24h assessment of arterial stiffness

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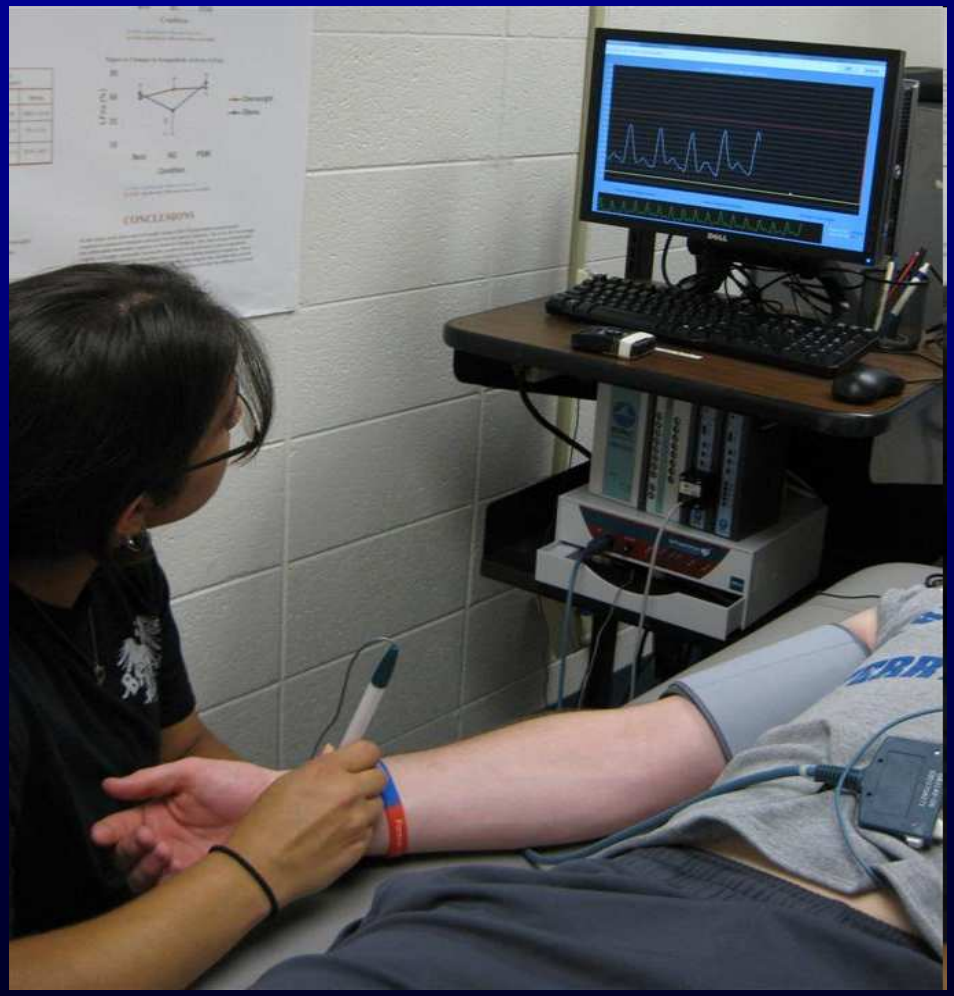
