Central versus Peripheral Blood Pressure

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- Central Artery vs Peripheral Artery
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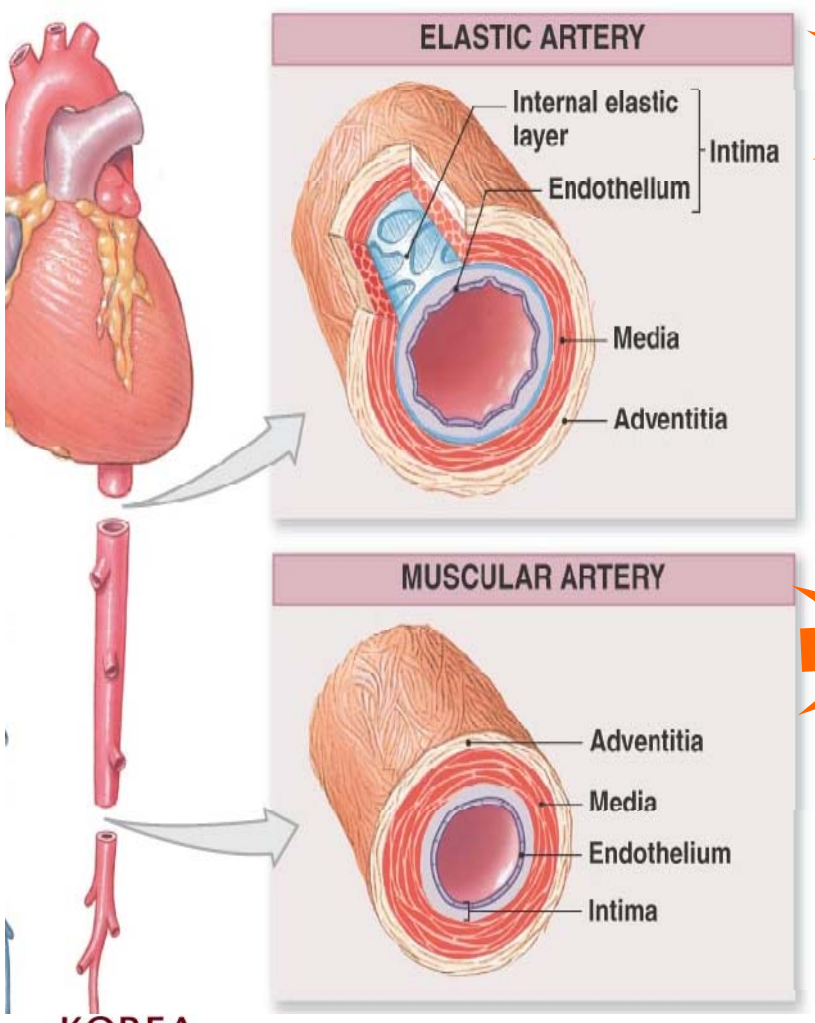
Central vs. Peripheral artery



- **Central artery**

  :aorta, carotid artery

- **Peripheral artery**

  : brachial, radial artery

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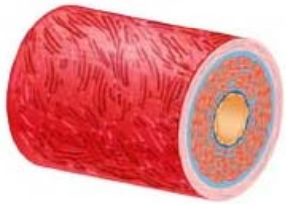
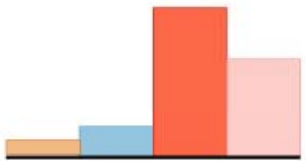
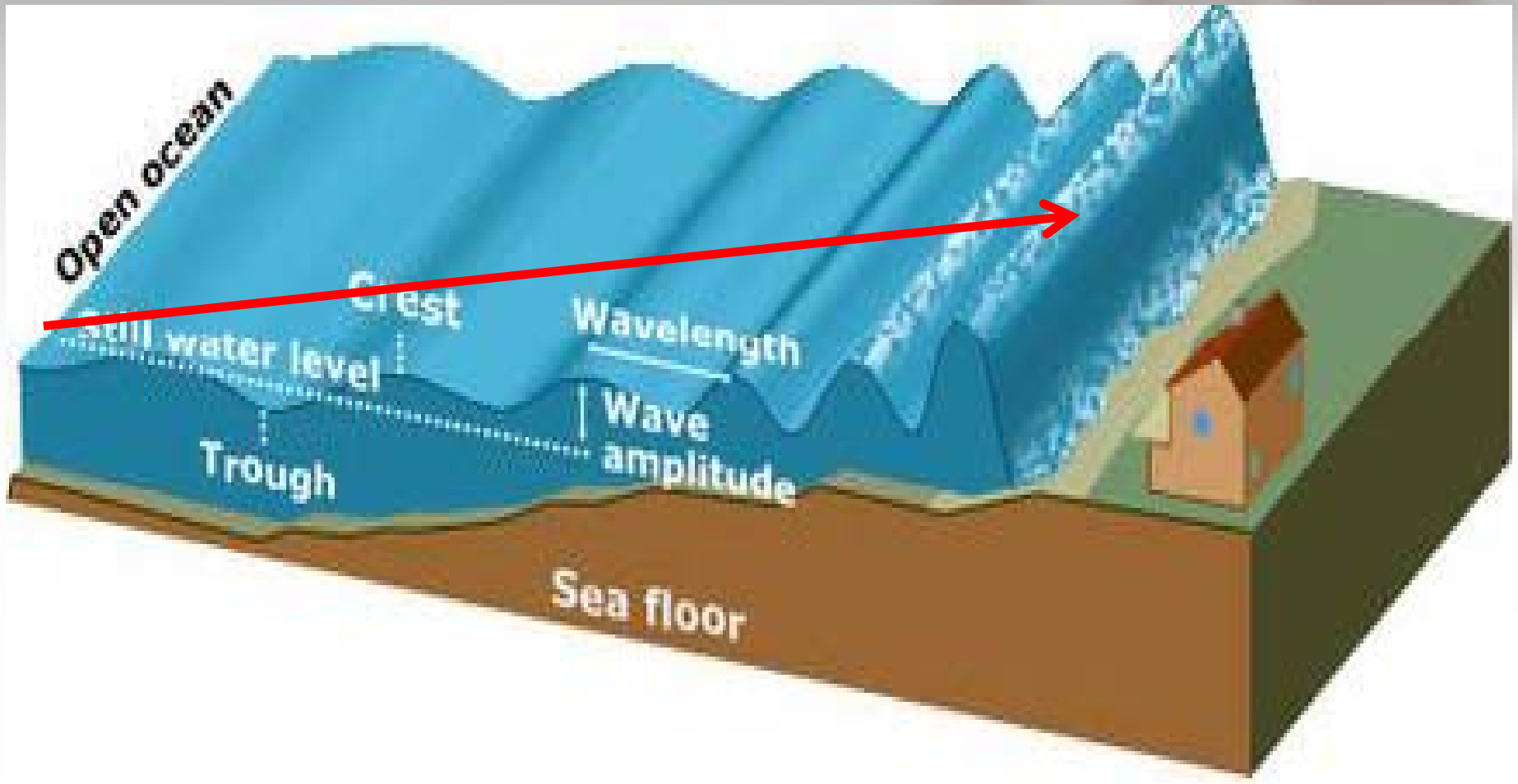
Central Vs Peripheral Artery

VESSEL TYPE/ ILLUSTRATION*	AVERAGE LUMEN DIAMETER (D) AND WALL THICKNESS (T)	RELATIVE TISSUE MAKEUP
 Elastic artery	D : 1.5 cm T : 1.0 mm	
 Muscular artery	D : 6.0 mm T : 1.0 mm	

- **Elastic (conducting) arteries**
  - more elastic fibers,
  - less smooth muscle
  - lose elasticity with aging
- **Muscular (distributing) arteries**
  - deliver blood to most organs
  - more smooth muscle cells
  - fewer elastic fibers

*. Benetos A et al. *Arterioscler Thromb.* 1993 13:90-7.
*. Avolio AP et al. *Circulation.* 1983;68:50 -58.

Blood Pressure Amplification



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Blood Pressure Amplification

8 m/sec

Young normal aorta

12 m/sec

Old stiff aorta

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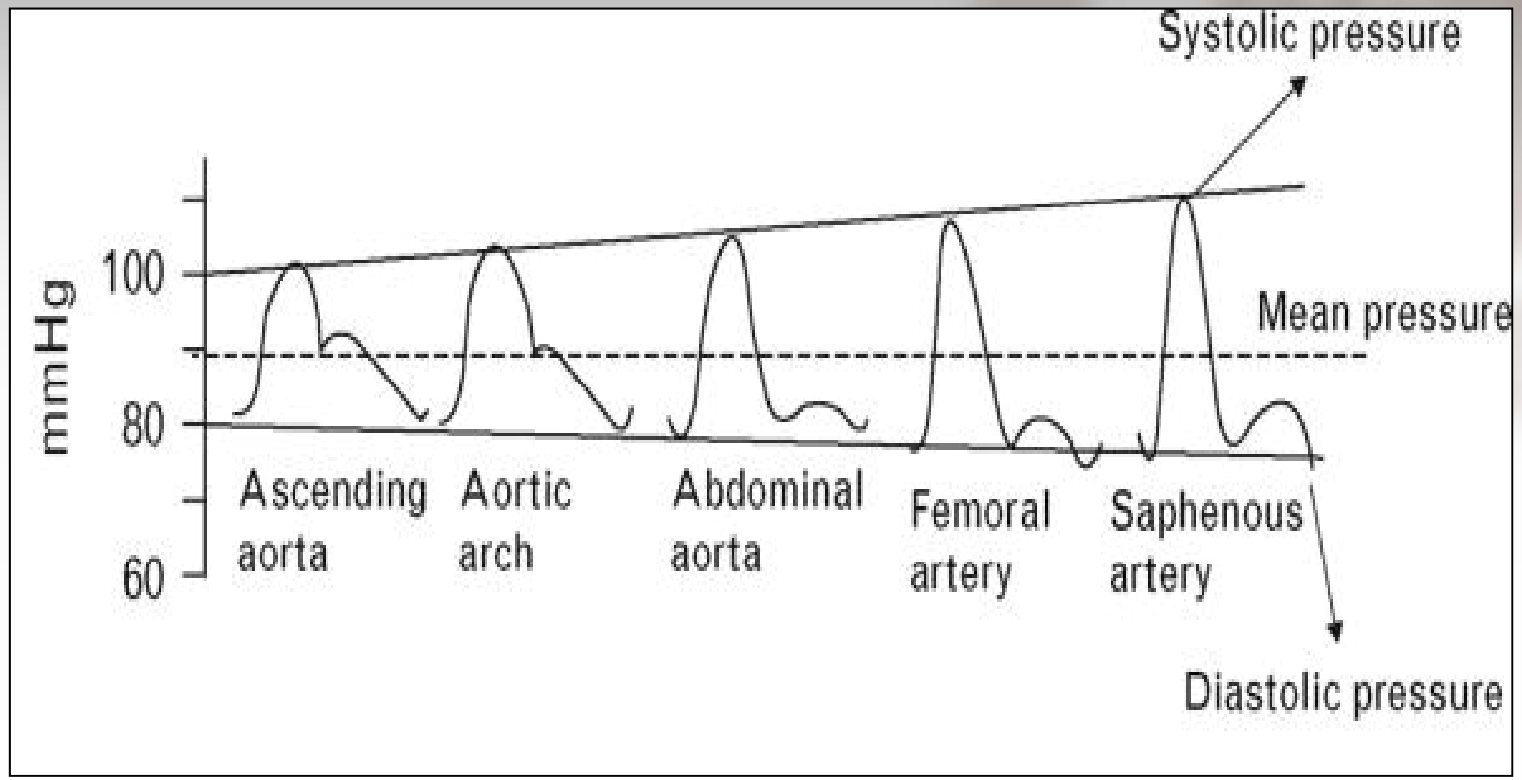
Blood Pressure Amplification



It is a traditional observation that BP waves differ markedly between central and peripheral sites of the arterial bed

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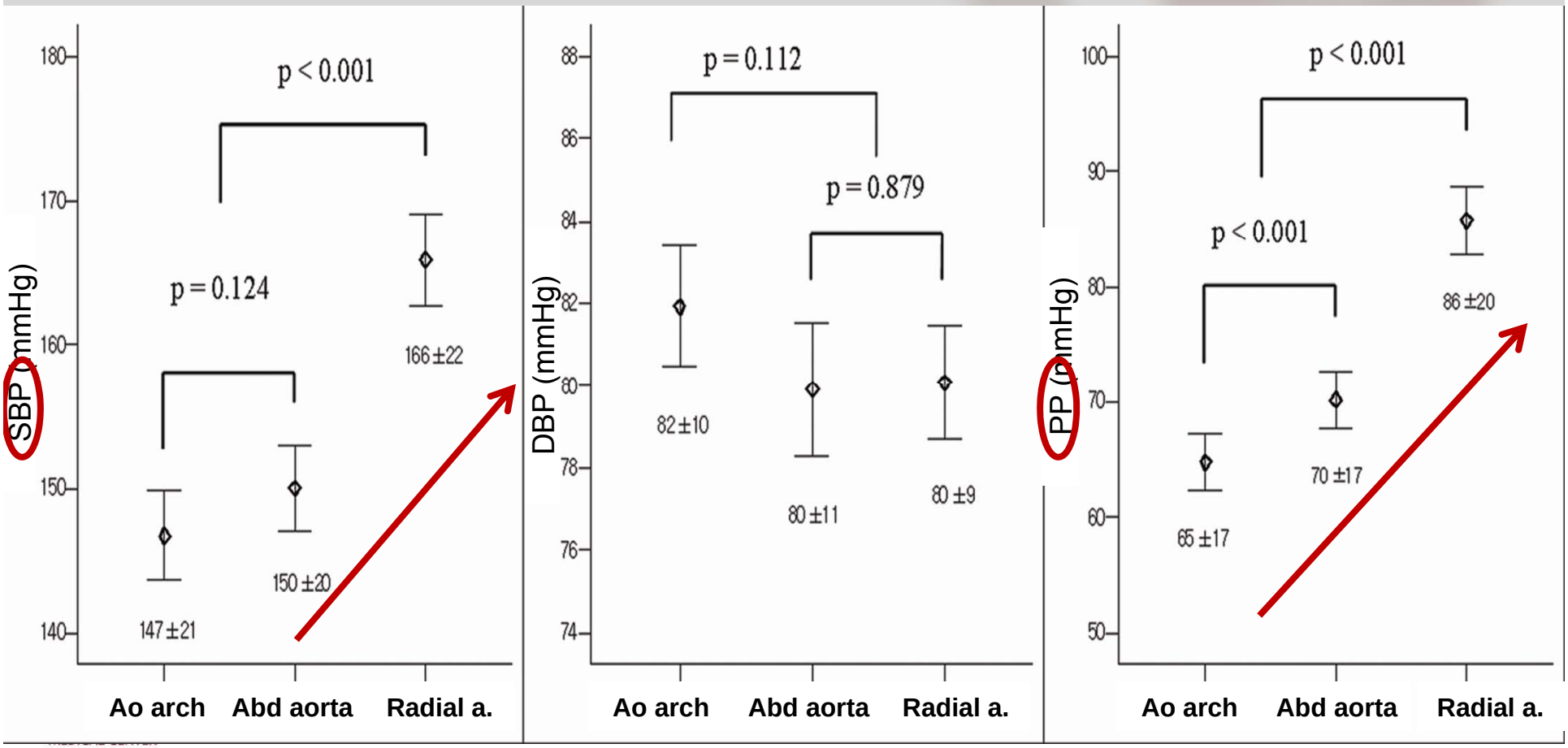
Journal of Hypertension. 2007;25:265-272

Blood Pressure in Each Arterial Tree.



Choi CU, Kim EJ et al. *J Hypertens* 2010 (In press).

Limitation of Brachial BP

◆ In younger people

- Overestimate the central systolic and pulse pressure

◆ In elderly

- Tachycardia, systolic heart failure, exercise, drug

➔ Difference between brachial pressures and central aortic pressures

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Clinical Implication of Central BP

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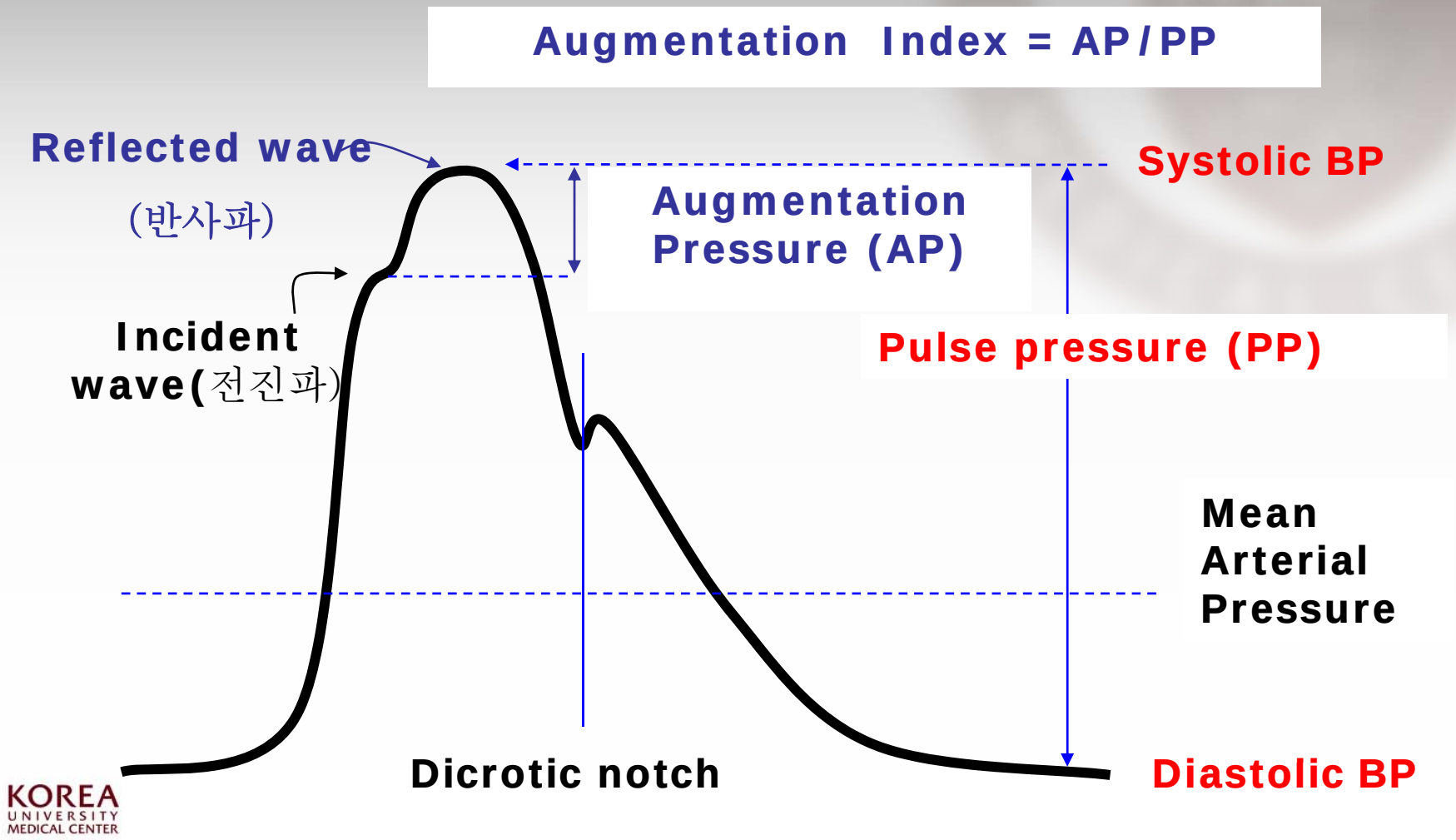
Central Arterial Pulse Wave



