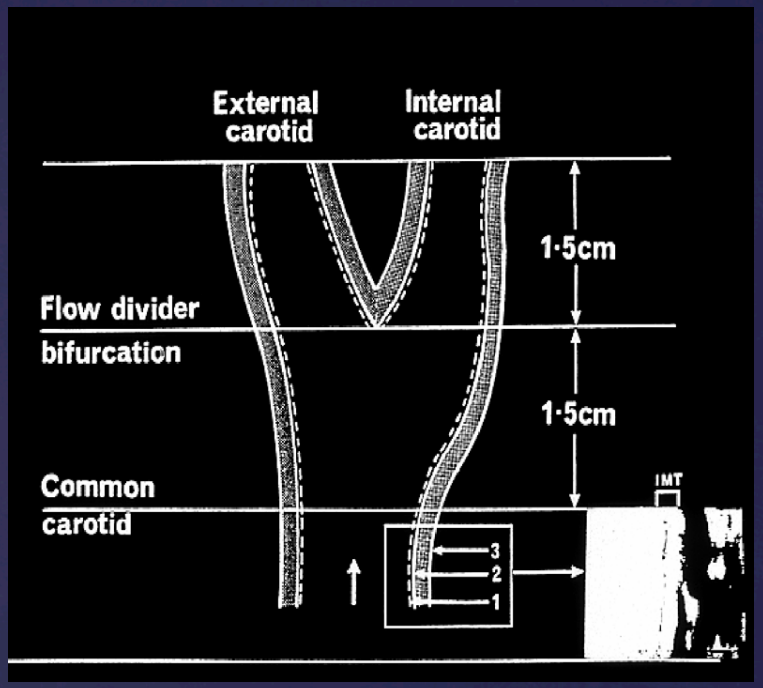
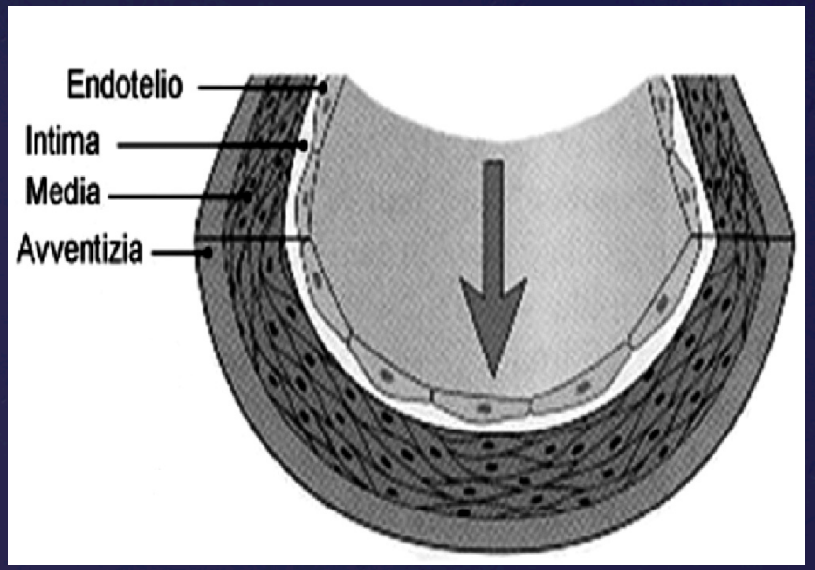
Imaging carotideo: dall'IMT alla pulse wave velocity

(Asymptomatic organ damage)

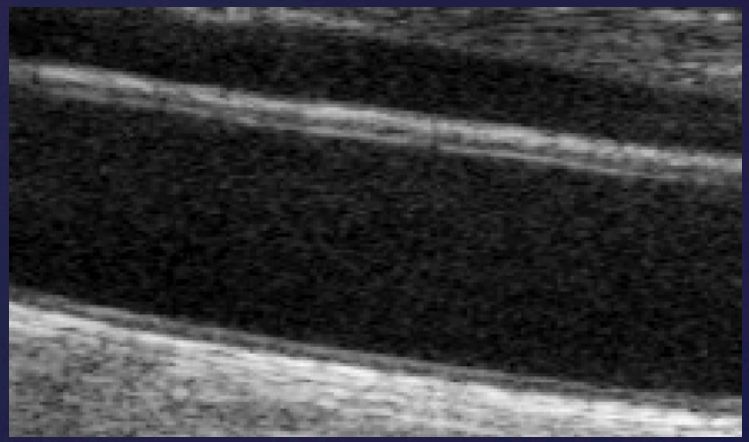
Olga Vriz

San Daniele del Friuli





carotid artery demonstrated a 'double-line' pattern: the first echogenic interface on the far wall corresponds to the lumen–intima interface and the second arises from the media–adventitia interface.



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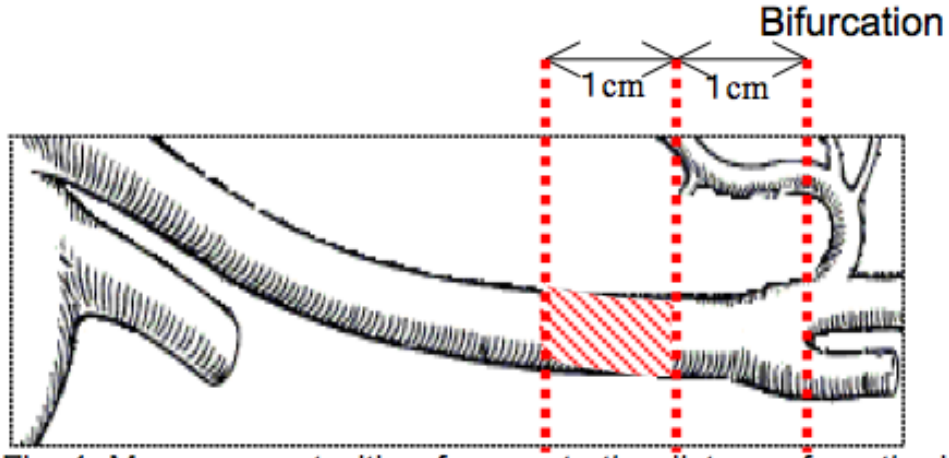
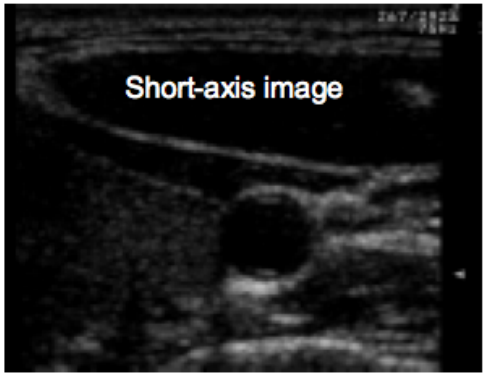
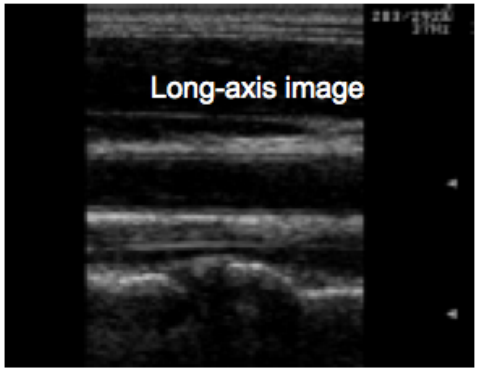


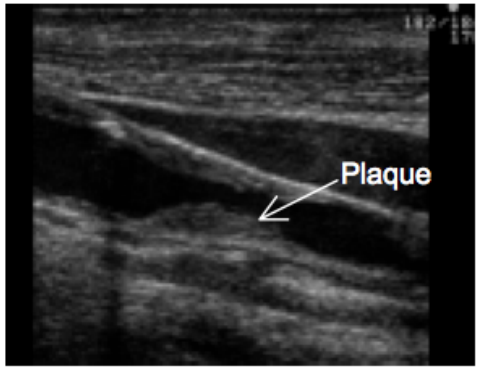
Fig. 1: Measurement with reference to the distance from the branch



Short-axis images have the advantage that the maximum diameter of a blood vessel can be easier to recognize.



Long-axis images have a smaller variation when the effect of vein pulsation is strong.



Avoid the part where plaques and others are formed.

Table 5 Recommend scanning protocol for evaluation of common carotid artery carotid intima-media thickness and detection of carotid plaques

Step	View	Area of interest	Technique	Use
1	Transverse B-mode scan (3-5 beat cine-loop in each segment)	From proximal CCA through middle of the internal carotid artery	Notch of transducer oriented to right of patient Slowly advance probe, keep vessel in center of screen, show double lines on near and far walls	Overview of vessel orientation, wall thickness, plaques, and surrounding structures
2	Internal and external carotid artery Doppler recordings (one frame of each)	Pulsed wave Doppler of proximal 1 cm of each branch	Sample volume parallel to flow by beam steering and angle correction of ≤60 degrees If narrowing is seen, obtain pre- and post-velocities to document severity	Verifies anatomic orientation and may identify significant stenosis if present
3	Longitudinal plaque screen scan (3-5 beat cine-loop from at least 3 different angles in each segment)	Near and far walls of CCA, bulb, and internal carotid artery segments	Rotate 90 degrees from transverse plane with notch of transducer oriented toward head of patient Circumferential plaque screen scan from anterior, lateral, and posterior imaging planes	Identification and description of plaques

4 Select best images of distal 1 cm of CCA far wall from each of 3 angles; review loops, then measure from R-wave gated still frames

Complementary angles better represent overall wall thickness

5 Measure images in triplicate by tracing far wall blood-intima and media-adventitia interfaces using leading edge–to–leading edge method (Figure 5)
-Measure 1-cm length
-Assure that measurements from each angle are within 0.05 mm of others
-Plaques should be traced as part of CIMT

Triplicate measurements insure consistency, averaging increases precision
If more variation is identified, critically review images; only trace images with clear boundaries that are over-gained and are imaged perpendicular to artery

ASE CONSENSUS STATEMENT

Use of Carotid Ultrasound to Identify Subclinical Vascular Disease and Evaluate Cardiovascular Disease Risk: A Consensus Statement from the American Society of Echocardiography Carotid Intima-Media Thickness Task Force

Endorsed by the Society for Vascular Medicine

James H. Stein, MD, FASE, Claudia E. Korcarz, DVM, RDCS, FASE, R. Todd Hurst, MD, Eva Lonn MD, MSc, FASE, Christopher B. Kendall, BS, RDCS, Emile R. Mohler, MD, Samer S. Najjar, MD, Christopher M. Rembold, MD, and Wendy S. Post, MD, MS,
Madison, Wisconsin; Scottsdale, Arizona; Hamilton, Ontario, Canada; Philadelphia, Pennsylvania; Baltimore, Maryland; and Charlottesville, Virginia

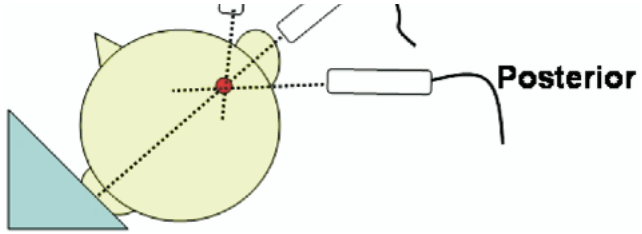


Figure 2 Head position and probe orientation for carotid ultrasound scanning, right-side example.

Transversal scanning: at least 3 IMT

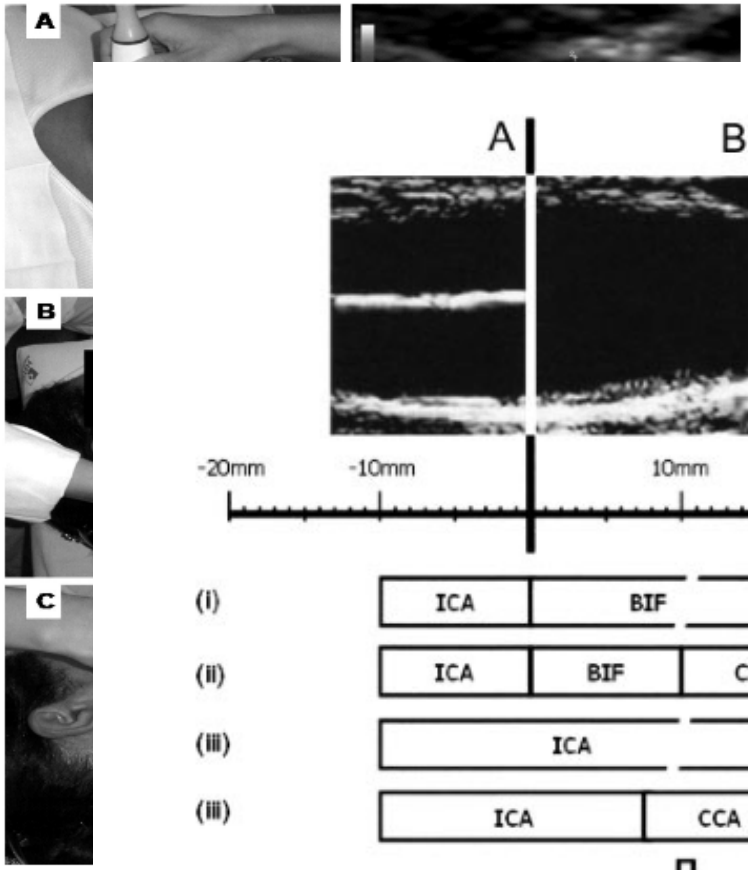


Figure 1
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g for
3:
Measurements of the F-wall right and/or left CCA

