Vascular Ultrasound:
Current state, current needs, future directions

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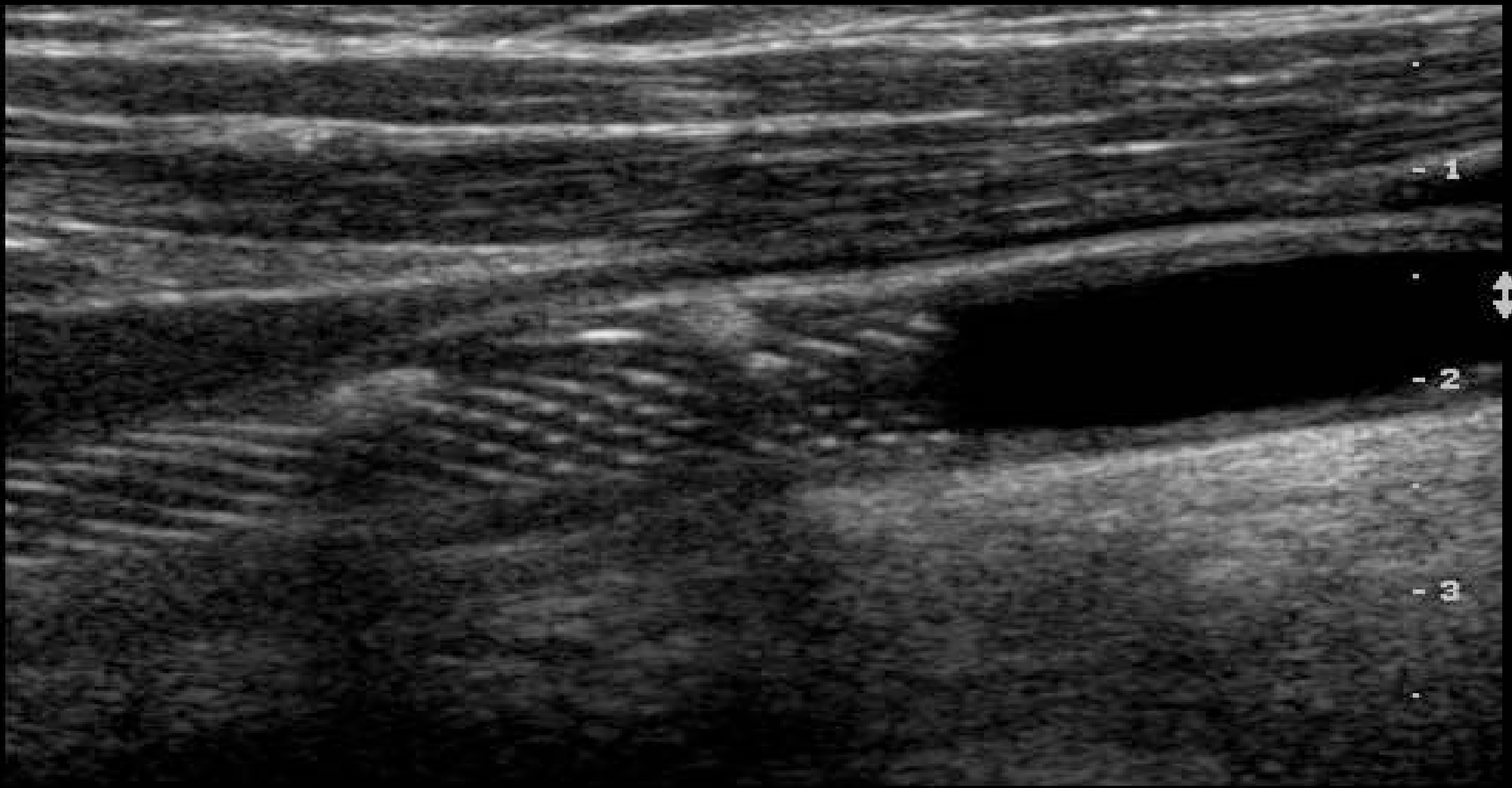
Disclosures

- Member, Intersocietal Accreditation Commission – Vascular Testing (unpaid)

Overview

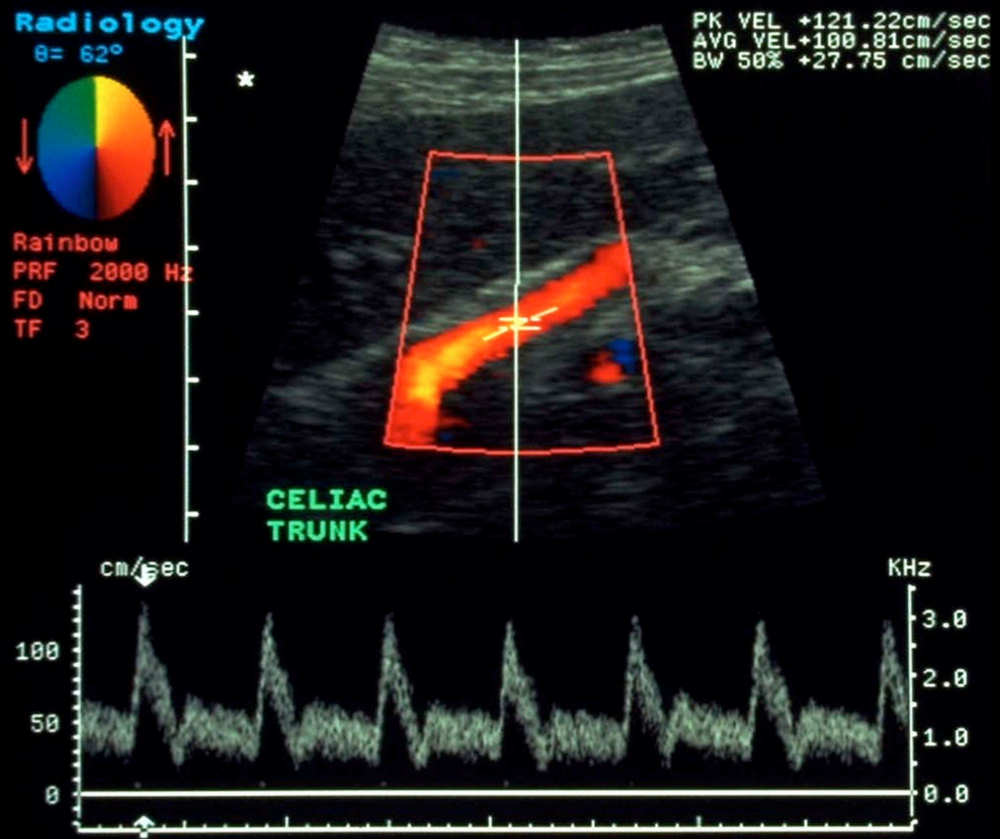
- What tests are done for vascular disease?
- What is the natural history of vascular disease using ultrasound?
- What shortcomings exist for US vascular diagnosis?
- What is the future direction of ultrasound? of vascular ultrasound?

Gray scale ultrasound
Carotid stent

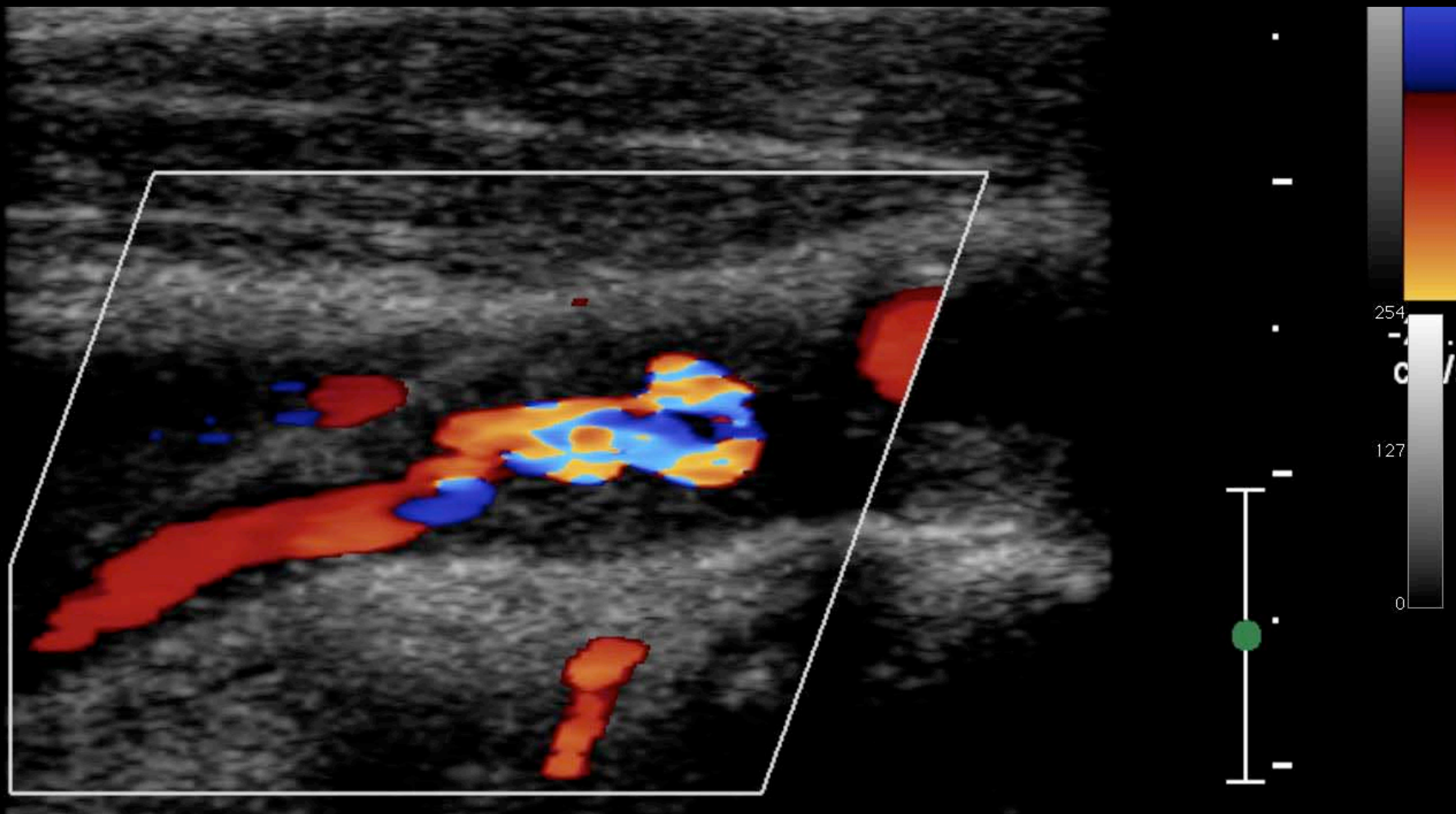


Spectral Doppler

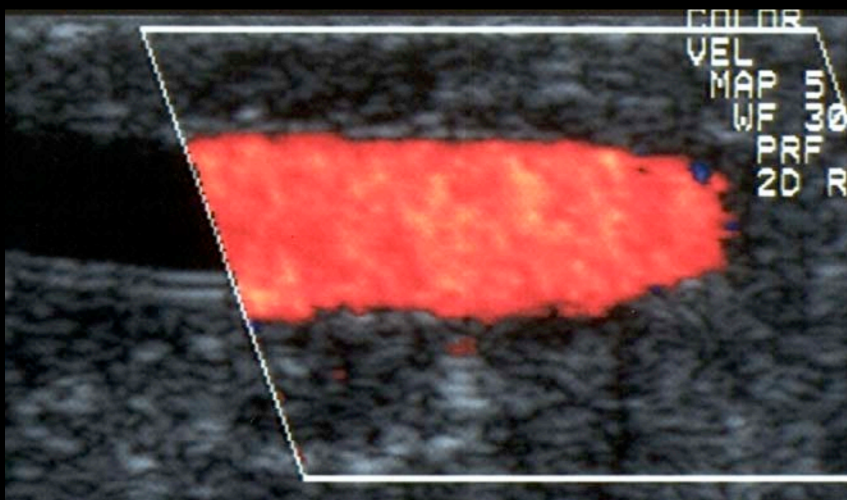
- Graphical representation of Doppler
- Makeup
  - Doppler frequency or velocity - y axis
  - Time - x axis
  - Strength of signal - gray scale
    - Number of reflectors

