Tractament quirúrgic de l'aorta ascendent: quan i com?

De la fisiopatologia al tractament quirúrgic

Josep Maria Alegret

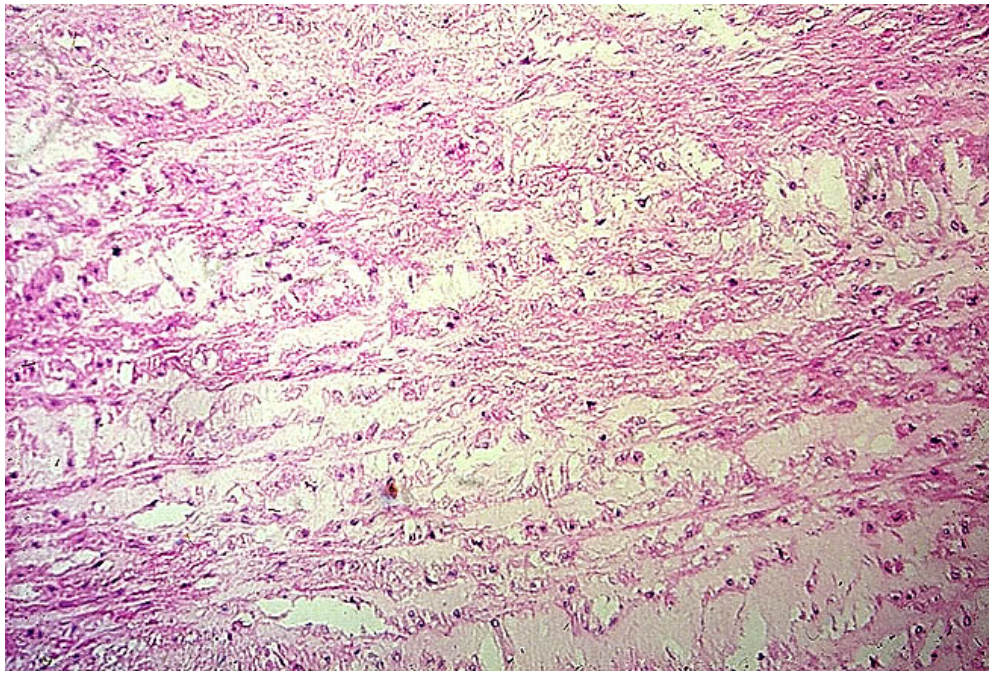
Hospital Universitari de Sant Joan de Reus, IISPV

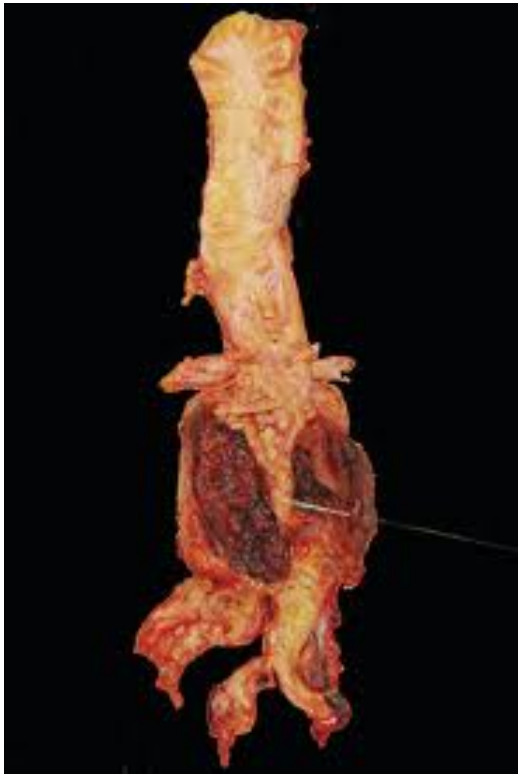
Imatge Cardíaca Corporació CRC

- Fisiopatologia dels aneurismes de l'Aorta toràcica (AAT) (ascendent)
- Tipus d'AAT
- Tractament mèdic
- Indicació quirúrgica

Fisiopatologia dels AAT

Aneurisma Ao toràcica vs Abdominal: Diferent causa, diferent histopatologia



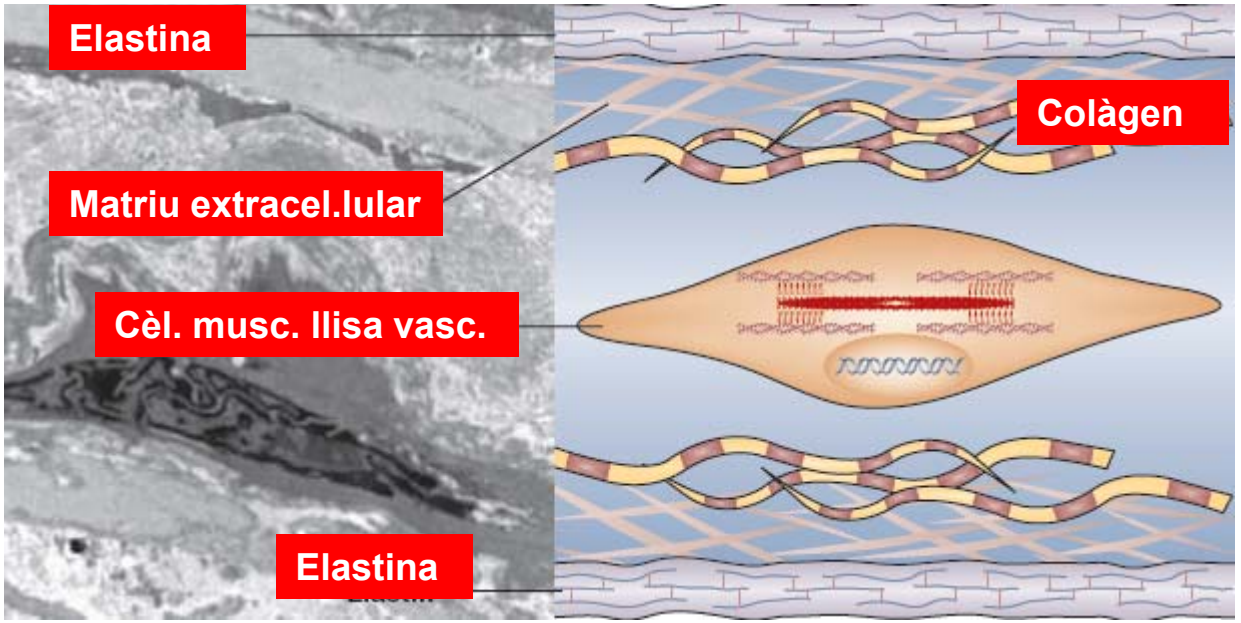


AAT *Degeneratiu*

AAA *Ateroscleròtic*

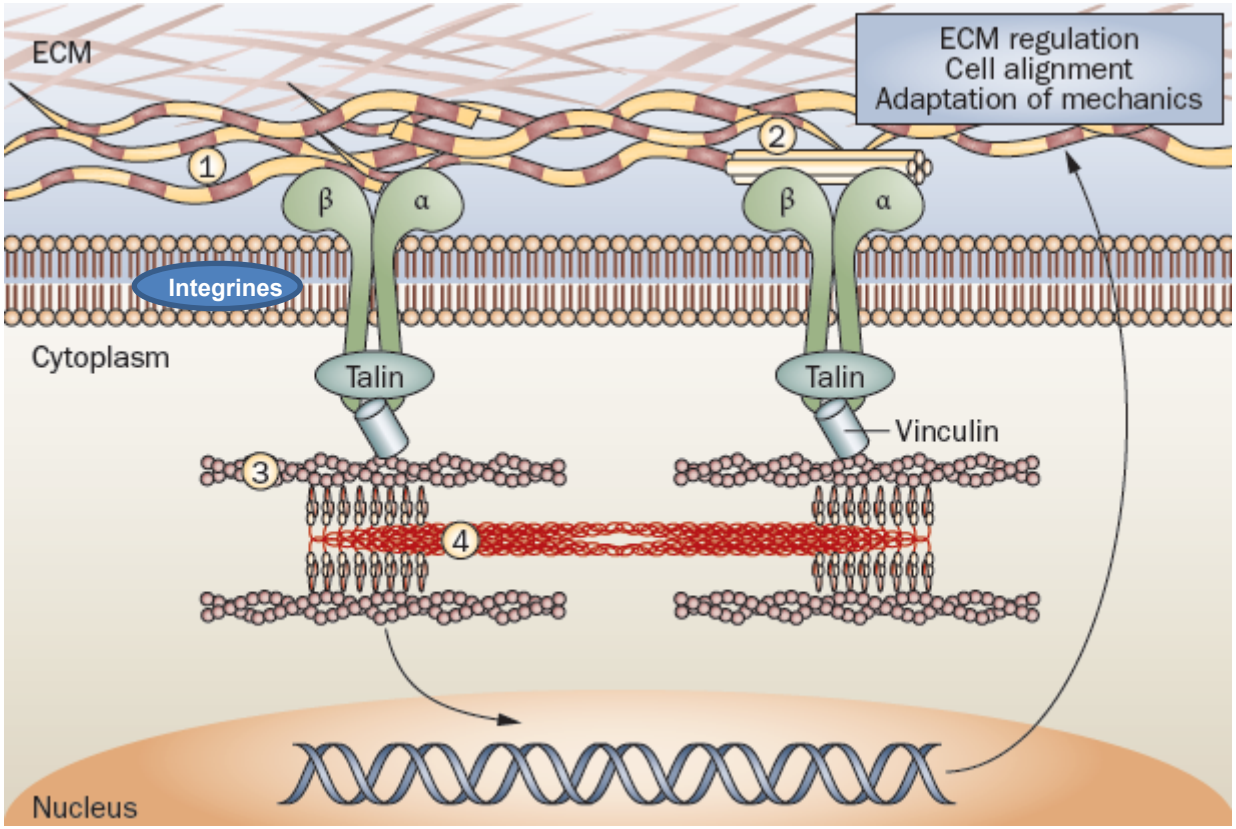
Hospital Universitari de Sant Joan Reus

Hospital Quirón Barcelona diagnòstic per la imatge

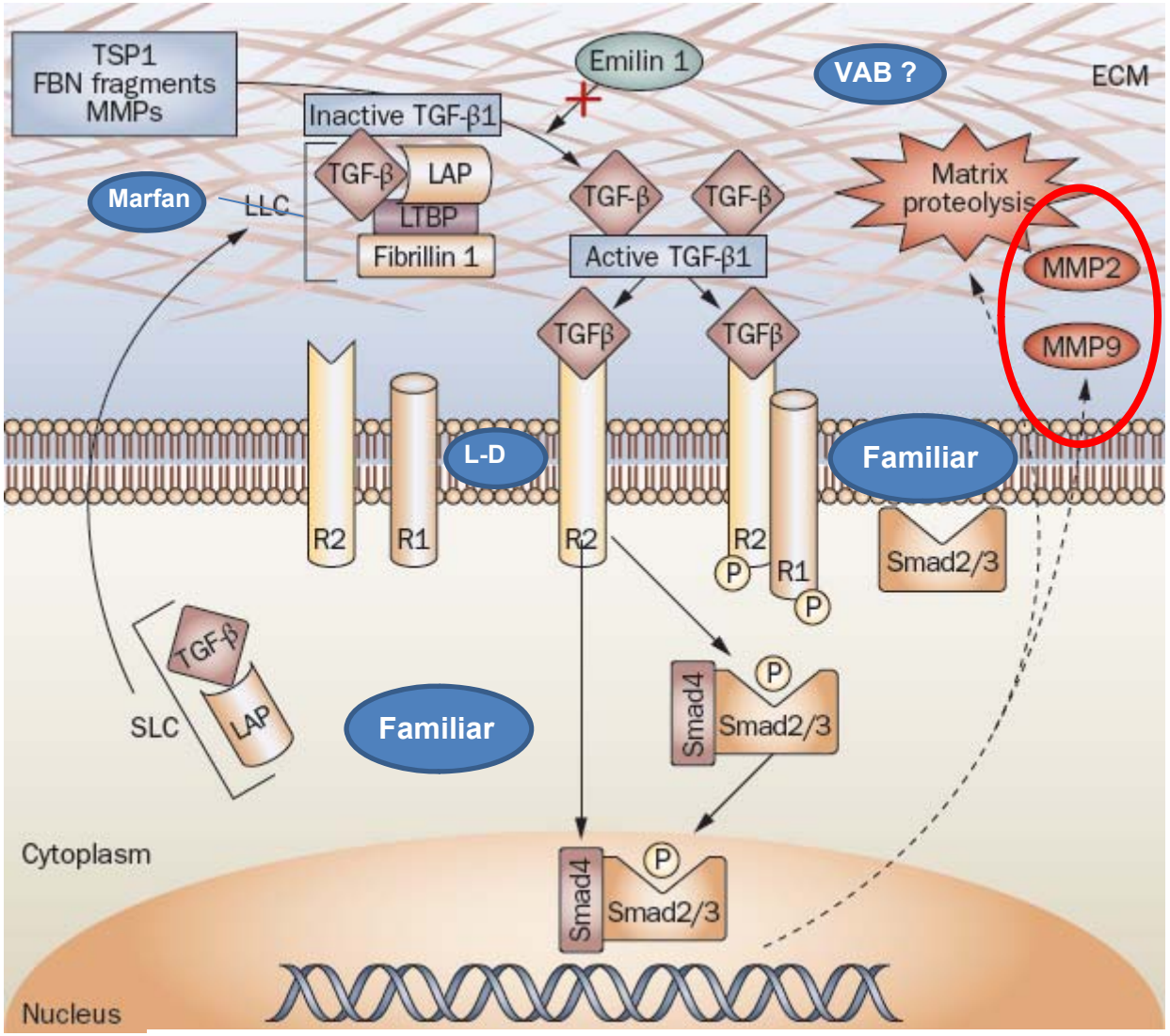


Unitat Lamelar

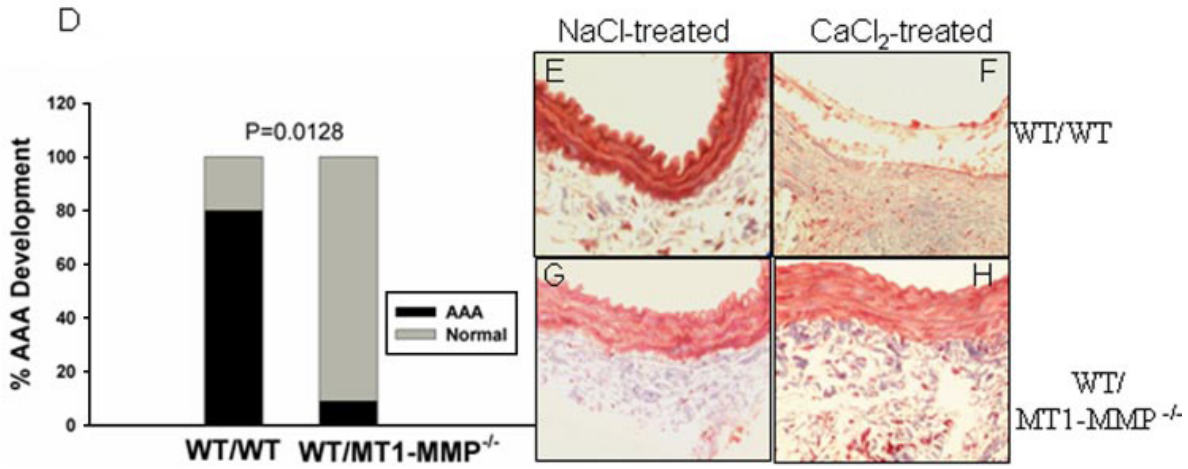
Mecanotransducció:
Interacció entre Matriu EC-receptors de membrana-citoesquelet

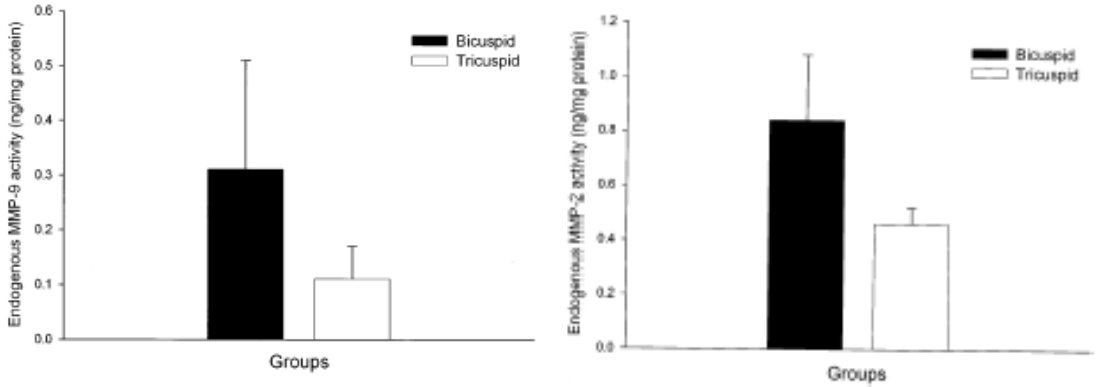


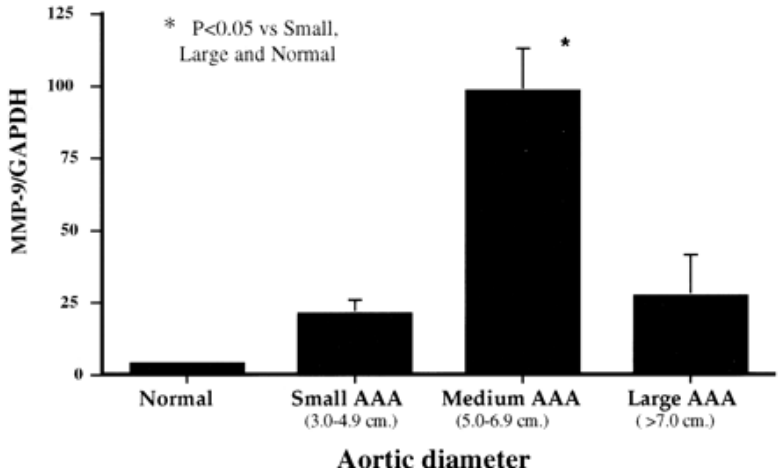
Paper clau de *TGF-β1* en la formació dels AAT



