Theoretical and practical questions in the evaluation of arterial function

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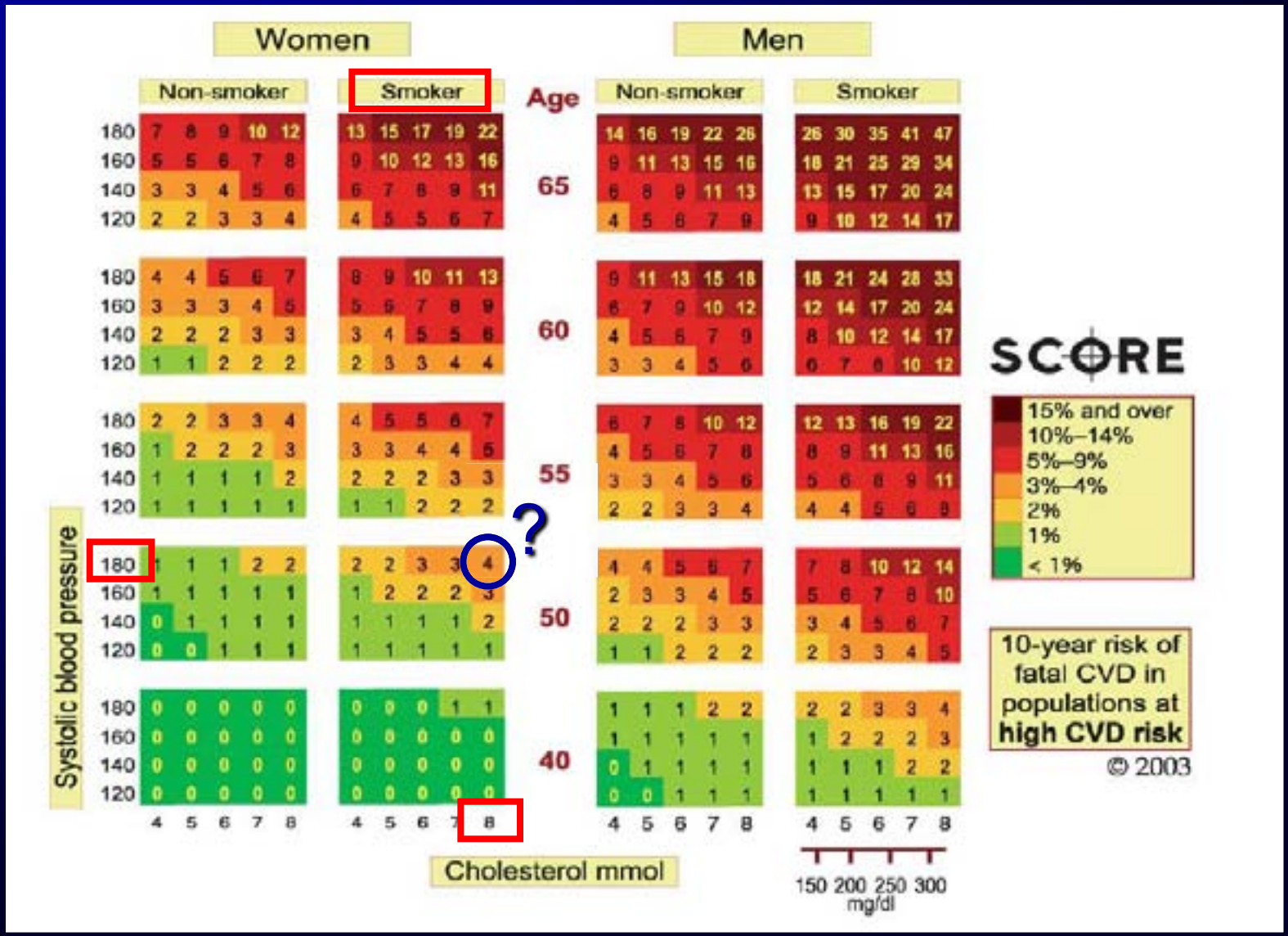
WHY DO WE HAVE TO EVALUATE THE ARTERIAL FUNCTION?

LIMITATIONS OF CV RISK SCORING



Limitations of traditional risk scores (SCORE, Framingham)

- They do not consider several established risk factors (obesity, diabetes, history of premature CHD in first degree relatives, lack of regular physical exercise, psychosocial factors, etc.)
- They are mainly focused on fatal and non-fatal CHD, however ischemic stroke is also a major cause of deaths and disability
- They do not consider the ethnicity
- More than 80% of the global CVD burden is in low-income and middle-income countries where the value of risk factors has not yet been established

Simon, A. and Levenson, J.:
*May **subclinical** arterial disease help to better detect and treat high-risk asymptomatic individuals?*
J Hypertens 2005, 23: 1939-1945

„....although traditional risk factors may account for 90% of the attributable cardiovascular risk, their prediction of CVD is weak probably because the susceptibility to CVD varies greatly among individuals."

„The detection of high CVD risk based on traditional risk factors alone probably fails to diagnose a number of high-risk conditions and therefore measurement of new potential risk factors has been proposed to improve the detection of high-risk asymptomatic subjects."

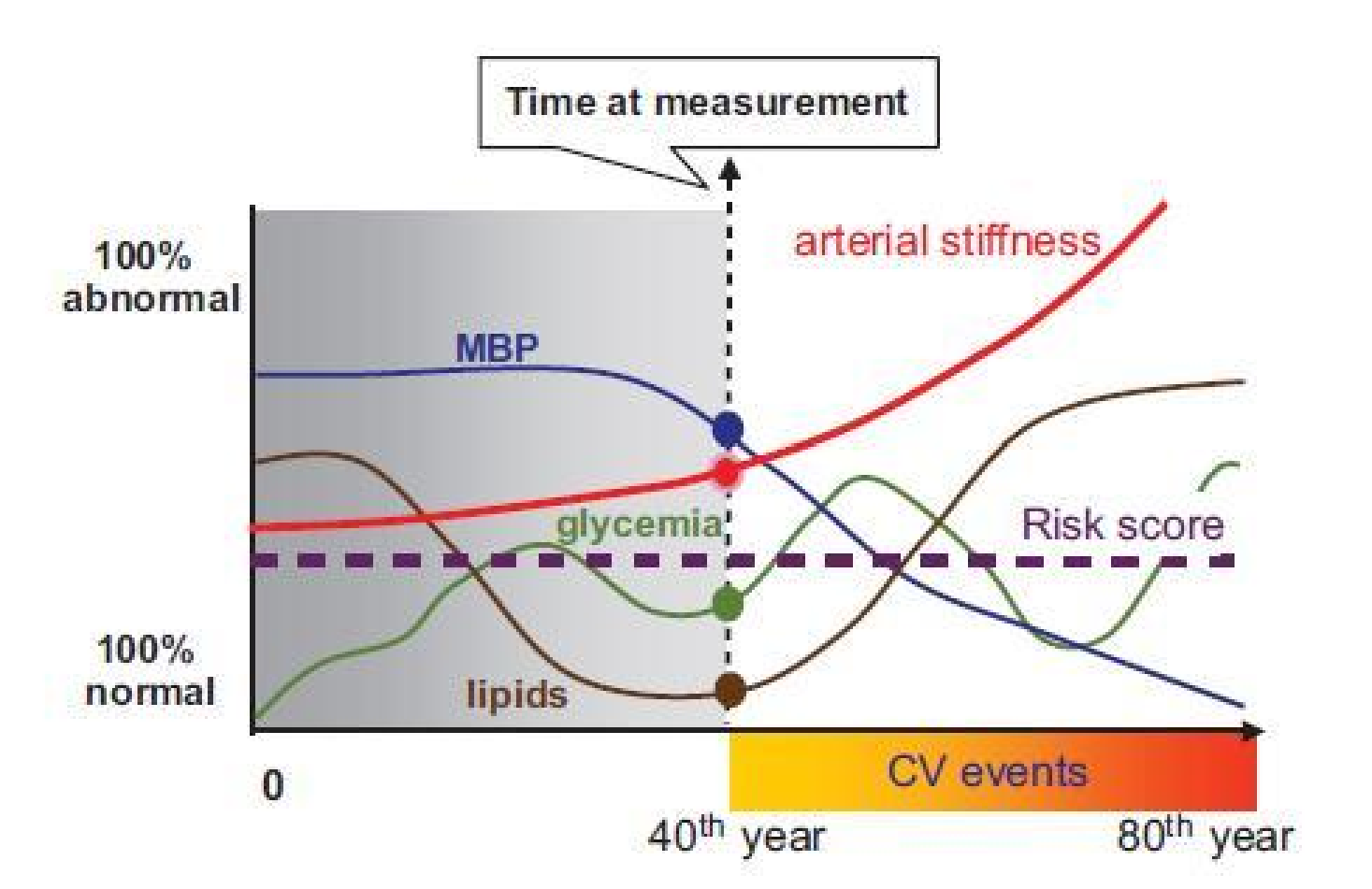
Simon, A. and Levenson, J.:
May subclinical arterial disease help to better detect and treat high-risk asymptomatic individuals?
J Hypertens 2005, 23: 1939-1945

„….vascular stiffness precedes atherosclerosis and is a risk factor for atherosclerosis.