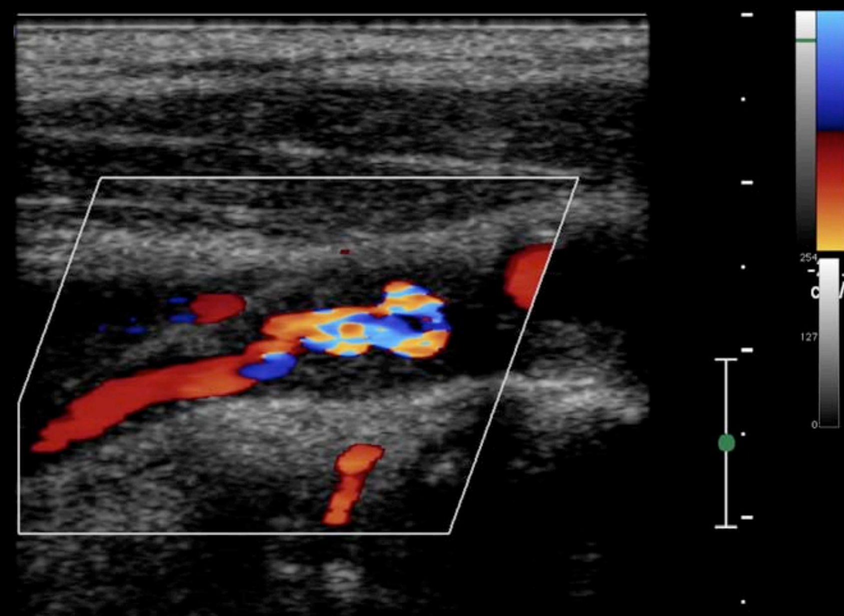


Color Doppler
Carotid stenosis



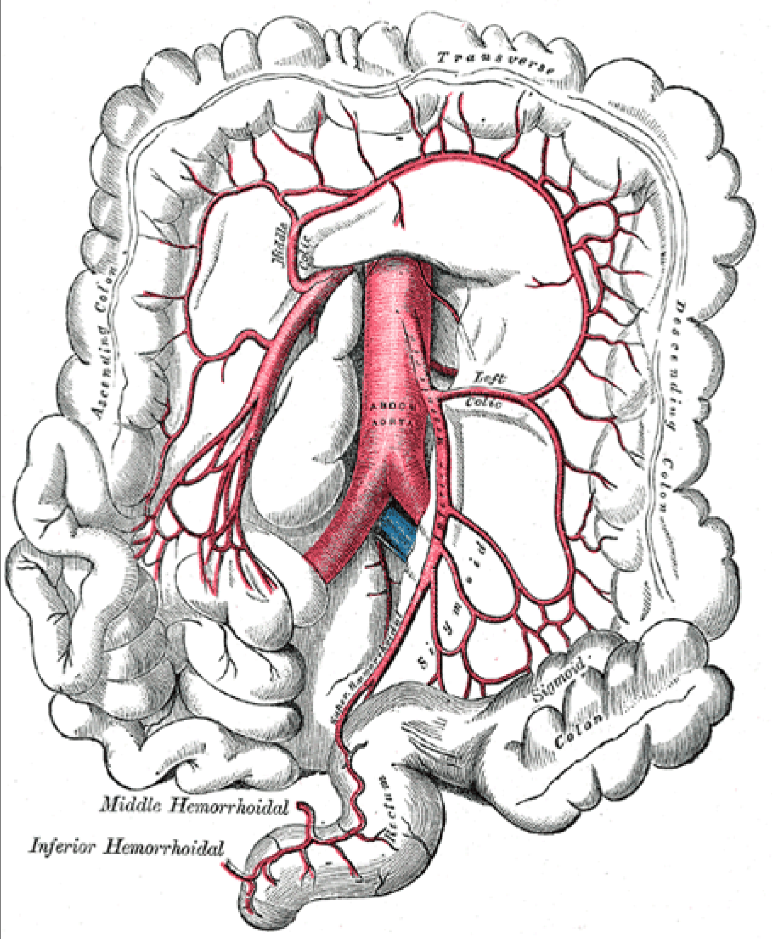
Color Doppler Artifacts

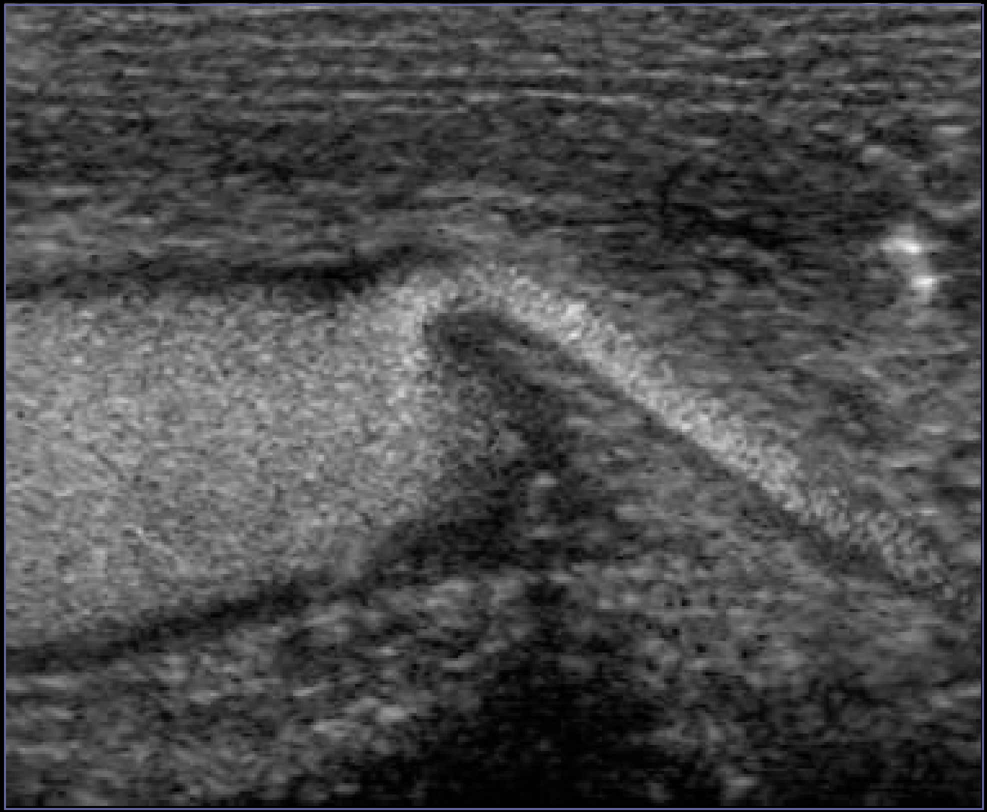




Vein of Galen "Aneurysm"

Inferior Mesenteric Artery





Current state

Tests

- Venous ultrasound – obstruction – DVT
- Carotid duplex ultrasound - stenosis
- Aortic ultrasound - aneurysm
- Abdominal Doppler
  - Renal arteries - stenosis
  - Liver (portal hypertension) – hypertension, flow direction
  - Ovaries and testes – increased or decreaed flow, tumors

Acting Surgeon General Issues 'Call to Action to Prevent Deep Vein Thrombosis and Pulmonary Embolism'

FOR IMMEDIATE RELEASE
Monday September 15, 2008

Contact: Office of Public Health and Science
(202) 205-0143

Acting Surgeon General Steven K. Galson, M.D., M.P.H., today issued a Call to Action to reduce the number of cases of deep vein thrombosis and pulmonary embolism in the United States.

Galson urged all Americans to learn about and prevent these treatable conditions.

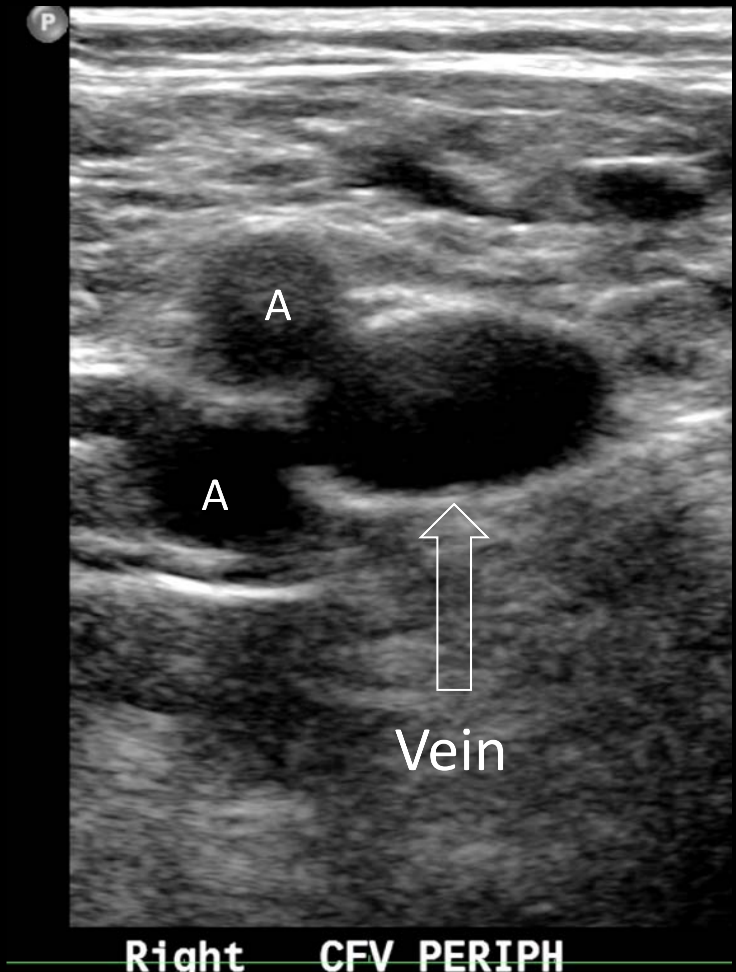
Deep vein thrombosis and pulmonary embolism affect an estimated 350,000 to 600,000 Americans each year, and the numbers are expected to increase as the U.S. population ages. Together, deep vein thrombosis and pulmonary embolism contribute to at least 100,000 deaths each