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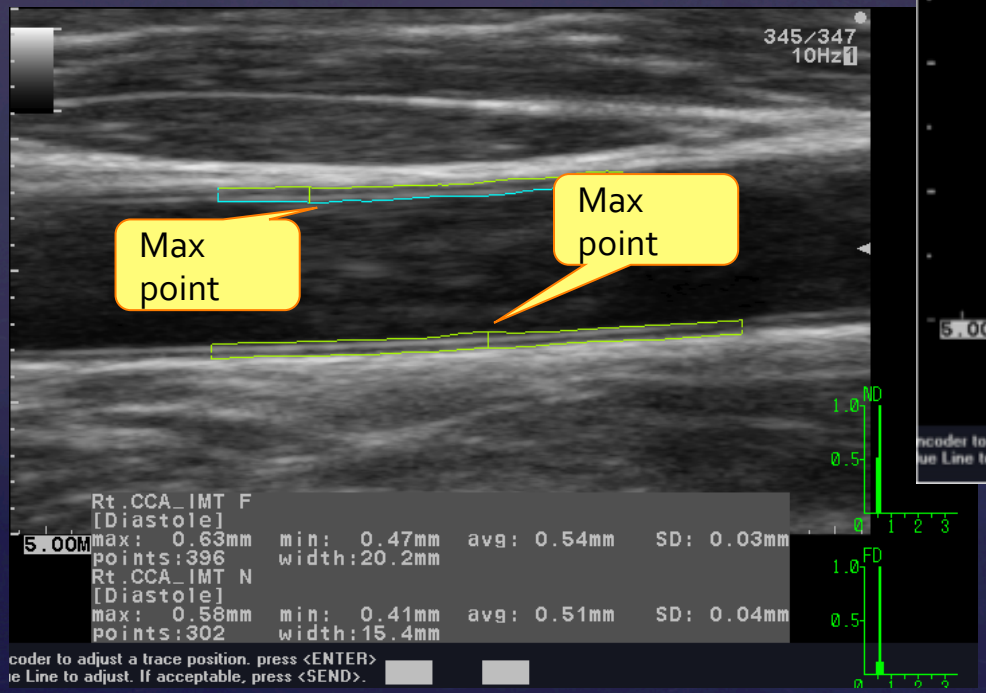
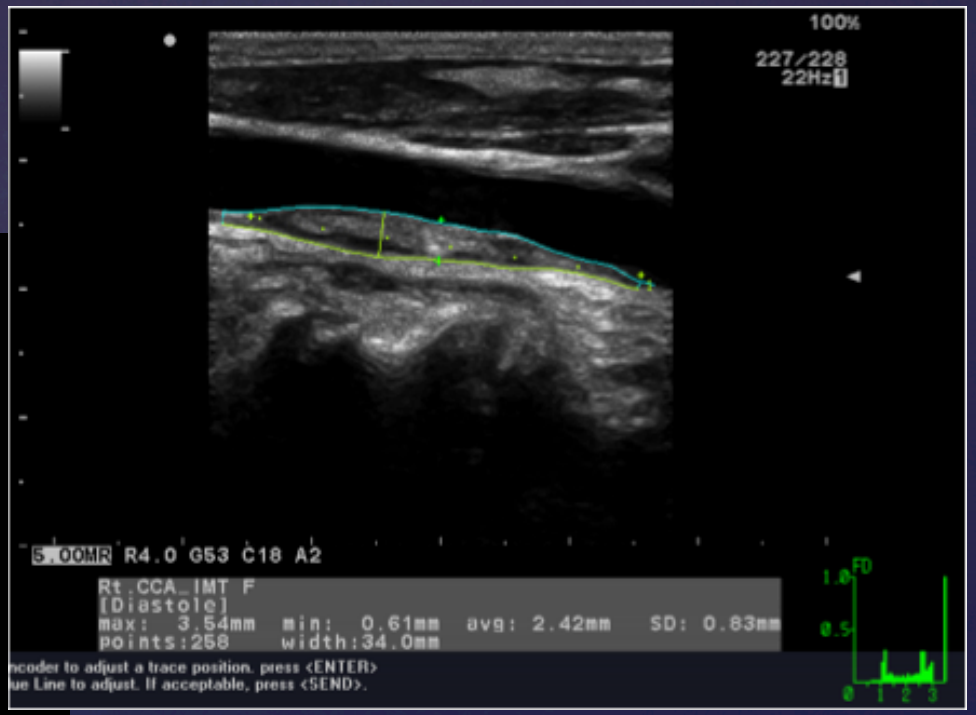
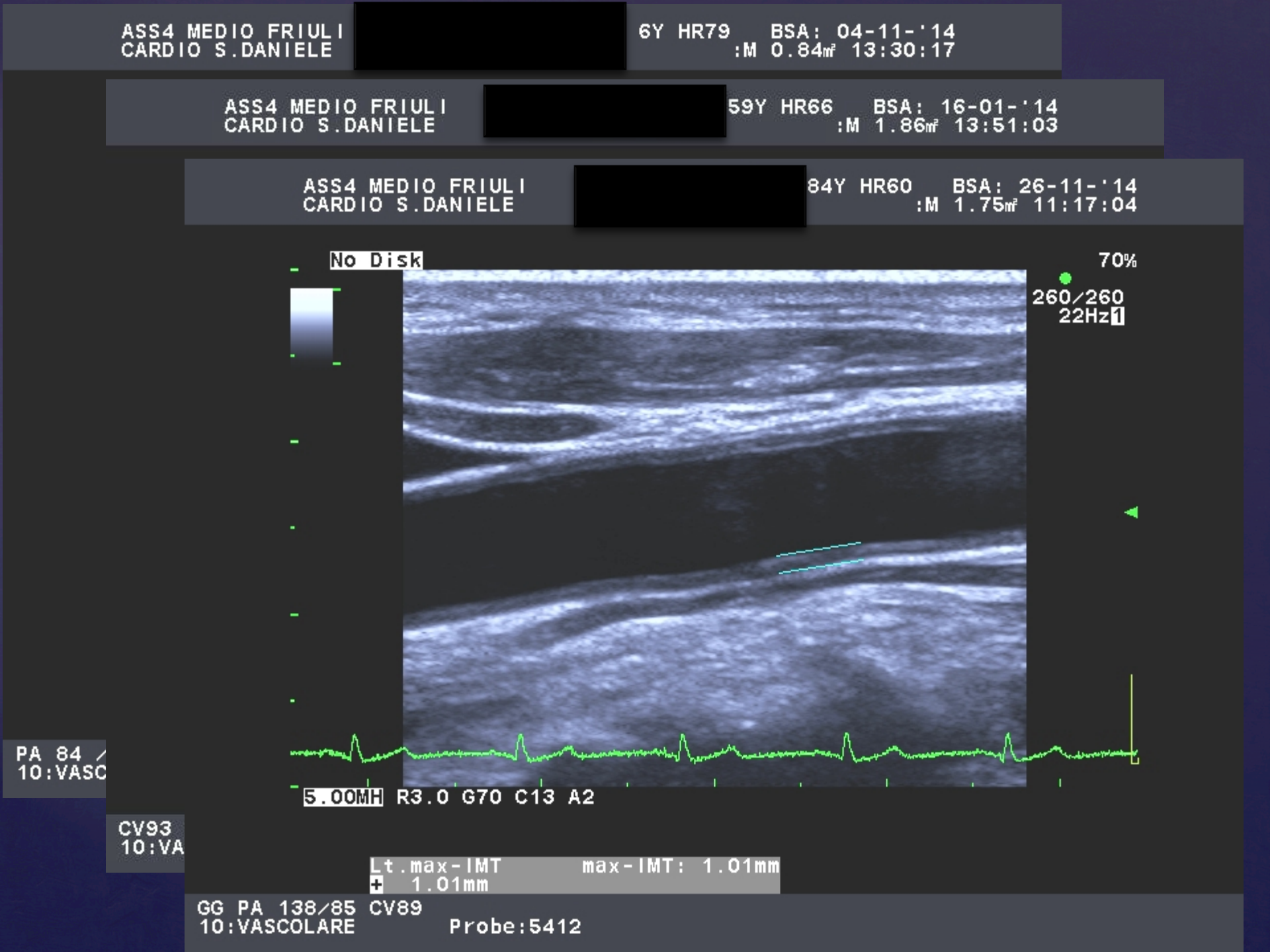
LETTURA AUTOMATICA

- Displayed items
  - max, min, average
  - SD, points (How many points are used for the result)
  - Width of ROI
  - Histogram






ASS4 MEDIO FRIULI
CARDIO S.DANIELE
[redacted]
6Y HR79 BSA: 04-11-'14
:M 0.84m² 13:30:17
ASS4 MEDIO FRIULI
CARDIO S.DANIELE
[redacted]
59Y HR66 BSA: 16-01-'14
:M 1.86m² 13:51:03
ASS4 MEDIO FRIULI
CARDIO S.DANIELE
[redacted]
84Y HR60 BSA: 26-11-'14
:M 1.75m² 11:17:04
No Disk
70%
260/260
22Hz 1
5.00MH R3.0 G70 C13 A2
PA 84 /
10:VASC
CV93
10:VA
Lt.max-IMT max-IMT: 1.01mm
1.01mm
GG PA 138/85 CV89
10:VASCOLARE Probe:5412

Carotid IMT varies through the cardiac cycle by 0.03 mm, thickest in end diastole and thinnest at peak systole.

In healthy middle-age adults, the distance from the CCA lumen – intima interface and the media–adventia interface measures 0.6–0.7 mm
3 segments each 1 cm lenghth: CCA , CB, ICA
Complex artery with different association for each segment with risk factors and outcome.

Atherosclerotic plaque: encroaches into the arterial lumen of at least 0.5 mm, thickness >1.5 mm from media-adventitia to intima-lumen interface

Lessons From the Past and Promises for the Future for Carotid Intima-Media Thickness

Michiel L. Bots, MD, PhD,* Kim Sutton-Tyrrell, DrPH†

What is CIMT

Which CIMT measurement is the best?

What is the best: near versus far wall?

What is the best: CCA alone versus adding the bifurcation and internal measurements?

Quantification of CIMT: manual versus semi-automated edge detection.

Use of CIMT in risk classification.

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(J Am Coll Cardiol 2012;60:1599–604) ©

2013 ACC/AHA Guideline on the Assessment of Cardiovascular Risk☆

Assessment of 10-Year Risk of a First Hard ASCVD Event

Table 2. NHLBI Grading of the Strength o Recommendations

E	**Expert opinion ("There is insufficient evidence or evidence is unclear or conflicting, but this is what the Work Group recommends.")** Net benefit is unclear. Balance of benefits and harms cannot be determined because of no evidence, insufficient evidence, unclear evidence, or conflicting evidence, but the Work Group thought it was important to provide clinical guidance and make a recommendation. Further research is recommended in this area.

Atherosclerotic cardiovascular disease event (ASCVD) of a

NHLBI Grade: E (Expert Opinion); ACC/AHA COR: IIb†, LOE: B†

Recommendation 2. Routine measurement of CIMT is not recommended in clinical practice for risk assessment for a first ASCVD event.

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2013 ESH/ESC Guidelines for the management of arterial hypertension

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3.7.2 Blood vessels

3.7.2.1 Carotid arteries

Ultrasound examination of the carotid arteries with measurement of intima media thickness (IMT) and/or the presence of plaques has been shown to predict the occurrence of both stroke and myocardial infarction, independently of traditional CV risk factors.[51,183–186] This holds true, both for the IMT value at the carotid bifurcations (reflecting primarily atherosclerosis) and for the IMT value at the level of the common carotid artery (reflecting primarily vascular hypertrophy). The relationship between carotid IMT and CV events is a continuous one and determining a threshold for high CV risk is rather arbitrary. Although a carotid IMT >0.9 mm has been taken as a conservative estimate of existing abnormalities in the 2007 Guidelines,[2] the threshold value for high CV risk was higher in the elderly patients of the Cardiovascular Health Study and in the middle-aged patients of the European Lacidipine Study on Atherosclerosis (ELSA) study (1.06 and 1.16 mm, respectively).[184,186] Presence of a plaque can be identified by an IMT ≥1.5 mm or by a focal increase in thickness of 0.5 mm or 50% of the surrounding carotid IMT value.[187] Although plaque has a strong independent predictive value for CV events,[51,183–185,188] presence of a plaque and increased carotid IMT added little to each other for predicting CV events and re-classifying patients into another risk category in the Atherosclerosis Risk In Communities (ARIC) study.[185] A recent systematic review concluded that the added predictive value of additional carotid screening may be primarily found in asymptomatic individuals at intermediate CV risk.[189]

Table 4 Factors—other than office BP—influencing prognosis; used for stratification of total CV risk in *Figure 1*

Asymptomatic organ damage
Pulse pressure (in the elderly) ≥60 mmHg
Electrocardiographic LVH (Sokolow–Lyon index >3.5 mV; RaVL >1.1 mV; Cornell voltage duration product >244 mV*ms), or
Echocardiographic LVH [LVM index: men >115 g/m²; women >95 g/m² (BSA)][a]
Carotid wall thickening (IMT >0.9 mm) or plaque
Carotid–femoral PWV >10 m/s
Ankle-brachial index <0.9
CKD with eGFR 30–60 mL/min/1.73 m² (BSA)
Microalbuminuria (30–300 mg/24 h), or albumin–creatinine ratio (30–300 mg/g; 3.4–34 mg/mmol) (preferentially on morning spot urine)

The combination of C-IMT+ carotid plaques improve the prediction of CVD

Screening for Heart Attack Prevention and Education (SHAPE) Task Force

- "The SHAPE Task Force strongly recommends screening of the at-risk asymptomatic population (men 45-75 years of age and women 55-75 years of age) for subclinical atherosclerosis to more accurately identify and treat patients at high risk for acute ischemic events, as well as to identify those at lower risk who may be treated more conservatively."