…….studies clearly demonstrate that **PWV** and the **augmentation index** are associated with the structural changes of atherosclerosis”

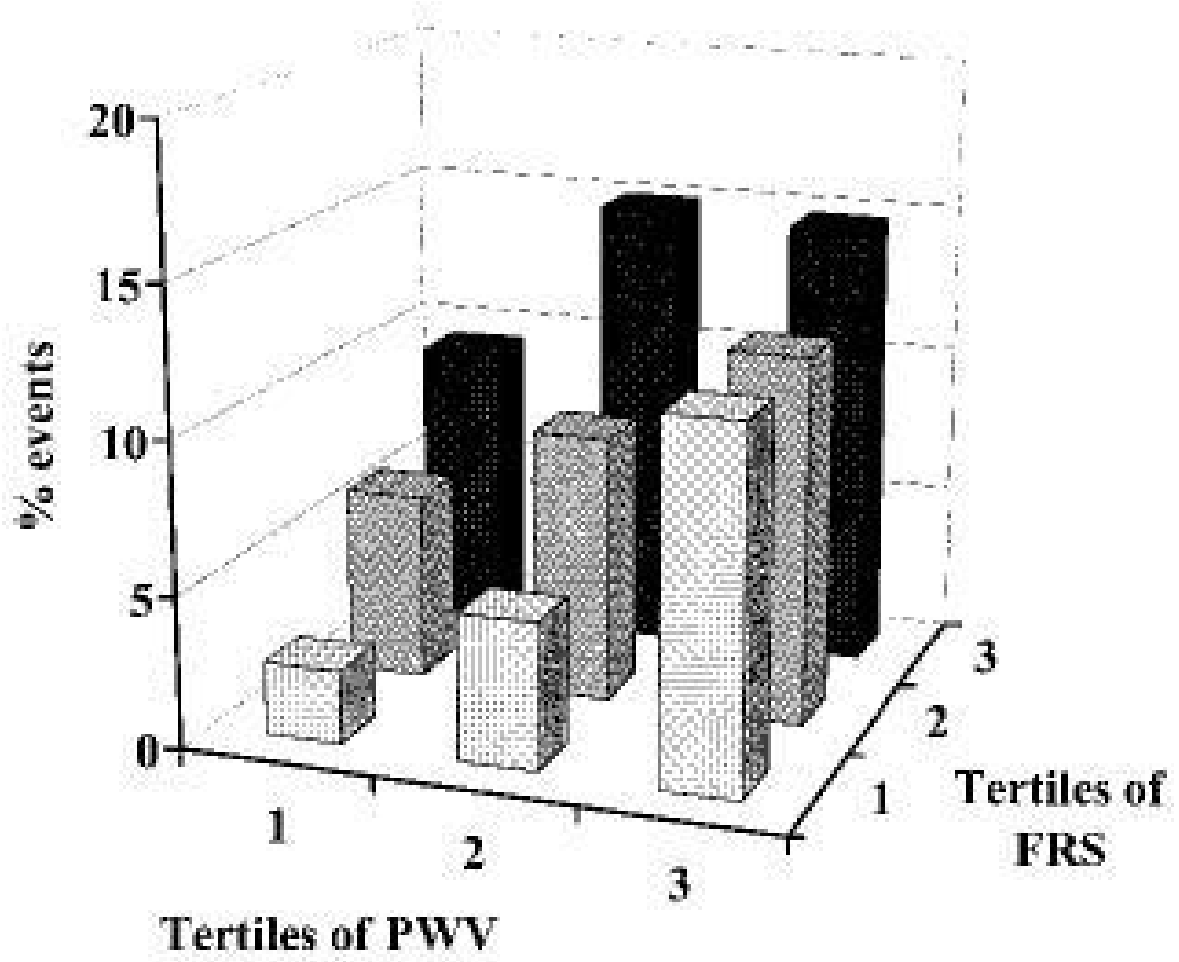
Davies, J.I., Struthers, A.D.:
***J Hypertens** 21:463-472; 2003*

Relation between classical CV risk factors and arterial stiffness



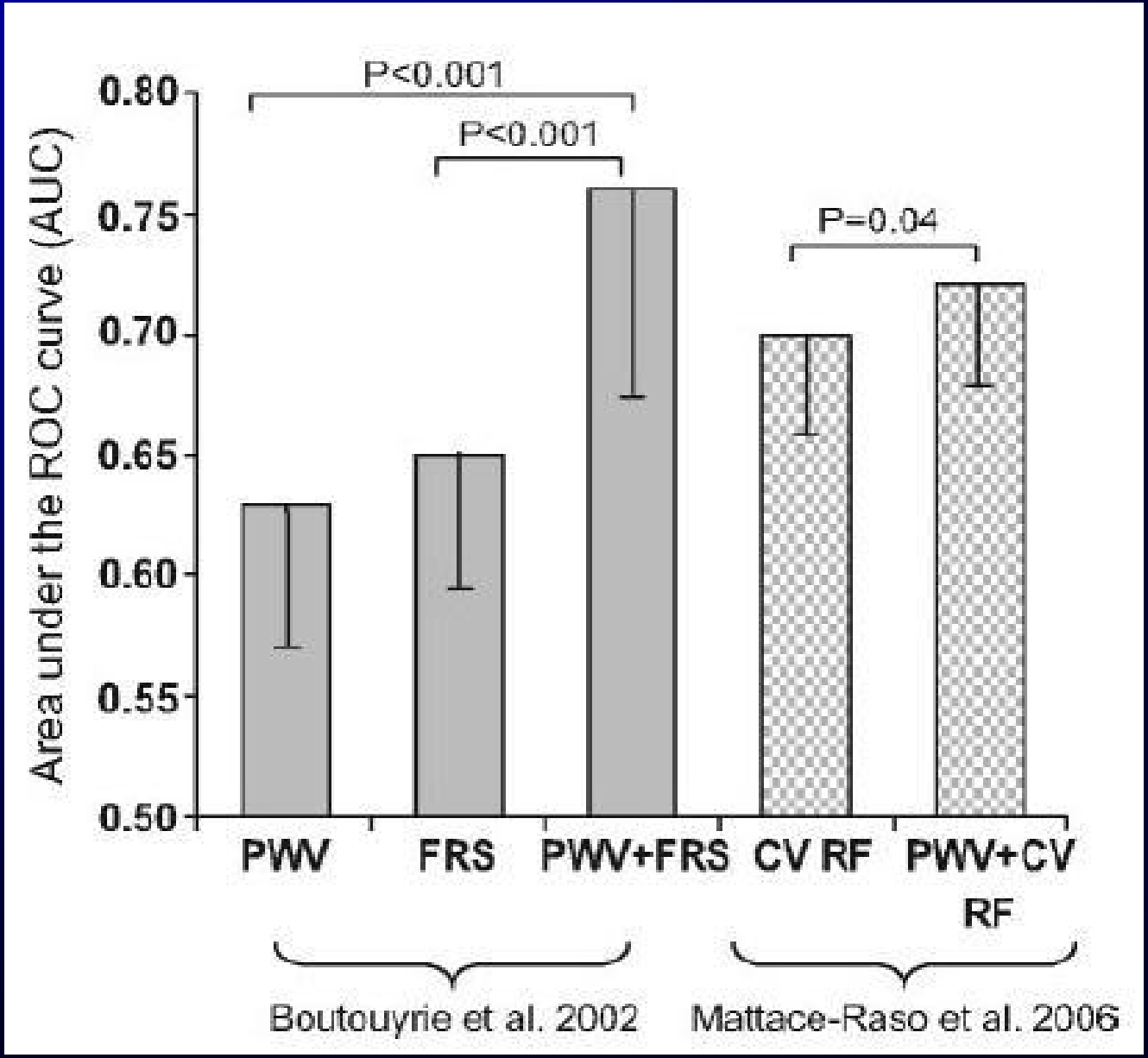
Peter M. Nilsson, Pierre Boutouyrie, Stéphane Laurent
***Hypertension*. 2009;54:3-10**

Additive predictive value of arterial stiffness



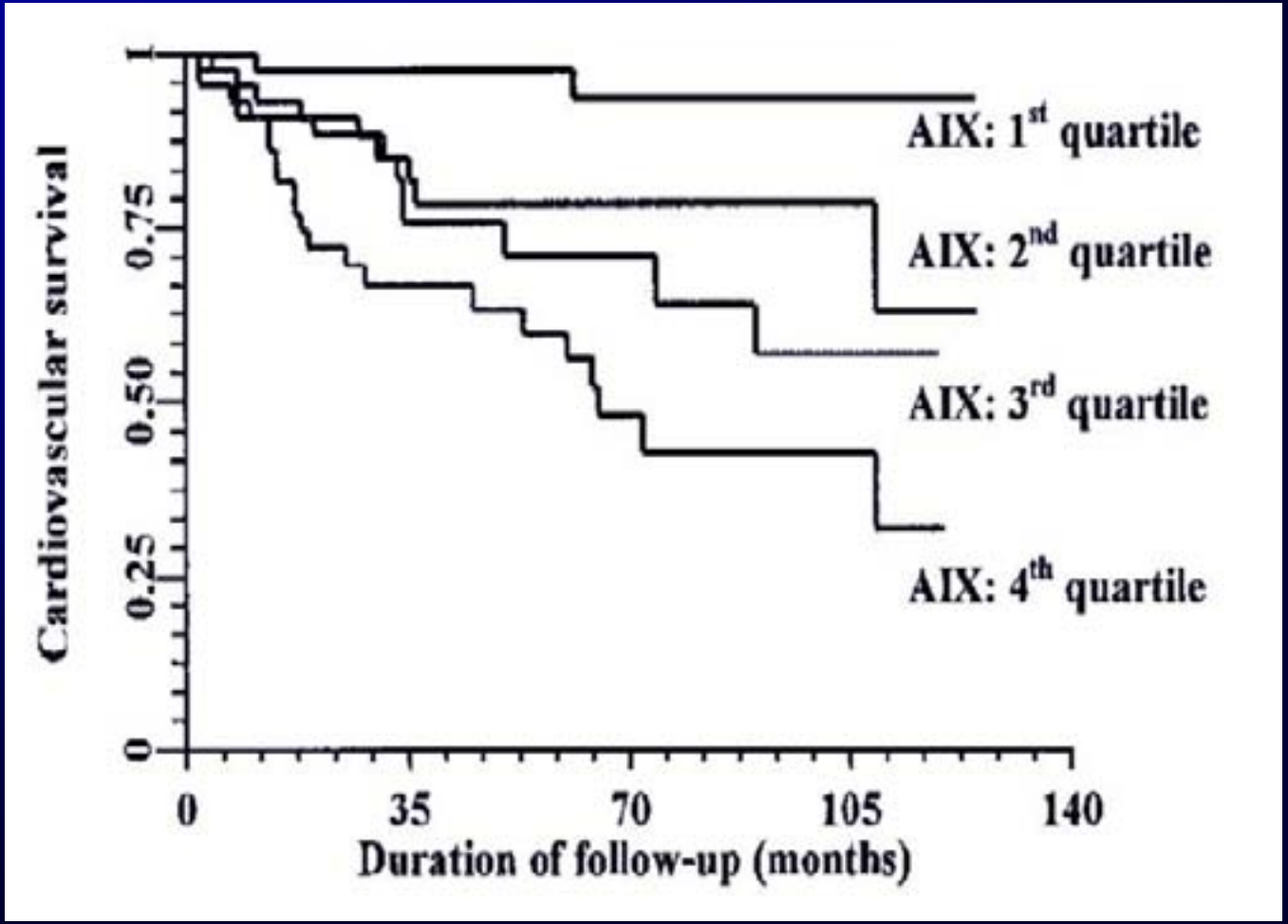
Boutouyrie et al: „Aortic Stiffness and Coronary Heart Disease" *Hypertension*. 2002;39:10-15.