Venous thromboembolic disease

- Ultrasound is the gold standard to diagnose deep venous thrombosis in the legs
- CT and NM are the major tests to diagnose its major complication, pulmonary embolism

- DVT and PE are associated with mortality, diagnosis of cancer, and chronic diseases

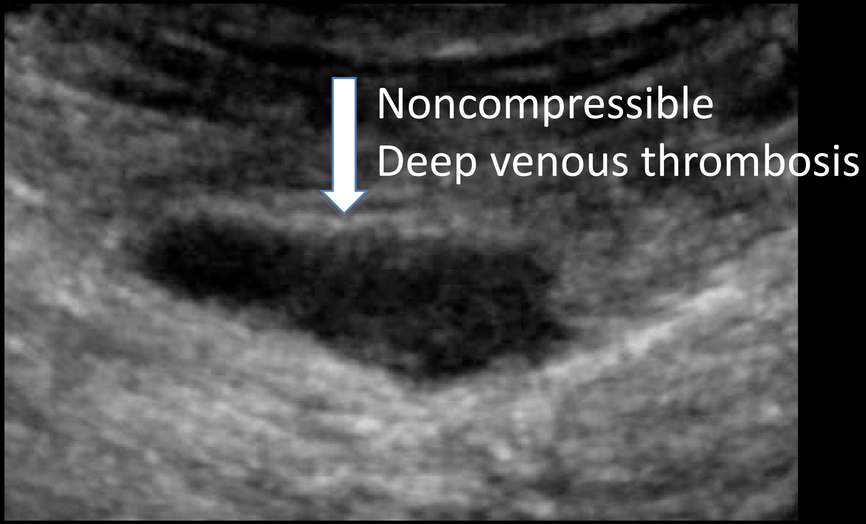
Venous US
Normal Compression



Noncompressible Vein: Causes

- Acute venous thrombosis (DVT)
- Scarring
- Inadequate compression

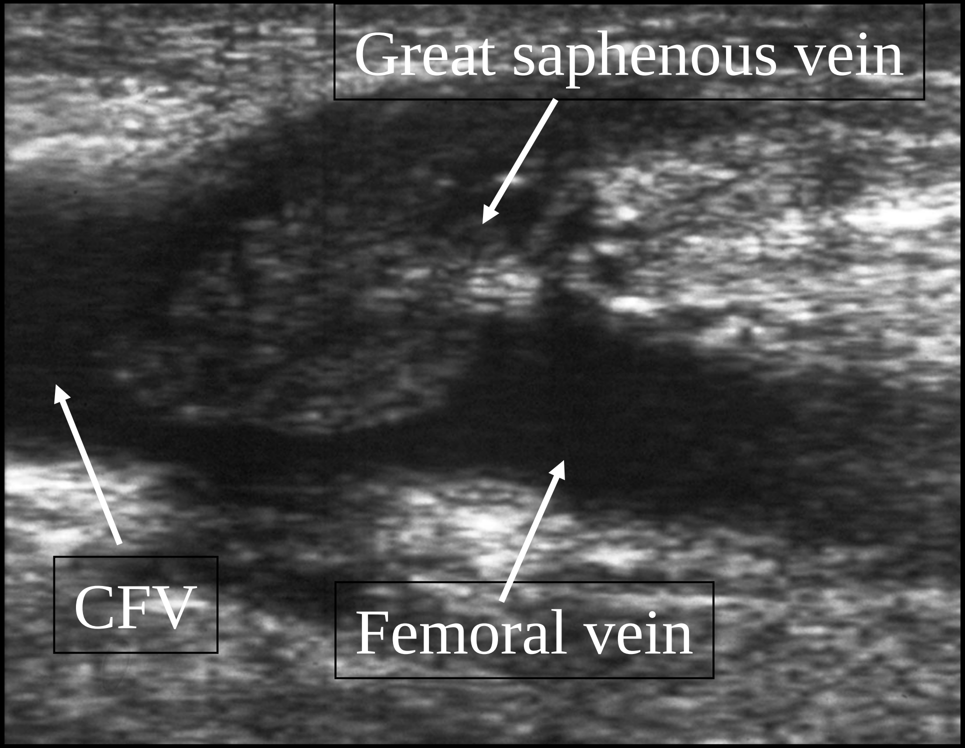
Without compression

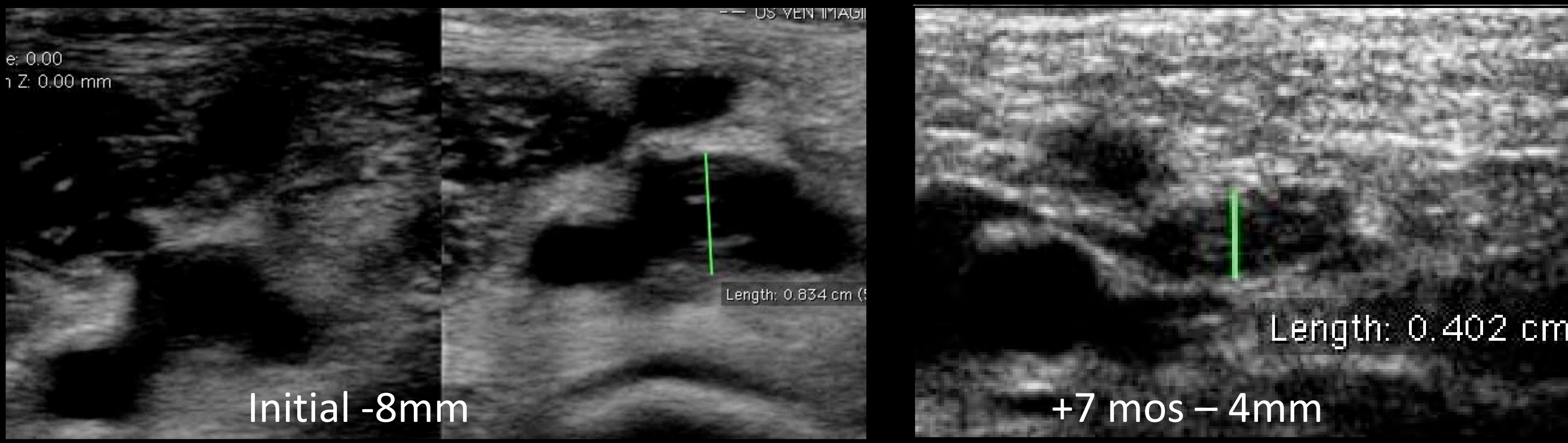


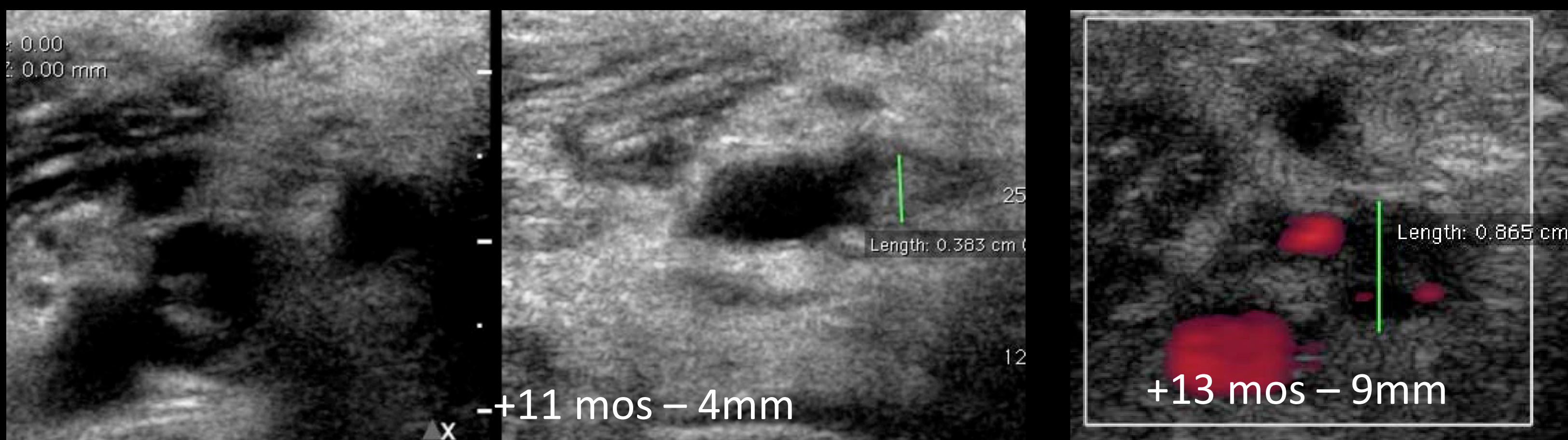
With compression

Acute Venous Thrombosis

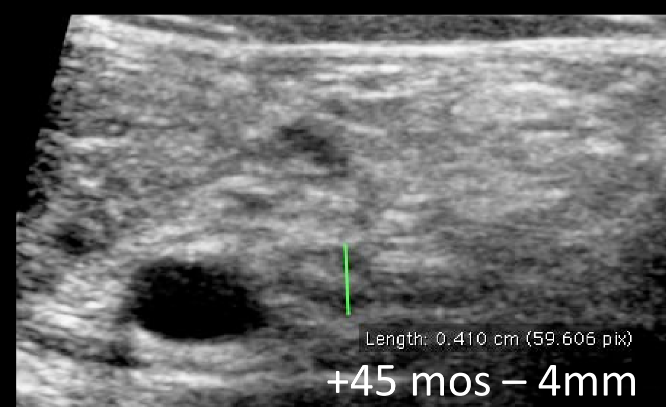
- Soft, deformable with compression
- Enlarges vein
- Smooth