The American Journal of Cardiology, Vol 98 (2A) July 17, 2006

Risk factors for the progression of carotid intima-media thickness over a 16-year follow-up period: The Malmö Diet and Cancer Study

M. Rosvall*, [1], M. Persson [1], G. Östling, P.M. Nilsson, O. Melander, B. Hedblad, G. Engström

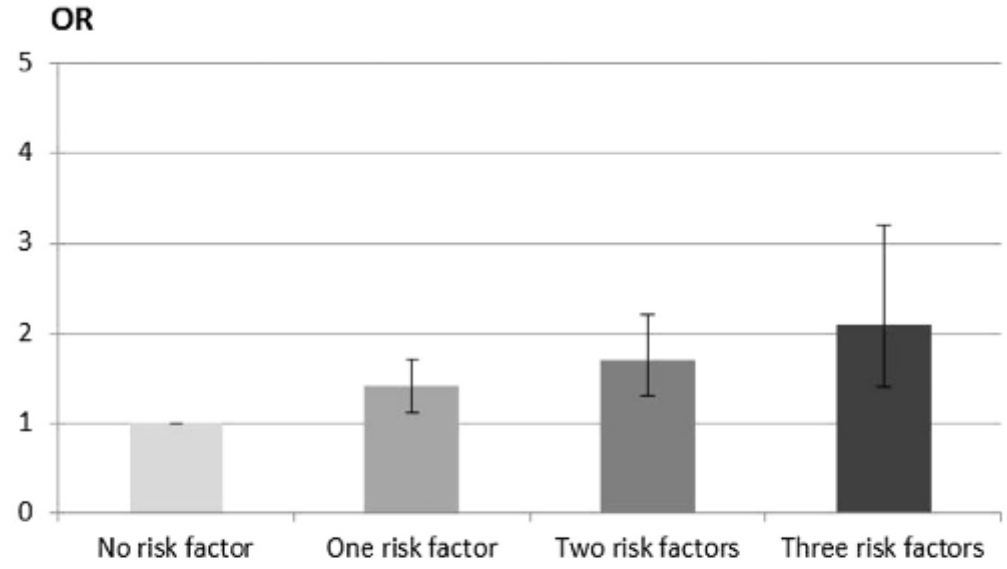

Fig. 1. Age, sex, and baseline IMT-adjusted odds ratios of high IMT CCA progression rate (>75th percentile) among subjects with 0, 1, 2, and 3 risk factors. Risk factors were hypertension (defined as systolic blood pressure >140 mm Hg or diastolic blood pressure >90 mm Hg or on antihypertensive treatment), LDL/HDL ≥3, and regular smoking. Data from the Malmö Diet and Cancer Study, 1991–1994 (baseline examination) and 2007–2012 (re-examination).

3426 sbj
Mean CCA IMT automatically measured
(max Far wall CB)

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Atherosclerosis 239 (2015) 615–621

Carotid intima-media thickness and presence or absence of plaque improves prediction of coronary heart disease risk in the Atherosclerosis Risk in Communities (ARIC) study

15792, 45-64 yrs
Cox proportional hazards model was used for 10-yr CHD risk

Table 3A: Number and percent reclassified in CHD risk categories and observed CHD risk* when C-IMT and plaque information are added to traditional risk prediction models (overall sample)

CHD risk by TRF only	CHD risk by TRF + C-IMT + plaque				
	<5%	5–10%	10–20%	>20%	All
	Number % KM 10-year risk(%)	Number % KM 10- year risk (%)	Number % KM 10-year risk (%)	Number % KM 10-year risk (%)	Number % KM 10-year risk (%)
≤5%, Low risk, n	5585	523	0	0	6108
	91.4	8.6	0.00	0.00	46.5
	2	5	.	.	2
5–10%, Low-intermediate risk, n	839	2340	563	0	3742
	22.4	62.5	15.1	0.00	28.5
	5	7	17	.	8
10–20% High-intermediate risk, n	0	627	1560	340	2527
	0.00	24.8	61.7	13.5	19.2
	.	11	15	24	15
>20% High risk, n	0	0	165	603	768
	0.00	0.00	21.5	78.5	5.8
	.	.	14	31	27
All, n	6264	3490	2288	943	13145
	48.9	26.6	17.4	7.2	100.0
	2	7	15	28	7

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Nambi et al

J Am Coll Cardiol. 2010

Carotid intima-media thickness progression to predict cardiovascular events in the general population (the PROG-IMT collaborative project): a meta-analysis of individual participant data

Lancet 2012; 379: 2053–62

Matthias W Lorenz, Joseph F Polak, Maryam Kavousi, Ellisiv B Mathiesen, Henry Völzke, Tomi-Pekka Tuomainen, Dirk Sander, Matthieu Plichart, Alberico L Catapano, Christine M Robertson, Stefan Kiechl, Tatjana Rundek, Moïse Desvarieux, Lars Lind, Caroline Schmid, Pronabesh DasMahapatra, Lu Gao, Kathrin Ziegelbauer, Michiel L Bots, Simon G Thompson, on behalf of the PROG-IMT Study Group

Summary

Background Carotid intima-media thickness (cIMT) is related to the risk of cardiovascular events in the general population. An association between changes in cIMT and cardiovascular risk is frequently assumed but has rarely been reported. Our aim was to test this association.

Methods We identified general population studies that assessed cIMT at least twice and followed up participants for myocardial infarction, stroke, or death. The study teams collaborated in an individual participant data meta-analysis. Excluding individuals with previous myocardial infarction or stroke, we assessed the association between cIMT progression and the risk of cardiovascular events (myocardial infarction, stroke, vascular death, or a combination of these) for each study with Cox regression. The log hazard ratios (HRs) per SD difference were pooled by random effects meta-analysis.

Findings Of 22 eligible studies, 16 with 36 984 participants were included. During a mean follow-up of 7·0 years, 1519 myocardial infarctions, 1339 strokes, and 2028 combined endpoints (myocardial infarction, stroke, vascular death) occurred. Yearly cIMT progression was derived from two ultrasound visits 2–7 years (median 4 years) apart. For mean common carotid artery intima-media thickness progression, the overall HR of the combined endpoint was 0·97 (95% CI 0·94–1·00) when adjusted for age, sex, and mean common carotid artery intima-media thickness, and 0·98 (0·95–1·01) when also adjusted for vascular risk factors. Although we detected no associations with cIMT progression in sensitivity analyses, the mean cIMT of the two ultrasound scans was positively and robustly associated with cardiovascular risk (HR for the combined endpoint 1·16, 95% CI 1·10–1·22, adjusted for age, sex, mean common carotid artery intima-media thickness progression, and vascular risk factors). In three studies including 3439 participants who had four ultrasound scans, cIMT progression did not correlate between occassions (reproducibility correlations between r=–0·06 and r=–0·02).

Interpretation The association between cIMT progression assessed from two ultrasound scans and cardiovascular risk in the general population remains unproven. No conclusion can be derived for the use of cIMT progression as a surrogate in clinical trials.

Carotid arterial stiffness

- Arterial stiffness is one of the main determinants of SBP, PP and then CV events.

- Arterial stiffness has also an independent predictive value for CV events in patients with hypertension, diabetes, end-stage renal disease in elderly subjects and in general population.

- Arterial stiffening and cardiac function should be considered as an unicum: *ventricular arterial coupling*

PWV as part of the HT stratification ….

Asymptomatic organ damage
Pulse pressure (in the elderly) ≥60 mmHg
Electrocardiographic LVH (Sokolow–Lyon index >3.5 mV; RaVL >1.1 mV; Cornell voltage duration product >244 mV*ms), or
Echocardiographic LVH [LVM index: men >115 g/m^2; women >95 g/m^2 (BSA)][a]
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CKD with eGFR 30–60 mL/min/1.73 m^2 (BSA)
Microalbuminuria (30–300 mg/24 h), or albumin–creatinine ratio (30–300 mg/g; 3.4–34 mg/mmol) (preferentially on morning spot urine)

ESH AND ESC GUIDELINES

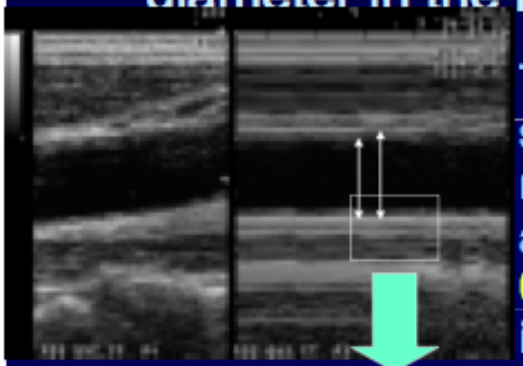
European Heart Journal (2013) **34**, 2159–2219
doi:10.1093/eurheartj/eht151

…and aortic disease

2014 ESC Guidelines on the diagnosis and treatment of aortic diseases

European Heart Journal Advance Access published August 29, 2014

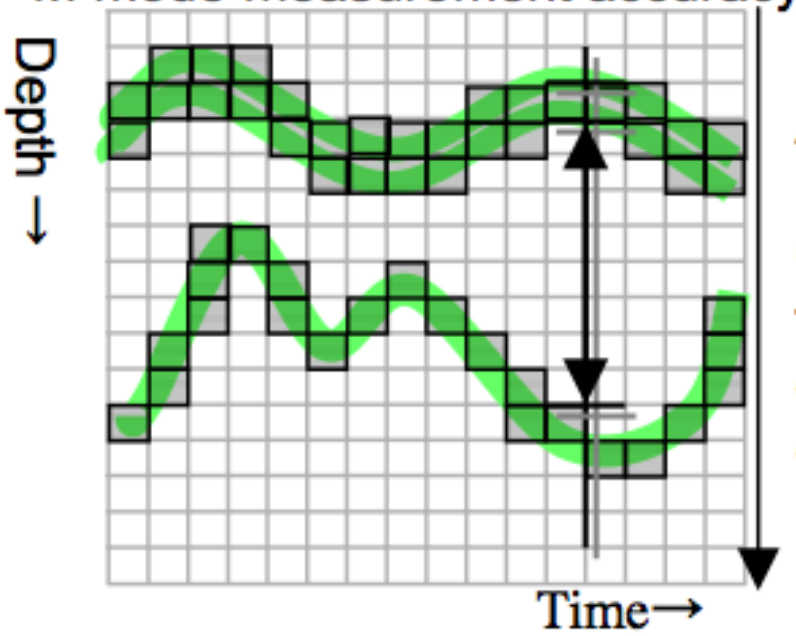
Measurement of blood vessel diameter in the M mode



The smallest step of measurement is approximately **0.1 mm** at best in the M mode.

In the ultrasound system, measurement is conducted by counting the number of pixels. Therefore, a resolution smaller than a pixel is not available.

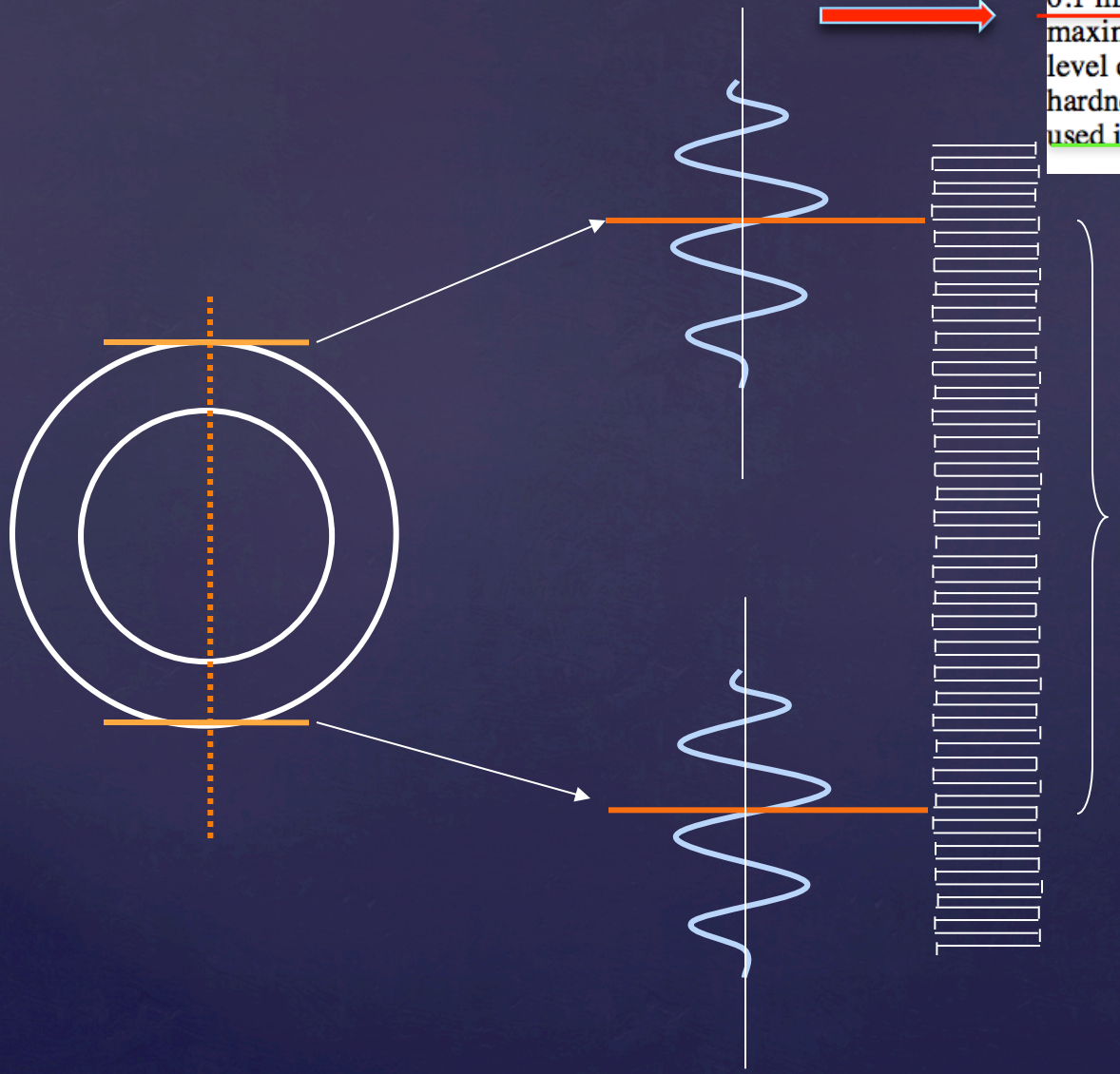
M mode measurement accuracy



Approx. 400 pixels.
0.1 mm/pixel for 4 cm diagnostic distance.