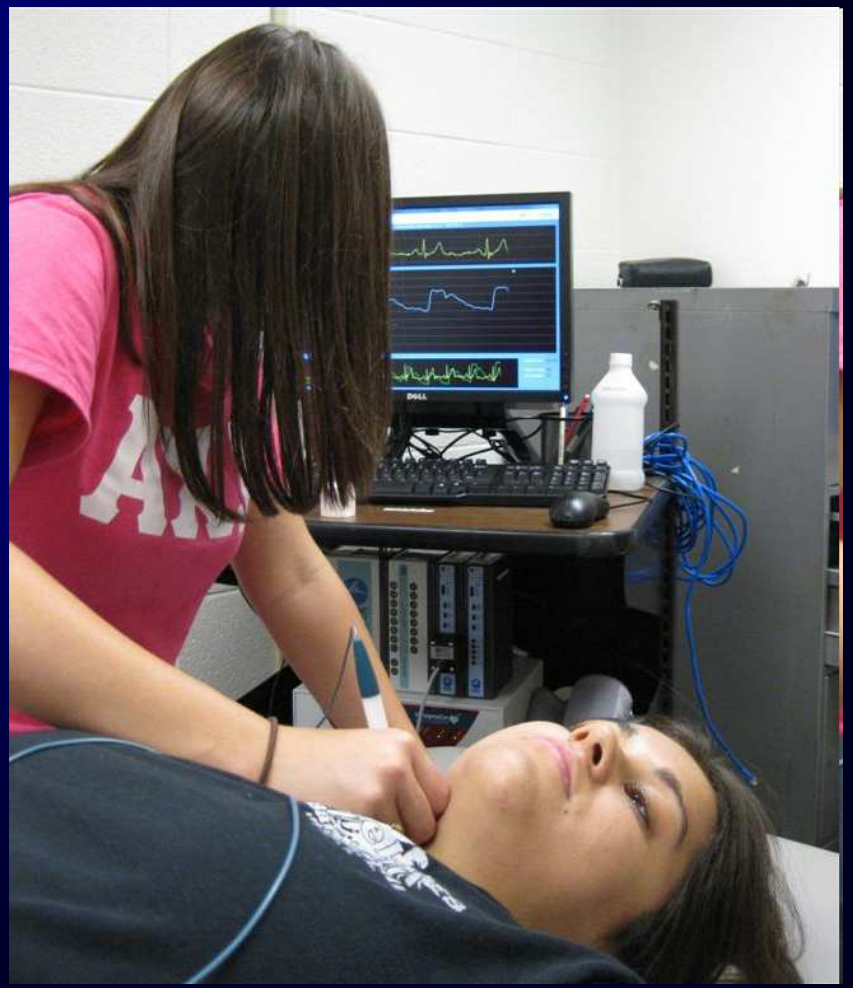
Conflict of interest:
M.I. is patent holder of the oscillometric, occlusive method measuring arterial stiffness