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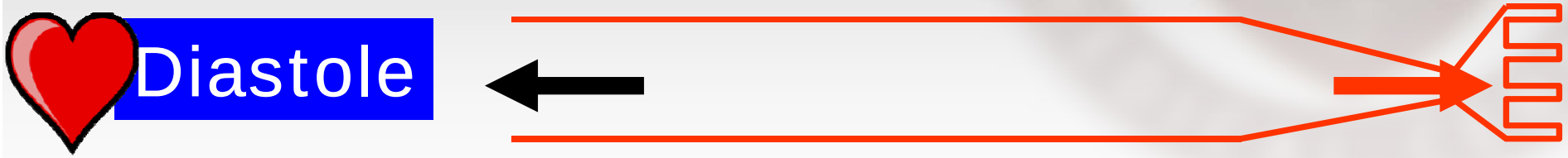
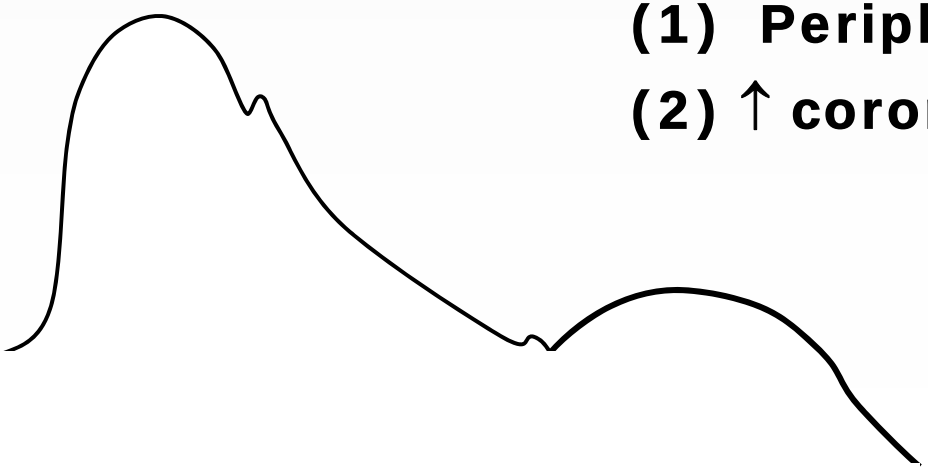
Pulse Wave Reflection

Young compliant arteries :
normal PW velocity 8 m/sec





(1) Peripheral amplification

(2) ↑ coronary blood flow

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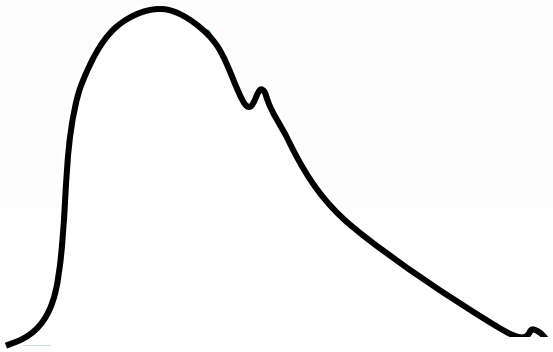
Pulse Wave Reflection

Elderly stiff arteries with ISH : increased PW velocity 12 m/sec



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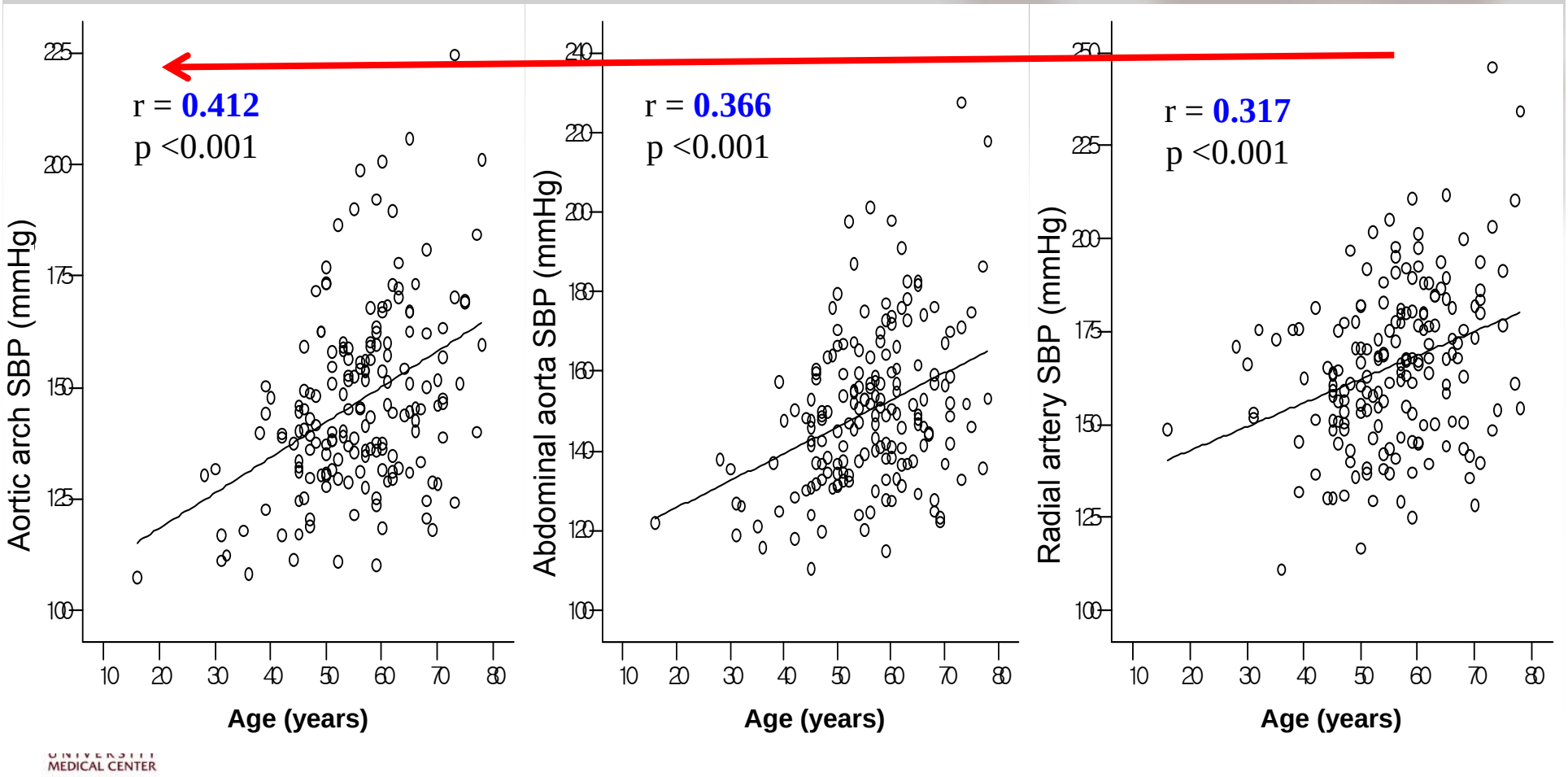
(1) Ventricular-vascular mismatch

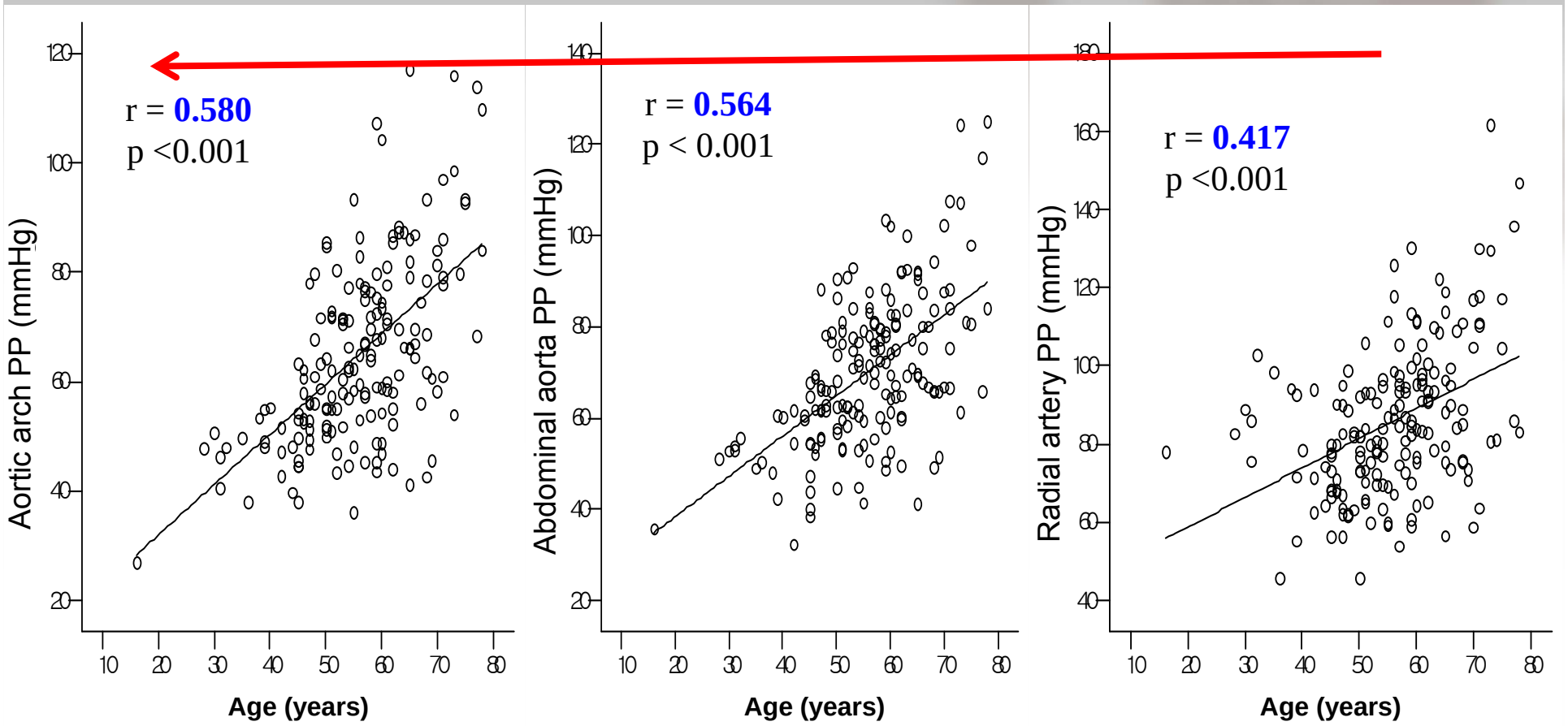
(2) The reflected wave augments aortic BP in late systole

Correlation between Ageing and SBP in Each Arterial Tree



Choi CU, Kim EJ et al. *J Hypertens* 2010 (In press).

Correlation between Ageing and PP in Each Arterial Tree



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Choi CU, Kim EJ et al. *J Hypertens* 2010 (In press).

Clinical Implication of Central BP

- **Increased Aortic SBP**
  - Increased vascular afterload and developed LVH
  - Decreased coronary perfusion pressure
  - Increased MVO2 and subendocardial ischemia
  - Increased endothelial dysfunction and atherogenesis

Clinical Implication of Central BP "Beyond BP Control"

- **HOPE, LIFE, ANBP2 studies**
- "**Beyond BP control**" are perhaps associated with arterial properties or central BP

Appropriateness of Measuring Central Pressure

- Brachial BP is not the perfect surrogate for central BP

- Central BP should be more closely related to the pathophysiology of cardiovascular disease

•Nichols. WW et al. Theoretical, Experimental and Clinical Principles. Fifth Edition. 2005:193-213, 339-386
•Safar. ME et al. Handbook of Hypertension: Arterial Stiffness and Wave Reflection. 2005.

Measurement of Central BP

Measurement of Central BP

◆Invasive method

- Fluid Field Catheter
- High Fidelity Catheter Micro-tip Pressure Transducer

◆Non-invasive methods

- Applanated arterial tonometry
- Echo-Tracking
- Plethysmography

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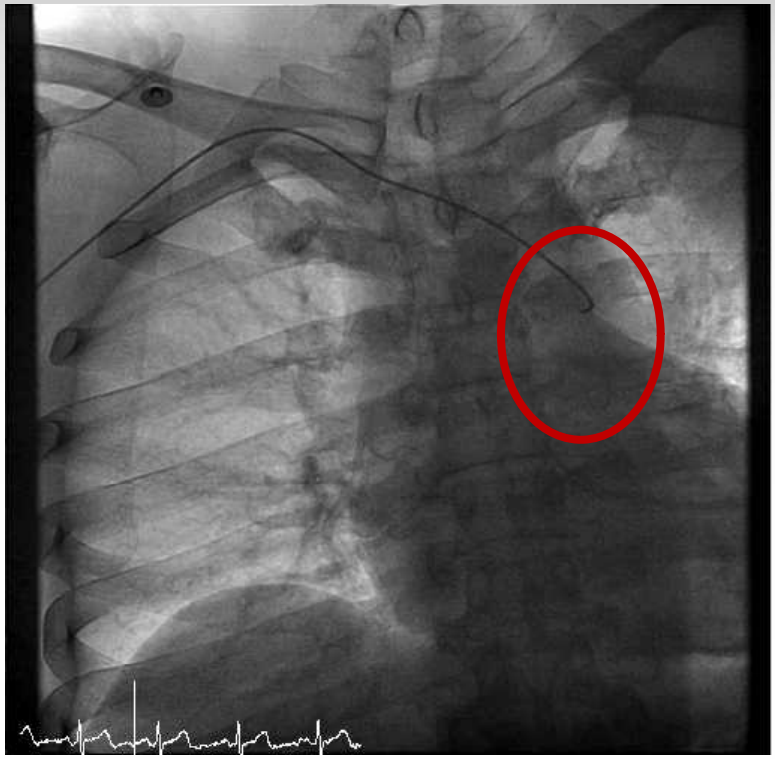
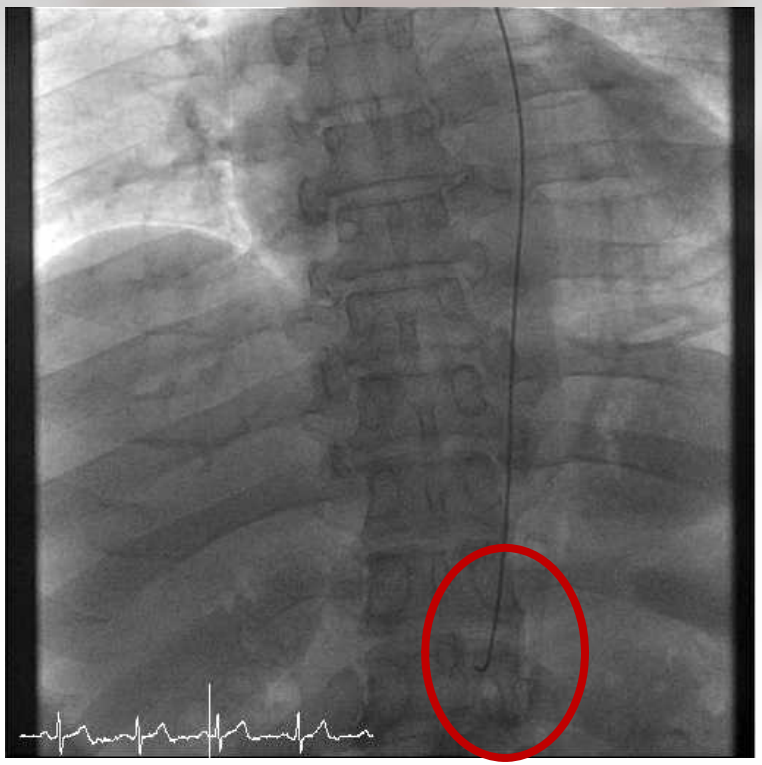
Invasive Method

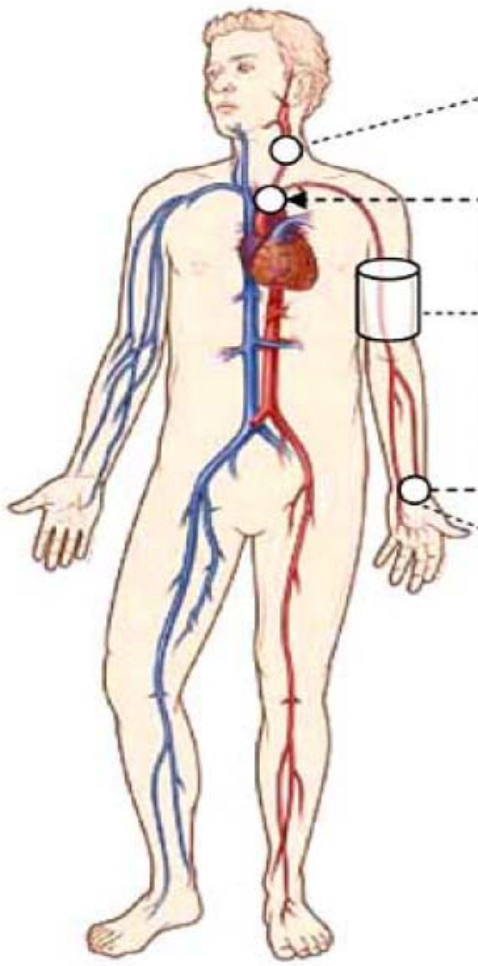
Aortic arch



Abdominal aorta



Non-invasive Method



Recording of carotid BP

Direct estimation of central pressures by applanation tonometry or echo-tracking.

Recording of brachial BP

Statistical derivation of central pressures by using regression equations.

Recording of radial BP

a) Indirect estimation of aortic pressures by applying generalized transfer functions to radial pressure waves.

b) Estimation of aortic systolic pressure by detection of 2nd systolic peak of the radial artery.