El-Hamamsy, I. & Yacoub, M. H. *Nat. Rev. Cardiol.* 6, 771–786 (2009)







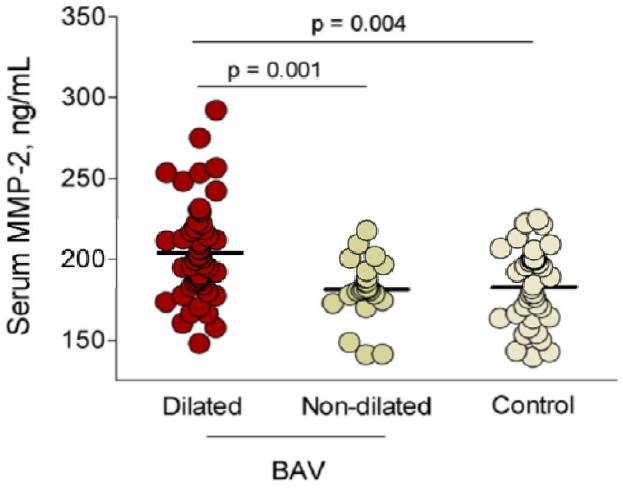
Major expressió de diverses **MMP** als AAT (MT1, **2**, 7, **9**, 13…)

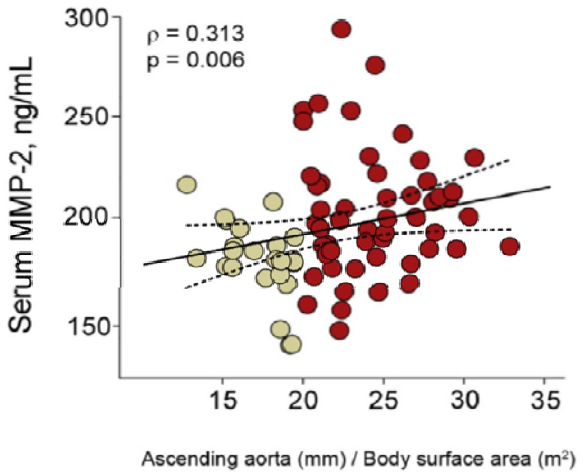
Heterogenicitat: diversos mecanismes d'activació i MMP implicades

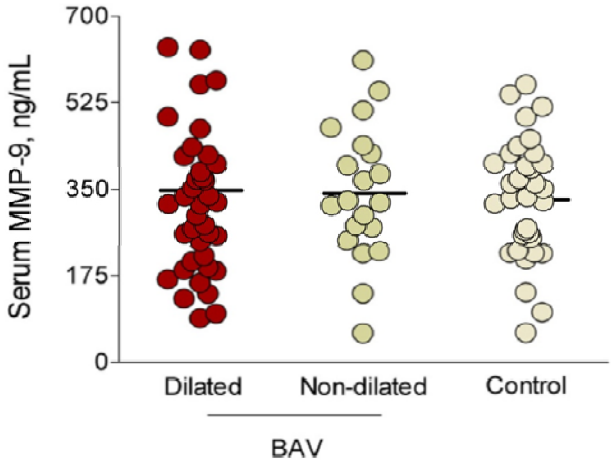
Boyurn J. *J Thor Cardiovasc Surg* 2004, Ikonomidis JS, *Thor Cardiovasc Surg* 2007

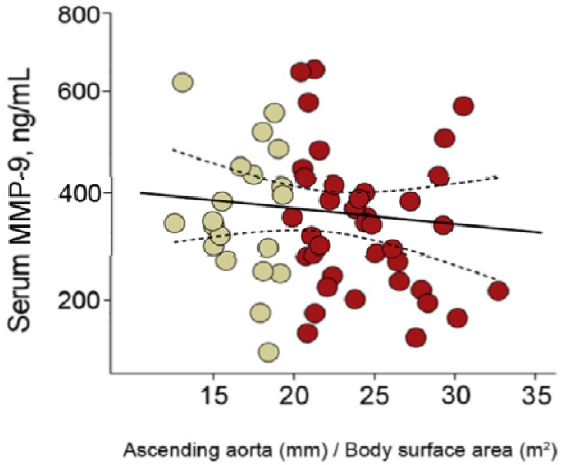
…..

MMP també identificables en sang perifèrica:
Majors nivells circulants de MMP-2 a la dilatació Ao ascendent per VAB



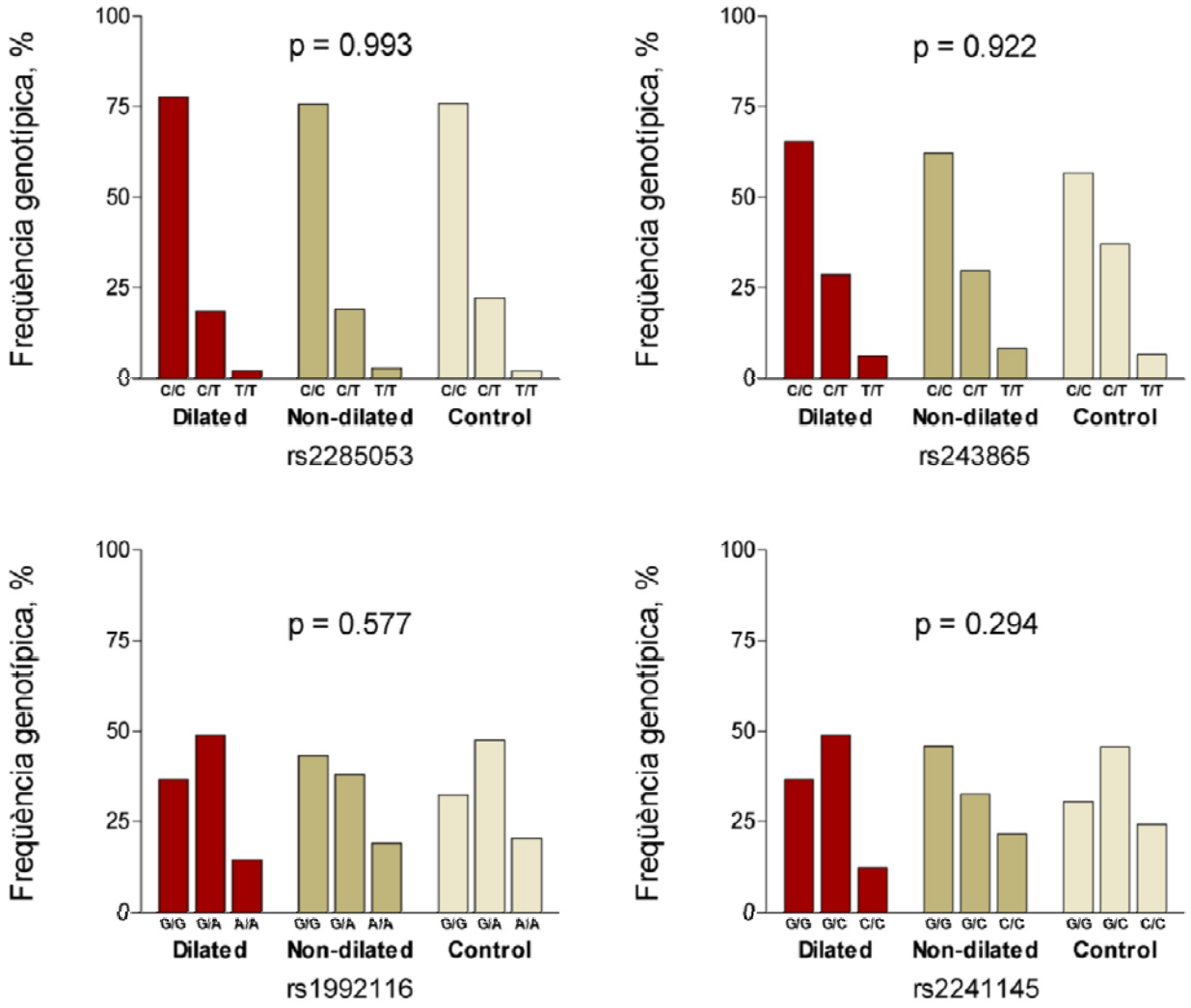




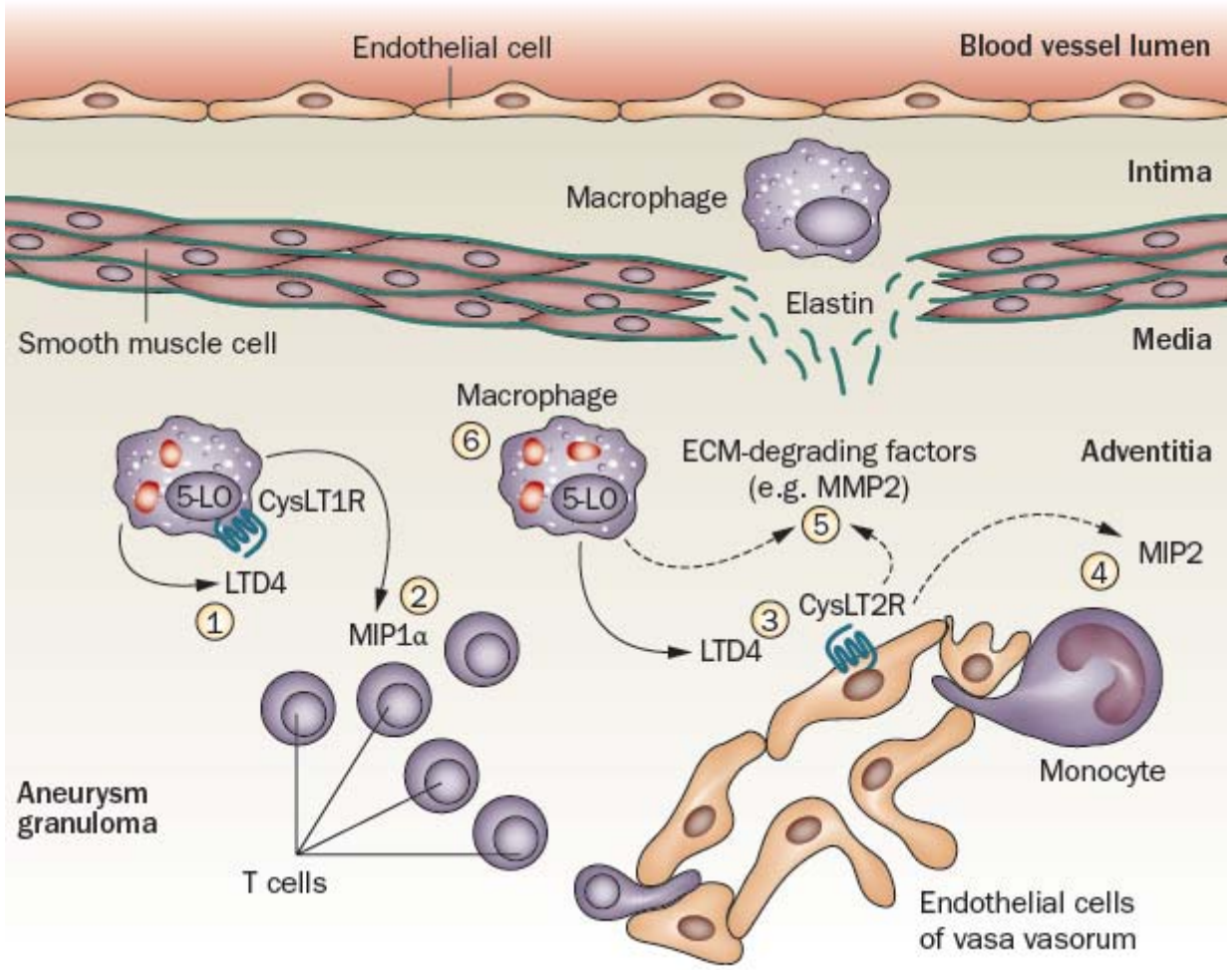


Alegret JM et al. Rev Esp Cardiol. Abstr. Supl. 2011

…sense que sigui degut a polimorfismes amb > expressió de MMP-2



Alegret JM et al. Rev Esp Cardiol. Abstr. Supl. 2011



Paper de la Inflamació ?

Zhao et al, Nat. Med 2004

Circulation. 2011 Sep 13;124(11 Suppl):S168-73.

Pravastatin reduces Marfan aortic dilation.

McLoughlin D, McGuinness J, Byrne J, Terzo E, Huuskonen V, McAllister H, Black A, Kearney S, Kay E, Hill AD, Dietz HC, Redmond JM.

Department of Surgery, Royal College of Surgeons in Ireland, Dublin, Ireland.

Abstract

BACKGROUND: The sequelae of aortic root dilation are the lethal consequences of Marfan syndrome. The root dilation is attributable to an imbalance between deposition of matrix elements and metalloproteinases in the aortic medial layer as a result of excessive transforming growth factor-beta signaling. This study examined the efficacy and mechanism of statins in attenuating aortic root dilation in Marfan syndrome and compared effects to the other main proposed preventative agent, losartan.

METHODS AND RESULTS: Marfan mice heterozygous for a mutant allele encoding a cysteine substitution in fibrillin-1 (C1039G) were treated daily from 6 weeks old with pravastatin 0.5 g/L or losartan 0.6 g/L. The end points of aortic root diameter (n=25), aortic thickness, and architecture (n=10), elastin volume (n=5), dp/dtmax (maximal rate of change of pressure) (cardiac catheter; n=20), and ultrastructural analysis with stereology (electron microscopy; n=5) were examined. The aortic root diameters of untreated Marfan mice were significantly increased in comparison to normal mice (0.161 ± 0.001 cm vs 0.252 ± 0.004 cm; P<0.01). Pravastatin (0.22 ± 0.003 cm; P<0.01) and losartan (0.221 ± 0.004 cm; P<0.01) produced a significant reduction in aortic root dilation. Both drugs also preserved elastin volume within the medial layer (pravastatin 0.23 ± 0.02 and losartan 0.29 ± 0.03 vs untreated Marfan 0.19 ± 0.02; P=0.01; normal mice 0.27 ± 0.02). Ultrastructural analysis showed a reduction of rough endoplasmic reticulum in smooth muscle cells with pravastatin (0.022 ± 0.004) and losartan (0.013 ± 0.001) compared to untreated Marfan mice (0.035 ± 0.004; P<0.01).

CONCLUSIONS: Statins are similar to losartan in attenuating aortic root dilation in a mouse model of Marfan syndrome. They appear to act through reducing the excessive protein manufacture by vascular smooth muscle cells, which occurs in the Marfan aorta. As a drug that is relatively well-tolerated for long-term use, it may be useful clinically.

PMID: 21911808 [PubMed - indexed for MEDLINE]

Am J Cardiol. 2011 Nov 15;108(10):1458-62.

Comparison of ascending aortic size in patients with severe bicuspid aortic valve stenosis treated with versus without a statin drug.

Goel SS, Tuzcu EM, Agarwal S, Aksoy O, Krishnaswamy A, Griffin BP, Svensson LG, Kapadia SR.

Department of Cardiovascular Medicine, Cleveland Clinic Foundation, Cleveland, Ohio.

Abstract

Ascending aortic dilation commonly occurs in patients with bicuspid aortic valve (BAV). Statins have been shown to reduce the expression of matrix metalloproteinases and slow the progression of abdominal aortic aneurysms. The role of statins in slowing ascending aortic dilation in patients with BAV is unknown. We sought to compare the ascending aortic dimensions in patients with BAV stenosis treated with versus without a statin. From our catheterization laboratory database, all patients undergoing preoperative coronary angiography before aortic valve with or without ascending aorta replacement for bicuspid aortic stenosis (AS) from 2004 to 2007 were identified. The ascending aortic size was measured on their preoperative transesophageal echocardiogram. Data on statin use were obtained from chart review, and the ascending aortic size was compared between patients taking and not taking a statin. The study sample included 147 patients, of whom 76 were treated with statins (mean age 62 ± 9 years, 72% men) and 71 were not (mean age 59 ± 12 years, 68% men). The total and low-density lipoprotein cholesterol and triglyceride levels were significantly lower in the statin group. The ascending aorta size was significantly lower in the statin subgroup of the pure severe AS group (3.6 ± 0.7 cm vs 3.9 ± 0.6 cm, p <0.01) but not in the mixed severe AS and severe aortic regurgitation group. In the pure severe AS group, significantly fewer patients taking a statin had an ascending aorta ≥4 cm (29% vs 52%, p <0.02). On multivariate analysis, statin use was the only independent predictor of aortic size and was associated with a 0.33-cm reduction in aortic size (95% confidence interval 0.06 to 0.59, p <0.01). In conclusion, patients with statin-treated BAV stenosis have a smaller ascending aortic size than patients with BAV untreated with statins.