What's eTRACKING : Principle

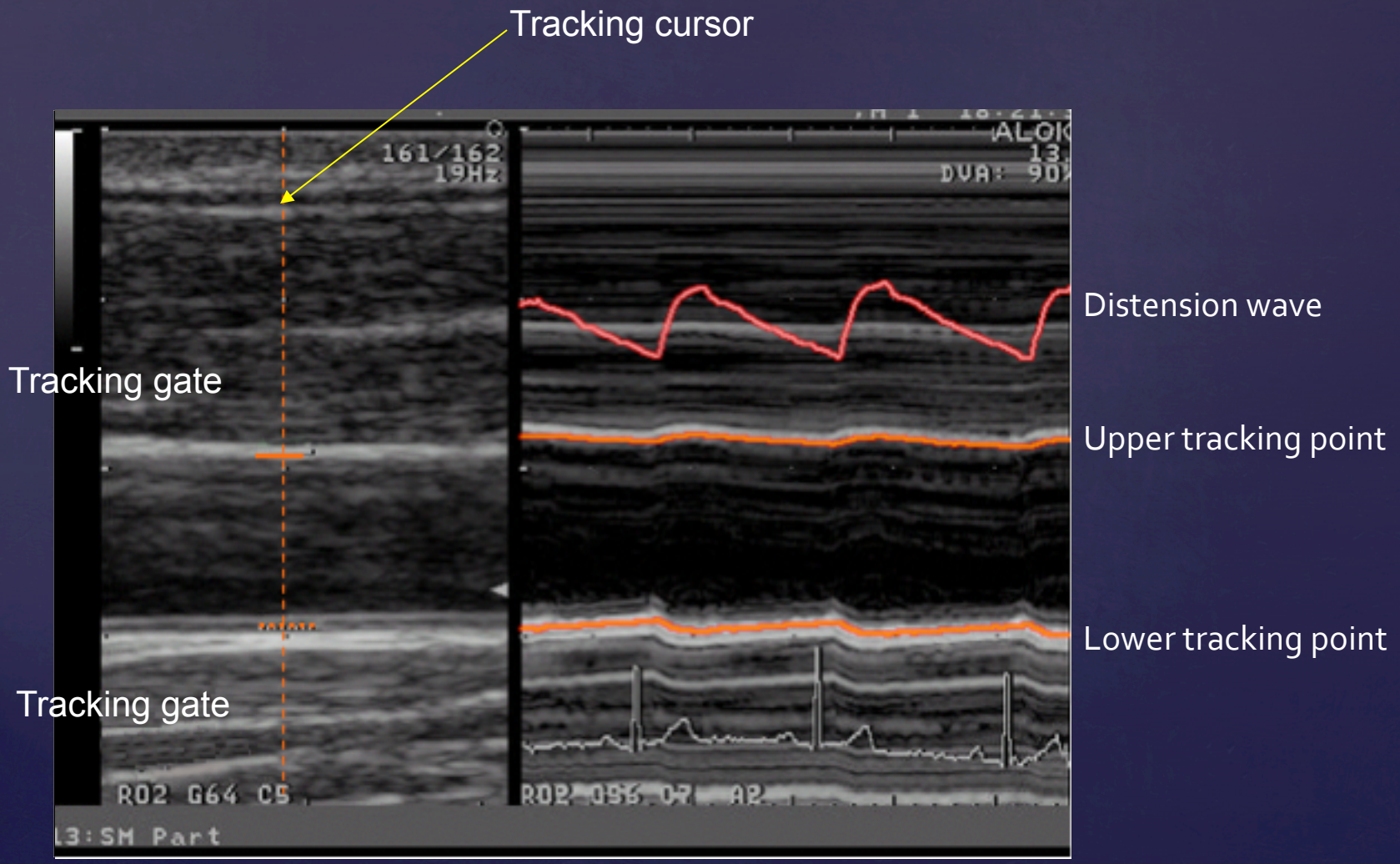
Even if the target area is enlarged, errors are inevitable by arbitrarily selecting the area of the membrane for measurement. Fineness of the M-mode measurement depends on distance resolution of the ultrasonic diagnostic unit and fineness (pixels) of the image. Generally, the smallest measuring step is approximately 0.1 mm. For hard arteries, the difference between maximum and minimum diameter is small, so that this level of accuracy is not enough to correctly evaluate the hardness. To solve the problem, RF echo tracking is used in eTRACKING.



The distance between two gates shows the vessel diameter.

At 10 MHz frequency precision is 0.01mm

Movement of the vessel wall is tracked automatically by simply setting the tracking gate on the B-mode image. Displays waveforms of changes in vessel diameter (distension wave) in real time.



Calculated parameters

Ps: systolic pressure
Pd: diastolic pressure
Ds: maximum vessel diameter
Dd: minimum vessel diameter

β (stiffness parameter): index of arterial stiffness

$\beta = \ln(Ps / Pd) / [(Ds - Dd) / Dd]$

Calculated from changes in vessel diameter and blood pressure
Arterial stiffness ↑ β value ↑
Less dependent on blood pressure

Ep (pressure-strain elasticity modulus): index of vessel elasticity

$Ep = (Ps - Pd) / [(Ds - Dd) / Dd]$

Calculated from changes in vessel diameter and blood pressure
Arterial elasticity ↓ Ep ↑
The value greatly varies with blood pressure even in the same blood vessel.

AC (arterial compliance): index of blood vessel compliance

$AC = \pi(Ds \times Ds - Dd \times Dd) / [4 (Ps - Pd)]$

Calculated from changes in blood vessel cross-sectional area and blood pressure
Arterial compliance ↑ AC ↑

PWVβ　One –Point Pulse wave velocity

PWV is mainly related to the elastic properties of the arterial wall.

PWV is well known as a parameter to assess the atherosclerosis.

Conventional PWV has been based on the two point measurement.

PWV is the velocity at which the pulse wave travel between known distance.

When the artery becomes stiffer, the PWV shows higher value.

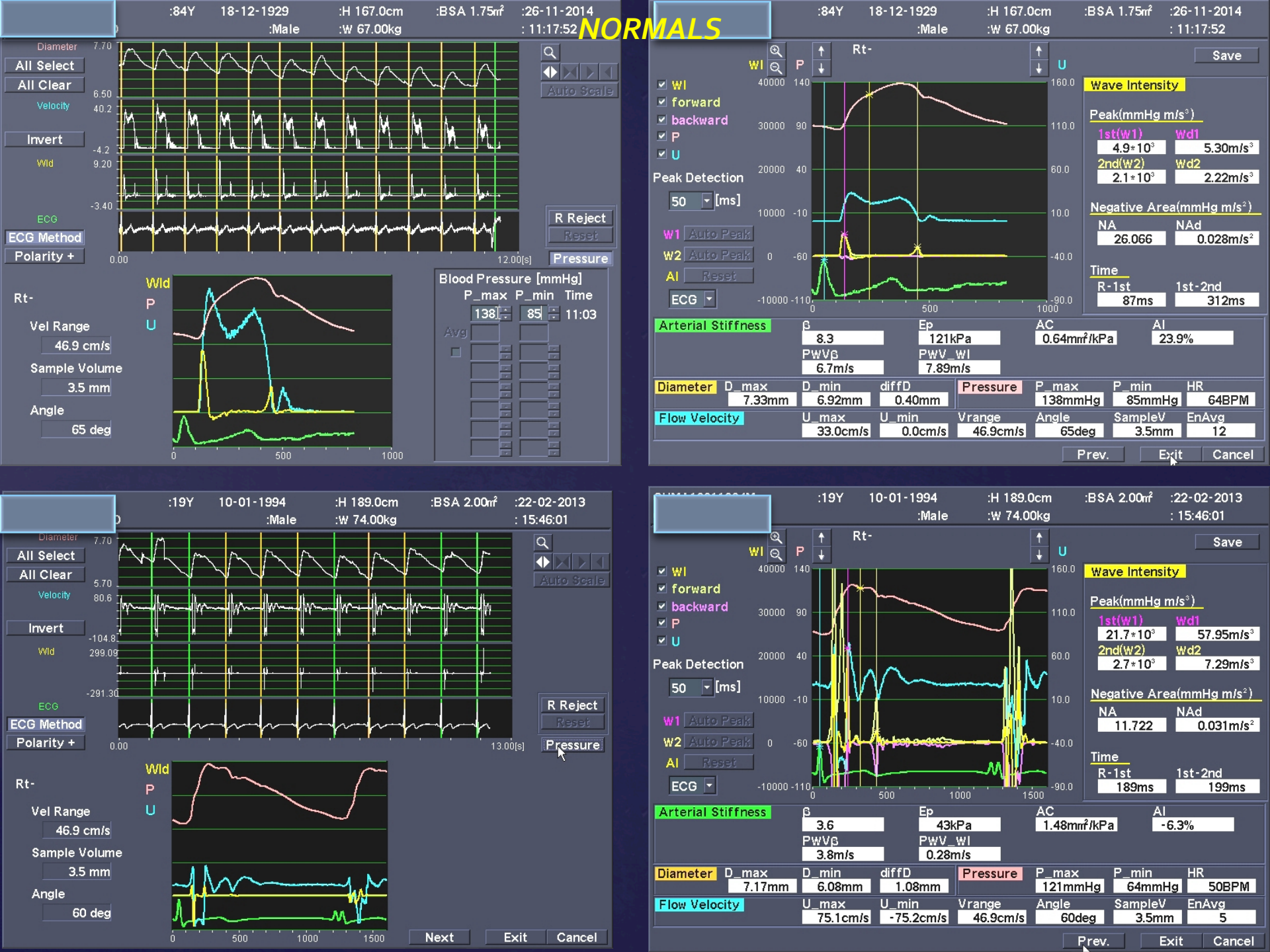
This time, we developed one point pulse wave velocity method derived from Stiffness parameter β

Definitional equation $C = \sqrt{(\beta P / 2\rho)}$

P：Diastolic blood pressure

ρ：blood density (1050 Kg/m^3)

"Non-invasive one-point measurement of local pulse wave velocity"
Sugawara, et al., Tokyo Women's Medical University
Saishin Igaku, 2003,6,279-288


NORMALS
:84Y 18-12-1929 :H 167.0cm :BSA 1.75m² :26-11-2014
:Male :W 67.00kg : 11:17:52
Diameter
All Select
All Clear
Velocity
Invert
Wid
ECG
ECG Method
Polarity +
R Reject
Reset
Pressure
Auto Scale
Rt-
Vel Range
46.9 cm/s
Sample Volume
3.5 mm
Angle
65 deg
Wid
P
U
Blood Pressure [mmHg]
P_max P_min Time
138 85 11:03
Avg
WI
forward
backward
P
U
Peak Detection
50 [ms]
W1 Auto Peak
W2 Auto Peak
AI Reset
ECG
Save
Wave Intensity
Peak(mmHg m/s³)
1st(W1) Wd1
4.9*10³ 5.30m/s³
2nd(W2) Wd2
2.1*10³ 2.22m/s³
Negative Area(mmHg m/s²)
NA NAd
26.066 0.028m/s²
Time
R-1st 1st-2nd
87ms 312ms
Arterial Stiffness
β 8.3
Ep 121kPa
AC 0.64mm²/kPa
AI 23.9%
PWVβ 6.7m/s
PWV_WI 7.89m/s
Diameter D_max 7.33mm D_min 6.92mm diffD 0.40mm
Pressure P_max 138mmHg P_min 85mmHg HR 64BPM
Flow Velocity U_max 33.0cm/s U_min 0.0cm/s Vrange 46.9cm/s Angle 65deg SampleV 3.5mm EnAvg 12
Prev. Exit Cancel
:19Y 10-01-1994 :H 189.0cm :BSA 2.00m² :22-02-2013
:Male :W 74.00kg : 15:46:01
Vel Range
46.9 cm/s
Sample Volume
3.5 mm
Angle
60 deg
Next Exit Cancel
Arterial Stiffness
β 3.6
Ep 43kPa
AC 1.48mm²/kPa
AI -6.3%
PWVβ 3.8m/s
PWV_WI 0.28m/s
Diameter D_max 7.17mm D_min 6.08mm diffD 1.08mm
Pressure P_max 121mmHg P_min 64mmHg HR 50BPM
Flow Velocity U_max 75.1cm/s U_min -75.2cm/s Vrange 46.9cm/s Angle 60deg SampleV 3.5mm EnAvg 5
Prev. Exit Cancel


ASS4 MEDIO FRIULI
CARDIO S.DANIELE
HR66 BSA: 13-11-'14
:M 1.92㎡ 15:19:23
27-09-1936 :H 172.0cm :BSA 1.92㎡ :13-11-2014
27-09-1936 :H 172.0cm :BSA 1.92㎡ :13-11-2014
:Male :W 79.00kg : 15:28:40
Rt-
WI
P
U
Save
WI
forward
backward
P
U
Peak Detection
50 [ms]
W1 Auto Peak
W2 Auto Peak
AI Reset
ECG
40000 160
30000 110
20000 60
10000 10
0 -40
-10000 -90
160.0
110.0
60.0
10.0
-40.0
-90.0
0
500
1000
Wave Intensity
Peak(mmHg m/s³)
1st(W1) 5.9*10³
Wd1 1.09m/s³
2nd(W2) 2.1*10³
Wd2 0.39m/s³
Negative Area(mmHg m/s²)
NA 31.277
NAd 0.006m/s²
Time
R-1st 183ms
1st-2nd 254ms
Arterial Stiffness
β 46.8
Ep 716kPa
AC 0.09mm²/kPa
AI 23.2%
PWVβ 16.3m/s
PWV_WI 3.26m/s
Diameter
D_max 6.58mm
D_min 6.52mm
diffD 0.07mm
Pressure
P_max 144mmHg
P_min 90mmHg
HR 76BPM
Flow Velocity
U_max 56.7cm/s
U_min -8.0cm/s
Vrange 46.9cm/s
Angle 75deg
SampleV 3.5mm
EnAvg 10
Prev.
Exit
Cancel

Arterial stiffness:regional and local stiffness

EHJ 2006;27:2588

Although carotid-femoral PWV and carotid stiffness provide similar information on the impact of ageing on large artery stiffness in normal subjects, this is not the case for high blood pressure and/or diabetes. In these cases, the aorta stiffened more than the carotid artery with age and other CV risk factors.[66] Thus, aortic stiffness and carotid stiffness cannot be used as interchangeable predictors in high-risk patients.