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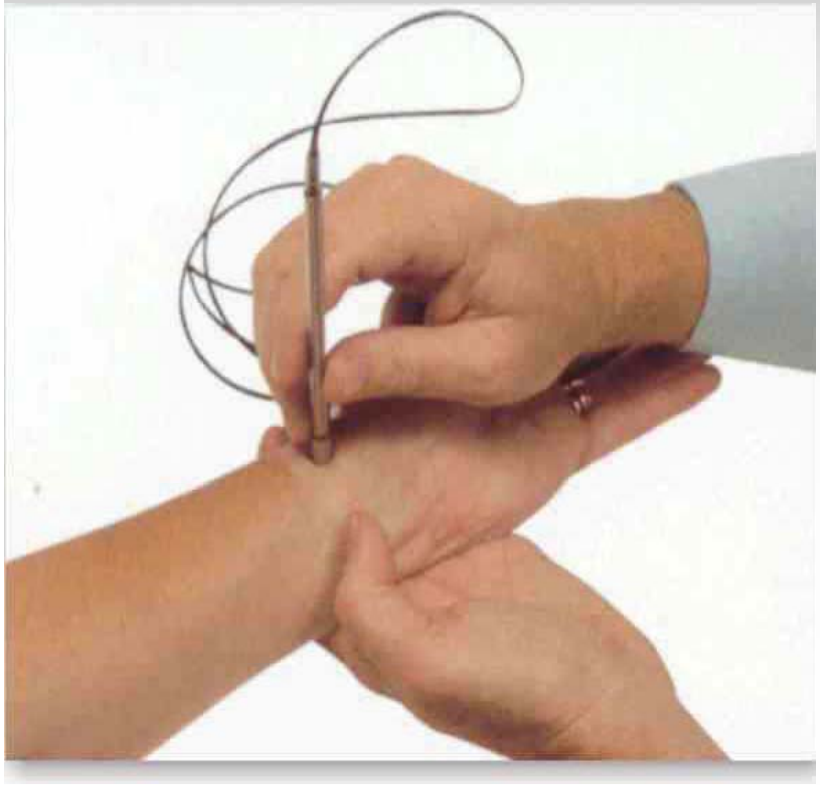
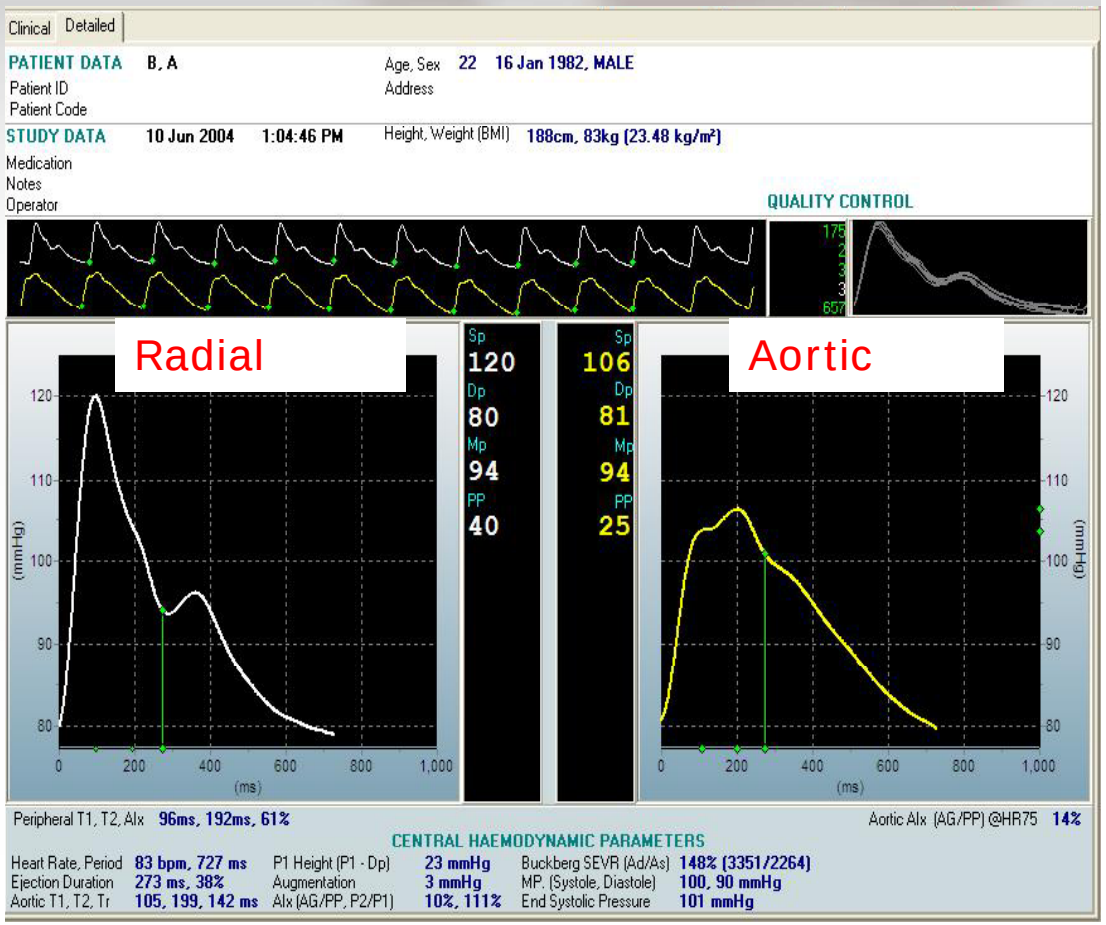
Current Pharmaceutical Design, 2009, 15, 245-253

Non-invasive Method (Sphygmocor)





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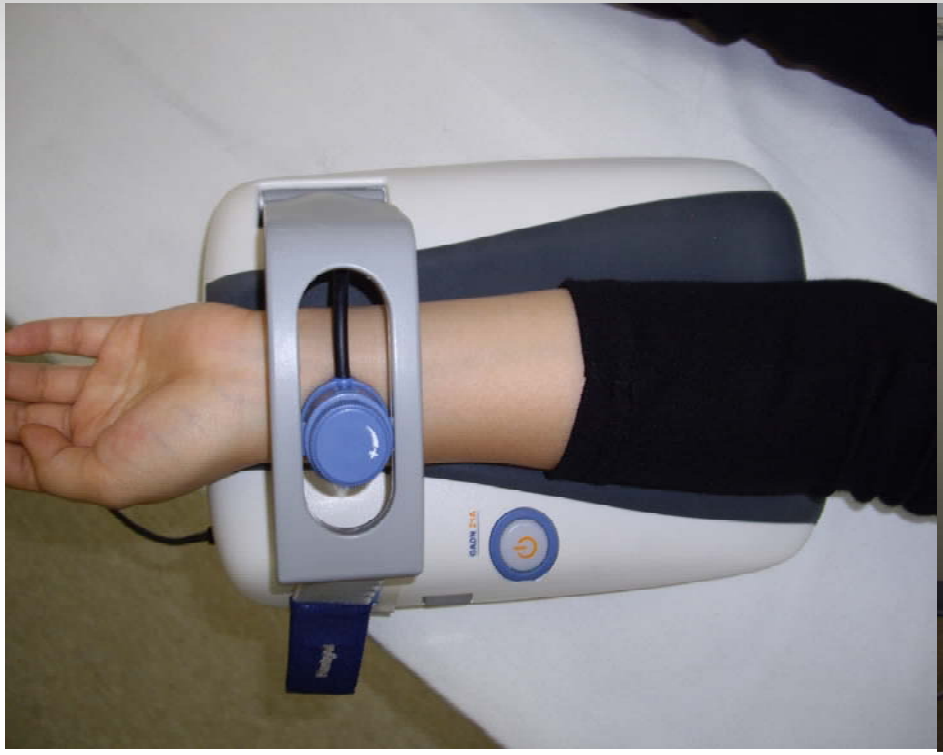
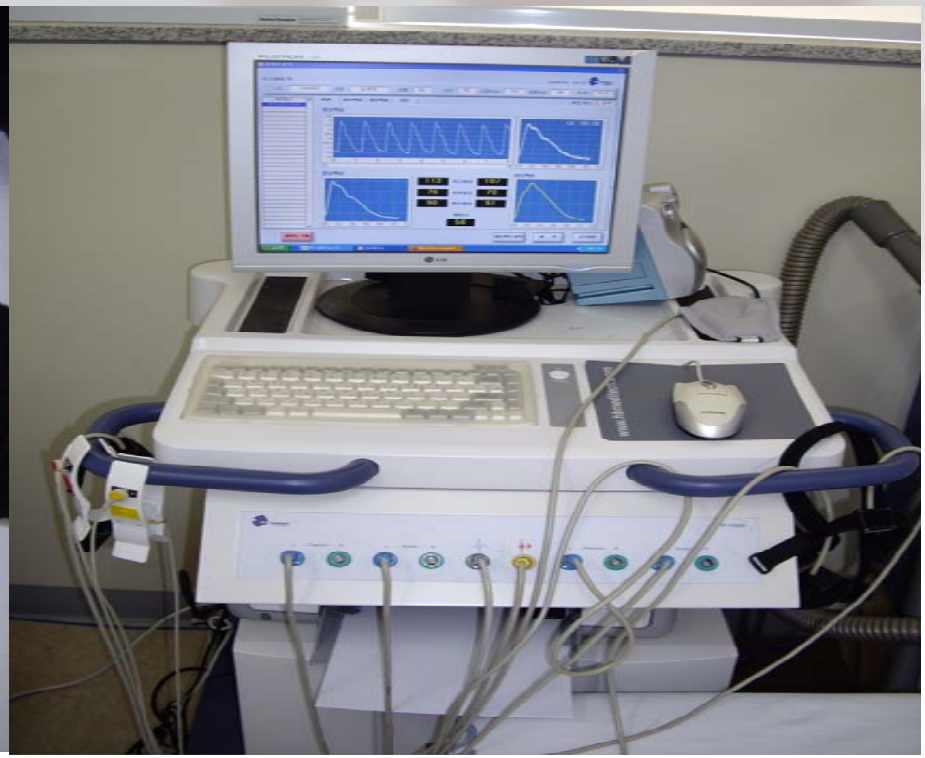
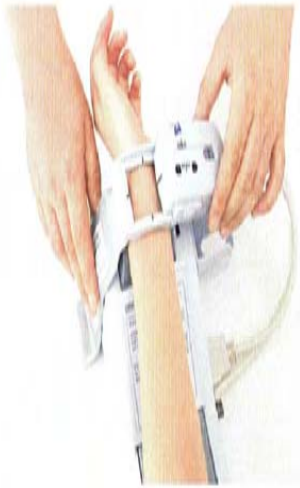
Non-invasive Method (GAON, Korea)

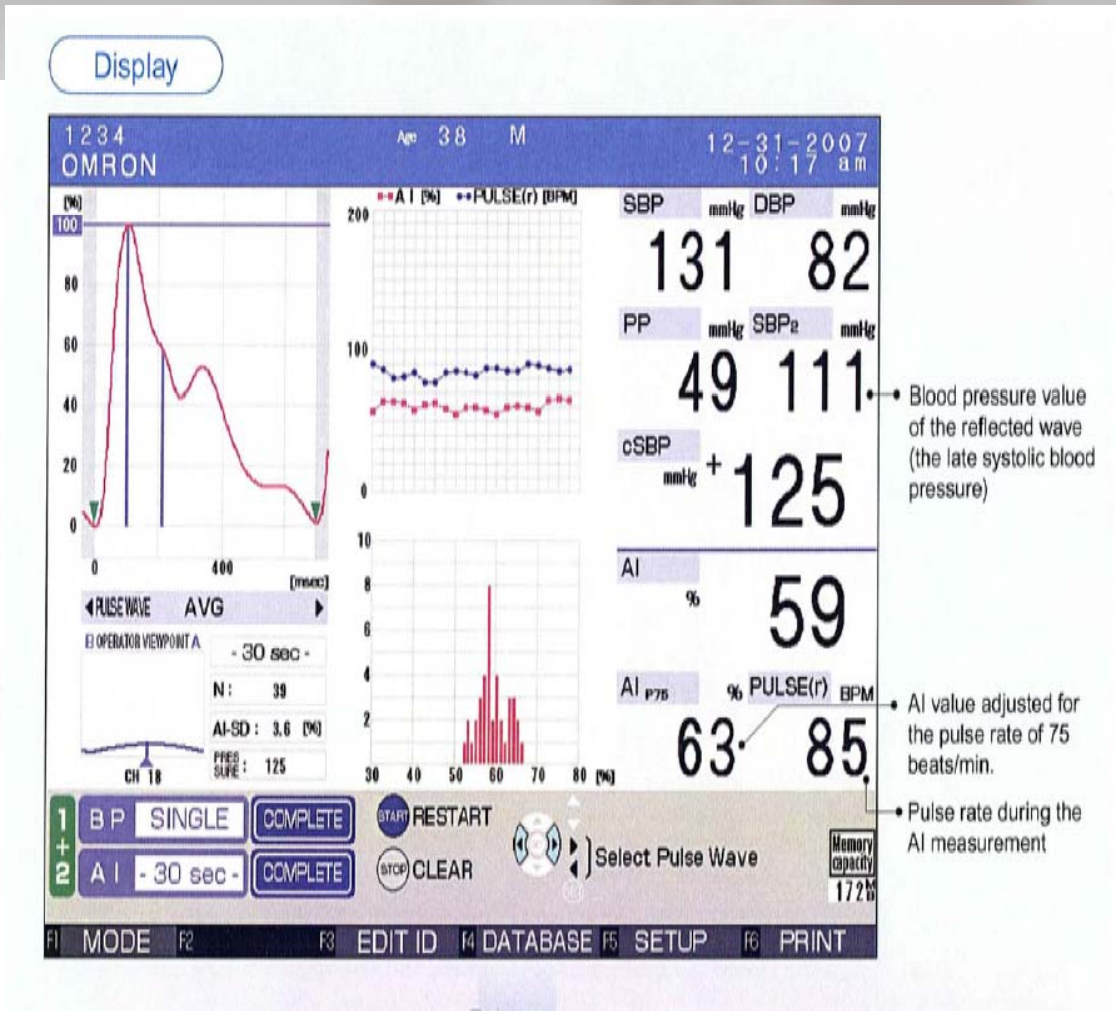




Non-invasive Method (HEM-9000AI, Japan)

Desksize suits for ambulatory practice with simple procedure





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Central Pressure as Marker of Disease

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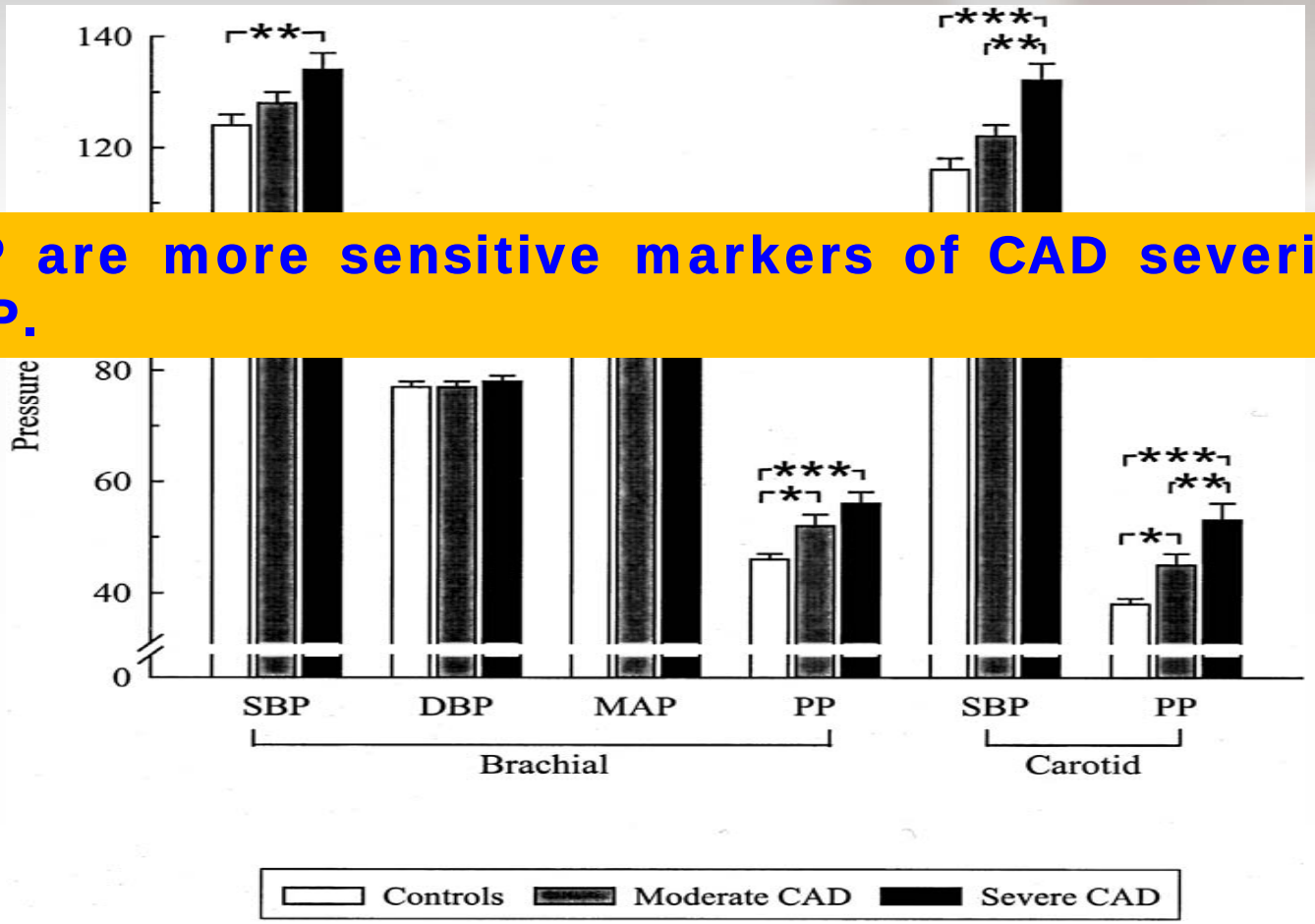
Carotid Pressure and CAD Severity

patients with CAD



Central BP are more sensitive markers of CAD severity than brachial BP.

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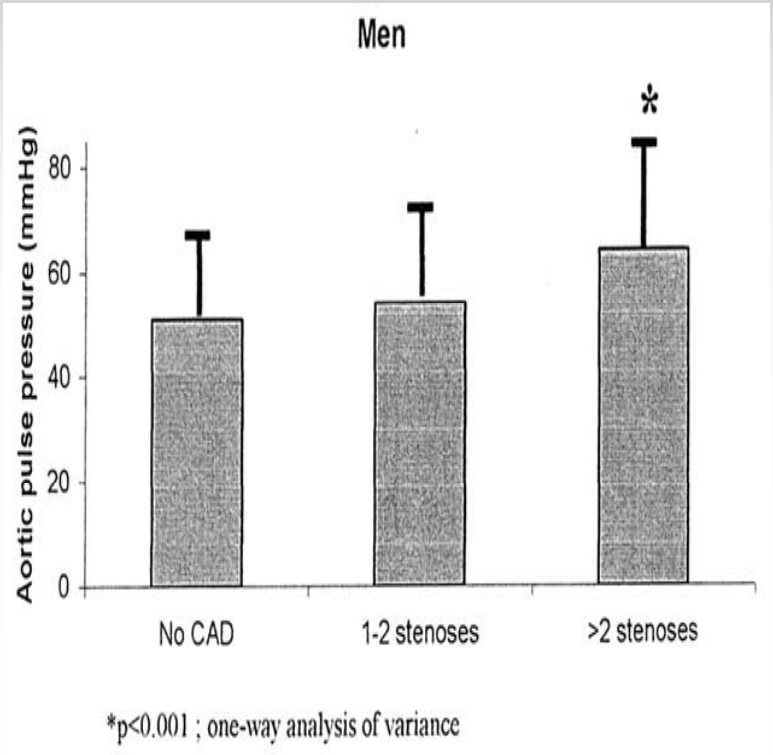
Waddell, T. K. et al. Hypertension 2001;38:927-931

Aortic PP and Extent of CAD

99 patients with CAD



*p<0.001 ; one-way analysis of variance

Table 3. Multiple logistic regression analysis of prediction of coronary artery disease

Characteristic	Relative Risk	95% CI	*P* Value
Men			
Age	1.04	1.00–1.07	.030
Pulse pressure (for a 1-mm Hg increase)	1.02	1.00–1.04	.043
Hypercholesterolemia	1.84	0.99–3.43	.053
Women			
Age	1.04	1.00–1.09	.046
PVD	9.47	0.94–95.5	.057

PVD = peripheral vascular disease.

aortic PP was significantly correlated with the presence and extent of CAD

Danchin N et al. Am J Hypertens. 2004;17:129-33.

Central Pressure and Vascular Disease (Strong Heart Study)

TABLE 2. Relations of Central and Brachial Blood Pressures and Arterial Stiffness to Carotid Hypertrophy and Extent of Atherosclerosis*

Variable	Intimal-Medial Thickness	Vascular Mass	Plaque Score
P value, brachial PP vs brachial SBP†	<0.001	<0.02	<0.001
P value, central PP vs central SBP†	<0.001	ns	<0.001
P value, central vs brachial SBP†	<0.001	<0.001	<0.001
P value, central vs brachial PP†	<0.002	<0.05	<0.001
P value, arterial stiffness vs brachial SBP†	<0.005	<0.001	<0.001
P value, arterial stiffness vs brachial PP†	ns	ns	<0.02

SBP indicates systolic blood pressure; PP, pulse pressure; ns, not significant.
*All correlations $P<0.001$.
†Correlations compared by Z statistics.

Central BP is more strongly related to vascular hypertrophy and extent of atherosclerosis than is brachial BP

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Roman MJ et al.Hypertension. 2007;50:197-203.

Central PP and Cardiac Mass

The greater change in LVM on Per/Ind was linked to central and not brachial BP.

Left ventricular mass change (%) — **469 hypertensive patients**

Adjustment (Adj) with	Adj mean (Per/Ind) (95% CI)	Adj mean (atenolol) (95% CI)	Adj mean (Per/Ind – atenolol) (95% CI for difference)
Carotid SBP (Model 1)	−6.6 (−10.2 to −3.0)	−1.4 (−5.7 to +2.8)	−5.17 (−10.88 to +0.53)
Brachial SBP (Model 2)	−6.2 (−9.9 to −2.5)	−2.0 (−6.4 to +2.4)	−4.19 (−10.00 to +1.62)
Carotid PP (Model 3)	−7.2 (−10.8 to −3.6)	−0.7 (−4.9 to +3.6)	**−6.50 (−12.31 to −0.71)**
Brachial PP (Model 4)	−6.3 (−10.0 to −2.6)	−1.8 (−6.2 to +2.6)	−4.53 % (−10.48 to +1.42)

Adjusted (Adj) means [95% confidence interval (CI)] (derived from the general linear model of Table 4) for: left ventricular mass (LVM) change (%) in the Per/Ind (perindopril and indapamide) group; LVM change (%) in the atenolol group; and LVM change (%) in the (Per/Ind – atenolol) groups. Only model 3 (carotid PP) reaches statistical significance. SBP, systolic blood pressure; PP, pulse pressure.

