Σεμινάριο Ομάδων Εργασίας ΕΚΕ, Φεβρουάριος 2010

Ομάδα Εργασίας Αορτής και Περιφερικών Αγγείων

Κλινική σημασία της αρτηριακής σκληρίας

Νικόλαος Αλεξόπουλος, Καρδιολόγος

Σισμανόγλειο ΓΝΑ

Γιατί μας ενδιαφέρει η αρτηριακή σκληρία;

Γιατί είναι ένας καλός δείκτης της λειτουργικής κατάστασης των αρτηριών μας

"A man is as old as his arteries"

Thomas Sydenham, 17ος αιώνας μ.Χ.

Βιολογική (αγγειακή) ηλικία ≠ χρονολογική ηλικία

(Risk Assessment Tool for Estimating Your 10-year Risk of Having a Heart Attack Version) - Mozilla Firefox

okmarks Tools Help

http://hp2010.nhlbihin.net/atpiii/calculator.asp?usertype=pub

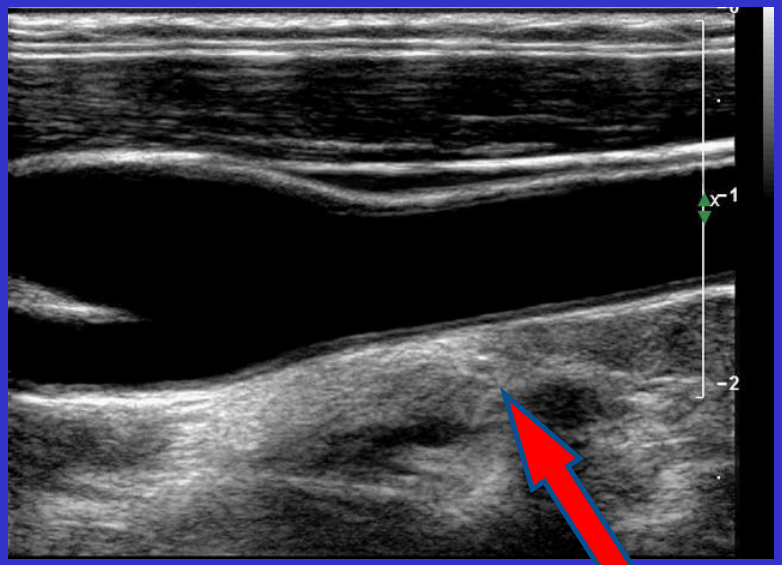
NATIONAL CHOLESTEROL EDUCATION PROGRAM
Third Report of the Expert Panel on
Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III)

Risk Assessment Tool for Estimating Your 10-year Risk of Having a Heart Attack

The risk assessment tool below uses information from the Framingham Heart Study to predict a person's chance of having a heart attack in the next 10 years. This tool is designed for adults aged 20 and older who do not have heart disease or diabetes. To find your risk score, enter your information in the calculator below.

Age:	years
Gender:	Female Male
Total Cholesterol:	mg/dL
HDL Cholesterol:	mg/dL
Smoker:	No Yes
Systolic Blood Pressure:	mm/Hg
Are you currently on any medication to treat high blood pressure.	No Yes

Calculate Your 10-Year Risk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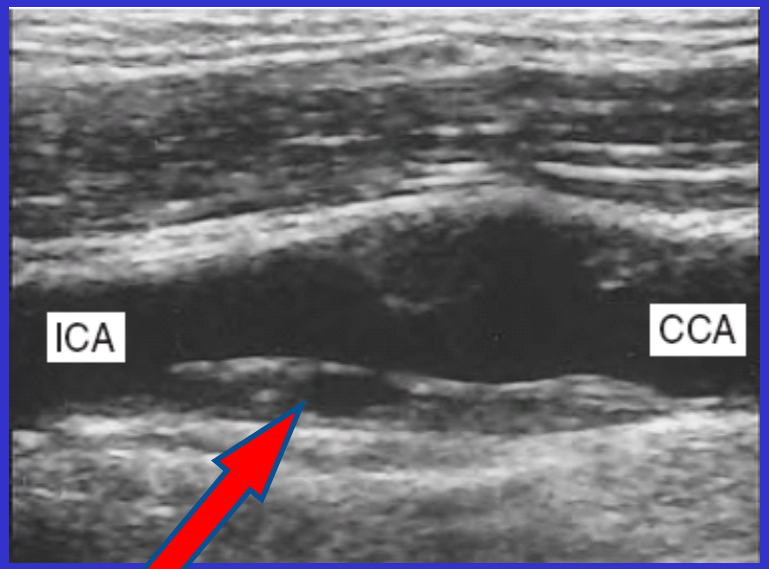
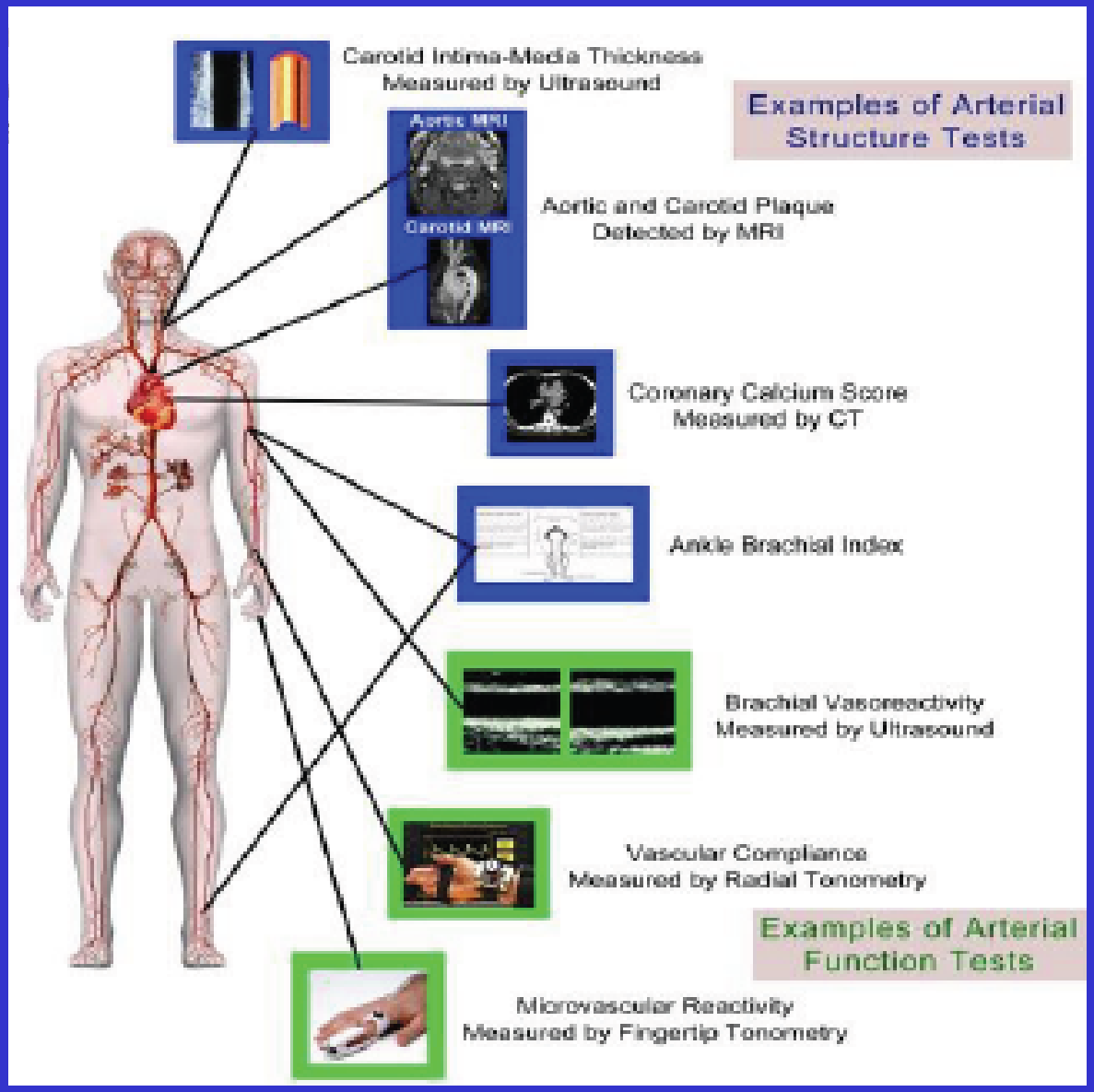

ICA
CCA

Ανίχνευση υποκλινικής αγγειακής βλάβης



Δείκτες αγγειακής ηλικίας

Η εκφύλιση των αρτηριών που επέρχεται με την ηλικία και την επίδραση των διαφόρων παραγόντων κινδύνου έχουν ως αποτέλεσμα:

Λειτουργικές μεταβολές →
- Ενδοθηλιακή λειτουργία
- Αρτηριακή σκληρία
- Κεντρικές πιέσεις

Δομικές μεταβολές → Πάχος έσω-μέσου χιτώνα

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Αρτηριακή σκληρία

European Heart Journal Advance Access published September 25, 2006

European Heart Journal
doi:10.1093/eurheartj/ehl254

EUROPEAN SOCIETY OF CARDIOLOGY®

Special article

Expert consensus document on arterial stiffness: methodological issues and clinical applications

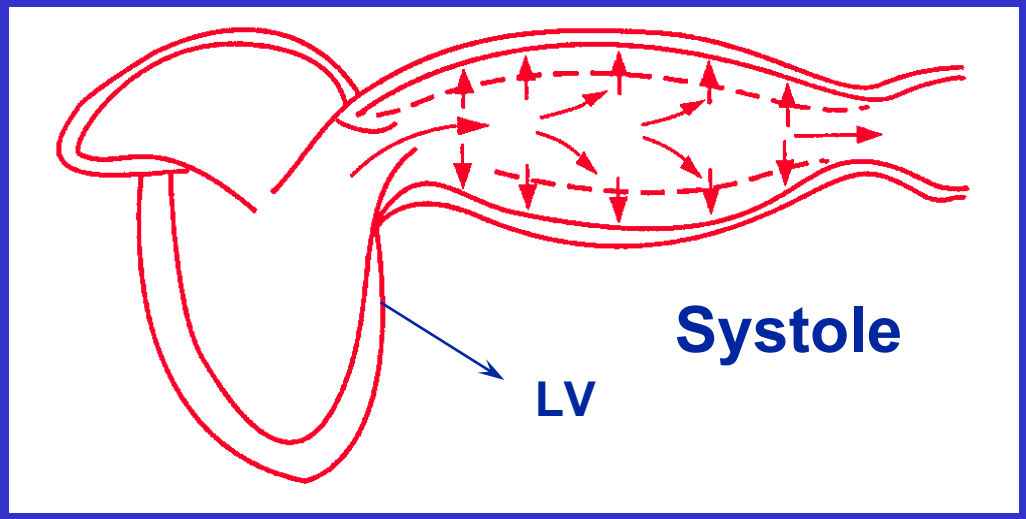
Stephane Laurent[1*], John Cockcroft[2], Luc Van Bortel[3], Pierre Boutouyrie[1], Cristina Giannattasio[4], Daniel Hayoz[5], Bruno Pannier[6], Charalambos Vlachopoulos[7], Ian Wilkinson[8], and Harry Struijker-Boudier[9] on behalf of the European Network for Non-invasive Investigation of Large Arteries

Αρτηριακή σκληρία – θεωρητική βάση

Left ventricular ejection generates a pulse wave which will propagate along the arterial walls at certain speed

Blood = incompressible fluid
Artery = elastic conduit

} ⟹ Propagation along the arterial tree

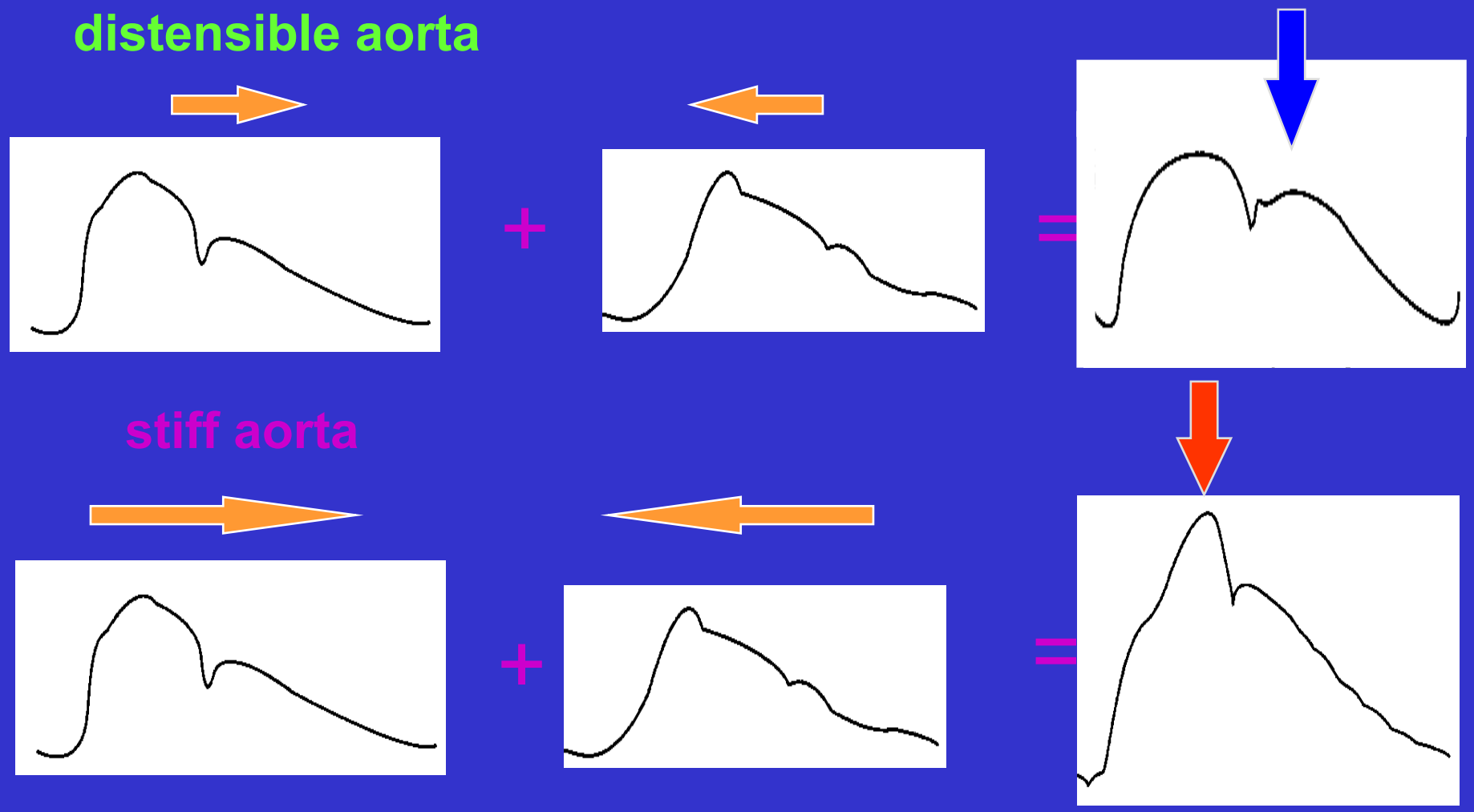


The propagation velocity is determined by:

The elastic and geometric properties of the arterial wall
The characteristics of the arterial wall structure

Αρτηριακή σκληρία – θεωρητική βάση

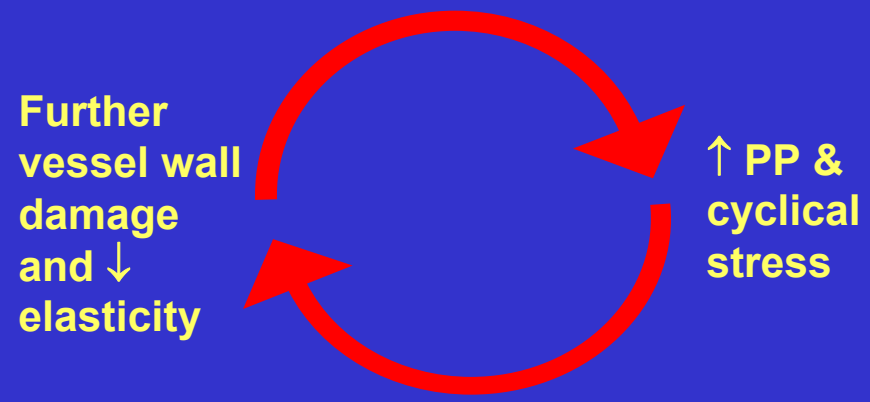
Arterial Stiffness and Wave Reflections

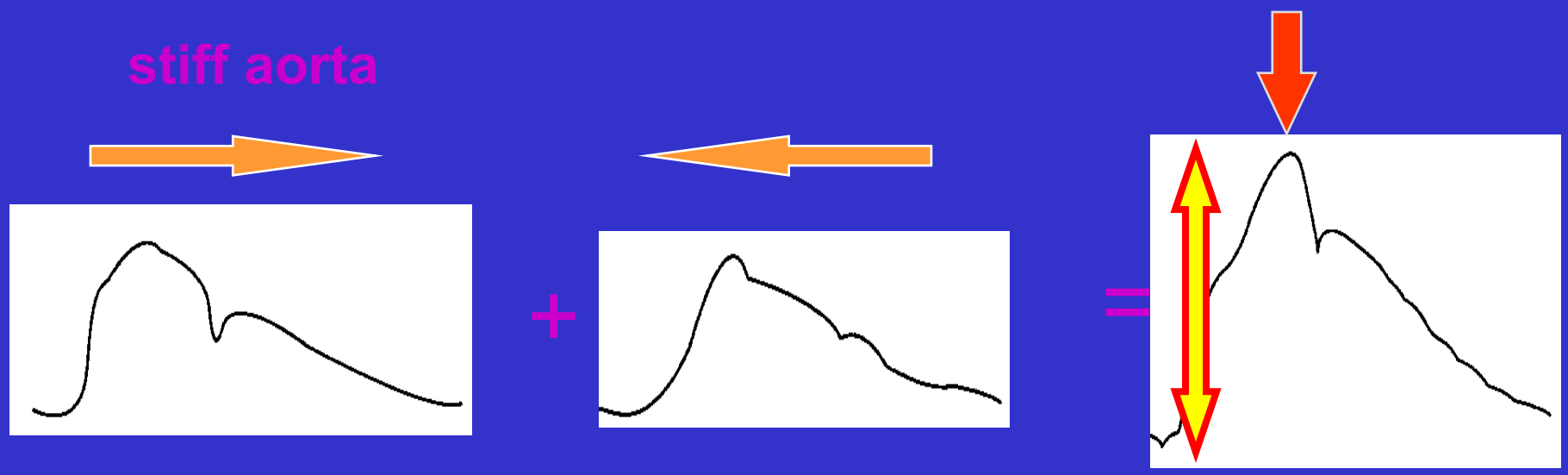


Vlachopoulos C, O'Rourke MF. Curr Probl Cardiol 2000

Αρτηριακή σκληρία – θεωρητική βάση

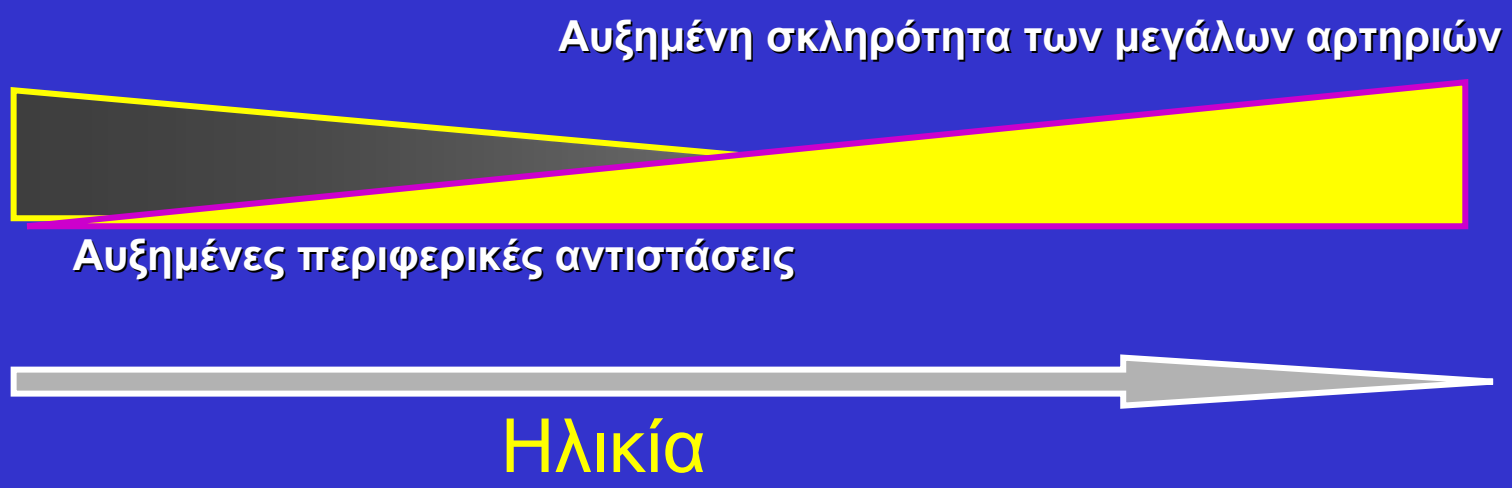
Arterial Stiffness and Wave Reflections





Vlachopoulos C, O'Rourke MF. Curr Probl Cardiol 2000

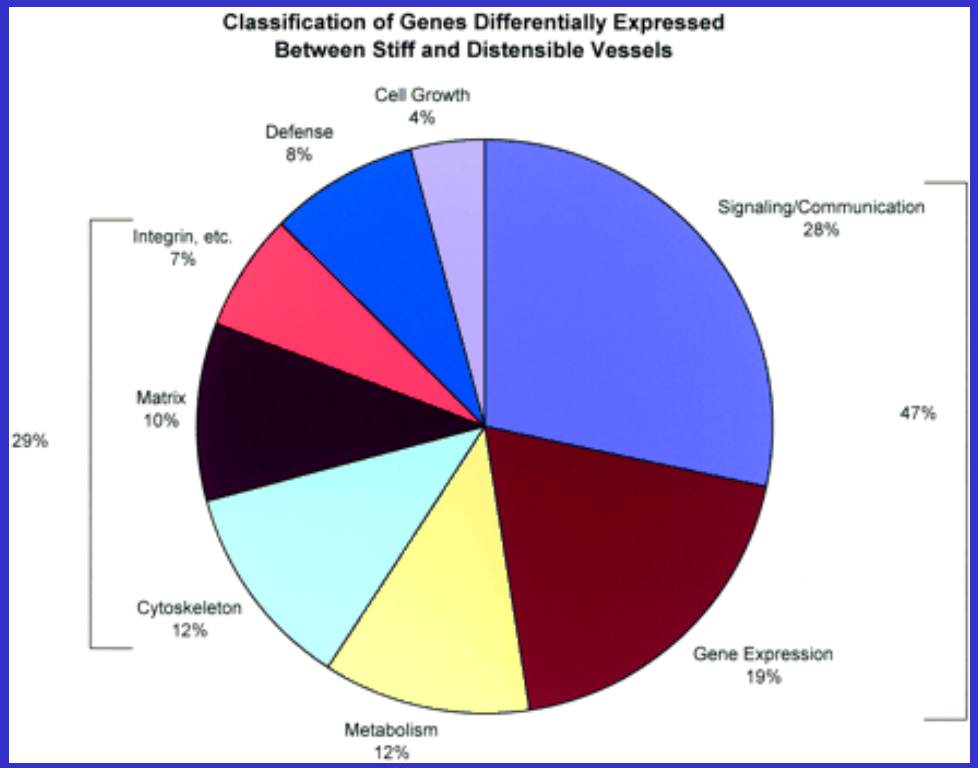
Υπέρταση - Παθοφυσιολογία



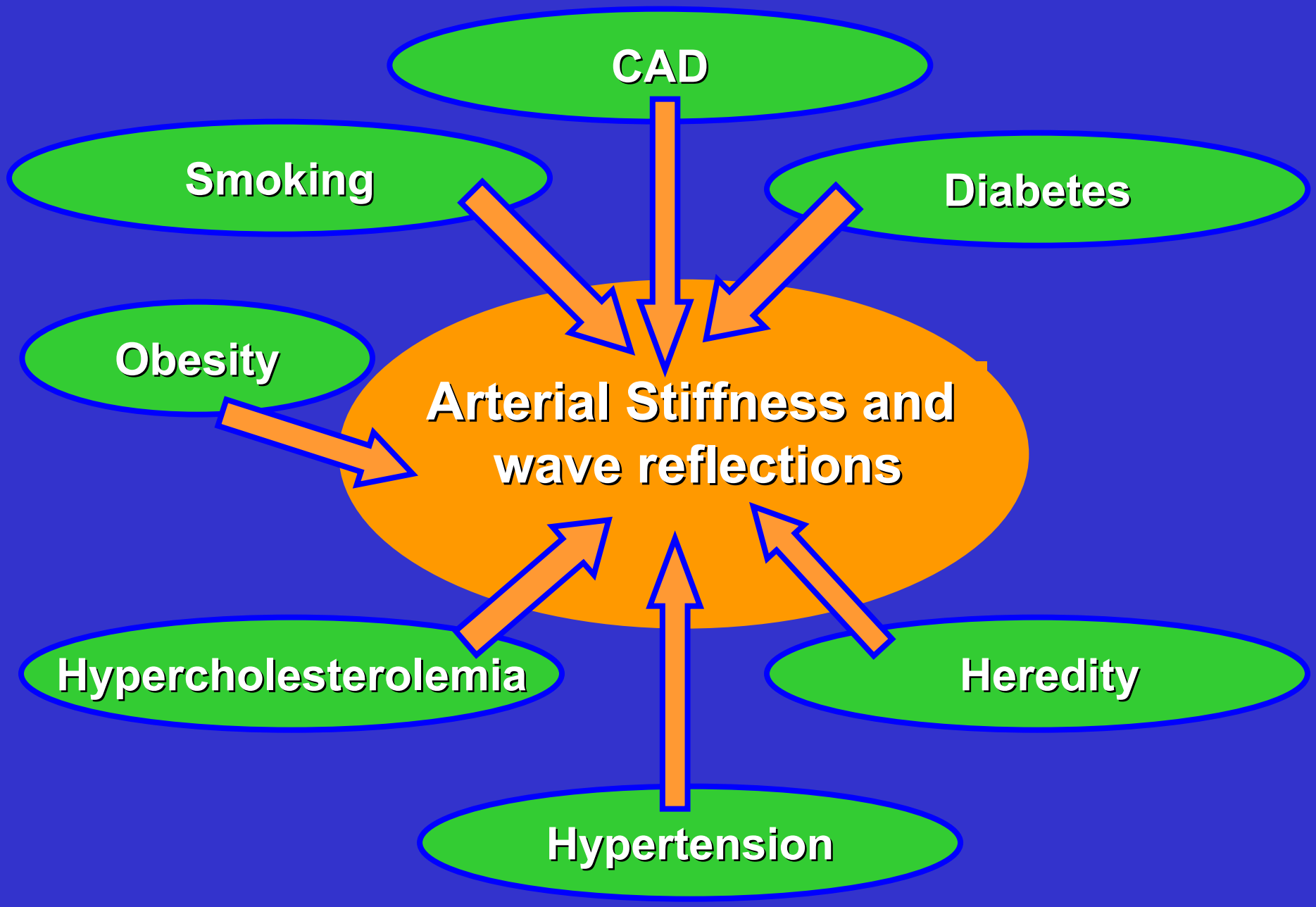
Vlachopoulos, O'Rourke. Curr Hypertens Rep 2000;2:271-9

Γενετική βάση αρτηριακής σκληρίας

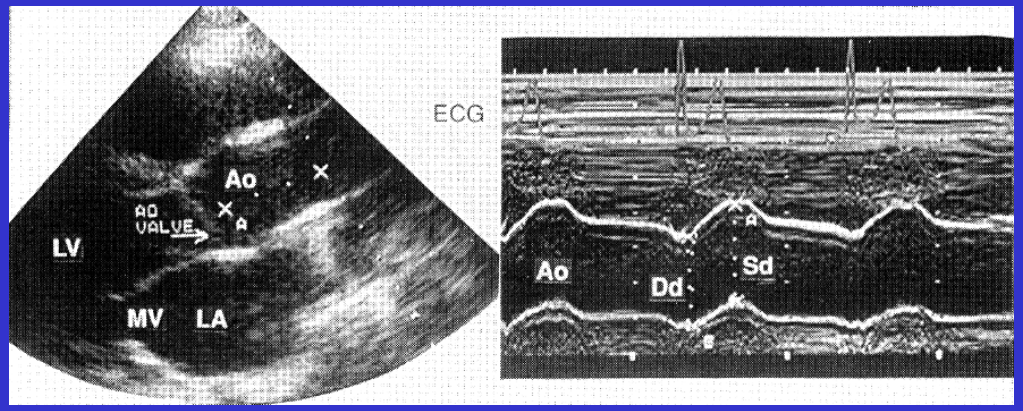
Διαφορετική έκφραση γονιδίων σε σκληρές από ότι σε ελαστικές αρτηρίες

