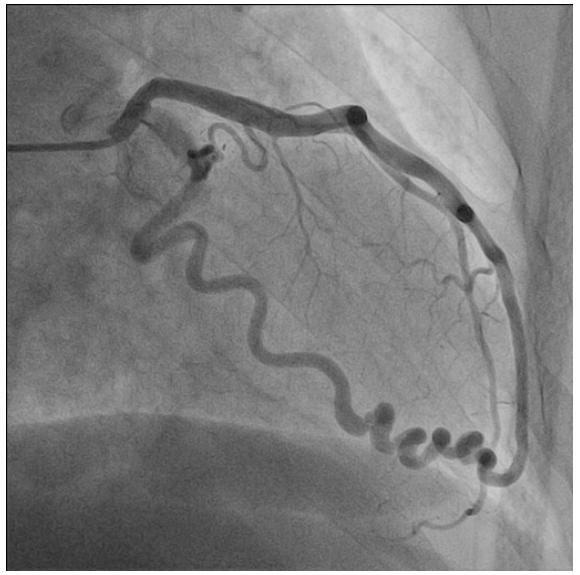


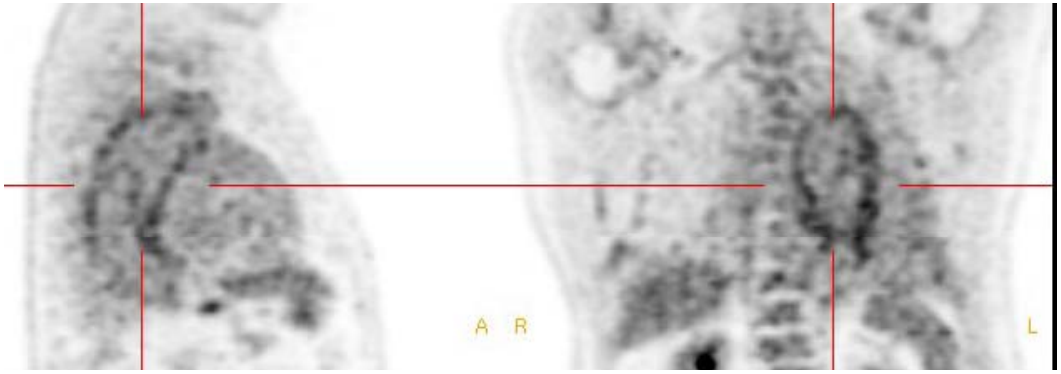


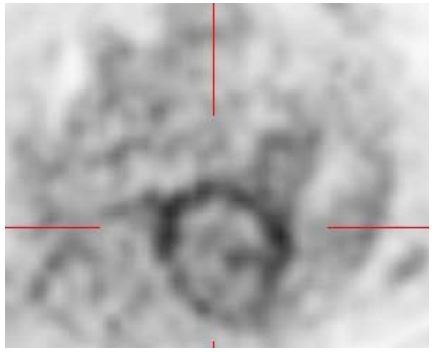
63 years, Male patient,with known coarctation.

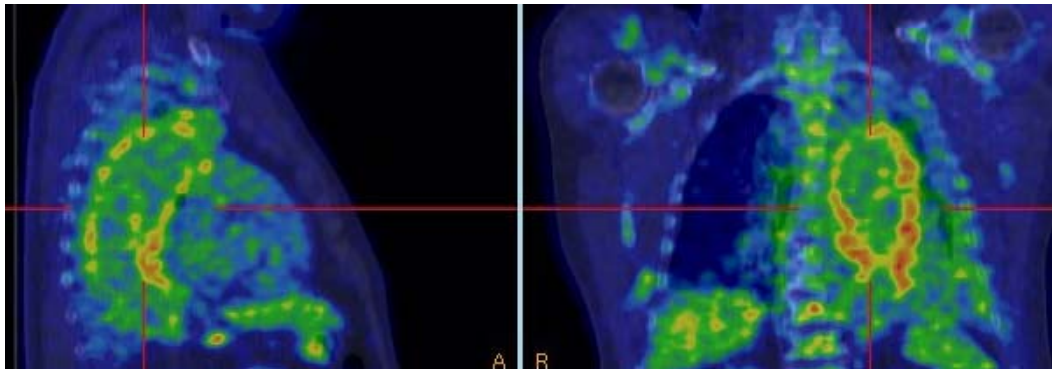


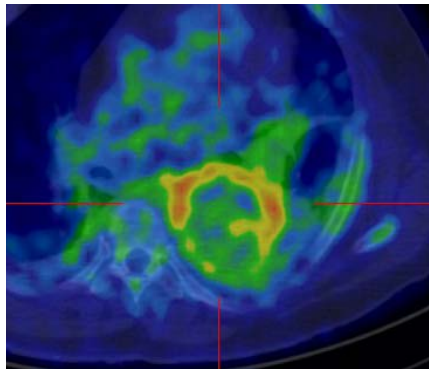




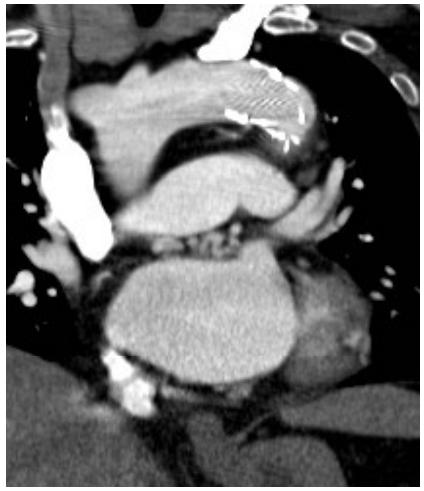
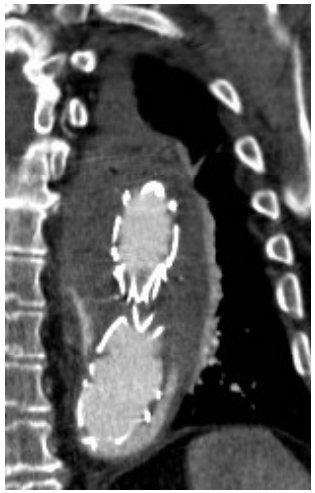
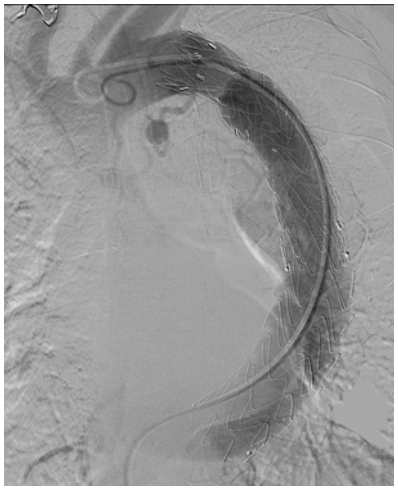
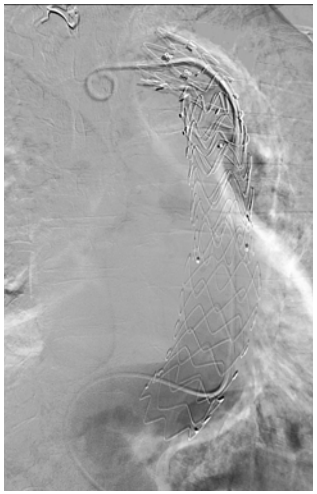
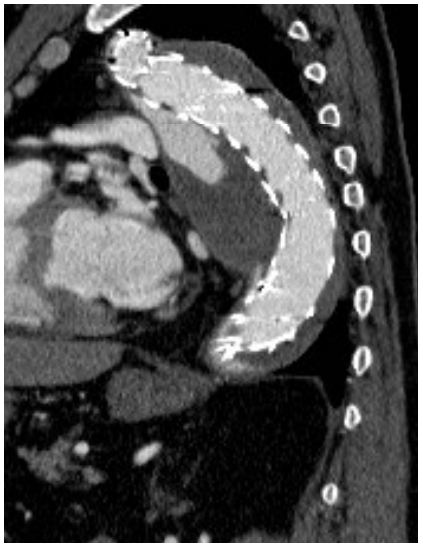
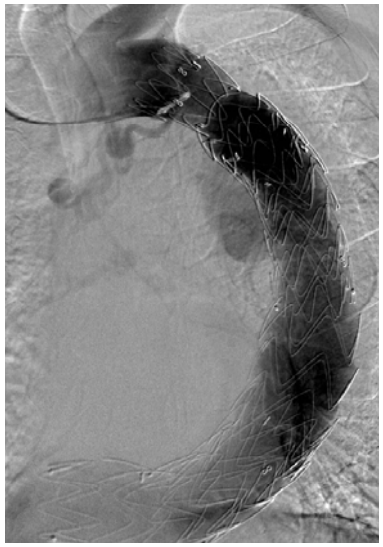
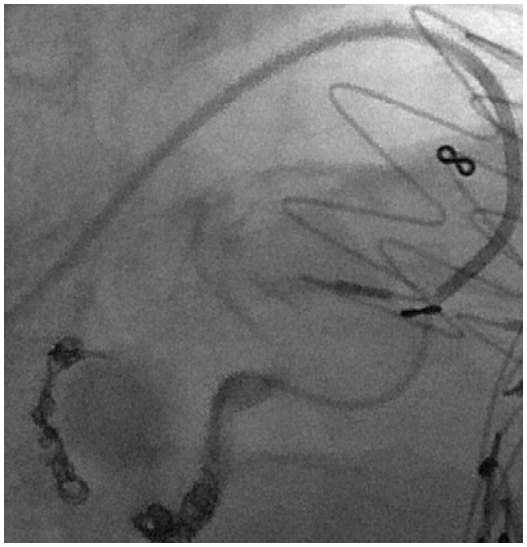
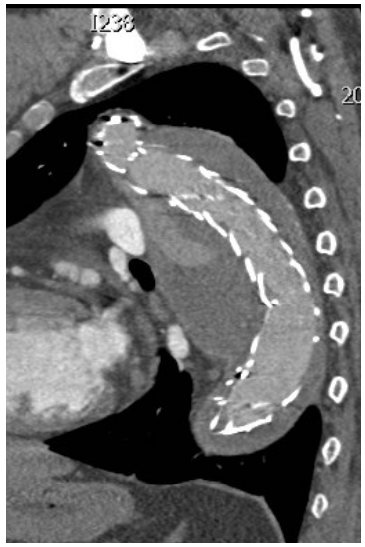

A R L




A B

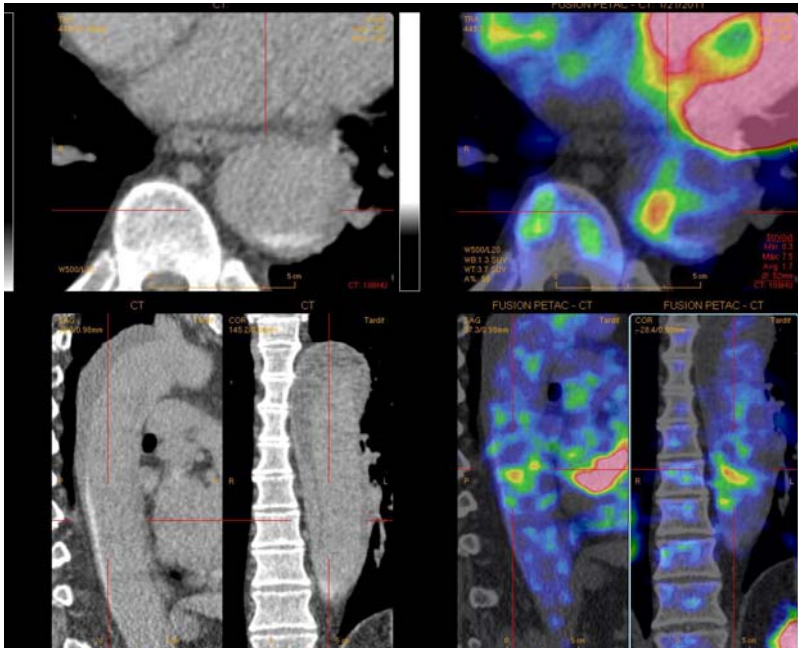


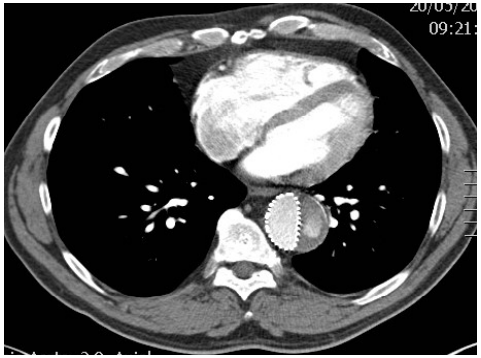
Outcome of the type B dissection
(with increased multifocal 18F-FDG uptake)

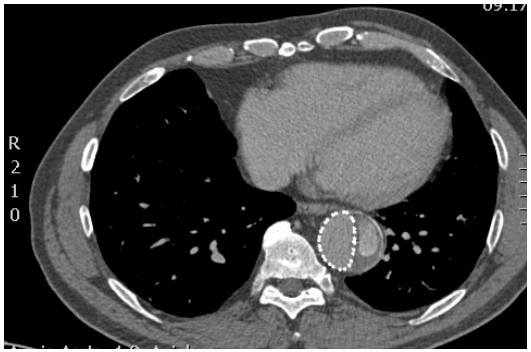









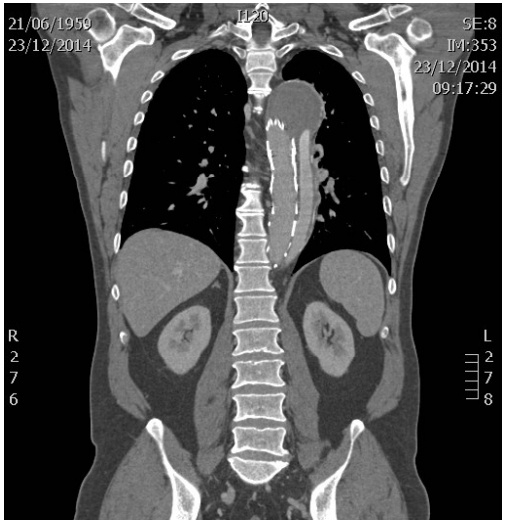
Familial Genome sequencies (MYLK) are in progress

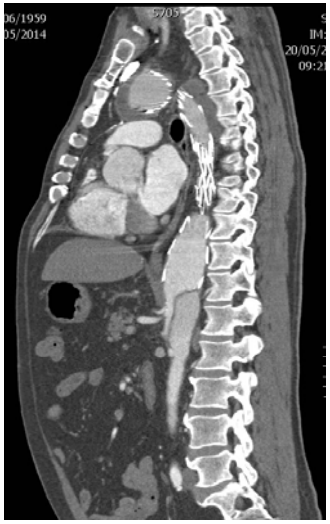
Outcome of the type A dissection with increased monofocal 18F-FDG uptake (Ration , SUV max/SUV Liver > 1)

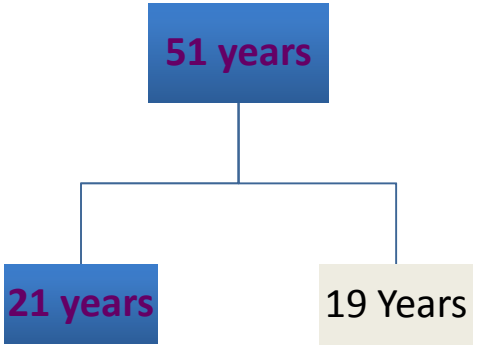












51 years male patient, following after Type A dissection.
TEVAR performed for Increasing diameter of aneurysm (64mm). (*ACTA* 2 mutation)

FACTEURS ETIOLOGIQUES DES DISSECTIONS AORTIQUES

Hypertension chronique

- tabagisme, dyslipidémie, drogues (cocaïne)

Maladie des tissus conjonctifs

- Marfan, Ehlers-Danlos,Turner,Loeys-Dietz

Anomalies cardio-vasculaires congénitales

- bicuspidie
- coarctation

Maladie vasculaire inflammatoire ou auto-immune

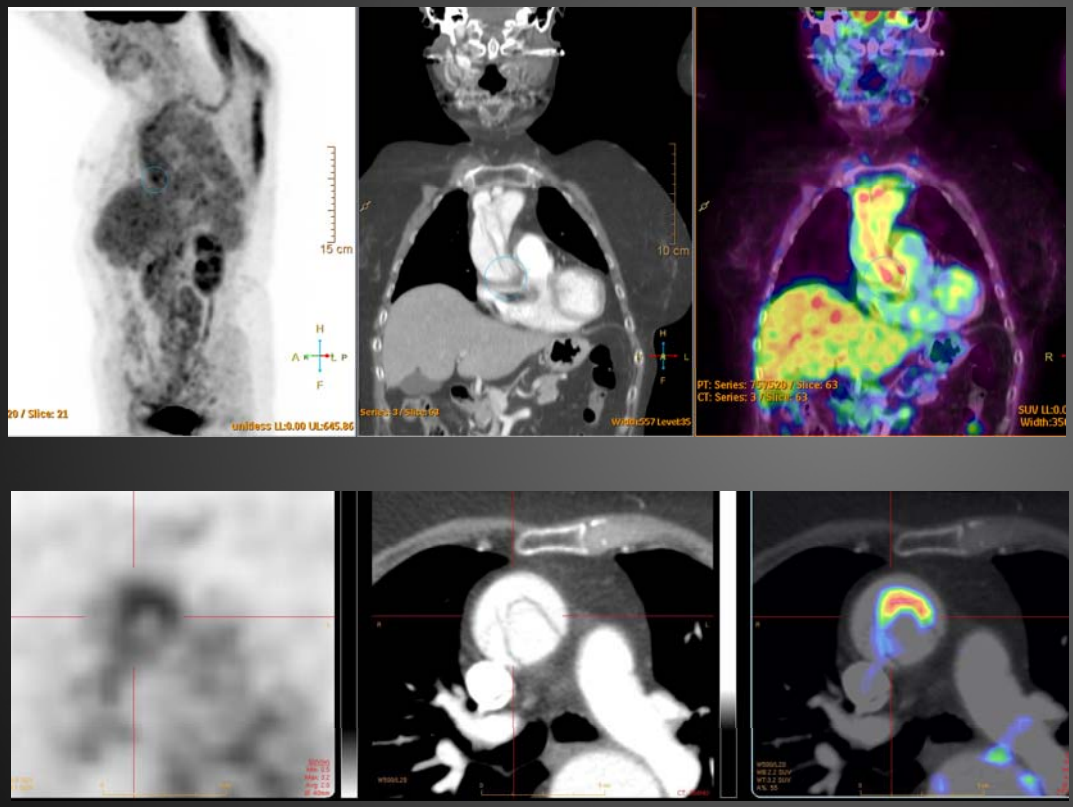
- "giant cell arteritis", Takayasu, Behcet

Trauma

- accident de roulage
- cathétérisme interventionnel
- chirurgie valvulaire/aortique

Enfin, la **grossesse** peut représenter une période dangereuse chez les personnes à risque.

Increased metabolic activity highlighted by PET-CT in the wall of the dissected ascending aorta in a patient with Horton disease



Bruls S,…Sakalihasan N. CIRCCVIM. 2013

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Acute type A aortic dissection and pregnancy: a population-based study

In an average female population of 34,1381 women in the age range of 15-45 years were followed up between 1994 and 2004 (total of 3755.195 person-years of observation).

: 15 patients with acute aortic dissection were identified, and an overall incidence of 0.4 case per 100 000 person-years was estimated

Thalmann M et al; Eur J Cardiothorac Surg. 2011 Jun;39(6):e159-63. Epub 2011 Feb 26

Acute type A aortic dissection and pregnancy: a population-based study

Table 1. Demographics, symptoms and underlying pathology of female patients under 45 years of age, suffering from acute aortic dissection. Assessment was performed via patient's, relative's interview and distinct medical chart review, as appropriate. Arterial hypertension was assumed in patients with documented history of hypertension or that with chronic intake of antihypertensive drugs. SCD: sudden cardiac death.