METHODOLOGICAL BACKGROUNG

24h AMBULATORY BP MONITORING

ARTERIAL STIFFNESS MEASUREMENT





AMBULATORY BP MONITORING

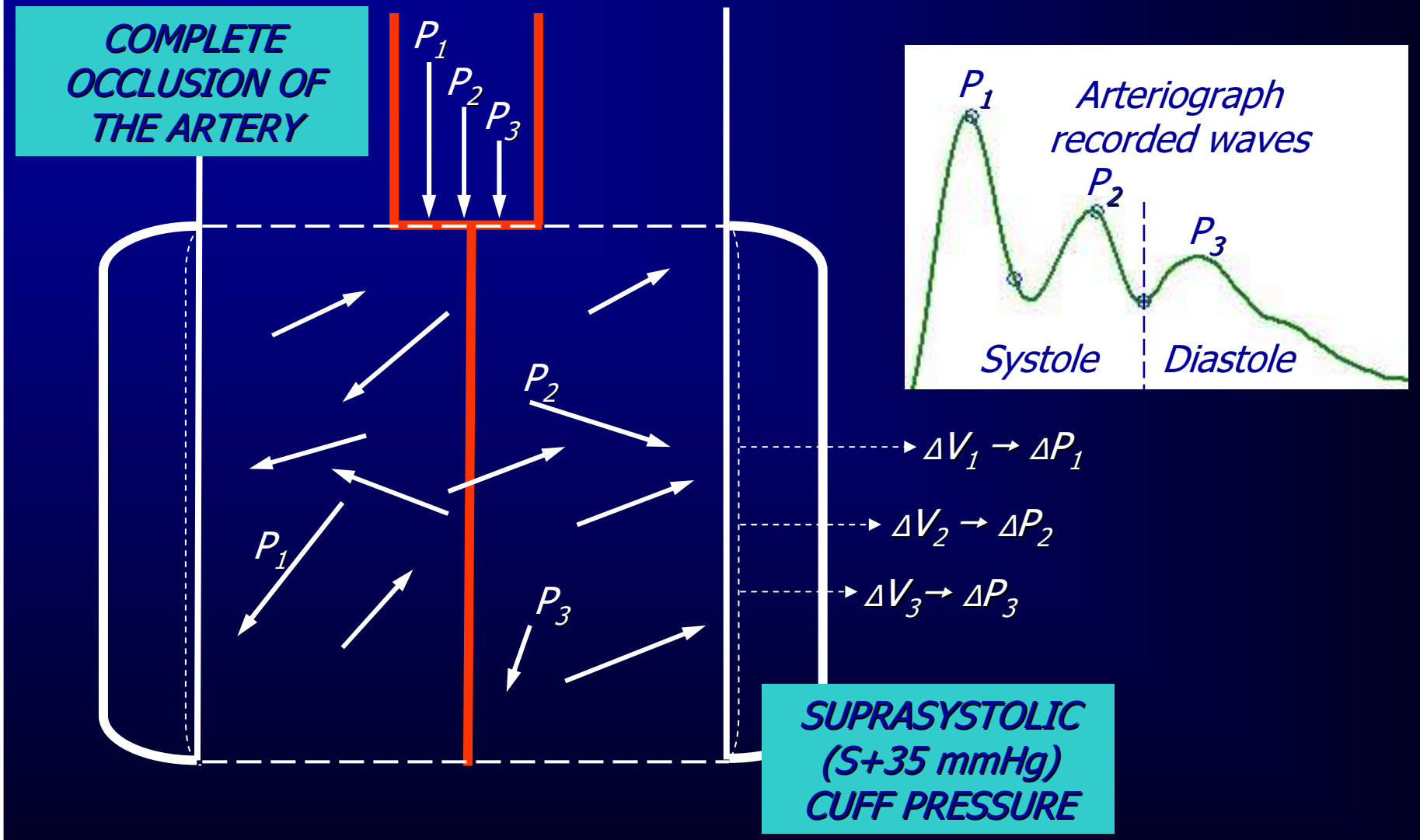
+

ARTERIAL STIFFNESS MEASUREMENT

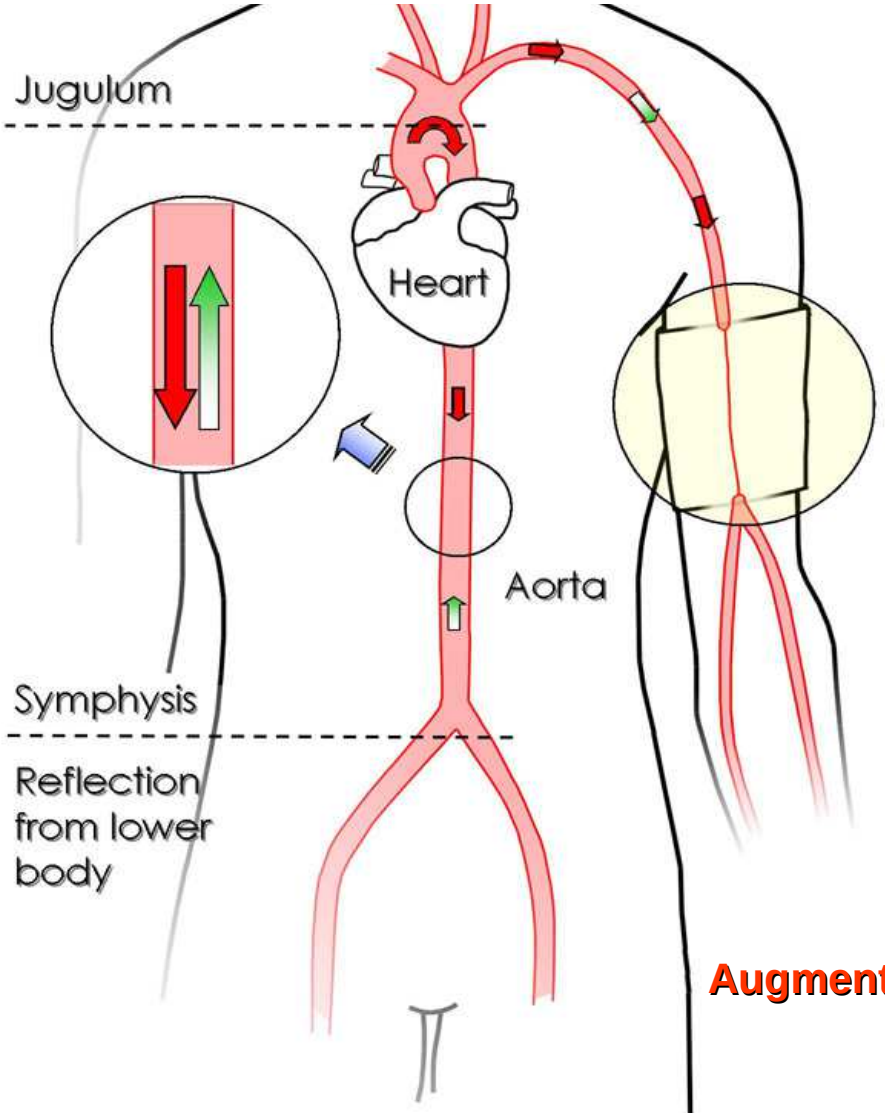
= ARTERIOGRAPH 24

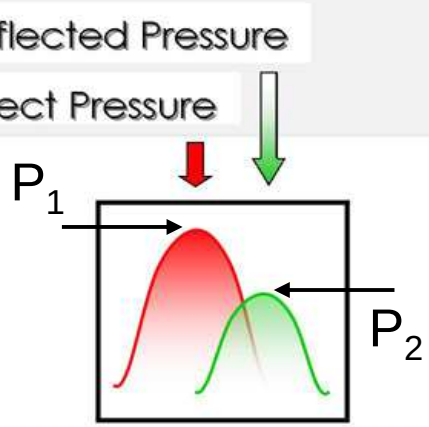
OCCLUSIVE, OSCILLOMETRIC METHOD

THE PRINCIPLE OF OPERATION OF ARTERIOGRAPH 24



The principle of measuring augmentation index with occlusive, oscillometric method