PMID: 22040719 [PubMed - in process]

Dilated aortic root is related to a global aortic dilating diathesis

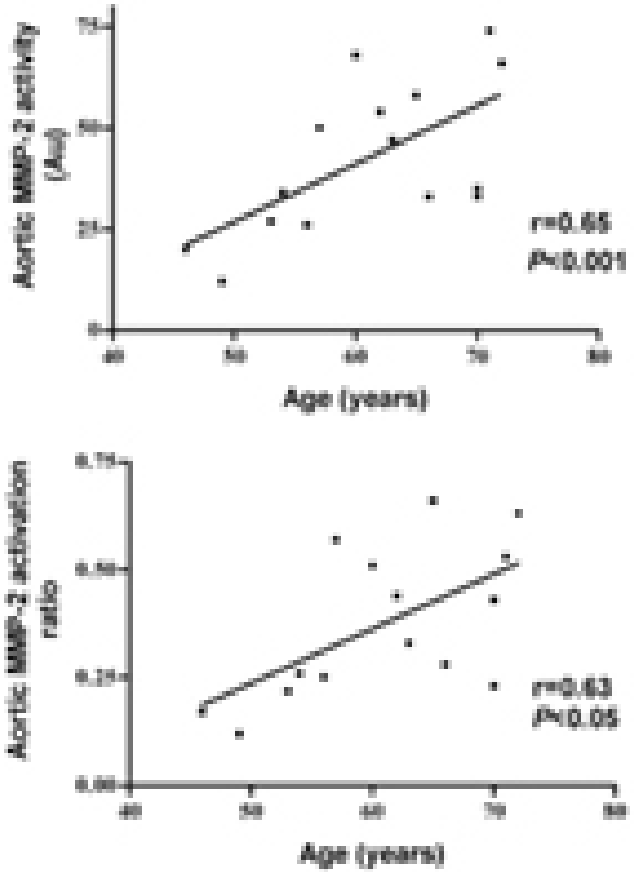
Josep M. Alegret, PhD,[a,b,d] Nahum Calvo, MD,[b] Carme Ligero, MD,[a] Raquel Palomares, MD,[a] Lidón Millá, MD,[b] Vicente Martín-Paredero, PhD,[c] Manuel Montero, MD,[b] and Jorge Joven, PhD,[d] *Reus and Tarragona, Spain*

Objective: The purpose of this study was to analyze the association between the dilatation of the aortic root and the diameters of the rest of the aorta and to identify some related factors that could be used to identify patients at higher risk of presenting with an aortic aneurysm.

Methods: In 71 consecutive patients with a dilated aortic root identified by transthoracic echocardiography, prospective helical computed tomography was performed. Aortic diameters were measured perpendicular to the flow at seven levels in the thoracic and abdominal aorta.

Results: Ascending aorta diameter showed a moderate correlation with aortic indexed diameters at the thoracic and abdominal level in tricuspid aortic valve patients (r ranging from 0.37-0.56), whereas in patients with a bicuspid aortic valve, a moderate correlation between the ascending aorta diameters and the thoracic descending aorta diameters was observed (r 0.51-0.53). In a multivariate analysis, age was independently related to indexed diameter at all aortic sites (β ranging from 0.06-0.12 per year), whereas aortic regurgitation was independently related only to thoracic aorta diameter (β ranging from 1.17-1.84). Age ($P < .0001$), body surface area ($P < .0001$), and grade of aortic valve regurgitation ($P = .001$) independently predicted aortic volume.

Conclusion: Different patterns of aortic diameters were observed in patients with dilated aortic root, depending on age, aortic valve morphology, and function. When a dilated aortic root is detected in older patients with a tricuspid aortic valve, an accurate cardiovascular survey that includes the entire aorta is needed. These results provide further evidence about the systemic nature of aortic dilatation. (J Vasc Surg 2010;52:867-72.)



L'envelliment s'associa a un increment de l'activitat de MMP-2 a l'aorta humana

McNulty M, Am J Hyper 2005

Circ Res. 2011 Oct 28;109(10):1115-9. Epub 2011 Sep 8.

MicroRNA-29 in Aortic Dilation: Implications for Aneurysm Formation.

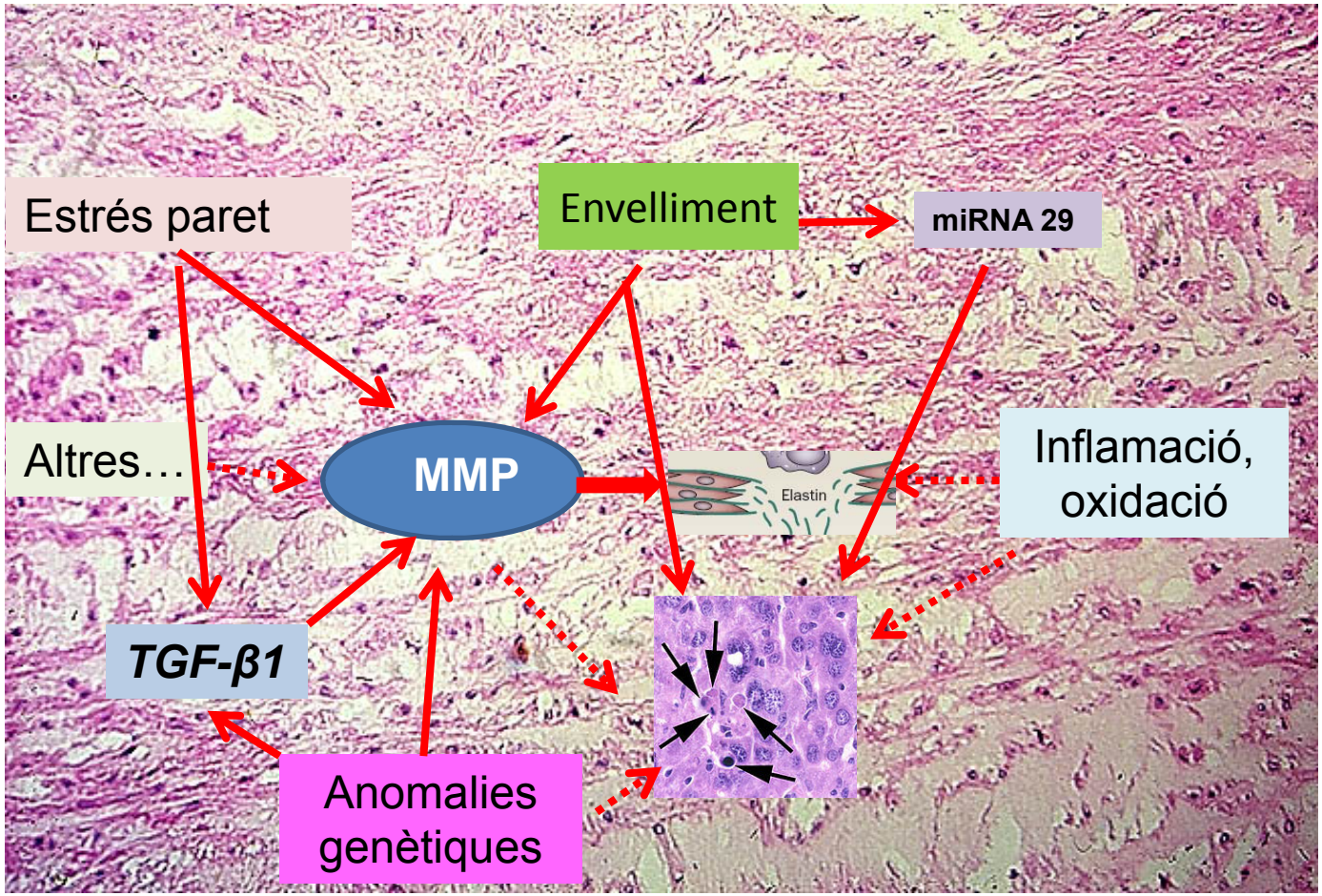
Boon RA, Seeger T, Heydt S, Fischer A, Hergenreider E, Horrevoets AJ, Vinciquerra M, Rosenthal N, Sciacca S, Pilato M, van Heijningen P, Essers J, Brandes RP, Zeiher AM, Dimmeler S.

Institute for Cardiovascular Regeneration, Centre for Molecular Medicine, Goethe University, Theodor Stern-Kai 7, 60590 Frankfurt, Germany. dimmeler@em.uni-frankfurt.de.

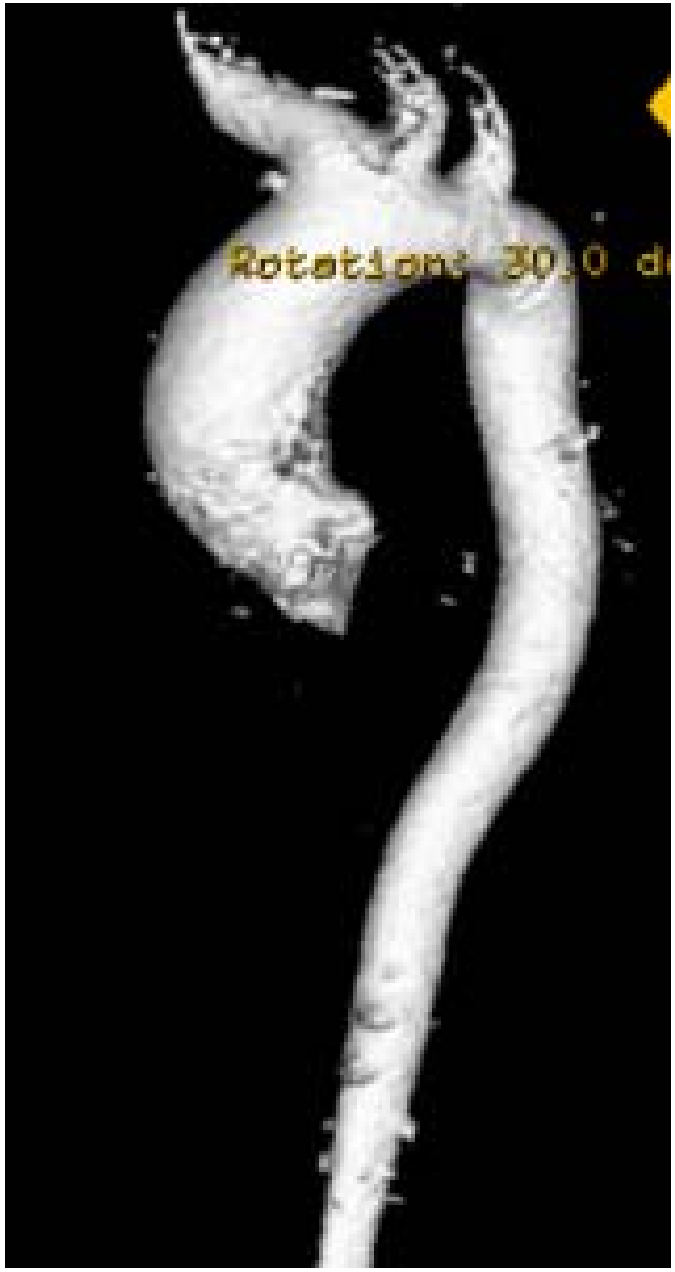
Abstract

Rationale: Aging represents a major risk factor for coronary artery disease and aortic aneurysm formation. MicroRNAs (miRs) have emerged as key regulators of biological processes, but their role in age-associated vascular pathologies is unknown. Objective: We aim to identify miRs in the vasculature that are regulated by age and play a role in age-induced vascular pathologies. Methods and Results: Expression profiling of aortic tissue of young versus old mice identified several age-associated miRs. Among the significantly regulated miRs, the increased expression of miR-29 family members was associated with a profound downregulation of numerous extracellular matrix (ECM) components in aortas of aged mice, suggesting that this miR family contributes to ECM loss, thereby sensitizing the aorta for aneurysm formation. Indeed, miR-29 expression was significantly induced in 2 experimental models for aortic dilation: angiotensin II-treated aged mice and genetically induced aneurysms in Fibulin-4(R/R) mice. More importantly, miR-29b levels were profoundly increased in biopsies of human thoracic aneurysms, obtained from patients with either bicuspid (n=79) or tricuspid aortic valves (n=30). Finally, LNA-modified antisense oligonucleotide-mediated silencing of miR-29 induced ECM expression and inhibited angiotensin II-induced dilation of the aorta in mice. Conclusion: In conclusion, miR-29-mediated downregulation of ECM proteins may sensitize the aorta to the formation of aneurysms in advanced age. Inhibition of miR-29 in vivo abrogates aortic dilation in mice, suggesting that miR-29 may represent a novel molecular target to augment matrix synthesis and maintain vascular wall structural integrity.

PMID: 21903938 [PubMed - in process]


Estrés paret
Envelliment
miRNA 29
Altres…
MMP
Elastin
Inflamació, oxidació
TGF-β1
Anomalies genètiques

Tipus d’aneurismes AAT



AAT

Sindròmic
(Marfan, L-D,
E-D, Turner…)

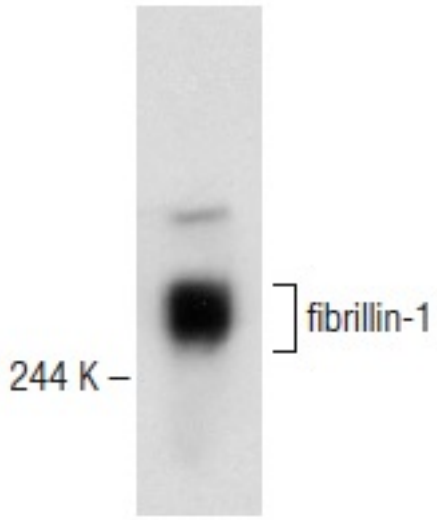
V.aórtica bicúspide

AAT no sindròmic

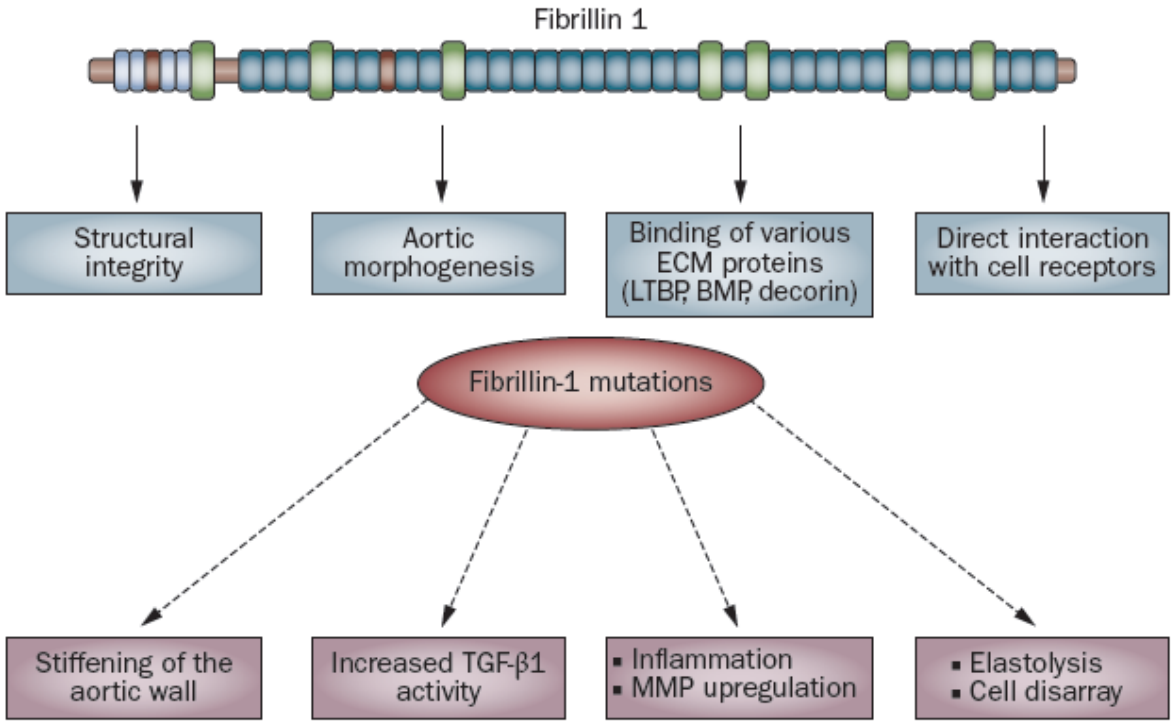
Familiar

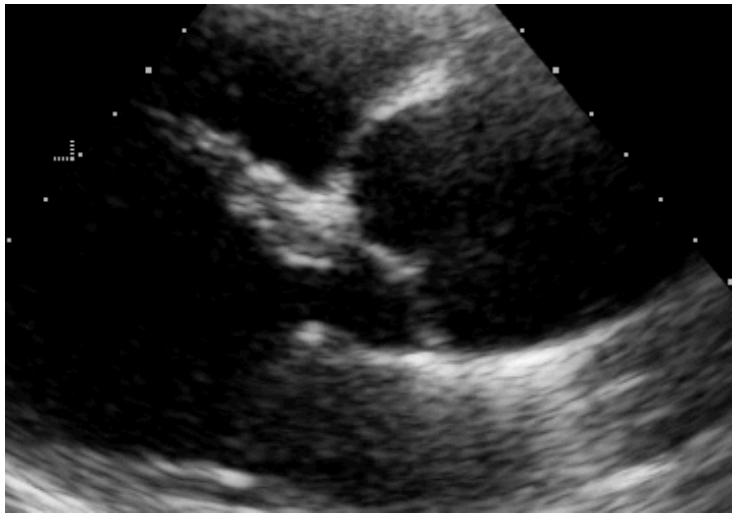
Esporàdic

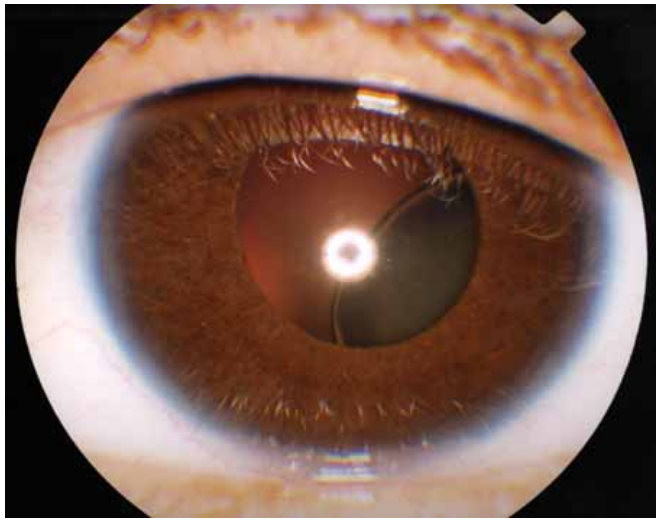
Síndrome de Marfan



Cromosoma 15q21.1
(Autosòmica Dominant)