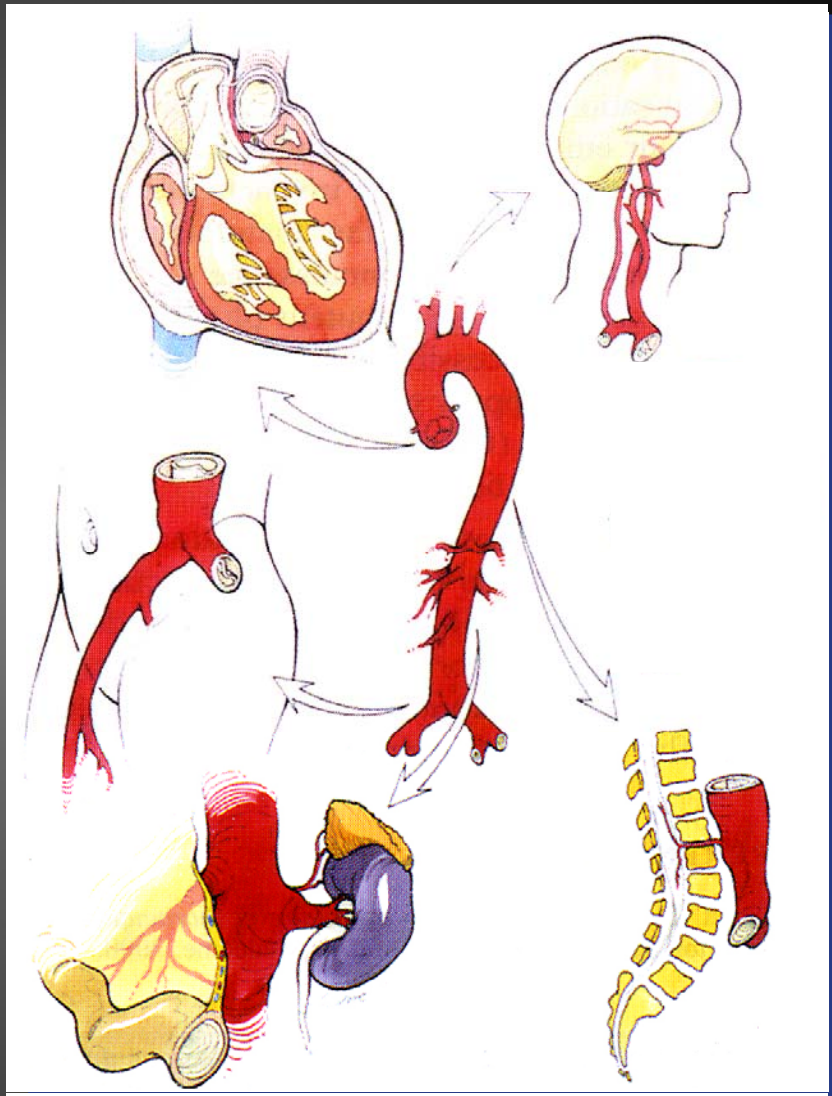
Patient (nr.)	Age (a)	Initial symptoms			SCD	Risk factors and aortic pathology					Preknown	Pregnancy (w)
		Chest pain	Syncope	Dyspnea		Hypertension	Marfan	Aortic coarctation	Cystic medial necrosis	Bicuspid aortic valve		
1	36	+	+	–	+	–	–	–	+	–	–	–
2	45	+	–	–	+	+	–	–	+	–	–	–
3	29	–	–	+	–	+	+	–	–	–	+	–
4	37	+	–	+	–	+	+	–	–	–	–	–
5	45	–	–	+	+	+	–	–	+	–	–	–
6	40	+	–	–	–	+	–	–	+	–	–	–
7	39	–	+	–	+	+	–	–	+	–	–	–
8	42	+	–	–	–	+	–	–	+	–	–	–
9	41	+	+	–	+	+	+	–	–	–	+	–
10	32	+	+	–	–	+	+	–	–	–	–	36
11	38	+	–	–	+	+	–	+	–	–	+	–
12	42	+	–	–	+	+	–	+	–	–	+	–
13	34	–	+	+	–	+	–	–	–	+	–	32
14	37	+	+	–	+	+	–	–	+	–	–	–
15	45	+	–	–	–	–	–	–	+	–	+	–
Cumulative count (%)		11 (73%)	6 (40%)	3 (20%)	8 (53%	14 (93%)	4 (27%)	2 (13%)	8 (53%)	1 (7%)	5 (33%)	2 (13%)

Thalmann M et al; Eur J Cardiothorac Surg. 2011 Jun;39(6):e159-63. Epub 2011 Feb 26

PRESENTATION CLINIQUE

LA DISSECTION AIGUË DE L'AORTE : LA GRANDE SIMULATRICE

R. LIMET



DOULEUR DANS LA DISSECTION AORTIQUE

- localisation thoracique antérieure ou interscapulaire
- "déchirante" ; "crucifiante"
- sévérité maximale d'emblée (≠ infarctus myocarde)
- migrante

manifestations vaso-vagales associées (sudation, vomissements, lipothymies)

SIGNES

1) anisosphygmie nouvelle (< 20%)
2) souffle diastolique aortique (40 - 50%, type A)
3) pouls paradoxal (épanchement péricardique)
4) choc cardiogénique (tamponnade, insuffisance aortique majeure, atteinte coronaire par compression ou dissection)
5) choc hypovolémique (rupture aortique extra-péricardique)
6) souffles abdominaux
7) disparition d'un ou des pouls fémoraux
8) hémiplégie, hémiparésie, paraplégie

sur les examens de routine :

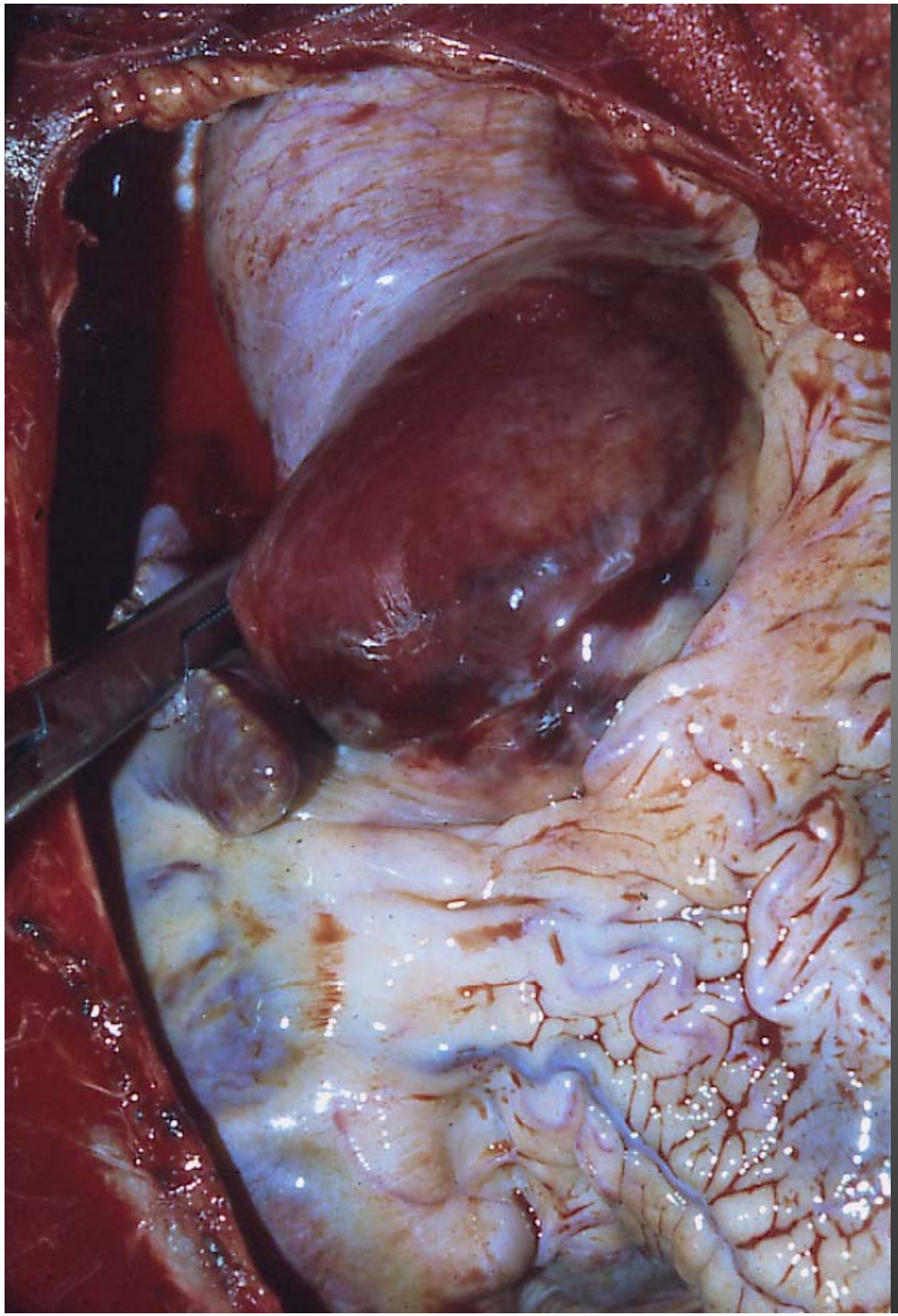
- ECG : normal, HVG ou ischémique
- Rx thorax : élargissement médiastinal, épanchement pleural

DIAGNOSTIC DIFFERENTIEL

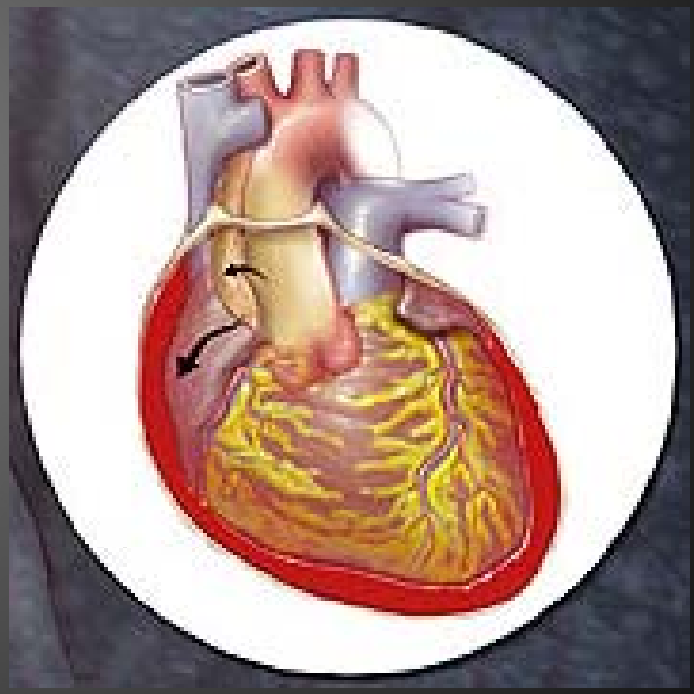
30% des anévrysmes disséquants ont reçu
un diagnostic erroné au début
→ **conséquences désastreuses**

IL FAUT ENVISAGER LA POSSIBILITE
D'UNE DISSECTION AORTIQUE EN CAS DE :

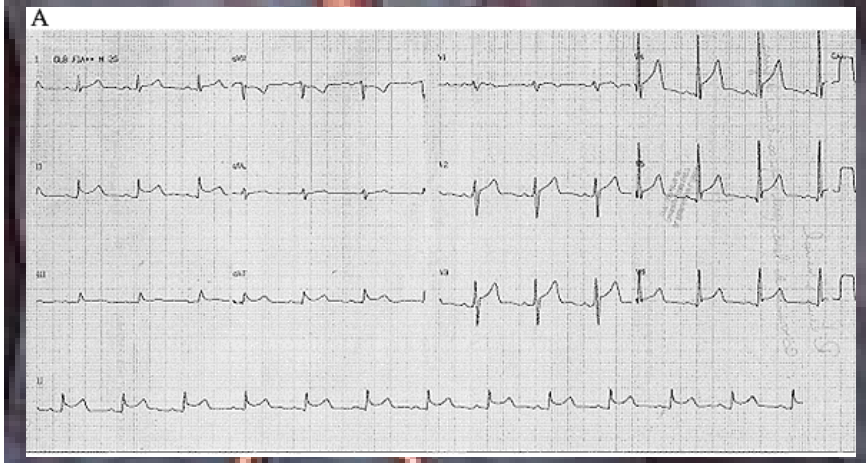
- syncope inexpliquée
- douleur thoracique
- douleur lombaire
- douleur abdominale
- AVC
- insuffisance cardiaque aiguë
- syndrome de malperfusion (mésentérique, rénal)
- paraplégie

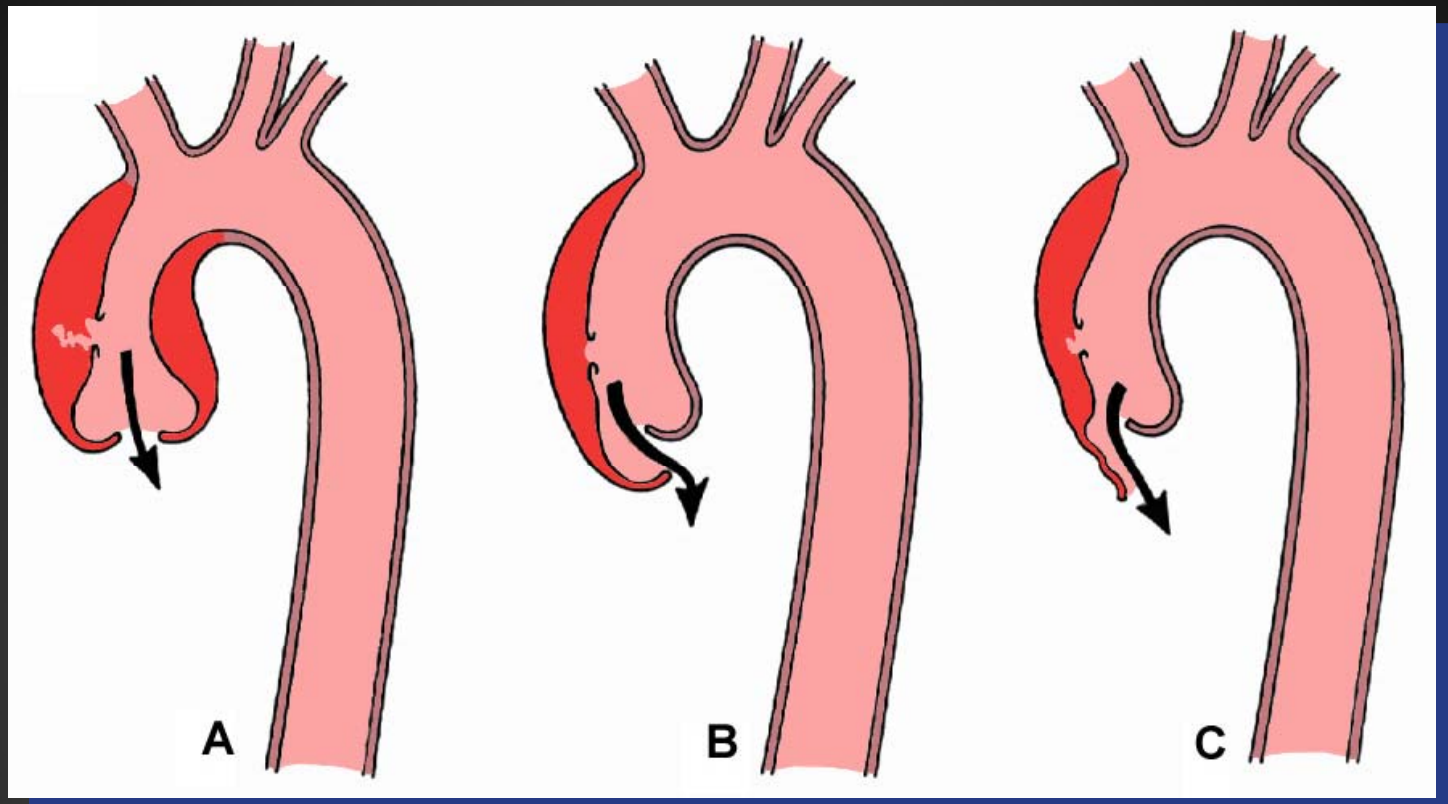


TAMPONNADE



ISCHEMIE MYOCARDIQUE





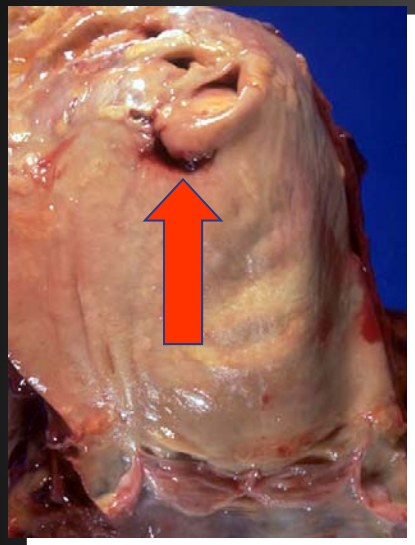
MECANISMES DE PRODUCTION DE L'INSUFFISANCE AORTIQUE

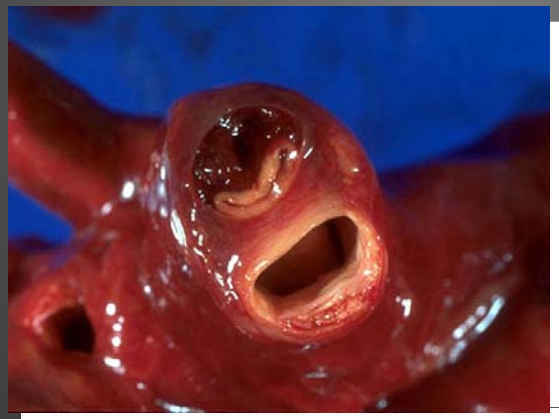
A) élargissement de la racine aortique, écarte les valves aortiques : insuffisance centrale

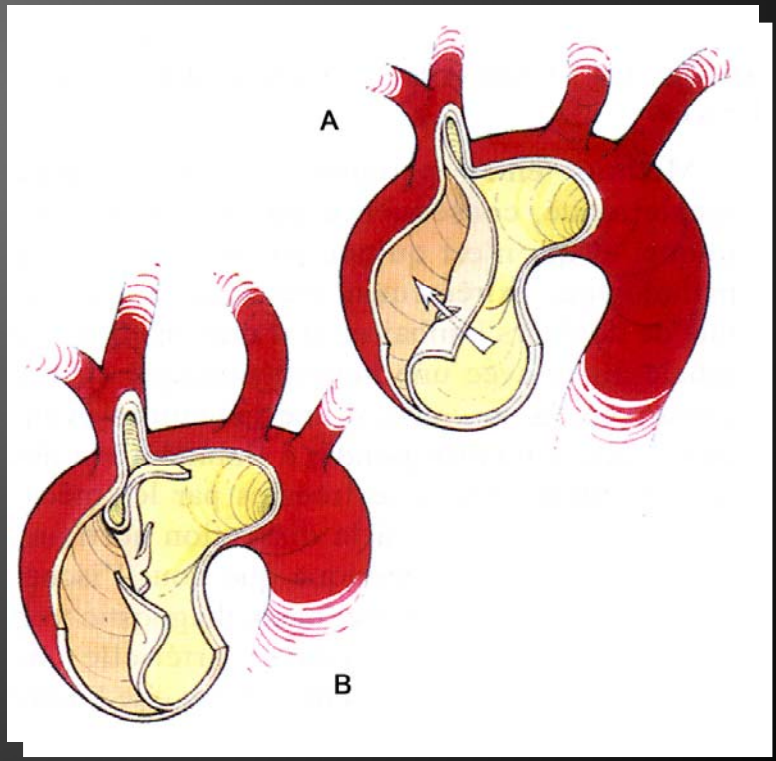
B) élargissement asymétrique de la racine, entraîne une valve au-dessous du plan de fermeture valvulaire

C) rupture mécanique de la base d'une valve provoquant son prolapse intraventriculaire

AVC PAR COMPRESSION DE LA CAROTIDE INTERNE DROITE







PARAPLEGIE

CAUSES ANATOMIQUES

vascularisation médullaire segmentaire

- collatéralité variable
- situation variable de l'artère d'Adamkievicz (D8-L3)

CAUSES PATHOLOGIQUES

- thrombi, dissection intimale

FACTEURS OPERATOIRES

- durée du clampage aortique
- hypotension prolongée

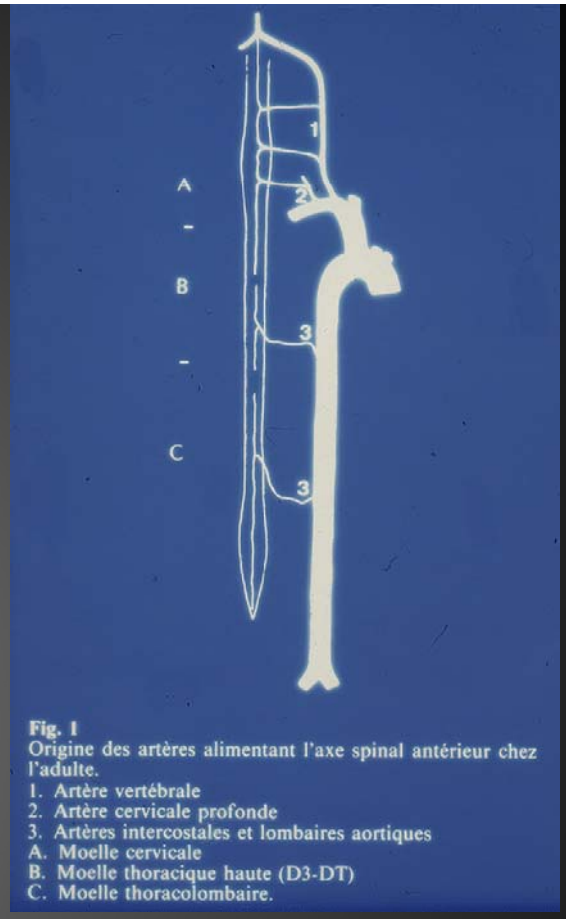
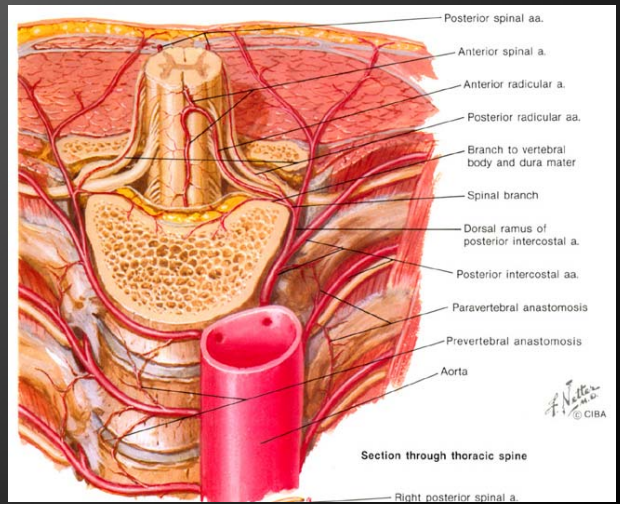


Fig. 1
Origine des artères alimentant l'axe spinal antérieur chez l'adulte.
1. Artère vertébrale
2. Artère cervicale profonde
3. Artères intercostales et lombaires aortiques
A. Moelle cervicale
B. Moelle thoracique haute (D3-DT)
C. Moelle thoracolombaire.



MOYENS DE DIAGNOSTIC

les examens complémentaires sont essentiels :

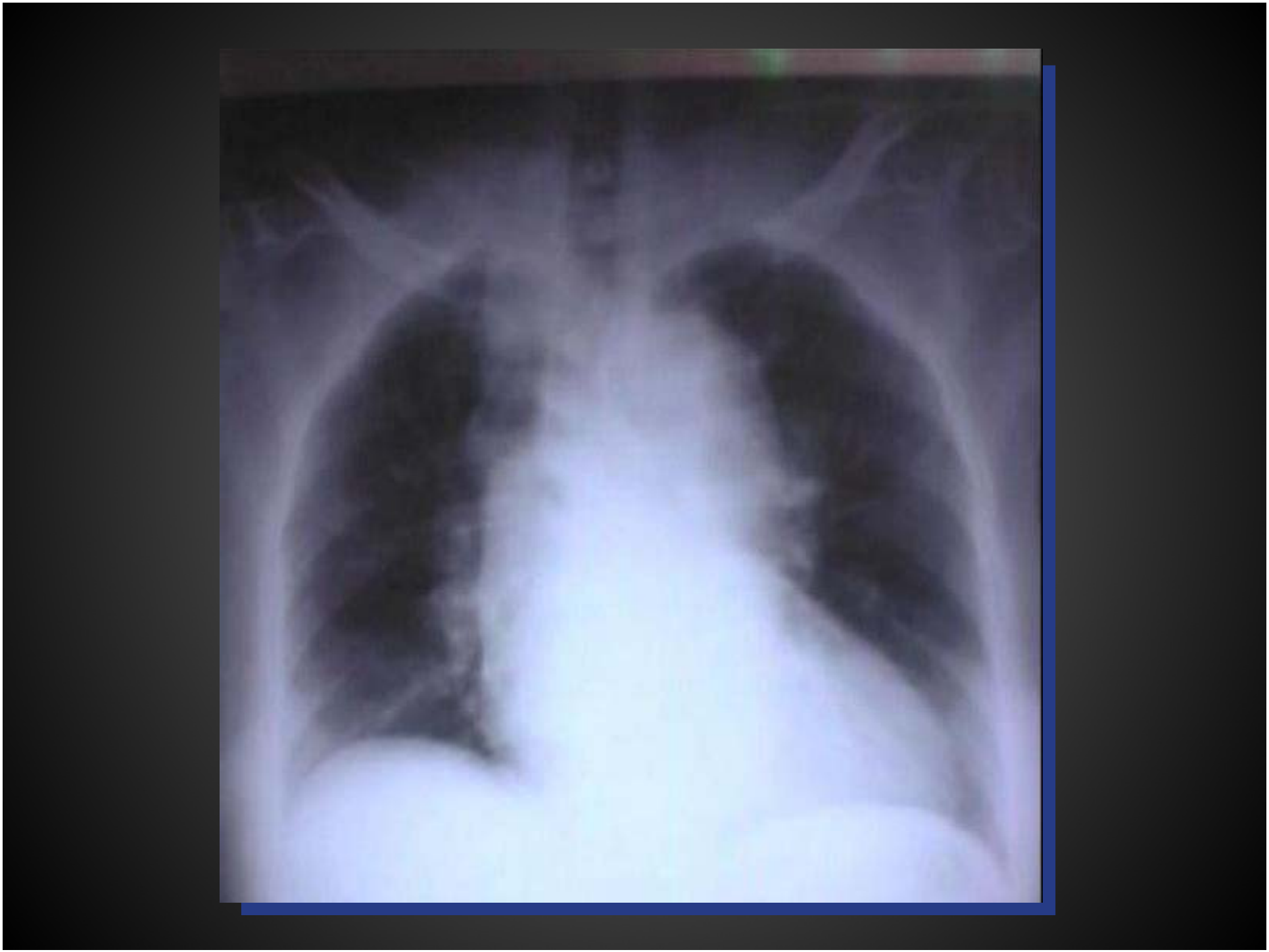
- pour confirmer le diagnostic
- pour déterminer le type A ou B
- pour préciser les modalités du traitement chirurgical éventuel

des dissections aortiques

parfois plusieurs examens sont nécessaires

QU'ATTENDONS-NOUS DES EXAMENS COMPLEMENTAIRES ?