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de Luca N et al. J Hypertens. 2004;22:1623-30.

Central Pressure as Predictors of Events

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Central Aortic Pressure and Clinical Outcomes (CAFE study)

Aortic PP may be a determinant of clinical outcomes

End Point in the CAFE Cohort

	χ^2	*P*	HR	95 % CI
Updated Cox proportional-hazards model adjusted for age and baseline risk factors				
Model 1 (305 events)				
Peripheral PP	3.83	0.050	1.10	1.00–1.22
Central PP	3.91	0.048	1.11	1.00–1.23
Augmentation	2.26	0.133	1.14	0.96–1.36
P_1 height	3.04	0.081	1.17	0.98–1.40
Model 2 (245 events)				
Peripheral PP	4.5	0.034	1.12	1.01–1.24
Central PP	5.0	0.026	1.13	1.02–1.26
Augmentation	4.2	0.040	1.21	1.01–1.45
P_1 height	2.5	0.114	1.16	0.96–1.40
Model 3 (225 events)				
Peripheral PP	4.1	0.044	1.12	1.00–1.25
Central PP	4.1	0.043	1.13	1.00–1.26
Augmentation	3.1	0.080	1.18	0.98–1.43
P_1 height	2.4	0.118	1.17	0.96–1.42

patients with hypertension

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Williams B et al. Circulation. 2006;113:1213-25

Central pressure and Clinical Outcome (Strong Heart Study)

TABLE 4. Multivariable Cox Models of Relation of Traditional Risk Factors and Central and Brachial Blood Pressures to Cardiovascular Outcome

Variable	HR (95% CIs)	HR (95% CIs)	HR (95% CIs)	HR (95% CIs)	HR (95% CIs)
Age, year	1.06 (1.04–1.07)*	1.05 (1.04–1.07)*	1.06 (1.04–1.07)*	1.05 (1.03–1.07)*	1.05 (1.04–1.07)*
Male gender	1.13 (0.87–1.45)	1.17 (0.91–1.52)	1.13 (0.88–1.46)	1.22 (0.94–1.58)	1.10 (0.83–1.45)
BMI, kg/m^2	0.99 (0.97–1.01)	0.99 (0.97–1.01)	0.99 (0.97–1.01)	0.99 (0.97–1.01)	0.99 (0.97–1.01)
Diabetes mellitus	2.48 (1.91–3.22)*	2.44 (1.88–3.17)*	2.47 (1.91–3.21)*	2.41 (1.86–3.13)*	2.42 (1.838–3.22)*
Heart rate, bpm	1.012 (1.001–1.022)‡	1.013 (1.002–1.023)‡	1.013 (1.008–1.143)‡	1.012 (1.001–1.022)‡	1.013 (1.001–1.025)‡
Brachial SBP	1.08 (1.02–1.14)‡				
Brachial PP		1.10 (1.03–1.18)†			
Central SBP			1.07 (1.01–1.14)‡		
Central PP				1.15 (1.07–1.24)*	
Arterial stiffness					1.06 (1.01–1.11)‡

All blood pressures per 10 mm Hg.
BMI indicates body mass index; SBP, systolic blood pressure; PP, pulse pressure.
*P<0.001; †P<0.01; ‡P<0.05; §P<0.005.

Central PP was more strong predictor of CVE than brachial PP

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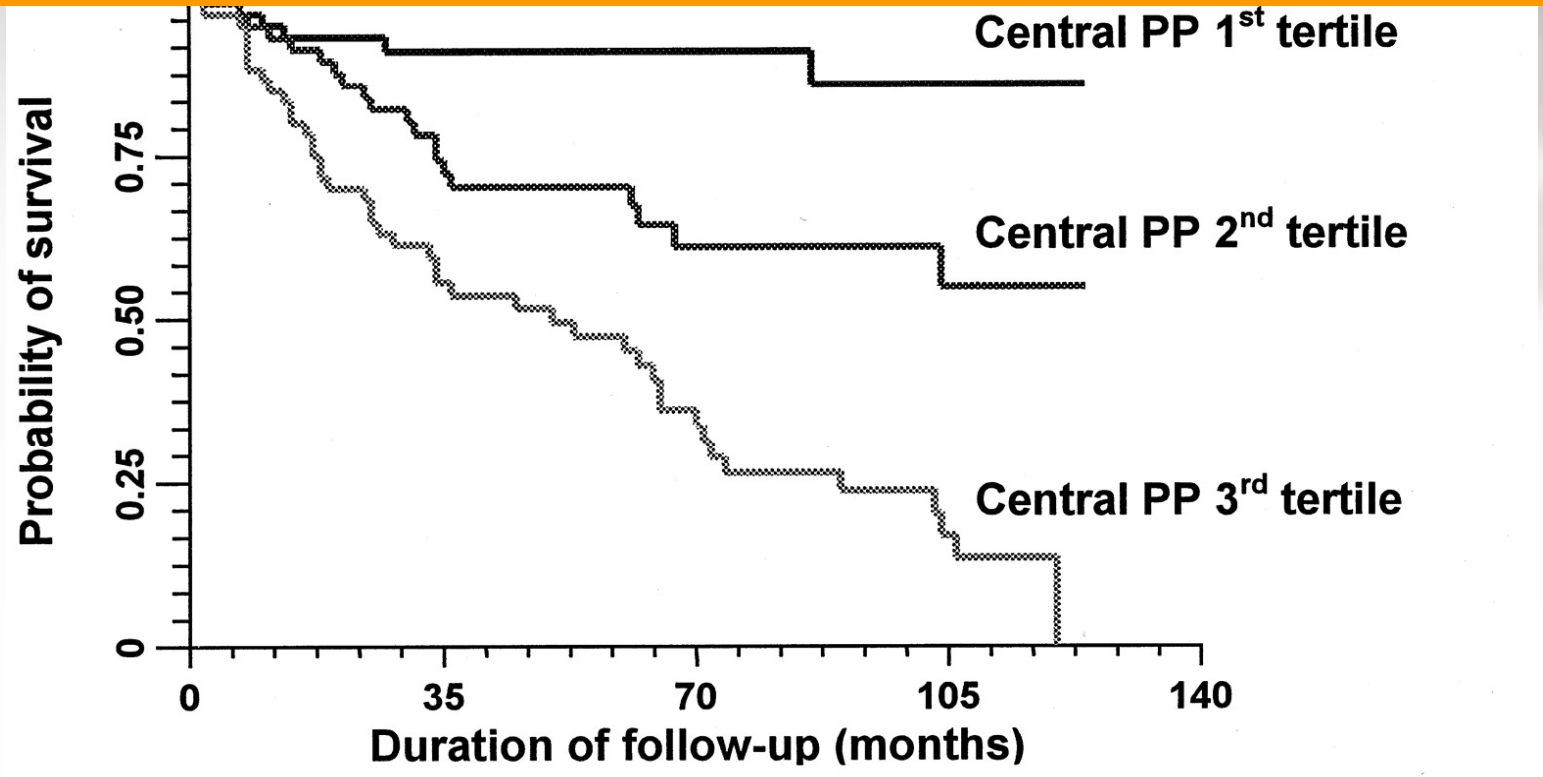
Roman MJ et al. Hypertension. 2007;50:197-203

Central PP and Mortality in ESRD

Carotid PP are strong independent predictors of all-cause mortality.



Probabilities of survival in the study population according to the level of central PP and PP amplification divided into tertiles

Safar ME et al.Hypertension. 2002;39:735-8

Aortic Pressures and Mortality

Patients With CAD

Predictors of all-cause mortality (n = 324)

Predictor*	Hazard Ratio (95% CI)	p Value
PP (per 10 mm Hg increase)*	1.18 (1.05–1.33)	0.004
Diastolic BP (per 10 mm Hg increase)	0.76 (0.62–0.94)	0.01
Peripheral vascular disease	1.96 (1.1–3.51)	0.02
Diabetes mellitus	1.87 (1.15–3.06)	0.01
Congestive heart failure	2.43 (1.42–4.16)	0.001
Left ventricular ejection fraction (per 10% increase)	0.74 (0.63–0.86)	<0.001
Aspirin use	0.59 (0.36–0.97)	0.03
Statin use	0.52 (0.31–0.90)	0.01
Hematocrit (per 10-point increase)	0.65 (0.48–0.90)	0.008
Digoxin use	2.41 (1.36–4.26)	0.002
Serum Creatinine (per mg/dl increase)	1.17 (1.04–1.31)	0.007
Modified Charlson score (per point increase)	1.10 (1.05–1.16)	<0.001

Aortic PP significantly predicted death

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Chirinos JA et al. Am J Cardiol. 2005;96:645-8.

Aortic arch PP & CAD in Chest Pain Pts

	Multivariate analysis		
	OR	95% CI	p
Age	1.071	1.022-1.123	0.004
Sex	3.940	1.632-9.512	0.002
DM	2.734	1.080-6.921	0.034
aoPWV			
Tertile 1, ≤720cm/s	1		
Tertile 2, 721-1044cm/s	2.740	1.036-7.245	0.042
Tertile 3, >1044cm/s	3.680	1.181-11.460	0.025
Abdominal PP	0.951	0.904-1.001	0.055
Aortic arch PP	1.058	1.008-1.111	0.022

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*. Multivariate analysis was performed after adjusting for age, sex, DM, dyslipidemia, HTN, smoking, **aoPWV, arPWV, radial PP, abdominal aorta PP and aortic arch PP.**

Choi CU, Kim EJ et al. *J Hypertens* 2010 (In press).

Central BP and Cardiovascular Events (ICARe Dicomano Study)

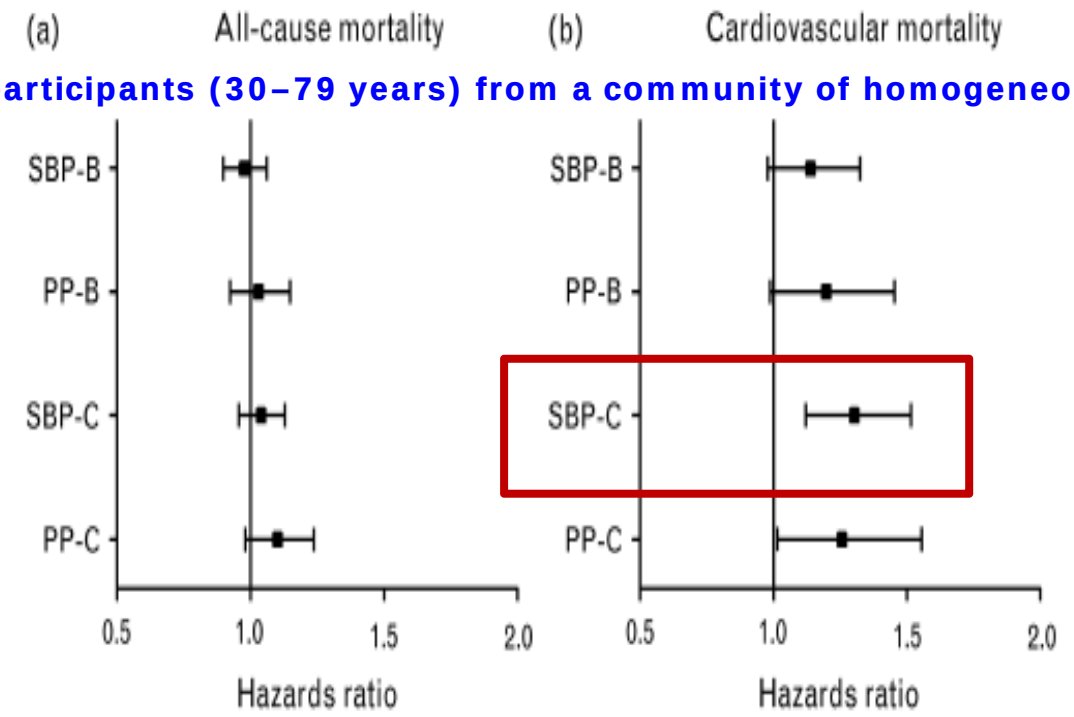
Table 6 Independent Predictors of Cardiovascular Events In Unselected Geriatric Population

	HR (95% CI)	p Value	HR (95% CI)	p Value	HR (95% CI)	p Value	HR (95% CI)	p Value
Brachial SBP (/10 mm Hg)		0.119						
Brachial PP (/10 mm Hg)				0.063				
Carotid SBP (/10 mm Hg)					1.19 (1.08-1.31)	<0.0001		
Carotid PP (/10 mm Hg)							1.23 (1.10-1.37)	<0.0001

Carotid BP was more strong predictor of CVE than brachial BP

Pini R et al. J Am Coll Cardiol. 2008;51:2432-9.

Central Pressure and Future Mortality



1272 participants (30–79 years) from a community of homogeneous Chinese.

Central SBP is more valuable than other BP variables in predicting CV mortality.

pulse pressure; SBP-B, brachial systolic blood pressure; SBP-C, central systolic blood pressure.

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Wang KL et al. J Hypertens. 2009;27:461-7.

Central PWV and Clinical Data

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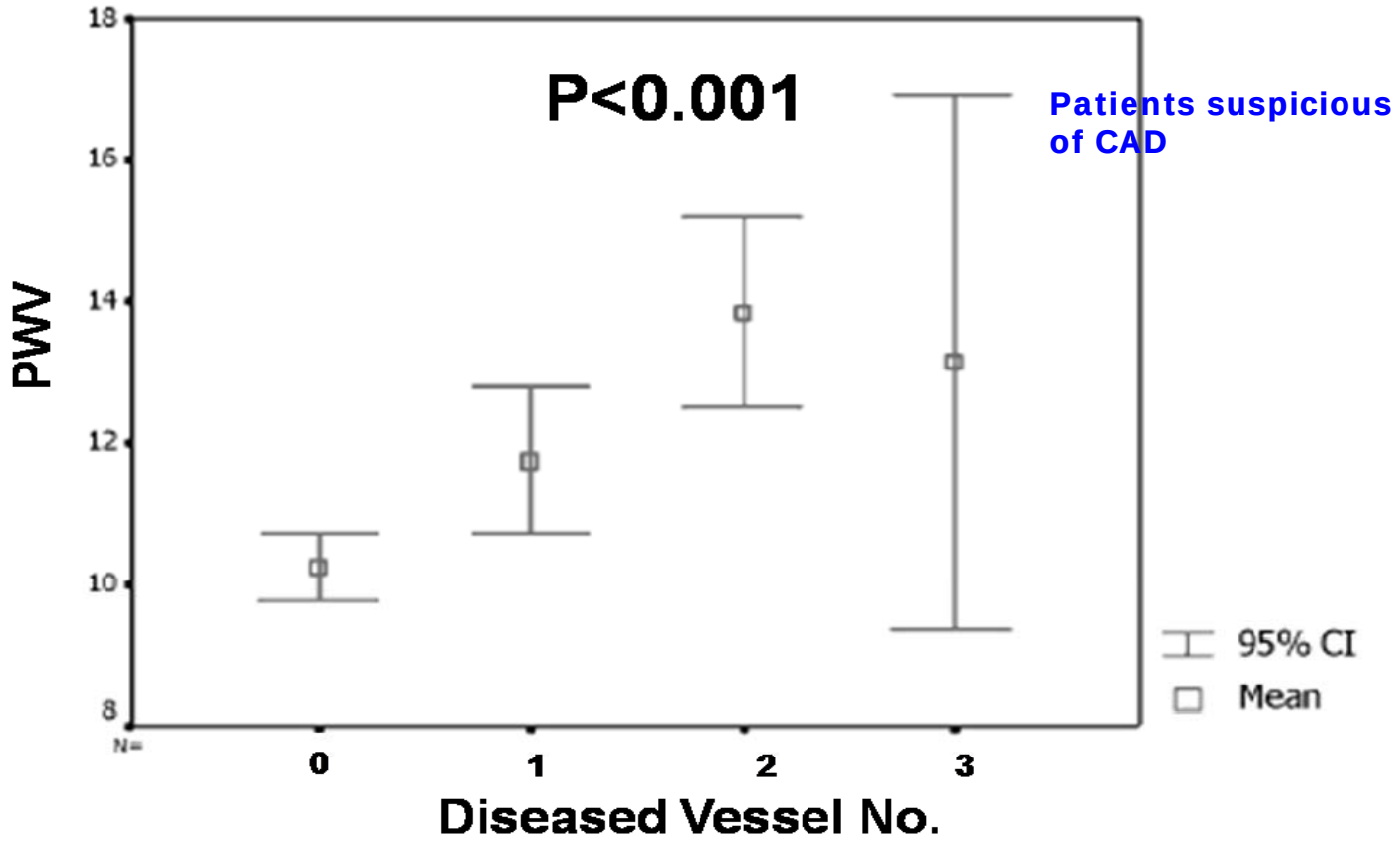
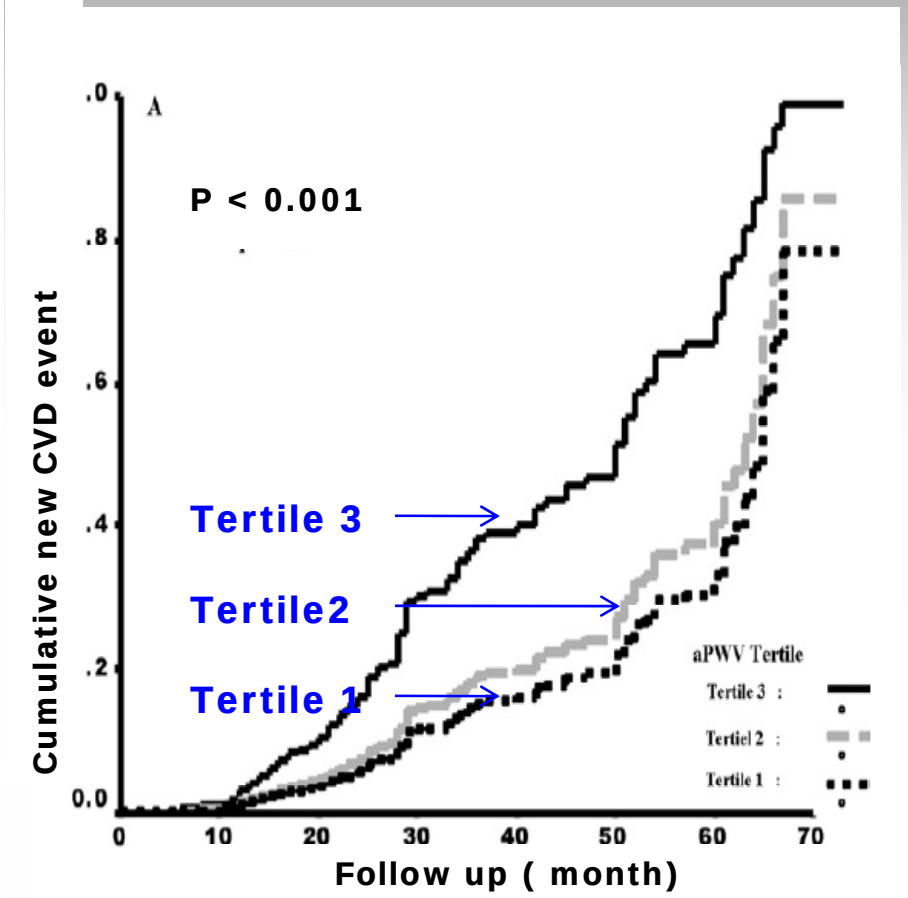
aPWV and Severity of CAD

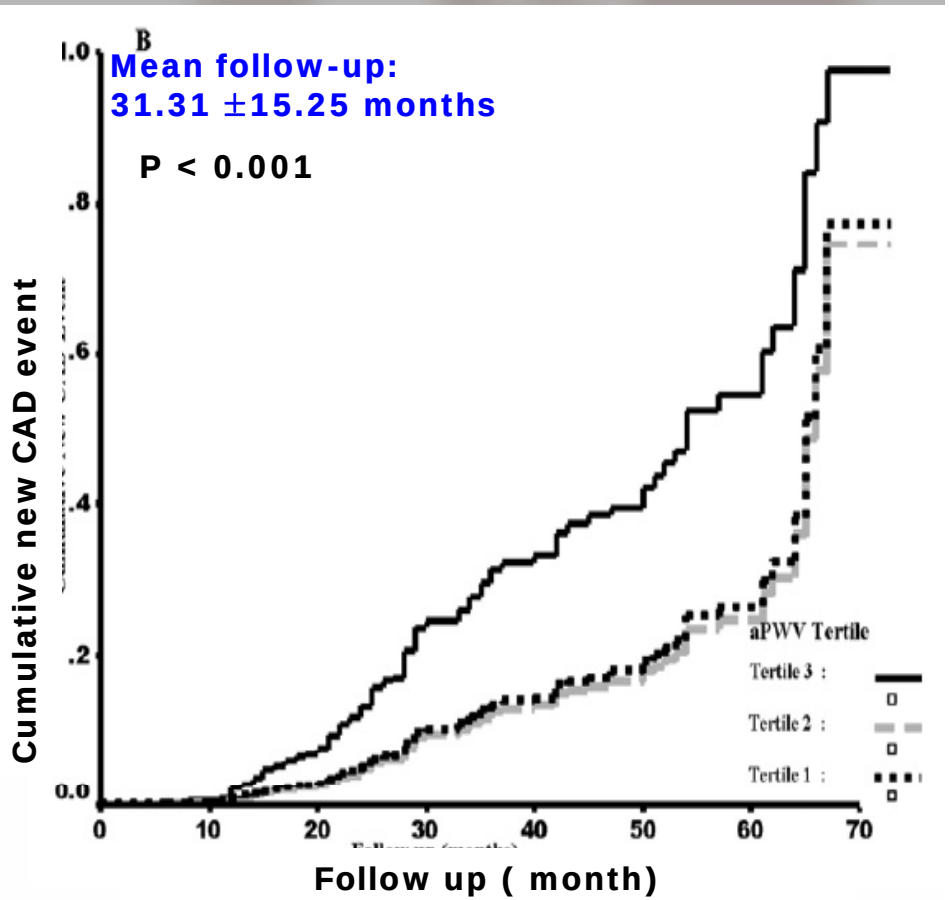


PWV is an independent risk marker for CAD and strongly associated with the severity of CAD.