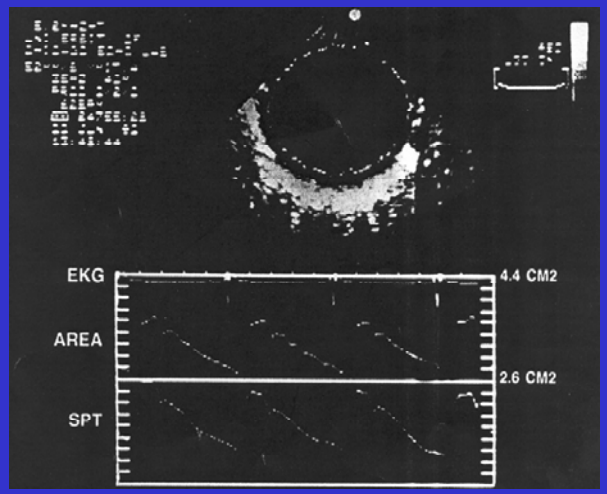


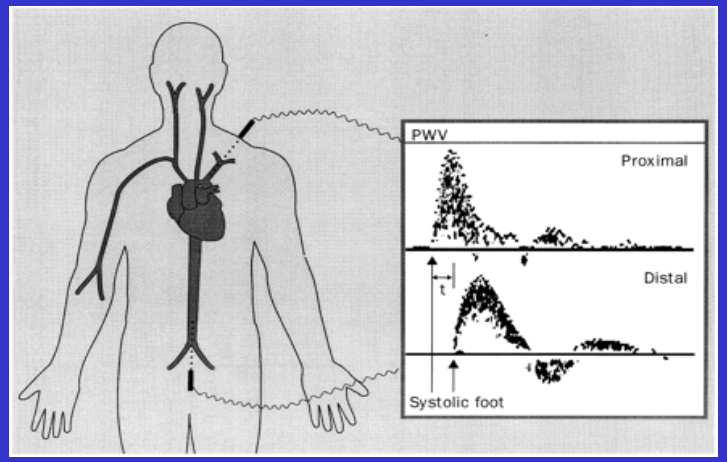
Durier S, et al. Circulation 2003

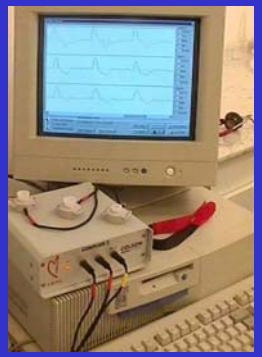

CAD
Smoking
Diabetes
Obesity
Arterial Stiffness and wave reflections
Hypercholesterolemia
Heredity
Hypertension

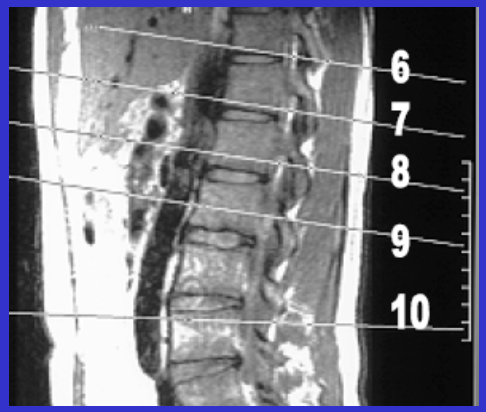
Μέθοδοι εκτίμησης αορτικής σκληρίας (τοπικής - περιοχικής)











Aortic stiffness – Why PWV ?

Consensus:

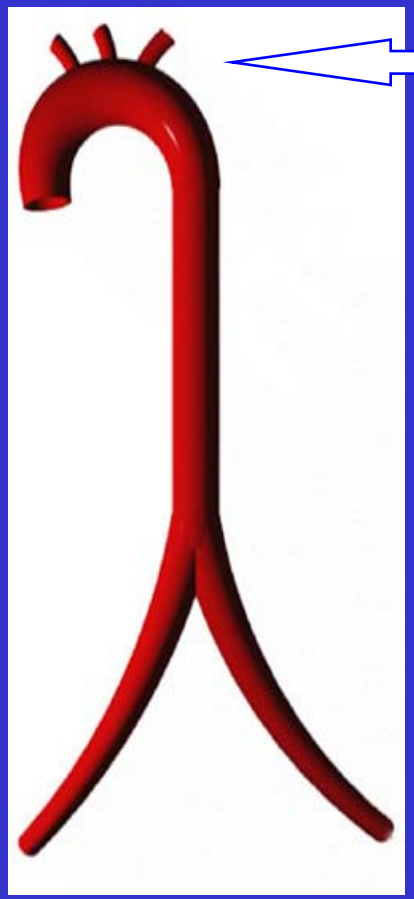
PWV is the gold standard measurement of arterial stiffness since:

- It is an independent predictor of CV morbidity and mortality in various populations
- It is a simple and reproducible method to determine arterial stiffness

Eur Heart J 2006;27:2588-2605

Μέτρηση καρωτιδομηριαίας ταχύτητας σφυγμικού κύματος

Ταχύτητα σφυγμικού κύματος



A
πιεσοϋποδοχέας

πιεσοϋποδοχέας
B

$$PWV = \frac{L\ (m)}{time\ (sec)}$$

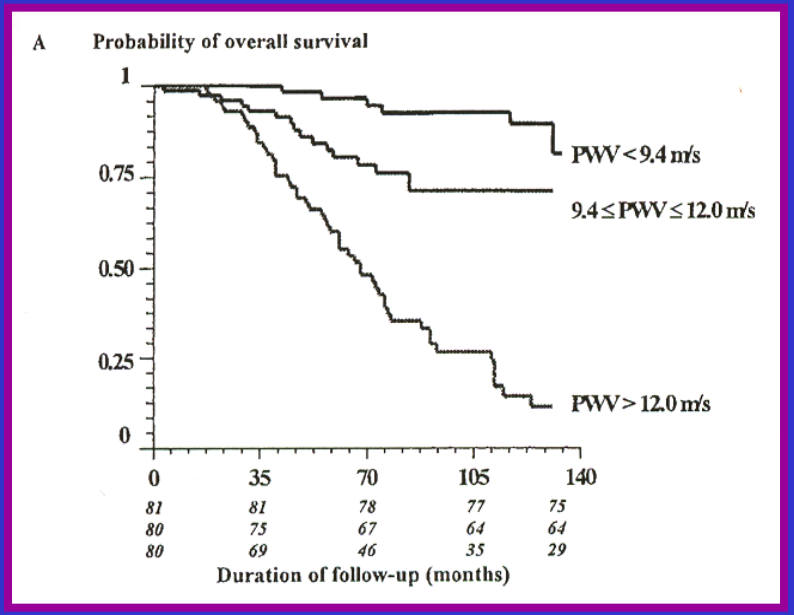
- **Εύκολη στη χρήση**
- **Αναπαραγώγιμη**
- **Σχετικά οικονομική**

Αορτική σκληρία – προγνωστική αξία

Author, year	Population-Sample size	Age (y)	Follow-up duration	Events
Blacher 1999	ESRD (N=241)	51.5±16.3	6 y	Deaths and CV deaths
Laurent 2001	Hypertension= (N=1,980)	50±13	9.3 y	Deaths and CV deaths
Meaume 2001	Subjects >70 y (N=141)	87.1±6.6	2.5 y	Deaths and CV deaths
Shoji 2001	ESRD (N=265)	55.4±10.5	5.3 y	Deaths and CV deaths
Boutouyrie 2002	Hypertension (N=1,045)	51±12	5.7 y	Coronary and CV events
Cruickshank 2002	Diabetes (N=394)	60±10	10.7 y	Deaths
Blacher 2003	ESRD (N=242)	52±16	6.5 y	Deaths and CV deaths
Laurent 2003	Hypertension (N=1,715)	51±13	7.9 y	Deaths, CV deaths and stroke deaths
Pannier 2005	ESRD (N=305)	53.1±16.2	5.8 y	CV deaths
Shokawa 2005	Ethnic minority (N=492)	63.7±8.8	10 y	Deaths and CV deaths
Sutton-Tyrrell 2005	Community based old adults (N=2,488)	73.7±2.9	4.6 y	Deaths, CV deaths and CV events
Mattace-Raso 2006	Community based adults (N=2,835)	71.7±6.7	4-9 y	Deaths and CV deaths
Willum-Hansen 2006	General population (N=1,678)	40-70 y	9.4 y	Deaths, CV deaths and CV events
Choi 2007	chest pain patients (N=497)	57.7±10.1	2.6 y	Deaths, CV deaths and CV events
Zoungas 2007	ESRD (N=207)	55±13	3.6 y	CV deaths and CV events
Terai 2008	Hypertension (N=676)	62±12	4.8 y	Deaths and CV events
Anderson 2009	Non-diabetic general population (N=174)	60±10	19.6 y	Deaths and CV events

Arterial stiffness & prognosis / ESRD

PWV



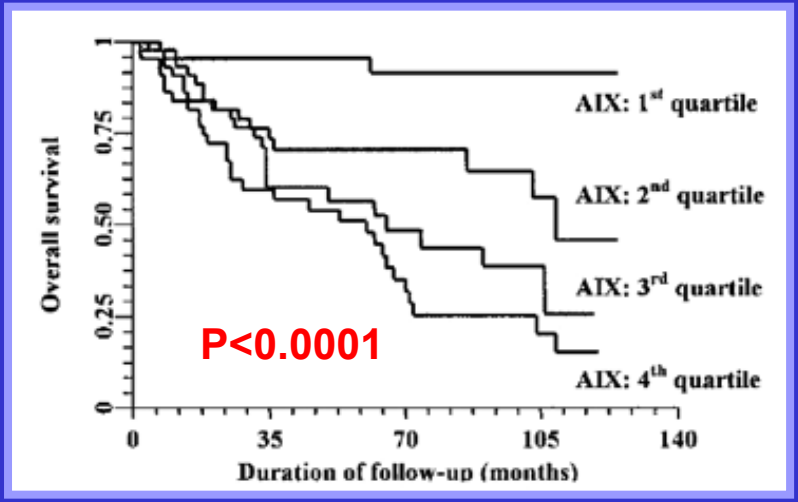
OR, all-cause mortality=5.4

PWV ↑ 1m/s ⇒ all-cause mortality ↑ 39%

N=241 pts ESRD/HD, aged 52±16 yrs.
Follow-up 72±41 mo

Blacher J, et al. *Circulation 1999*
Circulation 1998

WAVE REFLECTIONS



N=180 pts ESRD/HD, aged 54±16 yrs.
Measurements: PWV (Doppler),
AIX (applanation tonometry CA)
Follow-up 52±36 mo.

London GM, et al. *Hypertension 2001;38:434*

Arterial stiffness & prognosis / Hypertension

1.51

TABLE 5. Relative Risk of Cardiovascular Mortality According to Cardiovascular Risk Factors in Multivariate Analysis: Various Models Including PWV, PP, or SBP

Parameters	OR	Lower 95% CI	Higher 95% CI	*P*
Model 1 $CHI^2=97$				
Previous CVD, yes/no	8.33	4.33	16.02	<0.0001
Age, 10 y	1.69	1.25	2.30	<0.001
PWV, 5 m/s	1.51	1.08	2.11	0.03
Model 2 $CHI^2=95$				
Previous CVD, yes/no	8.09	4.19	15.61	<0.0001
Age, 10 y	1.72	1.27	2.34	<0.0001
PP, 10 mm Hg	1.19	0.99	1.42	0.06
Model 3 $CHI^2=96$				
Previous CVD, yes/no	8.32	4.33	16.02	<0.0001
Age, 10 y	1.82	1.36	2.45	<0.0001
SBP, 10 mm Hg	1.15	1.02	1.30	0.03

Diabetes (yes/no), included in each of the 3 models, was not significantly associated with cardiovascular mortality.

Laurent S, et al. *Hypertension 2001;37:1236*

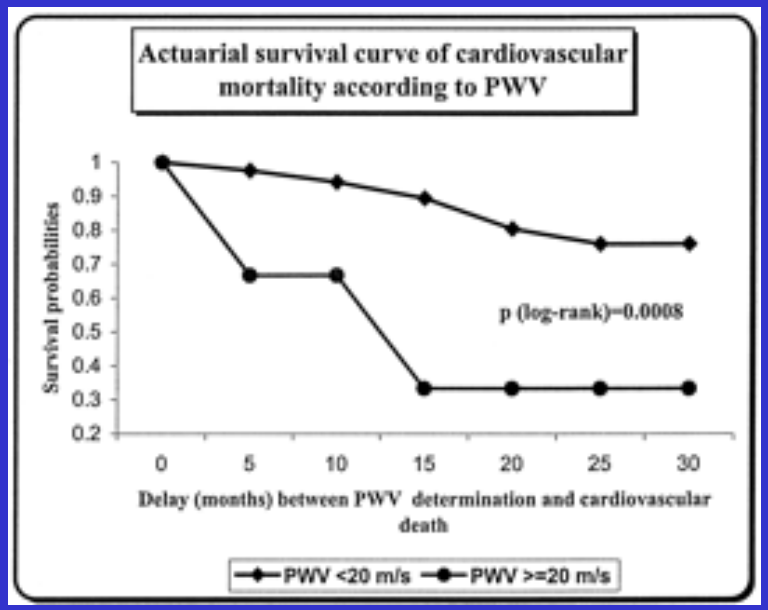
Arterial stiffness & prognosis / CAD +/- LV dysfunction

N=56 pts, CAD undergoing cath, mean age 54 yrs, LVEF 45%, Average follow-up: 3 yrs.
Endpoints:CHD events (unstable angina, AMI).