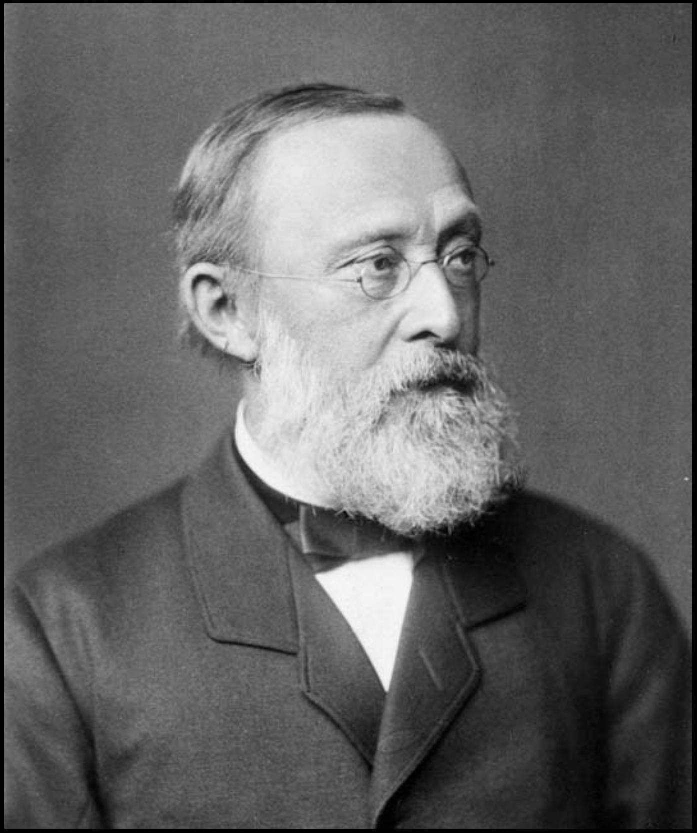


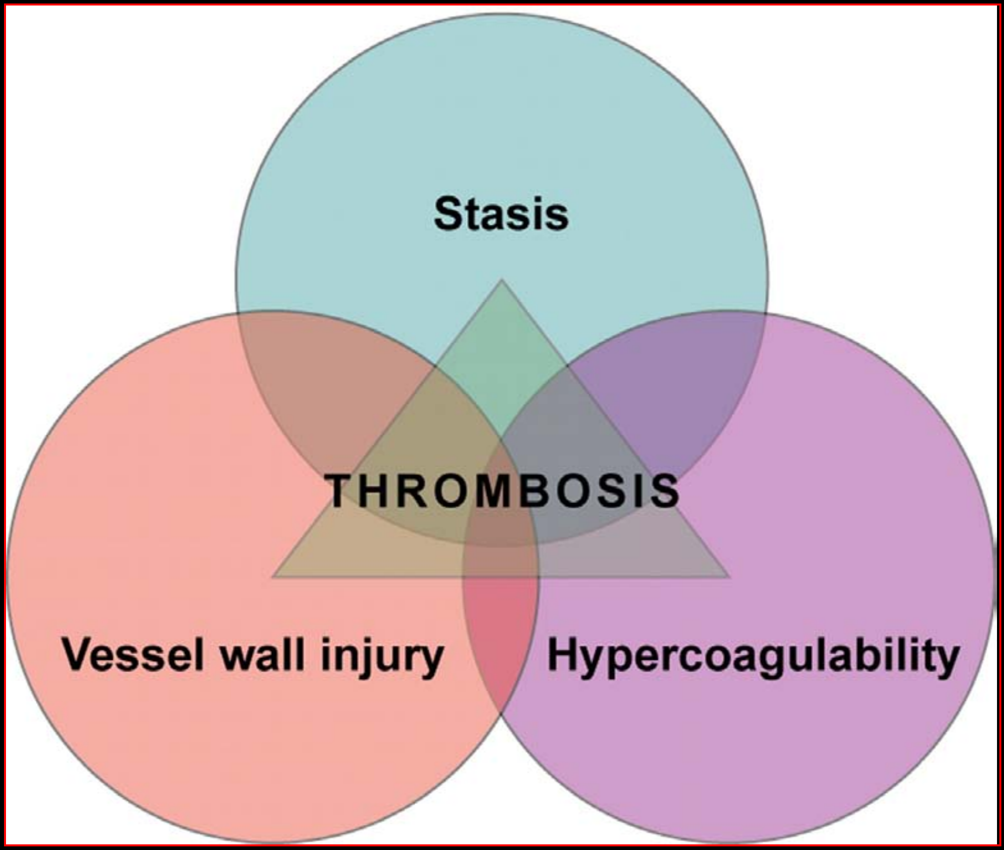


Popliteal vein recurrent thrombosis

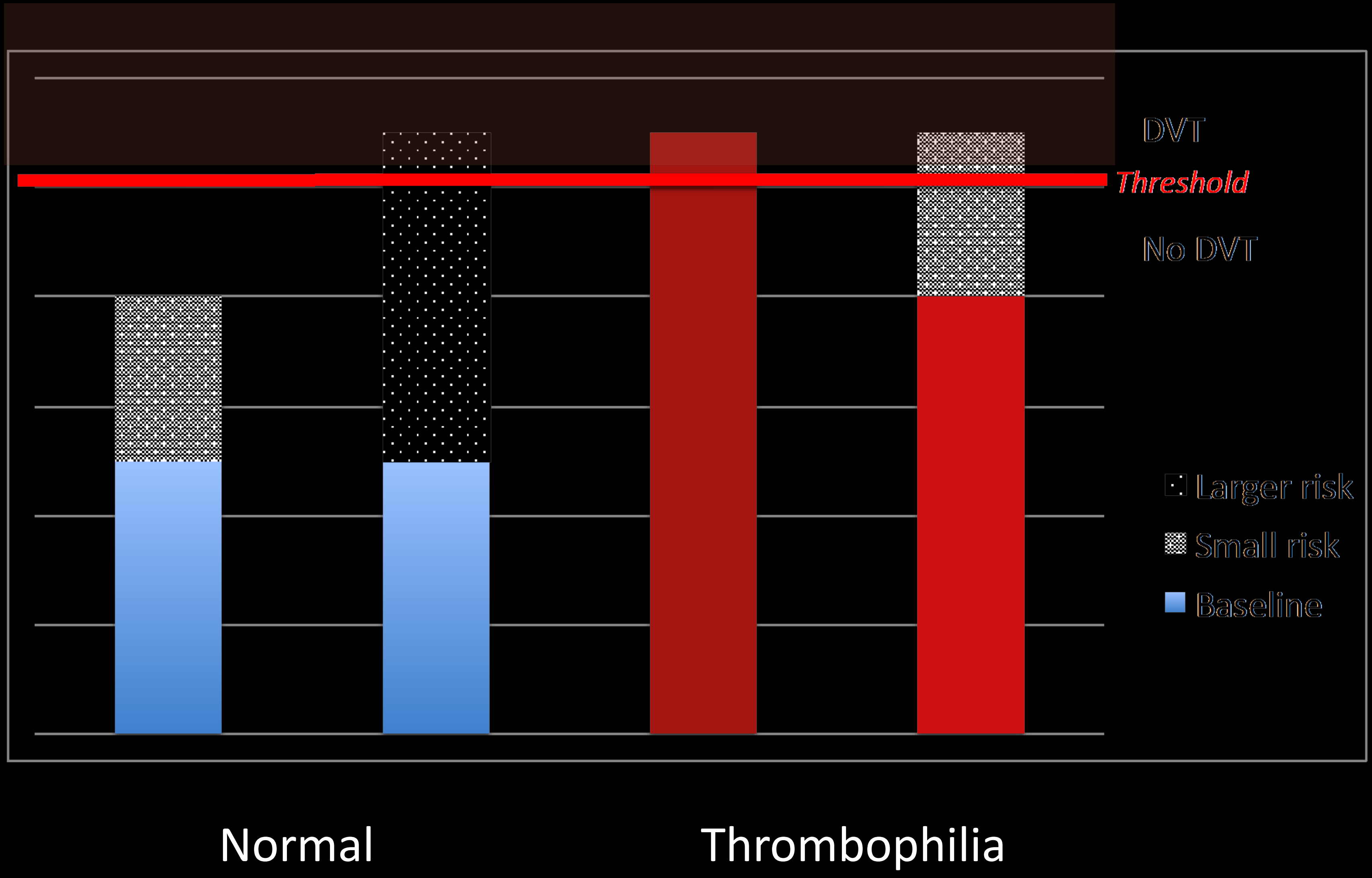


Virchow's Triad

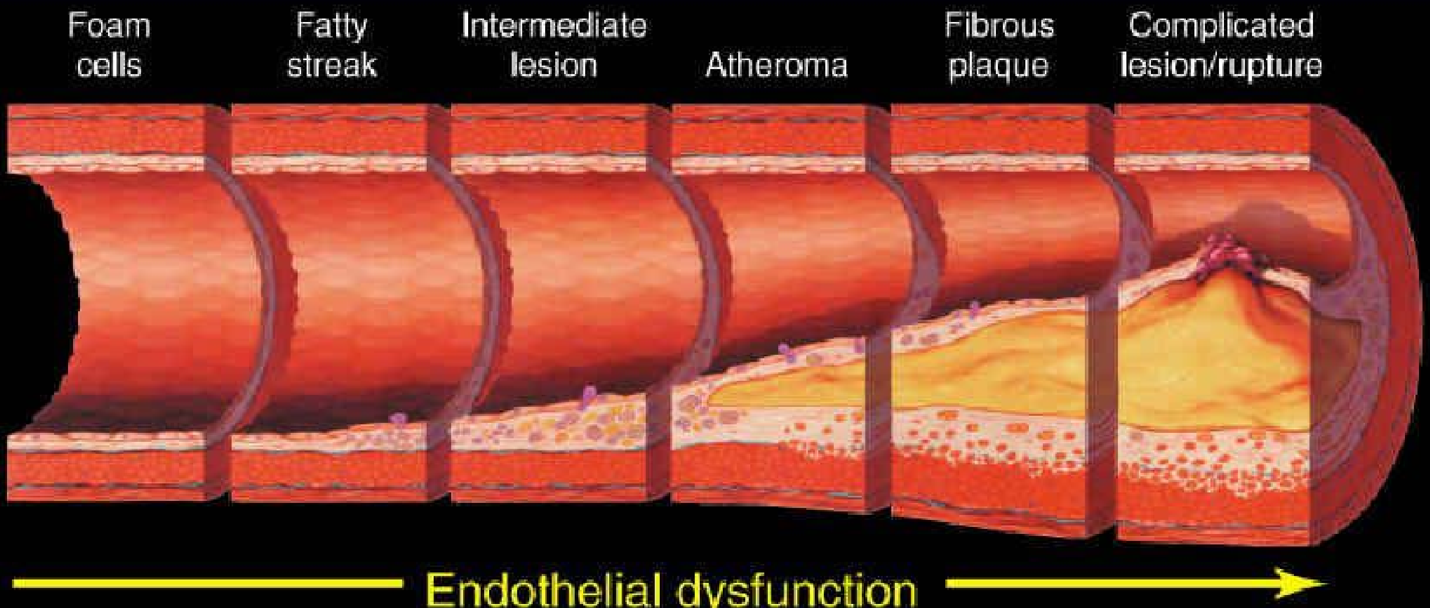




Development of DVT depends on baseline risk and risk events



Atherosclerosis timeline

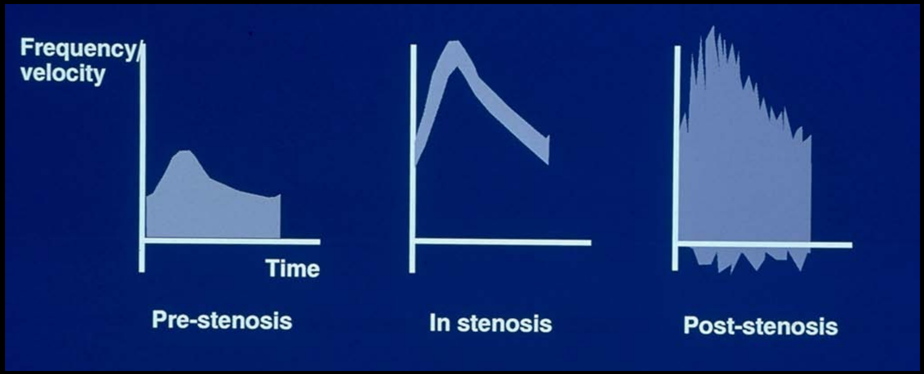


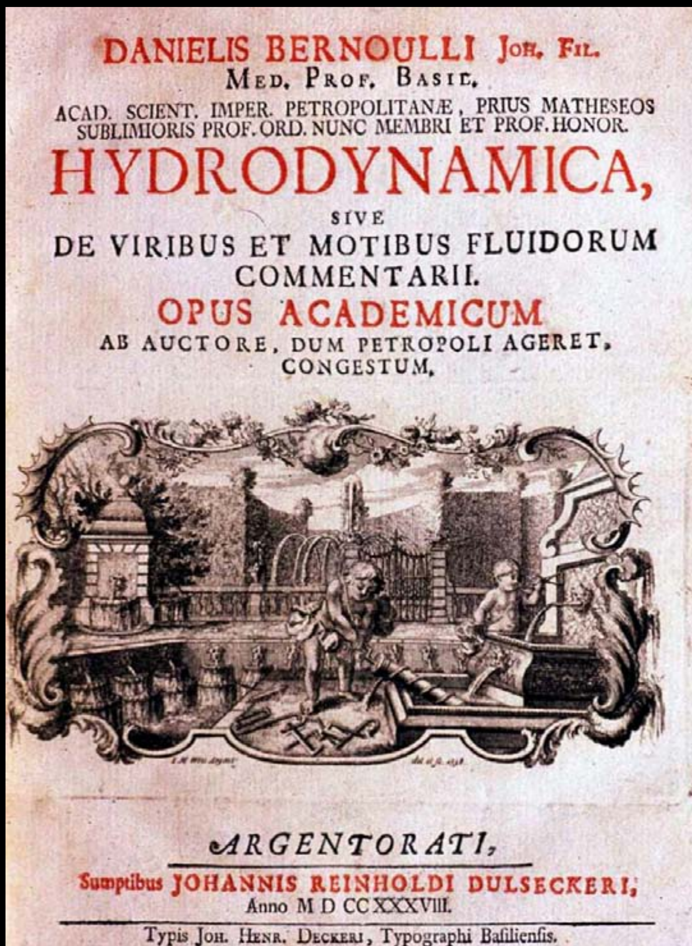
From first decade	From third decade	From fourth decade	
Growth mainly by lipid accumulation		Smooth muscle and collagen	Thrombosis, hematoma

Adapted from Pepine CJ. *Am J Cardiol.* 1998;82(suppl 104).