Additive predictive value of arterial stiffness



Aix, SBPao and PWVao; independent predictors of CV morbidity and mortality

Relation between Aix and CV survival



Hypertension 2001;38:434

Arterial Stiffness, Wave Reflections, and the Risk of Coronary Artery Disease

Thomas Weber, MD; Johann Auer, MD; Michael F. O'Rourke, MD; Erich Kvas, ScD; Elisabeth Lassnig, MD; Robert Berent, MD; Bernd Eber, MD

Background—Increased arterial stiffness, determined invasively, has been shown to predict a higher risk of coronary atherosclerosis. However, invasive techniques are of limited value for screening and risk stratification in larger patient groups.

Methods and Results—We prospectively enrolled 465 consecutive, symptomatic men undergoing coronary angiography for the assessment of suspected coronary artery disease. Arterial stiffness and wave reflections were quantified noninvasively using applanation tonometry of the radial artery with a validated transfer function to generate the corresponding ascending aortic pressure waveform. Augmented pressure (AP) was defined as the difference between the second and the first systolic peak, and augmentation index (AIx) was AP expressed as a percentage of the pulse pressure. In univariate analysis, a higher AIx was associated with an increased risk for coronary artery disease (OR, 4.06 for the difference between the first and the fourth quartile [1.72 to 9.57; $P<0.01$]). In multivariate analysis, after controlling for age, height, presence of hypertension, HDL cholesterol, and medications, the association with coronary artery disease risk remained significant (OR, 6.91; $P<0.05$). The results were exclusively driven by an increase in risk with premature vessel stiffening in the younger patient group (up to 60 years of age), with an unadjusted OR between AIx quartiles I and IV of 8.25 ($P<0.01$) and a multiple-adjusted OR between these quartiles of 16.81 ($P<0.05$).

Conclusions—AIx and AP, noninvasively determined manifestations of arterial stiffening and increased wave reflections, are strong, independent risk markers for premature coronary artery disease. **(*Circulation*. 2004;109:184-189.)**

Key Words: coronary disease ■ waves ■ arteries ■ arteriosclerosis

Relation between Aix and CV events in CHD

	Corrected Relative risk	Variables in the model
Aix (10% increase)	1,28 (1,09-1,50)	Age, height, drugs, MAP, HR, EF, diabetes, smoking, CRP, CHF, severity of CHD

Aix is a strong and independent risk factor of adverse cardiovascular outcomes !

N = 297 CHD, 1182 days follow-up,
Hypertension 2005;45:980

Central (aortic) SBP

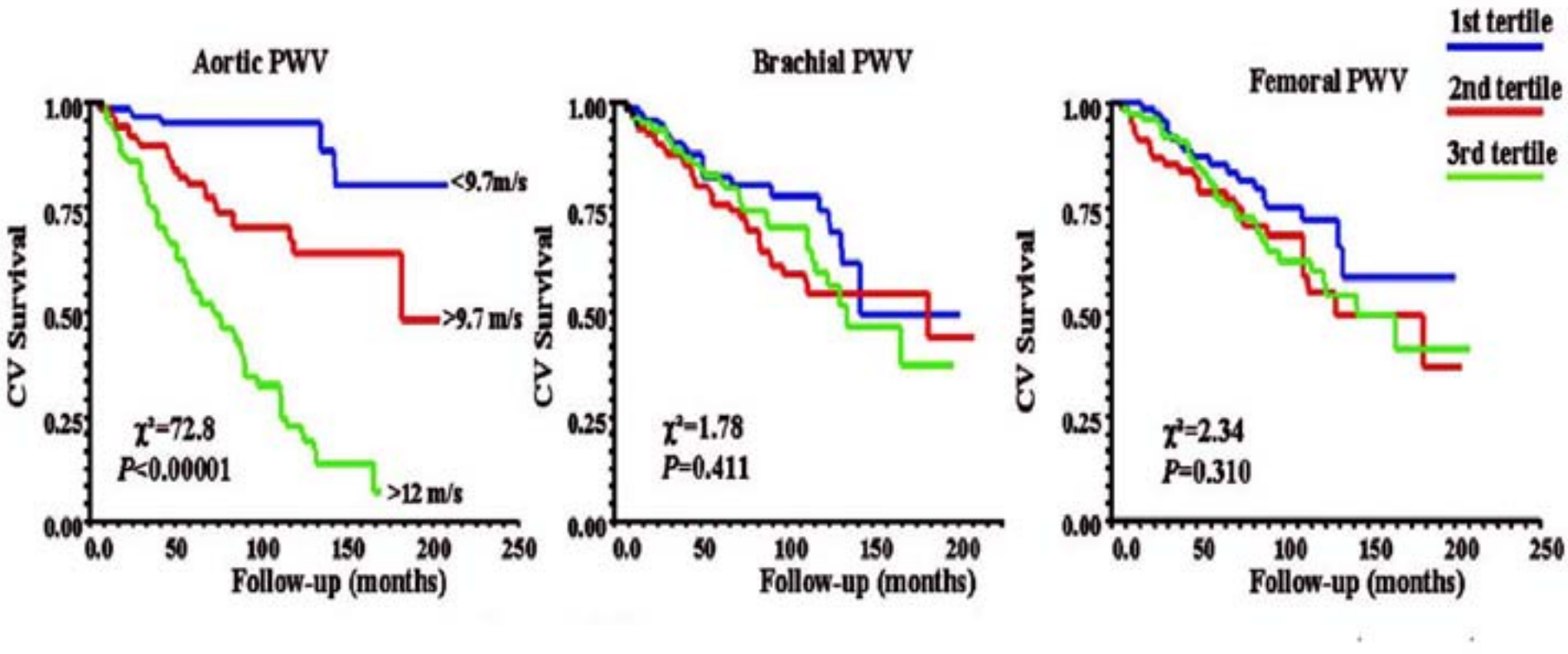
Central Pressure More Strongly Relates to Vascular Disease and Outcome Than Does Brachial Pressure

The Strong Heart Study

Mary J. Roman, Richard B. Devereux, Jorge R. Kizer, Elisa T. Lee, James M. Galloway, Tauqeer Ali, Jason G. Umans, Barbara V. Howard

Abstract—Brachial blood pressure is predictive of cardiovascular outcome; however central pressure may better represent the load imposed on the coronary and cerebral arteries and thereby bear a stronger relationship to vascular damage and prognosis. Relations of brachial and central pressures to carotid artery hypertrophy (intimal-medial thickness and vascular mass), extent of atherosclerosis (plaque score), and incident cardiovascular events were examined in the Strong Heart Study. Central pressures were calculated using radial applanation tonometry. Among 3520 participants, central and brachial pulse pressures were more strongly related to vascular hypertrophy and extent of atherosclerosis than were systolic pressures. Central pulse pressure was more strongly related to all 3 arterial measures than was brachial pulse pressure (r=0.364 versus 0.309 for plaque score; P<0.001 for comparison of Spearman correlation coefficient; r=0.293 versus 0.249 for intimal-medial thickness; P<0.002; r=0.320 versus 0.289 for vascular mass; P<0.05). Among the 2403 participants free of clinical cardiovascular disease at baseline, 319 suffered fatal or nonfatal cardiovascular events during mean follow-up of 4.8±1.3 years. After adjustment for age, gender, current smoking, body mass index, cholesterol:HDL ratio, creatinine, fibrinogen, diabetes, and heart rate, central pulse pressure predicted cardiovascular events more strongly than brachial pulse pressure (hazards ratio=1.15 per 10 mm Hg, χ^2=13.4, P<0.001 versus hazards ratio=1.10, χ^2=6.9, P=0.008). In conclusion, noninvasively-determined central pulse pressure is more strongly related to vascular hypertrophy, extent of atherosclerosis, and cardiovascular events than is brachial blood pressure. These findings support prospective examination of use of central blood pressure as a treatment target in future trials. (*Hypertension*. 2007;50:197-203.)

Increased PWV predicts CV mortality (ESRD)



N = 305, 70 months follow up, Hypertension 2005;45:596

Aortic Pulse Wave Velocity Is Associated With the Presence and Quantity of Coronary Artery Calcium

A Community-Based Study

Iftikhar J. Kullo, Lawrence F. Bielak, Stephen T. Turner, Patrick F. Sheedy II, Patricia A. Peyser

Abstract—We investigated the relationship of aortic pulse wave velocity (aPWV), a measure of central arterial stiffness, with the presence and quantity of coronary artery calcium (CAC) in a community-based sample of adults without prior history of heart attack or stroke (n=401, mean age 59.8 years, 53% men). ECG-gated waveforms of the right carotid and right femoral artery were obtained by applanation tonometry, and aPWV was calculated using established methods. CAC was measured noninvasively by electron beam computed tomography, and CAC score was calculated. aPWV was significantly correlated with log(CAC +1; r=0.41; P<0.0001) and pulse pressure (r=0.47; P<0.0001). Multivariable logistic and linear regression models were used to identify independent predictors of the presence and quantity of CAC, respectively. In multivariable logistic regression analyses, aPWV was associated with the presence of CAC (P=0.011) after adjustment for age, male sex, total cholesterol, high-density lipoprotein cholesterol, diabetes, history of smoking, systolic blood pressure, body mass index, and use of hypertension and statin medications. In multivariable linear regression analyses, aPWV was significantly associated with log(CAC +1) after adjustment for the covariates enumerated above (P<0.0001). aPWV remained significantly associated with both the presence and quantity of CAC even after the additional adjustment for diastolic blood pressure. We conclude that aPWV is related to subclinical coronary atherosclerosis independent of conventional risk factors (including indices of blood pressure) and may be a biomarker of cardiovascular risk in asymptomatic individuals. (*Hypertension*. 2006;47:174-179.)