Risk of events at FU
Higher tertile: 44%
Lower tertile: 5.6%

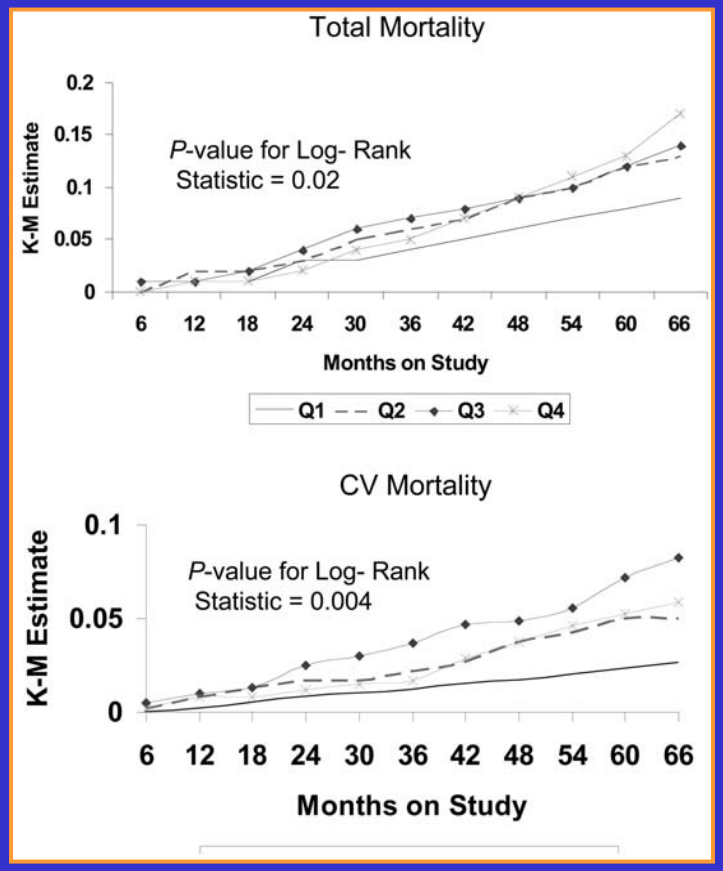
Stefanadis C et al. Eur Heart J 2000;21:390-6

Arterial stiffness & prognosis / General population – elderly



PWV ↑ 1m/s ⇒ all-cause mortality ↑ 19%

Maume S, et al. ATVB 2001

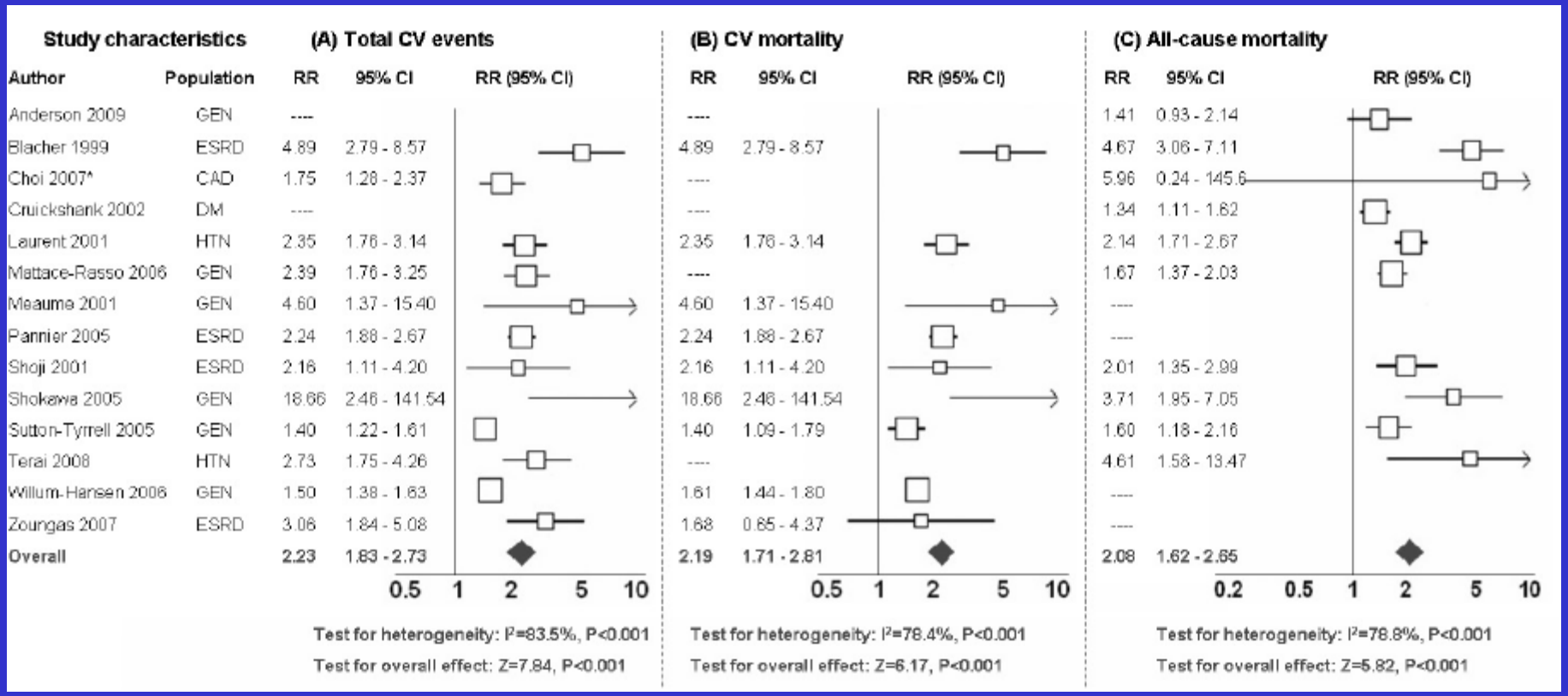


Relative Risk: 1.7

Sutton-Tyrrell K, et al. Circulation 2005

Aortic PWV as predictor of events

12,373 pts / mean FU 7.3 yrs

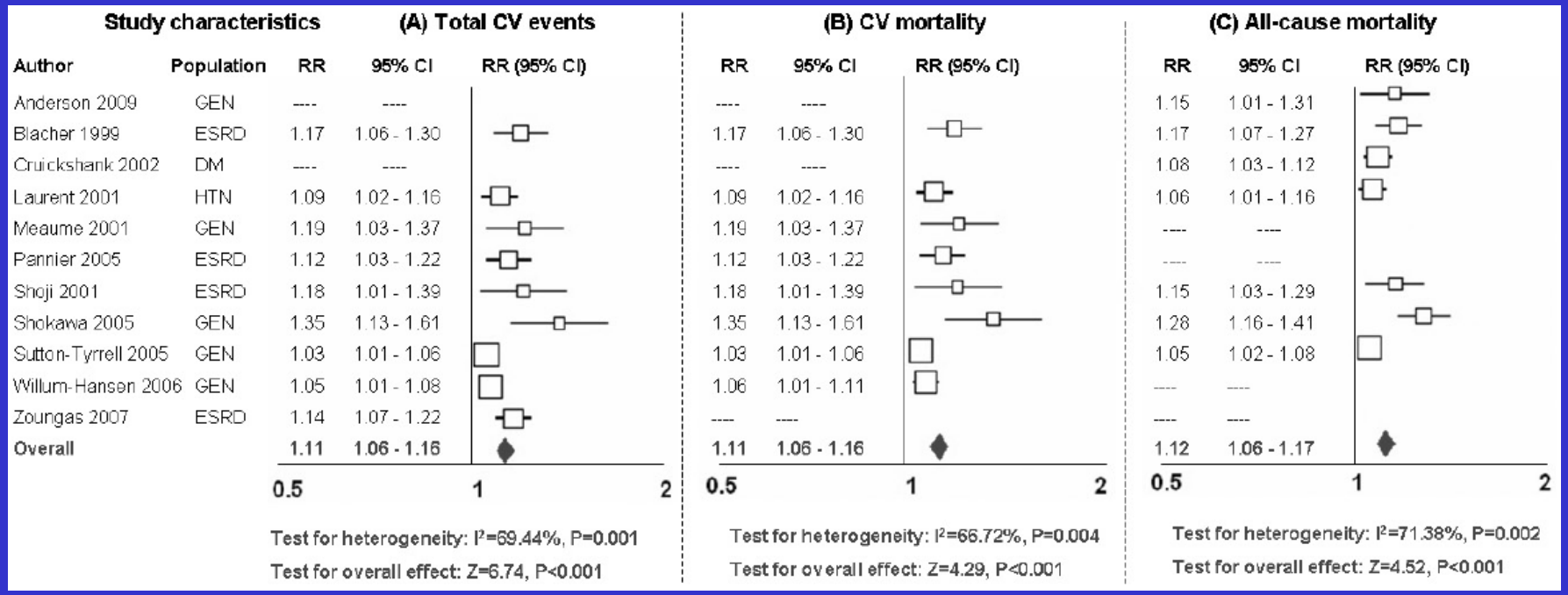


Relative Risk: 2.23 / 2.19 / 2.08

Vlachopoulos C / Aznaouridis K, and Stefanadis C. JACC 2010 (in press)

Aortic PWV as predictor of events

12,373 pts / mean FU 7.3 yrs

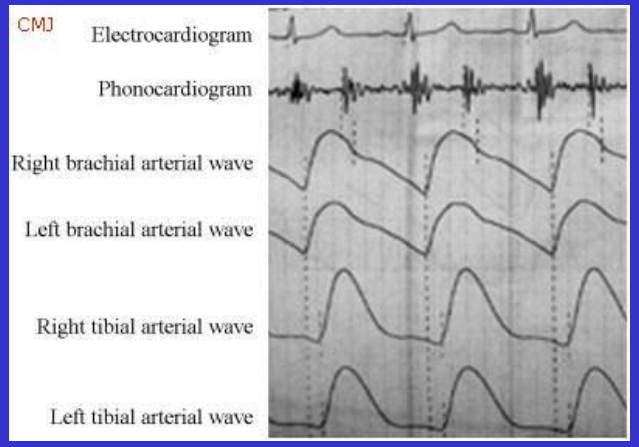


For 1 m/sec increase in PWV increase in RR: 11%

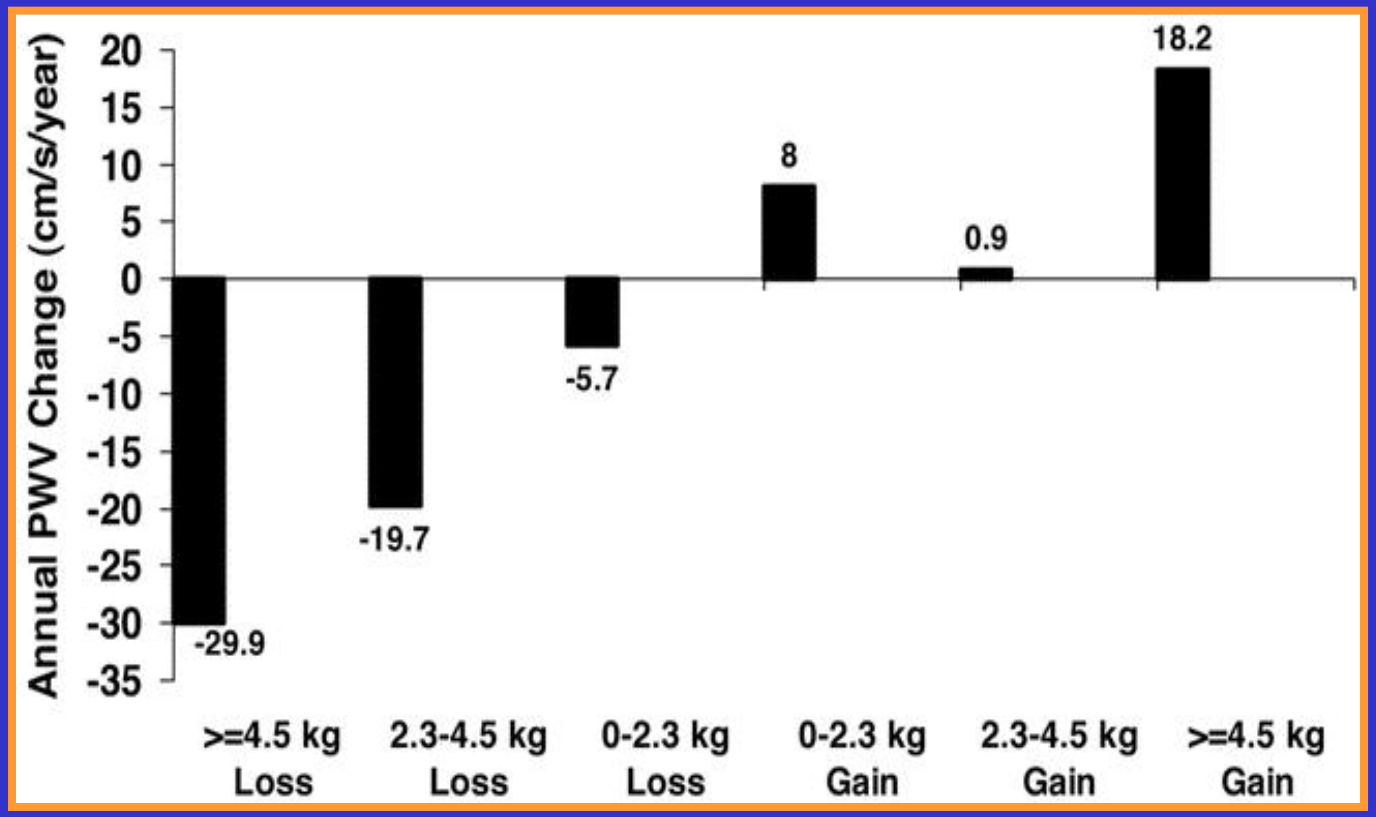
Vlachopoulos C / Aznaouridis K, and Stefanadis C. JACC 2010 (in press)

Παρακολούθηση της αντιαπόκρισης στη θεραπεία

Μεταβάλλεται η αορτική σκληρία με τη θεραπεία και η μεταβολή αυτή επηρεάζει την πρόγνωση;



Weight Loss and Arterial Stiffness



Young healthy adults

Effect controlled for BP

Larger effect on blacks

Wildman R, et al. Hypertension 2005

Απώλεια βάρους

Weight loss after bariatric surgery improves aortic elastic properties and left ventricular function in individuals with morbid obesity: a 3-year follow-up study

Ignatios Ikonomidis[a], Andreas Mazarakis[b], Costas Papadopoulos[a], Nikolaos Patsouras[b], Fotis Kalfarentzos[b], John Lekakis[a], Dimitrios T. Kremastinos[a] and Dimitrios Alexopoulos[b]