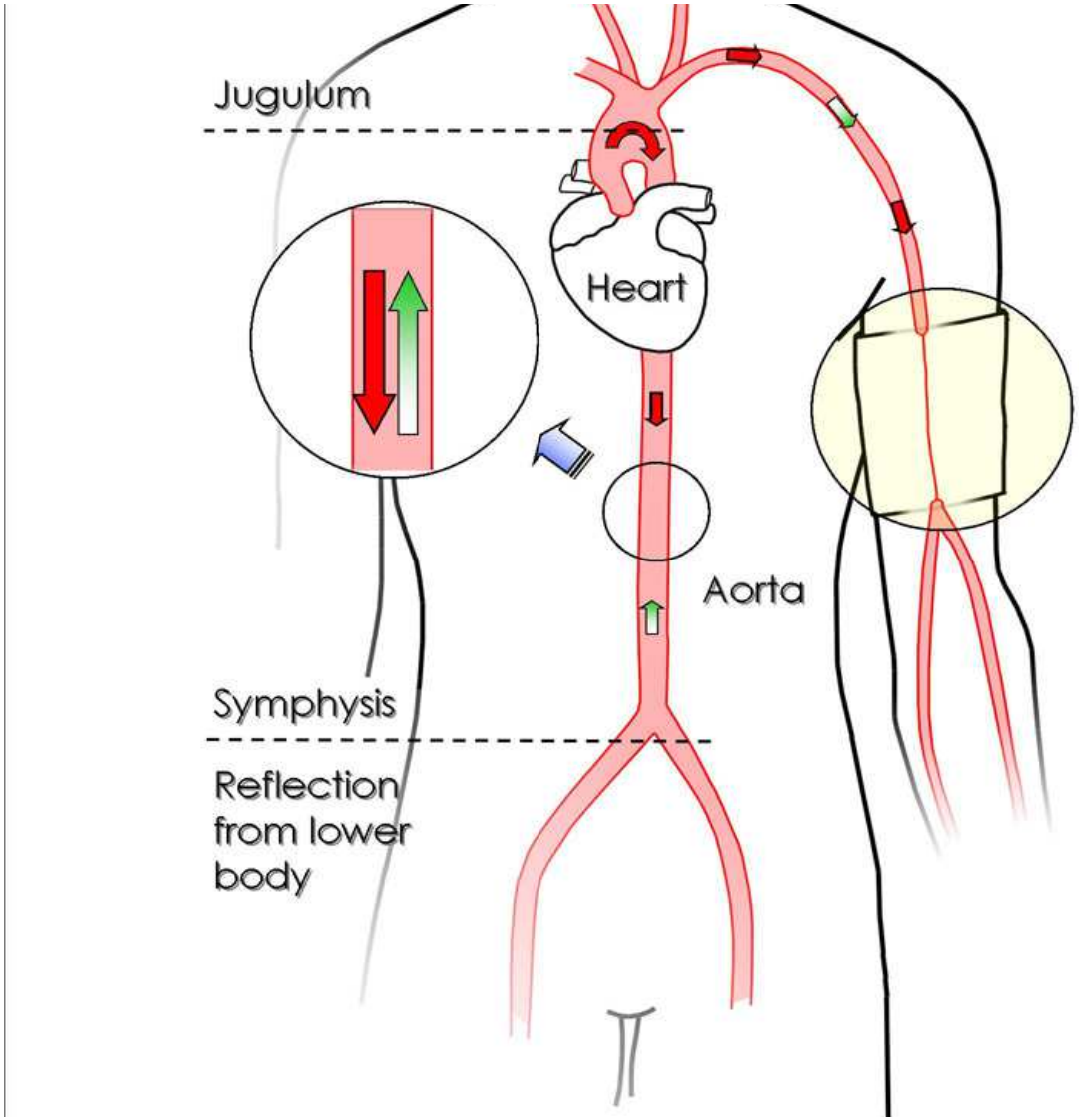
The **amplitude** of the reflected wave (P_2) is mainly determined by the actual peripheral arterial tone

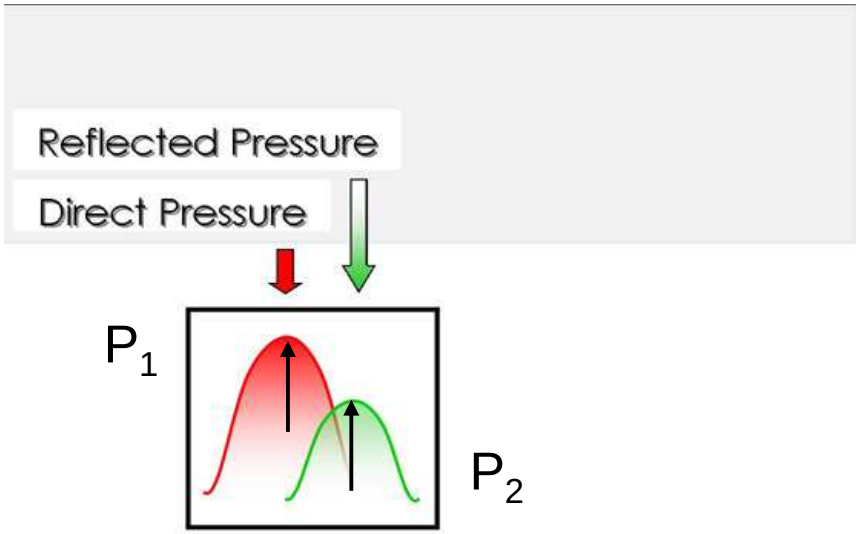
Sensor (Cuff)