Carotid and Aortic Stiffness

Determinants of Discrepancies

Anna Paini, Pierre Boutouyrie, David Calvet, Anne-Isabelle Tropeano, Brigitte Laloux, Stéphane Laurent

Abstract—Several studies have shown that aortic stiffness was an independent predictor for cardiovascular events. However, data are less consistent concerning carotid stiffness. We analyzed the determinants of the discrepancies between aortic and carotid stiffness in different populations with contrasting cardiovascular risk factors: 94 healthy normotensives (NT), 243 nondiabetic hypertensives (HT), and 126 patients with hypertension and type 2 diabetes (T2D). Aortic stiffness was measured with carotid-femoral pulse wave velocity. Common carotid stiffness was determined from the relative stroke change in diameter (measured with a high-resolution echotracking system) and carotid pulse pressure (measured with applanation tonometry) and was expressed in the same dimensions as pulse wave velocity (m/s). We identified the various factors explaining the discrepancies between aortic and carotid stiffness by multivariate analysis of the residuals of the correlation between aortic and carotid stiffness. The strength of the correlation between aortic and carotid stiffness became weaker as the number of cardiovascular risk factors increased (NT, r^2=0.41; HT, r^2=0.16; and T2D, r^2=0.11), whereas we observed the opposite for the discrepancies (residuals) between aortic and carotid stiffness, of which an increasing part was explained (11% in NT, 22% in HT, and 45% in T2D) primarily by aging. In conclusion, although carotid-femoral pulse wave velocity and carotid stiffness provided similar information on the impact of aging on large artery stiffness in normal subjects, this was not the case for high blood pressure and/or diabetes. In these cases, the aorta stiffened more than the carotid artery with age and other cardiovascular risk factors. (***Hypertension***. 2006;47:371-376.)

Local Stiffness of the Carotid and Femoral Artery Is Associated With Incident Cardiovascular Events and All-Cause Mortality

The Hoor

Thomas T. v
Jacqueline M
Coen D. A.

Maastricht ar

579 population based cohort study. F-up 7.6 yrs

JACC 2014

Table 2 Associations of Arterial Stiffness Indices of the Carotid, Femoral, and Brachial Arteries With Incident CV Events and All-Cause Mortality

Model	Carotid Artery			Femoral Artery		Brachial Artery	
	DC	YEM	CC	DC	CC	DC	CC
Incident Cardiovascular Events							
1	1.27 (1.02–1.57)	1.22 (1.04–1.42)	1.07 (0.88–1.30)	1.39 (1.07–1.81)	1.29 (0.99–1.67)	0.90 (0.74–1.08)	0.84 (0.71–1.01)
2	1.24 (0.98–1.58)	1.20 (1.02–1.42)	1.04 (0.86–1.28)	1.37 (1.04–1.80)	1.26 (0.97–1.65)	0.87 (0.72–1.06)	0.84 (0.70–1.00)
3	1.22 (0.95–1.56)	1.19 (1.00–1.41)	1.08 (0.88–1.31)	[illegible]–1.83)	1.25 (0.96–1.63)	0.88 (0.72–1.07)	0.85 (0.71–1.02)
All-Cause Mortality							
1	1.62 (1.24–2.13)	1.33 (1.15–1.55)	1.50 (1.16–1.95)	[illegible]–1.88)	1.59 (1.11–2.28)	0.98 (0.77–1.25)	1.06 (0.84–1.33)
2	1.52 (1.13–2.05)	1.27 (1.08–1.50)	1.41 (1.08–1.84)	1.27 (0.90–1.78)	1.52 (1.06–2.19)	0.92 (0.72–1.17)	1.02 (0.82–1.28)
3	1.51 (1.11–2.06)	1.28 (1.07–1.53)	1.43 (1.10–1.86)	1.27 (0.90–1.79)	1.47 (1.01–2.13)	0.90 (0.70–1.15)	0.99 (0.78–1.25)

Values are hazard ratio (95% confidence interval). Hazard ratios are indicated per 1 SD lower distensibility (DC) and compliance coefficient (CC) and per 1 SD higher Young's elastic modulus (YEM). Model 1: adjusted for age, sex, and glucose metabolism status. Model 2: model 1 plus mean arterial pressure. Model 3: model 2 plus prior cardiovascular disease, body mass index, triglycerides, total/HDL cholesterol ratio, estimated glomerular filtration rate, microalbuminuria, physical activity, and smoking habits. The number of participants and events available for analysis with incident CV events/all-cause mortality for carotid DC, CC, and YEM: 533 (130 events)/579 (96 events); for femoral DC and CC: 461 (111 events)/498 (77 events); and for brachial DC and CC: 478 (116 events)/517 (81 events).

Abbreviations as in Table 1.

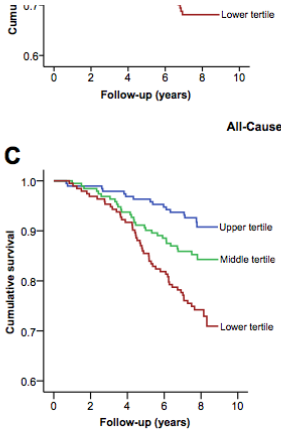


Figure 2 Kaplan-Meier Curves for Incident CV Events and All-Ca

Kaplan-Meier curves for incident cardiovascular (CV) events and all-cause mortality femoral arteries (**B and D**).

Table 3 Associations of the cfPWV, Aix, and SAC With Incident Cardiovascular Events and All-Cause Mortality

Model	cfPWV	Aix	SAC	
			Time-Decay Method	SV/Aortic PP
Incident Cardiovascular Events				
1	1.57 (1.29–1.92)	1.08 (0.89–1.31)	1.09 (0.88–1.35)	1.15 (0.90–1.47)
2	1.56 (1.27–1.93)	1.05 (0.86–1.28)	1.03 (0.81–1.30)	1.09 (0.84–1.43)
3	1.56 (1.23–1.98)	0.99 (0.81–1.22)	1.00 (0.79–1.26)	1.13 (0.87–1.47)
All-Cause Mortality				
1	1.27 (0.99–1.63)	1.05 (0.84–1.32)	1.22 (0.92–1.61)	1.05 (0.70–1.58)
2	1.18 (0.88–1.57)	1.19 (0.92–1.54)	1.06 (0.78–1.43)	1.05 (0.67–1.65)
3	1.13 (0.83–1.54)	0.93 (0.73–1.18)	1.04 (0.76–1.41)	1.01 (0.73–1.41)

Values are hazard ratio (95% confidence interval). Hazard ratios are indicated per 1 SD higher carotid femoral pulse wave velocity (cfPWV) and aortic augmentation index (Aix) and per 1 SD lower systemic arterial compliance (SAC). Model 1: adjusted for age, sex, and glucose metabolism status. Model 2: model 1 plus mean arterial pressure and heart rate. Model 3: model 2 plus prior cardiovascular disease, body mass index, triglycerides, total/HDL cholesterol ratio, estimated glomerular filtration rate, microalbuminuria, physical activity, and smoking habits. Number of participants and events available for analysis with incident cardiovascular events/all-cause mortality for cfPWV: 215 (53 events)/237 (36 events); for Aix: 500 (120 events)/543 (87 events); for SAC (time-decay method): 457 (110 events)/492 (77 events); and for SAC (SV/aortic PP) 448 (106 events)/482 (70 events).

PP = pulse pressure; SV = stroke volume; other abbreviations as in Table 1.

Carotid Artery Stiffness and Diastolic Function in Subjects without Known Cardiovascular Disease

Olga Vriz, MD, Eduardo Bossone, MD, PhD, FESC, FACC, Manola Bettio, MD, Daniela Pavan, MD, Scipione Carerj, MD, and Francesco Antonini-Canterin, MD, *Udine, Milan, Pordenone, and Messina, Italy*

JASE 2011

Background: The aim of this study was to investigate the relationship between carotid artery stiffness and diastolic function in a cohort of subjects without known cardiovascular risk factors and/or overt cardiovascular disease.

Methods: Ninety-two healthy subjects underwent transthoracic echocardiographic Doppler and carotid echo-tracking studies. Measurements of local arterial stiffness were obtained at left common carotid artery level; stiffness parameter (β), and pressure-strain elasticity modulus (Ep) were calculated as well as intima-media thickness (IMT).

Results: Stiffness parameter and Ep were correlated inversely with transmitral E wave ($P < .01$), E/A ratio, and septal Em ($P < .01$) and positively with A wave ($P < .001$). IMT was also associated with A wave, E/A ratio, Em, and Am but not with E wave. No association was found between IMT, β, and Ep. The correlation between arterial stiffness and left ventricular diastolic function remained significant after multivariate adjustment for age, sex, pulse pressure, and body mass index, but not with IMT.

Conclusions: In healthy subjects, changes in central carotid stiffness are in line with left ventricular diastolic function independently of age, sex, pulse pressure, and body mass index. (J Am Soc Echocardiogr

Carotid Artery Stiffness and Diastolic Function in Subjects without Known Cardiovascular Disease

Olga Vriz, MD, Eduardo Bossone, MD, PhD, FESC, FACC, Manola Bettio, MD, Daniela Pavan, MD, Scipione Carerj, MD, and Francesco Antonini-Canterin, MD, *Udine, Milan, Pordenone, and Messina, Italy*

JASE 2011

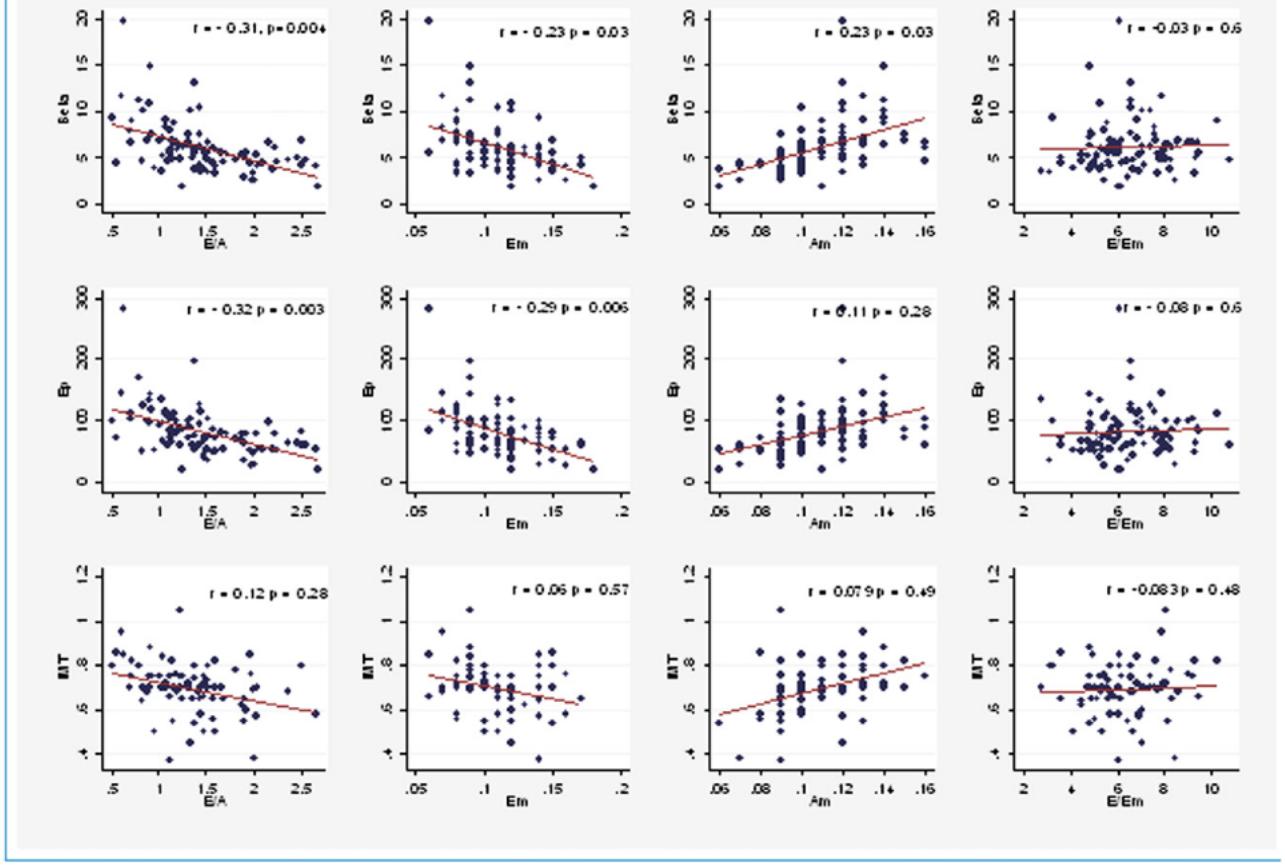


Figure 3 Correlations between arterial stiffness, IMT, and LV diastolic parameters adjusted by age.

Comparison of sequentially measured Aloka echo-tracking one-point pulse wave velocity with SphygmoCor carotid–femoral pulse wave velocity

Olga Vriz[1], Caterina Driussi[2], Salvatore La Carrubba[3], Vitantonio Di Bello[4], Concetta Zito[5], Scipione Carerj[5] and Francesco Antonini-Canterin[6]

SAGE Open Medicine
1: 2050312113507563

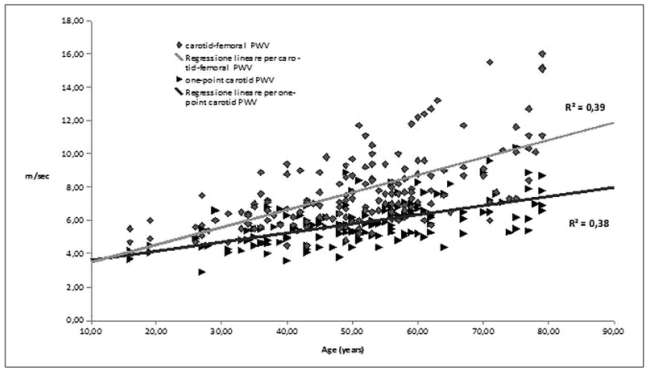


Figure 3. Relationship between carotid–femoral PWV and "one-point" carotid PWV with age.
PWV: pulse wave velocity.