New Ghent criteria for Marfan syndrome

Table 1. Revised Ghent criteria for the diagnosis of Marfan syndrome (MFS) and related conditions (14)

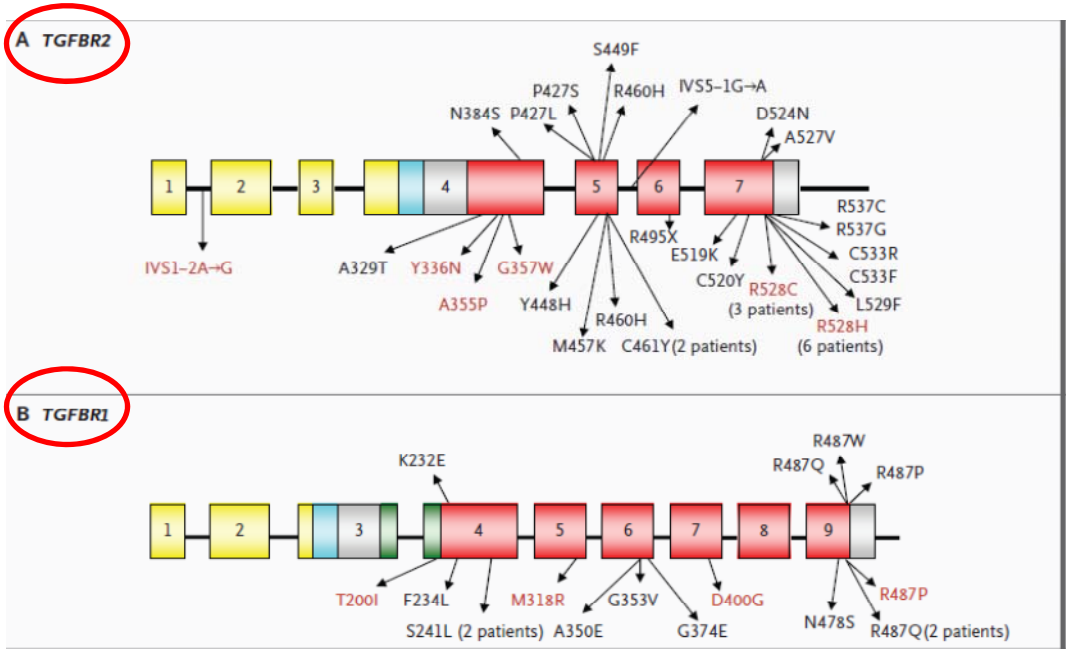
In the absence of a family history:
(1) Ao ($Z \geq 2$) AND EL = MFS
(2) Ao ($Z \geq 2$) AND *FBN1* = MFS
(3) Ao ($Z \geq 2$) AND Syst ($\geq$7 points) = MFS[a]
(4) EL AND *FBN1* with known Ao = MFS

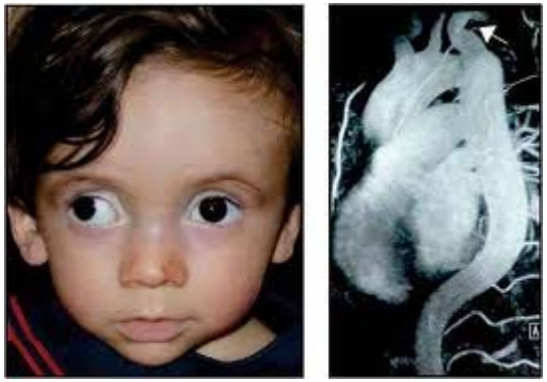
EL with or without Syst AND with an *FBN1* not known with Ao or no *FBN1* = ELS Ao ($Z < 2$) AND Syst ($\geq$5) with at least one skeletal feature without EL = MASS MVP AND Ao ($Z < 2$) AND Syst (>5) without EL = MVPS

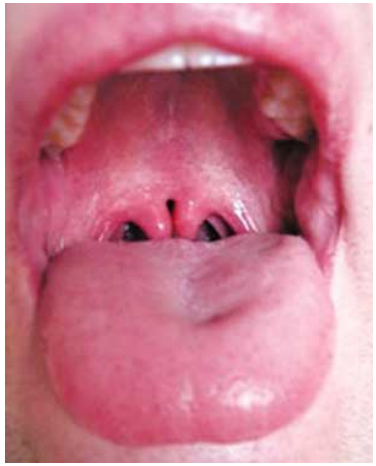
In the presence of a family history:
(5) EL AND FH of MFS (as defined above) = MFS
(6) Syst ($\geq$7 points) AND FH of MFS (as defined above) = MFS[a]
(7) Ao ($Z \geq 2$ above 20 years old, $\geq$3 below 20 years) + FH of MFS (as defined above) = MFS[a]

Loeys BL, J Med Genet 2010

Síndrome de Loeys-Dietz

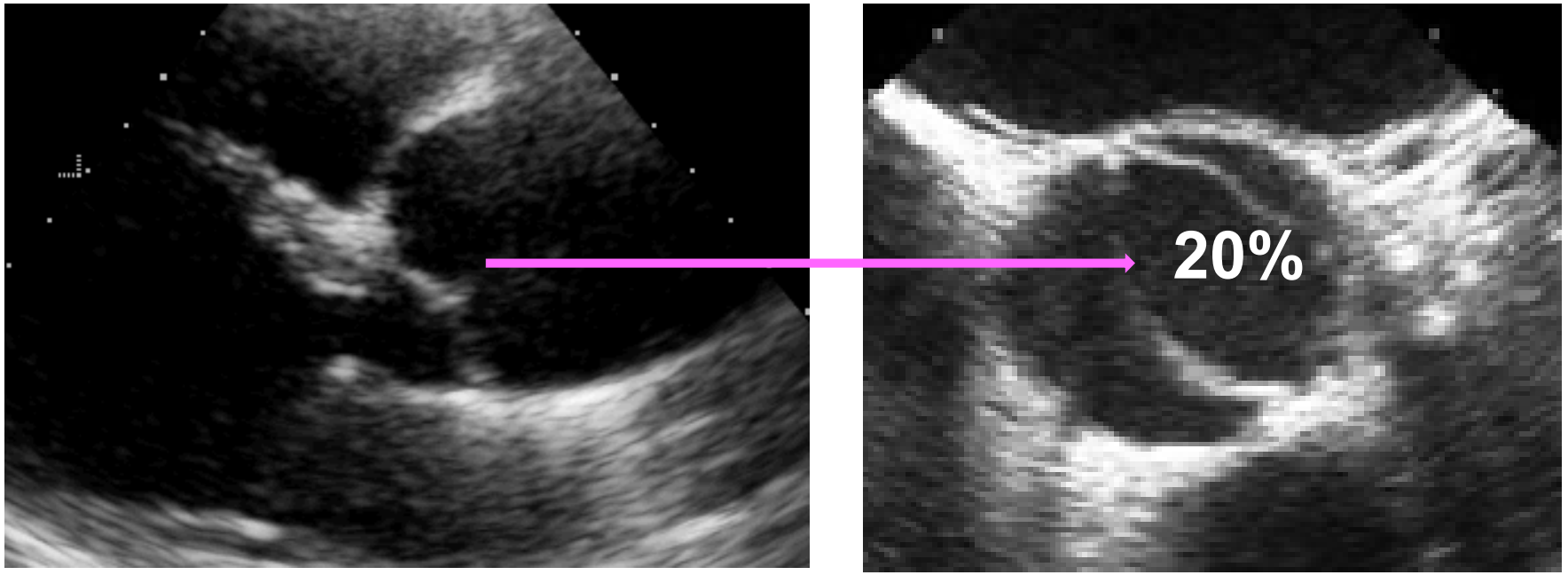






Loeys BL N Engl J Med 2006;355:788-98.

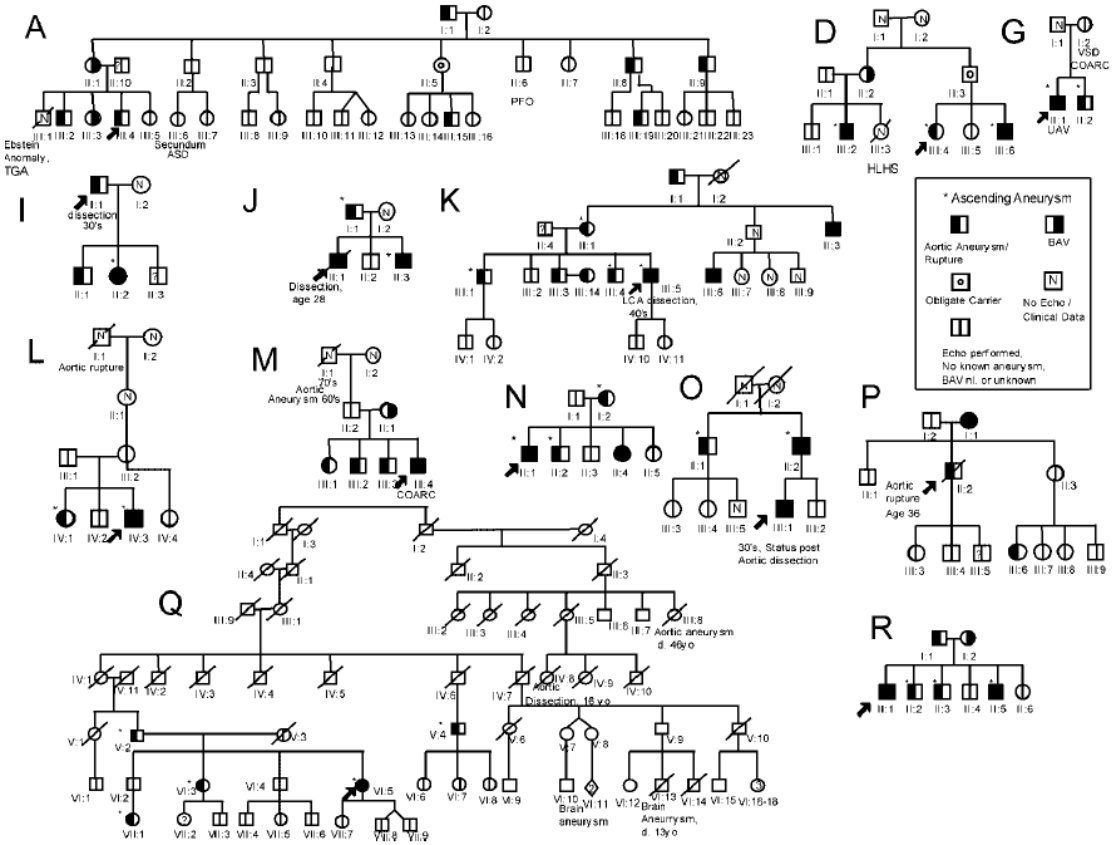
Vàlvula aòrtica bicúspide



Alegret JM. *Am J Cardiol* 2003;91:619-22

Familial Thoracic Aortic Dilation and Bicommissural Aortic Valve:

A Prospective Analysis of Natural History and Inheritance



13 families amb VAB

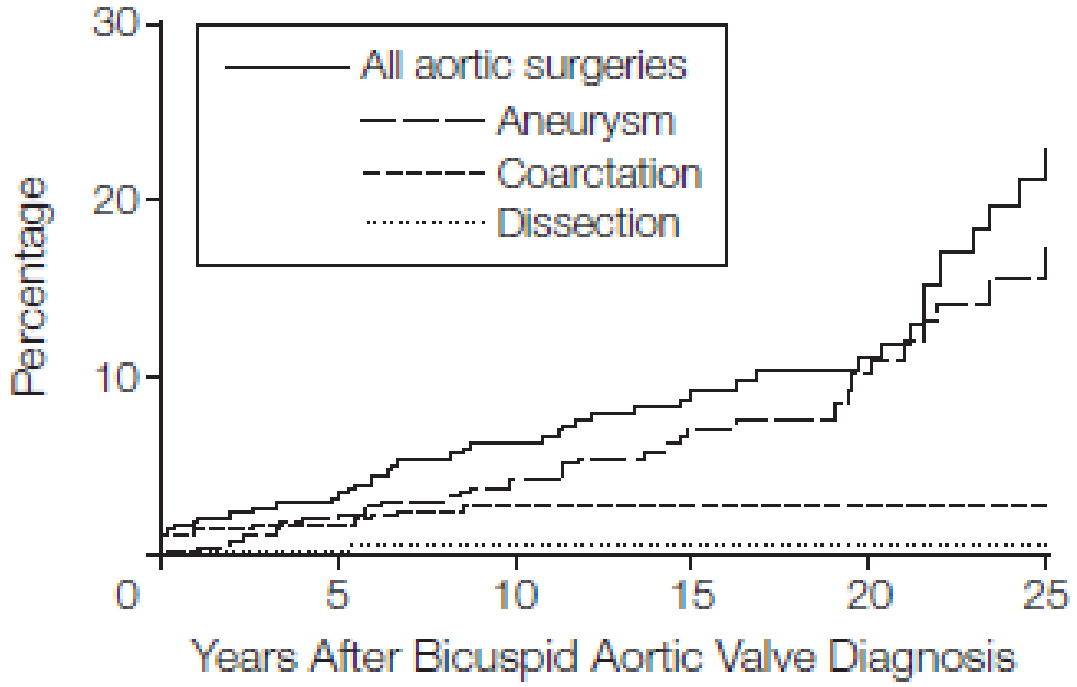
- 36% AAT

- 14% VAB

- 7 families amb disecció Ao (sense disfunció valvular)

(???)

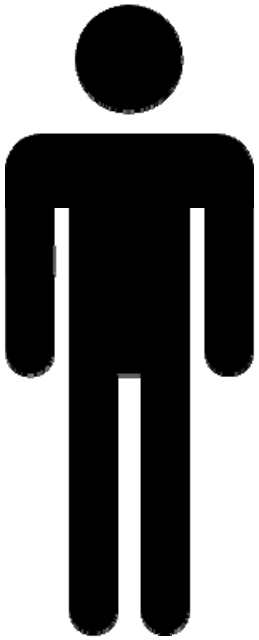
Loscalzo ML, Am J Med Gen 2007



2 DA de 416 VAB
(3.1 casos/10000 p/any)

Michelena HI, *JAMA. 2011;306(10):1104-1113*

AAT no sindròmic



11-19% Familiar

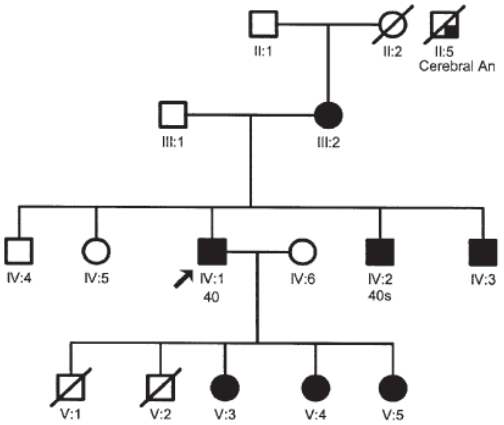
Esporàdic (st)

Hospital Universitari de Sant Joan Reus

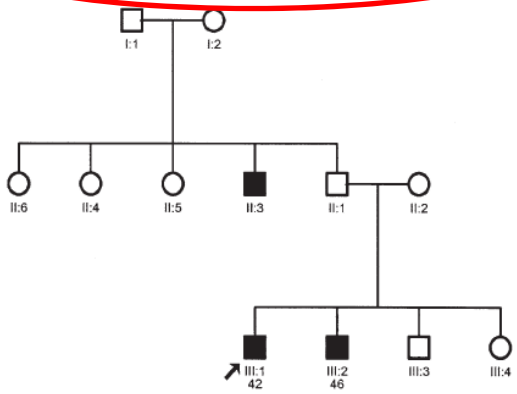
Hospital Quirón Barcelona diagnostic per la imatge

AAT Familiar

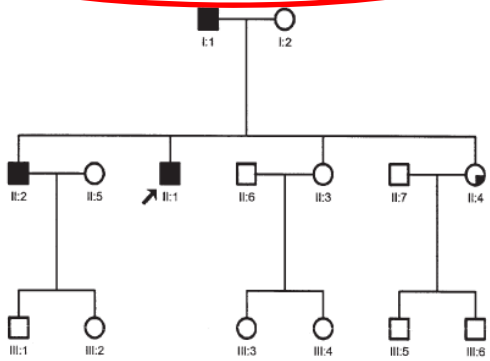
Autosomal Dominant



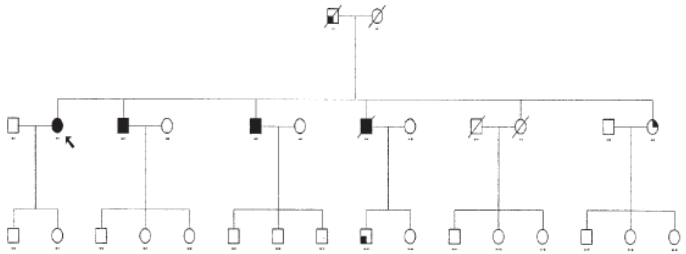
Autosomal Dominant with Decreased Penetrance