Lim HE, Park CG et al Blood Pressure 2004;13:369-75

aPWV & Future CVD in Chest Pain Pts

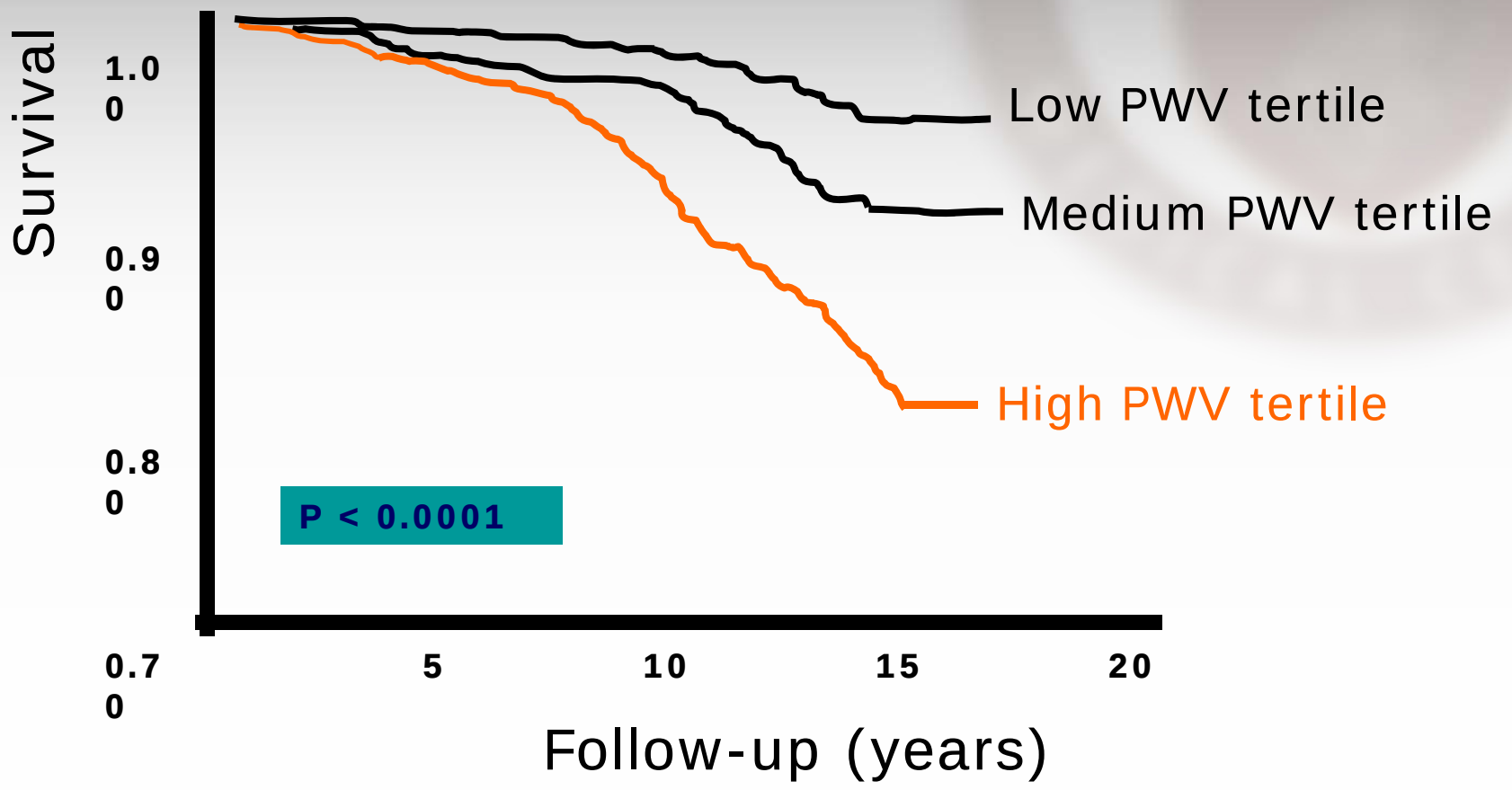




aPWV is an independent risk factor for future CV events and CAD

Choi CU, Park CG et al. Am J Hypertens. 2007;20:1163-9.

aPWV and All-cause Mortality in Hypertensive Subjects



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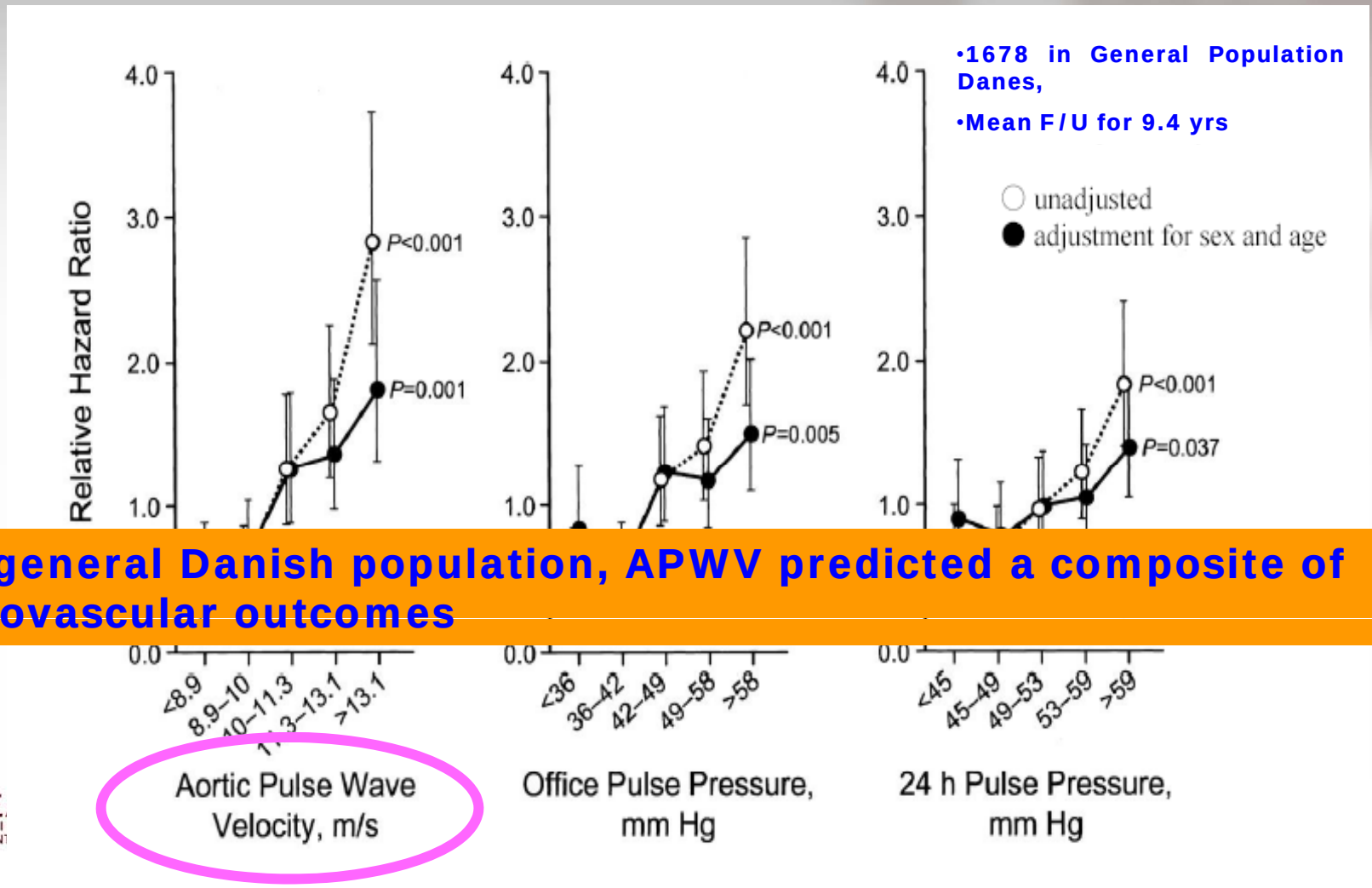
HYPERTENSION. 2001;37:1236-1241

aPWV and Composite Endpoint (CV mortality, CHD, Stroke)

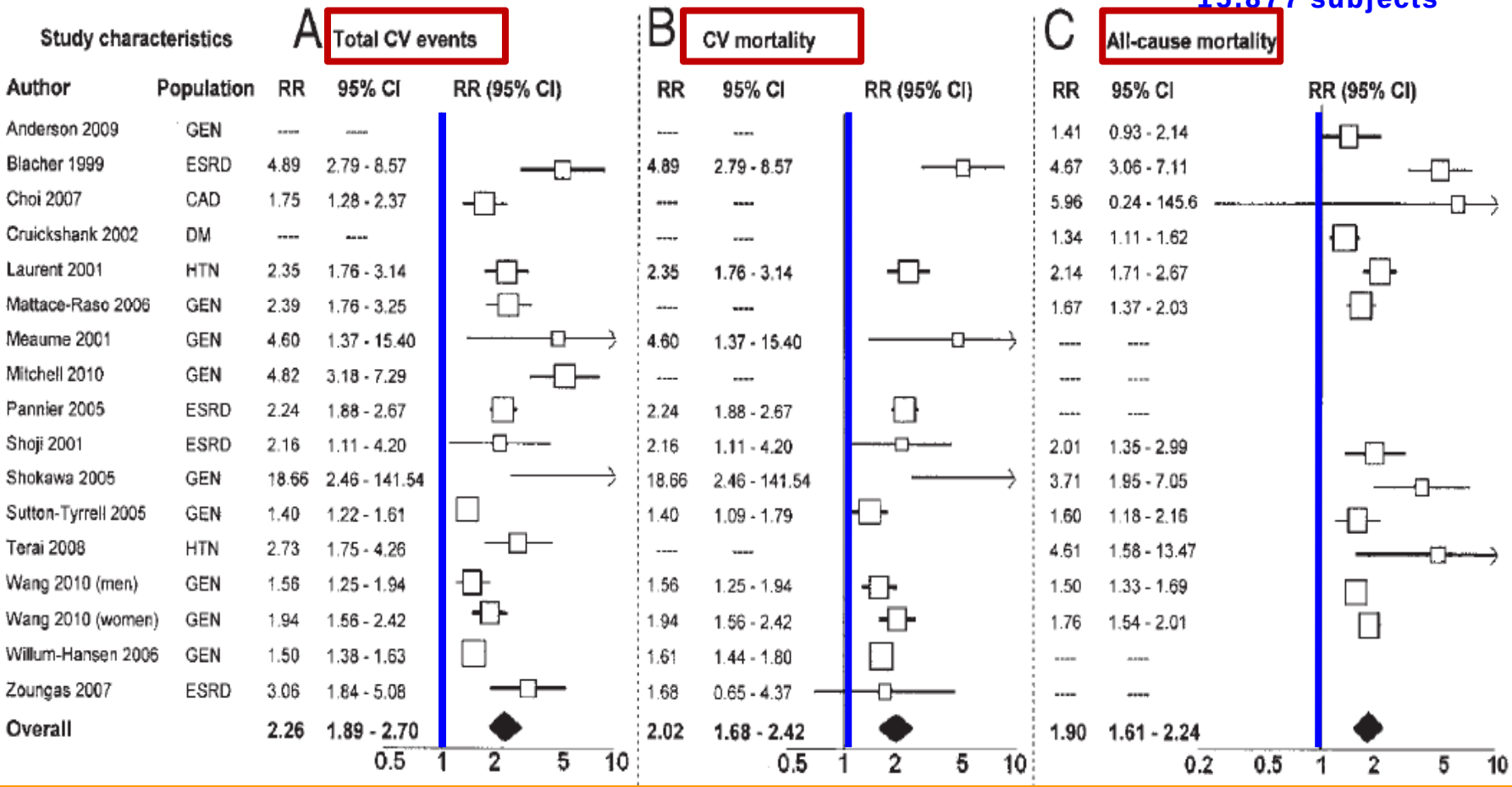


In a general Danish population, APWV predicted a composite of cardiovascular outcomes

CIRCULATION. 2006;113:664-670

aPWV and CVE,All-Cause Mortality Meta-analysis

15,877 subjects



Aortic PWV is a strong predictor of future CVE and all-cause mortality

Vlachopoulos C et al.J Am Coll Cardiol. 2010;55:1318-27

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Management of Central Blood Pressure

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Management of Central Blood Pressure

- Despite similar effects on brachial pressure, antihypertensive drugs have differential effects on central pressure.

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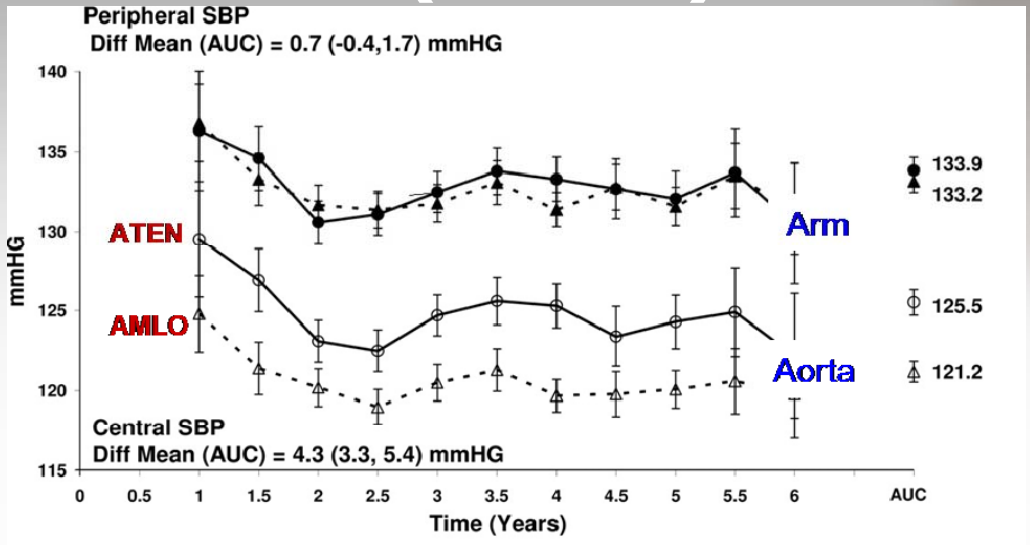
Hypertension. 2009;54:409-13.

O'Rourke M. Hypertension 1990; 15:339–347

The Conduit Artery Functional Evaluation (CAFE) ASCOT SUBSTUDY

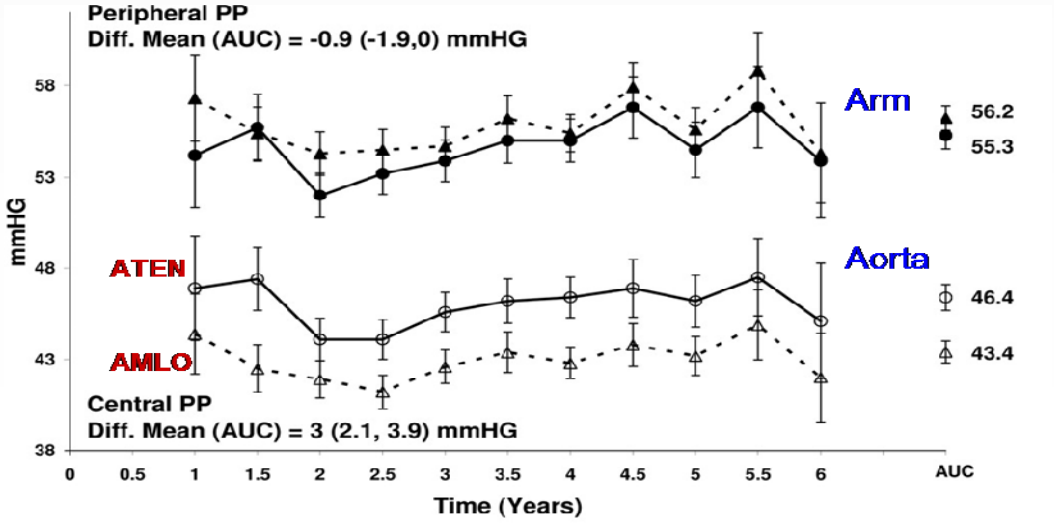
SBP



- ✓Amlodipine±Perindopril vs Atenolol±Thiazide
- ✓N=2073, 4 yr F/U

patients with HTN

PP



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CIRCULATION 2006;113:1213-25

Atenolol vs Eprosartan: Effects on Central BP

Table 2. Hemodynamic and biochemical parameters of study subjects

Hypertensive Subjects

Parameter	Baseline	Atenolol	Eprosartan	*P* value
Brachial systolic BP (mm Hg)	152 ± 2	135 ± 2*	136 ± 2*	.7
Brachial diastolic BP (mm Hg)	98 ± 2	90 ± 2*	92 ± 2*	.5
Brachial PP (mm Hg)	54 ± 1	45 ± 2*	44 ± 2*	.7
MAP (mm Hg)	117 ± 2	106 ± 1*	106 ± 2*	.9
Aortic systolic BP (mm Hg)	139 ± 3	128 ± 2*	123 ± 3*	.03
Aortic diastolic BP (mm Hg)	100 ± 2	91 ± 2*	92 ± 2*	.4
Aortic PP (mm Hg)	39 ± 1	37 ± 2	31 ± 2*	.005
PP amplification	1.38 ± 0.04	1.21 ± 0.04*	1.42 ± 0.04*	<.001
Heart rate (beats/min)	76 ± 3	57 ± 2*	76 ± 3	<.001
AIx (%)	22 ± 3	28 ± 2*	16 ± 3*	<.001
Aortic PWV (m/sec)	7.5 ± 0.3	6.8 ± 0.2*	7.1 ± 0.3*	.005

Inhibitors of the RAAS may cause a significantly greater fall of central pressures for a similar fall of brachial pressure

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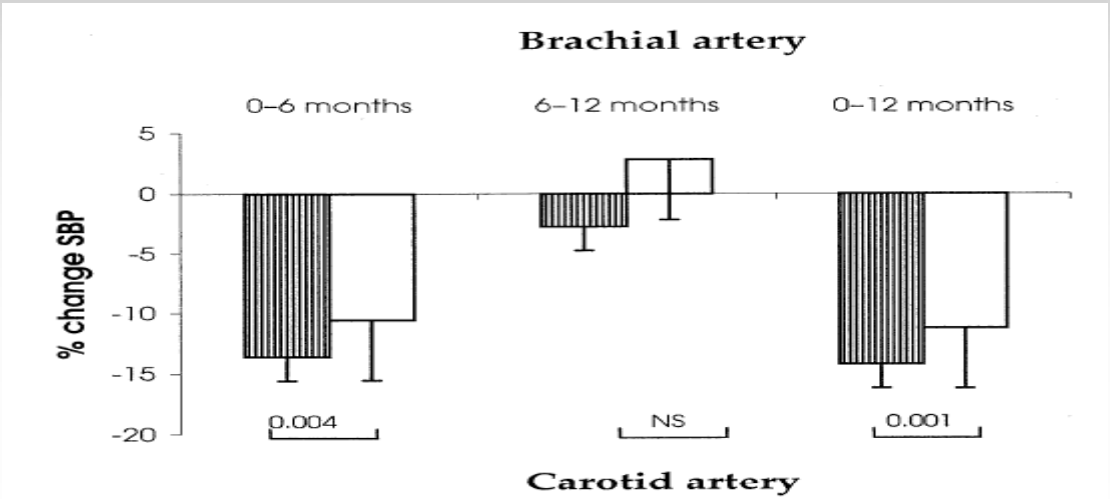
Dhakam Z et al. Am J Hypertens 2006;19:214-9.