AIX MEASUREMENT

Augmentation index (Aix) = $(P_2 - P_1 / PP) \times 100$

The principle of measuring aortic PWV with occlusive, oscillometric method





The **time** between the spikes of the direct (P_1) and reflected (P_2) pressure waves is equal with the transit time of the direct wave to the reflection site (bifurcation) and back.

Sensor (Cuff)

PWVao MEASUREMENT

PARAMETERS USED TO DESCRIBE ARTERIAL FUNCTION (STIFFNESS)

AORTIC PWV

RELATED TO THE AORTIC WALL STIFFNESS (RIGIDITY)

AUGMENTATION INDEX (aortic, carotid, brachial, radial)

BASICALLY RELATED TO THE VASCULAR TONE OF THE ARTERIOLES AND SMALL ARTERIES

CENTRAL SBP

RELATED TO THE PERIPHERAL (BRACHIAL) BP AND THE AIX (WAVE REFLECTION)

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

We conclude that **aortic PWV** 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.)

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 2.45 (1.29 to 4.66), respectively, after adjustment for age, gender, mean arterial pressure, and heart rate. Corresponding estimates for stroke were 1.22 (0.55 to 2.70) and 2.28 (1.05 to 4.96). Estimates decreased only slightly after adjustment

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

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, 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

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.)

Central (aortic) PP

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

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.