Duplex Doppler ultrasound is used to diagnose and grade stenoses

- Gray scale narrowing
- Color narrowing and color changes of elevated velocity
- Spectral Doppler in and beyond stenosis




DANIELIS BERNOULLI JOH. FIL.
MED. PROF. BASIL.
ACAD. SCIENT. IMPER. PETROPOLITANÆ, PRIUS MATHESEOS
SUBLIMIORIS PROF. ORD. NUNC MEMBRI ET PROF. HONOR.

HYDRODYNAMICA,

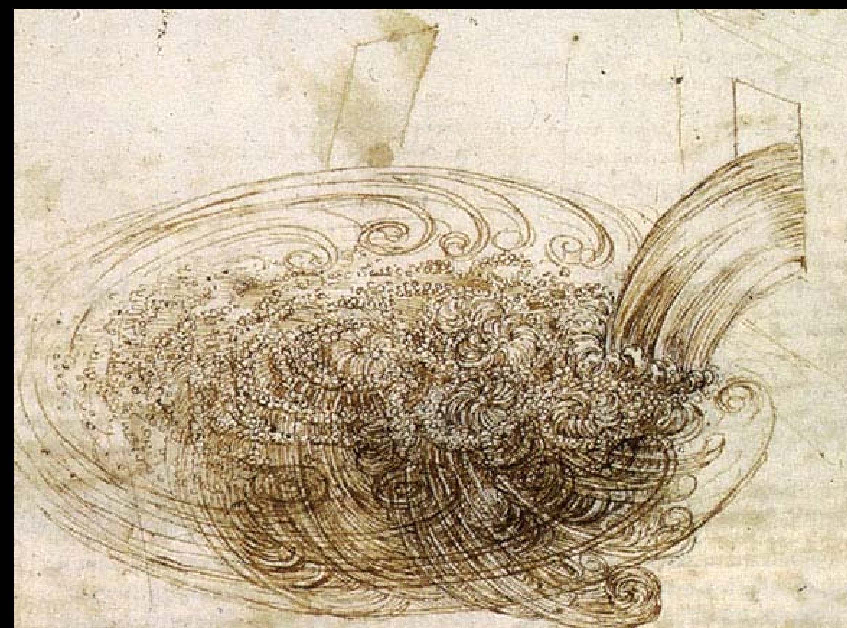
SIVE
DE VIRIBUS ET MOTIBUS FLUIDORUM
COMMENTARII.

OPUS ACADEMICUM
AB AUCTORE, DUM PETROPOLI AGERET,
CONGESTUM.

ARGENTORATI,

Sumptibus JOHANNIS REINHOLDI DULSECKERI,
Anno M D CC XXXVIII.

Typis JOH. HENR. DECKERI, Typographi Basiliensis.