J Hypertens 2007

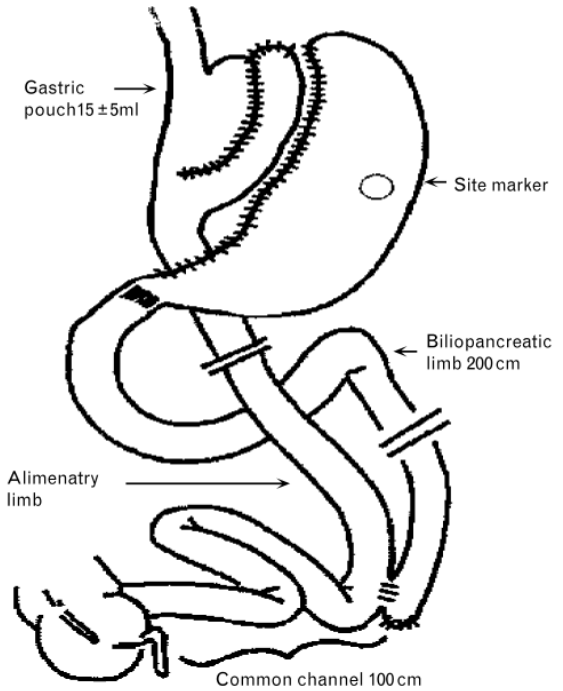
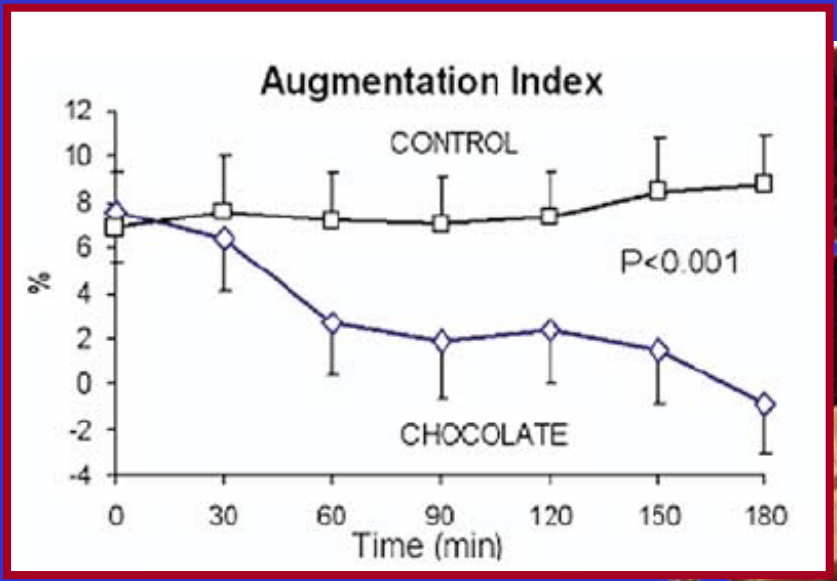


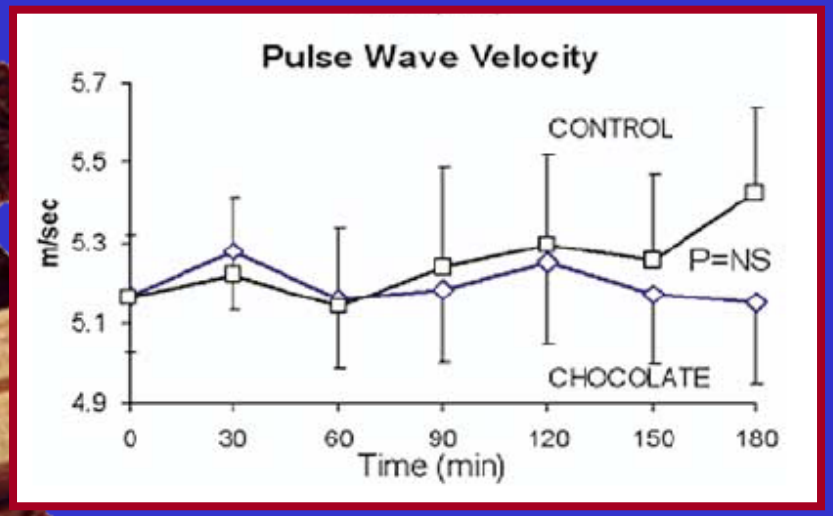
Diagram of biliopancreatic diversion with the Roux-en-Y gastric bypass procedure.

Η απώλεια βάρους μετά από εγχείρηση γαστρικού bypass συνοδεύτηκε από βελτίωση των ελαστικών ιδιοτήτων των αρτηριών

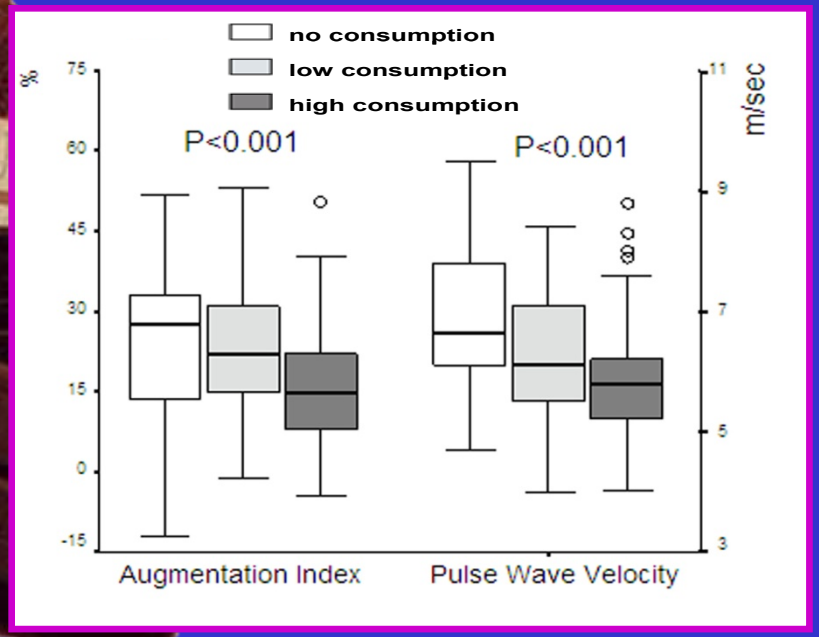
Dark Chocolate and Arterial Stiffness

Vlachopoulos C et al. Am J Hypertens 2005





100 mg dark chocolate, 74% in cocoa
acute study in healthy subjects



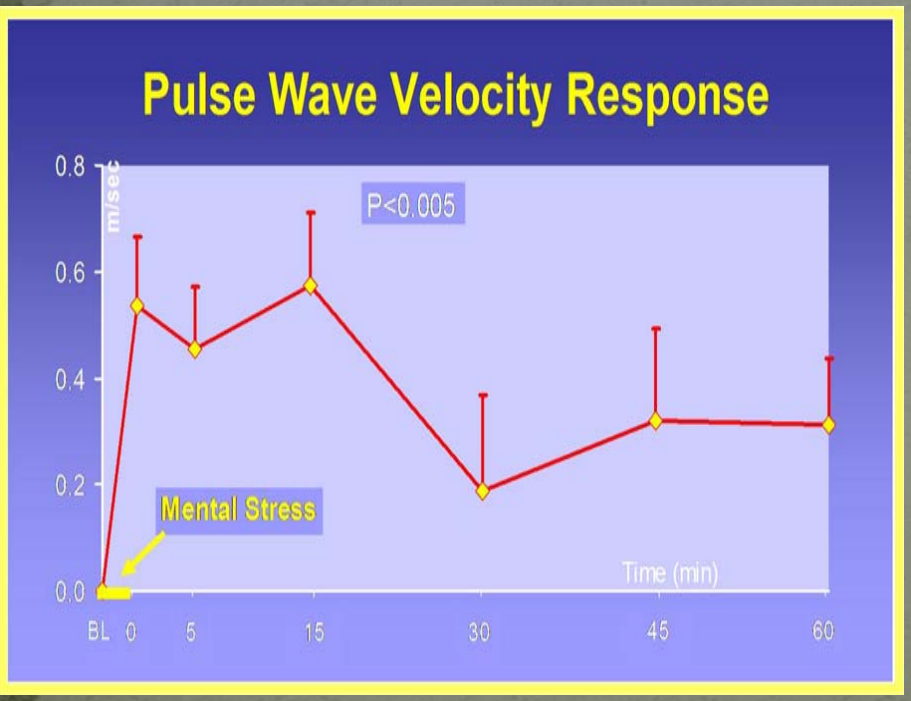
Vlachopoulos C, et al. Am J Cardiol 2007

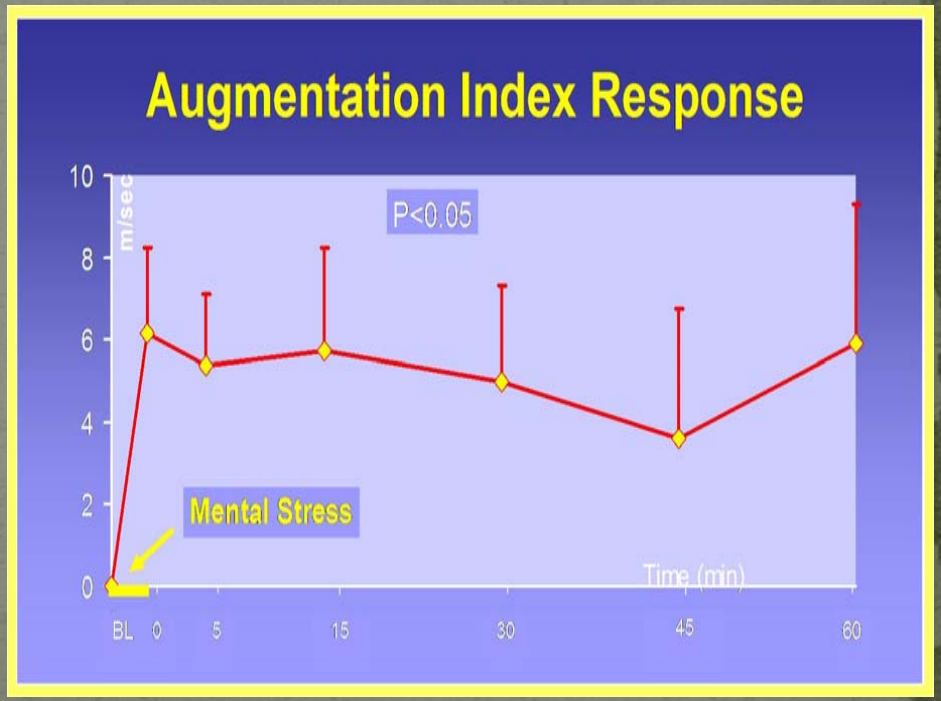
Οξύ stress και λειτουργικότητα των αρτηριών

Acute Mental Stress Has a Prolonged Unfavorable Effect on Arterial Stiffness and Wave Reflections

Charalambos Vlachopoulos, MD, Foteini Kosmopoulou, RN, Nikolaos Alexopoulos, MD, Nikolaos Ioakeimidis, MD, Gerasimos Siasos, MD, and Christodoulos Stefanadis, MD

Psychosomatic Medicine 68:231–237 (2006)





Λειτουργικότητα των αρτηριών

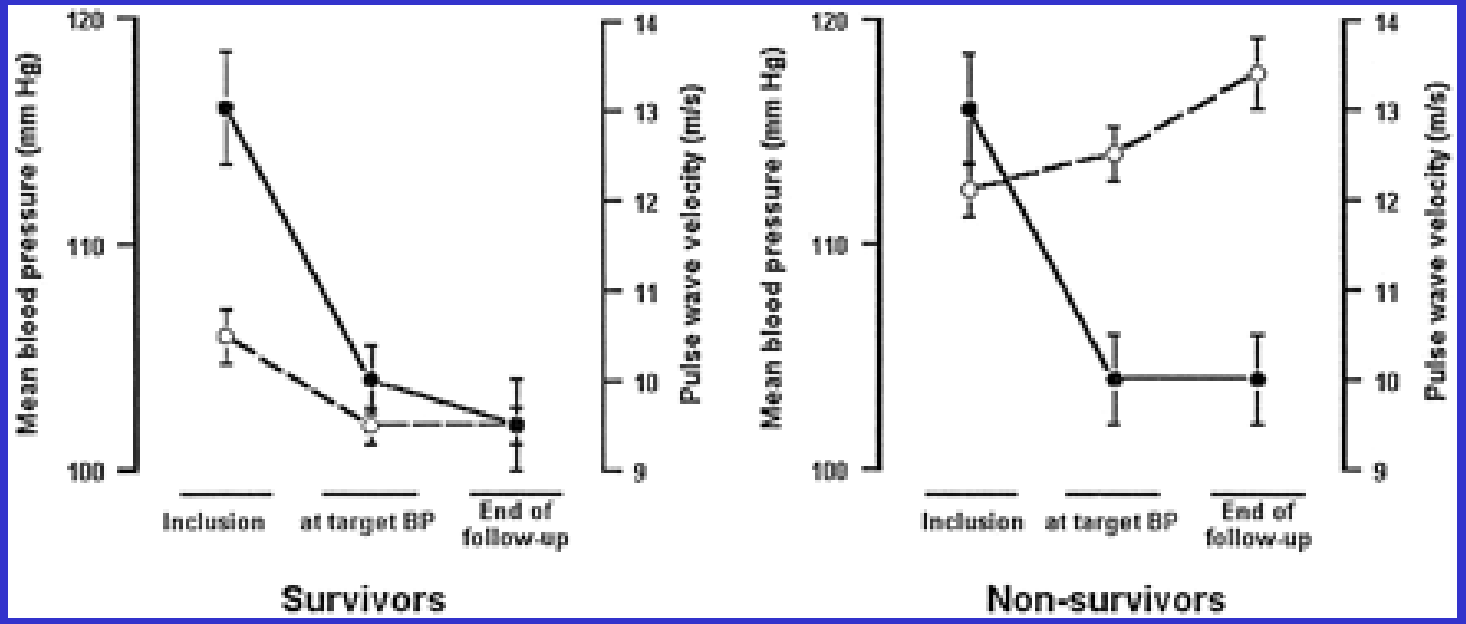
Divergent Effects of Laughter and Mental Stress on Arterial Stiffness and Central Hemodynamics

Charalambos Vlachopoulos, MD, Panagiotis Xaplanteris, MD, Nikolaos Alexopoulos, MD, Konstantinos Aznaouridis, MD, Carmen Vasiliadou, PhD, Katerina Baou, MD, Elli Stefanadi, MD, and Christodoulos Stefanadis, MD