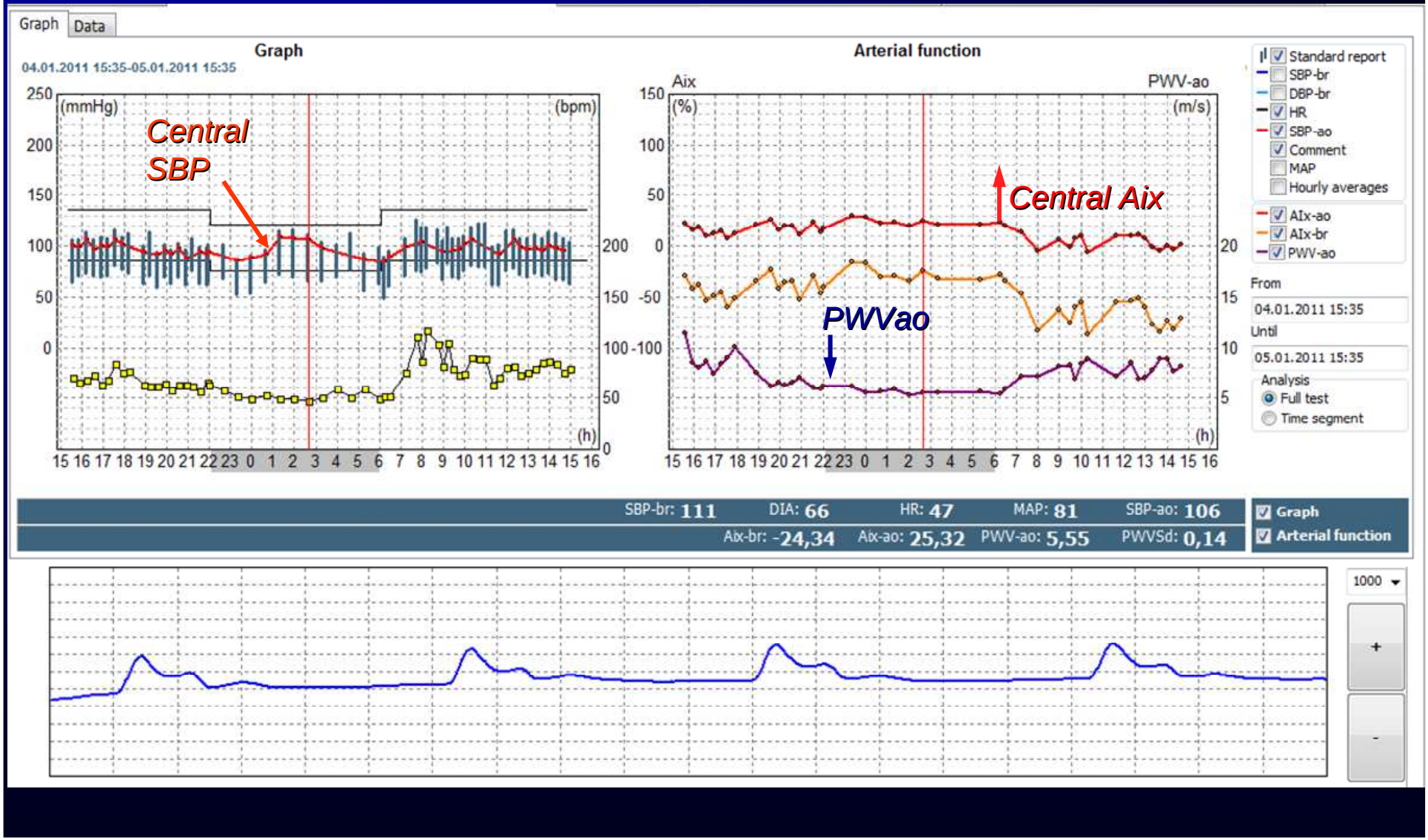
cholesterol:HDL ratio, creatinine, fibrinogen, diabetes, and heart rate, central pulse pressure predicted cardiovascular events more strongly than brachial pulse pressure (hazards ratio=1.15 per 10 mm Hg, χ^2=13.4, P<0.001 versus hazards ratio=1.10, χ^2=6.9, P=0.008). In conclusion, noninvasively-determined 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.)

BUT WHAT ABOUT 24h CHARACTERISTICS OF ARTERIAL STIFFNESS?

DOES ARTERIAL STIFFNESS HAVE CIRCADIAN RHYTHM?

FIRST RESULTS WITH ARTERIOGRAPH 24

The very first observations with 24h recording of arterial stiffness



The very first observations with 24h recording of arterial stiffness

Marked diurnal changes were seen

Awake

	SBP-br	DIA	MAP	PP		HR	
Mean	108	70	83	38	mmHg	74	/min
Max	126	83	95	50	mmHg	117	/min
Min	89	48	62	31	mmHg	49	/min
SD	8	7	7	4	mmHg	15	/min
DI	5	7	6	%			
PTE	0	0	0	%			
Load	0	0	0	mmHg*h			

	SBP-ao	PP-ao	
Mean	97	27	mmHg
Max	106	36	mmHg
Min	84	24	mmHg
SD	5	3	mmHg

	Aix-ao	Aix-br		PWV-ao	
Mean	10,91	-52,79	%	7,59	m/s
Max	25,98	-23,03	%	11,46	m/s
Min	-6,30	-86,79	%	5,44	m/s
SD	9,01	17,80	%	1,29	m/s

Asleep

	SBP-br	DIA	MAP	PP		HR	
Mean	103	65	78	38	mmHg	52	/min
Max	116	77	89	47	mmHg	59	/min
Min	87	52	64	32	mmHg	47	/min
SD	10	8	8	5	mmHg	4	/min
DI	5	7	6	%			
PTE	0	4	0	%			
Load	0	0	0	mmHg*h			