Bournoulli and Stenosis

Pre-stenosis

Total energy = Kinetic + potential

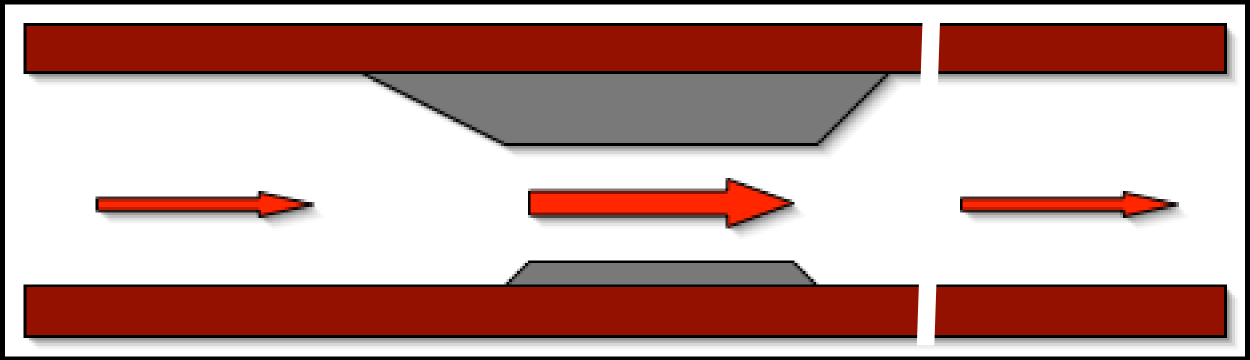
Stenosis

Potential energy very decreased

Post-stenosis

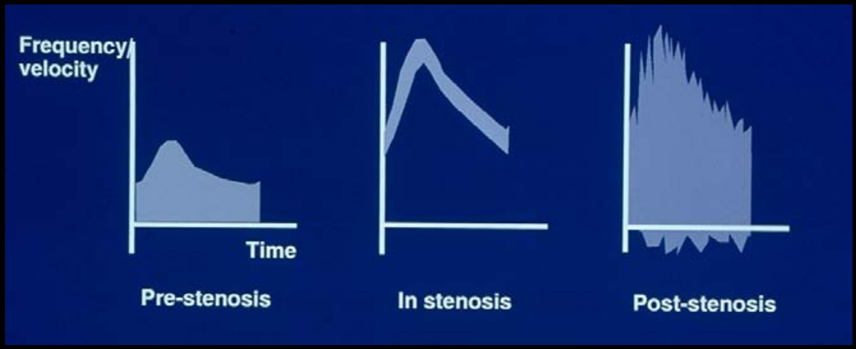
Total energy lower

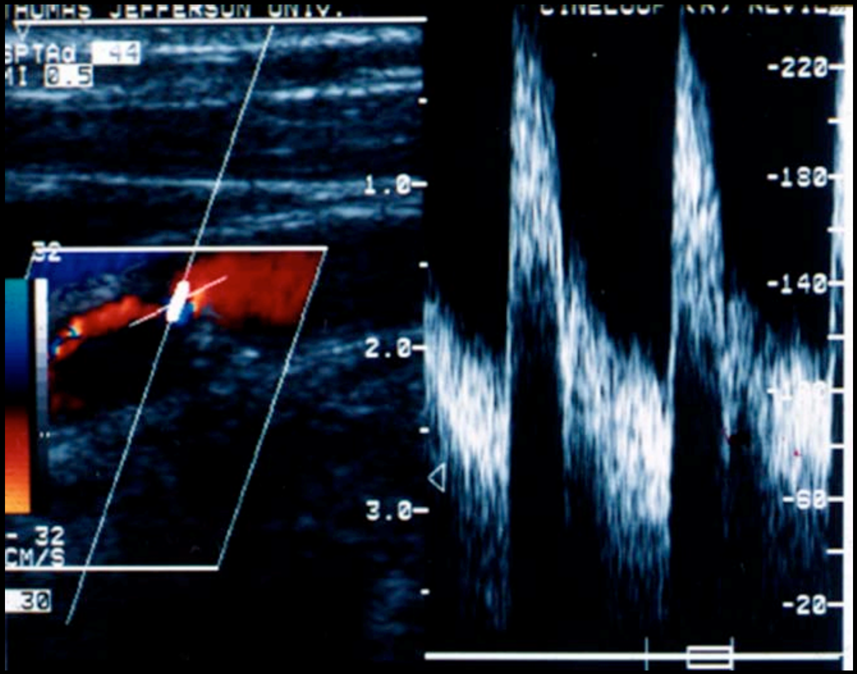
Potential energy decreased



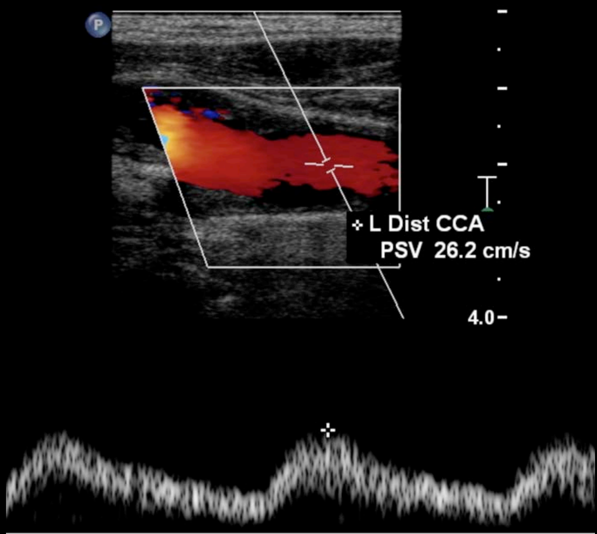
Kinetic energy very increased

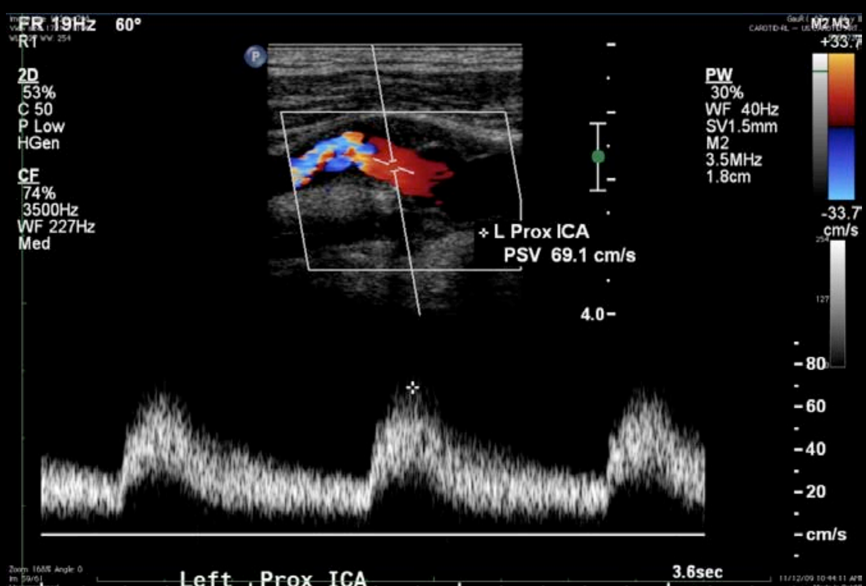
Increased Velocity in Stenosis

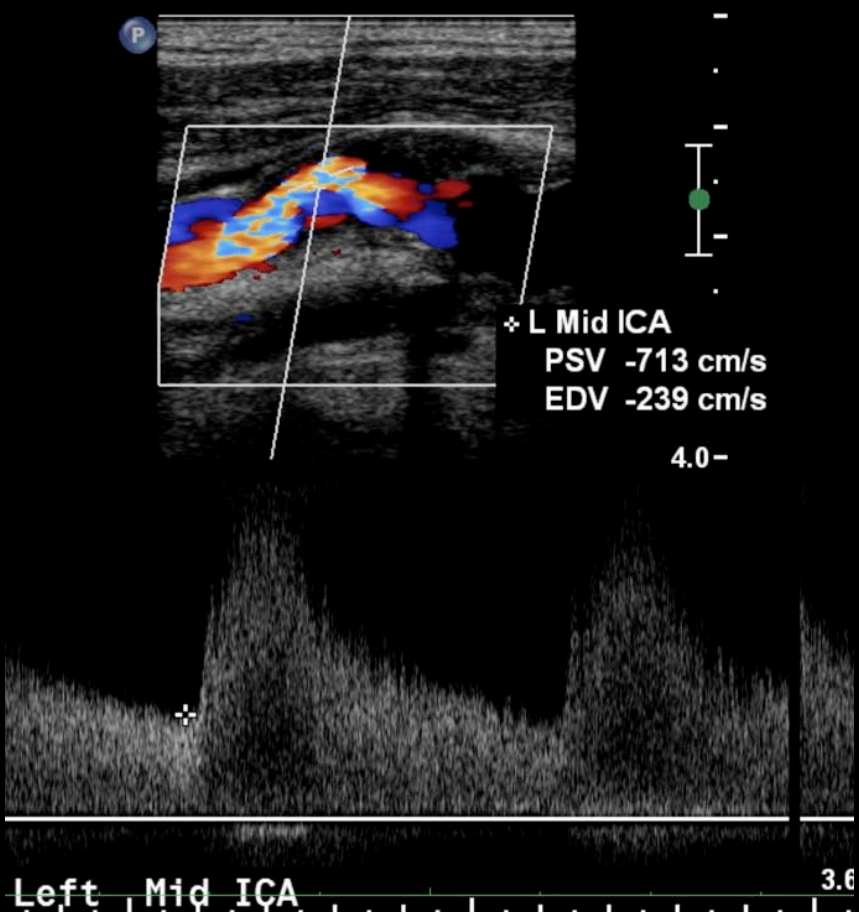




Pre and In the stenosis

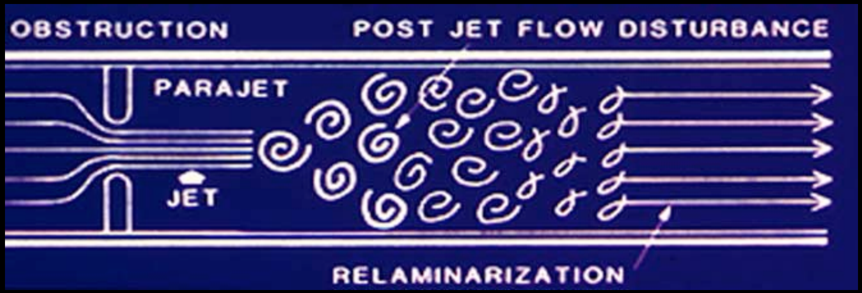




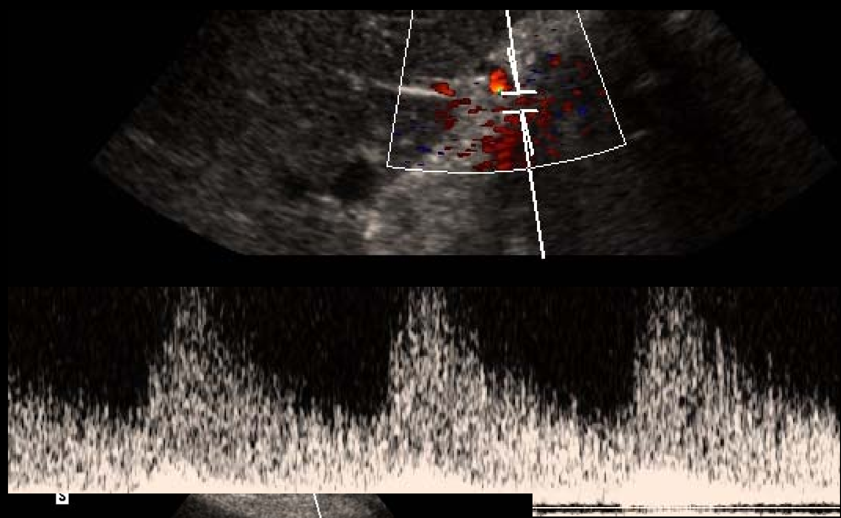


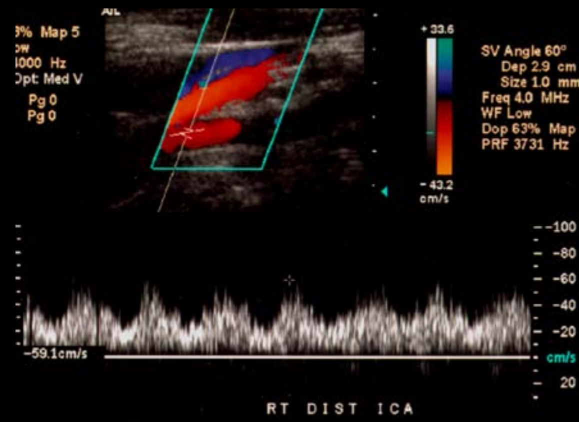
Beyond the stenosis

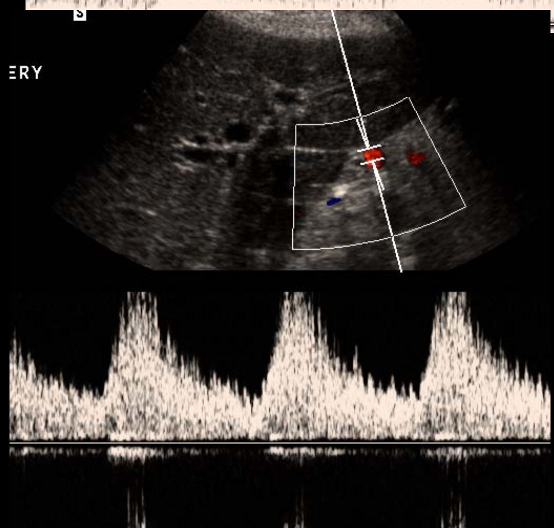
- Change from *small lumen to large lumen* destabilizes flow
  – Jet spreads out
- *High velocity* also destabilizing
  – Frank breakdown of regular flow disturbed flow (and evenually turbulence)

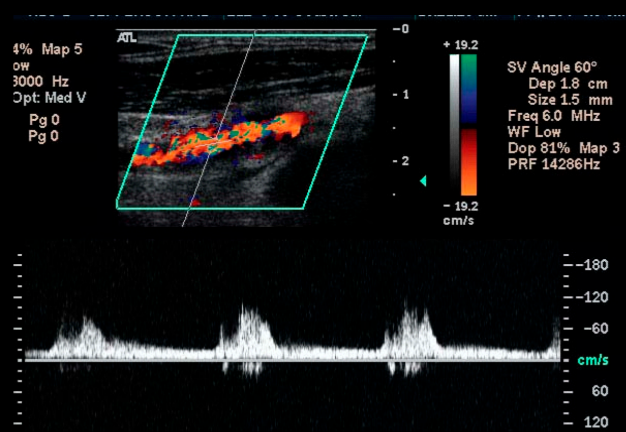


Post Stenotic Disturbed Flow









Criteria for Stenoses

- Some circulations use absolute velocity
  - Internal carotid artery
- Most circulations do not have standard velocities - Need ratios
- Some circulations use downstream effects in addition

- Peak systolic velocity ratio (velocity ratio)
  - Highest velocity in stenosis divided by velocity proximal to stenosis (in normal vessel)
  - IC:CC ratio
  - PSV ratio in arteries
  - Renal aortic ratio
- Intrarenal criteria