- confirmation de la dissection aortique
- atteinte ou intégrité de l'aorte ascendante
- niveau de dissection
- sites d’entrée et de réentrée
- thrombus dans la fausse lumière
- atteinte des branches aortiques
- insuffisance aortique valvulaire
- épanchement péricardique
- atteinte des artères coronaires



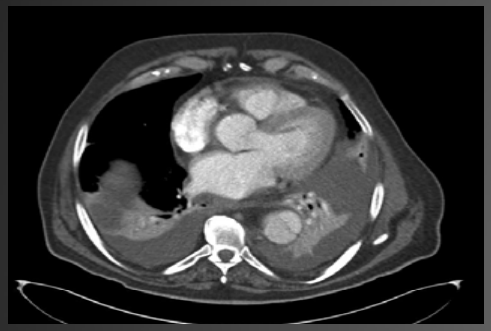
AORTOGRAPHIE

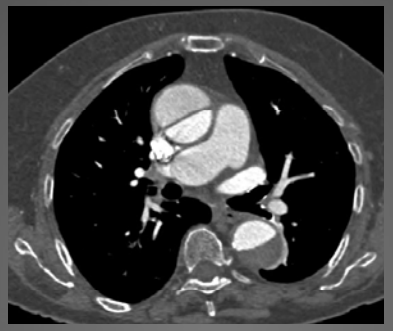
a été pendant longtemps la seule méthode possible ; maintenant elle n'est plus le premier choix

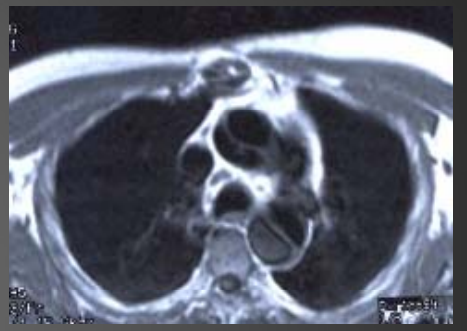
Faux-Négatifs : - thrombose de la fausse lumière
- opacification simultanée des 2 lumières
- déchirure intimale inhabituelle ou proximale par rapport au cathéter
- flap intimal non tangentiel aux rayons X

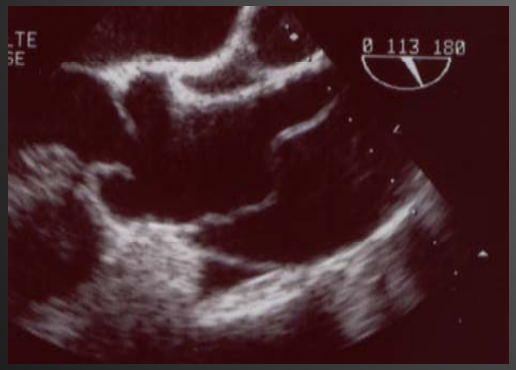
Diagnosis of Acute Aortic Syndrome

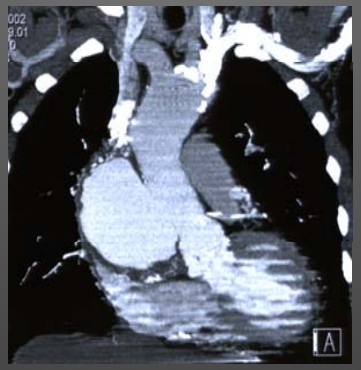
« conventional diagnostic tools »

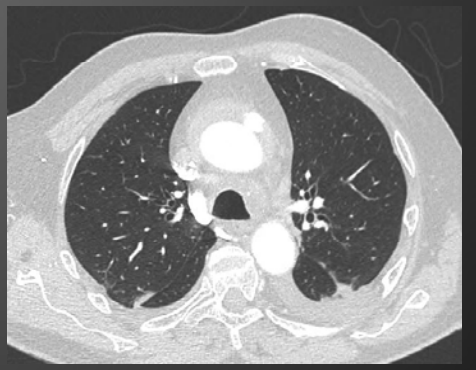












COMPARAISON
DES 4 TECHNIQUES UTILISEES

imagerie	sensibilité (%)	Classification de Stanford	
		Type A	Type B
IRM	100	100	100
CT scan	93	93	93
ETO	88	90	80
aortographie	87	87	87

TRAITEMENT

HISTOIRE NATURELLE DES DISSECTIONS

Type A

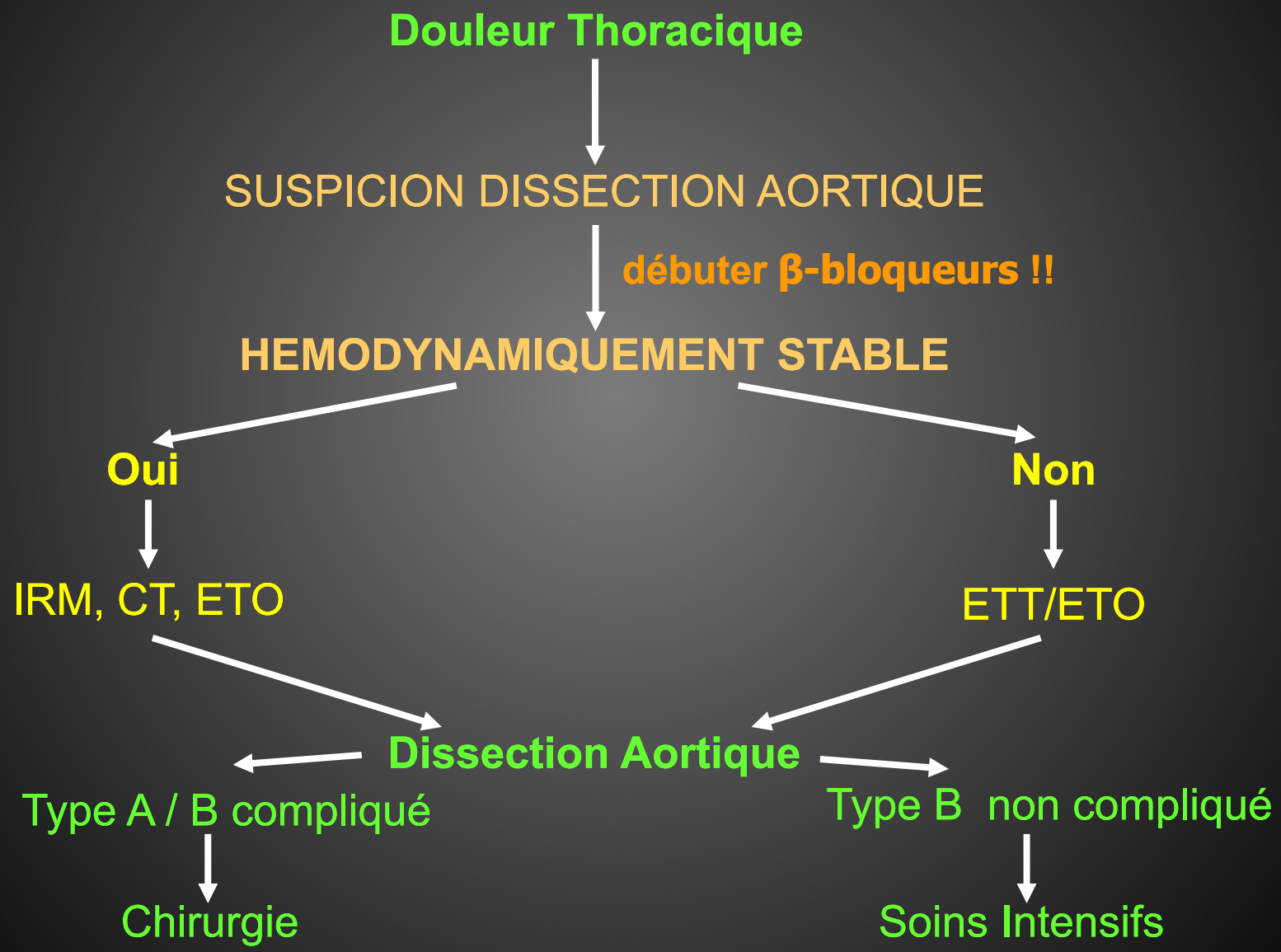
la mortalité est de 2% par heure au début !!!

- 33% de mortalité, après 24H
- 50% de mortalité, après 48H
- 75% de mortalité, à 2 semaines

Type B

non compliqué,	10% : 1er mois
compliqué,	20% : deux 1er jours
	30% : 1er mois

SCHEMA DECISIONNEL



Le traitement médical **IMMEDIAT** est impératif avant même qu'on ait terminé la mise au point et défini le type A ou B, car ce traitement s'attaque au moteur de la dissection en diminuant :

- la pression artérielle (PA) ⬅ hypot.
- la vitesse d'installation de la PA (dP/dt) ⬅ β-bloq.

Le traitement médical est continué jusqu'au moment de la chirurgie (A, et B compliqué) ou constitue l'essentiel du traitement (B non compliqué).

LE TRAITEMENT CHIRURGICAL IMPLIQUE :

1) la résection d'une partie de l'aorte
   - l'enlèvement de la porte d'entrée
   - l'oblitération de la fausse lumière

   toujours dans les types A et dans les types B compliqués ou menaçants

2) dans les types A, en outre, **souvent** RVA (ou plastie) + réimplantation des coronaires

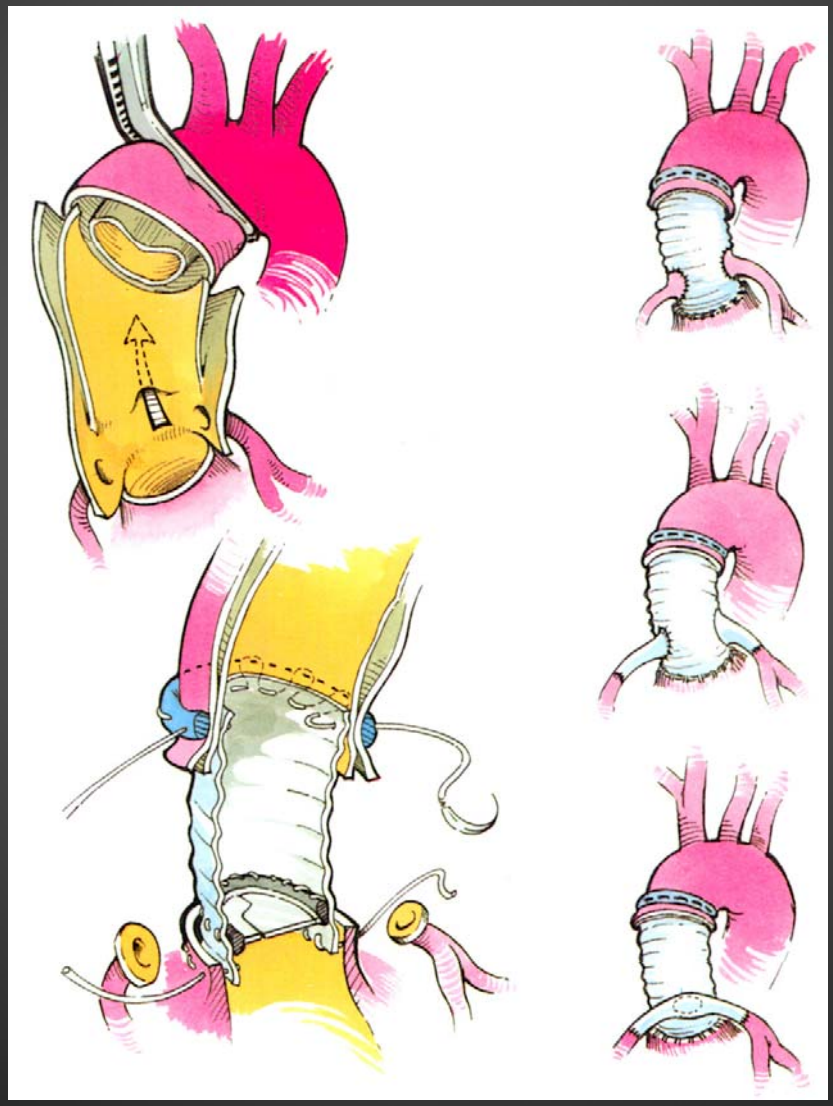
MODALITES DU TRAITEMENT CHIRURGICAL DES DISSECTIONS DE TYPE A

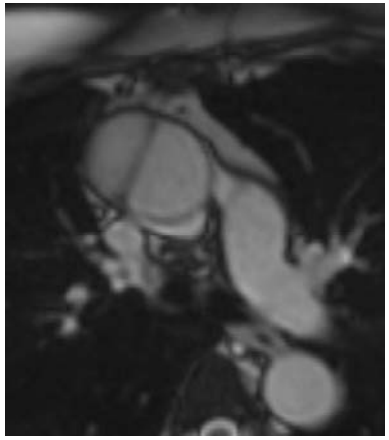
- circulation extra-corporelle avec canulation de la fémorale
- remplacement de l'aorte ascendante
  - remplacement valvulaire aortique
  - réimplantation des coronaires
    - opération de Bentall
  - ou resuspension de la valvule aortique et réimplantation des coronaires
    - opération de Tirone David
- arrêt circulatoire sous hypothermie profonde (18° C) pour les dissections complexes (Crawford - Kouchoukos)

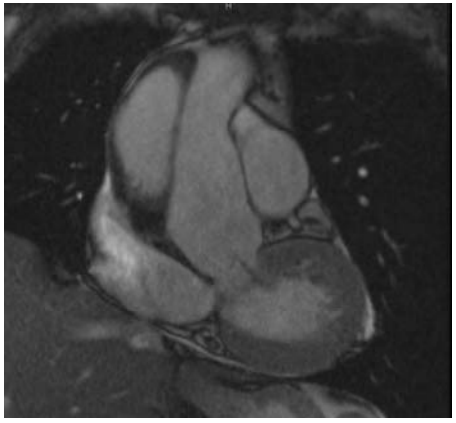
CHOIX DU TRAITEMENT DES DISSECTIONS AIGUES DE L'AORTE

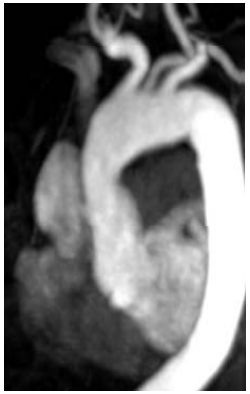
- CHIRURGIE
  - traitement impérieux de choix pour la dissection de type A
- CHIRURGIE DIRECTE OU ENDOVASCULAIRE ("stentgraft")
  - toute dissection de type B ???
  - dissections de type B compliquées :
    - rupture ou rupture imminente
    - formation d'un anévrysme sacculaire
    - ischémie viscérale (mésentérique, rénale)
- TRAITEMENT MEDICAL
  - dissection de type B non compliquée ????
  - dissection de type B subaiguë ou chronique ????
    (diagnostic tardif ≥ 2 semaines)

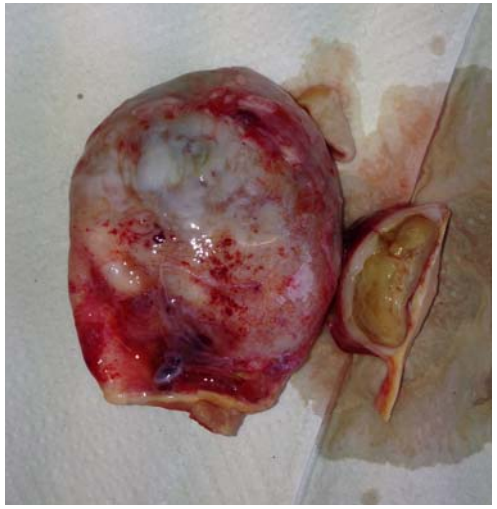
TECHNIQUES CHIRURGICALES (Type A)