Autosomal Dominant with Variable Expressivity

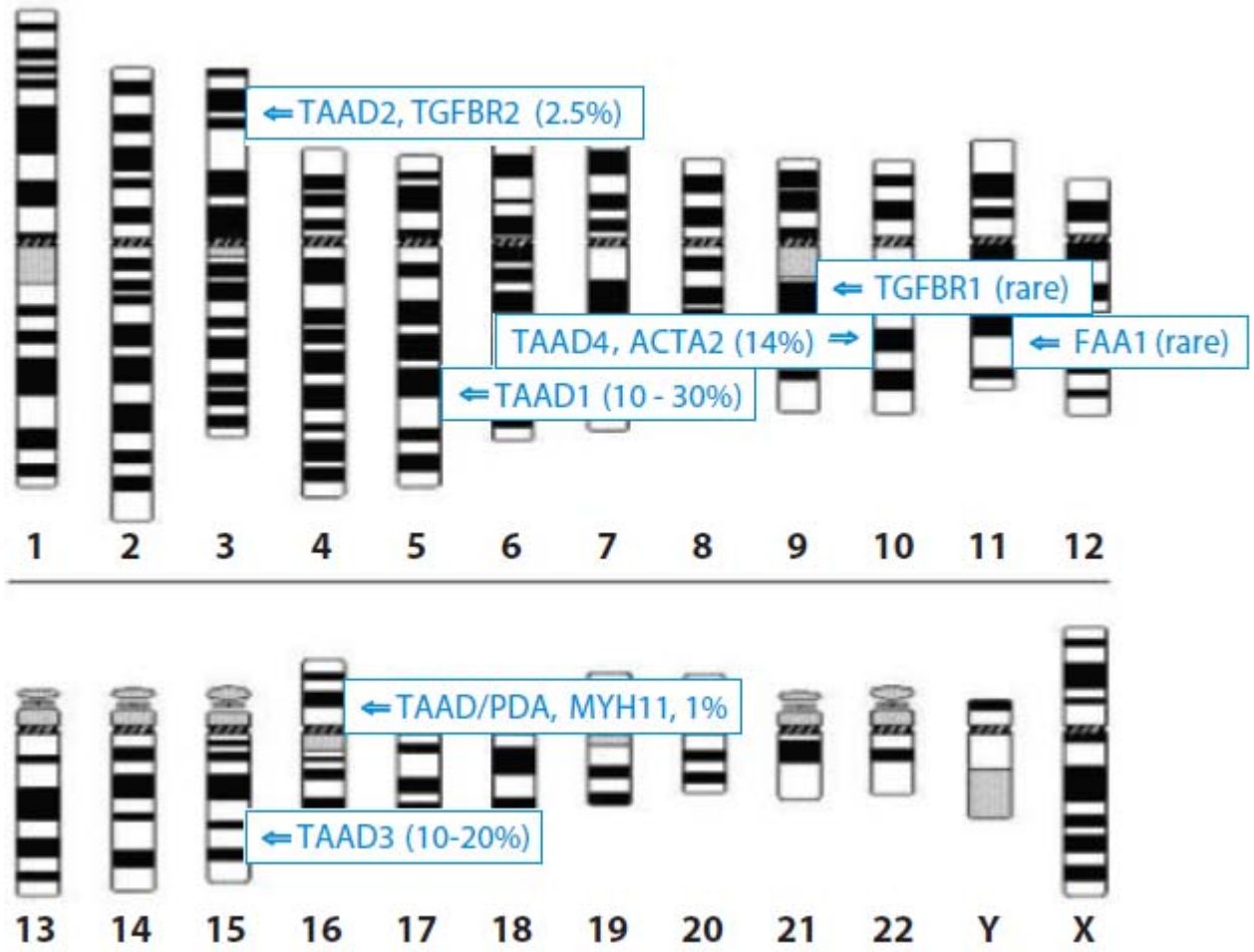


Autosomal Recessive



Davies RR, Ann Thorac Surg 2006;82:1400–6

Hospital Universitari de Sant Joan Reus



6 locus reconeguts

3 gens identificats:

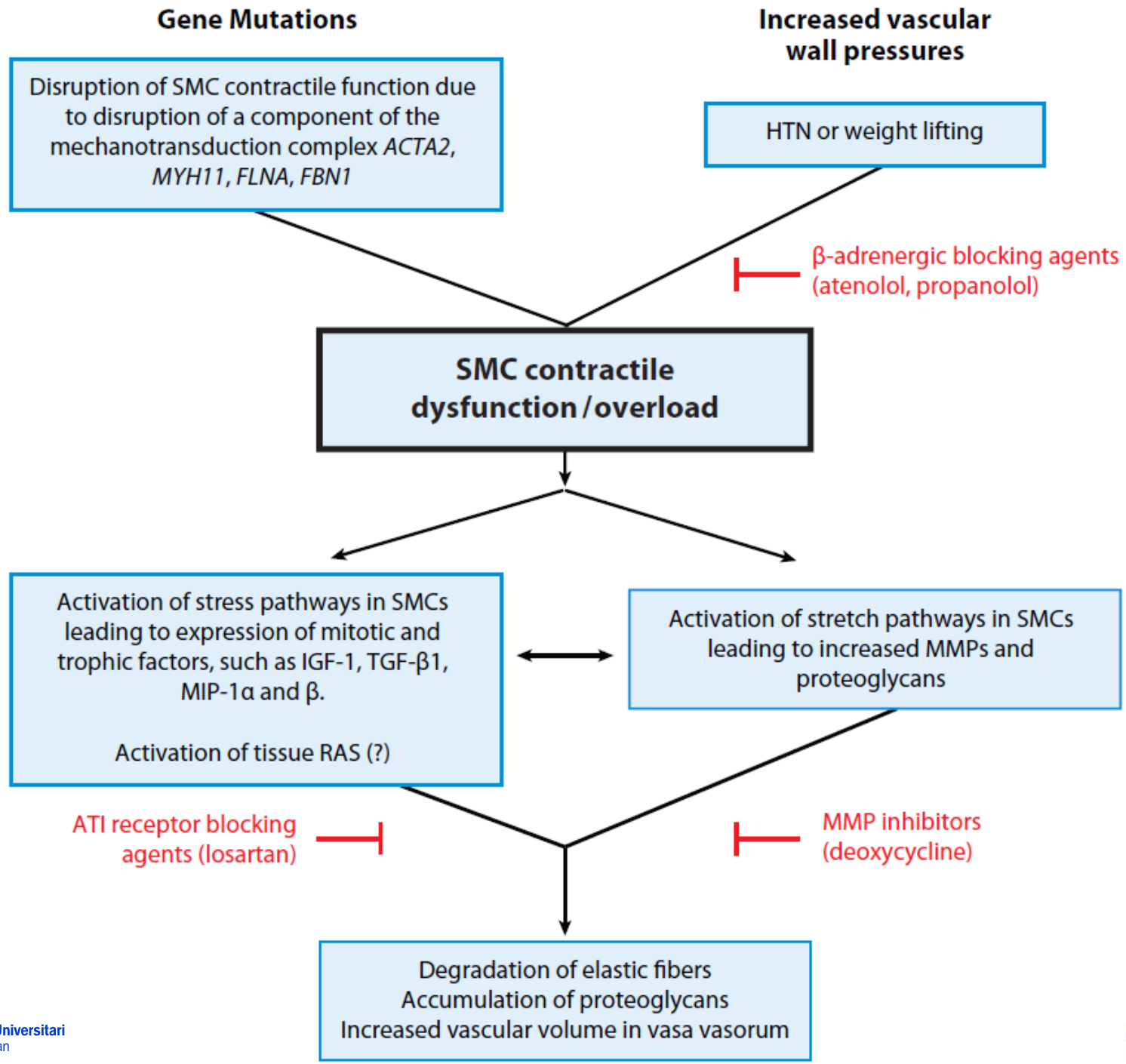
- **TGFBR2**
- **ACTA2**
- **MYH11**

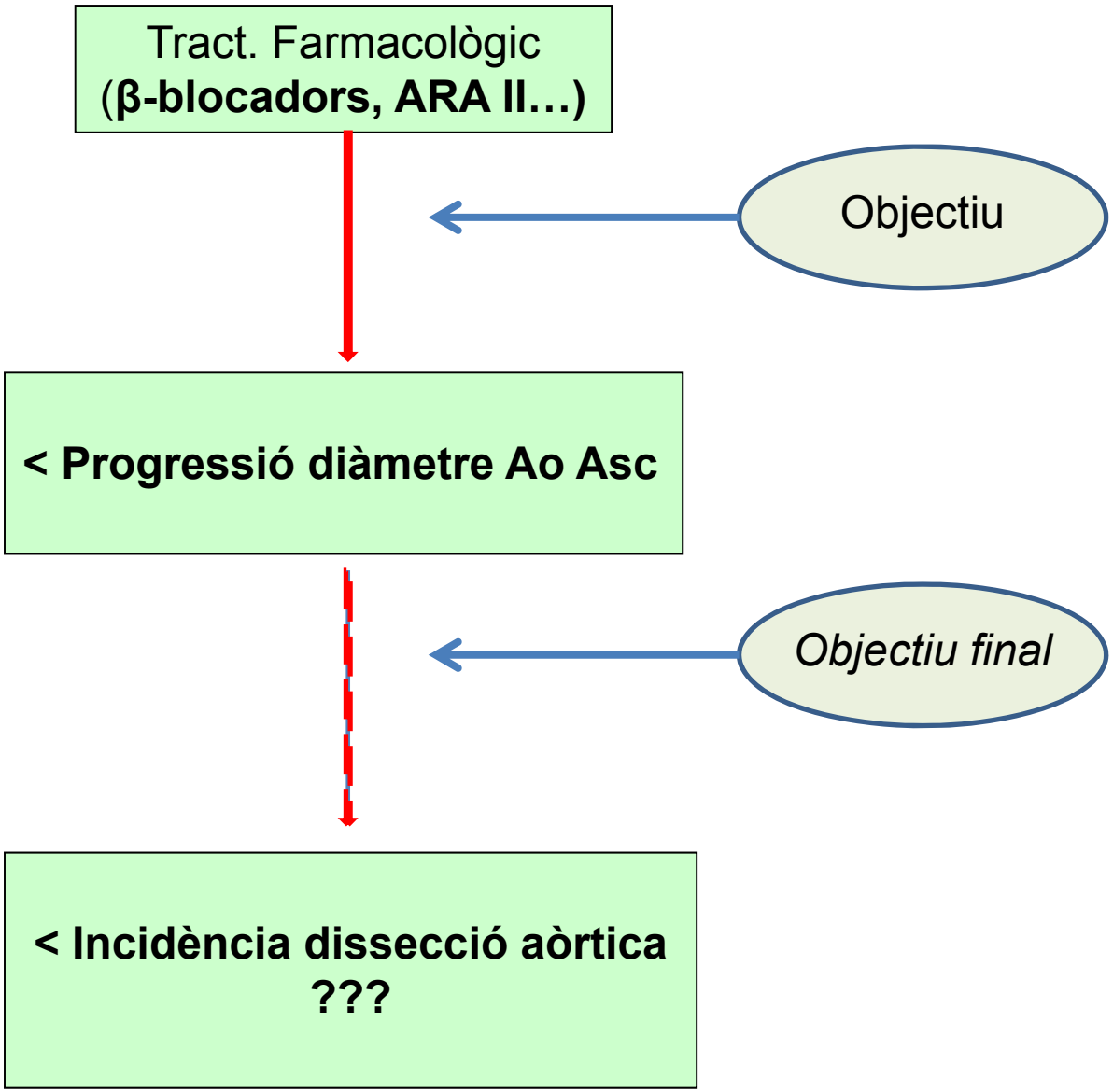
Identificable < 20%

< Edat que l'esporàdic

Milewicz 2008; Annu Rev Genomics Hum Genet

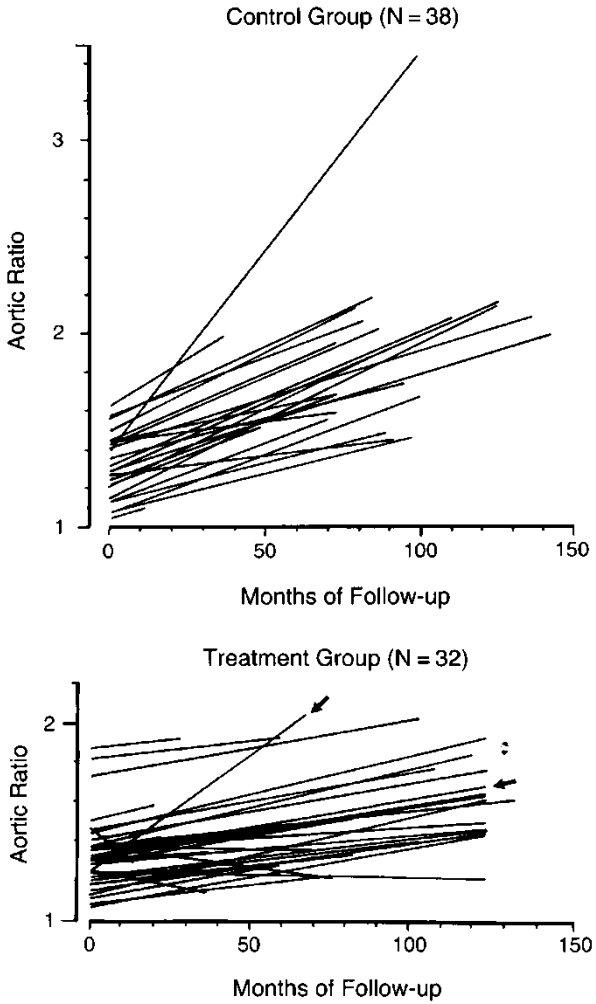
Tractament mèdic als AAT


Gene Mutations
Increased vascular wall pressures
Disruption of SMC contractile function due to disruption of a component of the mechanotransduction complex *ACTA2*, *MYH11*, *FLNA*, *FBN1*
HTN or weight lifting
β-adrenergic blocking agents (atenolol, propanolol)
SMC contractile dysfunction/overload
Activation of stress pathways in SMCs leading to expression of mitotic and trophic factors, such as IGF-1, TGF-β1, MIP-1α and β.
Activation of tissue RAS (?)
Activation of stretch pathways in SMCs leading to increased MMPs and proteoglycans
ATI receptor blocking agents (losartan)
MMP inhibitors (deoxycycline)
Degradation of elastic fibers
Accumulation of proteoglycans
Increased vascular volume in vasa vasorum

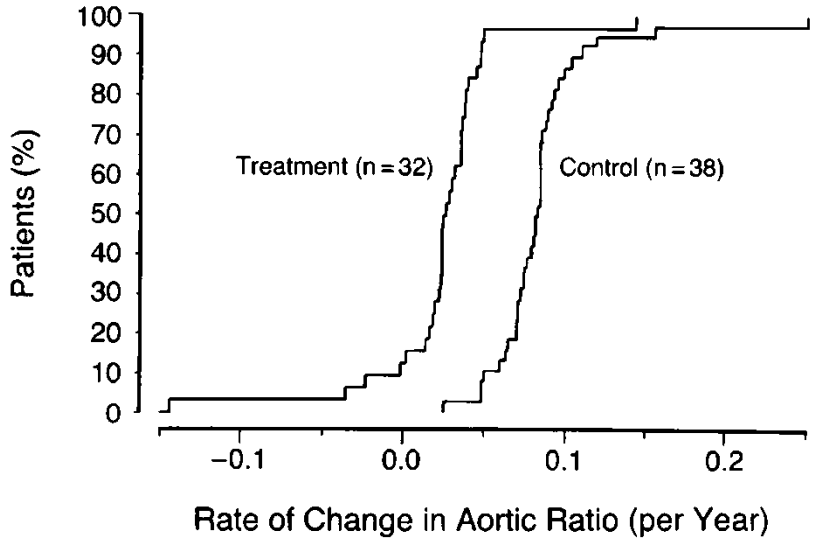


Hospital Universitari de Sant Joan Reus

CRC Hospital Quirón Barcelona diagnòstic per la imatge



Els **β-blocadors** enlenteixen la progressió de la dilatació aòrtica a la sínd. de Marfan



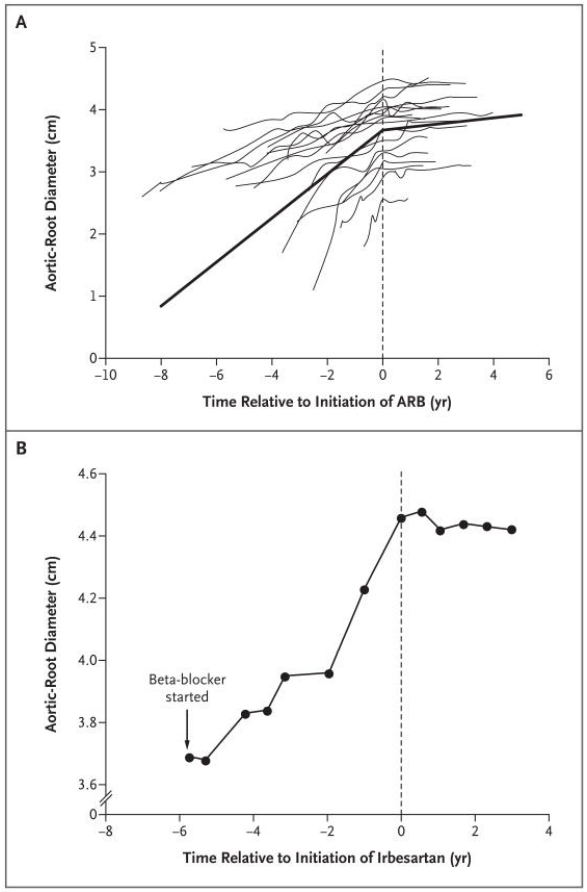
...pero altres estudis amb resultats negatius...