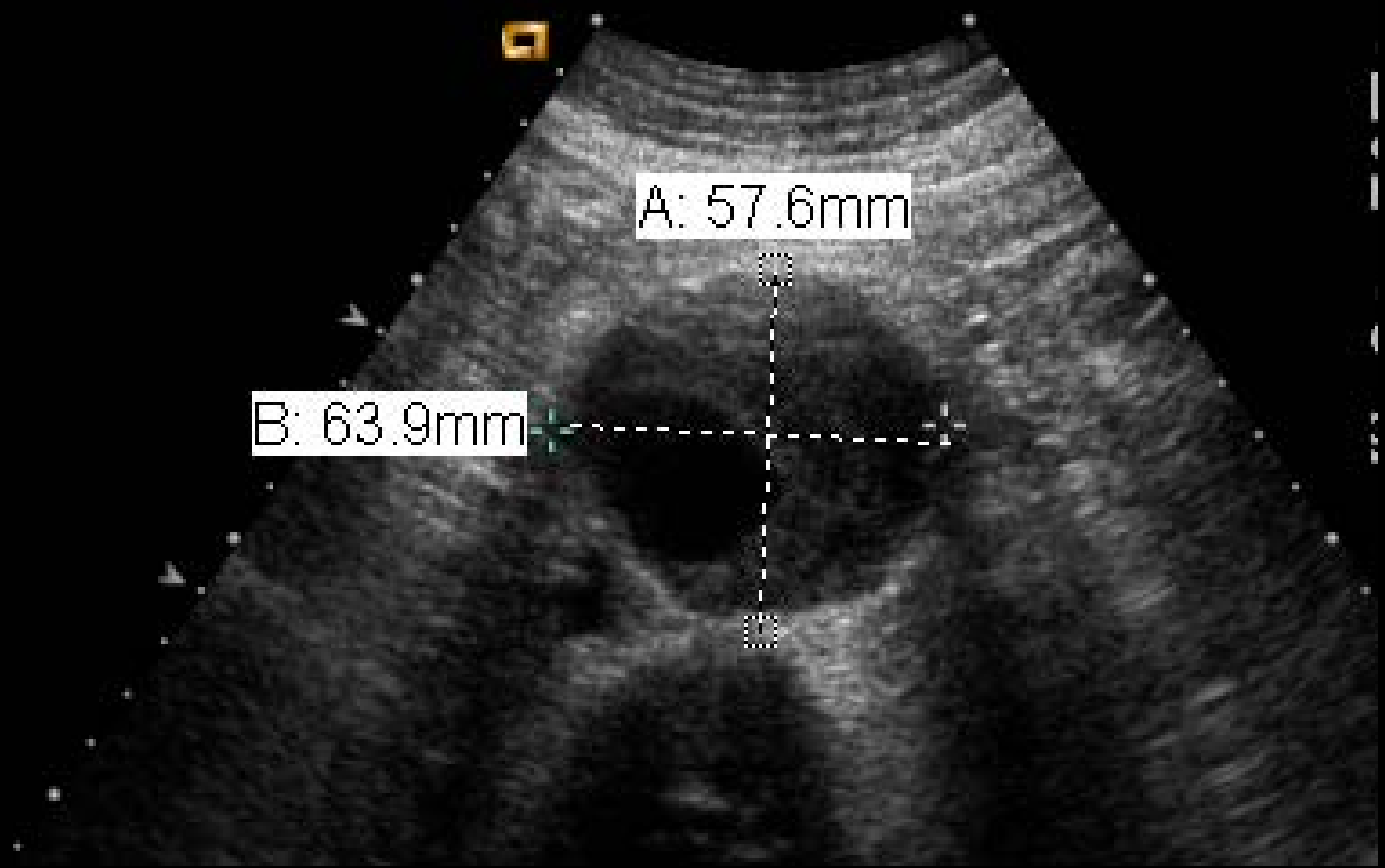
Abdominal aortic aneursym

- Abnormal dilatation of aorta
- If enlarges over 5 cm and is untreated, rupture may occur
- High mortality if rupture

- Approved for Medicare screening

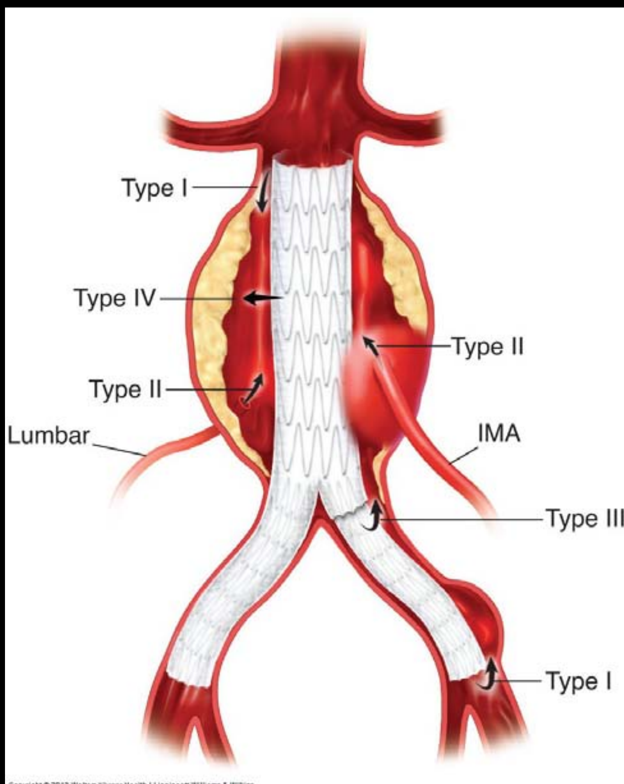
Abdominal Aortic Aneurysm (AAA)

AAA – gray scale ultrasound



AAA- Easy to measure, hard to acquire

Endoleaks


Type I
Type IV
Type II
Type II
Lumbar
IMA
Type III
Type I
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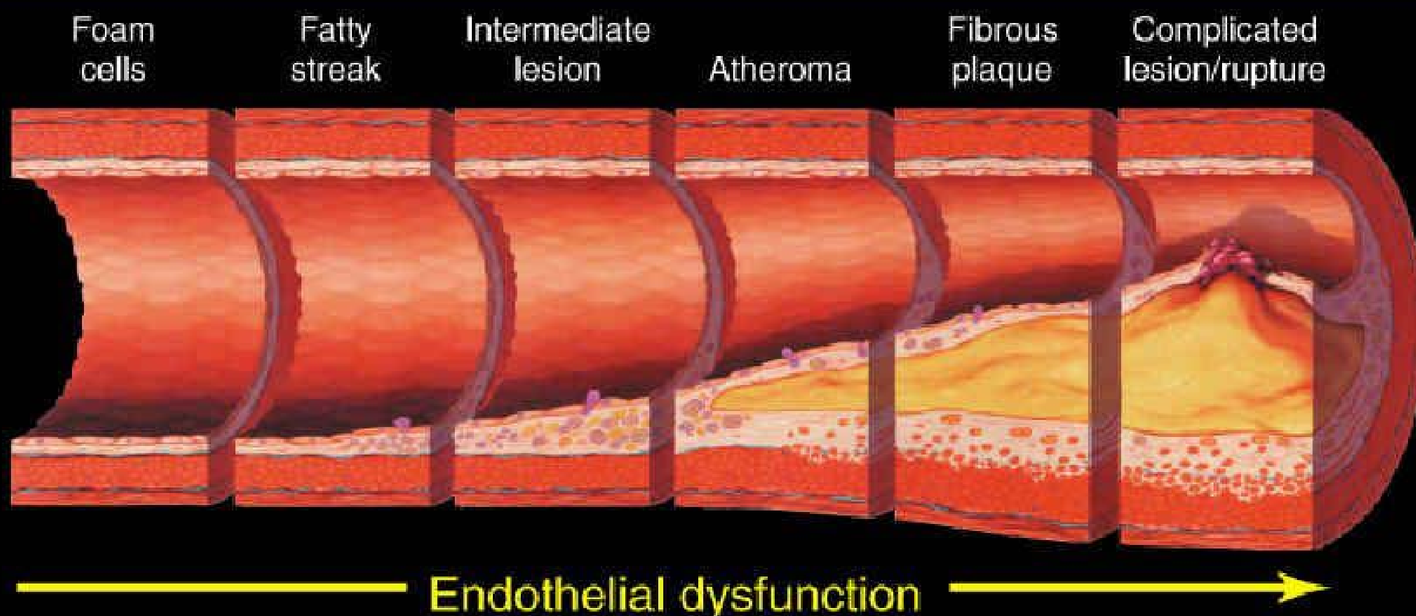
Atherosclerosis timeline



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

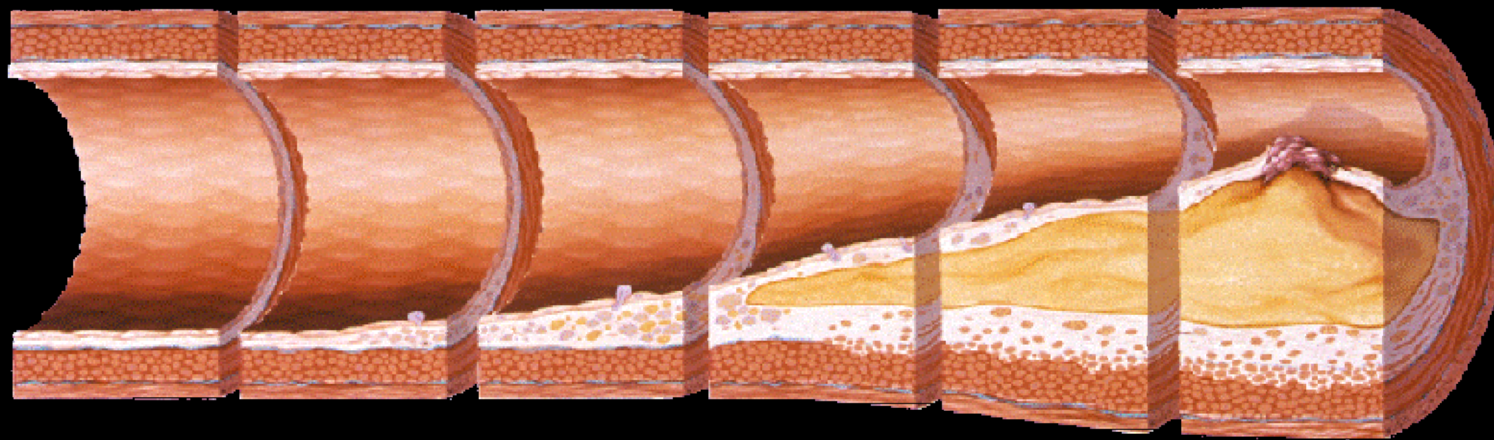
Adapted from Pepine CJ. *Am J Cardiol.* 1998;82(suppl 104).