STUDY (Denmark):
1678 patients, between 40-70 years,
9,4 years follow-up
endpoints: CV mortality, fatal and non-fatal CHD

Aortic PWV predicts cardiovascular outcomes above and beyond traditional risk factors, including 24h MAP (ABPM)

Circulation, 2006 Feb 7; 113(5):664-70

Prognostic Value of Aortic Pulse Wave Velocity as Index of Arterial Stiffness in the General Population

Tine Willum Hansen, MD, PhD; Jan A. Staessen, MD, PhD; Christian Torp Pedersen, MD, DMSc; Susanne Rasmussen, MD, PhD; Lutgarde Thijs, MSc; Hans Ibsen, MD, DMSc; Jørgen Jeppesen, MD, DMSc

In conclusion, in a general population of Western European extraction, APWV predicted a composite of cardiovascular outcomes above and beyond 24-hour mean arterial pressure and traditional risk factors. In combination with the previous studies in patients[3–8] and populations,[9,10] our present findings support the notion that measurement of arterial stiffness is useful in clinical practice for risk stratification.

Circulation. 2006;113:664-670

Arterial Stiffness and Risk of Coronary Heart Disease and Stroke

The Rotterdam Study

Francesco U.S. Mattace-Raso, MD, PhD; Tischa J.M. van der Cammen, MD, PhD; Albert Hofman, MD, PhD; Nicole M. van Popele, MD, PhD; Michiel L. Bos, MSc; Maarten A.D.H. Schalekamp, MD, PhD; Roland Asmar, MD, PhD; Robert S. Reneman, MD, PhD; Arnold P.G. Hoeks, PhD; Monique M.B. Breteler, MD, PhD; Jacqueline C.M. Witteman, PhD

Background—Arterial stiffness has been associated with the risk of cardiovascular disease in selected groups of patients. We evaluated whether arterial stiffness is a predictor of coronary heart disease and stroke in a population-based study among apparently healthy subjects.

Methods and Results—The present study included 2835 subjects participating in the third examination phase of the Rotterdam Study. Arterial stiffness was measured as aortic pulse wave velocity and carotid distensibility. Cox proportional hazards regression analysis was performed to compute hazard ratios. During follow-up, 101 subjects developed coronary heart disease (mean follow-up period, 4.1 years), and 63 subjects developed a stroke (mean follow-up period, 3.2 years). The risk of cardiovascular disease increased with increasing aortic pulse wave velocity index. Hazard ratios and corresponding 95% CIs of coronary heart disease for subjects in the second and third tertiles of the aortic pulse wave velocity index compared with subjects in the reference category were 1.72 (0.91 to 3.24) and

Conclusions - Aortic pulse wave velocity is an independent predictor of coronary heart disease and stroke in apparently healthy subjects. (***Circulation***, **2006;113**:657-663)

Conclusions—Aortic pulse wave velocity is an independent predictor of coronary heart disease and stroke in apparently healthy subjects. (***Circulation***. **2006;113:657-663.**)

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)

Authors/Task Force Members: Giuseppe Mancia, Co-Chairperson (Italy), Guy de Backer, Co-Chairperson (Belgium), Anna Dominiczak (UK), Renata Cifkova (Czech Republic), Robert Fagard (Belgium), Giuseppe Germano (Italy), Guido Grassi (Italy), Anthony M. Heagerty (UK), Sverre E. Kjeldsen (Norway), Stephane Laurent (France), Krzysztof Narkiewicz (Poland), Luis Ruilope (Spain), Andrzej Rynkiewicz (Poland), Roland E. Schmieder (Germany), Harry A.J. Struijker Boudier (Netherlands), Alberto Zanchetti (Italy)

Journal of Hypertension 2007, 25:1105–1187

Table 2 Factors influencing prognosis

Risk factors	Subclinical Organ Damage
• Systolic and diastolic BP levels • Levels of pulse pressure (in the elderly) • Age (M > 55 years; W > 65 years) • Smoking • Dyslipidaemia - TC > 5.0 mmol/l (190 mg/dl) or: - LDL-C > 3.0 mmol/l (115 mg/dl) or: - HDL-C: M < 1.0 mmol/l (40 mg/dl), W < 1.2 mmol/l (46 mg/dl) or: - TG > 1.7 mmol/l (150 mg/dl) • Fasting plasma glucose 5.6–6.9 mmol/L (102–125 mg/dl) • Abnormal glucose tolerance test • Abdominal obesity (Waist circumference > 102 cm (M), > 88 cm (W)) • Family history of premature CV disease (M at age < 55 years; W at age < 65 years)	• 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
Diabetes Mellitus	**Established CV or renal disease**
• Fasting plasma glucose ≥ 7.0 mmol/l (126 mg/dl) on repeated measurement, or • Postload plasma glucose > 11.0 mmol/l (198 mg/dl)	• Cerebrovascular disease: ischaemic stroke; cerebral haemorrhage; transient ischaemic attack • Heart disease: myocardial infarction; angina; coronary revascularization; heart failure • Renal disease: diabetic nephropathy; renal impairment (serum creatinine M > 133, W > 124 mmol/l); proteinuria (> 300 mg/24 h) • Peripheral artery disease • Advanced retinopathy: haemorrhages or exudates, papilloedema

Note: the cluster of three out of 5 risk factors among abdominal obesity, altered fasting plasma glucose, BP ≥ 130/85 mmHg, low HDL-cholesterol and high TG (as defined above) indicates the presence of metabolic syndrome

Box 6 Laboratory investigations

Routine tests

- Fasting plasma glucose
- Serum total cholesterol
- Serum LDL-cholesterol
- Serum HDL-cholesterol
- Fasting serum triglycerides
- Serum potassium
- Serum uric acid
- Serum creatinine
- Estimated creatinine clearance (Cockroft-Gault formula) or glomerular filtration rate (MDRD formula)
- Haemoglobin and haematocrit
- Urinalysis (complemented by microalbuminuria via dipstick test and microscopic examination)
- Electrocardiogram

Recommended tests

- Echocardiogram
- Carotid ultrasound
- Quantitative proteinuria (if dipstick test positive)
- Ankle-brachial BP Index
- Fundoscopy
- Glucose tolerance test (if fasting plasma glucose >5.6 mmol/L (100 mg/dL)
- Home and 24 h ambulatory BP monitoring
- Pulse wave velocity measurement (where available)

Extended evaluation (domain of the specialist)

- Further search for cerebral, cardiac, renal and vascular damage. Mandatory in complicated hypertension
- Search for secondary hypertension when suggested by history, physical examination or routine tests: measurement of renin, aldosterone, corticosteroids, catecholamines in plasma and/or urine; arteriographies; renal and adrenal ultrasound; computer-assisted tomography; magnetic resonance imaging

Box 7 Position statement: Searching for subclinical organ damage

Due to the importance of subclinical organ damage as an intermediate stage in the continuum of vascular disease and as a determinant of total cardiovascular risk, signs of organ involvement should be sought carefully by appropriate techniques:

1. Heart – Electrocardiography should be part of all routine assessment of subjects with high BP in order to detect left ventricular hypertrophy, patterns of "strain", ischaemia and arrhythmias. Echocardiography is recommended when a more sensitive detection of left ventricular hypertrophy is considered useful. Geometric patterns can be defined echocardiographically, of which concentric hypertrophy carries the worse prognosis. Diastolic dysfunction can be evaluated by transmitral Doppler.
2. Blood vessels – Ultrasound scanning of carotid arteries is recommended when detection of vascular hypertrophy or asymptomatic atherosclerosis is deemed useful. Large artery stiffening (leading to isolated systolic hypertension in the elderly) can be measured by pulse wave velocity. It might be more widely recommended if its availability were greater. A low ankle-brachial BP index signals advanced peripheral artery disease.
3. Kidney – Diagnosis of hypertension-related renal damage is based on a reduced renal function or an elevated urinary excretion of albumin. Estimation

Table 3 High/Very high risk subjects

- BP ≥ 180 mmHg systolic and/or ≥ 110 mmHg diastolic
- Systolic BP > 160 mmHg with low diastolic BP (< 70 mmHg)
- Diabetes mellitus
- Metabolic syndrome
- ≥ 3 cardiovascular risk factors
- One or more of the following subclinical organ damages:
  - Electrocardiographic (particularly with strain) or echocardiographic (particularly concentric) left ventricular hypertrophy
  - Ultrasound evidence of carotid artery wall thickening or plaque
  - Increased arterial stiffness
  - Moderate increase in serum creatinine
  - Reduced estimated glomerular filtration rate or creatinine clearance
  - Microalbuminuria or proteinuria
- Established cardiovascular or renal disease