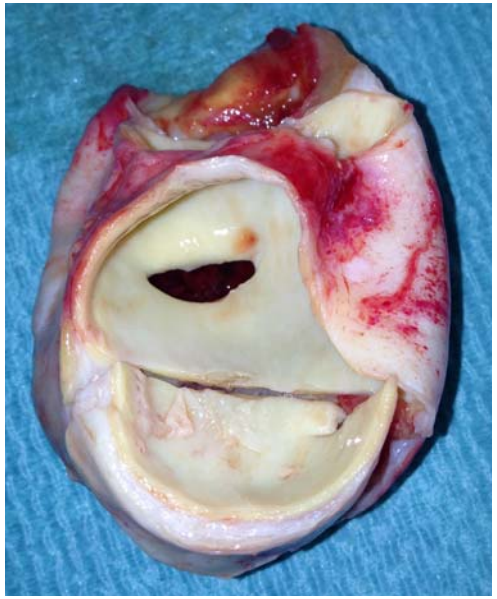


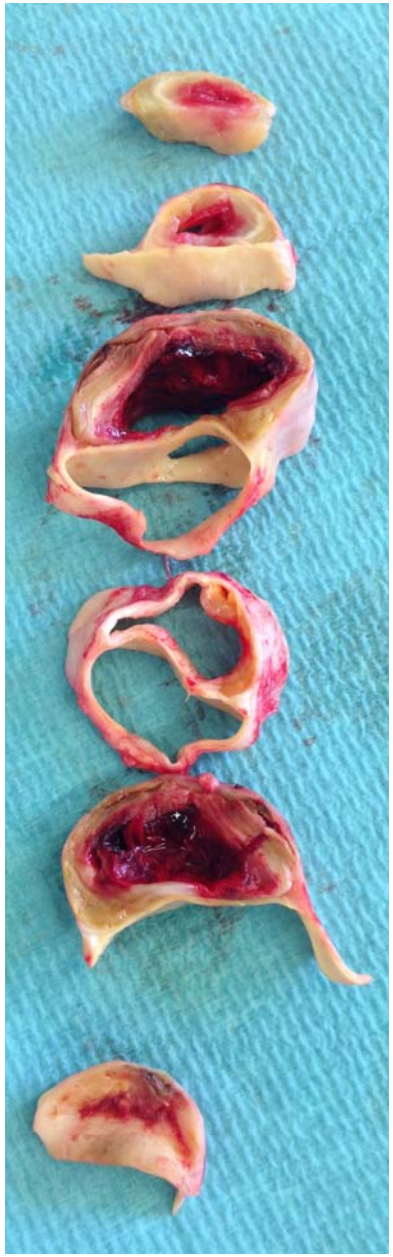




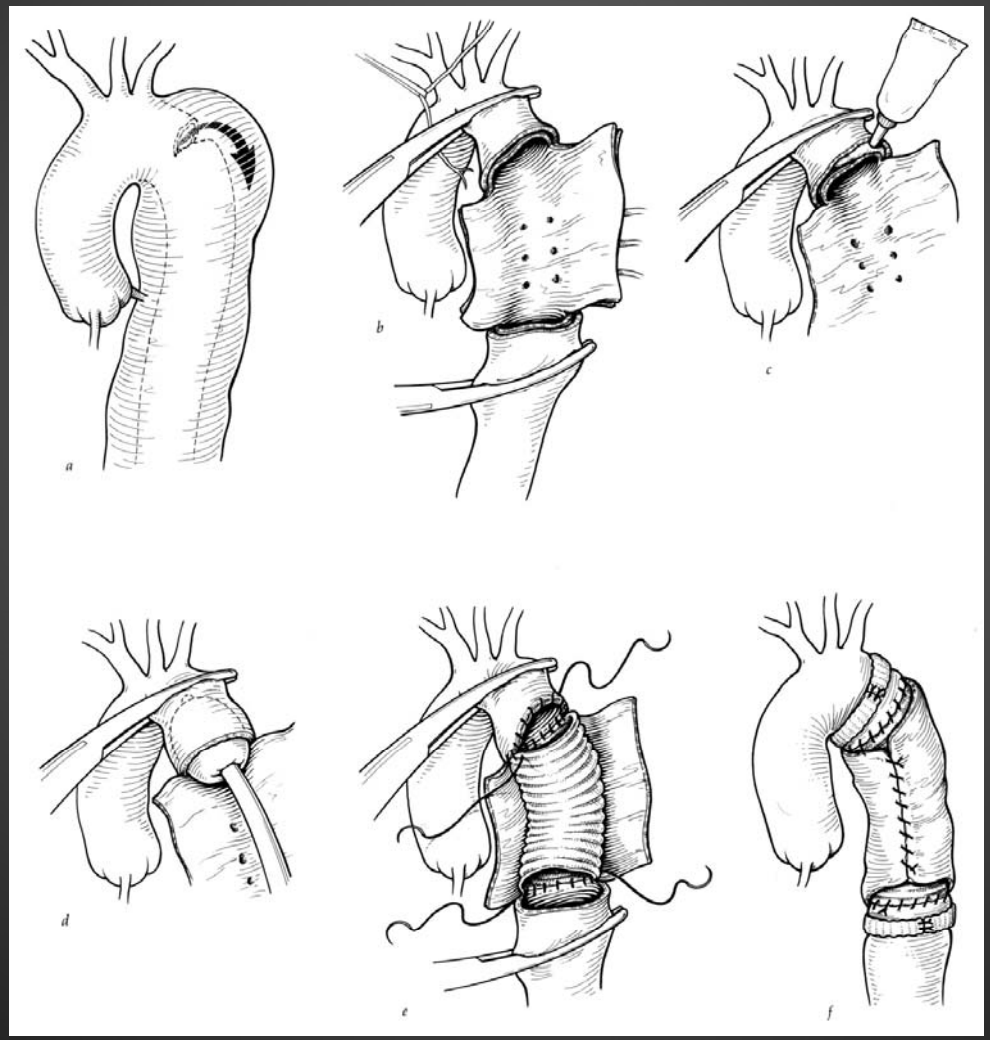


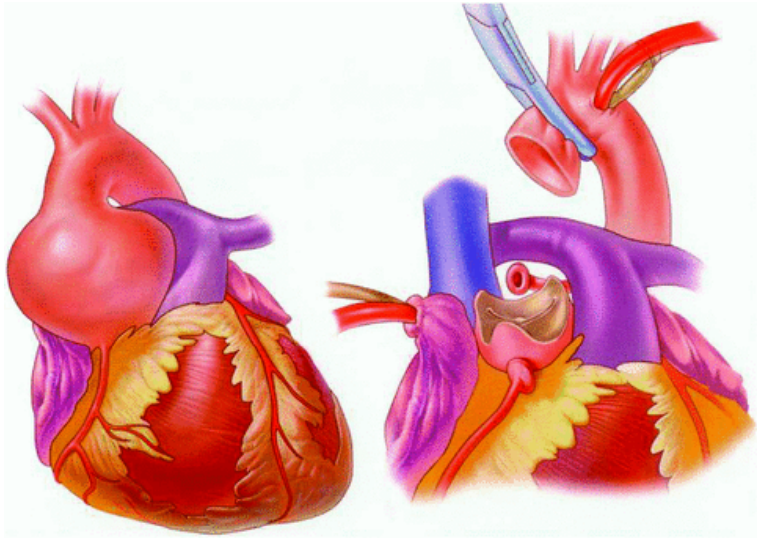


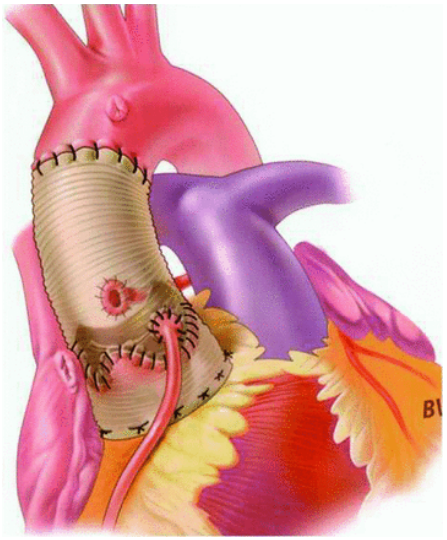


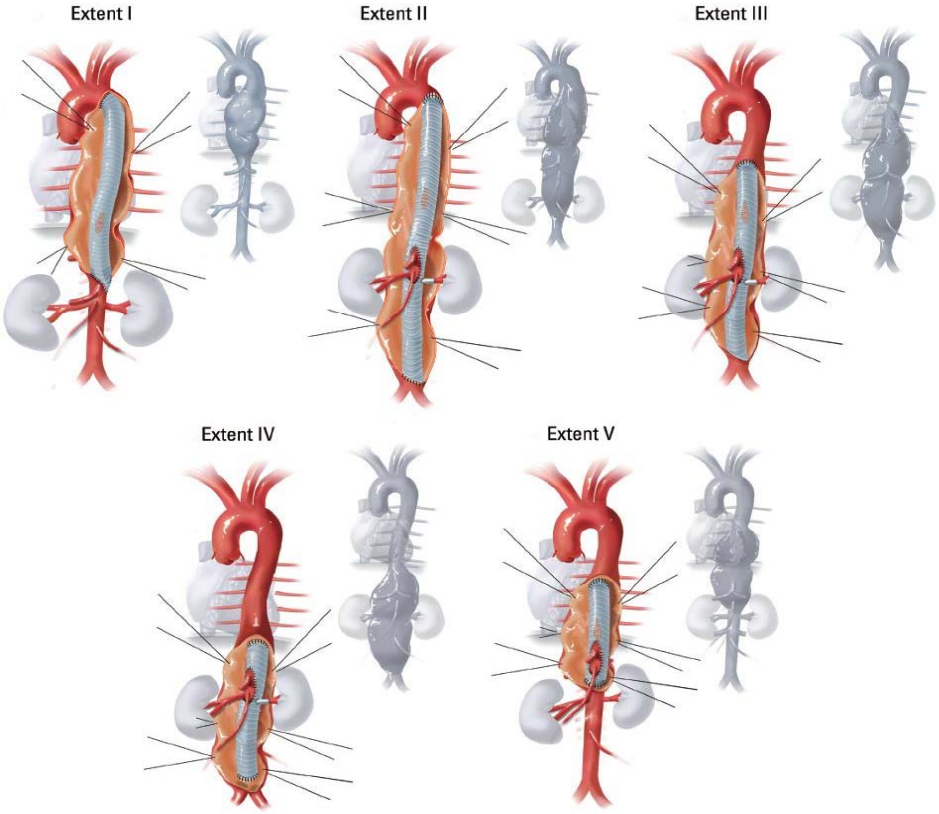


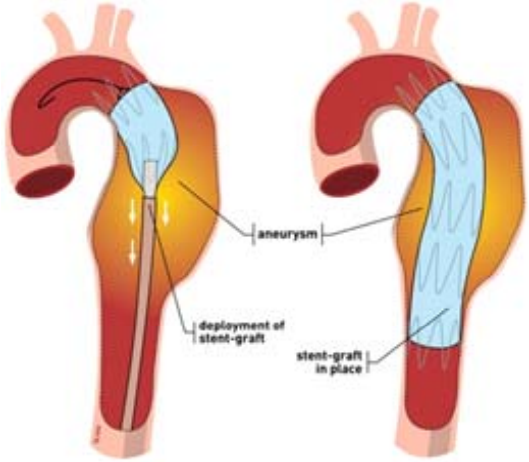
TECHNIQUES CHIRURGICALES (Type B)



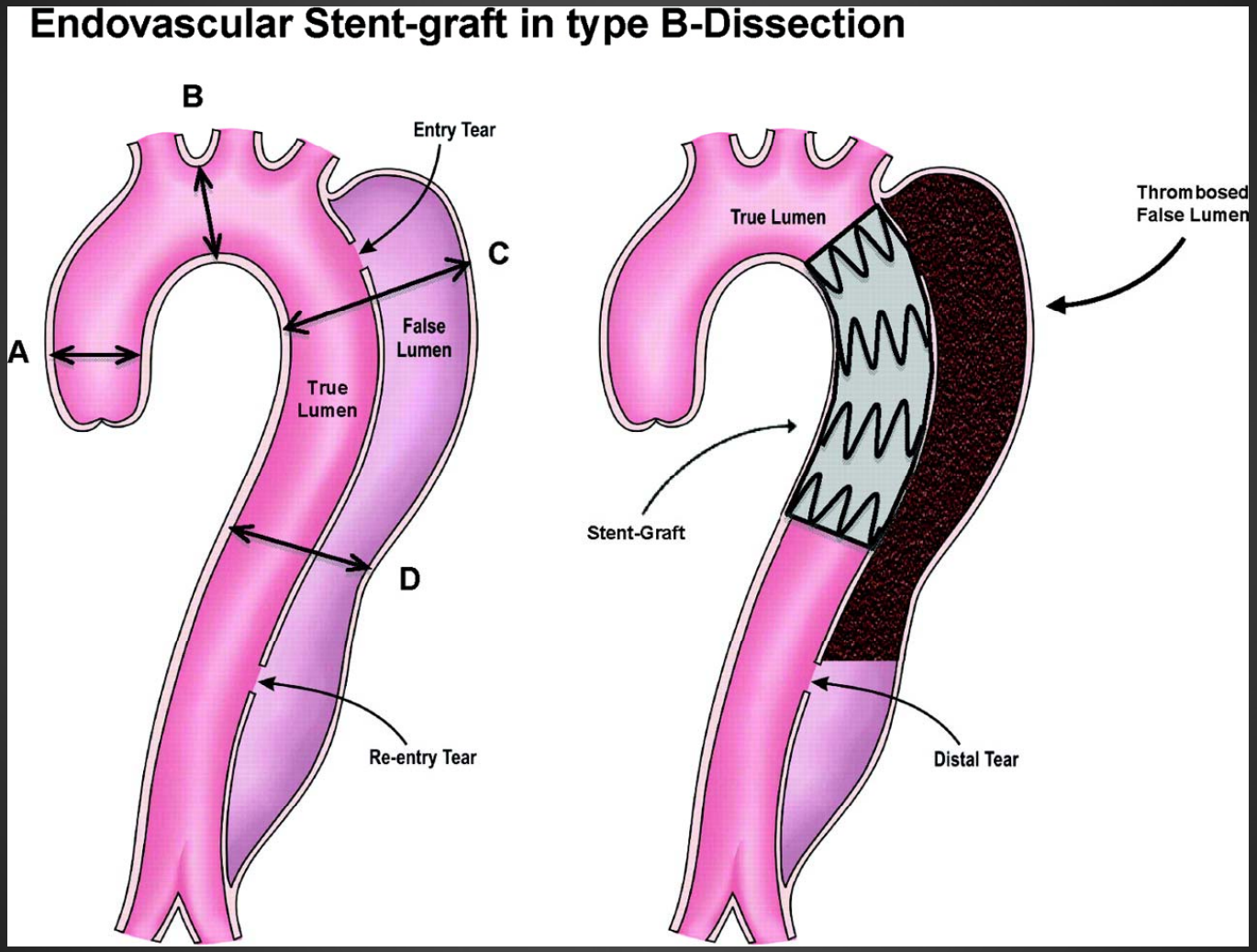





Extent I
Extent II
Extent III
Extent IV
Extent V


aneurysm
deployment of stent-graft
stent-graft in place

Endovascular stent graft in type B dissection



Nienaber, C. A. et al. Circulation 2009;120:2519-2528

Circulation

American Heart Association
Learn and Live

Case 1

83 year-old woman
Arterial Hypertension
Diabetes
COPD
Dorsal & left scapular pain

Δ: Acromioclavicular Arthritis

Θ : Medical treatment against scapular pain

Pace – Maker control by her Cardiologist

T T E: Type A Aortic dissection

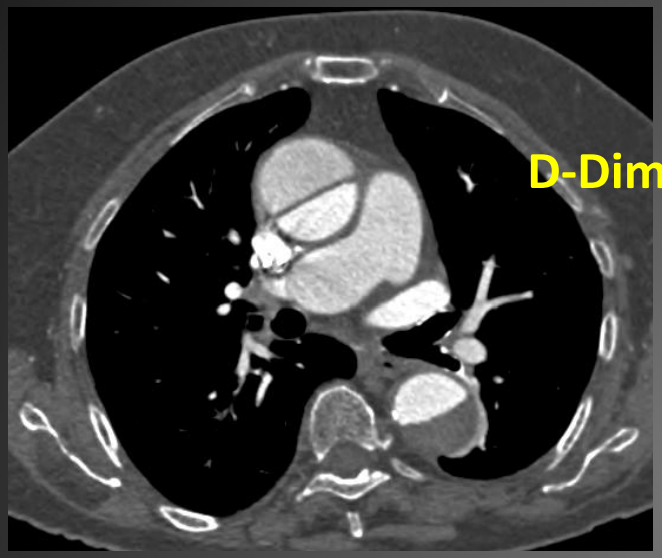
At admission:

Clinics : Thoracic and dorsal pain,

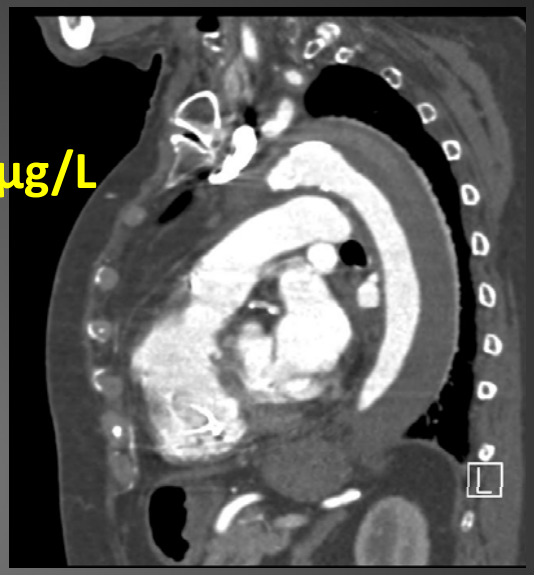
Blood test: NI except D-Dimer 1780 μg/L

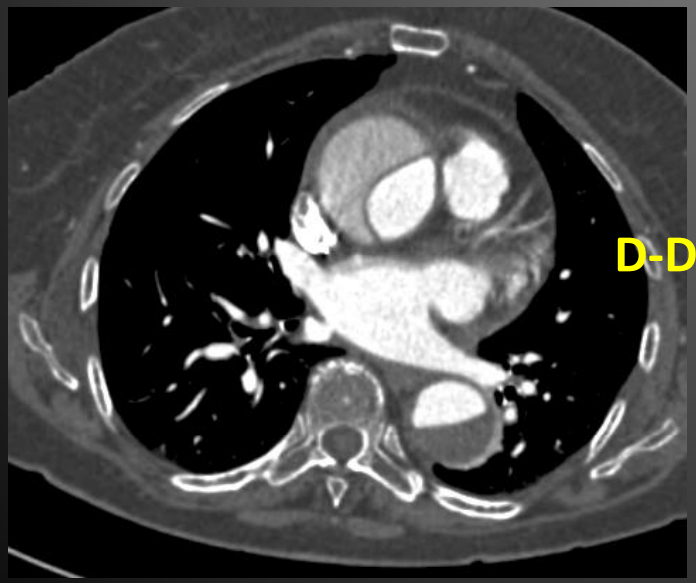
CT Angiography: Confirmation of Type A dissection

Case 1,Type A Aortic dissection

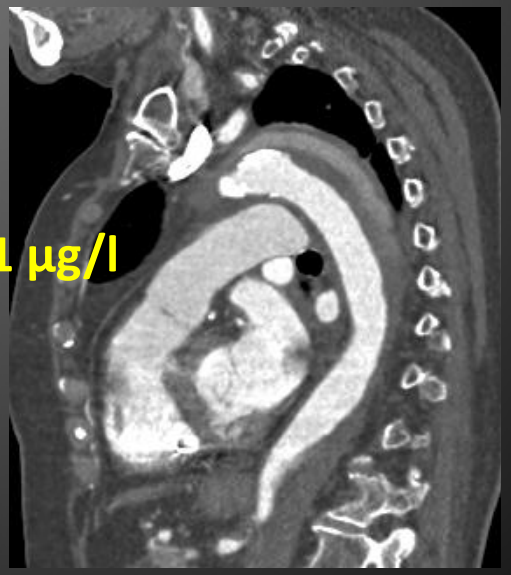


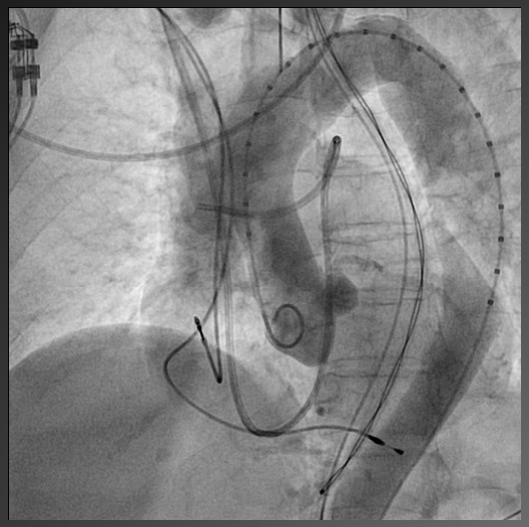
D-Dimer 1708 µg/L

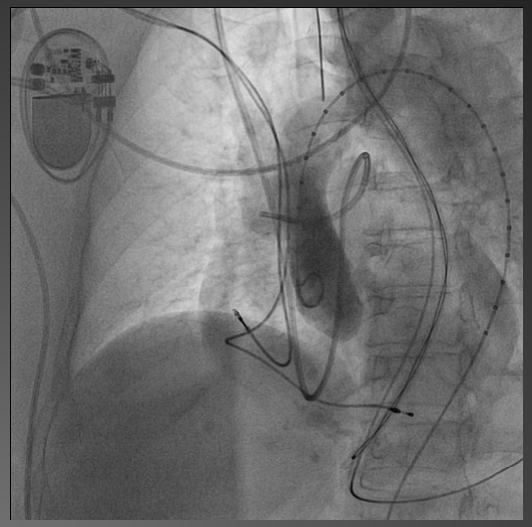


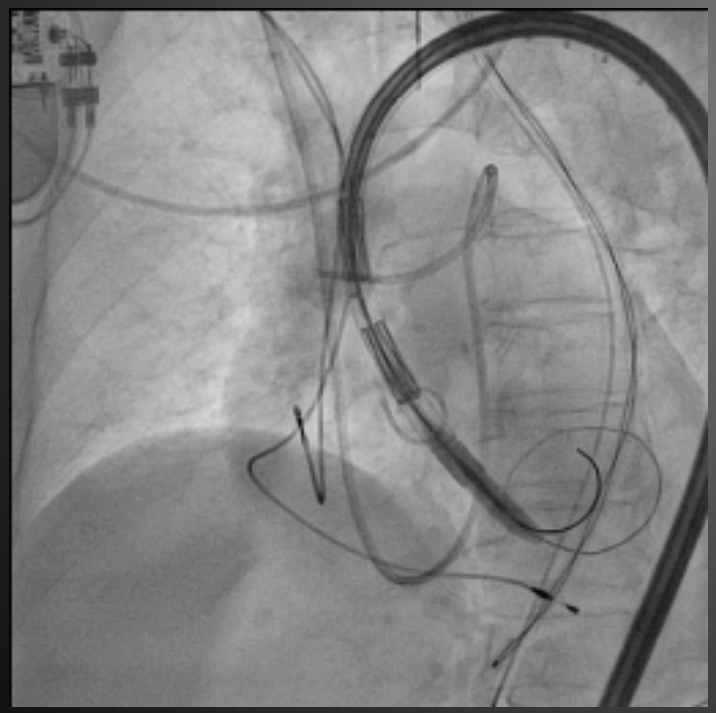


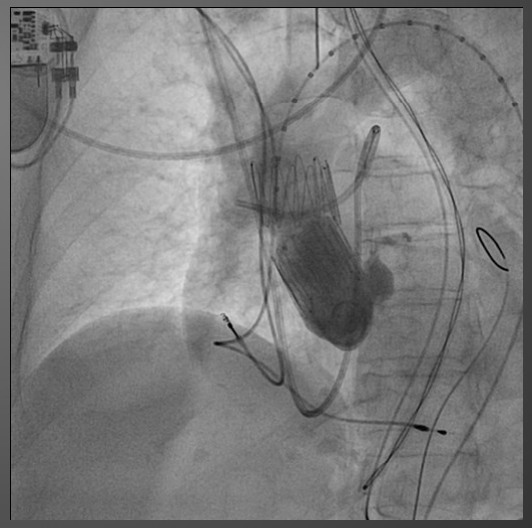
D-Dimer 5871 µg/l





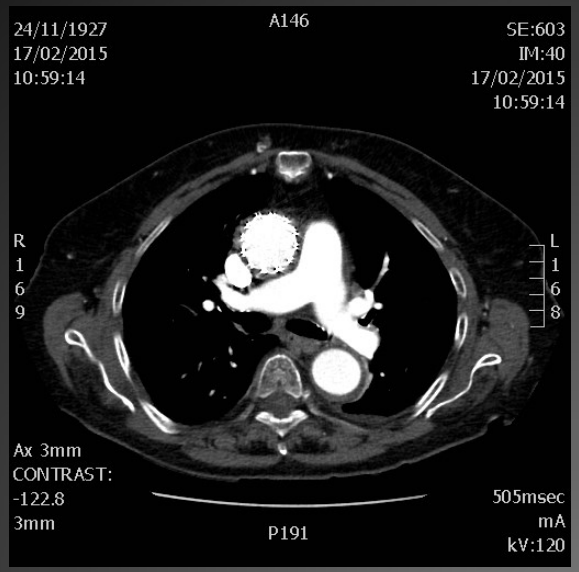


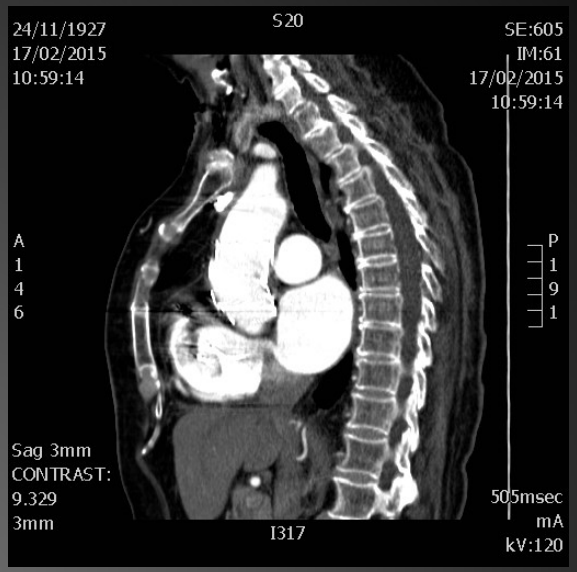


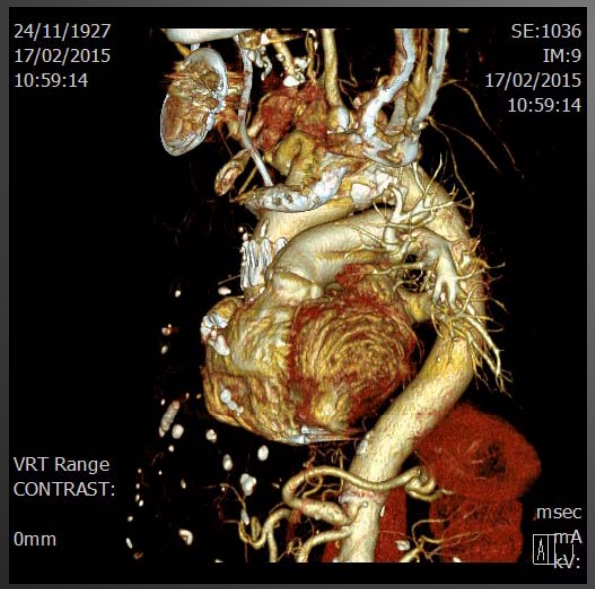


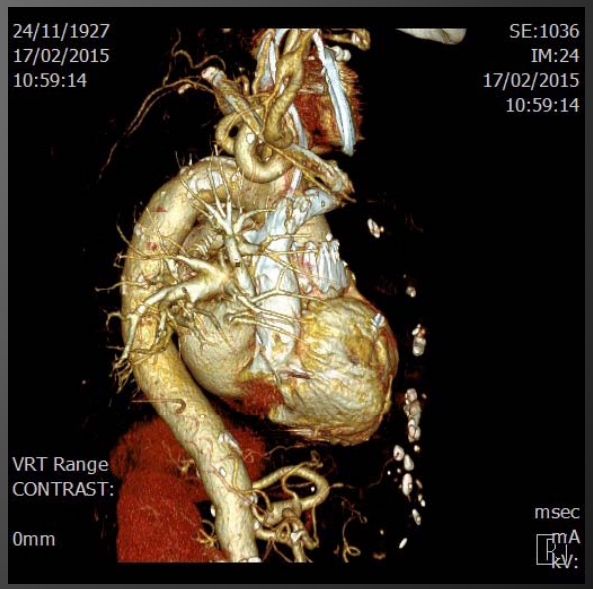
TEVAR in (type A) proximal aortic dissection, Ready for a broader application?