Psychosomatic Medicine 2009

Prognostic role of PWV – changes with therapy

ESRD pts: Despite similar reduction in MBP, only those who survived reduced aortic pulse wave velocity



Guerin AP, et al. *Circulation* 2001;103:987-992

European Heart Journal
doi:10.1093/eurheartj/ehm236

2007 Guidelines for the Management of Arterial Hypertension

Table 4 Availability, prognostic value and cost of some markers of organ damage (scored from 0 to 4 pluses)

Markers	CV predictive value	Availability	Cost
Electrocardiography	++	++++	+
Echocardiography	+++	+++	++
Carotid Intima-Media Thickness	+++	+++	++
Arterial stiffness (Pulse wave velocity)	+++	+	++
Ankle-Brachial index	++	++	+
Coronary calcium content	+	+	++++
Cardiac/Vascular tissue composition	?	+	++
Circulatory collagen markers	?	+	++
Endothelial dysfunction	++	+	+++
Cerebral lacunae/White matter lesions	?	++	++++
Est. Glomerular Filtration Rate or Creatinine Clearance	+++	++++	+
Microalbuminuria	+++	++++	+

Υποκλινική βλάβη οργάνων-στόχων

- **2007** ESH/ESC Guidelines for the management of hypertension

Subclinical organ damage

- Electrocardiographic LVH (Sokolow-Lyon >38 mm; Cornell >2440 mm*ms) or:
- Echocardiographic LVH° (LVMI M ≥ 125 g/m^2, W ≥ 110 g/m^2)
- Carotid wall thickening (IMT > 0.9 mm) or plaque
- Carotid-femoral pulse wave velocity >12 m/s
- Ankle/brachial BP index <0.9
- Slight increase in plasma creatinine:
  M: 115–133 μmol/l (1.3–1.5 mg/dl);
  W: 107–124 μmol/l (1.2–1.4 mg/dl)

- Low estimated glomerular filtration rate† (<60 ml/min/1.73 m^2) or creatinine clearance◇ (<60 ml/min)
- Microalbuminuria 30–300 mg/24 h or albumin-creatinine ratio: ≥22 (M); or ≥31 (W) mg/g creatinine

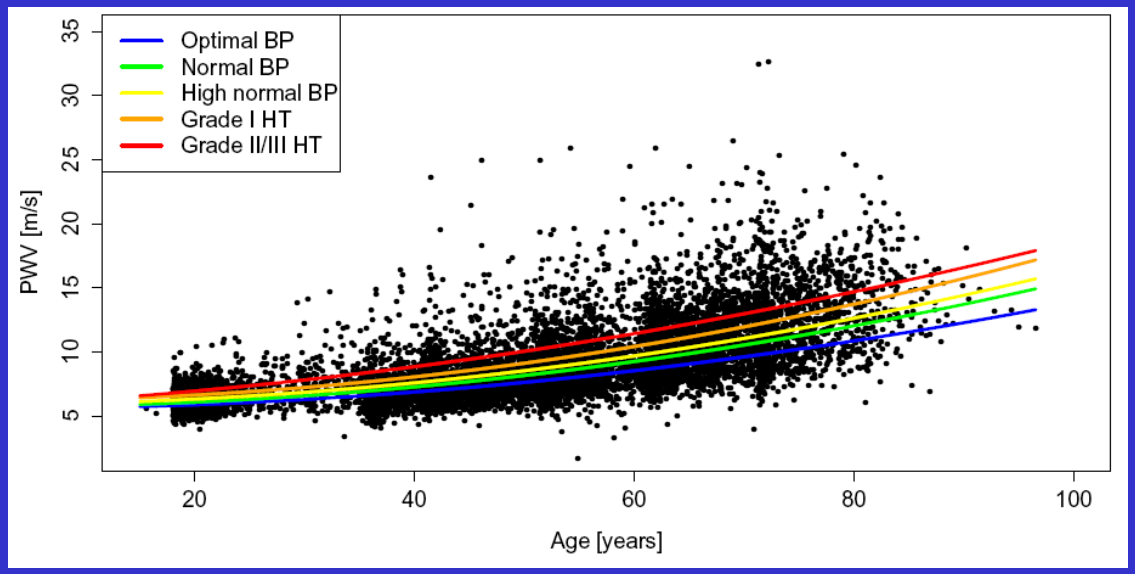
PWV: Φυσιολογικές τιμές;

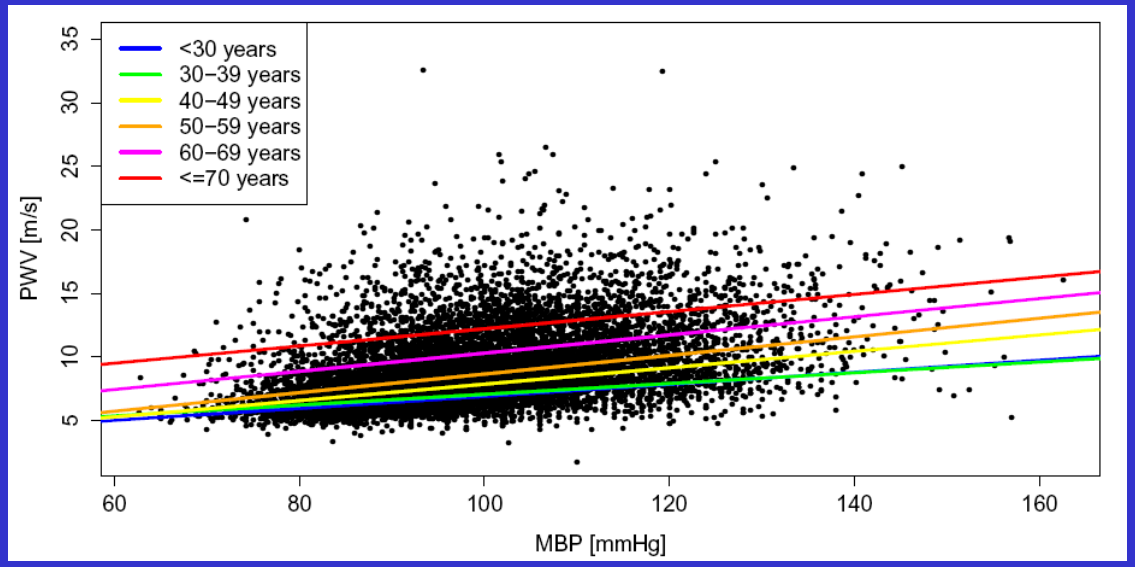
Reference values for Arterial Stiffness Collaboration

13 European Centers

24,484 (total)/ 11,092 (PWV) subjects

PWV: Φυσιολογικές τιμές;





Reference values for Arterial Stiffness Collaboration Project (under revision)

Δείκτες αγγειακής ηλικίας

Η εκφύλιση των αρτηριών που επέρχεται με την ηλικία και την επίδραση των διαφόρων παραγόντων κινδύνου έχουν ως αποτέλεσμα:

Λειτουργικές μεταβολές →
- Ενδοθηλιακή λειτουργία
- Αρτηριακή σκληρία
- Κεντρικές πιέσεις

Δομικές μεταβολές → Πάχος έσω-μέσου χιτώνα

Central Pressures and Indices

Central Blood Pressure Measurements and Antihypertensive Therapy

A Consensus Document

Enrico Agabiti-Rosei, Giuseppe Mancia, Michael F. O'Rourke, Mary J. Roman, Michel E. Safar, Harold Smulyan, Ji-Guang Wang, Ian B. Wilkinson, Bryan Williams, Charalambos Vlachopoulos

July 2007

Hypertension

Journal of the American Heart Association

American Heart Association®

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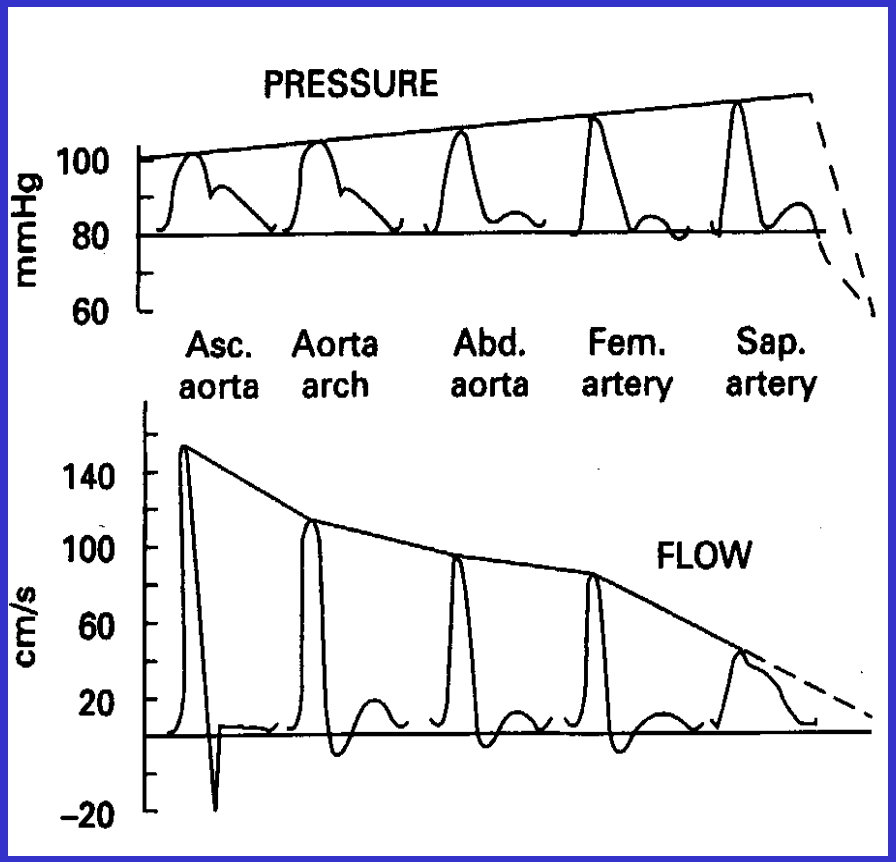
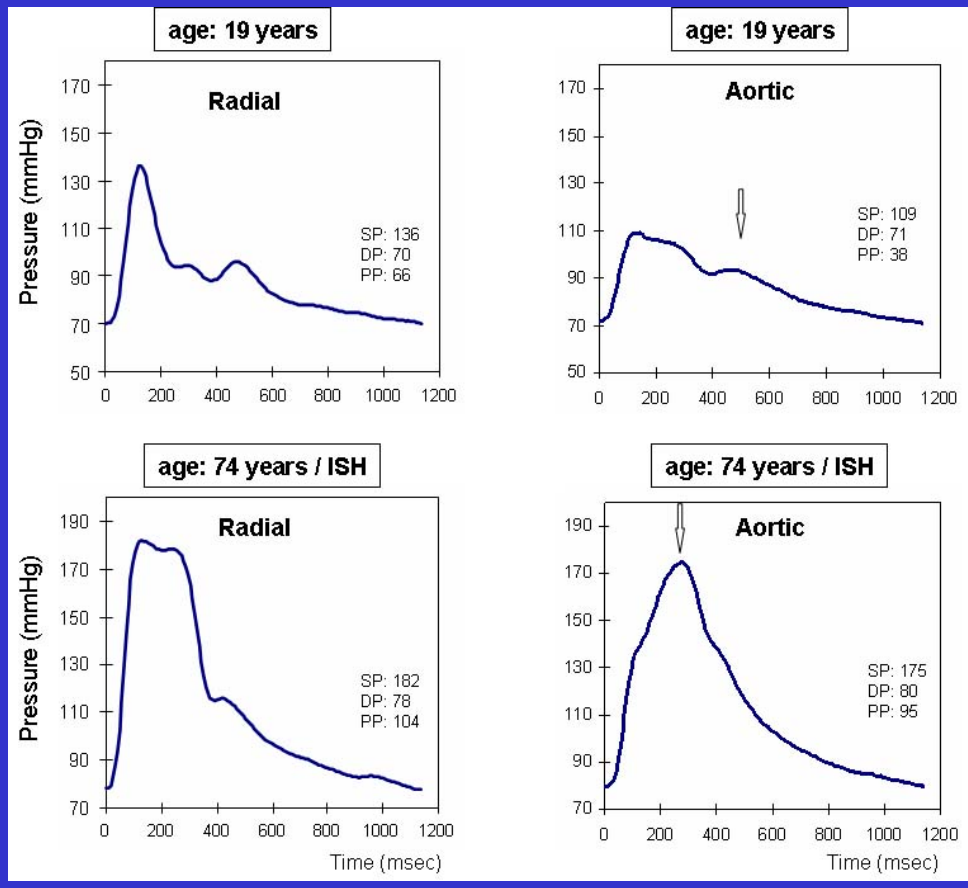
Κεντρικές πιέσεις – θεωρητικό υπόβαθρο

- amplification

- peripheral BP may overestimate central SP and PP, especially in young subjects





CBPs: theoretical basis

- Aortic systolic BP=LV systolic P

  reflects LV afterload

- Aortic diastolic BP throughout diastole

  determines coronary filling

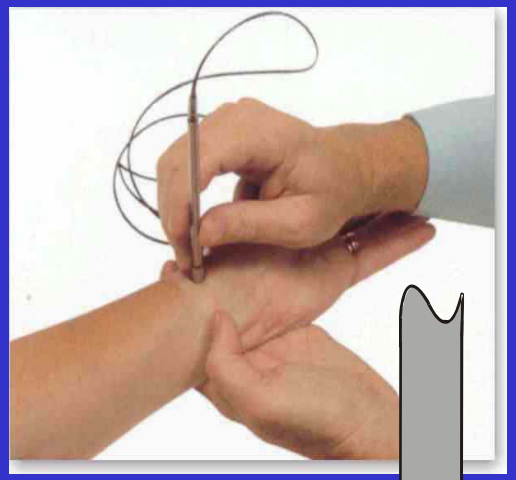
- Elastic-type arteries (aorta-carotids)