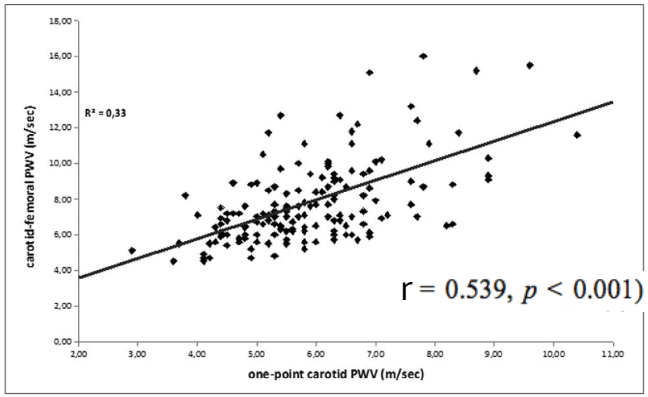


Figure 4. Relationship between carotid–femoral PWV and "one-point" carotid PWV obtained using SphygmoCor and echo-tracking system.
PWV: pulse wave velocity.

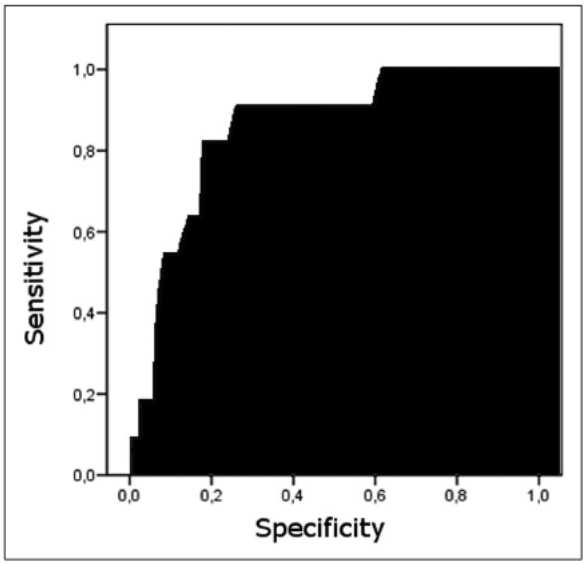


Figure 5. Receiver operating characteristic (ROC) curve analysis of "one-point" carotid PWV of 6.65 m/s as the best predictor of carotid–femoral PWV more than 12 m/s (sensitivity = 0.818, specificity = 0.819). Area under the curve = 0.85.
PWV: pulse wave velocity.

Ospedale San Antonio - San Daniele del Friuli

Interplay between arterial stiffness and diastolic function: a marker of ventricular-vascular coupling

Concetta Zito[a], Moemen Mohammed[a], Maria Chiara Todaro[a], Bijoy K. Khandheria[b], Maurizio Cusmà-Piccione[a], Giuseppe Oreto[a], Pietro Pugliatti[a], Mohamed Abusalima[c], Francesco Antonini-Canterin[d], Olga Vriz[e] and Scipione Carerj[a]

Aims We evaluated the interplay between left ventricular diastolic function and large-artery stiffness in asymptomatic patients at increased risk of heart failure and no structural heart disease (Stage A).

Methods We divided 127 consecutive patients (mean age 49 ± 17 years) with risk factors for heart failure who were referred to our laboratory to rule out structural heart disease into two groups according to presence (Group 1, $n = 35$) or absence (Group 2, $n = 92$) of grade I left ventricular diastolic dysfunction. Doppler imaging with high-resolution echo-tracking software was used to measure intima-media thickness (IMT) and stiffness of carotid arteries.

Results Group 1 had significantly higher mean age, blood pressure, left ventricular mass index, carotid IMT and arterial stiffness than Group 2 ($P<0.05$). Overall, carotid stiffness indices (β-stiffness index, augmentation index and elastic modulus) and 'one-point' pulse wave velocity each showed inverse correlation with E-wave velocity, E′ velocity and E/A ratio, and direct correlation with A-wave velocity, E-wave deceleration time and E/E′ ratio ($P<0.05$). Arterial compliance showed negative correlations with the echocardiographic indices of left ventricular diastolic function ($P<0.05$). On logistic regression analysis, age, hypertension, SBP, pulse pressure, left ventricular mass index, carotid IMT and stiffness parameters were associated with grade I left ventricular diastolic dysfunction ($P<0.05$ for each). However, on multivariate logistic analysis, only 'one-point' pulse wave velocity and age were independent predictors ($P = 0.038$ and $P = 0.016$, respectively).

Conclusion An independent association between grade I left ventricular diastolic dysfunction and increased arterial stiffness is demonstrated at the earliest stage of heart failure. Hence, assessment of vascular function, beyond cardiac function, should be included in a comprehensive clinical evaluation of these patients.

J Cardiovasc Med 2014, 15:000–000

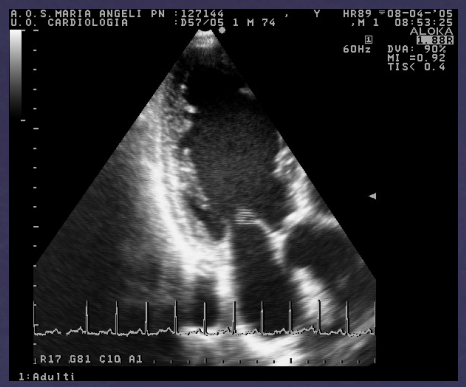
PWV: gr 1 8 m/s, gr 2 6 m/s

FIC Federazione Italiana di Cardiologia
Italian Federation of Cardiology

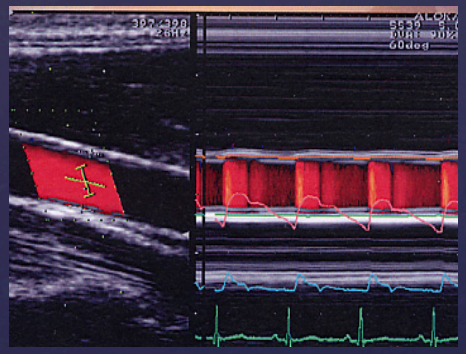
Conclusion 1

- ✓ The presence of subclinical arterosclerosis (increse C-IMT, increse carotid stiffness) is a disease itselfe over an additional predictive value.
- ✓ C-IMT and arterial stiffness have incremental value on risk assessment over FRS especially among intermediate risk cohort
- ✓ C-IMT measurements need to be better standarized to be constant and rialable in every-day clinical practice

Conclusion 2
Clinical application of arterial stiffness in cardiac disease assessment



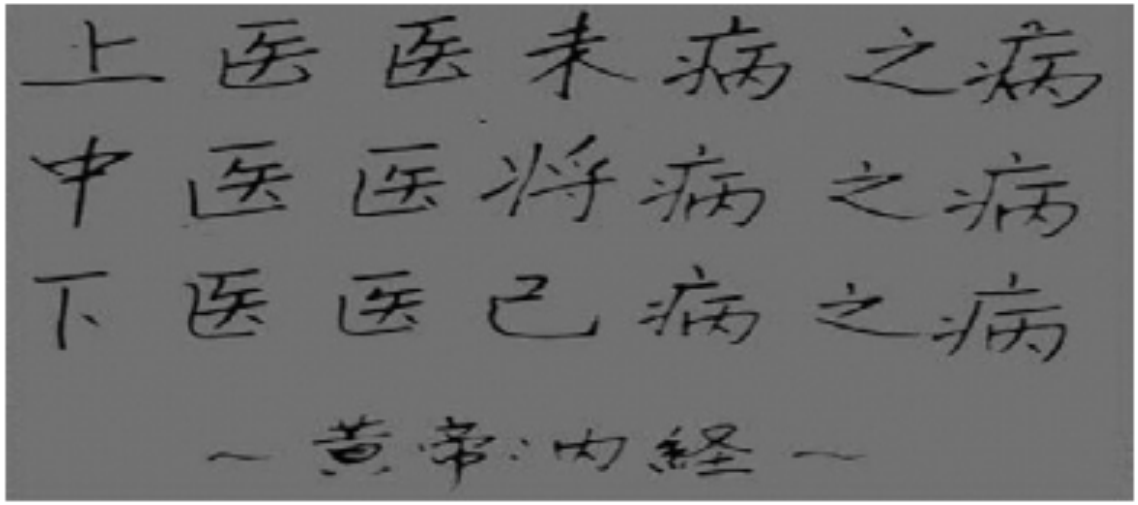
Cardiac anatomy and function



Arterial anatomy and function

1 machine

2 information



Huang Dee: Nai-Ching (2600 BC, First Medical Text)

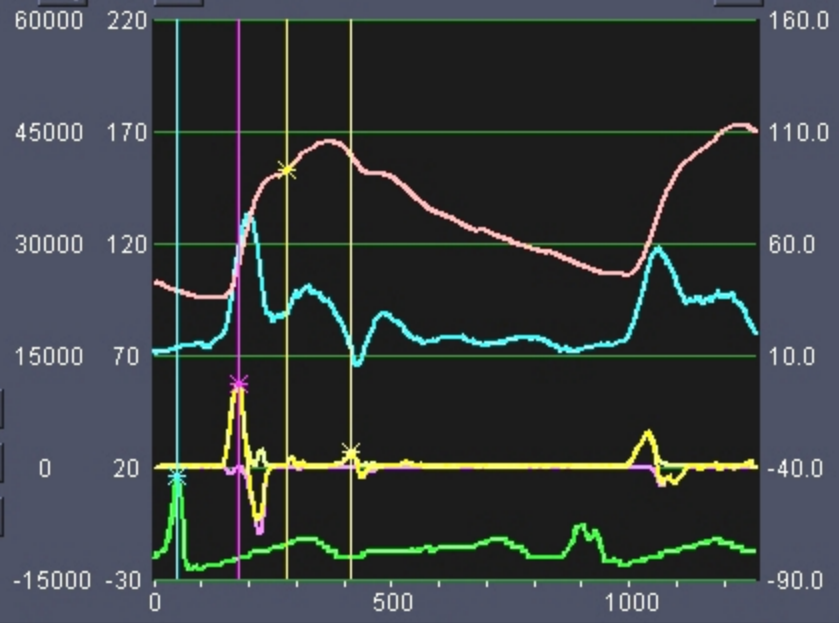
Translation: superior doctors prevent the disease; mediocre doctors treat the disease before evident; inferior doctors treat the full-blown disease.

Grazie

OBJA20011976M :39Y 20-01-1976 :H 180.0cm :BSA 2.06㎡ :15-04-2015
OBREGON-MONTANO JACKSON :Male :W 86.00kg : 13:10:10

Save

WI P Rt- U



- [x] WI
- [x] forward
- [x] backward
- [x] P
- [x] U

Peak Detection
50 [ms]

W1 Auto Peak
W2 Auto Peak
AI Reset
ECG

Wave Intensity

Peak(mmHg m/s³)

1st(W1)	Wd1
11.0*10³	10.35m/s³
2nd(W2)	Wd2
2.0*10³	1.79m/s³

Negative Area(mmHg m/s²)

NA	NAd
192.55	0.175m/s²

Time

R-1st	1st-2nd
128ms	237ms

Arterial Stiffness

β	Ep	AC	AI
8.3	140kPa	0.53㎟/kPa	17.9%
PWVβ	PWV_WI		
7.1m/s	8.66m/s		

Diameter

D_max	D_min	diffD
7.22mm	6.77mm	0.45mm

Pressure

P_max	P_min	HR
165mmHg	95mmHg	69BPM

Flow Velocity

U_max	U_min	Vrange	Angle	SampleV	EnAvg
73.2cm/s	4.7cm/s	46.9cm/s	60deg	3.5mm	12

Prev. Exit Cancel

Carotid intima media thickness and coronary atherosclerosis linkage in symptomatic intermediate risk patients evaluated by coronary computed tomography angiography☆

Andrea Igoren Guaricci [a,*], Teresa Arcadi [b], Natale Daniele Brunetti [a], Erica Maffei [b], Deodata Montrone [a], Chiara Martini [a], Maria De Luca [a], Fiorella De Rosa [a], Domenico Cocco [a], Massimo Midiri [c], Filippo Cademartiri [b,d], Luca Macarini [e], Matteo Di Biase [a], Gianluca Pontone [f]

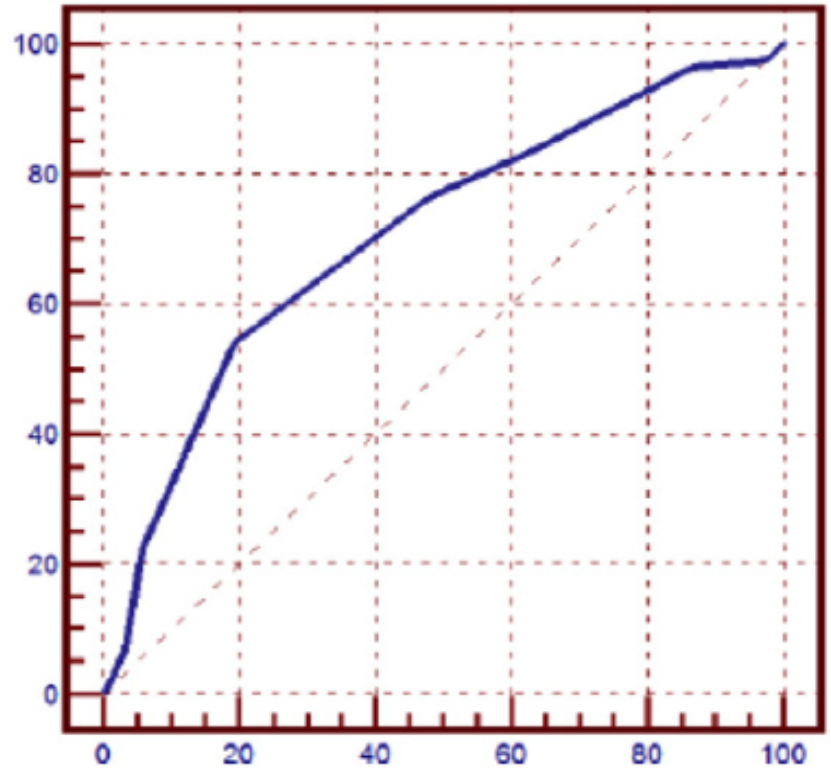


Fig. 4. Predictive power of Carotid Intima Media Thickness (CIMT) — Receiver operating curve (ROC) of predictive model of CIMT for occurrence of combined endpoint of presence of coronary artery plaques determining CAD ≥ 50% and/or mixed plaques and/or remodeled plaques showed the best predicting power with a cutoff value of >1.3 corresponding to a positive and negative predictive values of 100% and 69%, respectively.