Christoph A. Nienaber[1], Natzi Sakalihasan[2], Rachel E. Clough[3], Mohamed Aboukoura[4], Enrico Mancuso[1], Nick Cheshire, Ulrich Peter Rosendahl[1], Cesare Quarto[1], John Pepper[1]









TEVAR in (type A) proximal aortic dissection, Ready for a broader application?

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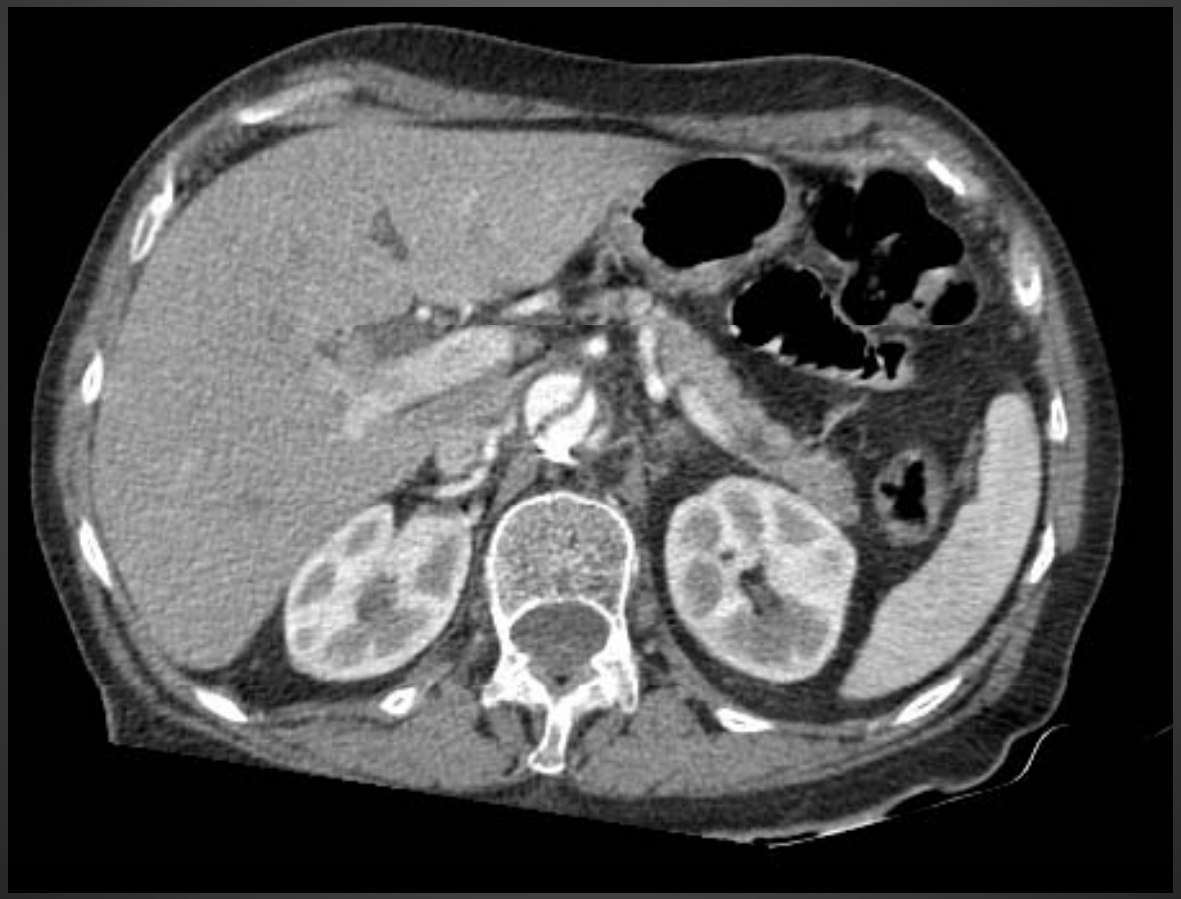
Case 2

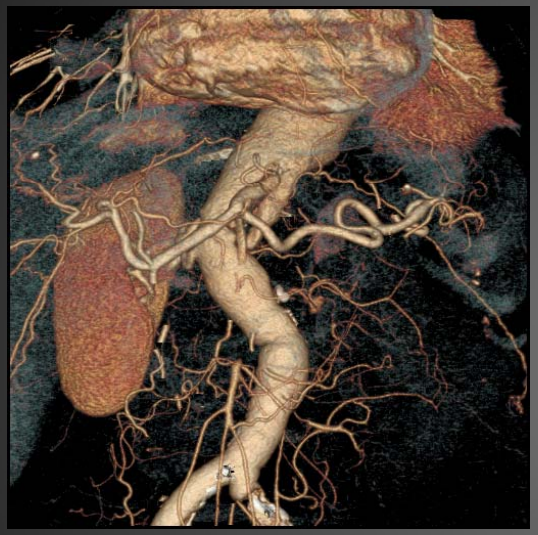
68 year-old woman
smoker
Arterial Hypertension
Syncope followed by paraplegia during 1h.

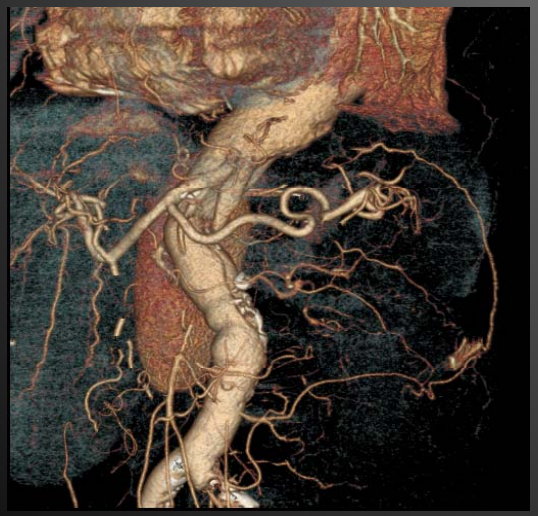
At admission:
Paraplegia replaced by paresthesia
Cerabral CT: Normal
Thoracic and dorsal pain,
Blood test: NI except D-Dimer 1570 µg/L
CT Angiography: Type B dissection

Case 2, Type B dissection

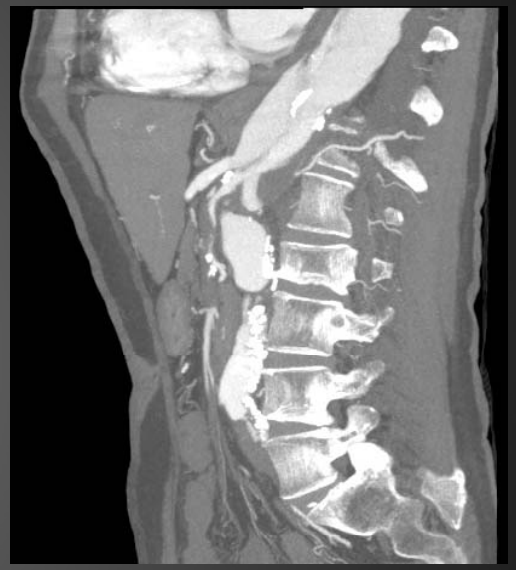


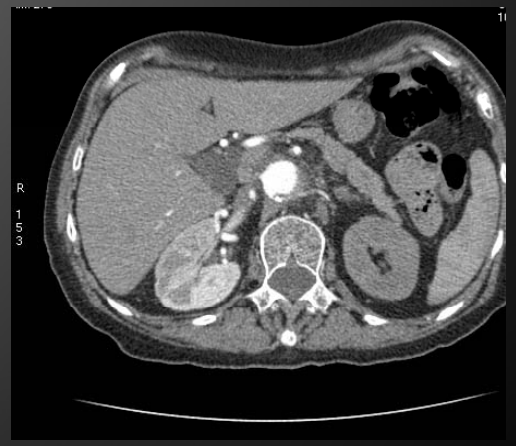
Complicated Type B dissection



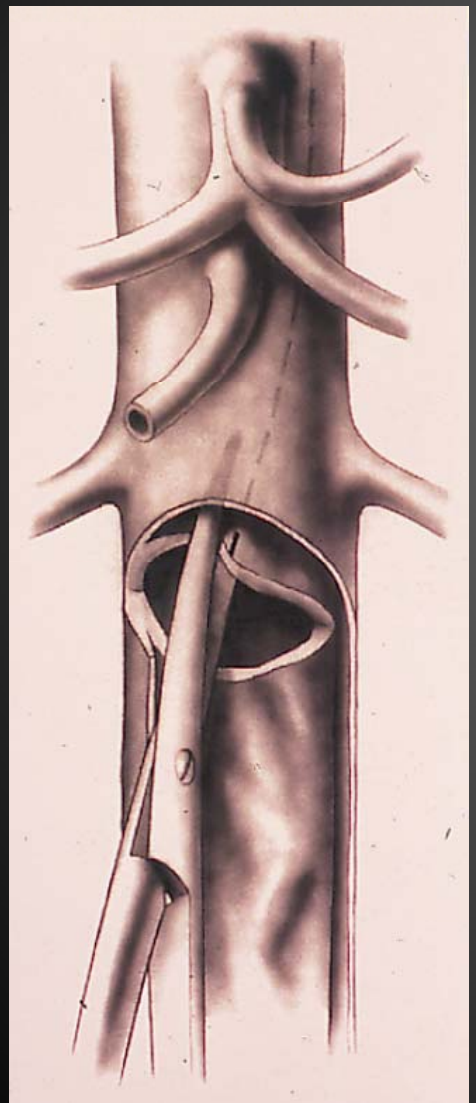


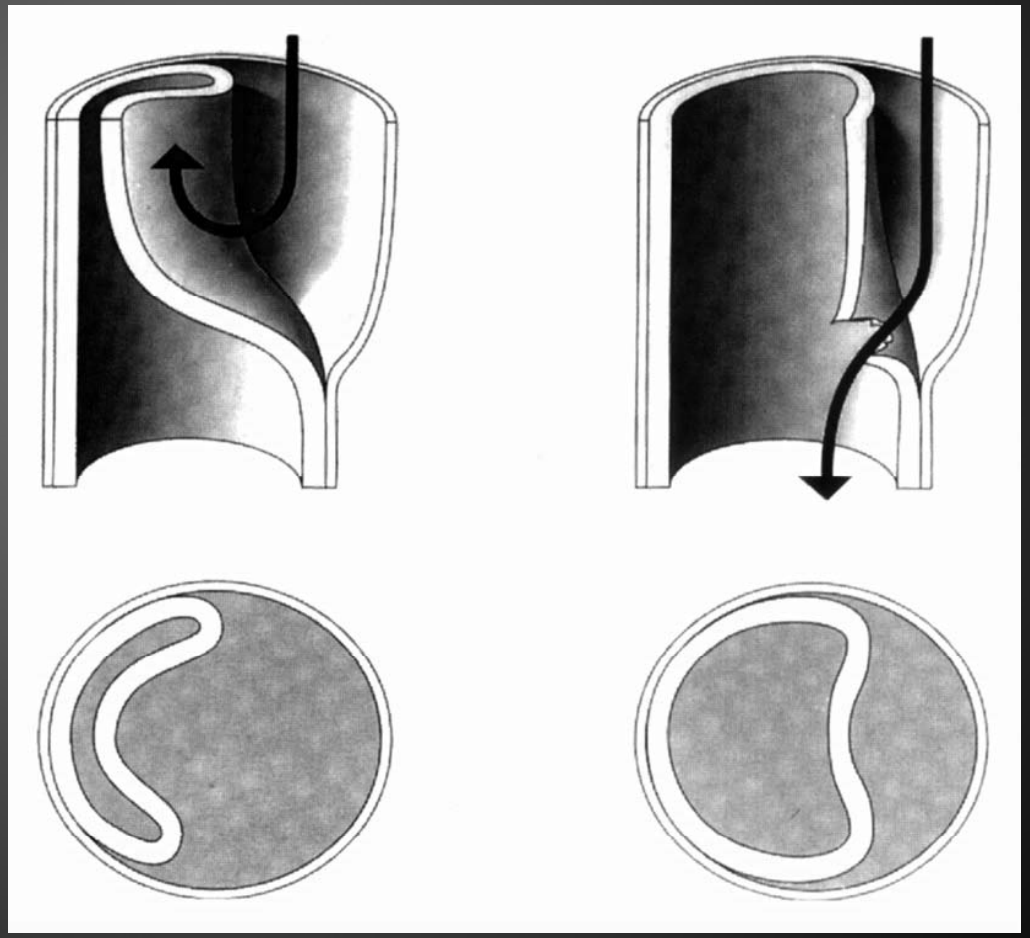
D-Dimer 1692 µg/l



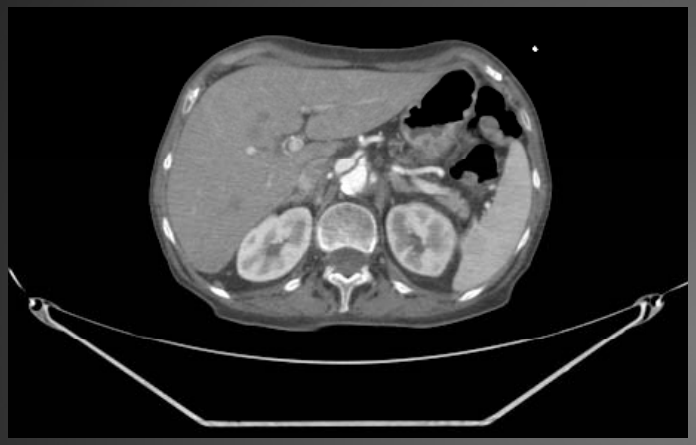


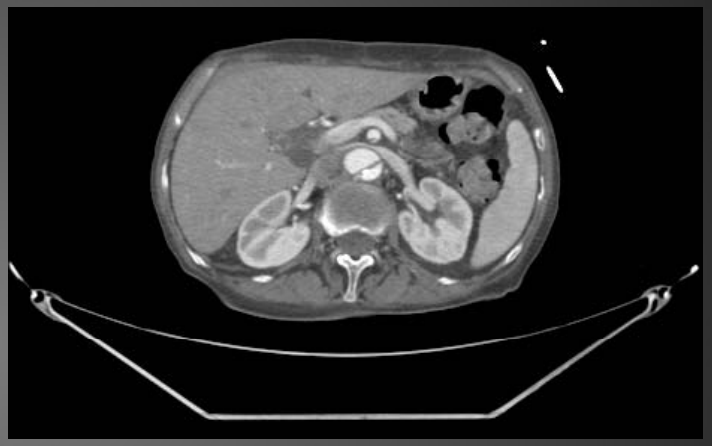
SURGICAL FENESTRATION



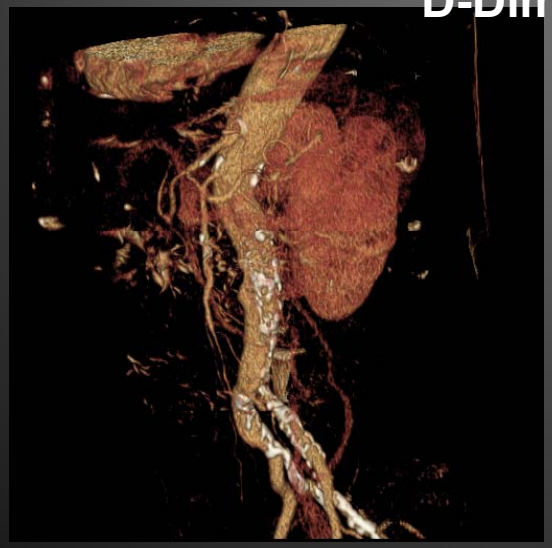


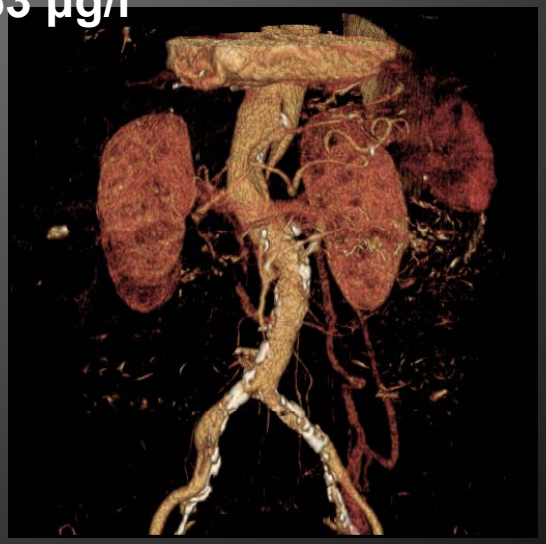
Surgical treatment of complicated type B dissection





D-Dimer 963 µg/l

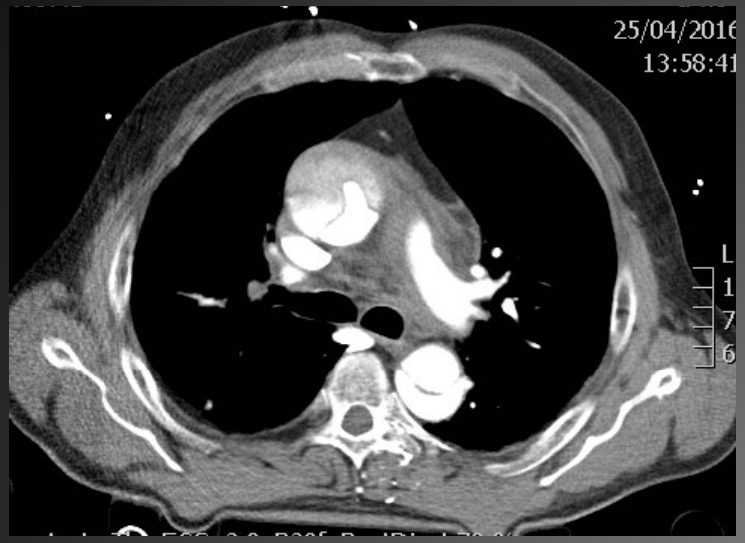




CASE 3

73 year-old woman
smoker
Arterial Hypertension
Followed TAA , 46mm (25/01/2016)
Dorsal pain with irradiation to two legs,
Transfer to hospital

At admission:
Thoracic and dorsal pain,
Hypotension
CT Angiography: Type A dissection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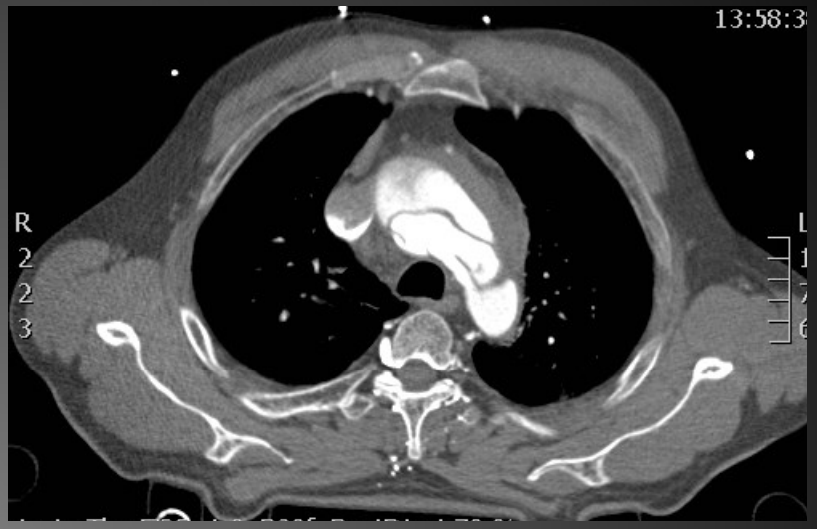
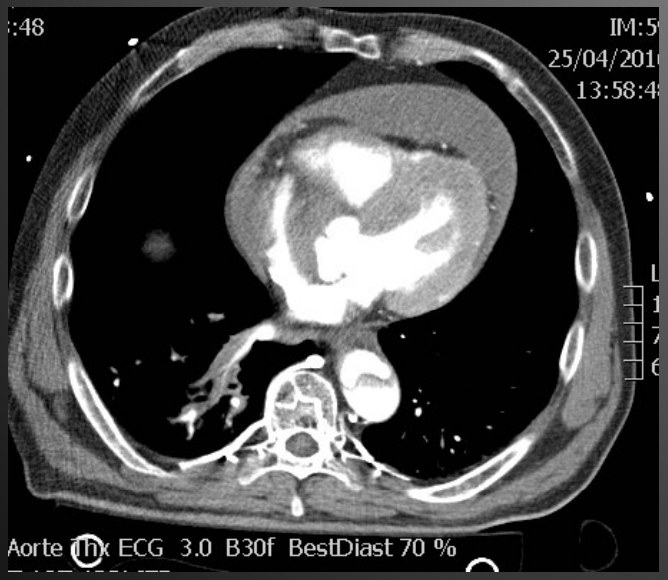
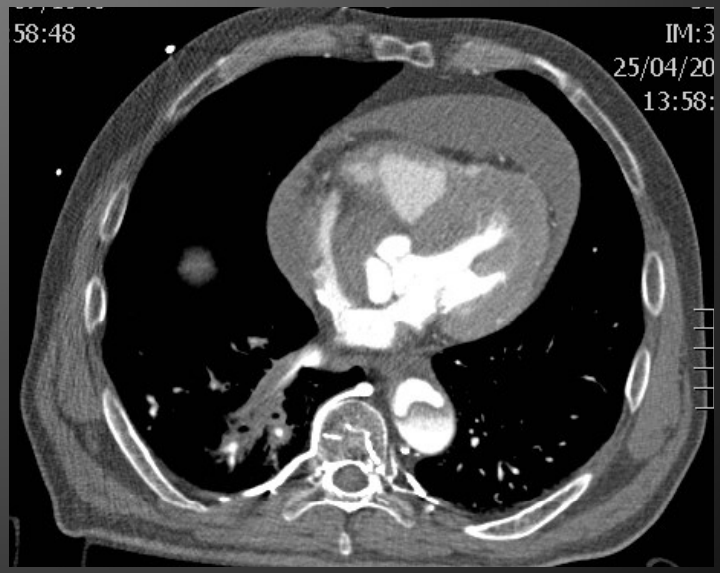