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	+++	++++	+

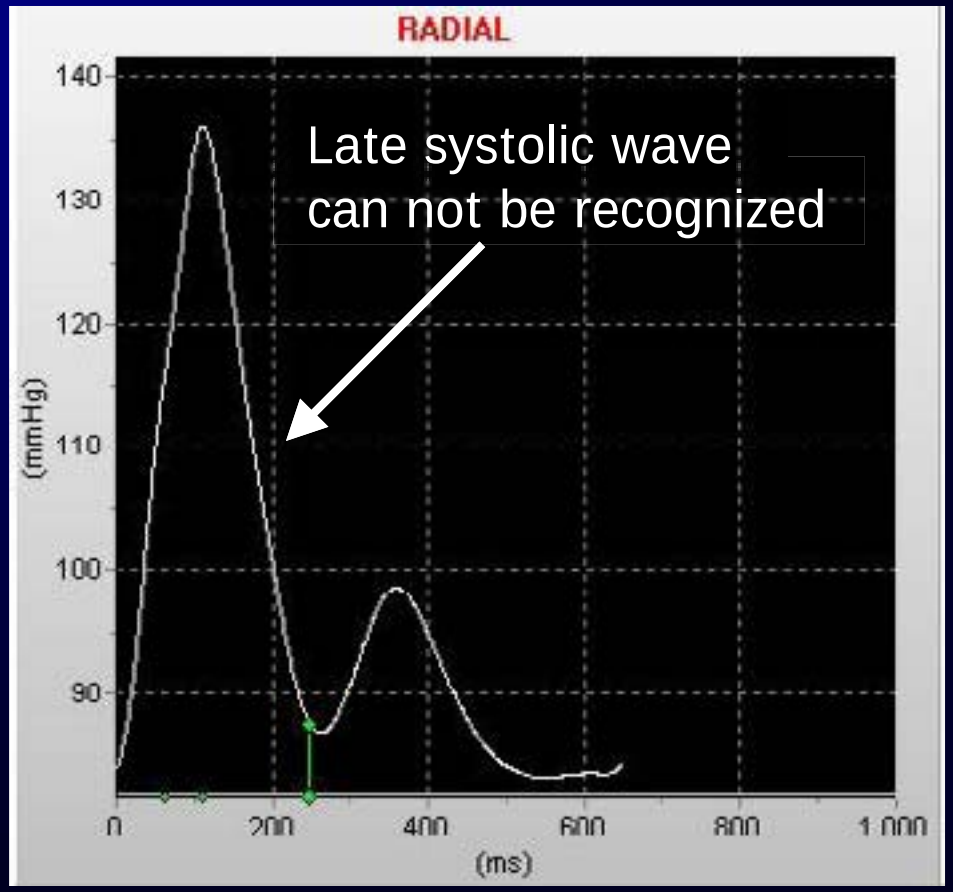
WE DID NOT HAVE A SIMPLE, FAST, USER IDEPENDENT, RELIABLE NON-INVASIVE METHOD, WHICH COULD BE SUITABLE FOR THE EVERYDAY CLINICAL PRACTICE TO MEASURE Aix, SBPao, PWVao