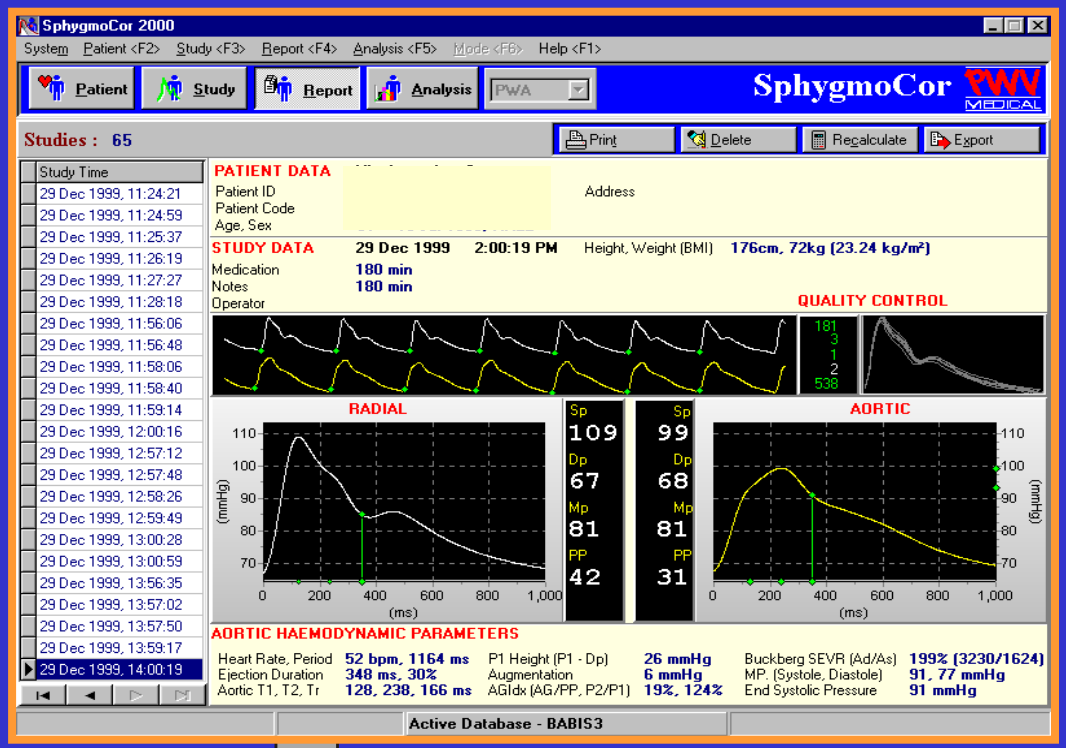
  degenerate with aging and hypertension

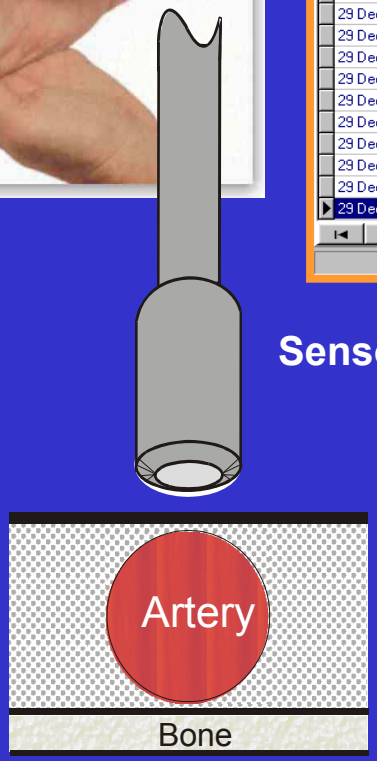
Central BPs are physiologically more relevant than peripheral BPs to the pathogenesis of CV disease

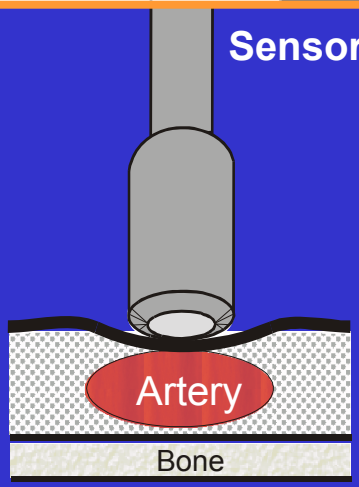
Μέτρηση κεντρικών πιέσεων

Sphygmocor









Οι κεντρικές πιέσεις ως δείκτες νόσου

Cross-sectional and longitudinal studies

First author	Year, country	Population	Design	Parameter	End-point
Saba*†	1993, USA	Normotensives	Cross-sectional	Carotid AIx	LVMI, carotid thickness
Boutouyrie*†	1999, France	Hypertensives	Cross-sectional	Carotid PP	Carotid thickness
Boutouyrie*†	2000, France	Hypertensives	Longitudinal (9-month FU)	Carotid PP	Carotid IMT reduction with treatment
Roman	2000, USA	Normotensives, Hypertensives	Cross-sectional	Carotid systolic BP	Relative LV wall thickness
Waddell*†	2001, Australia	CAD	Cross-sectional	Carotid BP	Extent of CAD
Nishijima*	2001, Japan	Suspected CAD	Cross-sectional	Aortic fractional PP	Incident CAD
Nurnberger	2002, Germany	Healthy + CVD	Cross-sectional	Carotid AIx	CV risk scores
Philippe*	2002, France	CAD	Cross-sectional	Aortic PP	Extent of CAD
Hayashi*	2002, Japan	Suspected CAD	Cross-sectional	Aortic AIx	Incident CAD
De Luca †	2004, REASON study	Hypertensives	Longitudinal (1-year FU)	Carotid PP	LVMI reduction
Weber	2004, Austria	Suspected CAD	Cross-sectional	Aortic AP, AIx	Incident CAD
Jankowski*	2004, Poland	CAD	Cross-sectional	Aortic BP	Extent of CAD
Danchin*	2004, France	Suspected CAD	Cross-sectional	Aortic PP	Incidence and extent of CAD
Booth	2004, UK	Systemic vasculitis	Cross-sectional	Aortic AIx	Disease activity
Roman	2007, USA	High-risk pts	Cross-sectional	Aortic PP	Carotid IMT and mass
Hashimoto †	2007, Japan	Hypertensives	Longitudinal (1-year FU)	Aortic AIx	LVMI reduction with treatment
Dart	2007, Australia	Eledrly hypertensives	Longitudinal (4-year FU)	Carotid/Brachial BP	BP with treatment
Jiang	2007, China	Hypertensives	Longitudinal (2-mo FU)	Central SP	BP with treatment

* Measured invasively

Incremental value

Κεντρικές πιέσεις και πρόγνωση

Author, year	Population-Sample size	Age (y)	Follow-up duration (longitudinal studies)	Events	Index
Lu 2001*	Stable CAD /angioplasty (N=87)	72.5±5.1	6.1±4.1m	Restenosis	Aortic PP, PPf and PI
London 2001	ESRD (N=180)	54±16	52±36m	Deaths and CV deaths	Carotid AIx
Safar 2002	ESRD (N=180)	54±16	52±36m	Deaths and CV deaths	carotid SBP, PP, brachial-carotid PP amplification
Ueda 2004*	CAD / angioplasty (N=103)	62±9	6m	Restenosis	Aortic AIx, Aortic inflection time
Chirinos 2005*	CAD or non-obstructive coronary atherosclerosis (N=297)	63.8±10.3	40±14m	Deaths and CV events	Aortic PP, AP, AIx
Weber 2005	CAD / angioplasty (N=262)	65±10	24m	Deaths and CV events	HR- corrected aortic AIx
Dart 2006	elderly female hypertensives (N=484)	72±5	49m (median)	CV events	carotid SBP, PP AIx
Covic 2006	ESRD (N=92)	42.6±11.2	61±25m	Deaths	HR- corrected aortic AIx
Roman 2007	American Indians free of CVD (N=2,403)	63.5±7.5	58±16m	Deaths, CV deaths and CV events	Aortic SBP, PP
Jankowski 2008*	Subjects undergoing non-emergency coronary angiography (N=1,109)	52.7±19.2	52.7±19.2m	Deaths, CV deaths and CV events	Aortic PP PPf
Pini 2008	Community-dwelling individuals ≥ 65 y (N=398)	73±6	94±24m	Deaths, CV deaths and CV events	Aortic SBP, PP, AIx
Wang 2009	Unselected chinese community 30-74 y (N=1272)	51±12	120m	All cause deaths, CV deaths, LVMI, IMT, eGFR	Aortic SBP, PP, AIx

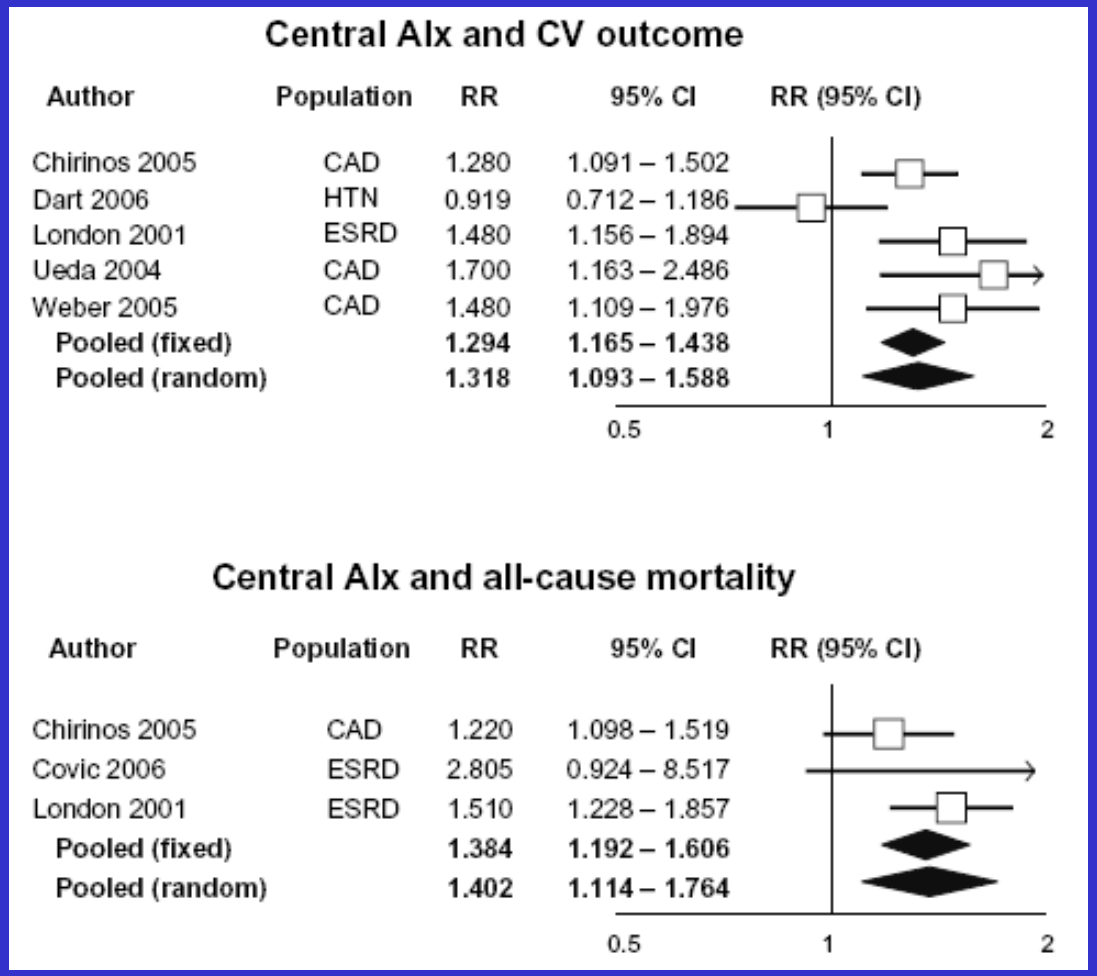
* Measured invasively

Negative value

Incremental value

CBPs/indices as predictors of events

5,648 pts / mean FU 45 months

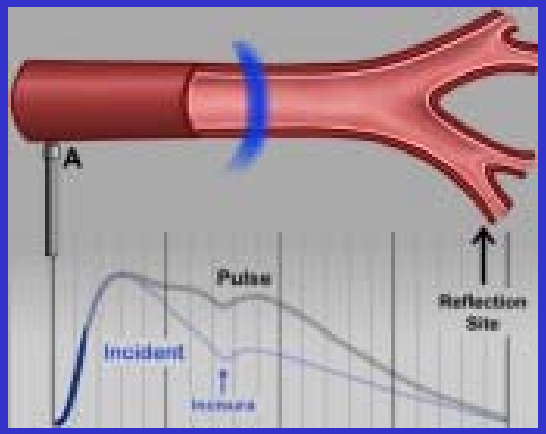


For 10% increase in AIx increase in RR: 38%

Vlachopoulos C / Aznaouridis K, et al. *Eur Heart J 2010*

Παρακολούθηση της αντιαπόκρισης στη θεραπεία

Μεταβάλλεται η αορτική σκληρία με τη θεραπεία και η μεταβολή αυτή επηρεάζει την πρόγνωση;



ASCOT endpoints

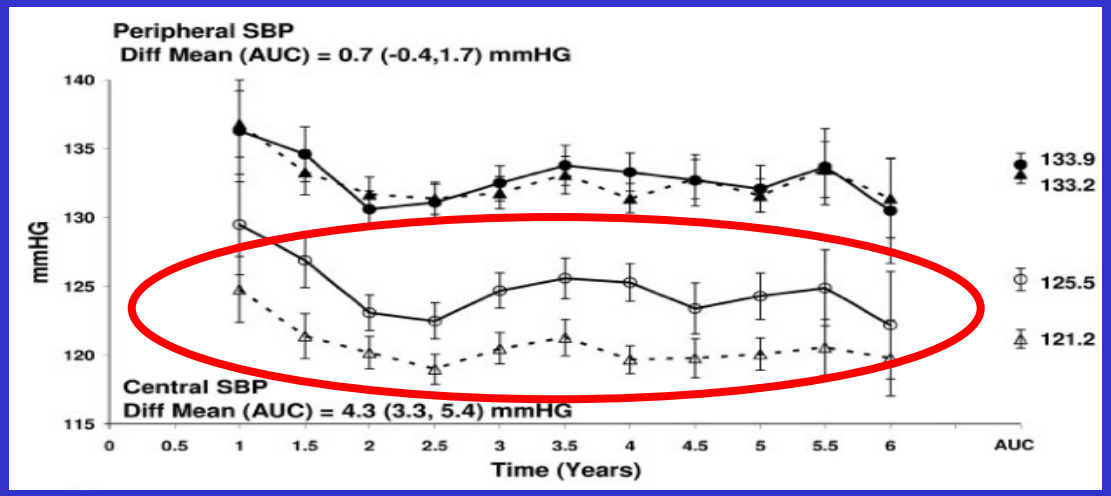
	ΣK (95% CI)
Primary	
Non-fatal MI (incl silent) + fatal CHD	0.90 (0.79-1.02)
Secondary	
Non-fatal MI (exc. Silent) +fatal CHD	0.87 (0.76-1.00)
Total coronary end point	0.87 (0.79-0.96)
Total CV event and procedures	0.84 (0.78-0.90)
All-cause mortality	0.89 (0.81-0.99)
Cardiovascular mortality	0.76 (0.65-0.90)
Fatal and non-fatal stroke	0.77 (0.66-0.89)
Fatal and non-fatal heart failure	0.84 (0.66-1.05)
Tertiary	
Silent MI	1.27 (0.80-2.00)
Unstable angina	0.68 (0.51-0.92)
Chronic stable angina	0.98 (0.81-1.19)
Peripheral arterial disease	0.65 (0.52-0.81)
Life-threatening arrhythmias	1.07 (0.62-1.85)
New-onset diabetes mellitus	0.70 (0.63-.078)
New-onset renal impairment	0.85 (0.75-0.97)
Post hoc	
Primary end point+coronary revasc procs	0.86 (0.77-0.96)
CV death + MI + stroke	0.84 (0.76-0.92)