13:58:3
R
2
2
3


:48
IM:5
25/04/201
13:58:4
Aorte thx ECG 3.0 B30f BestDiast 70 %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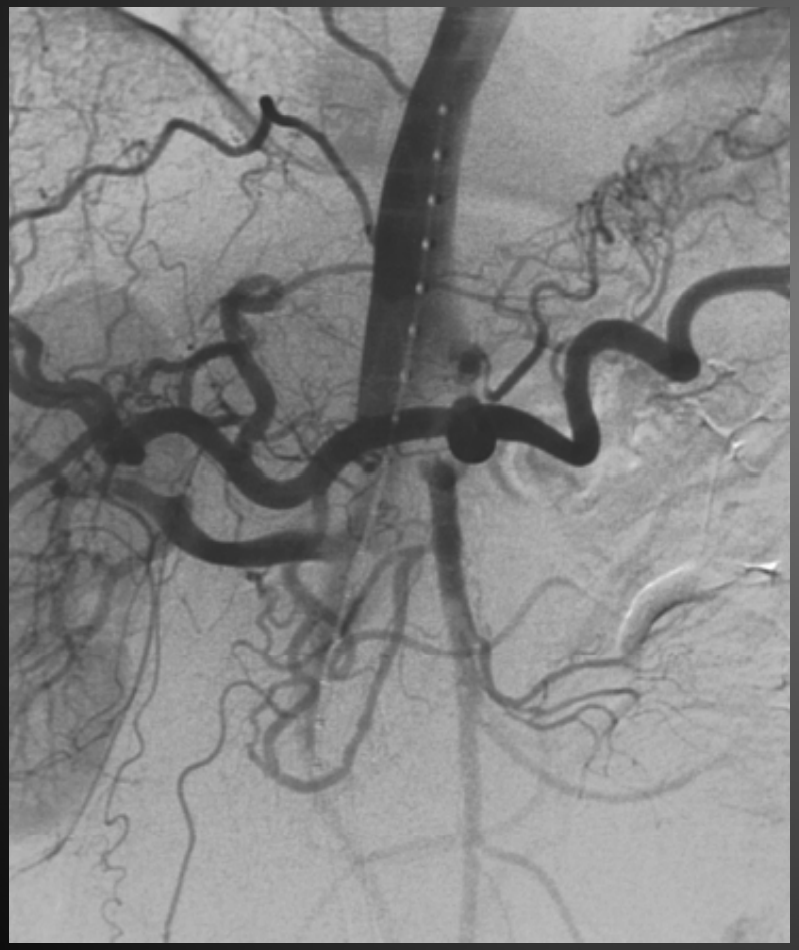
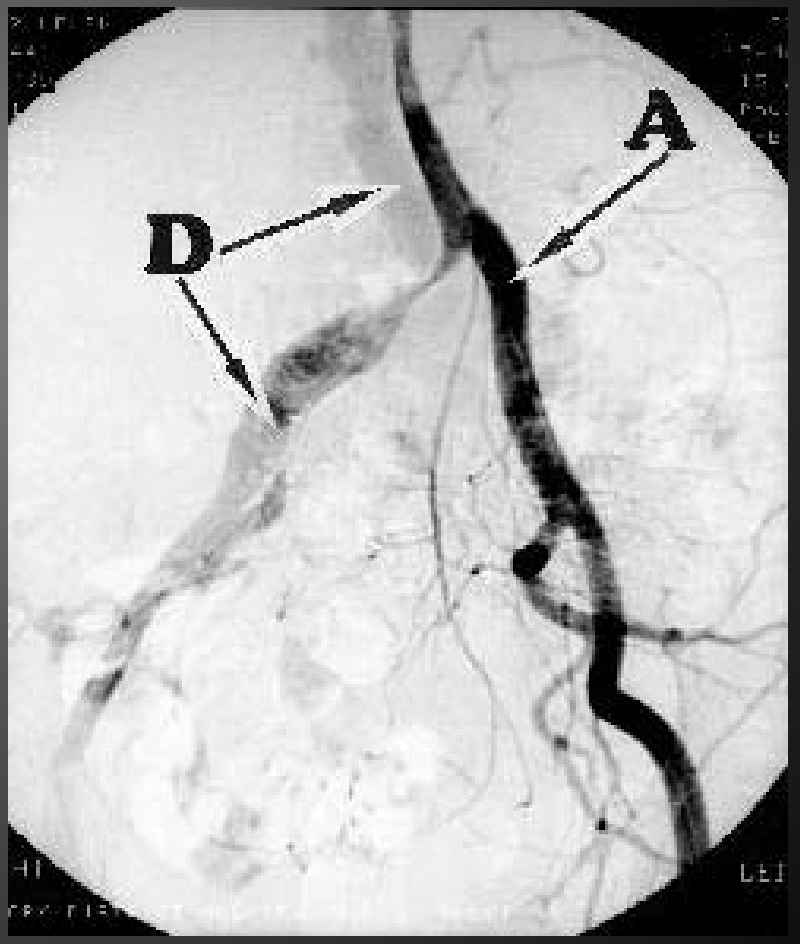
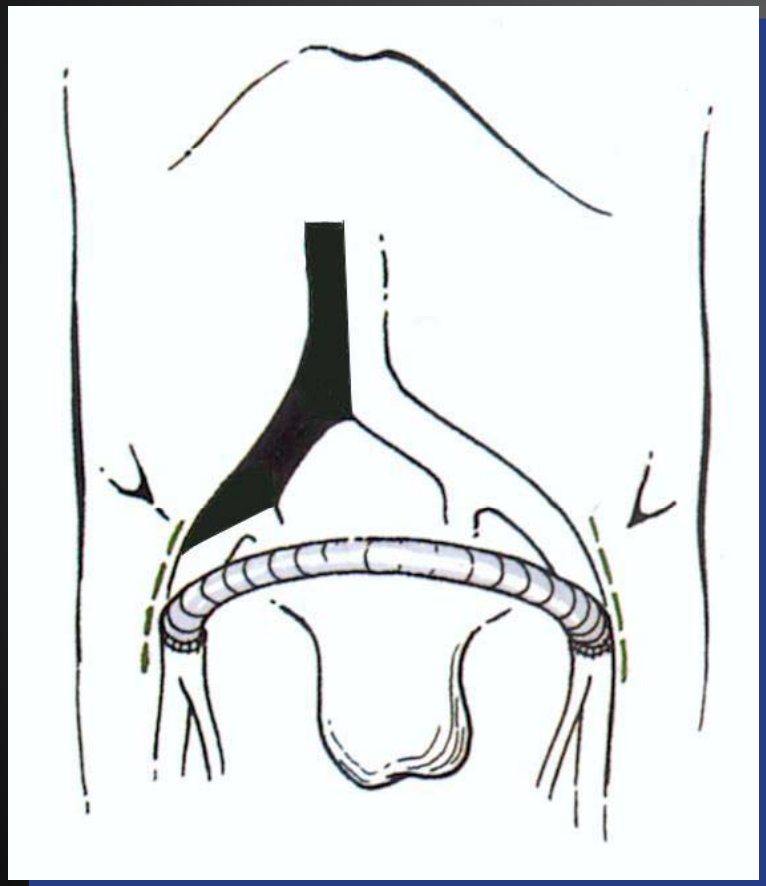
25/04/20
13:58:

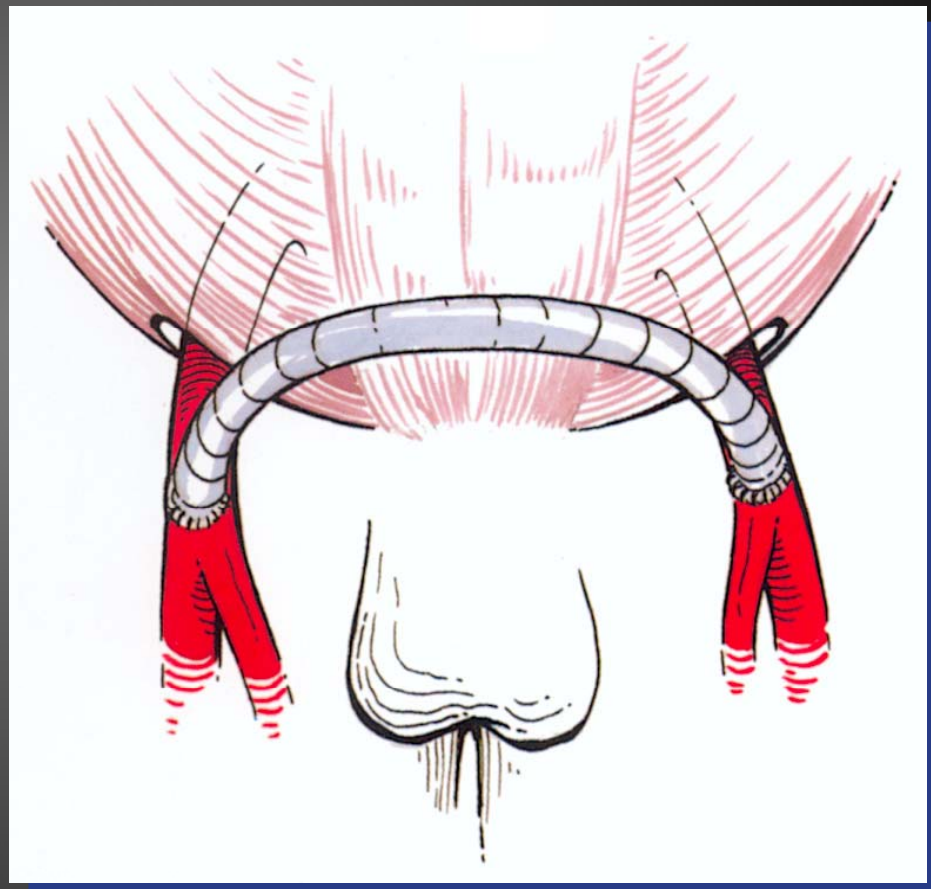
Complicated Type B dissection



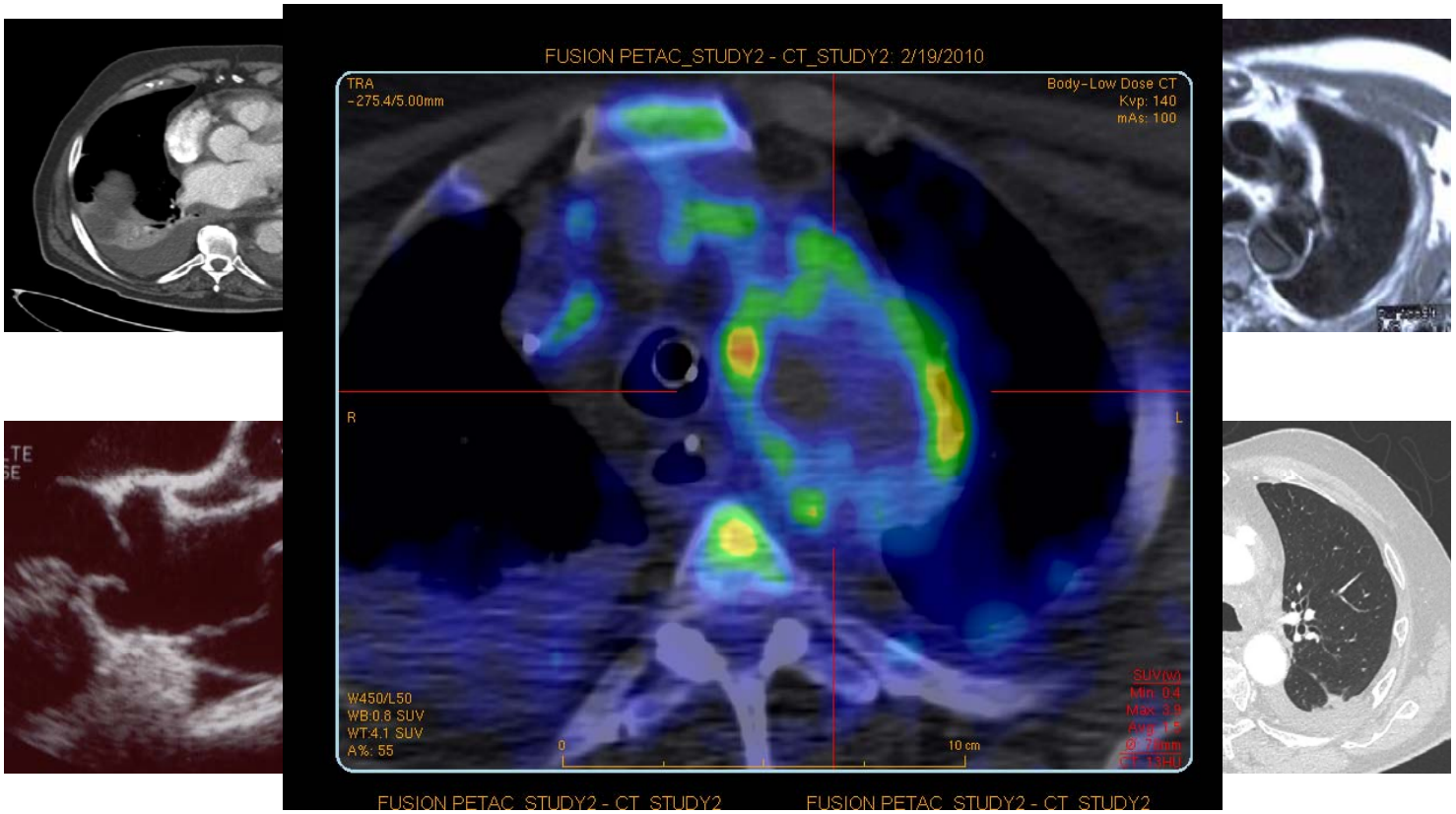


PONTAGE EXTRA-ANATOMIQUE

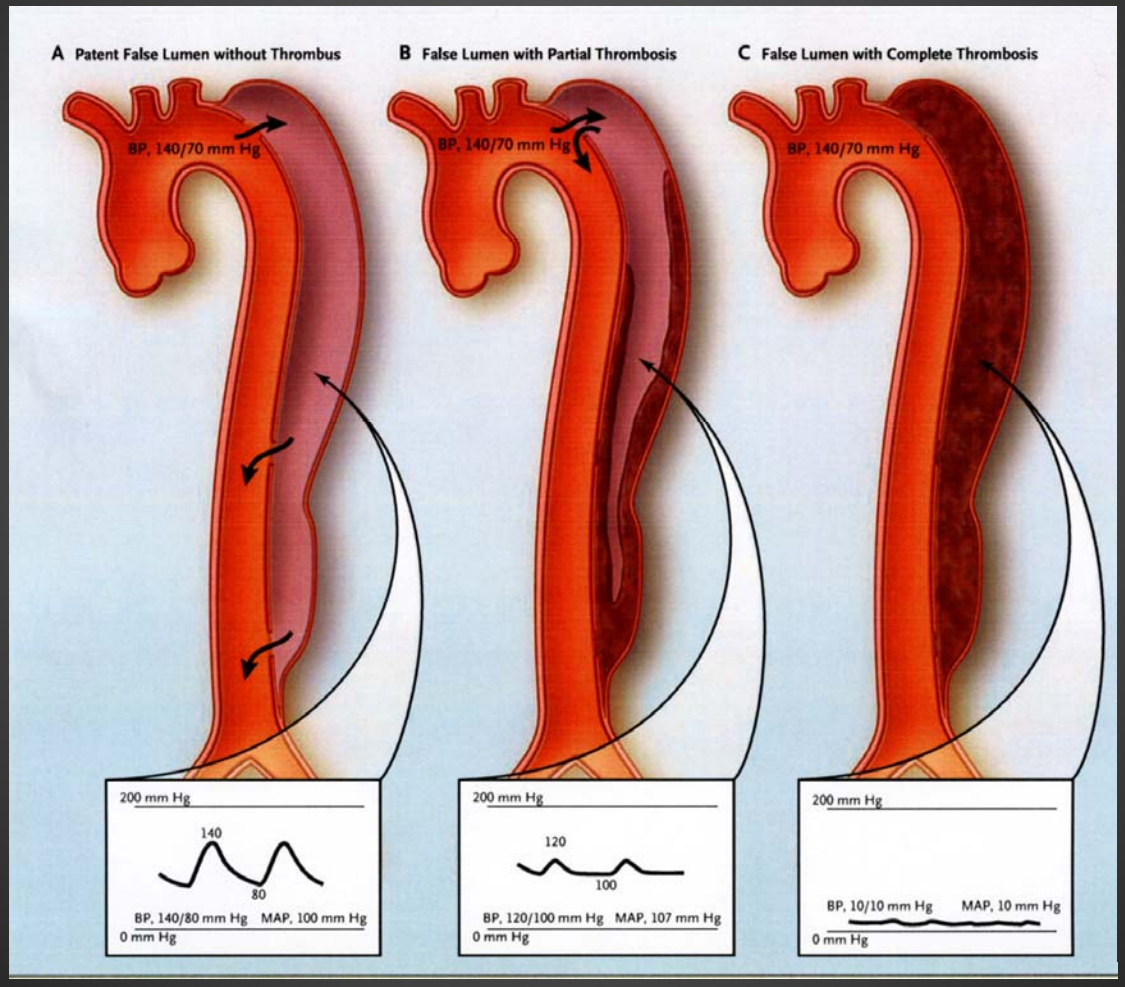




Can we predict the complications in Type B dissections?

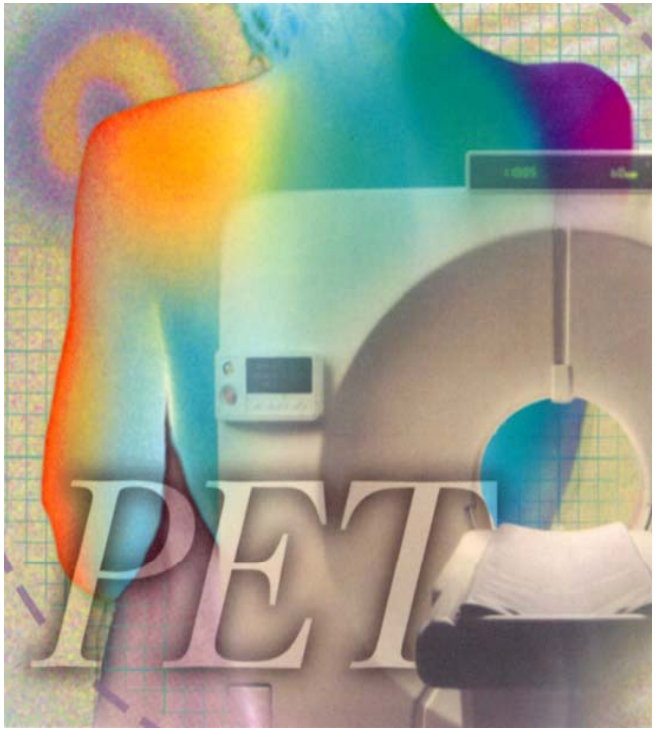


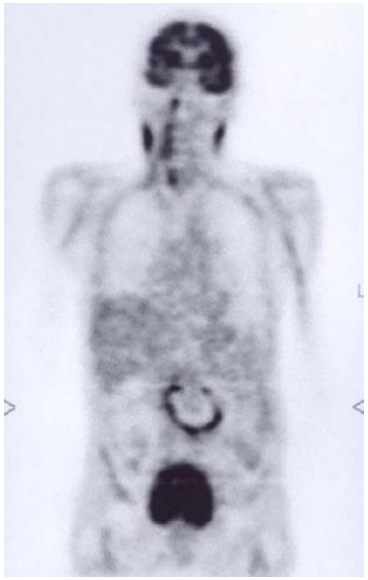
Prognosis of the Type B aortic dissection

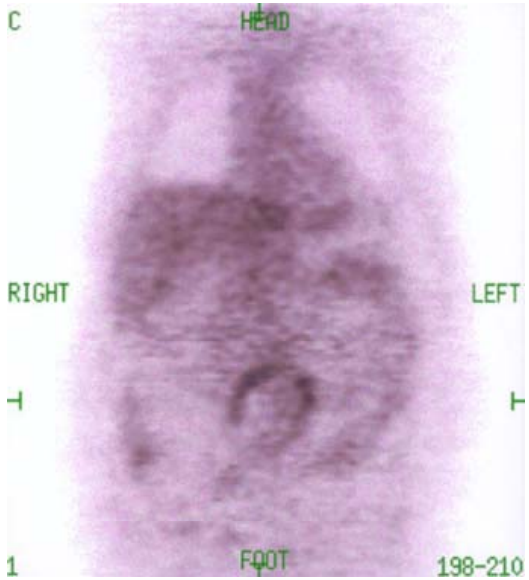


Thomas T. Tsai et all, N Engl J Med 357;4 July 26, 2007

Positron Emission Tomography (PET) with 18F-Flurodeoxyglucose (FDG)







Sakalihasan et al, Eur J Vasc Endovasc Surg 2002
Sakalihasan et al, Sem Vasc Surg 2004

Defawe OD,… Sakalihasan N,Clin Nucl Med 2005

Xu Y,.. Sakalihasan N Eur J Vasc Endovasc Surg 2010

Courtois A et al; JNM 2013, Nchimi A,… Sakalihasan N. Circ Cardiovasc Imaging. 2013

Courtois A,.. Sakalihasan N. Mol Med. 2014 Dec 10. doi: 10.2119/molmed.2014.00065.

(Tissue PET) Vascular metabolic imaging and peripheral plasma biomarkers in the evolution of chronic aortic dissections

Material & Methods

23 Patients (17 M & 6 F)
(Mean FU 16,7 months)

55 PET-CT examinations
89 CT examinations

Biological analysis:

Fibrinolysis: **PAP, D-Dimers**
Thrombosis: **TAT, P-Selectine**
Neutrophil: **MMP9,Elastase,MPO**

Sakalihasan N et al; Eur Heart J Cardiovasc Imaging.2015 Jan 14.pii:jeu283.

Results:

Demographics, risk factors and medical treatment of study patients at baseline, follow-up time and secondary complications (N=23)

Characteristic	N (%)	Mean ± SD
Age (years)		62.3 ± 11.5
Sex (male)	17 (74)	
BMI (kg/m²)		28.4 ± 4.8
Current smoking	5 (22)	
Stopped smoking	12 (52)	
Diabetes mellitus	4 (17)	
Arterial hypertension	19 (83)	
Dyslipidemia [a]	13 (57)	
Acute myocardial infarction	6 (26)	
Angina pectoris	4 (17)	
Peripheral artery disease	4 (17)	
Renal insufficiency [b]	1 (4)	
COPD	8 (35)	
Other arterial aneurysms	8 (35)	
Previous type A dissection	6 (26)	
Aspirin	18 (78)	
β-blockers	14 (61)	
Statins	16 (70)	
Aortic diameter (mm)		47.2 ± 14.5
Follow-up duration (months)		16.7 ± 8.0
Secondary complications	17 (74)	

COPD indicates chronic obstructive pulmonary disease

[a] Increased serum levels of triglycerides and/or low-density lipoprotein cholesterol

[b] clearance of creatinine < 60 mL/min

Sakalihasan N et al; Eur Heart J Cardiovasc Imaging.2015 Jan 14.pii:jeu283.

Results:

Distribution of secondary complications observed during follow-up (N=23 patients)

Complication	Number (%)
Rapid growth	4 (17.4)
Progression of dissection	10 (43.5)
Aneurysm	12 (52.2)
Refractory hypertension	2 (8.7)
Persistent Pain	2 (8.7)
Extra-aortic bleeding	1 (4.3)
Visceral ischemia	2 (8.7)
Total	33 (100)

Sakalihasan N et al; Eur Heart J Cardiovasc Imaging.2015 Jan 14.pii:jeu283.