Low-dose Combination of Perindopril/Indapamide in Hypertensive Subjects: Comparison with Atenolol



Perindopril/Indapamide

Atenolol

patients with HTN

Under Per/Ind, but not atenolol, normalization of brachial SBP is achieved with a significantly greater reduction of central SBP

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REASON trial, London GM et al. J Am Coll Cardiol 2004;43:92-9.

Comparison of the Effects of Antihypertensive Agents on Central BP

Table 2. Hemodynamic Indices Before and After the 10-Week Active Therapy Period 59 pts (>60yrs) with ISH

	Perindopril		Atenolol		Lercanidipine		Bendrofluazide	
Parameter	Placebo	10 wk	Placebo	10 wk	Placebo	10 wk	Placebo	10 wk
Peripheral SBP, mm Hg	153±3	136±4*	156±2	138±4*	146±2	133±3*	154±3	140±3*
Peripheral DBP, mm Hg	80±2	75±2*	84±2	76±3*	80±2	79±3	85±2	82±3
Peripheral PP, mm Hg	72±4	61±4*	72±3	62±3*	66±3	54±4*	69±4	58±4*
Central SBP, mm Hg	140±4	123±4*	144±3	130±4*	132±2	118±3*	139±2	126±2*
Central PP, mm Hg	58±4	46±3*	59±2	53±3	51±3	38±4*	53±4	42±3*

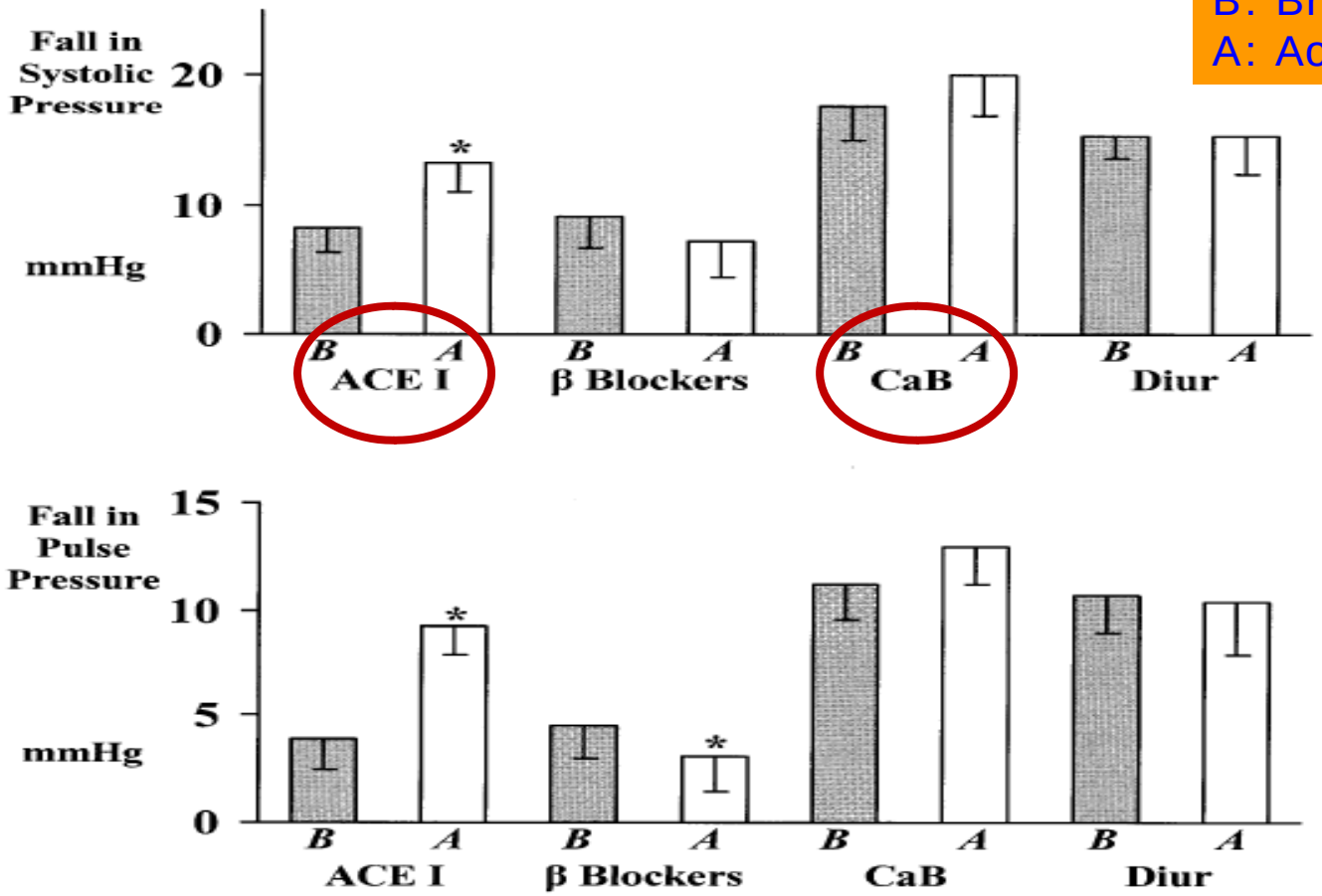
Central PP was only reduced significantly by perindopril, lercanidipine, and bendrofluazide, whereas atenolol had no effect

Hypertension. 2009;54:409-13.

Effect of Different Antihypertensive Drug Classes on Central Aortic Pressure

patients with HTN

B: Brachial artery
A: Aortic root



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Morgan T et al. Am J Hypertens. 2004;17:118-23.

Effect of Antihypertensive Drug on Central Hemodynamics

Drug class	Aortic PWV	Wave reflection	Carotid stiffness
Diuretics	↔	↔	↓/↔
β-Blockers			
Without vasodilating effect	↓	↑/↔	↔
With vasodilating effect	↓	↓	↓/↔
α-Blockers	↔	↓	NA
Calcium channel blockers			
Dihydropiridines	↓/↔	↓	↓/↔
Non-dihydropiridines	↓	↓	↓
ACE inhibitors	↓	↓	↓
Angiotensin II receptor blockers	↓	↓	↓/↔
Aldosterone antagonists	↓/↔	↓	NA

↑—increased; ↓—reduced; ↔—no change; ACE—angiotensin-converting enzyme; NA—not available; PWV—pulse wave velocity.

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Current Hypertension Reports 2009;11:190–196

Limitation of Central Pressure

◆Determination of normal values for wave reflections indices and central BPs is mandatory.

◆Extension of the existing data regarding the superiority of central BP over and above brachial BP in a wider range of populations and disease states is desirable

Summary & Conclusion I

◆ Central Pressures

- Differ significantly from peripheral pressures
- More closely related to the pathophysiology of CVD
- Central BP estimation is feasible by simple and reproducible methods
- Subjected to greater changes by drugs than peripheral BP.

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Summary & Conclusion II

- The assessment of central BP, together with aortic PWV, may give new additional data for the stratification of CV risk in the management of HTN and CVD.

- The prognostic value of central BP in large-scale prospective clinical trials remains a great challenge.

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Thank you for your attention!

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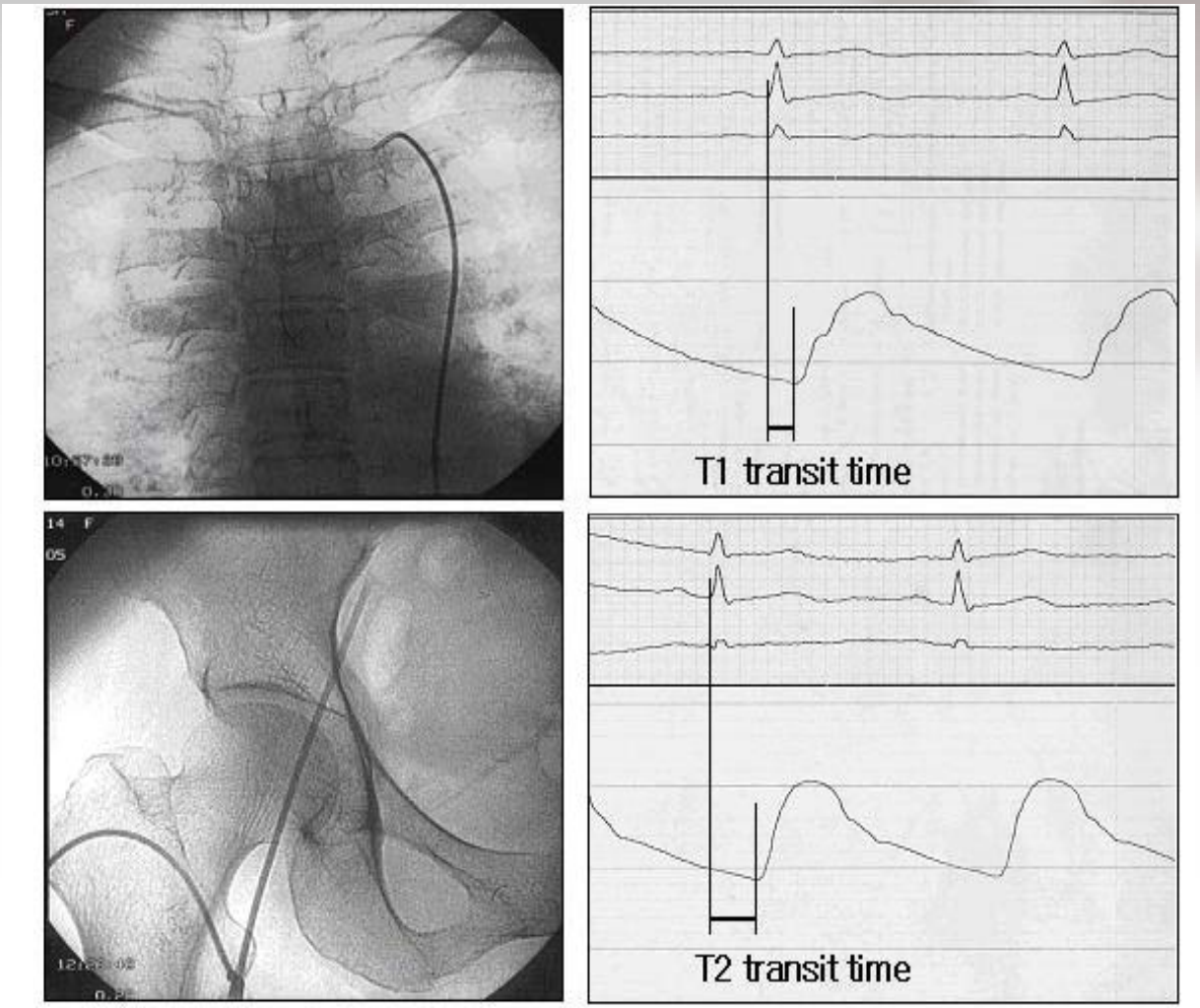
- Back up

PULSE WAVE VELOCITY: Invasive method



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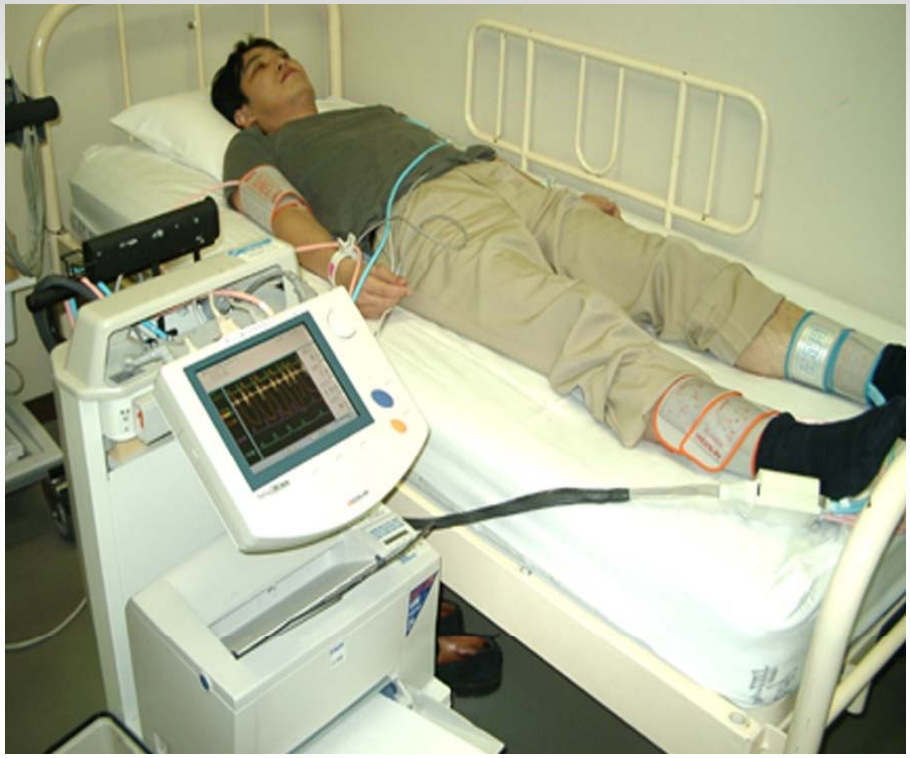
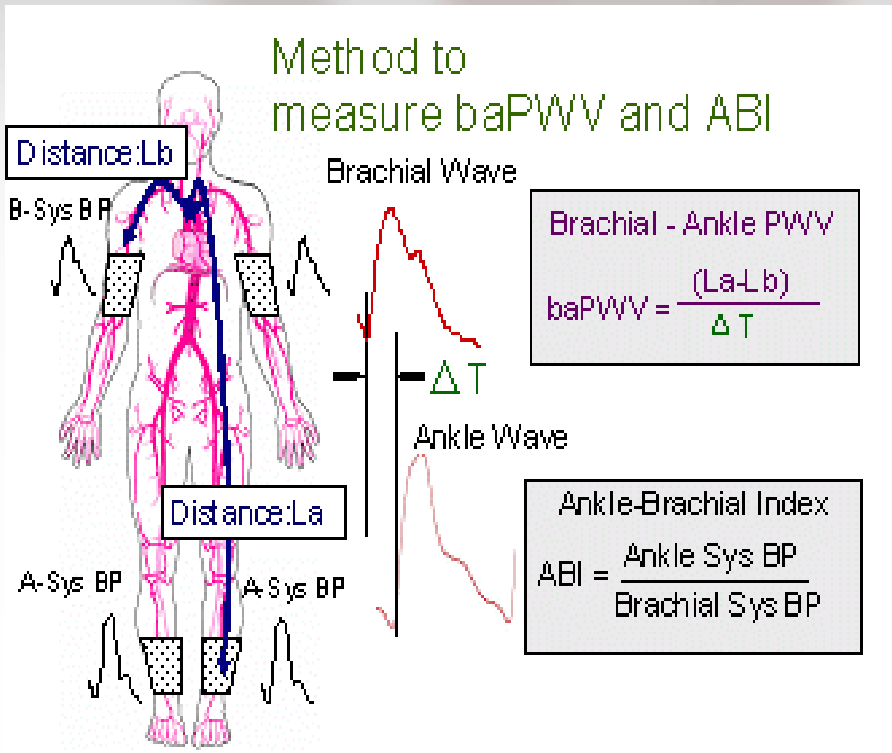
PULSE WAVE VELOCITY: Non-invasive method





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Association between Age and Each Blood Pressure Using Multivariate Analysis

	Total (n=175)		
Age-Associations	β	p	R^2
Aortic arch SBP	0.58	<0.001	0.268
Aortic arch DBP	-0.22	0.004	0.145
Aortic arch PP	0.80	<0.001	0.403
Abd aorta SBP	0.53	0.001	0.197
Abd aorta DBP	-0.30	0.001	0.128
Abd. aorta PP	0.83	<0.001	0.374
Radial artery SBP	0.53	0.002	0.153
Radial artery DBP	-0.21	0.005	0.106
Radial artery PP	0.74	<0.001	0.216

***.Multivariate regression analysis was done after co-adjusting for sex, CAD, diabetes mellitus, dyslipidemia, the use of antihypertensives, heart rate and smoking.**

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Choi CU, Kim EJ et al. *J Hypertens* 2010 (In press).

Association between Age and Each Blood Pressure Using Multivariate Analysis

	CAD (+, n=71)			CAD(-, n=104)		
Age-Associations	β	p	R^2	β	p	R^2
Aortic arch SBP	0.54	0.042	0.260	0.55	0.003	0.299
Aortic arch DBP	-0.36	0.019	0.211	-0.17	0.062	0.161
Aortic arch PP	0.89	<0.001	0.402	0.72	<0.001	0.425
Abd aorta SBP	0.64	0.021	0.243	0.44	0.016	0.245
Abd aorta DBP	-0.43	0.007	0.218	-0.26	0.015	0.122
Abd aorta PP	0.56	<0.001	0.374	0.70	<0.001	0.448
Radial artery SBP	0.63	0.042	0.256	0.43	0.033	0.103
Radial artery DBP	-0.42	0.004	0.184	-0.13	0.141	0.115
Radial artery PP	0.45	<0.001	0.353	0.57	0.001	0.157

***.Multivariate regression analysis was done after co-adjusting for sex, diabetes mellitus, dyslipidemia, the use of antihypertensives, heart rate and smoking.**

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Choi CU, Kim EJ et al. *J Hypertens* 2010 (In press).

Association between Age and Each Blood Pressure Using Multivariate Analysis

	HTN Tx (+, n=61)			HTN Tx (-, n=71)		
Age-Associations	β	p	R^2	β	p	R^2
Aortic arch SBP	0.44	0.021	0.241	0.71	0.006	0.387
Aortic arch DBP	-0.35	<0.001	0.196	-0.04	0.753	0.119
Aortic arch PP	0.79	<0.001	0.395	0.75	<0.001	0.472
Abd aorta SBP	0.44	0.026	0.197	0.58	0.019	0.246
Abd aorta DBP	-0.45	<0.001	0.197	-0.13	0.362	0.060
Abd. aorta PP	0.50	<0.001	0.400	0.71	<0.001	0.402
Radial artery SBP	0.42	0.041	0.154	0.54	0.056	0.220
Radial artery DBP	-0.31	0.001	0.165	-0.01	0.676	0.117
Radial artery PP	0.37	<0.001	0.221	0.59	0.016	0.302

***.Multivariate regression analysis was done after co-adjusting for sex, CAD, diabetes mellitus, dyslipidemia, heart rate and smoking.**

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Choi CU, Kim EJ et al. *J Hypertens* 2010 (In press).

the question follows as to whether central aortic pressures should be measured more routinely in clinical practice

- first, can central aortic pressure be measured in routine clinical practice?
- Second, the only point in measuring central aortic pressures would be if the measurement provided more accurate information than brachial BP about the patients' risk and a better assessment of their response to treatment.