Fig. 2. Carotid
(Panel B), nur
CAD: Coronar

San Daniele del Friuli

Carotid Intima-Media Thickness and Antihypertensive Treatment

A Meta-Analysis of Randomized Controlled Trials

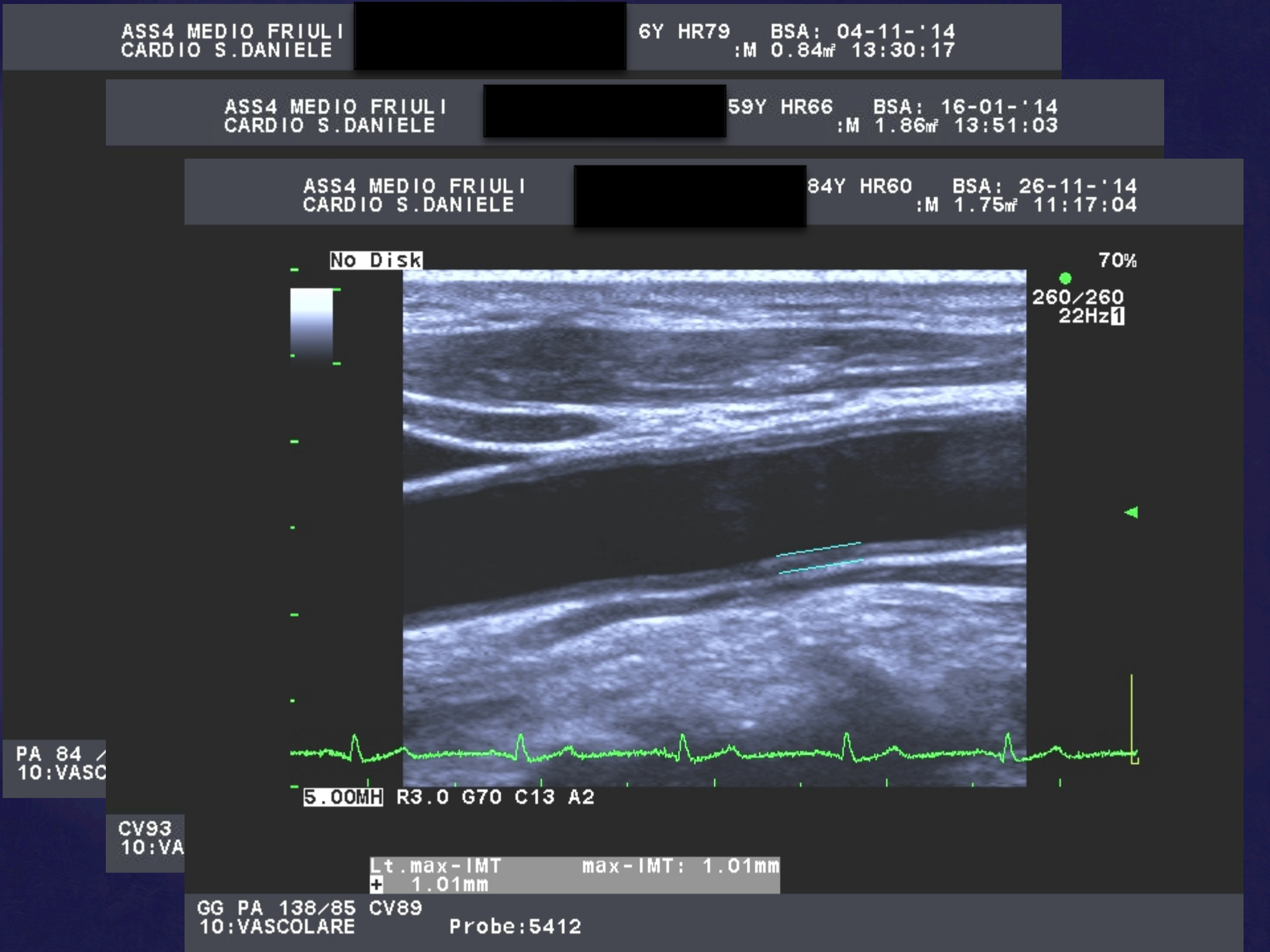
Ji-Guang Wang, MD, PhD; Jan A. Staessen, MD, PhD; Yan Li, MD, PhD;
Luc M. Van Bortel, MD, PhD; Tim Nawrot, PhD; Robert Fagard, MD, PhD;
Franz H. Messerli, MD; Michel Safar, MD

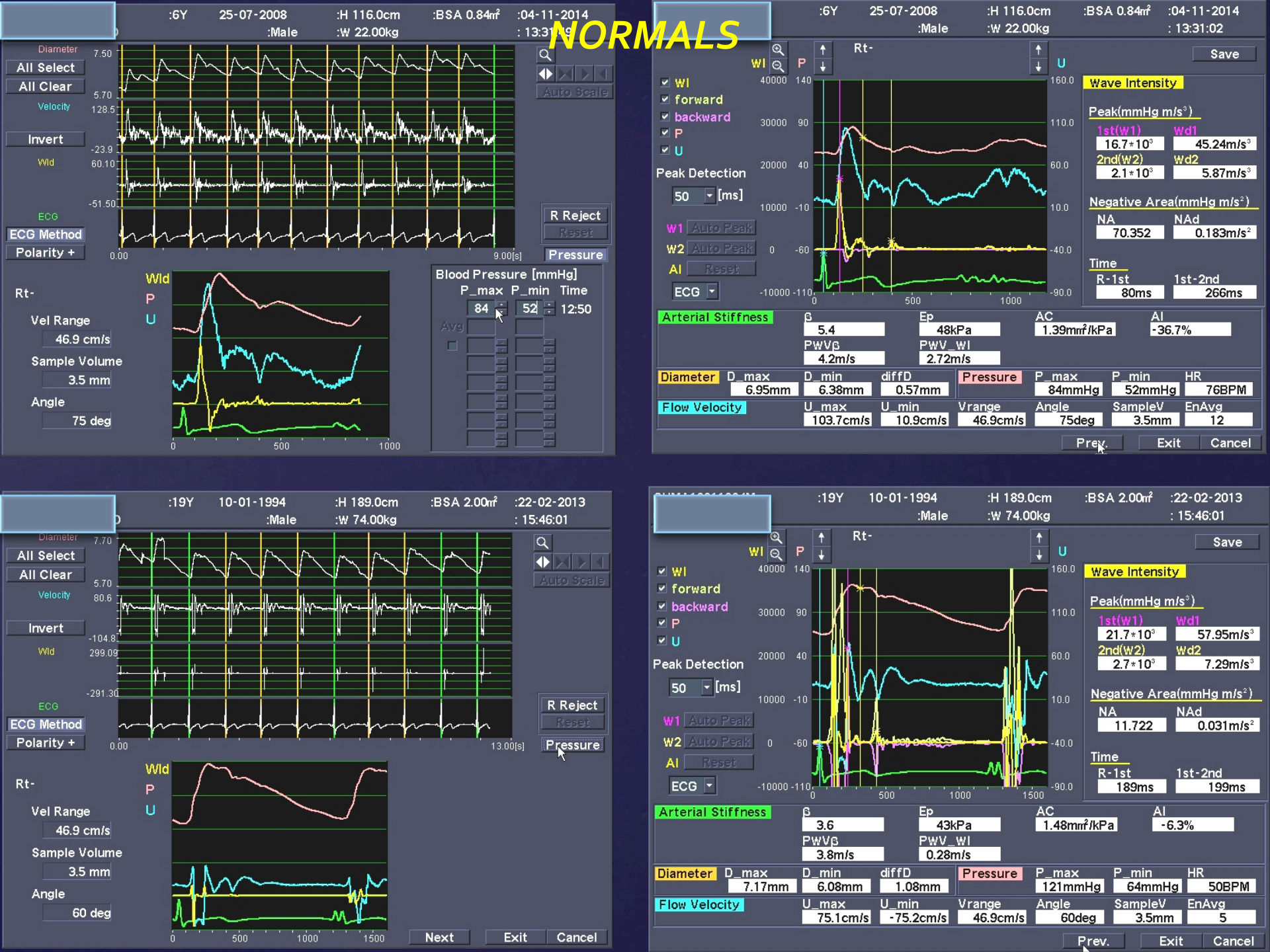
Background and Purpose—Hypertension promotes carotid intima-media thickening. We reviewed the randomized controlled trials that evaluated the effects of an antihypertensive drug versus placebo or another antihypertensive agent of a different class on carotid intima-media thickness.

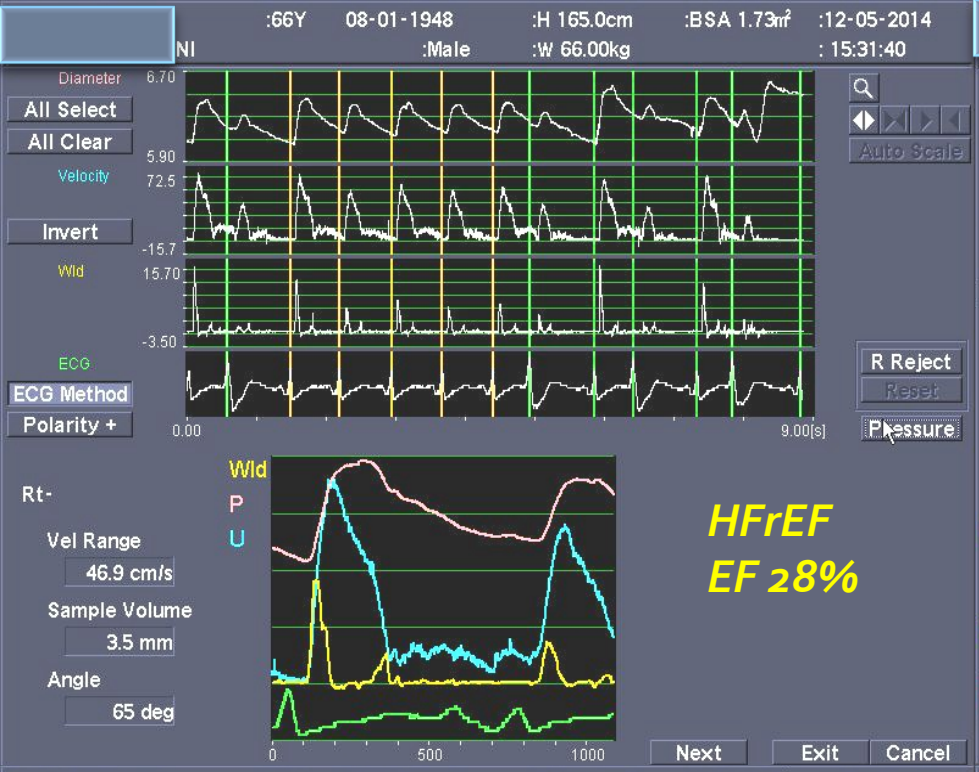
Methods—We searched the PubMed and the Web of Science databases for randomized clinical trials, published in English before 2005, and included 22 trials.

Results—In 8 trials including 3329 patients with diabetes or coronary heart disease, antihypertensive treatment initiated with an angiotensin-converting enzyme (ACE) inhibitor, a β-blocker, or a calcium-channel blocker (CCB), compared with placebo or no-treatment, reduced the rate of intima-media thickening by 7 μm/year (P=0.01). In 9 trials including 4564 hypertensive patients, CCBs, ACE inhibitors, an angiotensin II receptor blocker or an α-blocker, compared with diuretics or β-blockers, in the presence of similar blood pressure reductions, decreased intima-media thickening by 3 μm/year (P=0.03). The overall beneficial effect of the newer over older drugs was largely attributable to the decrease of intima-media thickening by 5 μm/year (P=0.007) in 4 trials of CCBs involving 3619 patients. In 5 trials including 287 patients with hypertension or diabetes, CCBs compared with ACE inhibitors did not differentially affect blood pressure, but attenuated intima-media thickening by 23 μm/year (P=0.02). The treatment induced changes in carotid intima-media thickness correlated with the changes in lumen diameter (P=0.02), but not with the differences in achieved blood pressure (P>0.53).