Results:

Predictive value of false lumen morphology and PET/CT imaging findings at baseline for the occurrence of secondary complications (N=23)

Variable		No complication N=6	Complication N=17	P-value
False lumen	Patent	5 (50)	5 (50)	0.0046
	Partial thrombosis	0 (0)	12 (100)	
	Complete thrombosis	1 (100)	0 (0)	
PET/CT	Negative	4 (57)	3 (43)	0.045
	Positive	2 (12)	14 (88)	
SUV_{max}		2.9 ± 0.54	3.5 ± 0.69	0.073
SUV_{RL}		0.72 ± 0.18	0.96 ± 0.30	0.069
Diameter (mm)		35.7 ± 10.1	51.3 ± 17.8	0.0095
Aortic dilation	No	5 (62)	3 (38)	0.0086
	Yes	1 (7)	14 (93)	

Sakalihasan N et al; Eur Heart J Cardiovasc Imaging.2015 Jan 14.pii:jeu283.

Results:

Overall associations between aneurysmal progression, false lumen morphology, PET/CT imaging, aortic diameters and biomarkers levels recorded during follow-up of the cohort (N=23)

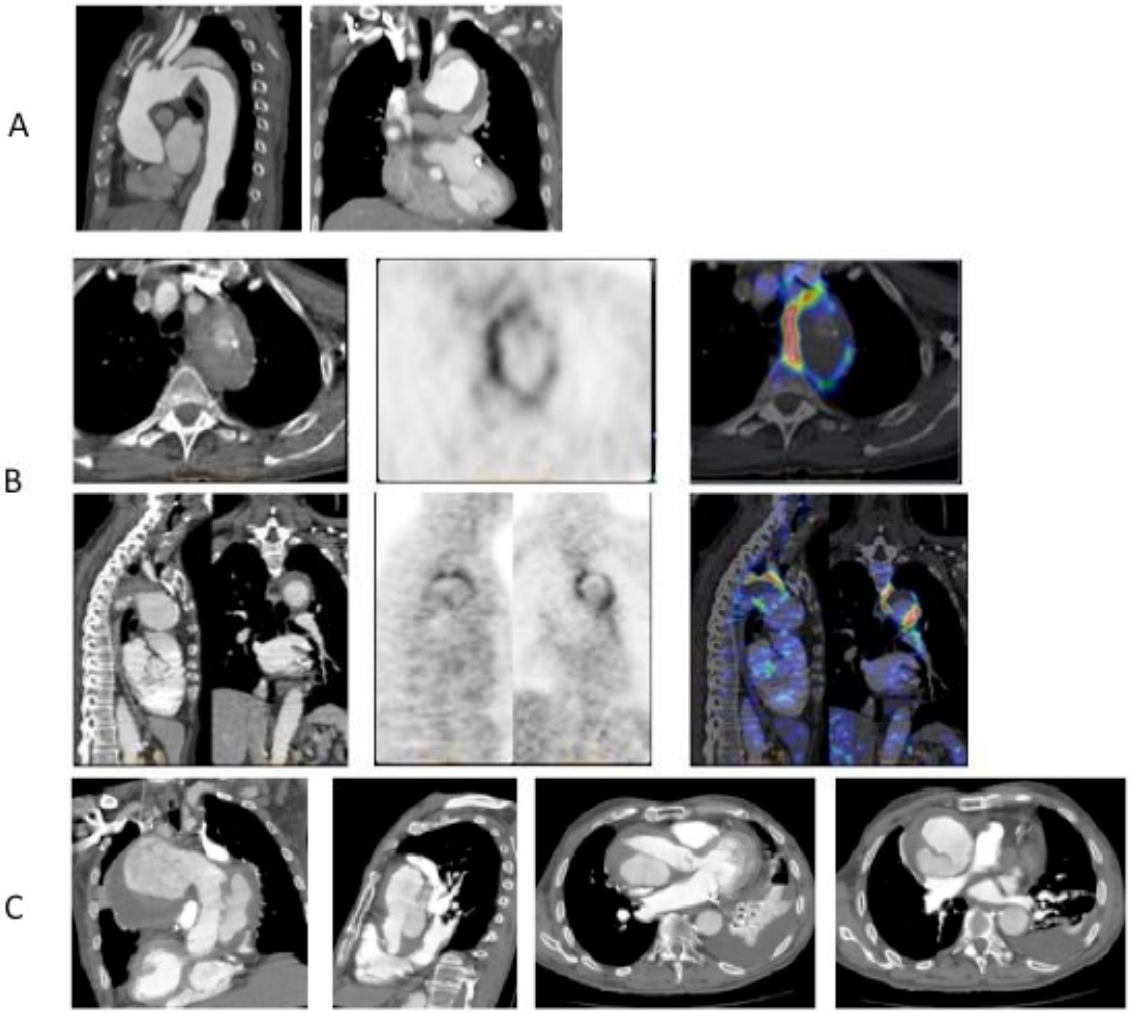
Variable		Aneurysm[a] No N=11	Aneurysm[a] Yes N=12	P-value[b]
False lumen	Patent	9	1	<0.0001
	Partial thrombosis	0	11	
	Complete thrombosis	2	0	
SUV_{max}		2.8 ± 0.5	3.4 ± 0.9	0.0090
SUV_{RL}		0.72 ± 0.15	0.95 ± 0.30	0.0029
Aortic diameter (mm)		36.4 ± 10.4	54.7 ± 12.3	<0.0001
P-selectin (ng/mL)		34.3 ± 16.2	55.1 ± 21.6	0.0009
TAT (ng/mL)		7.5 ± 8.5	18.6 ± 14.8	0.0075
D-dimers (µg/L)		838 ± 563	3117 ± 2048	0.0006
PAP (ng/mL)		259 ± 129	934 ± 1026	<0.0001
MPO (ng/mL)		45.2 ± 44.7	32.0 ± 25.8	0.27
Elastase (ng/mL)		84.4 ± 39.1	91.5 ± 46.4	0.54
MMP9 (ng/mL)		7.9 ± 4.1	5.5 ± 5.1	0.014
Ferritin (ng/mL)		222 ± 113	354 ± 350	0.64
Transferrin (ng/mL)		2.5 ± 0.38	2.5 ± 0.55	0.85

[a] In each group, mean and SD values were calculated over all observations recorded during follow-up of the 23 cohort patients (55 PET/CTs and 52 biological samples)

[b] For comparing the two groups, P-values were obtained from the general linear mixed model (GLMM) which accounts for repeated values within patients?

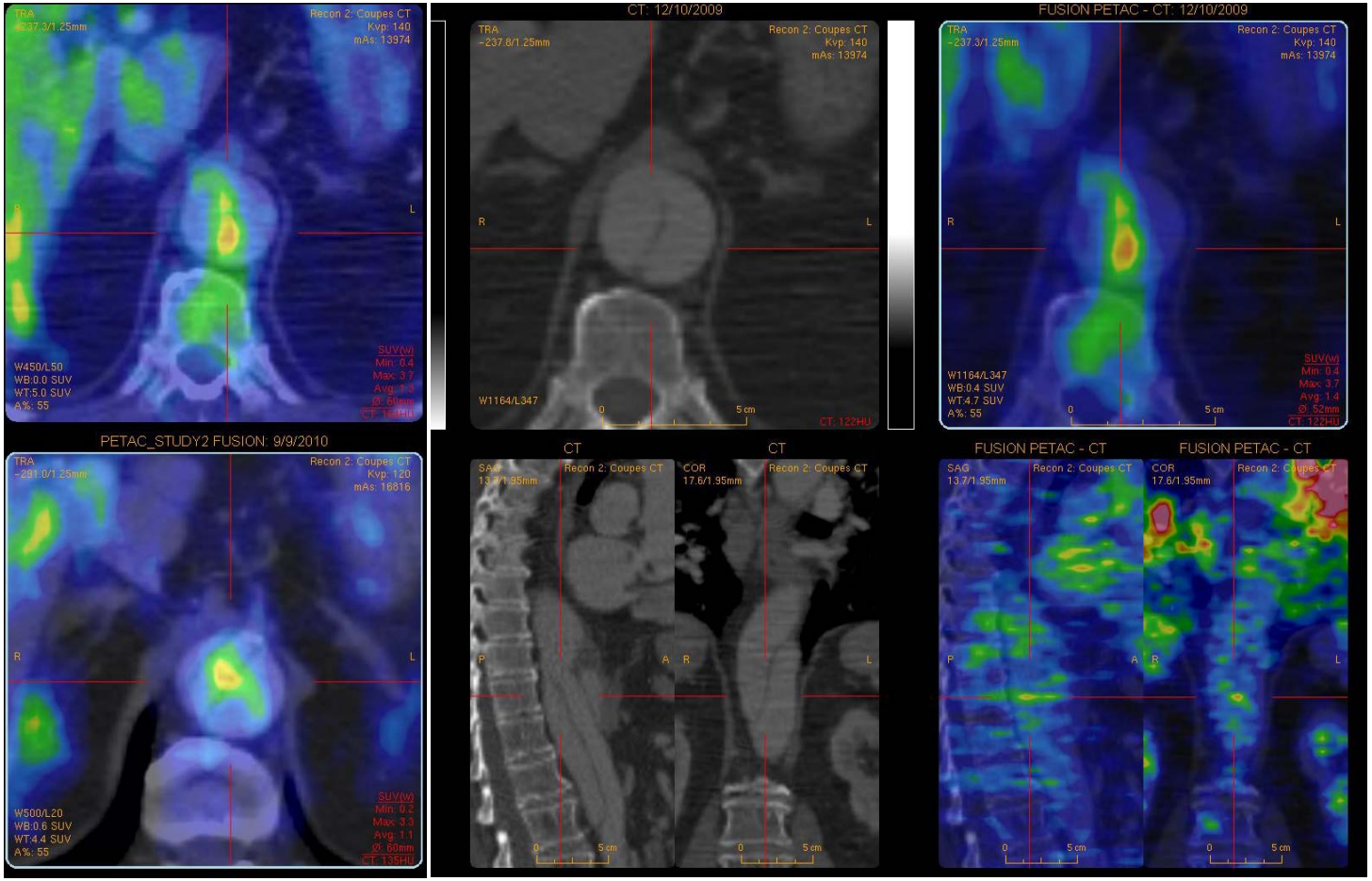
Sakalihasan N et al; Eur Heart J Cardiovasc Imaging.2015 Jan 14.pii:jeu283.

Outcome of the type B dissection (with retrograde dissection)

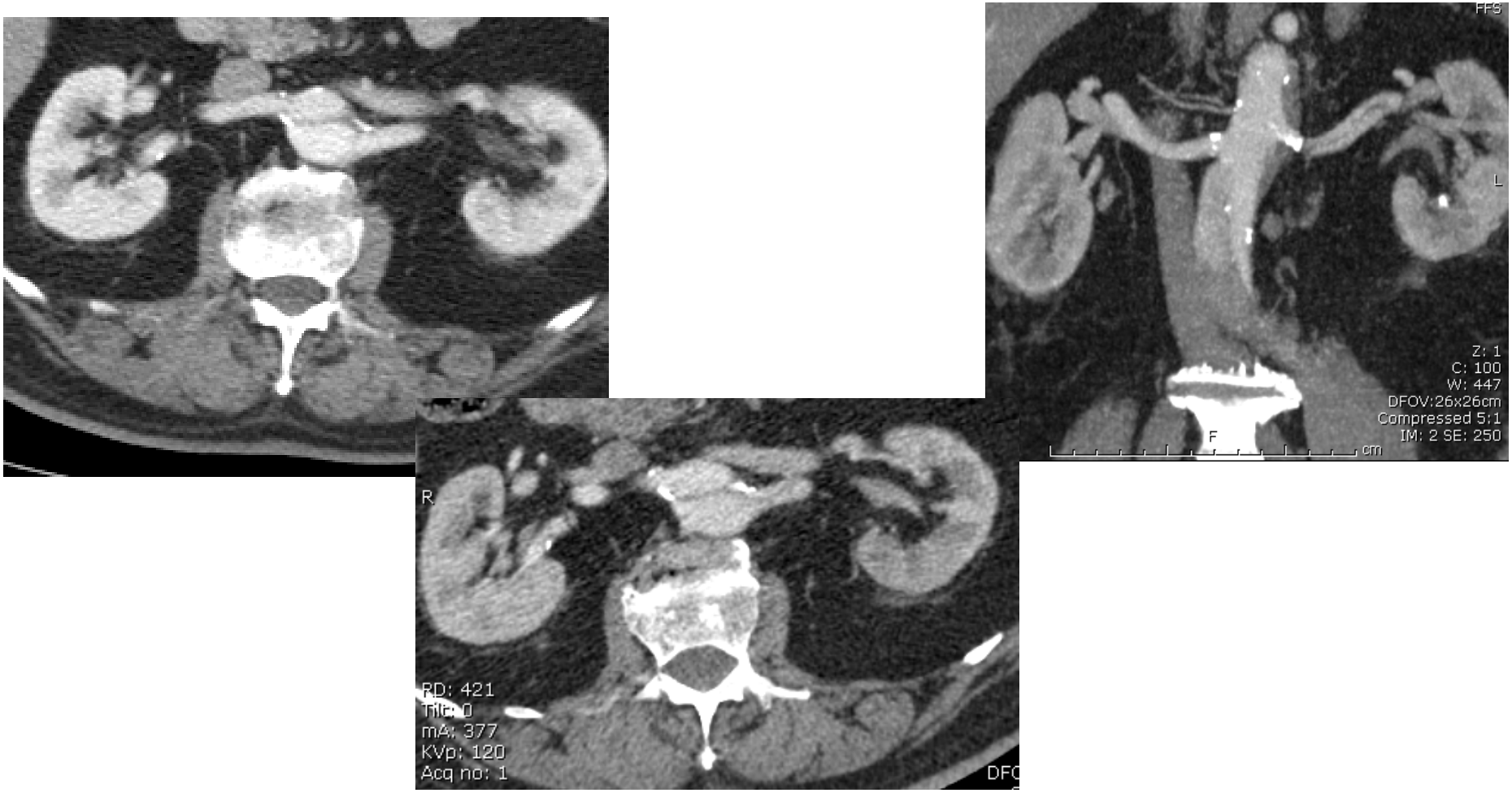


Sakalihasan N et al; Eur Heart J Cardiovasc Imaging.2015 Jan 14.pii:jeu283.

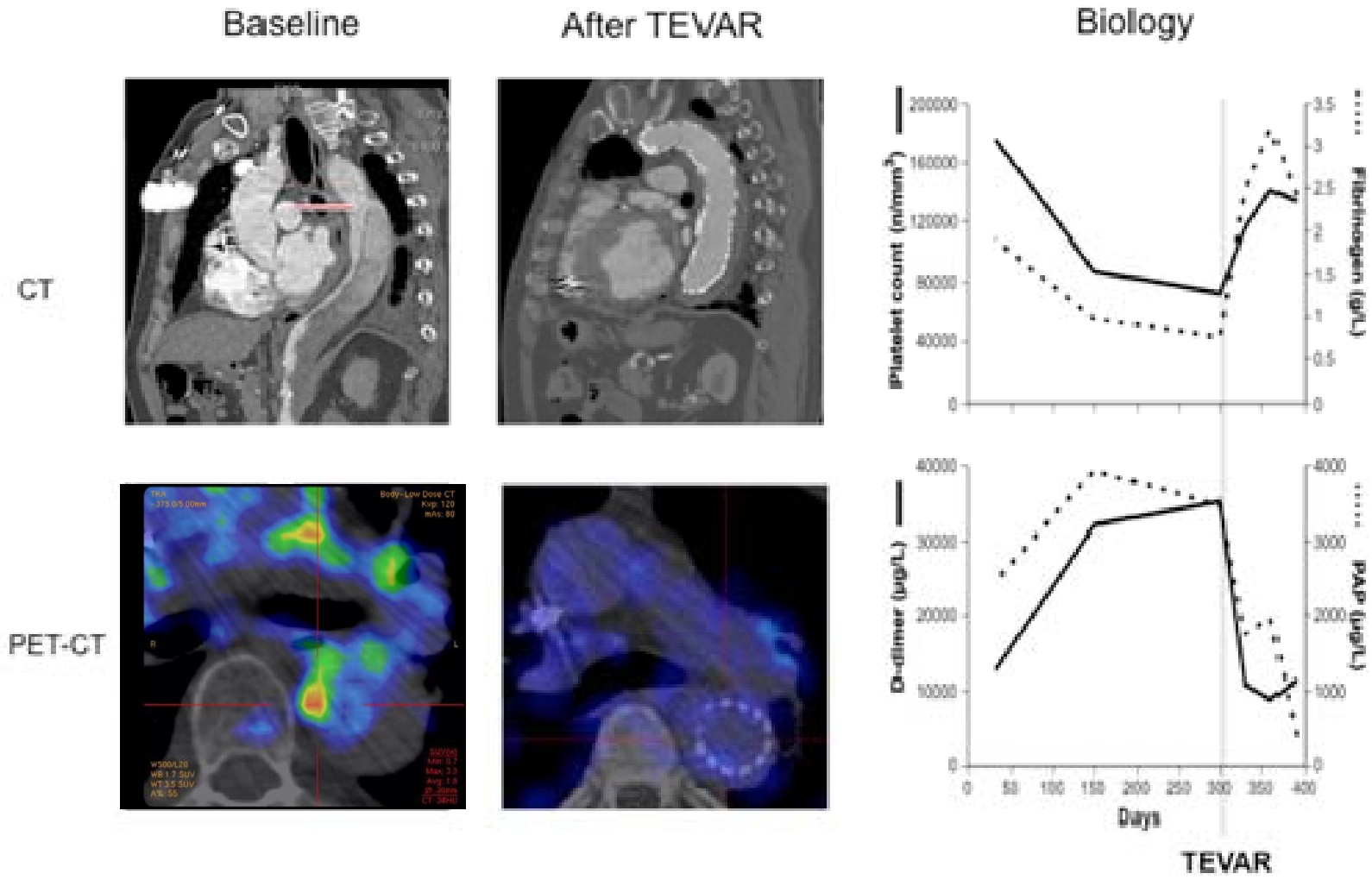
Patent false lumen without thrombus with (+) uptake



Outcome of the type B dissection (with progression of dissection)

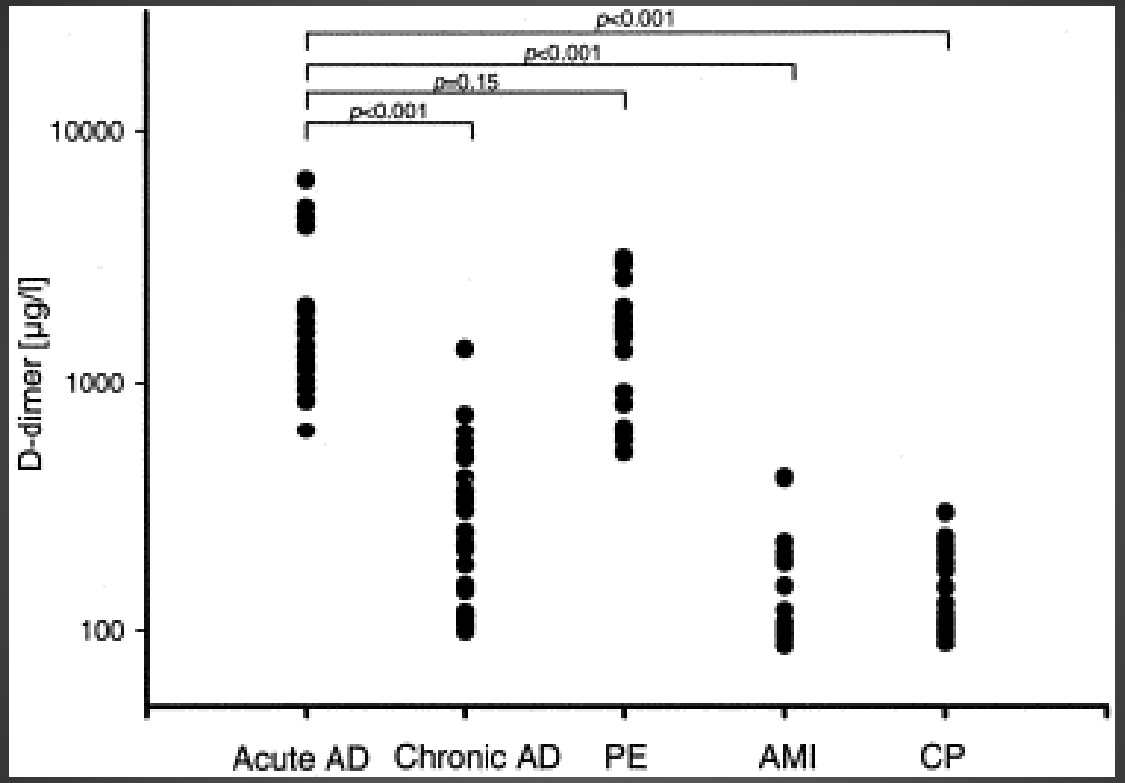


Outcome of the type B dissection (with monofocal 18F-FDG uptake after TEVAR)



Sakalihasan N et al; Eur Heart J Cardiovasc Imaging.2015 Jan 14.pii:jeu283.

Diagnostic and prognostic value of circulating D-Dimers in patients with acute aortic dissection.



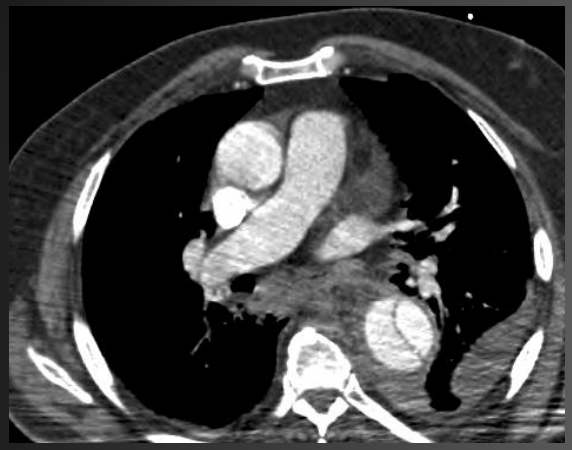
Comparison of D-dimer values between the different patient groups (p values adjusted according to Bonferoni).
AD = aortic dissection; AMI = acute myocardial infarction; CP = chest pain; PE = pulmonary embolism.

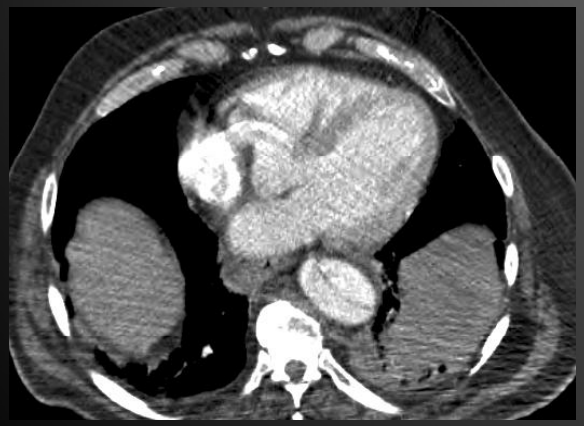
Ohlmann, Patrick et al;Critical Care Medicine. 34(5):1358-1364, May 2006.