BECAUSE THE AVAILABLE METHODS ARE

- **DIFFICULT,**
- **INCONVENIENT,**
- **EXPENSIVE,**
- **REQUIRE PROPERLY TRAINED STAFF**

PWVcf measurement with ECG gated pulsed wave Doppler

Applanation tonometry



2001
CREATING A RESEARCH CONSORTIUM

Hungarian Academy of Sciences

Institute of Material Sciences

Semmelweis Medical University

Institute of Human Physiology

Semmelweis Medical University

1st Department of Internal Medicine

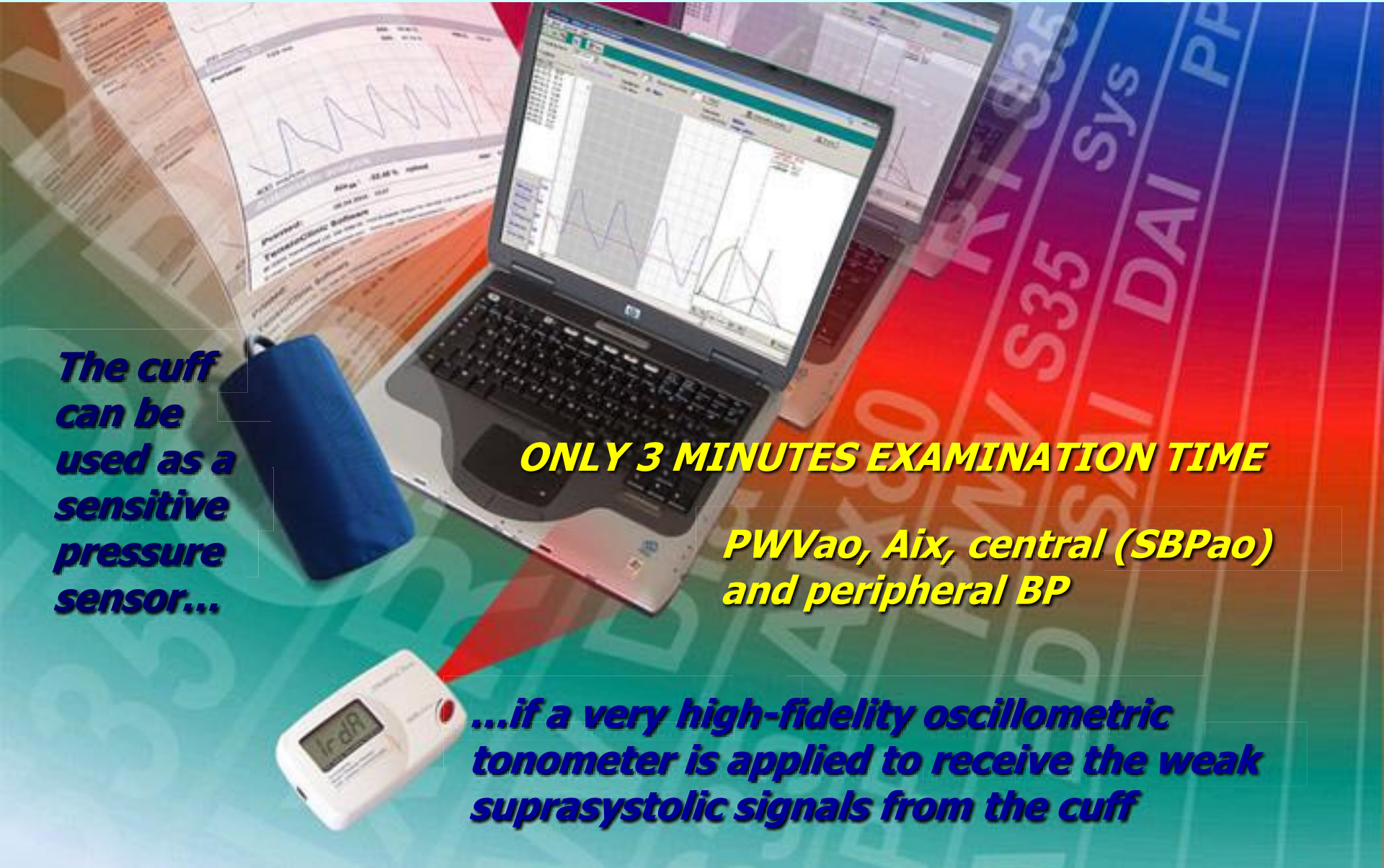
St. Emeritus Hospital, Budapest

1st Department of Internal Medicine

TensioMed Arterial Stiffness Centre

Consortium leader

ARTERIAL FUNCTION (STIFFNESS) EVALUATION WITH OSCILLOMETRIC OCCLUSIVE METHOD



The cuff can be used as a sensitive pressure sensor…

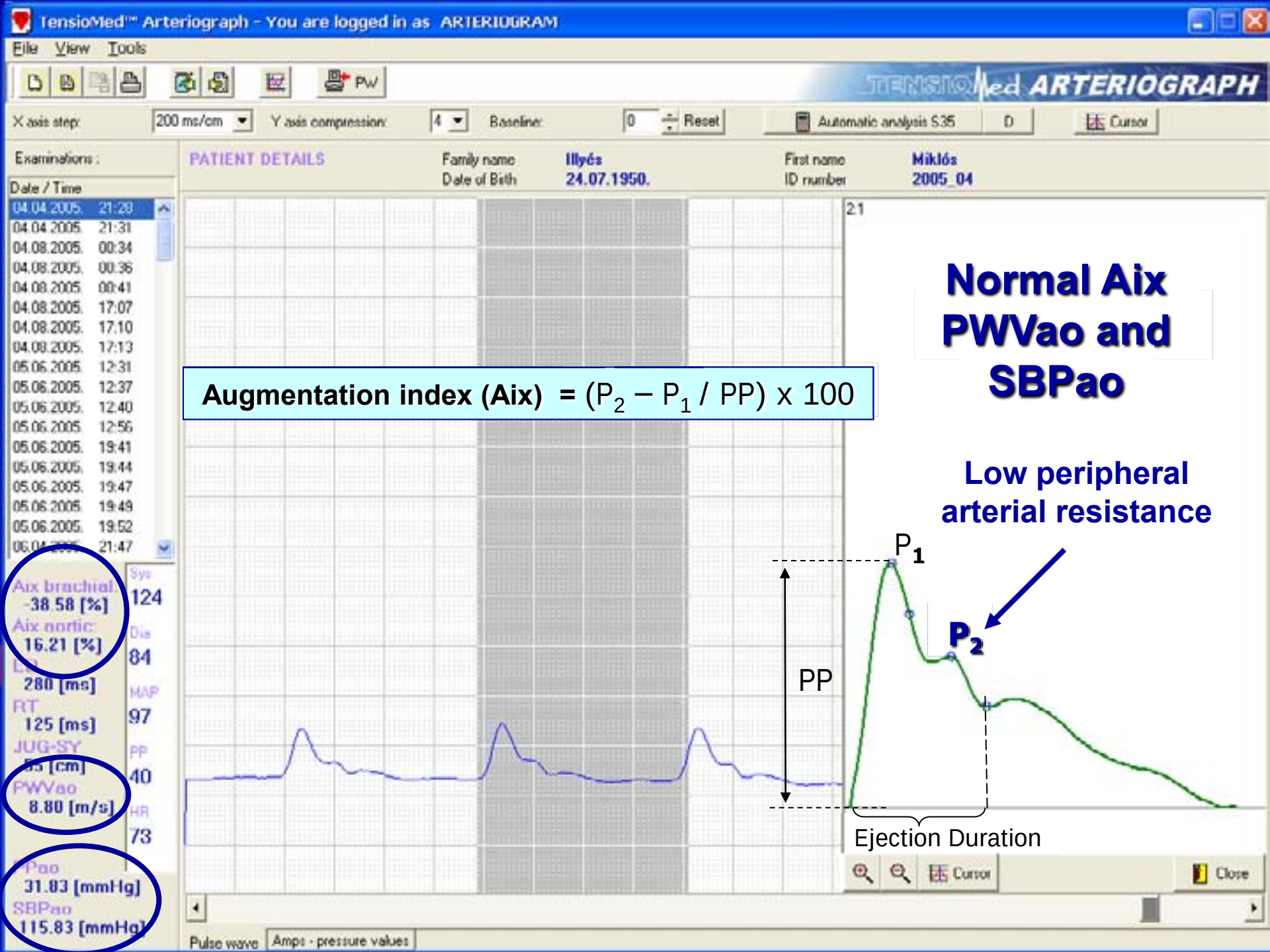
ONLY 3 MINUTES EXAMINATION TIME

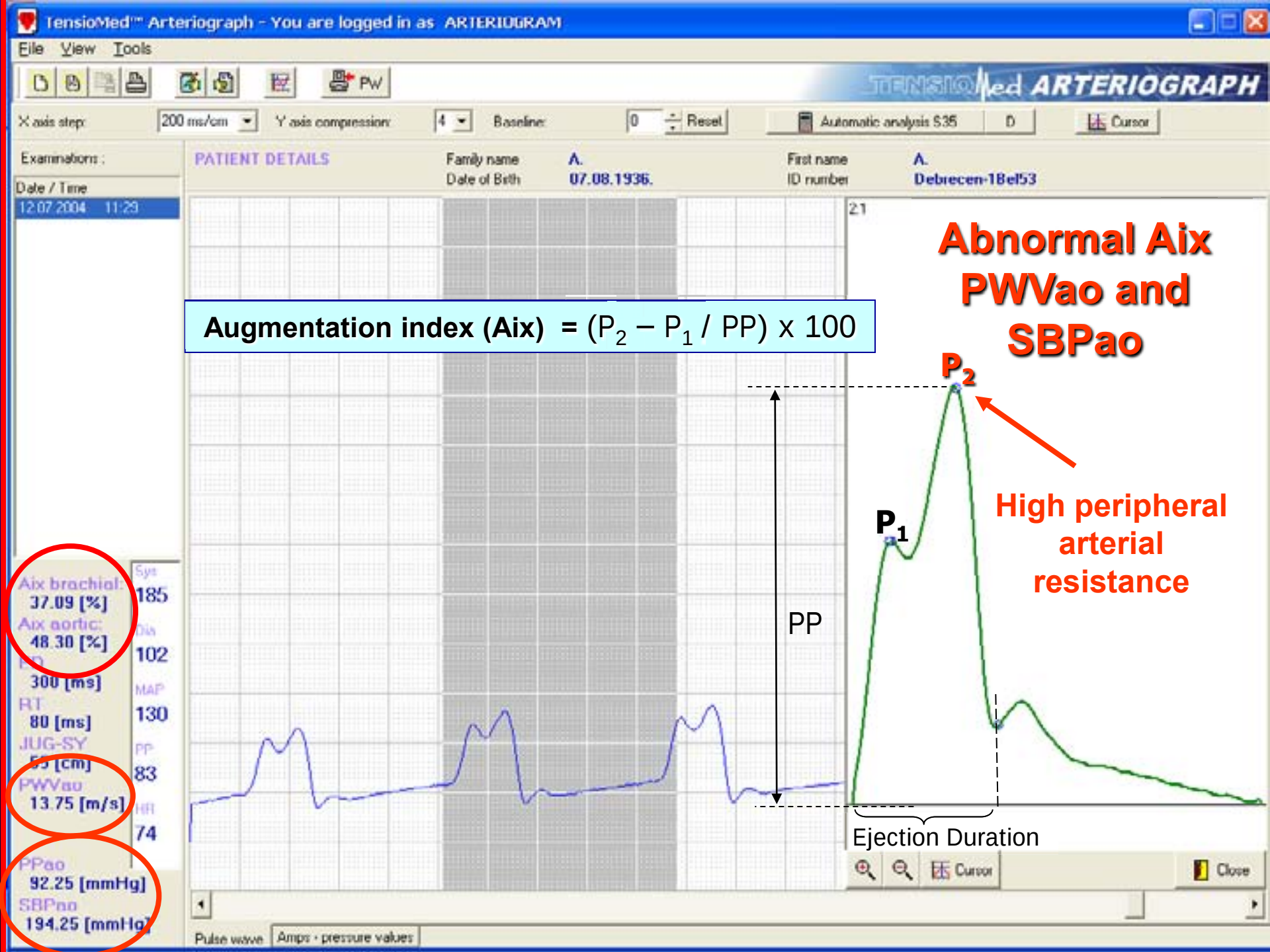
PWVao, Aix, central (SBPao) and peripheral BP

…if a very high-fidelity oscillometric tonometer is applied to receive the weak suprasystolic signals from the cuff

ARTERIOGRAPH MEASURED PARAMETERS DESCRIBING ARTERIAL FUNCTION

1. **AUGMENTATION INDEX (Aix)**
   WHICH REFLECTS TO THE TPR AND **ENDOTHEL FUNCTION**
2. **AORTIC PULSE WAVE VELOCITY (PWVao)**
   WHICH REFLECTS TO THE **STIFFNESS OF THE AORTIC WALL**
3. **SAI, DAI AND DRA**
   BY MEASURING THE SYSTOLIC AND DIASTOLIC AREA INDEX AND DIASTOLIC REFLECTION AREA THE **LEFT CORONARY FILLING CONDITION** CAN BE ASSESSED
4. **SYSTOLIC/DIASTOLIC BP AND HR**
5. **CENTRAL (AORTIC) SBP**


TensioMed™ Arteriograph - You are logged in as ARTERIOGRAM
File View Tools
TENSIOMED ARTERIOGRAPH
X axis step: 200 ms/cm
Y axis compression: 4
Baseline: 0
Reset
Automatic analysis S35
D
Cursor
Examinations :
Date / Time
PATIENT DETAILS
Family name Illyés
Date of Birth 24.07.1950.
First name Miklós
ID number 2005_04
Aix brachial -38.58 [%]
Aix aortic: 16.21 [%]
280 [ms]
RT 125 [ms]
JUG-SY
PWVao 8.80 [m/s]
Pao 31.83 [mmHg]
SBPao 115.83 [mmHg]
Sys 124
Dia 84
MAP 97
PP 40
HR 73
Augmentation index (Aix) = (P2 – P1 / PP) x 100
Normal Aix
PWVao and
SBPao
Low peripheral
arterial resistance
P1
P2
PP
Ejection Duration
Cursor
Close
Pulse wave
Amps - pressure values


TensioMed™ Arteriograph - You are logged in as ARTERIOGRAM
File View Tools
TENSIOMed ARTERIOGRAPH
Examinations :
Date / Time
12.07.2004 11:29
PATIENT DETAILS
Family name A.
Date of Birth 07.08.1936.
First name A.
ID number Debrecen-1Bel53
Augmentation index (Aix) = (P2 – P1 / PP) x 100
Abnormal Aix
PWVao and
SBPao
P2
P1
PP
High peripheral
arterial
resistance
Ejection Duration
Aix brachial:
37.09 [%]
Aix aortic:
48.30 [%]
300 [ms]
RT
80 [ms]
PWVao
13.75 [m/s]
PPao
92.25 [mmHg]
SBPao
194.25 [mmHg]
185
102
130
83
74
Cursor
Close
Pulse wave
Amps - pressure values

IS THIS NEW OSCILLOMETRIC METHOD (ARTERIOGRAPH)

USEFUL FOR SCREENING

PRECLINICAL MACROVASCULAR ATHEROSCLEROSIS?

Patients' characteristics (n = 643)

	Mean	SD	Min	Max
Age (ys)	57,2	11,2	20	86
SBP (mmHg)	134,1	17,6	97,0	226,0
DBP (mmHg)	81,6	9,9	53,0	121,0
HR (beat/min)	71,9	10,7	43	115
BMI	27,1	4,3	17,5	44,1
Total chol. (mmol/l)	5,6	1,2	2,0	10,7
Smoker (%)	16,2			
PWVao (m/s)	10,0	2,2	5,6	17,1
Carotis positive (%)	319 (49,6)			
Carotis negative (%)	324 (50,4)			

Carotis protocol

Both extracranial carotis systems were screened completely.
The results were considered to be positive for asymptomatic atherosclerosis if