Amlodipine ± perindopril better **Atenolol ± thiazide better**

The area of the yellow square is proportional to the amount of statistical information

Παρακολούθηση της αντιαπόκρισης στη θεραπεία

CAFÉ Study



- BP-lowering drugs can have substantially different effects on central aortic pressures and hemodynamics despite a similar impact on brachial BP

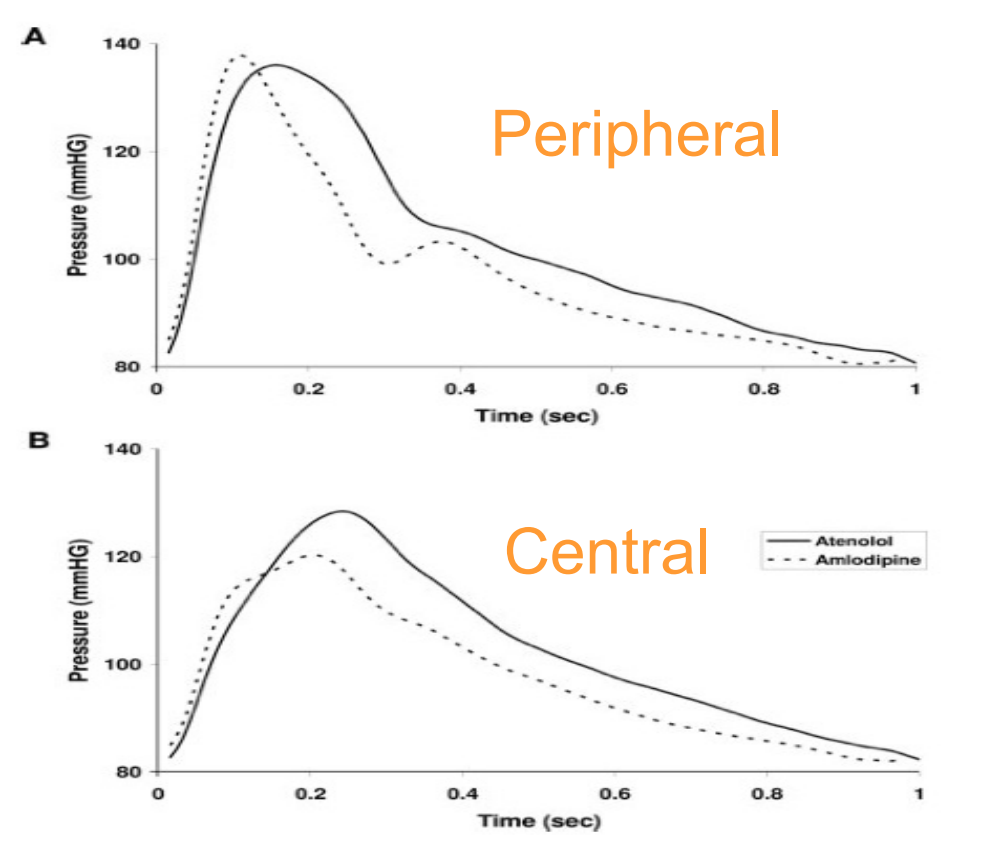
- Central aortic pressure is an independent determinant of outcome

- Central aortic pressure may comprise a treatment target

Williams B, et al. Circulation 2006

Παρακολούθηση της αντιπόκρισης στη θεραπεία

CAFÉ Study



Williams B, et al. Circulation 2006

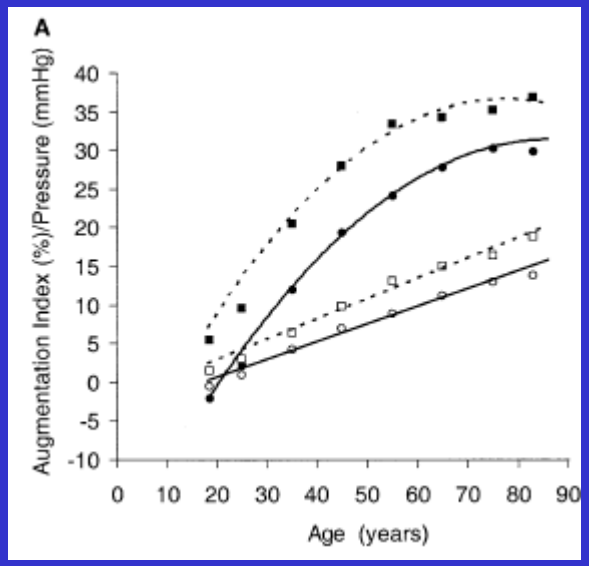
Κεντρικές πιέσεις – ανακλώμενα κύματα: φυσιολογικές τιμές

Need for reference values/cut-offs

Published:

ACCT 2005

EPOGH 2006



Ongoing:

European Network on Large Artery Investigation

NIA

Framingham

CBPs: Where do we stand today?

European Heart Journal
doi:10.1093/eurheartj/ehm236

ESC and ESH Guidelines

2007 Guidelines for the Management of Arterial Hypertension

The Task Force for the Management of Arterial Hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC)

3.1.7 Central blood pressure

Due to the variable superimposition of incoming and reflected pressure waves along the arterial tree, aortic systolic and pulse pressure (i.e. the pressure exerted at the level of the heart, brain and kidney) may be different from the conventionally measured brachial pressure.[162] Furthermore, the claim has long been made that peripheral and central systolic and pulse pressures may be differently affected by antihypertensive drugs.[163] The need for invasive measurement of central blood pressure has confined this issue to research. However, recently a method has been described to non-invasively estimate aortic blood pressure by calculating the 'augmentation index' from the pulse wave pressure contour recorded from a peripheral artery.[164,165] Use of this method has confirmed that the effects of antihypertensive drugs on central systolic and pulse pressure do not invariably reflect those seen at the brachial artery level.[166,167] Furthermore, the results obtained in a large substudy performed within a randomized trial have shown that central pulse pressure as assessed from the 'augmentation index' is significantly related to cardiovascular events.[166] However, the prognostic role of central as opposed to peripheral blood pressure needs to be further confirmed in more large-scale observational and interventional studies.

" Though a wider use of PWV and AIx measurements may add further precision to the assessment of arterial damage, the availability of these techniques is largely limited to research centres."

CBPs: Where do we stand today?

Central Blood Pressure Measurements and Antihypertensive Therapy

A Consensus Document

Enrico Agabiti-Rosei, Giuseppe Mancia, Michael F. O'Rourke, Mary J. Roman, Michel E. Safar, Harold Smulyan, Ji-Guang Wang, Ian B. Wilkinson, Bryan Williams, Charalambos Vlachopoulos

Hypertension
Journal of the American Heart Association
American Heart Association
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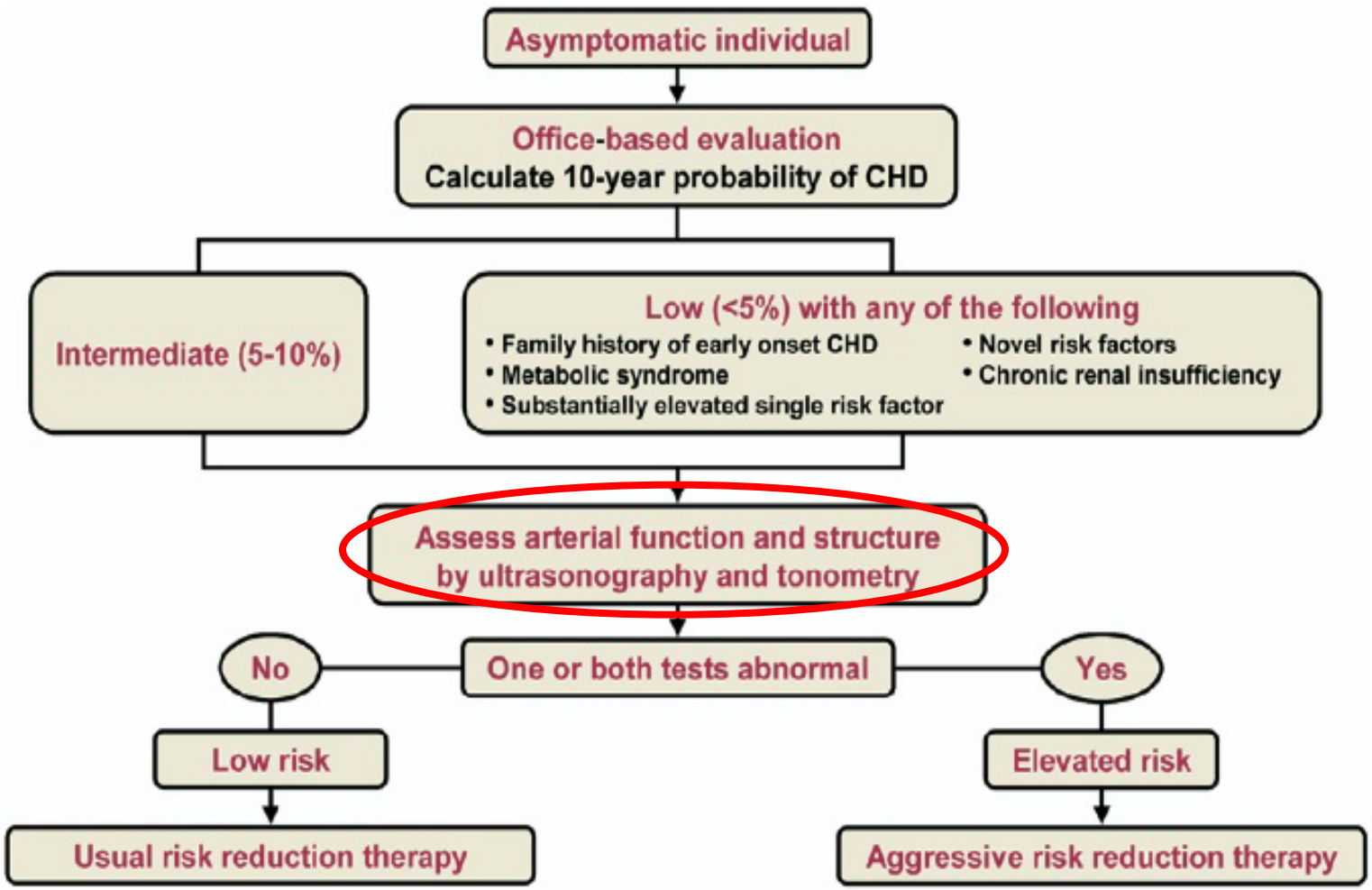
July 2007

is desirable. Inclusion of a parameter in patient assessment and management should serve various purposes, such as advancement of science, physician education, and practicality of use in a range of settings, whereas cost should also be taken into consideration. Assessment of central pressures meets these criteria to a varying degree at present. *Definition of terms such as central and peripheral BP, arterial stiffness, wave reflections, and systolic BP and PP amplification should be readily available to both clinicians and researchers and introduced in the guidelines on hypertension and cardiovascular risk.* Although brachial BP remains our point of reference, there is a definite sense that efforts to investigate

Arterial Stiffness: clinical tool or research toy?



Kullo and Malik. JACC 2007

Stiffness in clinical practice

Blood pressure (mmHg)					
Other risk factors, OD or Disease	Normal SBP 120–129 or DBP 80–84	High normal SBP 130–139 or DBP 85–89	Grade 1 HT SBP 140–159 or DBP 90–99	Grade 2 HT SBP 160–179 or DBP 100–109	Grade 3 HT SBP ≥180 or DBP ≥110
No other risk factors	Average risk	Average risk	Low added risk	Moderate added risk	High added risk
1–2 risk factors	Low added risk	Low added risk	Moderate added risk	Moderate added risk	Very high added risk
3 or more risk factors, MS, OD or Diabetes	Moderate added risk	High added risk	High added risk	High added risk	Very high added risk
Established CV or renal disease	Very high added risk	Very high added risk	Very high added risk	Very high added risk	Very high added risk

- 2007 ESH/ESC Guidelines for the management of hypertension

Stiffness in clinical practice

Blood pressure (mmHg)					
Other risk factors OD or disease	Normal SBP 120–129 or DBP 80–84	High normal SBP 130–139 or DBP 85–89	Grade 1 HT SBP 140–159 or DBP 90–99	Grade 2 HT SBP 160–179 or DBP 100–109	Grade 3 HT SBP≥180 or DBP≥110
No other risk factors	No BP intervention	No BP intervention	Lifestyle changes for several months then drug treatment if BP uncontrolled	Lifestyle changes for several weeks then drug treatment if BP uncontrolled	Lifestyle changes + Immediate drug treatment
1–2 risk factors	Lifestyle changes	Lifestyle changes	Lifestyle changes for several weeks then drug treatment if BP uncontrolled	Lifestyle changes for several weeks then drug treatment if BP uncontrolled	Lifestyle changes + Immediate drug treatment
≥3 risk factors, MS or OD	Lifestyle changes	Lifestyle changes and consider drug treatment	Lifestyle changes + Drug treatment	Lifestyle changes + Drug treatment	Lifestyle changes + Immediate drug treatment
Diabetes	Lifestyle changes	Lifestyle changes + Drug treatment			
Established CV or renal disease	Lifestyle changes + Immediate drug treatment	Lifestyle changes + Immediate drug treatment	Lifestyle changes + Immediate drug treatment	Lifestyle changes + Immediate drug treatment	Lifestyle changes + Immediate drug treatment

- 2007 ESH/ESC Guidelines for the management of hypertension