Amplification차이

- Mean BP and diastolic BP change little from the aortic root to brachial artery and represent the tonic component of BP.
- In contrast, systolic BP is the more dynamic, pulsatile pressure component and is amplified with increasing distance from the aortic root. This systolic and pulse pressure amplification process is principally a function of the timing of pressure wave reflections in the circulation.
- These reflections are more likely to augment systolic and pulse pressure as the pressure wave moves from the aortic root and closer to reflecting sites at the periphery [1]. This amplification phenomenon is more pronounced in younger people with healthy conduit arteries who thus have greater brachial systolic and pulse pressures relative to their corresponding central aortic pressures.
- Aortic:brachial pulse pressure amplification can be quite marked in young healthy people, and atypical brachial:aortic ratio is 1.5.
- The amplification process diminishes with ageing, principally due to aortic stiffening and an increased pulse wave velocity. Thus, with ageing or aortic stiffening or both, central aortic pressures are closer to the brachial pressures but are rarely the same

Aortic PP was a Significant Risk Factor for the Extent of CAD

Table 4. Multiple regression analysis of the number of diseased coronary vessels

Factor	Regression Coefficient	R^2	*P*
Gender (0 male, 1 female)	−0.55	0.022	.013
Aortic pulse pressure (mm Hg)	0.0125	0.10	.023
Dyslipidemia (1 yes/0 no)	0.286	0.059	—
Age (y)	−0.011	0.028	—
Diabetes mellitus (1 yes/0 no)	0.249	0.024	—
Aortic mean BP (mm Hg)	0.005	0.005	—
Current smoker (1 yes/0 no)	0.09	0.0006	—

Dependent variable: number of diseased vessels ($r^2 = 0.24$; $P < .001$). Abbreviation as in Table 3.

Philippe F et al. Am J Hypertens. 2002;15:672-7.

Asc. Aorta SBP are Related to Extent of CAD

Table 3. Odds ratios for the association between each hemodynamic parameter and the risk of three-vessel coronary artery disease (CAD) (1 = three-vessel CAD, 0 = one- or two-vessel CAD)

Parameter	Odds Ratio (95% CI)	
	Univariate	Multivariate*
Sphygmomanometer brachial BP		
Systolic BP per 10 mm Hg	1.15 (1.03–1.28)	
Diastolic BP per 10 mm Hg	1.11 (0.92–1.36)	
Mean BP per 10 mm Hg	1.18 (1.00–1.40)	
Pulse pressure per 10 mm Hg	1.20 (1.04–1.38)	
FSP per 0.1	1.27 (0.99–1.62)	
FDP per 0.1	0.62 (0.38–1.02)	
Invasively measured ascending aortic BP		
Systolic BP per 10 mm Hg	1.16 (1.06–1.27)	
Diastolic BP per 10 mm Hg	0.93 (0.77–1.12)	
Mean BP per 10 mm Hg	1.12 (0.96–1.30)	
Pulse pressure per 10 mm Hg	1.28 (1.15–1.44)	1.15 (1.01–1.30)
FSP per 0.1	1.58 (1.30–1.92)	1.28 (1.03–1.60)
FDP per 0.1	0.40 (0.27–0.59)	0.61 (0.39–0.95)

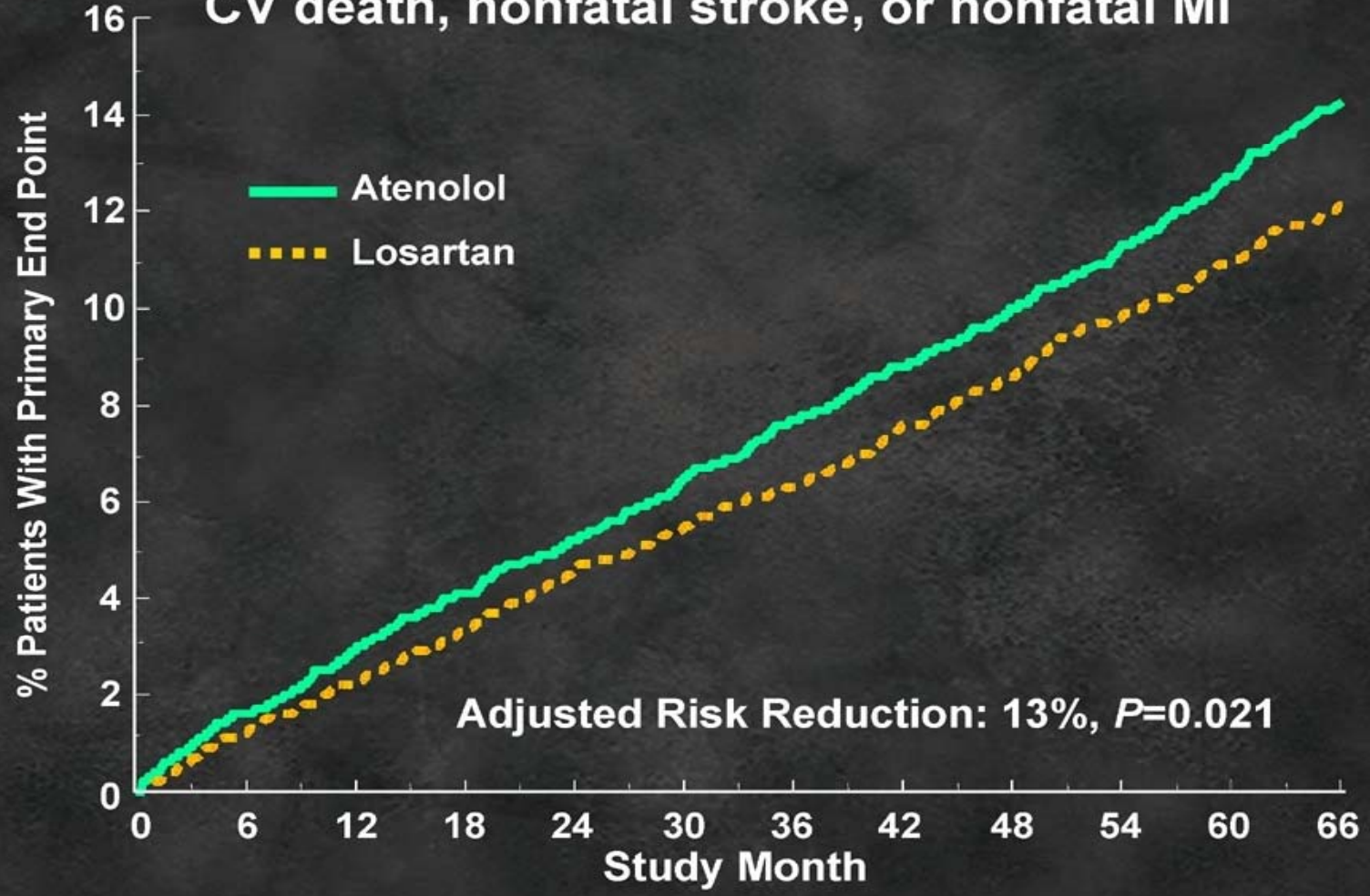
Jankowski P et al. Am J Hypertens. 2004;17:641-6.

Implications for Cuff (Brachial) BP

- Inaccurate indicator of hypertension ?
- Poor indicator of central systolic BP, cardiac load, and LVH ?
- Poor indicator of renal and cerebral microcirculatory pulsatile loads and organ damage ?
- Poor indicator of beneficial effects of antihypertensive drugs ?

LIFE: Primary End Point

Kaplan-Meier estimates of the primary end point of time to CV death, nonfatal stroke, or nonfatal MI



ANBP2 study 결과

All Subjects

End Point	Hazard Ratio (95% CI)	P Value
All cardiovascular events or death from any cause	0.89 (0.79–1.00)	0.05
First cardiovascular event or death from any cause	0.89 (0.79–1.01)	0.06
Death from any cause	0.90 (0.75–1.09)	0.27

Male Subjects

End Point	Hazard Ratio (95% CI)	P Value
All cardiovascular events or death from any cause	0.83 (0.71–0.97)	0.02
First cardiovascular event or death from any cause	0.83 (0.71–0.97)	0.02
Death from any cause	0.83 (0.66–1.06)	0.14

Female Subjects

End Point	Hazard Ratio (95% CI)	P Value
All cardiovascular events or death from any cause	1.00 (0.83–1.21)	0.98
First cardiovascular event or death from any cause	1.00 (0.83–1.20)	0.98
Death from any cause	1.01 (0.76–1.35)	0.94

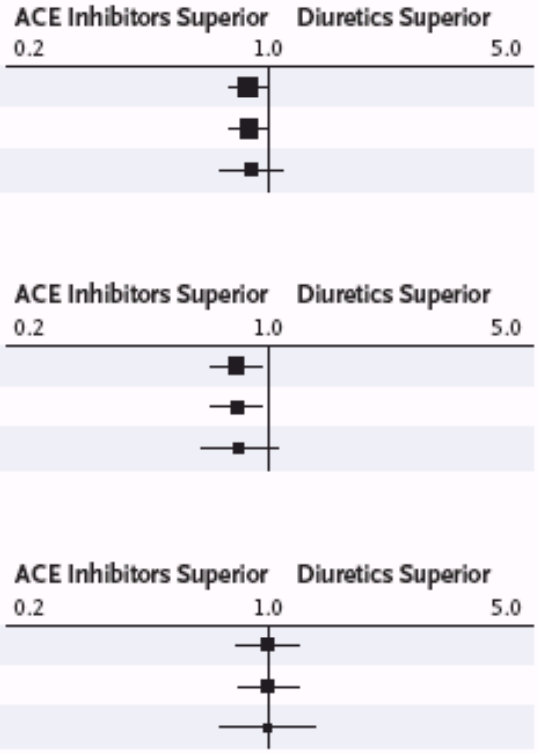
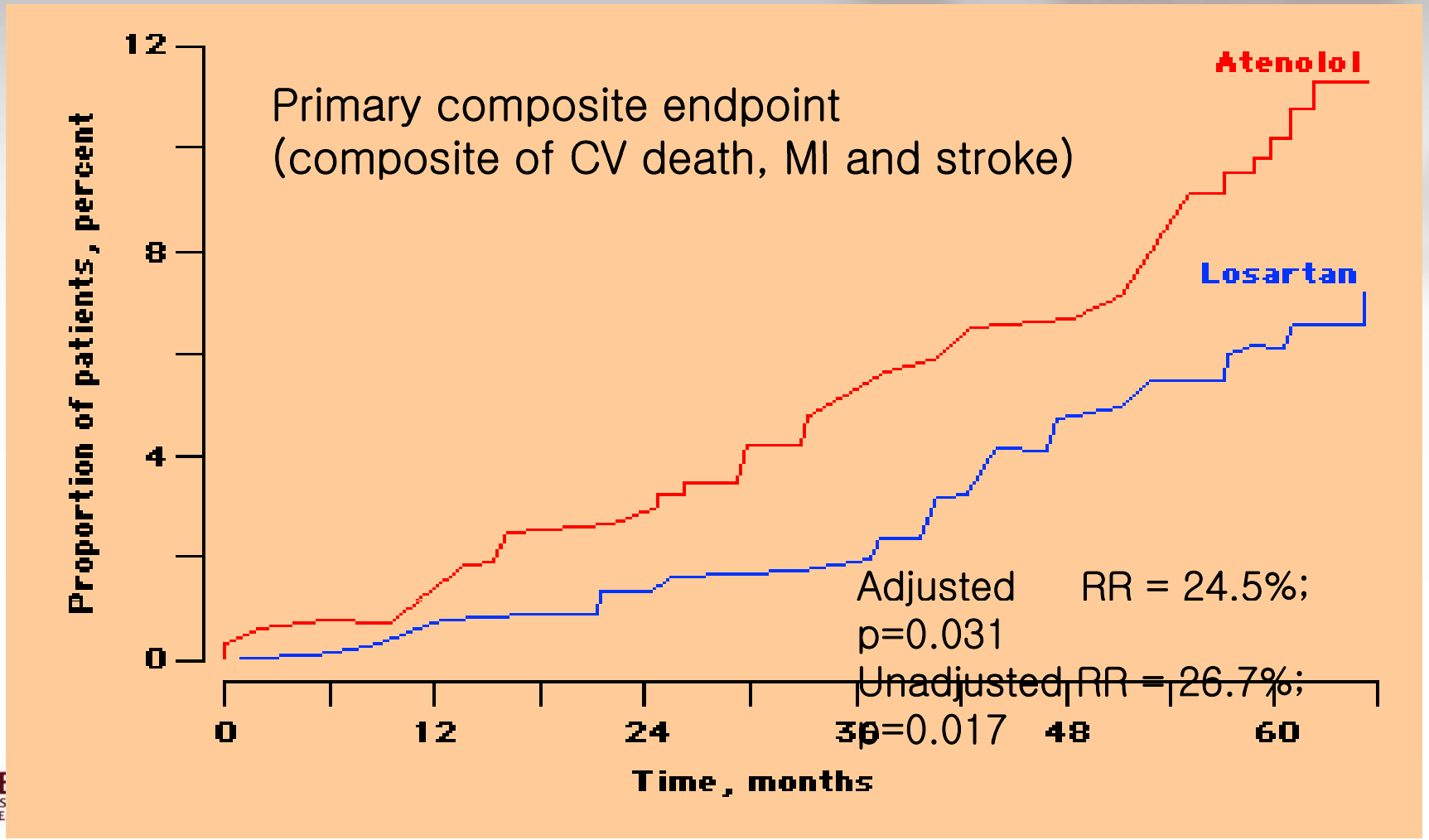


Figure 3. Primary End Points among All Subjects, Male Subjects, and Female Subjects.
ACE denotes angiotensin-converting enzyme, and CI confidence interval.

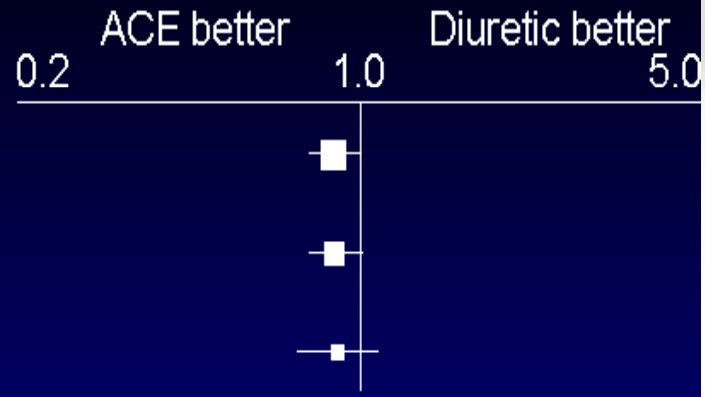
LIFE - Cardiovascular Benefits of Losartan Confirmed in Diabetic Subgroup



Lindholm LH et al *Lancet* 2002;359:1004-1010

ANBP 2 Primary Result

	Hazard Ratio (95% CI)	p
Overall Treatment Effect	0.89 (0.79,1.00)	0.05
First Cardiovascular or Death	0.89 (0.79,1.01)	0.06
Death	0.90 (0.75,1.09)	0.27

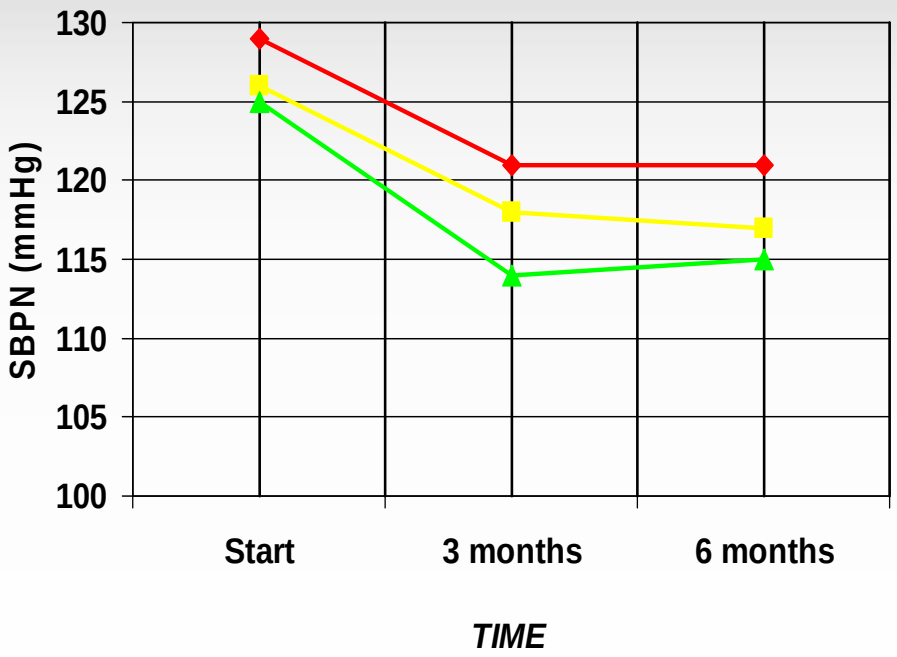


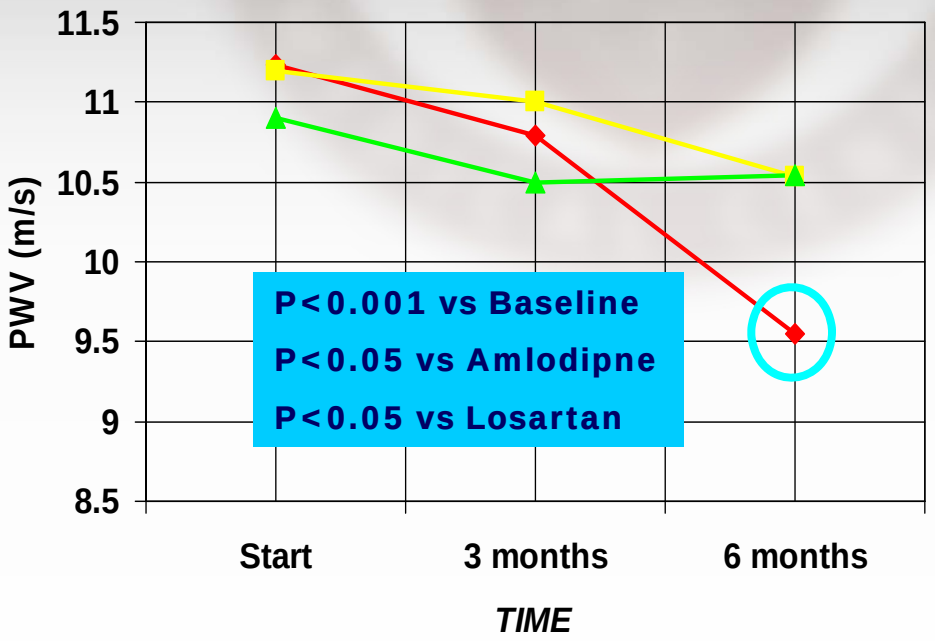
Rate per 1000 patient years

Adjusted for age, gender

aPWV and antihypertensive therapy
ACEI vs CCB vs ARB

Different effects on PWV for the same Systolic BP reduction





G1 = ACEI (Quinapril 20mg)

G2 = CCB (Amlodipine 10mg)

G3 = ARB (Losartan 100mg)