- ≥ 1 mm echogenic (calcificated) plaque was seen

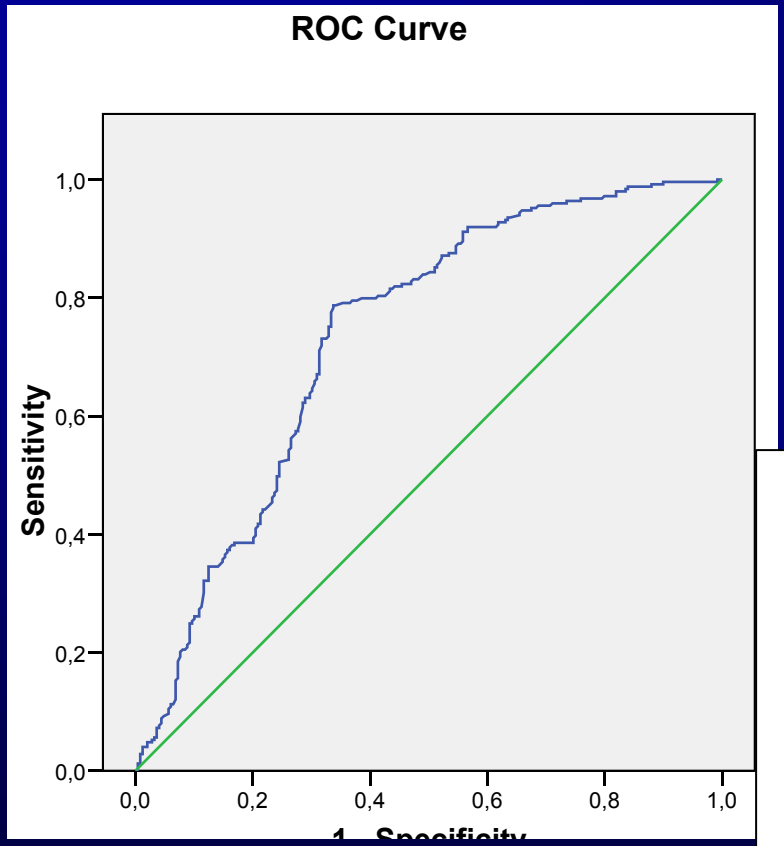
and/or

- ≥ 1,3 mm IMT was measured

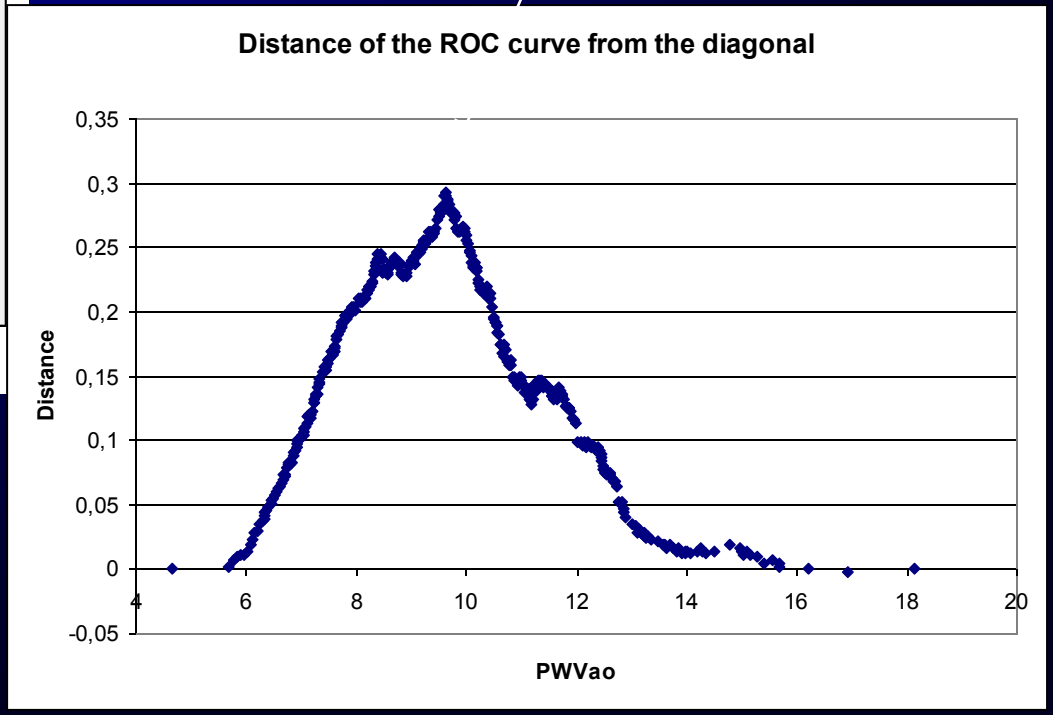
Arterial stiffness (PWVao) protocol

- A new, validated, oscillometric, user independent device (Arteriograph) was used, which can perform a complete arterial function analysis within 3 minutes with a single upper arm cuff.
- The measurements were independent from the user
- The patients had at least 10 minutes rest before the examination
- Caffeine intake, smoking was not allowed 3 hours before of the measurement
- Alcohol intake was not allowed 10 hours before of the examination
- Patients with arrhythmia and nitrate use were excluded

Results – PWVao threshold



The optimal threshold of PWVao proved to be **9,62** m/s with ROC



Results

Asymptomatic Population

	Overall	Male	Female
Sensitivity (%)	**76,5**	**68,1**	**84,9**
Specificity (%)	**64,5**	**79,7**	**55,5**
P. pred. value (%)	**68,0**	**73,6**	**65,2**
N. pred. value (%)	**73,6**	**68,5**	**78,9**
Relative Risk*	**2,6**	**2,4**	**3,1**
Odds ratio**	**6,0**	**6,8**	**7,0**

*Relative Risk shows that among patients with high (>9,62 m/s) PWVao, how many times more probable the disease (e.g. carotis atherosclerosis) than with low PWVao subjects

**Odds ratio shows that how many times higher the chance to diagnose the disease (e.g. carotis atherosclerosis) properly, than improperly

The prevalence of carotid atherosclerosis was 49% in the studied population

TensioMed™ Arteriograph

Arterial Stiffness Report

Patient details

Name:

Date of Birth: 1950.09.04. **Height:** 170 cm **BMI:** 20.76 **Cholesterol:** 6.70 mmol/l

Sex: Female **Weight:** 60 kg **Smoker:** No

Comment:

10 year risk of fatal CVD (EHRC score): 1% (low risk)

Low CV risk, according to SCORE stratification

Study data

Examination date and time: 2007.05.24. 14:05 **JUG-SY:** 52 cm

Operator's name: DR. BENCZÚR BÉLA

Sys: 117 mmHg **Dia:** 76 mmHg **HR:** 67 /min **MAP:** 90 mmHg **PP:** 41 mmHg

Results S...

Elevated PWV = high CV risk !!!

PWVao: 10.51 m/s **SBPao:** 111.35 mmHg

Aix aortic: 22.67 % **RT:** 99 ms **SD PWVao:** 0.27 m/s

Examination:

- **Carotid Ultrasound – positive (plaque)**

Conclusion:

- By screening an *apparently healthy* subject with Arteriograph *asymptomatic arterial disease* could be detected
- Further examination (carotid US) may confirm the diagnosis
- Active therapy and patient follow-up is mandatory to prevent MACE

TensioMed™ Arteriograph

Arterial Stiffness Report

Patient details

Name:

Date of Birth: 07.10.1938. **Height:** 167 cm **BMI:** 30.48 **Cholesterol:**

Sex: Female **Weight:** 85 kg **Smoker:**

Comment: Bisoprolol

β-blocker as antihypertensive drug

Study data

Examination date and time: 12.01.2006. 09:45 **JUG-SY:** 58 cm

Operator's name: ARTERIOGRAM

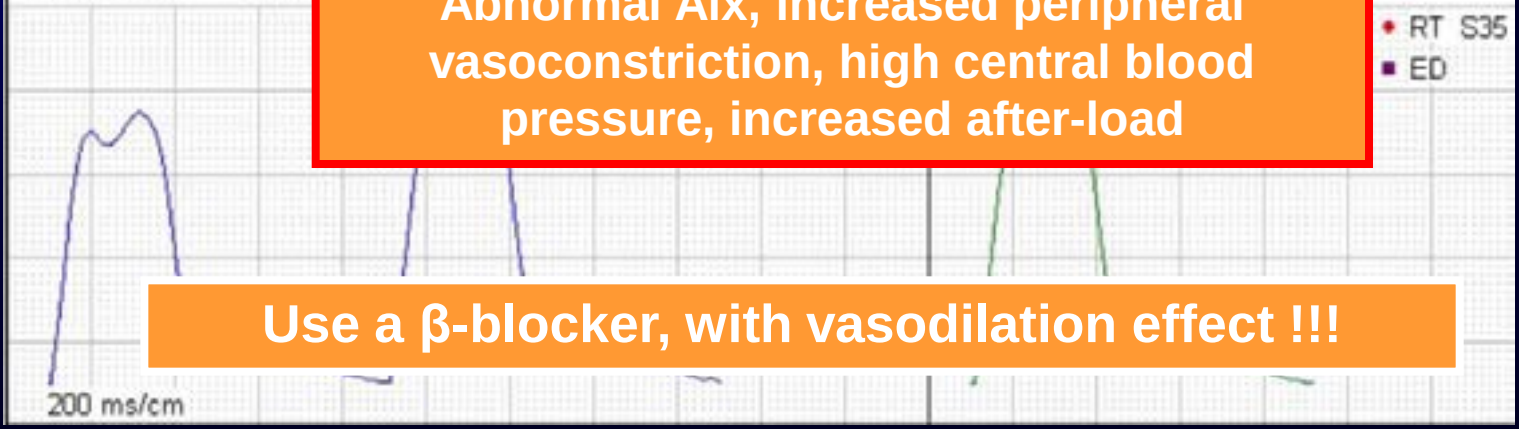
Sys: 157 mmHg **Dia:** 94 mmHg **HR:** 68 /min **MAP:** 115 mmHg **PP:** 63 mmHg

Results S 35

Aix brachial: 9.07 % **ED:** 345 ms **PWVao:** 12.00 m/s **SBPao:** 160.05 mmHg

Aix aortic: 36.4

Abnormal Aix, increased peripheral vasoconstriction, high central blood pressure, increased after-load



Use a β-blocker, with vasodilation effect !!!