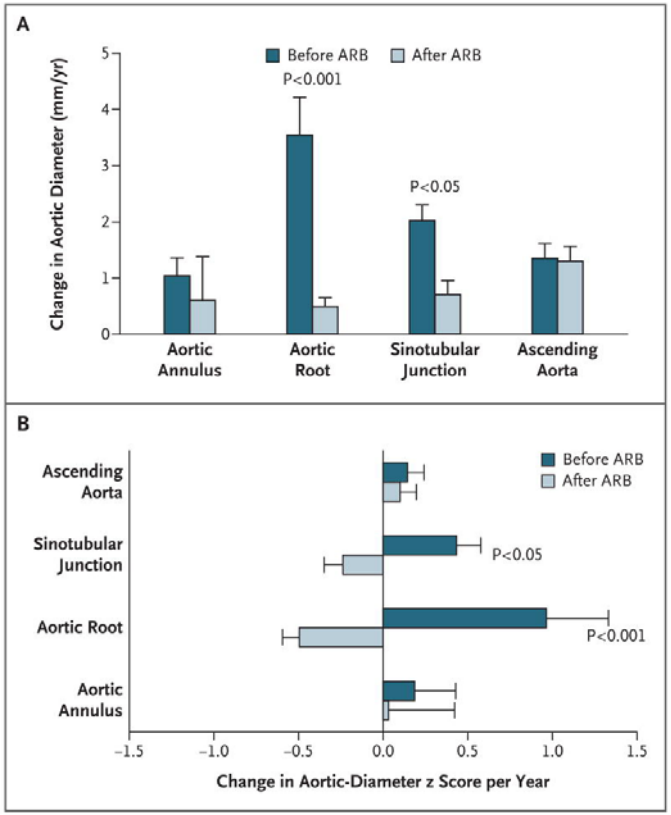
Yetman AT, Am J Cardiol 2005

Shores J, NEJM 1994

ORIGINAL ARTICLE

Angiotensin II Blockade and Aortic-Root Dilation in Marfan's Syndrome

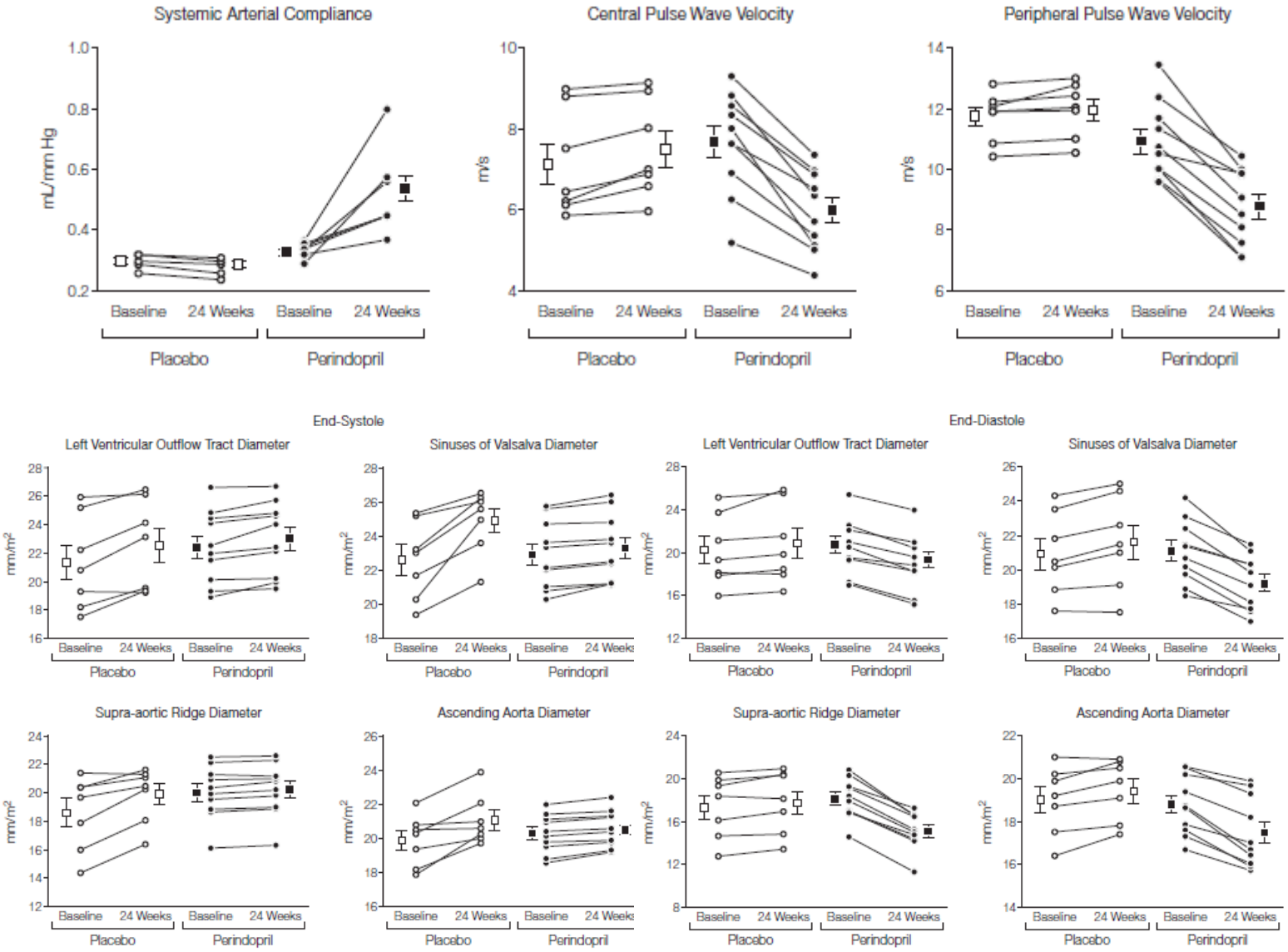




Losartan en**18** p. pediàtrics...
Retrospectiu...

Brooke BS, NEJM 2008

Hospital Universitari de Sant Joan Reus



Perindopril redueix la rigidesa aòrtica i la progressió dels diàmetres…
en 10 pacients amb Marfan… Ahimastos AA, JAMA 2007

Rev Esp Cardiol. 2011;64(6):492–498

Artículo original

Valoración de la eficacia y la seguridad del losartán frente al atenolol en la prevención de la dilatación de la aorta en el síndrome de Marfan

Alberto Forteza[a,*], Arturo Evangelista[b], Violeta Sánchez[a], Gisela Teixidó[b], Diana García[a], Paz Sanz[a], Laura Gutiérrez[b], Jorge Centeno[a], José Rodríguez-Palomares[b], José Cortina[a] y David García-Dorado[b]

[a] *Unidad de Marfan, Hospital Universitario 12 de Octubre, Madrid, España*
[b] *Unidad de Marfan, Hospital Universitario Vall d'Hebron, Barcelona, España*

Radonic *et al. Trials* 2010, **11**:3
http://www.trialsjournal.com/content/11/1/3

TRIALS

STUDY PROTOCOL **Open Access**

Losartan therapy in adults with Marfan syndrome: study protocol of the multi-center randomized controlled COMPARE trial

Hospital Universitari de Sant Joan Reus

CRC Hospital Quirón Barcelona diagnòstic per la imatge

Indicació del tractament quirúrgic als AAT

Journal of the American College of Cardiology
© 2010 by the American College of Cardiology Foundation and the American Heart Association, Inc.
Published by Elsevier Inc.

Vol. 55, No. 14, 2010
ISSN 0735-1097/10/$36.00
doi:10.1016/j.jacc.2010.02.015

PRACTICE GUIDELINE: FULL TEXT

2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM Guidelines for the Diagnosis and Management of Patients With Thoracic Aortic Disease

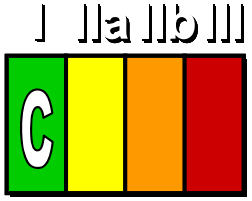
A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, American Association for Thoracic Surgery, American College of Radiology, American Stroke Association, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of Thoracic Surgeons, and Society for Vascular Medicine

Endorsed by the North American Society for Cardiovascular Imaging

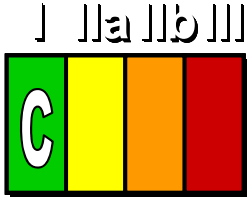
Hospital Universitari de Sant Joan Reus

CRC Hospital Quirón Barcelona diagnòstic per la imatge

Recomanacions ACC/AHA
Aneurisma de l'aorta ascendente



Els pacients asintomàtics amb aneurisma toràcic degeneratiu, disecció aòrtica crònica, hematoma intramural, úlcera penetrant, aneurisma micòtic, o pseudoaneurisma, amb diàmetres de l'aorta ascendent o dels sinus de Valsalva ≥ 5.5 cm, han de ser avaluats per a cirurgia reparadora aòrtica.

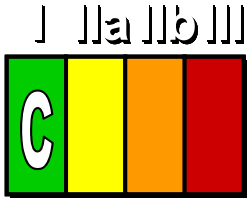


Els pacients amb sínd. de **Marfan** o altres malalties mediades genèticament (sind. de Ehlers-Danlos, sind. de Turner, **vàlvula aòrtica bicúspide**, o aneurisma/disecció de la aorta toràcica **familiar**) haurien de ser tractats quirúrgicamente amb diàmetres menors (**4.0 a 5.0 cm**) per evitar una disecció o rotura aòrtica.

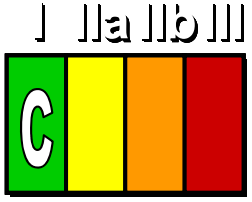
Hospital Universitari de Sant Joan Reus

CRC Hospital Quirón Barcelona diagnòstic per la imatge

Recomanacions ACC/AHA
Aneurisma de l'aorta ascendente



Els pacients amb un augment del diàmetre aòrtico ≥0.5 cm/any haurien de ser avaluats per IQ.



Els pacients sotmesos a reparació o **recanvi valvular aòrtic** i amb diàmetres de la Ao ascendent o de l'arrel aòrtica >4.5 cm haurien de ser avaluats per cirurgia de l'aorta.

Hospital Universitari de Sant Joan Reus

CRC Hospital Quirón Barcelona diagnòstic per la imatge

40. Clementi M, Notari L, Borghi A, Tenconi R. Familial congenital bicuspid aortic valve: a disorder of uncertain inheritance. Am J Med Genet 1996;62:336–8.
41. Glick BN, Roberts WC. Congenitally bicuspid aortic valve in multiple family members. Am J Cardiol 1994;73:400–4.
42. Cripe L, Andelfinger G, Martin J, Shooner K, Benson DW. Bicuspid aortic valve is heritable. J Am Coll Cardiol 2004;44:138–43.
43. Garg V, Muth AN, Ransom JF, et al. Mutations in *NOTCH1* cause aortic valve disease. Nature 2005;437:270–4.
44. Mohamed SA, Aherrahrou Z, Liptau H, et al. Novel missense mutations (p.T596M and p.P1797H) in *NOTCH1* in patients with bicuspid aortic valve. Biochem Biophys Res Commun 2006;345: 1460–5.
45. Martin LJ, Ramachandran V, [illegible] linkage to human chromosomal [illegible] aortic valve and associated cardi[illegible] 2007;121:275–84.
46. Guo DC, Pannu H, Tran-Fadulu V, et al. Mutations in smooth muscle alpha-actin (*ACTA2*) lead to thoracic aortic aneurysms and dissections. Nat Genet 2007;39:1488–93.
47. Huntington K, Hunter AG, Chan KL. A prospective study to assess the frequency of familial clustering of congenital bicuspid aortic valve. J Am Coll Cardiol 1997;30:1809–12.
48. Warnes CA, Williams RG, Bashore TM, et al. ACC/AHA 2008 guidelines for the management of adults with congenital heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Develop Guidelines on the Management of Adults With Congenital Heart Disease). J Am Coll Cardiol 2008;52:e1–121.
49. Chan KL, Stinson WA, Veinot JP. Reliability of transthoracic echocardiography in the assessment of aortic valve morphology: 2002;88:348–51.
63. Robicsek F, Thubrikar MJ, Cook JW, Fowler B. The congenitally bicuspid aortic valve: how does it function? Why does it fail? Ann Thorac Surg 2004;77:177–85.
64. Beppu S, Suzuki S, Matsuda H, Ohmori F, Nagata S, Miyatake K. Rapidity of progression of aortic stenosis in patients with congenital bicuspid aortic valves. Am J Cardiol 1993;71:322–7.
65. Fernandes SM, Khairy P, Sanders SP, Colan SD. Bicuspid aortic valve morphology and interventions in the young. J Am Coll Cardiol 2007;49:2211–4.
66. Otto CM, Burwash IG, Legget ME, et al. Prospective study of asymptomatic valvular aortic stenosis. Clinical, echocardiographic, and exercise predictors of outcome. Circulation 1997;95:2262–70.
[illegible], et al. Outcome of 622 [illegible]ly significant aortic steno- [illegible]n 2005;111:3290–5.
68. Rosenhek R, Binder T, Porenta G, et al. Predictors of outcome in severe, asymptomatic aortic stenosis. N Engl J Med 2000;343:611–7.
69. Roman MJ, Devereux RB, Niles NW, et al. Aortic root dilatation as a cause of isolated, severe aortic regurgitation. Prevalence, clinical and echocardiographic patterns, and relation to left ventricular hypertrophy and function. Ann Intern Med 1987;106:800–7.
70. Roberts WC, Morrow AG, McIntosh CL, Jones M, Epstein SE. Congenitally bicuspid aortic valve causing severe, pure aortic regurgitation without superimposed infective endocarditis. Analysis of 13 patients requiring aortic valve replacement. Am J Cardiol 1981;47: 206–9.
71. Pachulski RT, Chan KL. Progression of aortic valve dysfunction in 51 adult patients with congenital bicuspid aortic valve: assessment and follow up by Doppler echocardiography. Br Heart J 1993;69: 237–40.

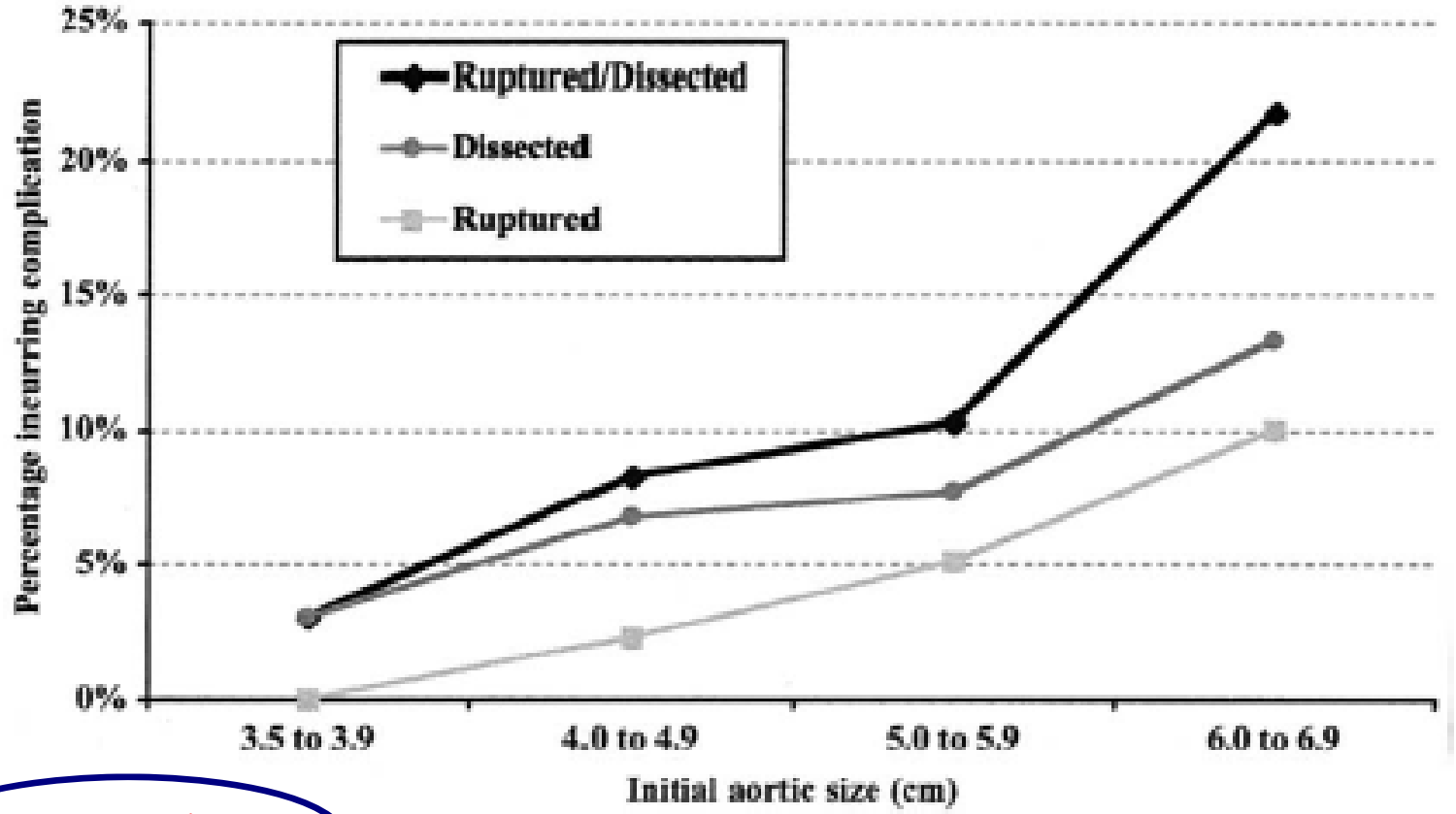
No evidències…

Guidelines on the management of valvular heart disease

The Task Force on the Management of Valvular Heart Disease of the European Society of Cardiology

	Class
Severe AR	
Symptomatic patients (dyspnoea, NYHA class II, III, IV or angina)	IB
Asymptomatic patients with resting LVEF ≤50%	IB
Patients undergoing CABG or surgery of ascending aorta, or on another valve	IC
Asymptomatic patients with resting LVEF >50% with severe LV dilatation:	
End-diastolic dimension >70 mm or	IIaC
ESD >50 mm (or >25 mm/m^2 BSA)[a]	IIaC
Whatever the severity of AR	
Patients who have aortic root disease with maximal aortic diameter[b]	
≥45 mm for patients with Marfan's syndrome	IC
≥50 mm for patients with bicuspid valves	IIaC
≥55 mm for other patients	IIaC