Prognosis of the Type B aortic dissection

Admission

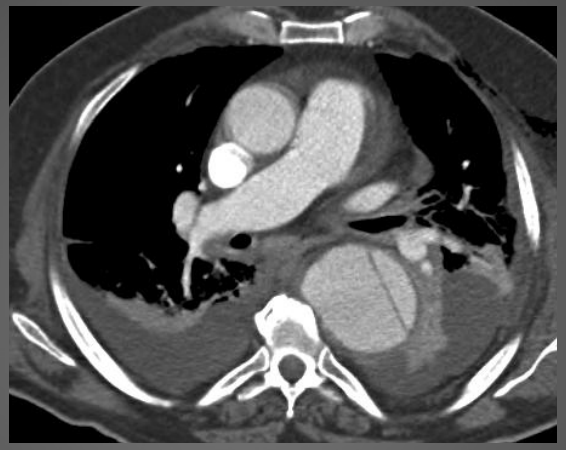
CT

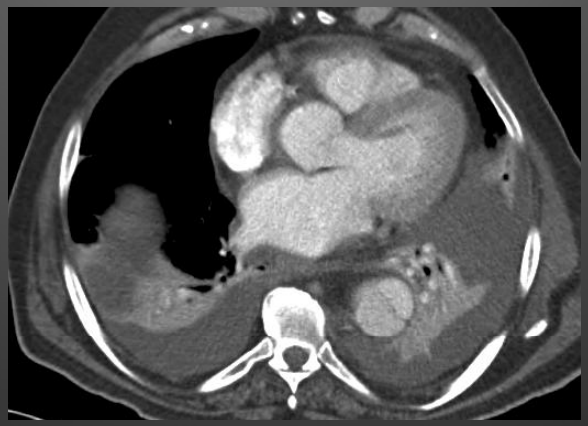




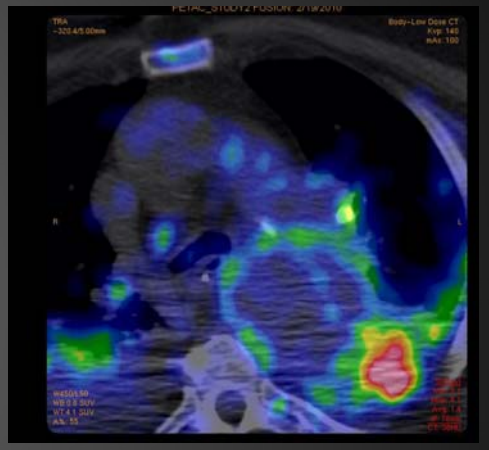
1 week later

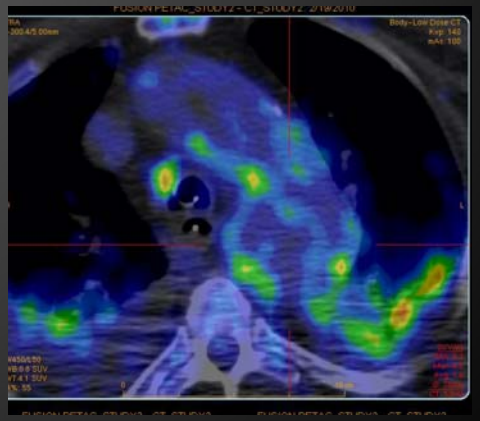
CT



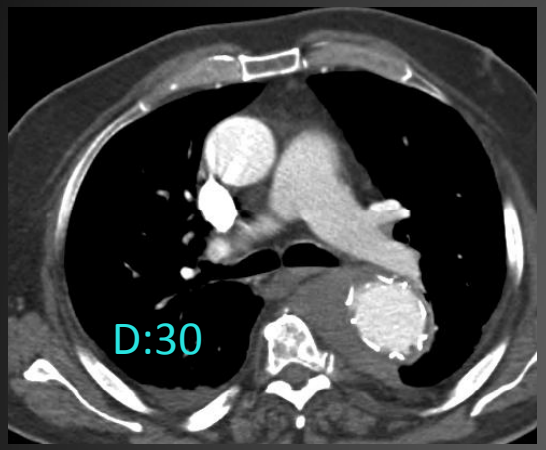


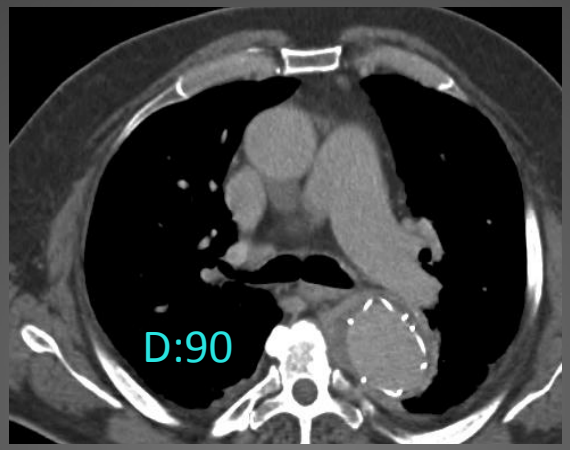
PET

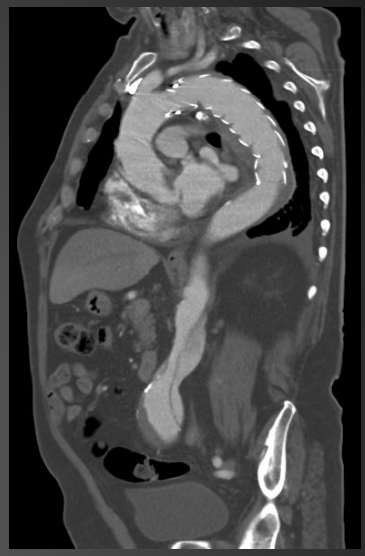


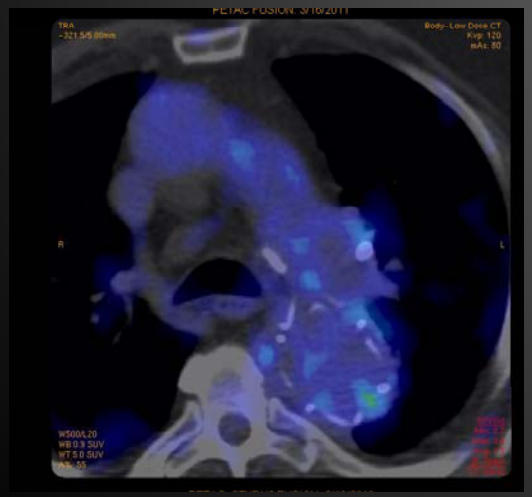


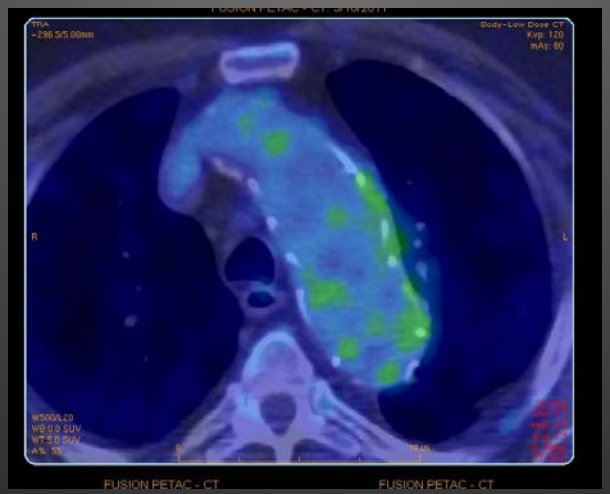
Outcome of the type B dissection after TEVAR

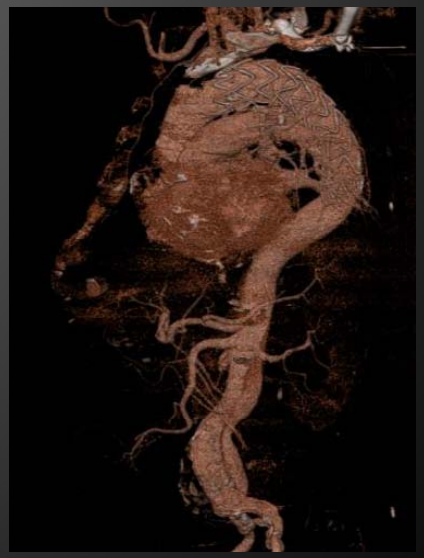




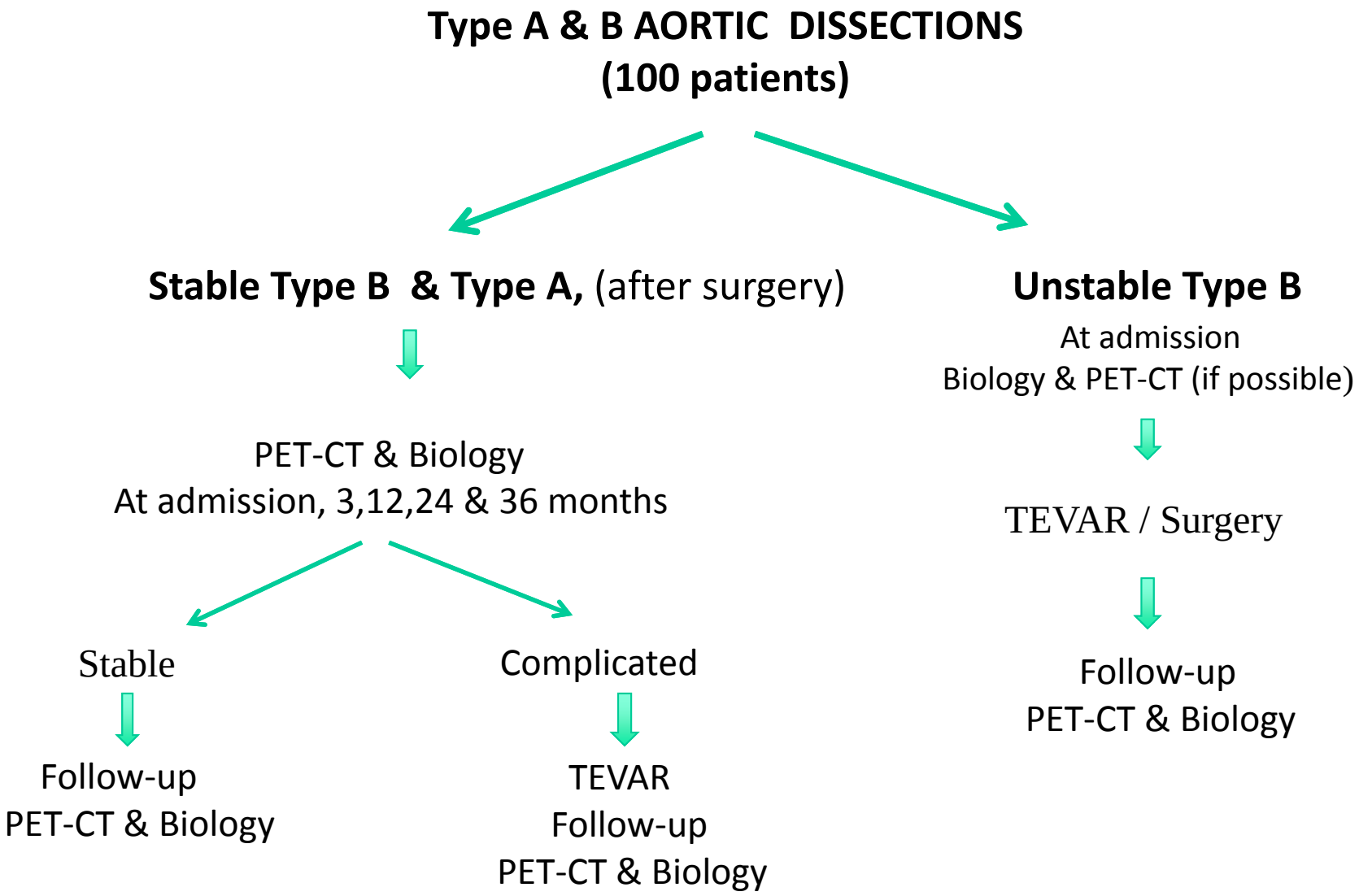


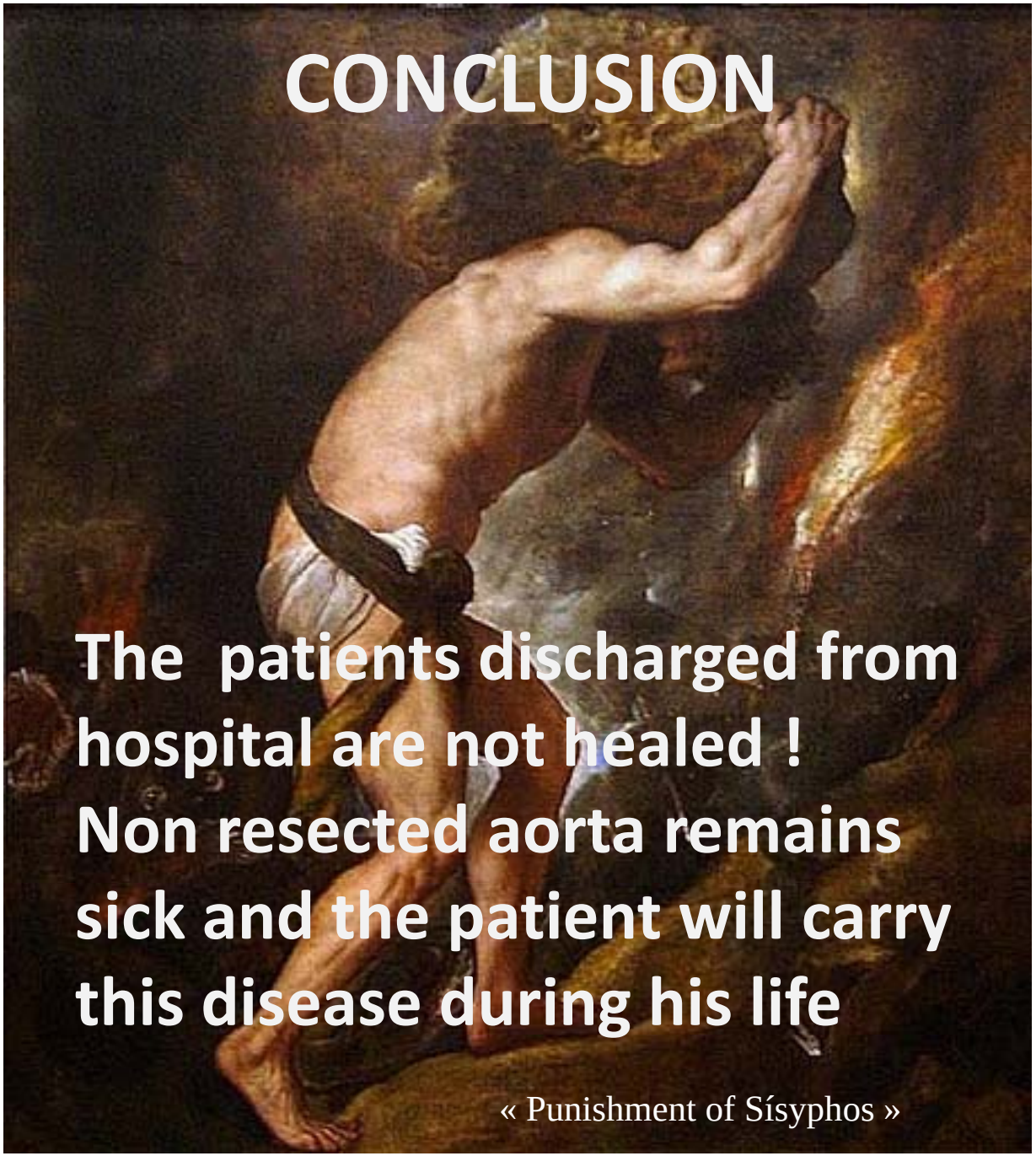






Ongoing FAD & LIDIA studies
(Fighting Aneurysmal Diseases) & (Liege study on Dissected Aorta)




CONCLUSION
The patients discharged from hospital are not healed !
Non resected aorta remains sick and the patient will carry this disease during his life
« Punishment of Sísyphos »

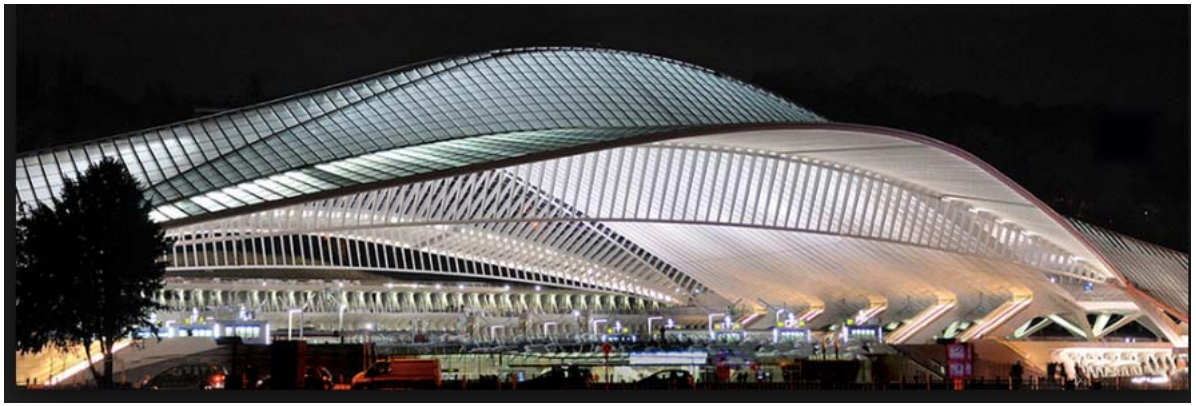
First announcement

5th International Meeting on Aortic Diseases

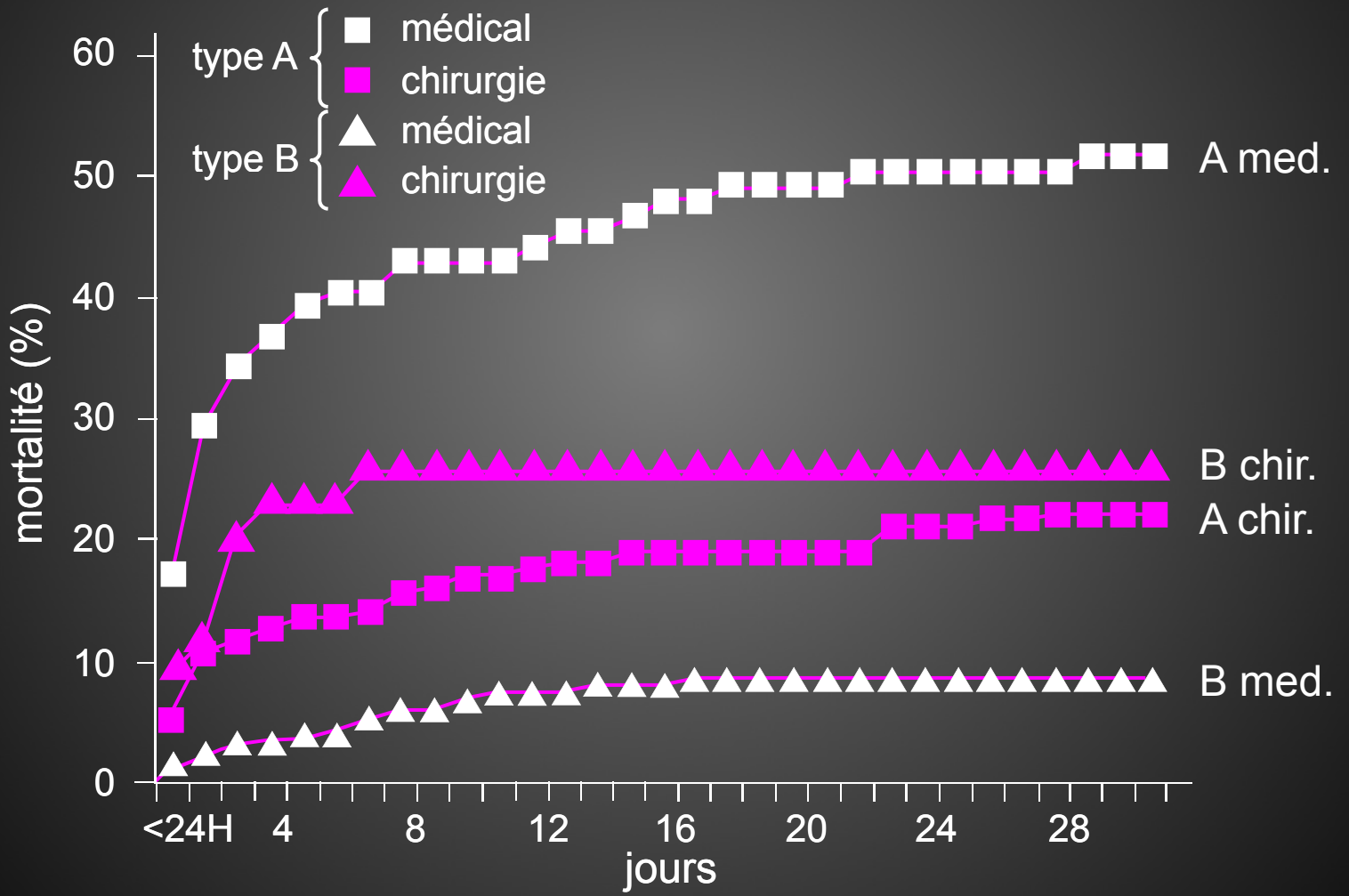
New insights into an old problem
CHU Liège, APF

September 15-17
2016
Crowne Plaza Hotel
Liège, Belgium

www.chuliege-imaa.be



MORTALITE HOSPITALIERE APRES TRAITEMENT



Nienaber CA, Eagle KA, Circulation 2003

Visual Analyses at Segment Level and FDG uptake quantification,Standardized Uptake Values (SUV)

Monofocal,bifocal uptake

$$SUV_{BW} = \frac{tissue\ concentration\ (MBq/mL)}{injected\ dose\ (MBq)/\ patient\ body\ weight\ (g)}$$

$$SUV_{BSA} = \frac{tissue\ \ concentration\ \ (MBq/mL)}{injected\ \ dose\ \ (MBq)/BSA\ \ (m^2)}$$

$$with\ \ BSA = 0.007184 \times body\ \ weight^{0.425} \times body\ \ height^{0.725}$$

Multifocal uptake

$$SUV_{LBM} = \frac{tissue\ \ concentration\ \ (MBq/mL)}{injected\ \ dose\ \ (MBq)/LBM}$$

$$with\ LBM_{women} = (1.07 \times body\ weight) - 148(body\ weight/body\ height)^2$$

$$and\ LBM_{men} = (1.1 \times body\ weight) - 120(body\ weight/body\ height)^2$$

Quantitative and Qualitative Evaluation of Total PET-CT Examinations

	Mean SUV max	Mean SUV liver	Mean Ration SUV max/SUV liver
FDG Uptake + 26 PET-CT	3.40	3.70	0.94
FDG Uptake - 26 PET-CT	2.72	3.92	0.70
P<	0.00017	0.19	0.00014

Sakalihasan N et al; Eur Heart J Cardiovasc Imaging.2015 Jan 14.pii:jeu283.

Conclusion

Our translational clinical investigation demonstrates that, PET-CT has potential to predict the outcome of patients with « chronic » asymptomatic type B dissection.

PET/CT can be instrumental in therapeutic decision-making, given that it predicts the complications after acute phase of dissection.

Nevertheless, further largest prospective studies are needed.