TensioMed™ Arteriograph

Arterial Stiffness Report

The same subject with modified therapy

β-blocker with vasodilation effect

Patient details

Name:

Date of Birth: 07.10.1938. **Height:** 167 cm **BMI:** 29.40 **Cholesterol:** 3.90 mmol/l

Sex: Female **Weight:** 82 kg

Comment: Nebivolol

10 year risk of fatal CVD (EHRC score): 3% (medium risk)

Study data

Examination date and time: 29.11.2006. 13:45 **JUG-SY:** 54 cm

Operator's name: ARTERIOGRAM

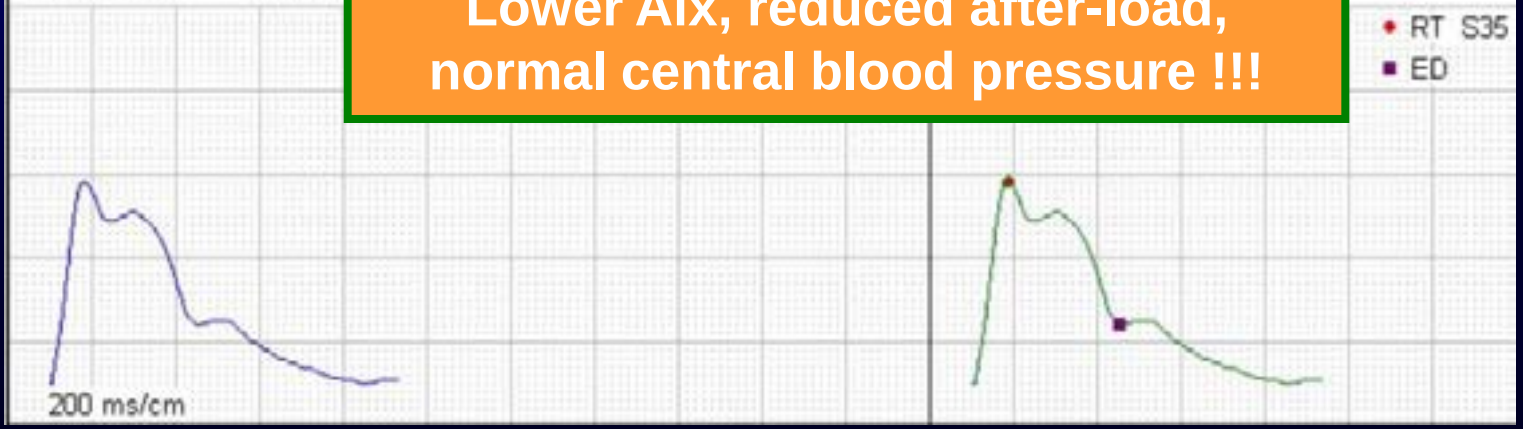
Sys: 141 mmHg **Dia:** 77 mmHg **HR:** 70 /min **MAP:** 98 mmHg **PP:** 64 mmHg

Results S 35

Aix brachial: -15.03 % **ED:** 355 ms **PWVao:** 9.80 m/s **SBPao:** 134.81 mmHg

Aix aortic: 26.20 %

Lower Aix, reduced after-load, normal central blood pressure !!!



INDICATIONS OF MEASURING ARTERIAL FUNCTION

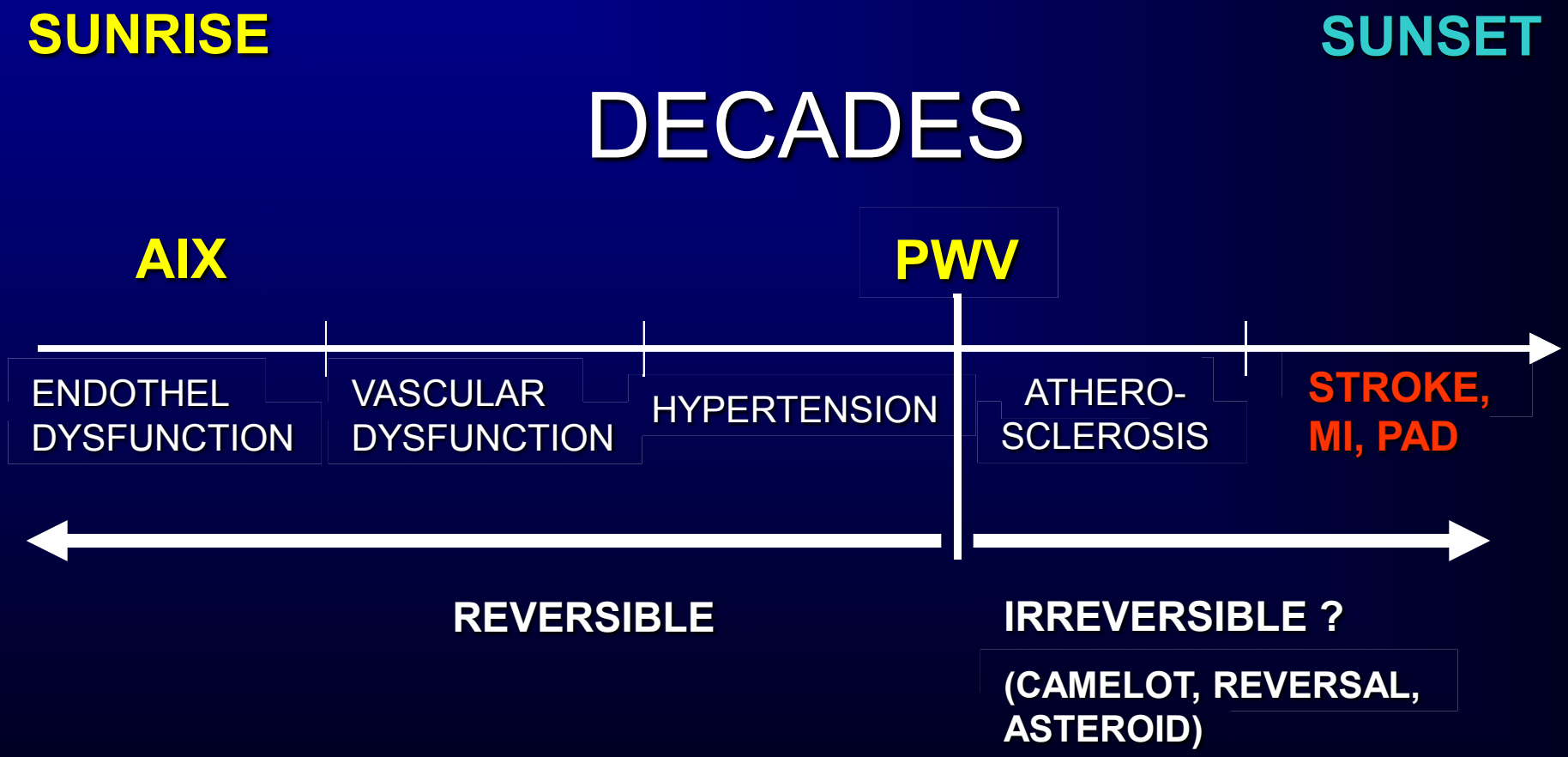
TO SCREEN FOR EARLY ATHEROSCLEROSIS AMONG ASYMPTOMATIC, APPARENTLY „HEALTHY" SUBJECTS FOR IMPROVING CV RISK ASSESSMENT

AND

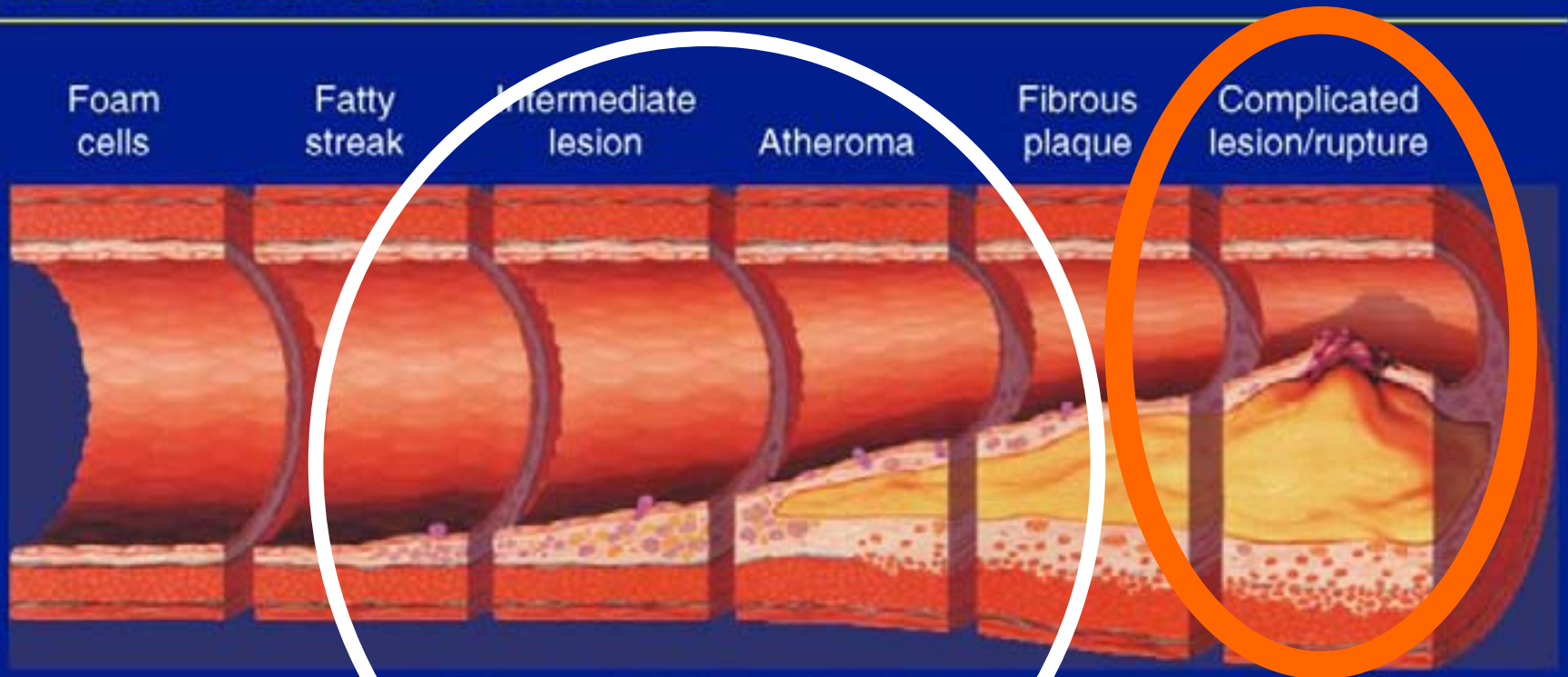
TO ASSESS THE EFFECT OF THE THERAPY ON VASCULAR FUNCTION AMONG PATIENTS WHERE ATHEROSCLEROSIS HAS BEEN ESTABLISHED (CAD, POST MI, STROKE, PAD)

THE METHOD CAN NOT BE USED FOR DETECTING LOCAL STENOSIS IN A CERTAIN ARTERY !

The genesis of atherosclerosis



Atherosclerosis timeline



From first decade	From third decade	From fourth decade	
Growth mainly by lipid accumulation		Smooth muscle and collagen	Thrombosis, hematoma

Pepine CJ. *Am J Cardiol.* 1998;82(suppl 10A):23S-27S.


THANKS FOR YOUR
ATTENTION !