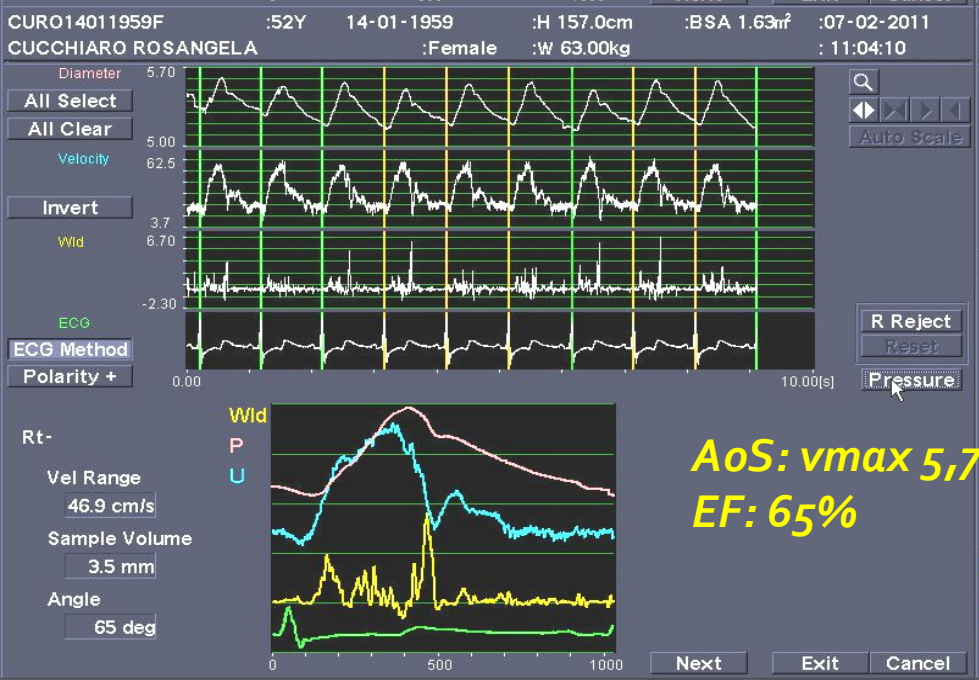
Conclusions—CCBs reduce carotid intima-media thickening. This mechanism might contribute to their superior protection against stroke. (***Stroke*. 2006;37:1933-1940.)**

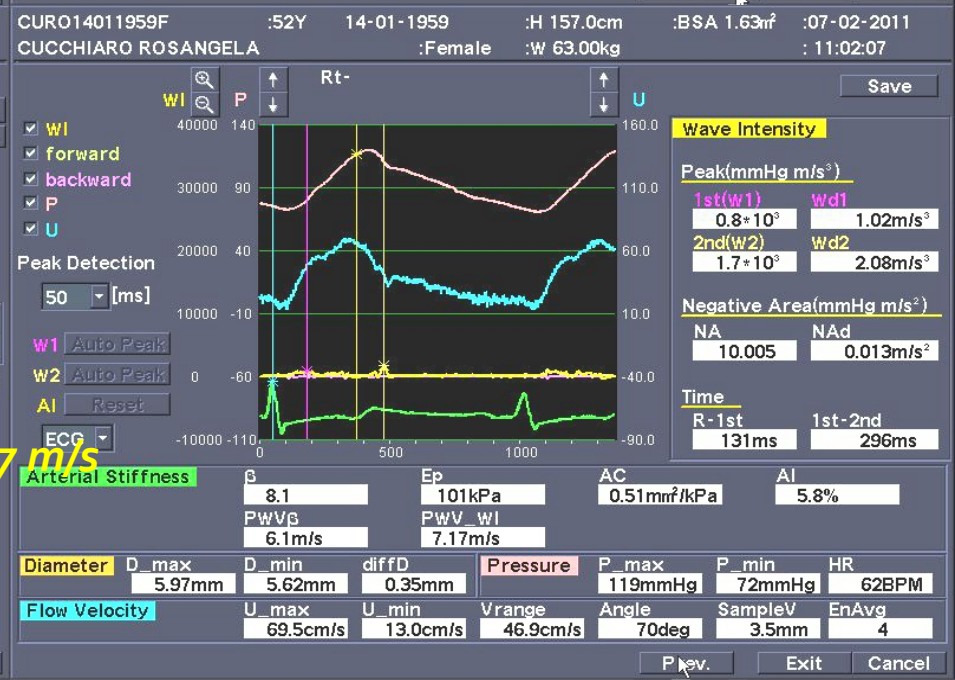

ASS4 MEDIO FRIULI
CARDIO S.DANIELE
[redacted]
6Y HR79
:M
BSA: 04-11-'14
0.84m² 13:30:17
ASS4 MEDIO FRIULI
CARDIO S.DANIELE
[redacted]
59Y HR66
:M
BSA: 16-01-'14
1.86m² 13:51:03
ASS4 MEDIO FRIULI
CARDIO S.DANIELE
[redacted]
84Y HR60
:M
BSA: 26-11-'14
1.75m² 11:17:04
No Disk
70%
260/260
22Hz 1
5.00MH R3.0 G70 C13 A2
PA 84 /
10:VASC
CV93
10:VA
Lt.max-IMT
+ 1.01mm
max-IMT: 1.01mm
GG PA 138/85 CV89
10:VASCOLARE
Probe:5412


NORMALS
:6Y 25-07-2008 :H 116.0cm :BSA 0.84㎡ :04-11-2014
:Male :W 22.00kg : 13:31:02
Diameter
All Select
All Clear
Velocity
Invert
Wld
ECG
ECG Method
Polarity +
7.50
5.70
128.5
-23.9
60.10
-51.50
0.00
9.00[s]
Auto Scale
R Reject
Reset
Pressure
Rt-
Vel Range
46.9 cm/s
Sample Volume
3.5 mm
Angle
75 deg
Wld
P
U
0
500
1000
Blood Pressure [mmHg]
P_max P_min Time
84 52 12:50
Avg
Rt-
WI
P
U
WI
forward
backward
P
U
Peak Detection
50 [ms]
W1 Auto Peak
W2 Auto Peak
AI Reset
ECG
40000 140
30000 90
20000 40
10000 -10
0 -60
-10000 -110
160.0
110.0
60.0
10.0
-40.0
-90.0
0
500
1000
Save
Wave Intensity
Peak(mmHg m/s³)
1st(W1) Wd1
16.7*10³ 45.24m/s³
2nd(W2) Wd2
2.1*10³ 5.87m/s³
Negative Area(mmHg m/s²)
NA NAd
70.352 0.183m/s²
Time
R-1st 1st-2nd
80ms 266ms
Arterial Stiffness
β Ep AC AI
5.4 48kPa 1.39㎟/kPa -36.7%
PWVβ PWV_WI
4.2m/s 2.72m/s
Diameter D_max D_min diffD
6.95mm 6.38mm 0.57mm
Pressure P_max P_min HR
84mmHg 52mmHg 76BPM
Flow Velocity U_max U_min Vrange Angle SampleV EnAvg
103.7cm/s 10.9cm/s 46.9cm/s 75deg 3.5mm 12
Prev. Exit Cancel
:19Y 10-01-1994 :H 189.0cm :BSA 2.00㎡ :22-02-2013
:Male :W 74.00kg : 15:46:01
Diameter
All Select
All Clear
Velocity
Invert
Wld
ECG
ECG Method
Polarity +
7.70
5.70
80.6
-104.8
299.09
-291.30
0.00
13.00[s]
Auto Scale
R Reject
Reset
Pressure
Rt-
Vel Range
46.9 cm/s
Sample Volume
3.5 mm
Angle
60 deg
Wld
P
U
0
500
1000
1500
Next Exit Cancel
Rt-
WI
P
U
WI
forward
backward
P
U
Peak Detection
50 [ms]
W1 Auto Peak
W2 Auto Peak
AI Reset
ECG
40000 140
30000 90
20000 40
10000 -10
0 -60
-10000 -110
160.0
110.0
60.0
10.0
-40.0
-90.0
0
500
1000
1500
Save
Wave Intensity
Peak(mmHg m/s³)
1st(W1) Wd1
21.7*10³ 57.95m/s³
2nd(W2) Wd2
2.7*10³ 7.29m/s³
Negative Area(mmHg m/s²)
NA NAd
11.722 0.031m/s²
Time
R-1st 1st-2nd
189ms 199ms
Arterial Stiffness
β Ep AC AI
3.6 43kPa 1.48㎟/kPa -6.3%
PWVβ PWV_WI
3.8m/s 0.28m/s
Diameter D_max D_min diffD
7.17mm 6.08mm 1.08mm
Pressure P_max P_min HR
121mmHg 64mmHg 50BPM
Flow Velocity U_max U_min Vrange Angle SampleV EnAvg
75.1cm/s -75.2cm/s 46.9cm/s 60deg 3.5mm 5
Prev. Exit Cancel



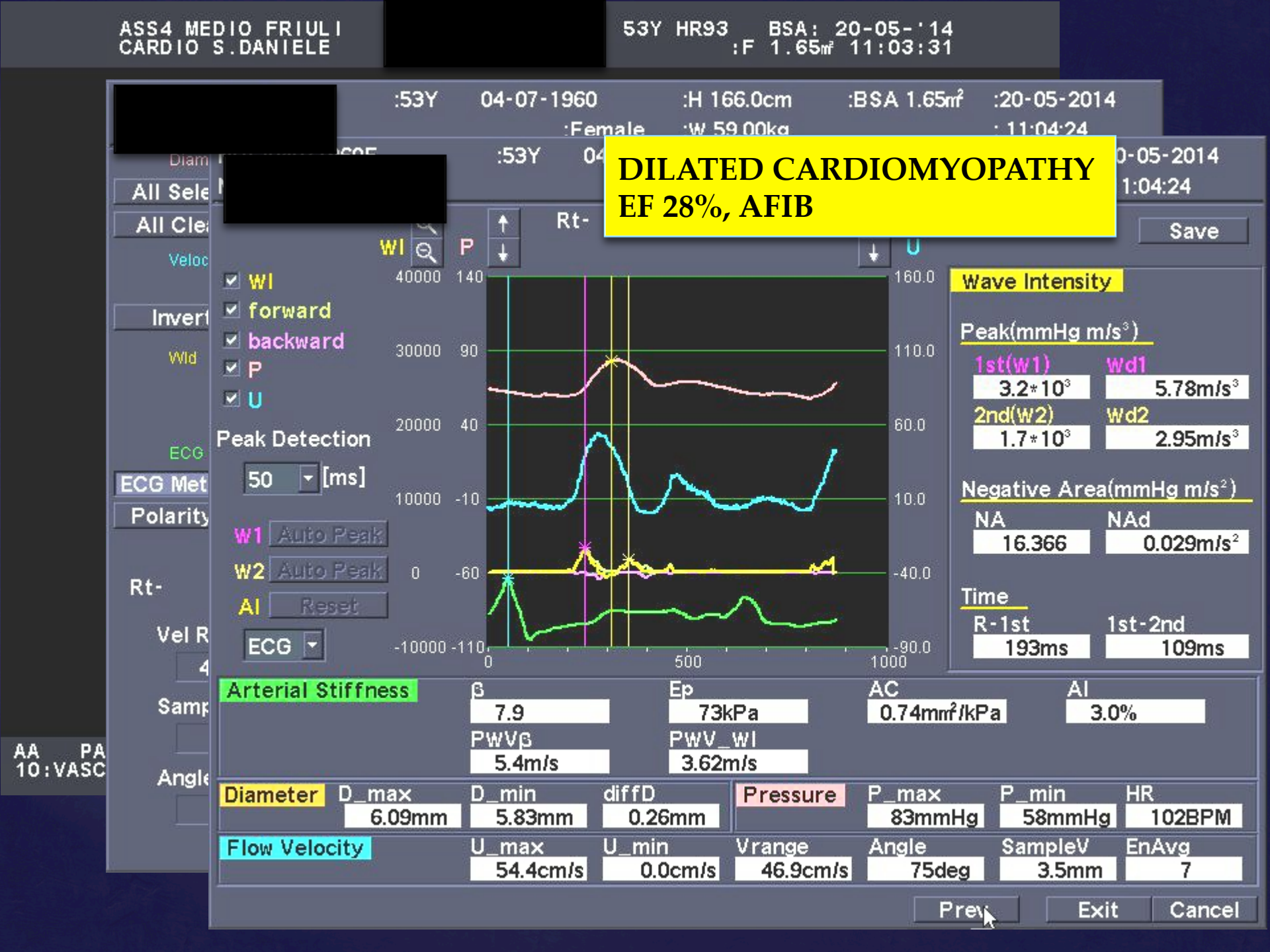
HFrEF
EF 28%





AoS: vmax 5,7 m/s
EF: 65%


ASS4 MEDIO FRIULI
CARDIO S.DANIELE
67Y HR77
BSA: 24-10-'13
:M 2.04㎡ 11:40:49
:67Y 09-02-1946 :H 168.0cm :BSA 2.04㎡ :24-10-2013
:Male :W 95.00kg : 11:41:41
Diameter 9.20
AMILOIDOSIS
Save
WI
P
Rt-
WI
forward
backward
P
U
Peak Detection
50 [ms]
W1 Auto Peak
W2 Auto Peak
AI Reset
ECG
40000 140
30000 90
20000 40
10000 -10
0 -60
-10000 -110
0 500 1000
110.0
60.0
10.0
-40.0
-90.0
Wave Intensity
Peak(mmHg m/s³)
1st(W1) 17.3*10³
Wd1 17.70m/s³
2nd(W2) 0.7*10³
Wd2 0.71m/s³
Negative Area(mmHg m/s²)
NA 15.376
NAd 0.015m/s²
Time
R-1st 88ms
1st-2nd 241ms
Arterial Stiffness
β 9.6
Ep 128kPa
AC 0.90mm²/kPa
AI 3.0%
PWVβ 7.0m/s
PWV_WI 7.11m/s
Diameter
D_max 8.88mm
D_min 8.49mm
diffD 0.40mm
Pressure
P_max 124mmHg
P_min 79mmHg
HR 80BPM
Flow Velocity
U_max 43.9cm/s
U_min -0.6cm/s
Vrange 46.9cm/s
Angle 70deg
SampleV 3.5mm
EnAvg 8
Prev.
Exit
Cancel
PA 86/49
10:VASCOLA


ASS4 MEDIO FRIULI
CARDIO S.DANIELE
53Y HR93 BSA: 20-05-'14
:F 1.65㎡ 11:03:31
:53Y 04-07-1960 :H 166.0cm :BSA 1.65㎡ :20-05-2014
:Female :W 59.00kg :11:04:24
DILATED CARDIOMYOPATHY
EF 28%, AFIB
Save
WI
P
U
Rt-
WI
forward
backward
P
U
Peak Detection
50 [ms]
W1 Auto Peak
W2 Auto Peak
AI Reset
ECG
40000 140
30000 90
20000 40
10000 -10
0 -60
-10000 -110
160.0
110.0
60.0
10.0
-40.0
-90.0
0
500
1000
Wave Intensity
Peak(mmHg m/s³)
1st(W1) 3.2*10³
Wd1 5.78m/s³
2nd(W2) 1.7*10³
Wd2 2.95m/s³
Negative Area(mmHg m/s²)
NA 16.366
NAd 0.029m/s²
Time
R-1st 193ms
1st-2nd 109ms
Arterial Stiffness
β 7.9
Ep 73kPa
AC 0.74mm²/kPa
AI 3.0%
PWVβ 5.4m/s
PWV_WI 3.62m/s
Diameter
D_max 6.09mm
D_min 5.83mm
diffD 0.26mm
Pressure
P_max 83mmHg
P_min 58mmHg
HR 102BPM
Flow Velocity
U_max 54.4cm/s
U_min 0.0cm/s
Vrange 46.9cm/s
Angle 75deg
SampleV 3.5mm
EnAvg 7
Prev
Exit
Cancel