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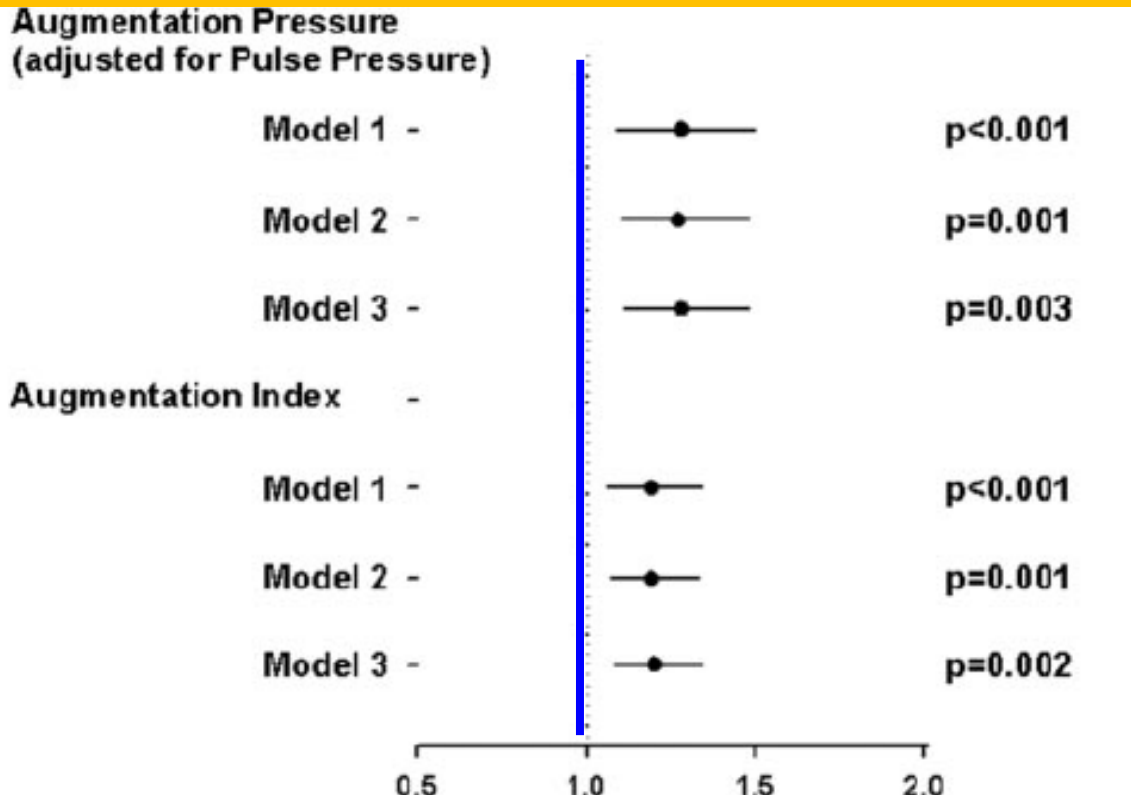
Rajzer & al Am J Hypertens 2003;16:439-44

Aortic Pressure Augmentation and Adverse Cardiovascular Events

Aortic AP independently predicts adverse outcomes



Patients With CAD

HR for Adverse Cardiovascular Events

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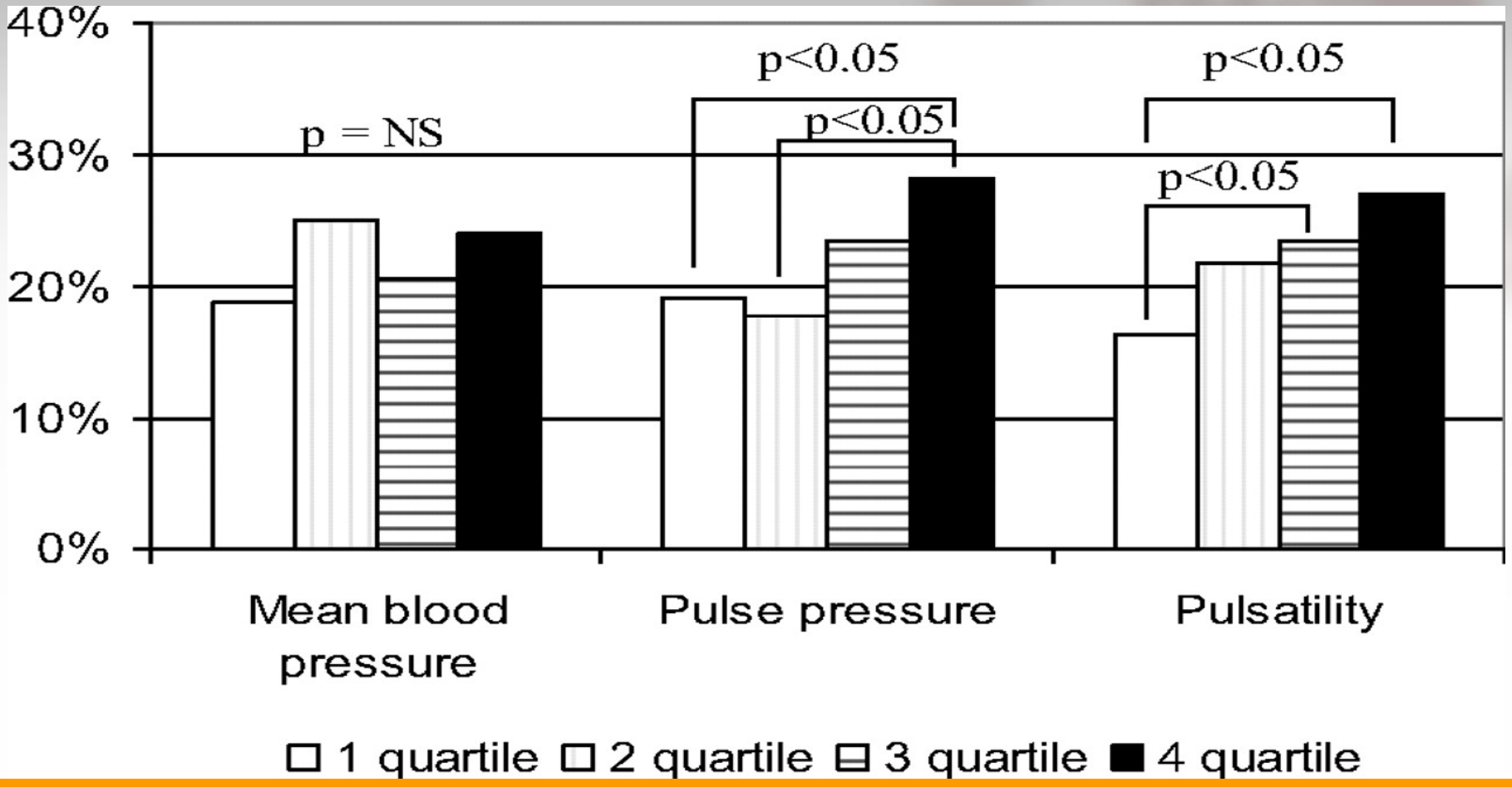
Chirinos JA et al. Hypertension. 2005;45:980-5.

Central Pressure and Cardiovascular Events in CAD Pts



Central PP was independently related to the CVE

Jankowski P et al. Hypertension. 2008;51:848-55.

Table 1 Central aortic pressures and major clinical outcomes in recent clinical studies

Patient group	Follow-up (months)	Outcome	Number of events	Dominant BP variable in multivariate analysis	Hazard ratio per 10 mmHg[a] or SD (95% CI)
End-stage renal disease	52	All cause mortality	70	Central pulse pressure (C)	1.4 (1.1–1.8)
Male coronary heart disease patients	39	All cause mortality	64	Central pulse pressure (D)	1.18[a] (1.05–1.33)
Treated hypertensive patients	36	Composite of CV and renal events	305	Central pulse pressure (R)	1.11[a] (1.0–1.21)
Unselected cohort of American Indians	58	Fatal and nonfatal CV events	319	Central pulse pressure (R)	1.15[a] (1.07–1.24)
Unselected elderly population	96	CV mortality	45	Central systolic pressure (C)	1.33[a] (1.03–1.72)
Coronary heart disease patients	54	Fatal and nonfatal CV events	246	Central pulse pressure (D)	1.25 (1.09–1.43)
Unselected Chinese community	120	CV mortality	130	Central systolic pressure (C)	1.30[a] (1.12–1.52)

Estimation of central systolic blood pressure using an oscillometric blood pressure monitor

Hao-Min Cheng[1,2], Kang-Ling Wang[2,3], Ying-Hwa Chen[2,3], Shing-Jong Lin[1,4], Lung-Ching Chen[2,3], Shih-Hsien Sung[2,3], Philip Yu-An Ding[2,3], Wen-Chung Yu[2,3], Jaw-Wen Chen[1,4] and Chen-Huan Chen[1,2,4]

Current noninvasive techniques for assessing central aortic pressure require the recording of an arterial pressure wave using a high-fidelity applanation tonometer. We therefore developed and validated a novel method to estimate the central aortic systolic pressure using an oscillometric blood pressure monitor alone. Invasive high-fidelity right brachial and central aortic pressure waves, and left-brachial pulse volume plethysmography from an oscillometric blood pressure monitor, were obtained at baseline and 3 min after administration of sublingual nitroglycerin in 100 patients during cardiac catheterization. In the initial 50 patients (Generation Group), Central systolic blood pressure was predicted by a multi-variate prediction model generated from the comprehensive analysis of the invasive brachial pressure wave, including brachial late-systolic shoulder pressure value and parameters related to wave reflection and arterial compliance. Another prediction model was similarly constructed from the noninvasively calibrated pulse volume plethysmography. Both models were validated in the subsequent 50 patients (Validation Group) with results: r=0.98 (P<0.001) and mean difference=0.5 ± 4.5 (95% confidence interval −8.3 to 9.3) mm Hg for the invasive model, and r=0.93 (P<0.001) and mean difference=−0.1 ± 7.6 (95% confidence interval −15.0 to 14.8) mm Hg for the noninvasive model. Thus, our results indicate that central aortic systolic blood pressure could be estimated by analysis of the noninvasive brachial pressure wave alone from an oscillometric blood pressure monitor.

Hypertension Research advance online publication, 26 March 2010; doi:10.1038/hr.2010.37

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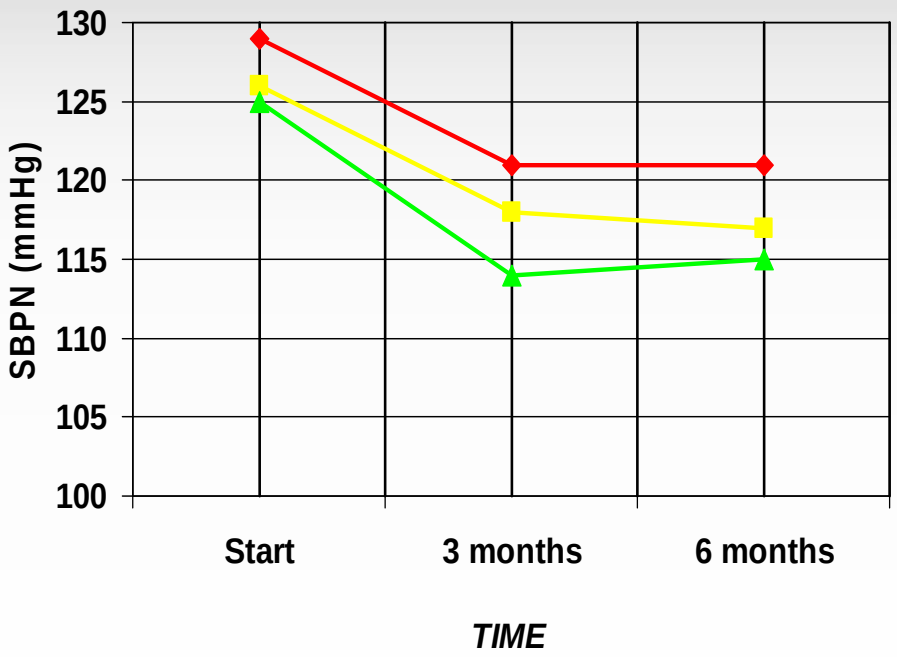
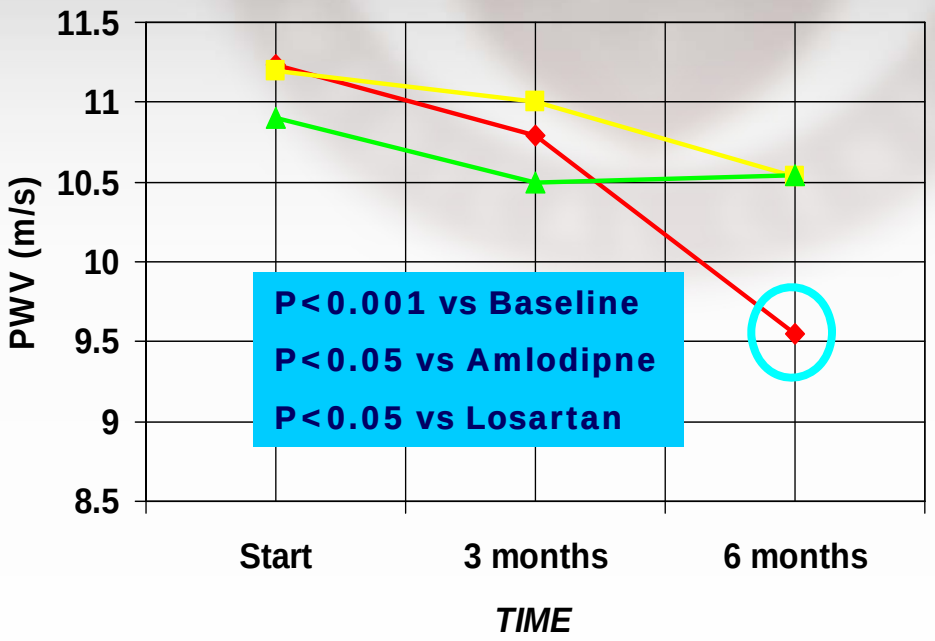
aPWV and Antihypertensive Therapy
ACEI vs CCB vs ARB

Different effects on PWV for the same Systolic BP reduction





G1 = ACEI (Quinapril 20mg)

G2 = CCB (Amlodipine 10mg)

G3 = ARB (Losartan 100mg)

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Rajzer & al Am J Hypertens 2003;16:439-44

The Anti-Hypertensive Role of Statins

Table 1 Data from various trials on the effects of statins on blood pressure

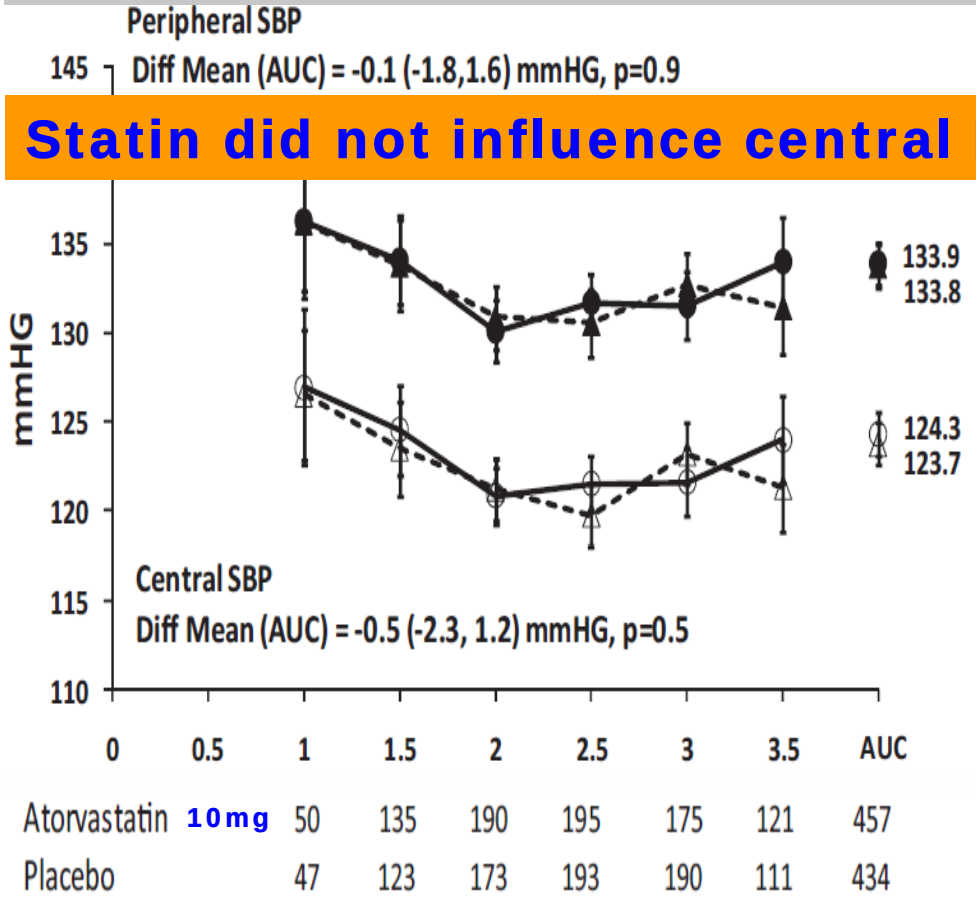
Principal investigators	Type of study/ study design	Data extrapolated from trial(s)						
		Patient characteristic	BP value	Placebo		Statin		'P' value
				Baseline	Treated	Baseline	Treated	
Glorioso et al 1999	Double blinded, randomized, cross over trial	N=25 HTN + ↑Chol	Systolic Diastolic	149±6 97±2	149±6 97±2	149±6 96±2	141±5 91±4	0.001 0.001
Sposito et al 1999	Randomized clinical trial without blinding	N=70 HTN + ↑ Chol	Systolic Diastolic	149±8 102±2	137±6 87±8	153±9 100±3	130±5 81±4	<0.01 <0.01
Borghi et al 2000	Double blinded, randomized placebo controlled trial	N=135 HTN + ↑ Chol + Normal Chol.	Systolic Diastolic	157.7±24 95.7±8	147.2±7 89.8±4	160.7±21 94.1±9	142.5±17 84.1±5	<0.001 <0.05
Prasad et al 2003	Retrospective cohort study of renal	N=113 HTN + ↑ Chol Renal Transpl.	Systolic Diastolic	133.9±14 83.9±8	133.1±17 82.5±10	135±16 82.5±10	128.3±15 79.4±9	0.005 0.05

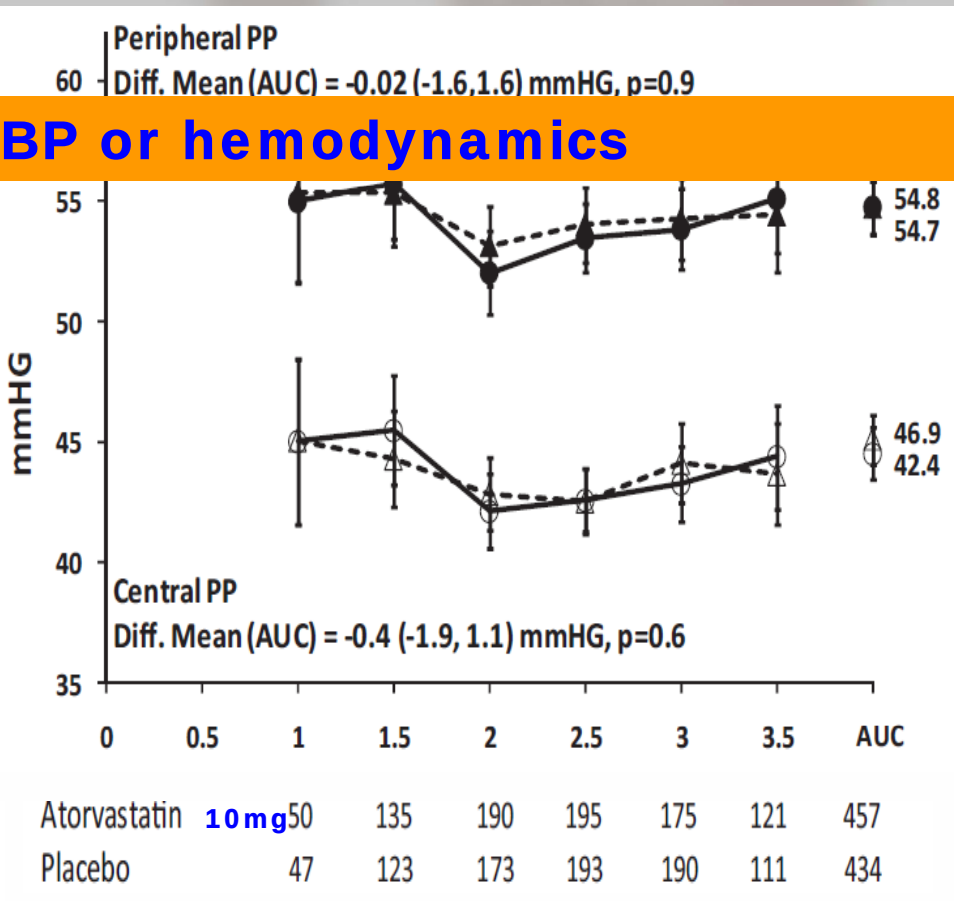
Statins may provide substantial reduction of blood pressure in the Hypertensive, independent of their lipid-lowering effect.

Cardiovasc Drugs Ther 2007;21:161–169

Impact of Statin Therapy on Central Aortic Pressures (CAFE-LLA)

Statin did not influence central BP or hemodynamics





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Circulation 2009;119;53-61