Vahanian A. Eur Heart J 2007

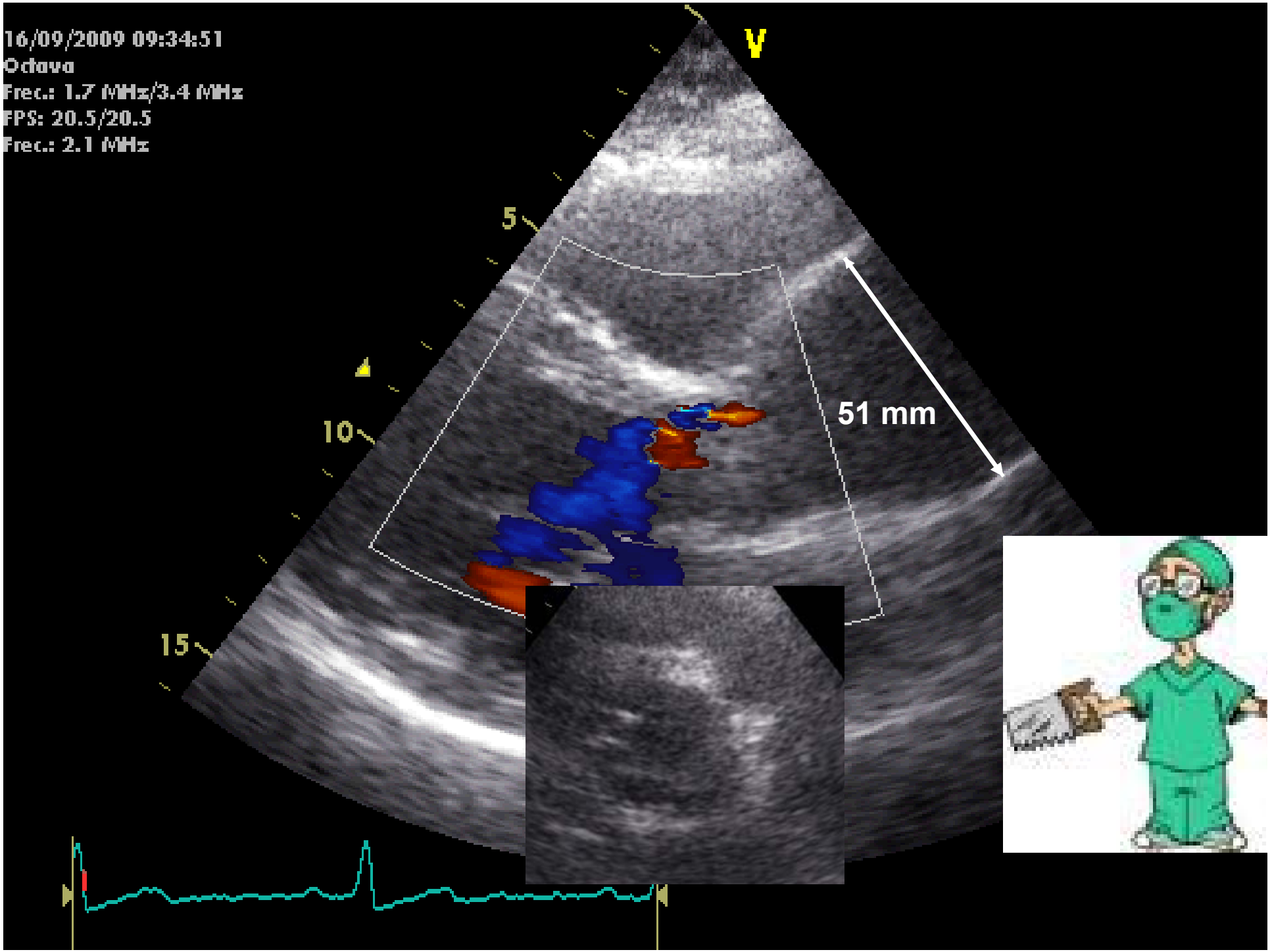


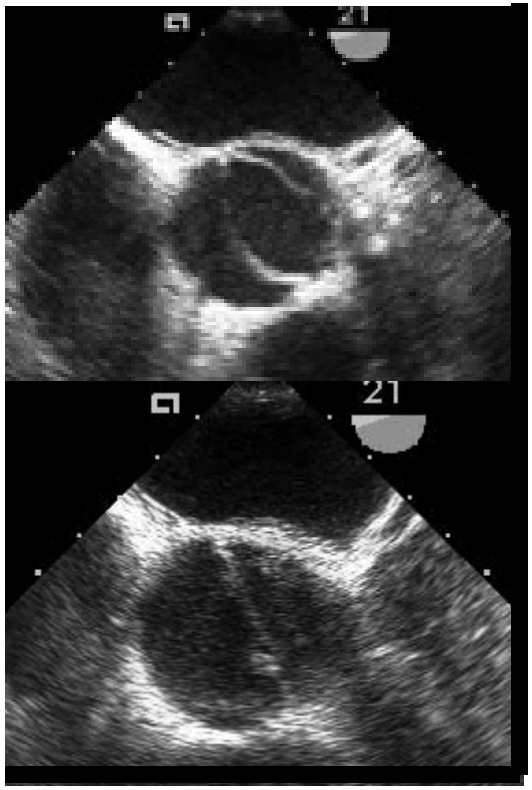
AAT > 6 cm

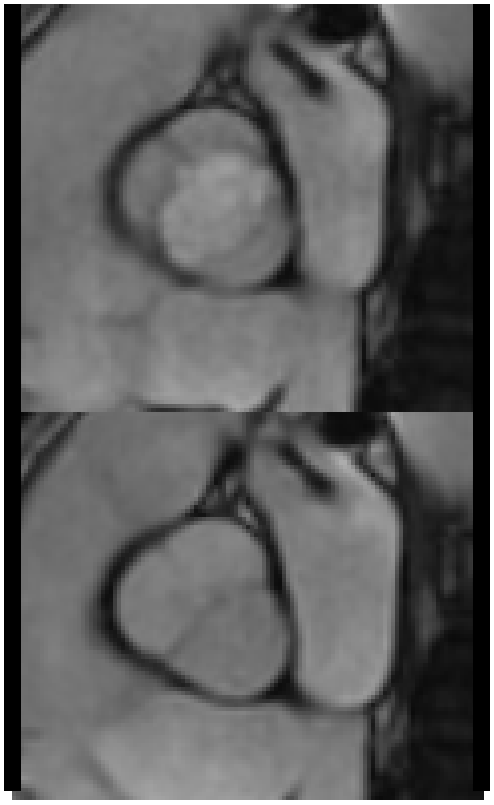
- 16% anual mort, disecció o ruptura

- OR 27 per ruptura

Davies RR. *Ann Thorac Surg* 2002


16/09/2009 09:34:51
Octava
Frec.: 1.7 MHz/3.4 MHz
FPS: 20.5/20.5
Frec.: 2.1 MHz
V
5
10
15
51 mm





ETE ?

CARDIO-RM ?

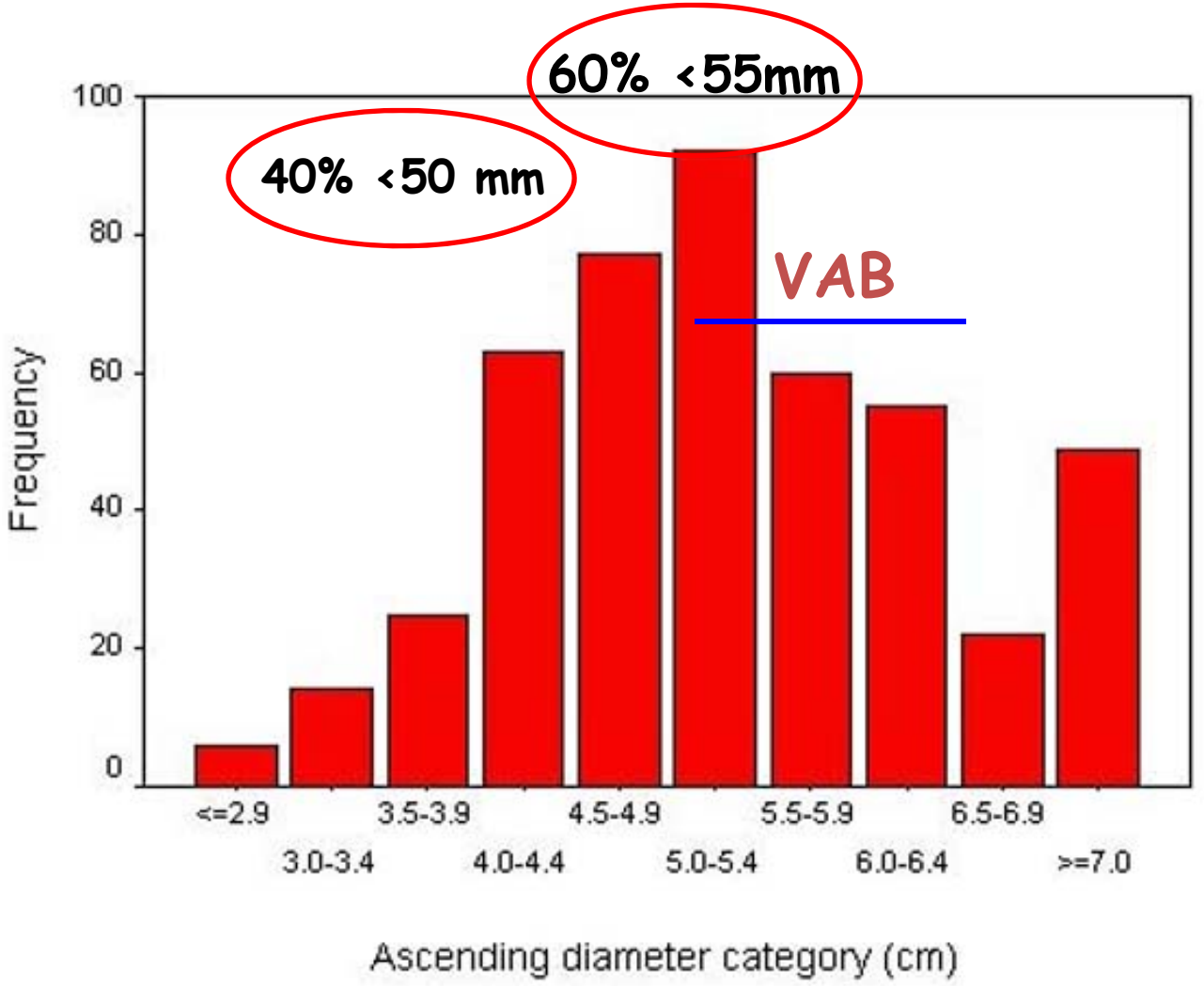
Hospital Universitari de Sant Joan Reus

Alegret JM, Int J Cardiovasc Imag 2005

Gleeson TG, J Magn Reson Imag 2008

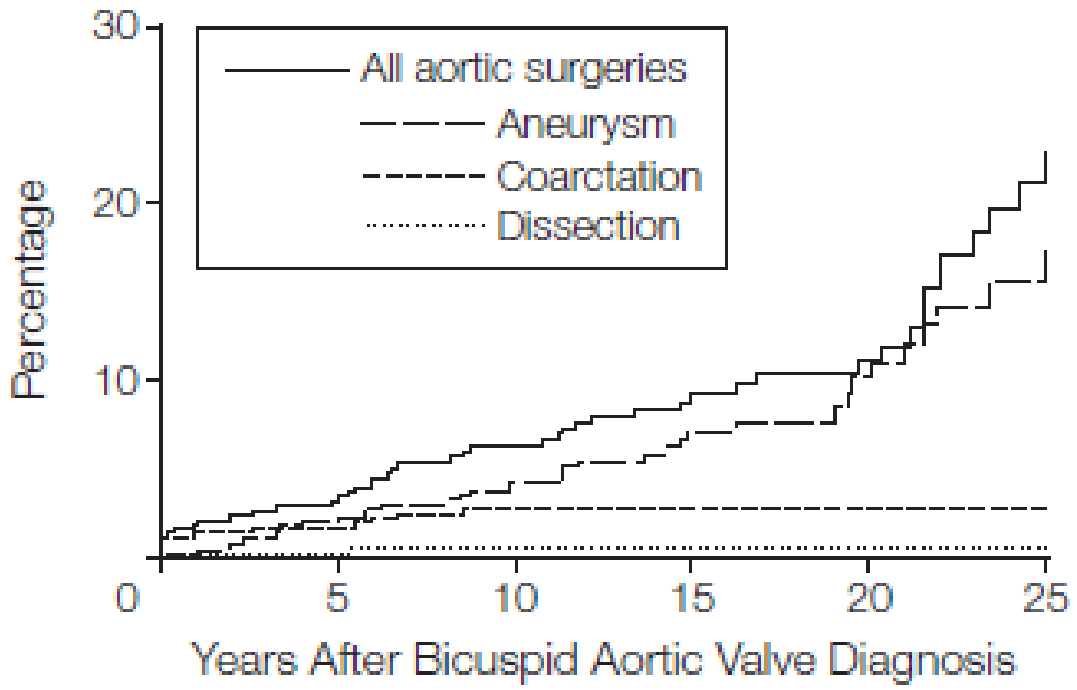
Quines evidències tenim per a recomanar IQ amb menors diàmetres a “bicúspides” vs “tricúspides”?

REGISTRE IRAD



Pape LA. *Circulation* 2007

Risc de DA a la v. aòrtica bicúspide



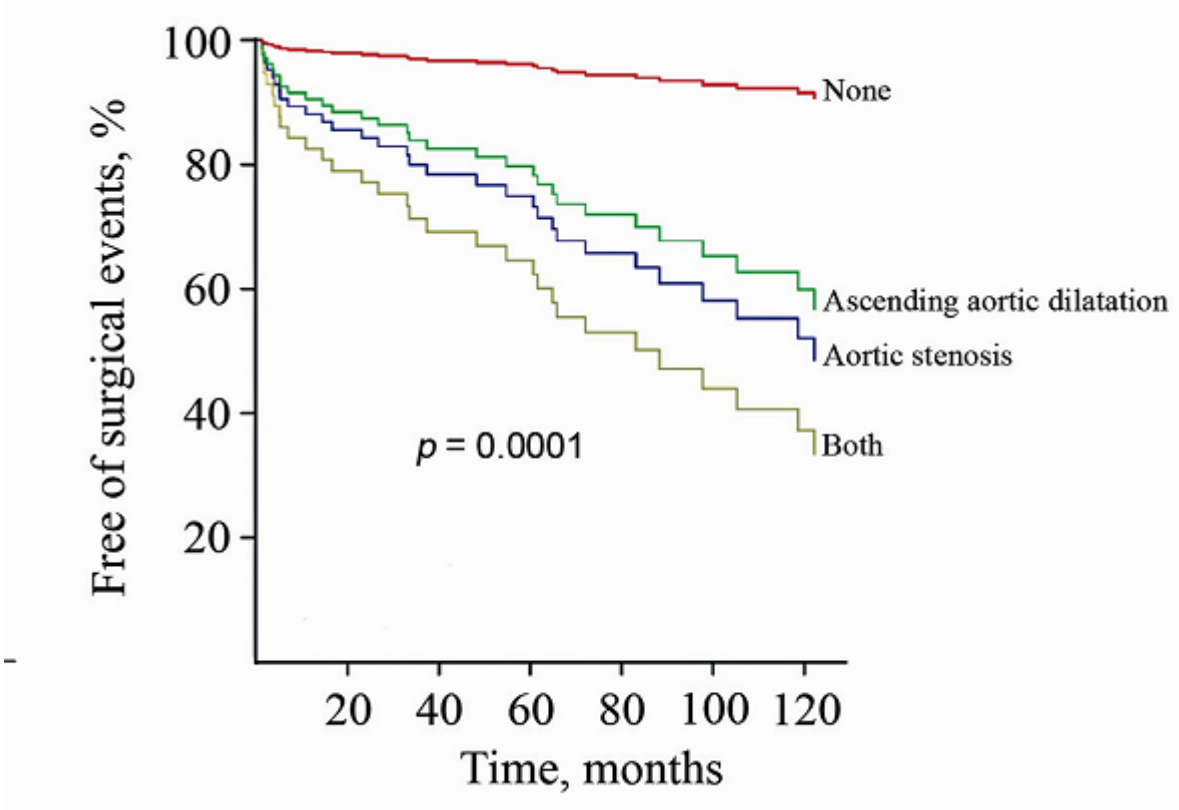
2 DA de 416 VAB
(3.1 casos/10000 p/any)

Michelena HI, *JAMA. 2011;306(10):1104-1113*

Hospital Universitari de Sant Joan Reus

CRC Hospital Quirón Barcelona diagnòstic per la imatge

Risc de DA a la v. aòrtica bicúspide

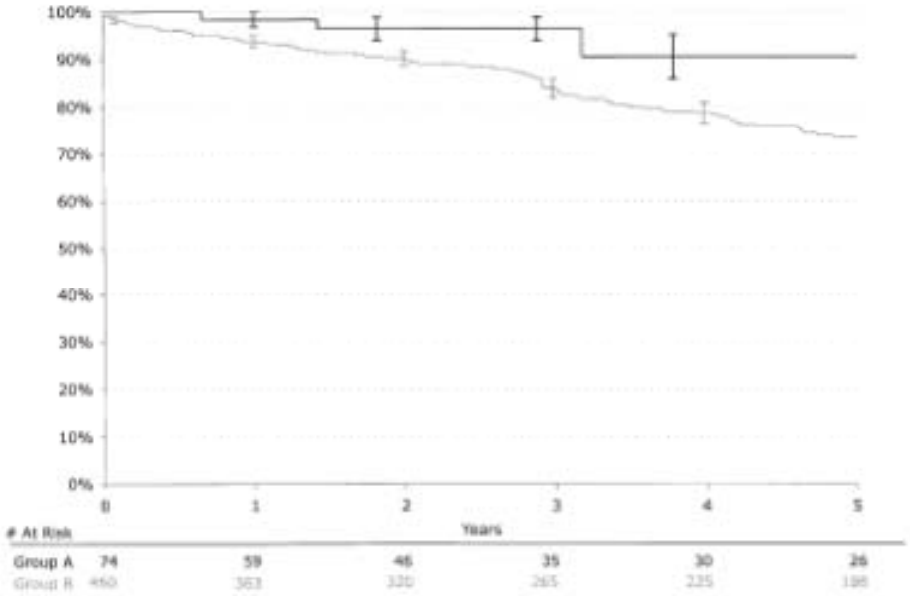


0 DA de 120 VAB
Seguiment mitjà 6 anys

Alegret JM et al. Eur Heart J, Abstr. Suppl. 2011

Hi ha altres factors a tenir presents en la indicació d'IQ en pacients amb VAB i dilatació aòrtica?