	SBP-ao	PP-ao	
Mean	97	32	mmHg
Max	109	40	mmHg
Min	85	28	mmHg
SD	10	4	mmHg

	Aix-ao	Aix-br		PWV-ao	
Mean	23,94	-27,06	%	5,66	m/s
Max	29,67	-15,74	%	6,19	m/s
Min	20,33	-34,19	%	5,23	m/s
SD	3,61	7,14	%	0,28	m/s

Preliminary results of the first study on the circadian rhythm of the arterial stiffness

Asklepeion Hospital Athens, Greece and University of Pécs, Hungary

		Mean	N	SD	P values
SBP brachial	daytime	130,2	21	16,3	<0,001
	nighttime ↓	118,6	21	13,9	
DBP brachial	daytime	80,8	21	13,8	<0,001
	nighttime ↓	70,4	21	11,5	
HR	daytime	70,5	21	8,7	<0,001
	nighttime ↓	62,2	21	8,5	
Aortic SBP	daytime	124,7	21	19,5	<0,001
	nighttime ↓	115,4	21	17,1	
Aortic Aix	daytime	24,4	21	16,7	<0,001
	nighttime ↑	**WHY?**			
Brachial Aix	daytime				<0,001
	nighttime ↑				
Aortic PWV	daytime	8,7	21	1,5	0,006
	nighttime ↓	8,1	21	1,8	

A. Manolis and M.Illyés: Unpublished data

$$R = \frac{U}{T}$$

$$TPR = \frac{MAP}{CO\ (HR*SV)}$$

Awake

	SBP-br	DIA	MAP	PP		HR	
Mean	108	70	83	38	mmHg	74	/min
Max	126	83	95	50	mmHg	117	/min
Min	89	48	62	31	mmHg	49	/min
SD	8	7	7	4	mmHg	15	/min
DI	5	7	6		%		
PTE	0	0	0		%		
Load	0	0	0		mmHg*h		

	SBP-ao	PP-ao	
Mean	97	27	mmHg
Max	106	36	mmHg
Min	84	24	mmHg
SD	5	3	mmHg

	Aix-ao	Aix-br		PWV-ao	
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Asleep

	SBP-br	DIA	MAP	PP		HR	
Mean	103	65	78	38	mmHg	52	/min
Max	116	77	89	47	mmHg	59	/min
Min	87	52	64	32	mmHg	47	/min
SD	10	8	8	5	mmHg	4	/min
DI	5	7	6		%		
PTE	0	4	0		%		
Load	0	0	0		mmHg*h		

	SBP-ao	PP-ao	
Mean	97	32	mmHg
Max	109	40	mmHg
Min	85	28	mmHg
SD	10	4	mmHg

	Aix-ao	Aix-br		PWV-ao	
Mean	23,94	-27,06	%	5,66	m/s
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Min	20,33	-34,19	%	5,23	m/s
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AWAKE

SBP = 108 DBP = 70

Aix = TPR MAP = 83

TPR = 1,24

CO = 6660

HR = 74 SV = 90

ASLEEP

SBP = 103 DBP = 65

Aix = TPR MAP = 78

TPR = 2,49

CO = 3120

HR = 52 SV = 60

Conclusions

On the basis of our preliminary results, we were able to describe first, that the arterial stiffness parameters exhibit marked circadian rhythm.

The augmentation index (Aix) increases very significantly during night (referring to the increased arterial resistance), while the aortic wall stiffness (aortic pulse wave velocity - PWVao) decreases significantly (referring to the diminished lateral tension against the aortic wall due to the reduced cardiac output and BP).

The observed phenomenon might be explained by the physiological adaptation of the circulation to the reduced cardiac performance during night i.e. during inactive, sleeping period.

Thanks for your attention!