L’ estenosi aòrtica, factor associat amb disecció Ao en VAB

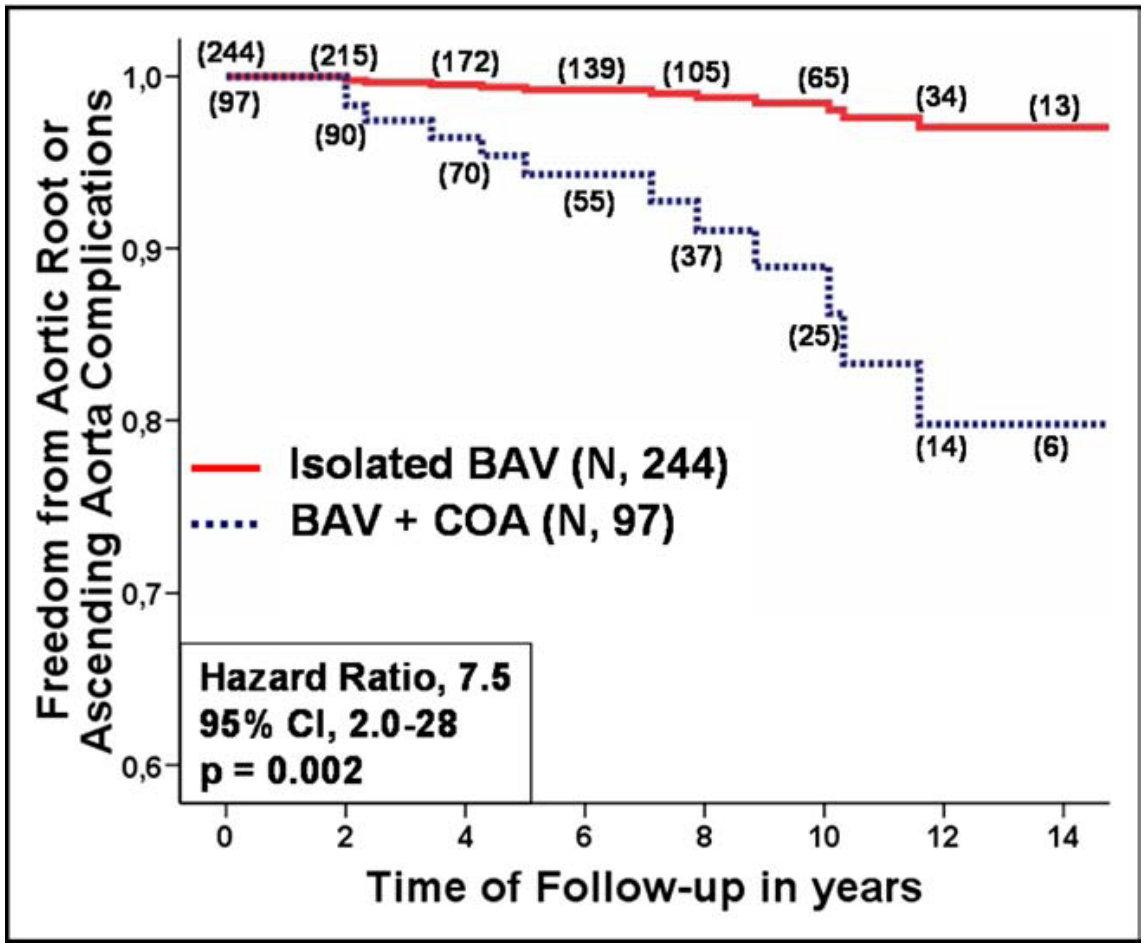


Davies RR. Ann Thorac Surg 2007

Hospital Universitari de Sant Joan Reus

Hospital Quirón Barcelona diagnostic per la imatge

La coartació aòrtica a la VAB augmenta el risc de DA



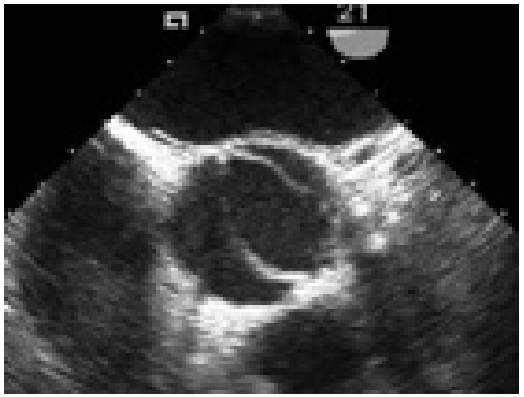
Oliver JM; Am J Cardiol 2009

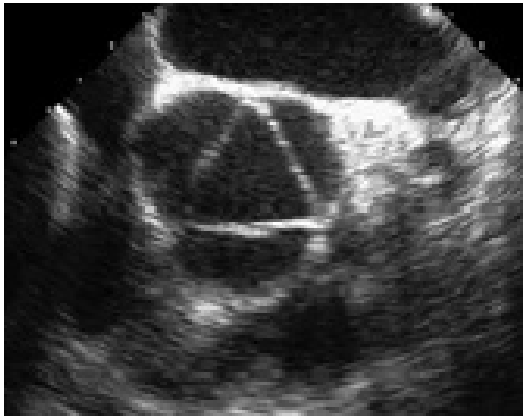
Hospital Universitari de Sant Joan Reus

Hospital Quirón Barcelona diagnostic per la imatge

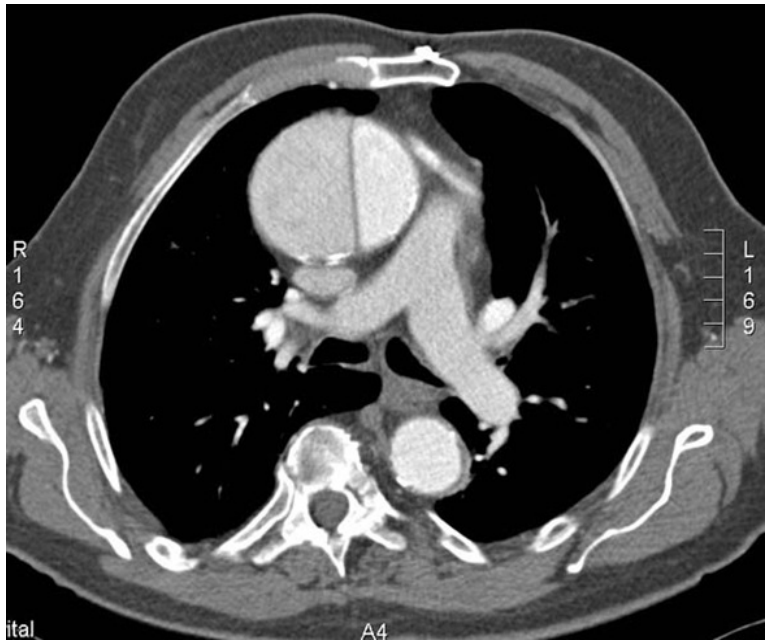
?

Alguns interrogants
de la pràctica clínica...

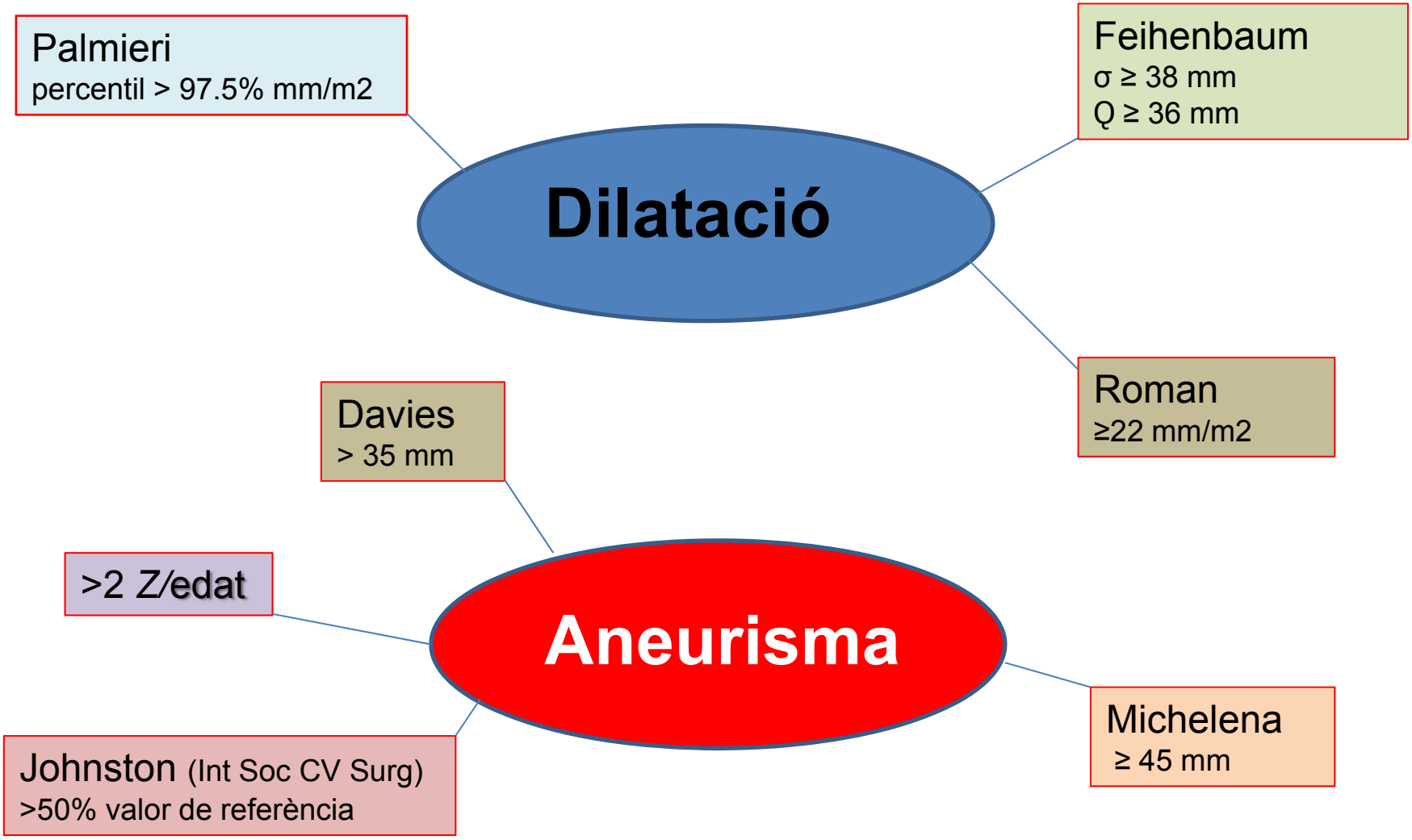




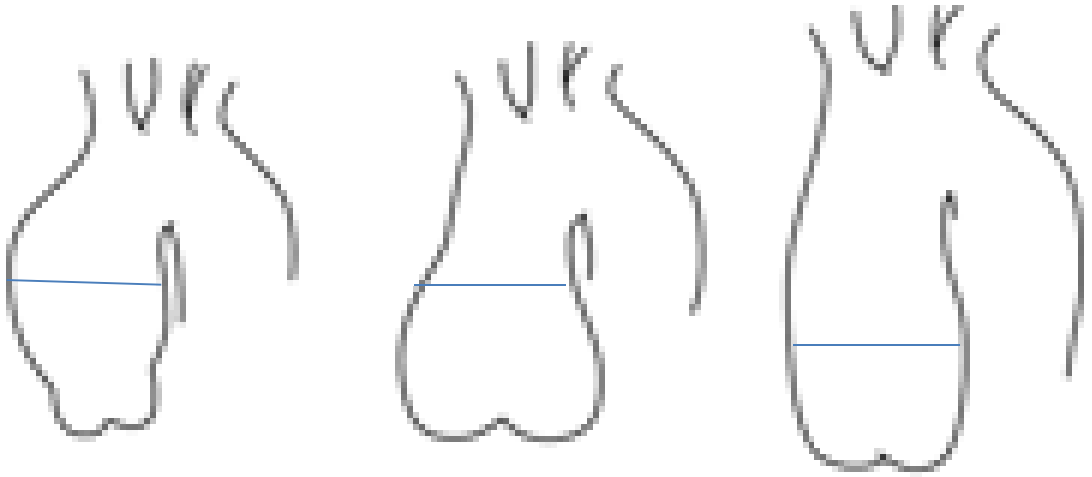
≠ Risc?



Què és *dilatació, aneurisma*?

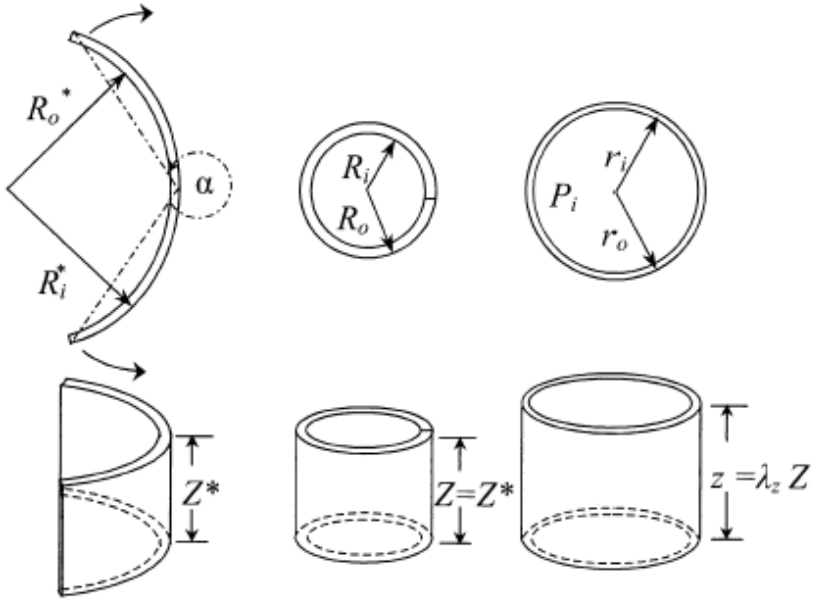


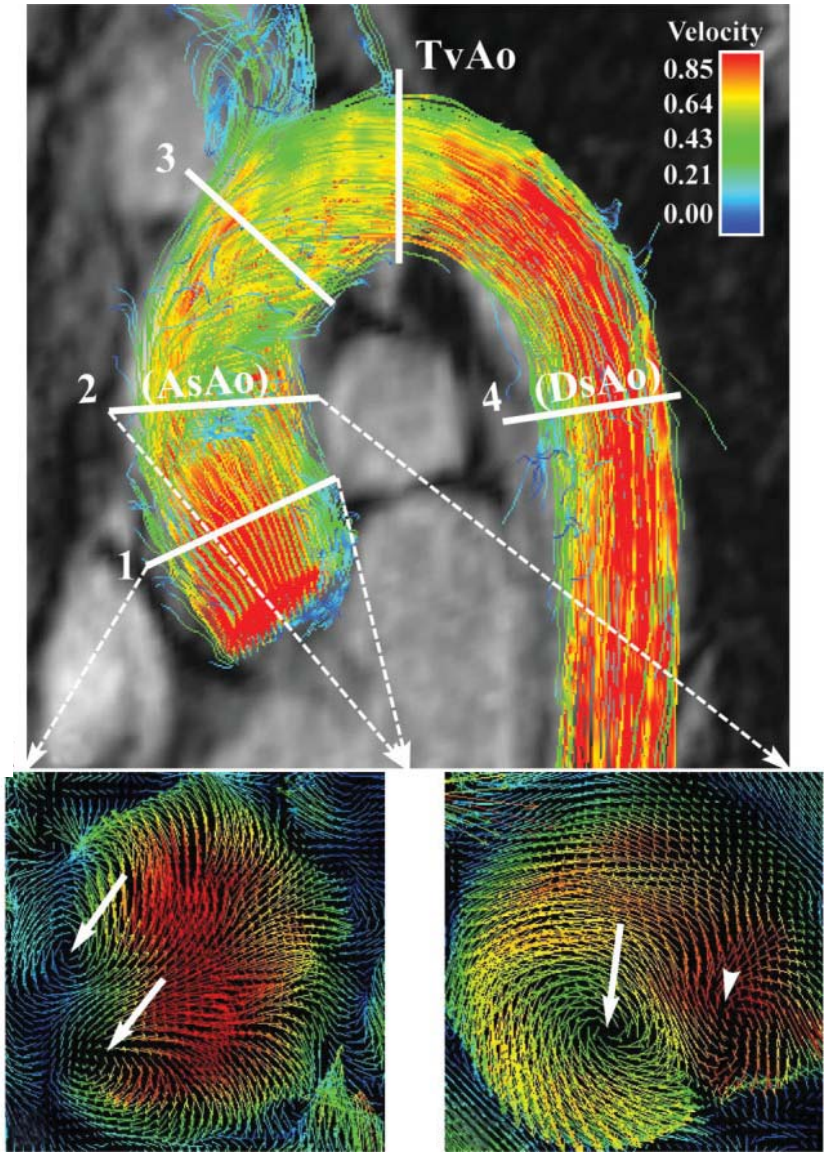

55 mm?
Cal indexar en la decisió d'IQ?



En tenim prou amb descriure el diàmetre màxim?
Té alguna utilitat el volum aòrtic?

Podrem quantificar els factors mecànics locals implicats en el risc de DA ?





El laboratori pot ser útils en la identificació dels AAT i l'estratificació de risc de DA?

Genètica
(FBN1, ACTA2, TGBR1, TGBR2, MYH11…)

Biomarcadors
(Dimer D, MMP, TGF*β,* expresió gènica*…)*

ESC GUIDELINES

Aorta toràcica 2012

????