Natural history of atherosclerosis

- Preclinical disease
  - Flow mediated dilatation, intima media thickness
  - Location of plaque
- Clinical disease
  - Degree of stenosis
  - Plaque characterization
  - Prediction of disease

Atherosclerosis Timeline

Foam Cells | Fatty Streak | Intermediate Lesion | Atheroma | Fibrous Plaque | Plaque Rupture



Cholesterol Accumulation - Inflammation → Thrombosis

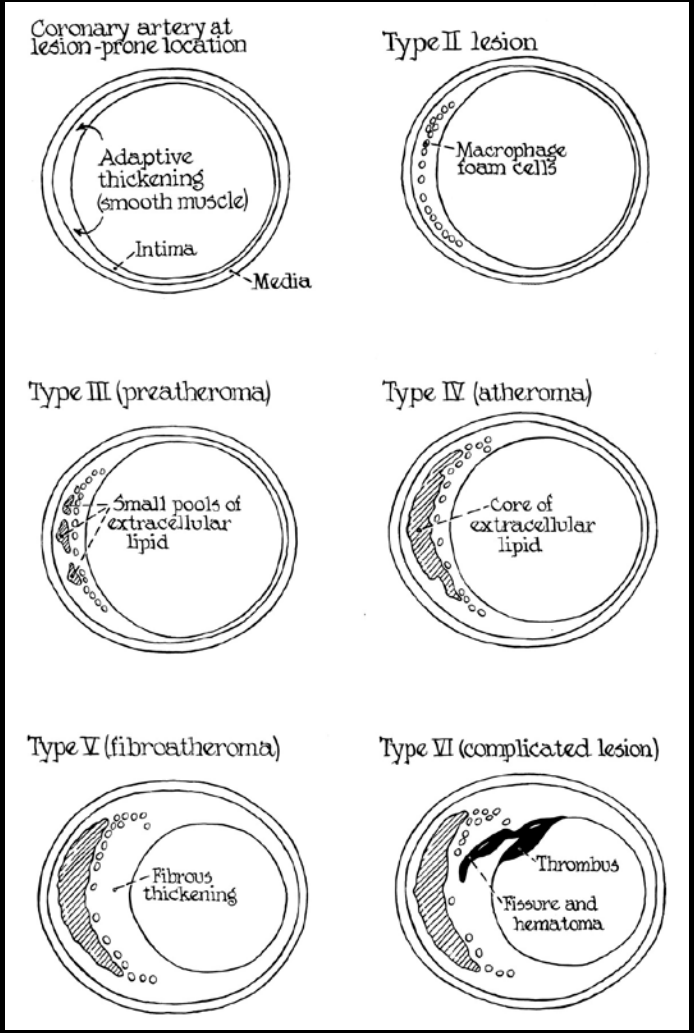
Decades	Seconds

Normal

Asymptomatic Atherosclerosis

Symptomatic Atherosclerosis:
Angina
Myocardial Infarction
Stroke

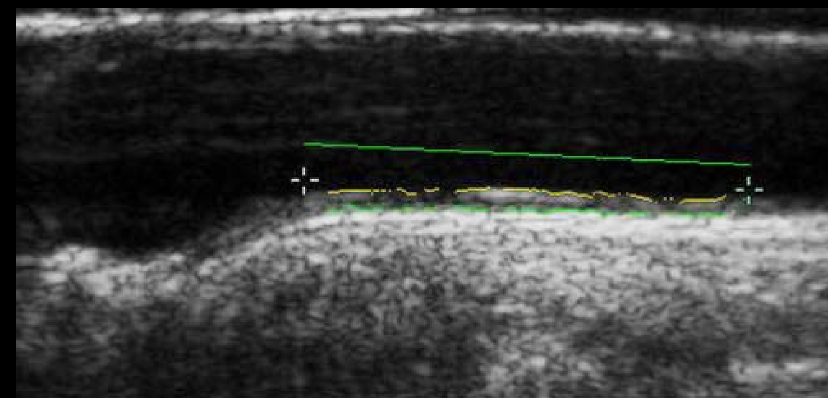
Modified from Stary HC et al, Circulation 92:1355, 1995

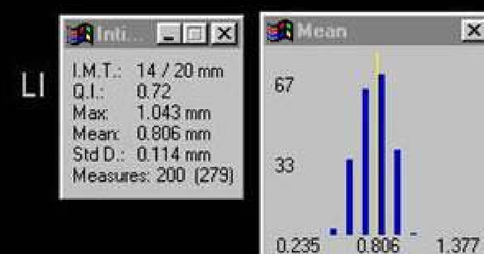


American Heart Association

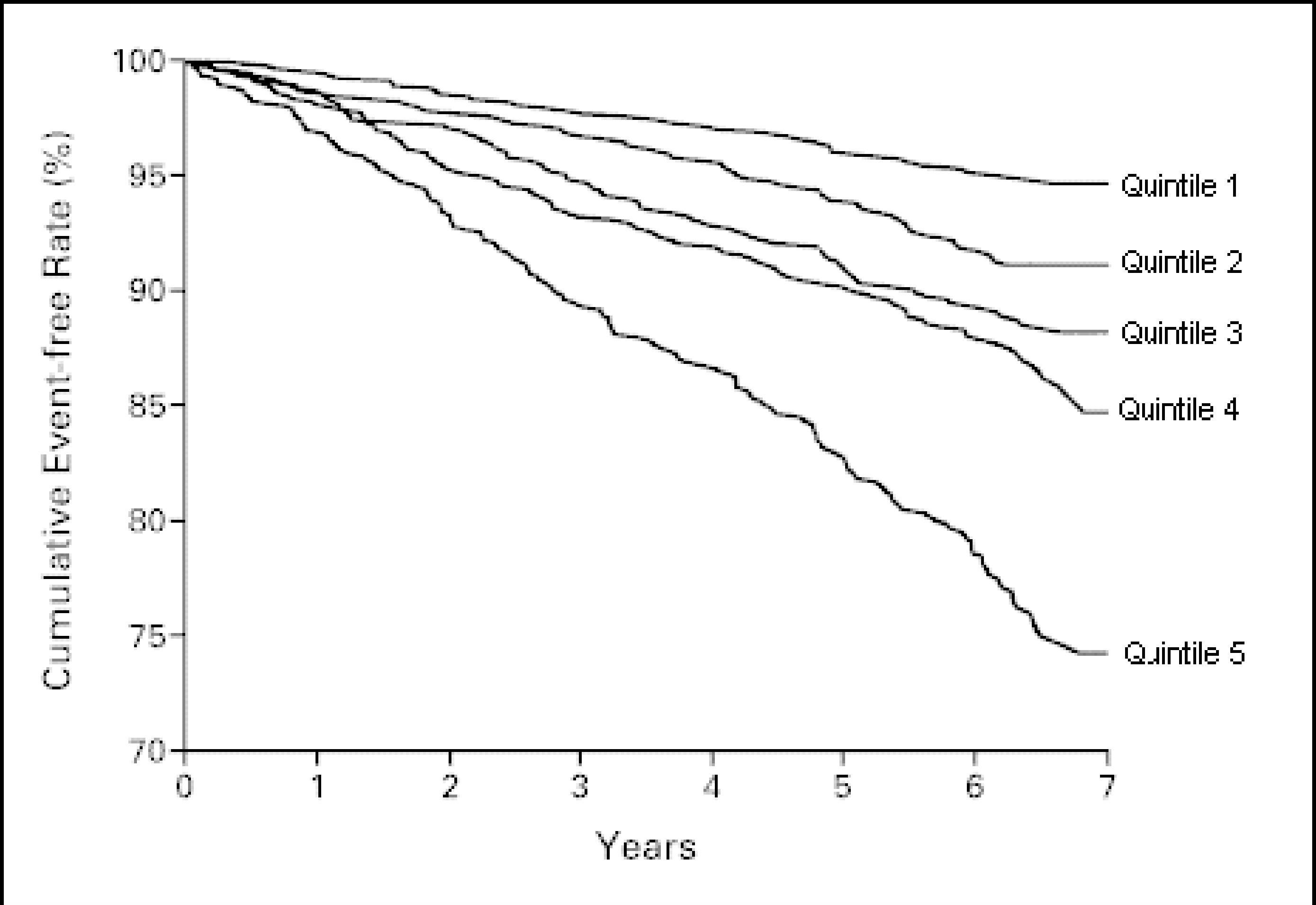
Intima Media Thickness

- Used in epidemiological studies
  - Strong predictor of future cardiovascular events
  - Additive to some traditional cardiovascular risk factors
- Used in pharmaceutical studies
- How can it be applied to the individual?
  - Reproducible clinically ?
  - How does it compare to other tests ?e.g. history, lipids, CRP, CT coronary calcium scoring?
  - One time or serial test ?





O’Leary et al
NEJM 1999:340:14-22



Weird Doppler from the bulb

Of the other three patients, one demonstrated 10–20% narrowing in an ECA but had a normal arch, siphon, and intracranial circulation. The other two demonstrated 10–20% narrowing in the carotid sinus but had normal siphons and intracranial circulations; one of these two patients demonstrated a mild (10–20%) subclavian stenosis.

In the nine patients with unilateral flow separation, the angiograms demonstrated increased distribution and severity of disease compared with the patients with bilateral flow separation. The bifurcation displaying flow separation was normal in six of

the nine patients and
diameter reduction i
eral aspect); the othe
into the mid-ICA, w
rowing. The opposit
three of the nine pati
eter reduction was
diameter reductions
lumen were noted. O
a 10–20% lesion in
similar lesion in the s
seen in six of the ni

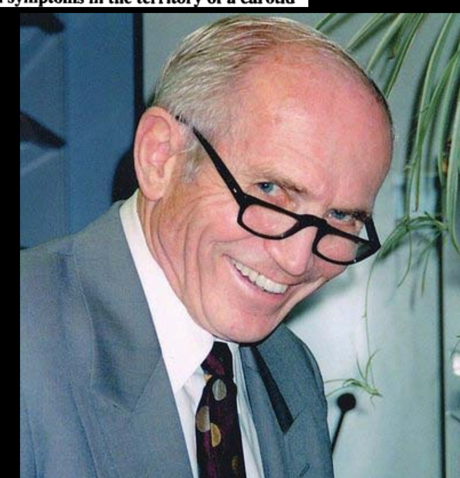
FIGURE 8.

Original Contributions

Diagnostic Significance of Flow Separation in the Carotid Bulb

Stephen C. Nicholls, MD, David J. Phillips, PhD,
Jean F. Primozich, BS, Ramona L. Lawrence, BS, Ted R. Kohler, MD,
Thomas G. Rudd, MD, and D. Eugene Strandness Jr., MD

Pulsatile blood flow within the normal carotid sinus involves at least two distinct components. That near the flow divider is laminar and antegrade, whereas a boundary layer separation zone in the posterolateral aspect exhibits transient blood flow reversal. It is now possible to document these flow velocity components using pulsed Doppler ultrasound methods. When atherosclerosis develops, it preferentially involves the posterolateral bulb region, obliterating the normal configuration of the sinus with consequent loss of the flow separation zone. It was therefore hypothesized that if flow separation could be detected, it should be predictive of a normal angiogram. To assess this, we evaluated 20 symptomatic patients and two with only bruits found by duplex scanning to have flow separation in either one or both carotid bulbs and who also underwent cerebral angiography. Initial diagnoses were stroke in seven, reversible ischemic neurologic deficit in one, transient ischemic attack in 12, and bruit in two. Flow separation was bilateral in 13 patients (59%). There were 15 patients with symptoms in the territory of a carotid



Flow separation

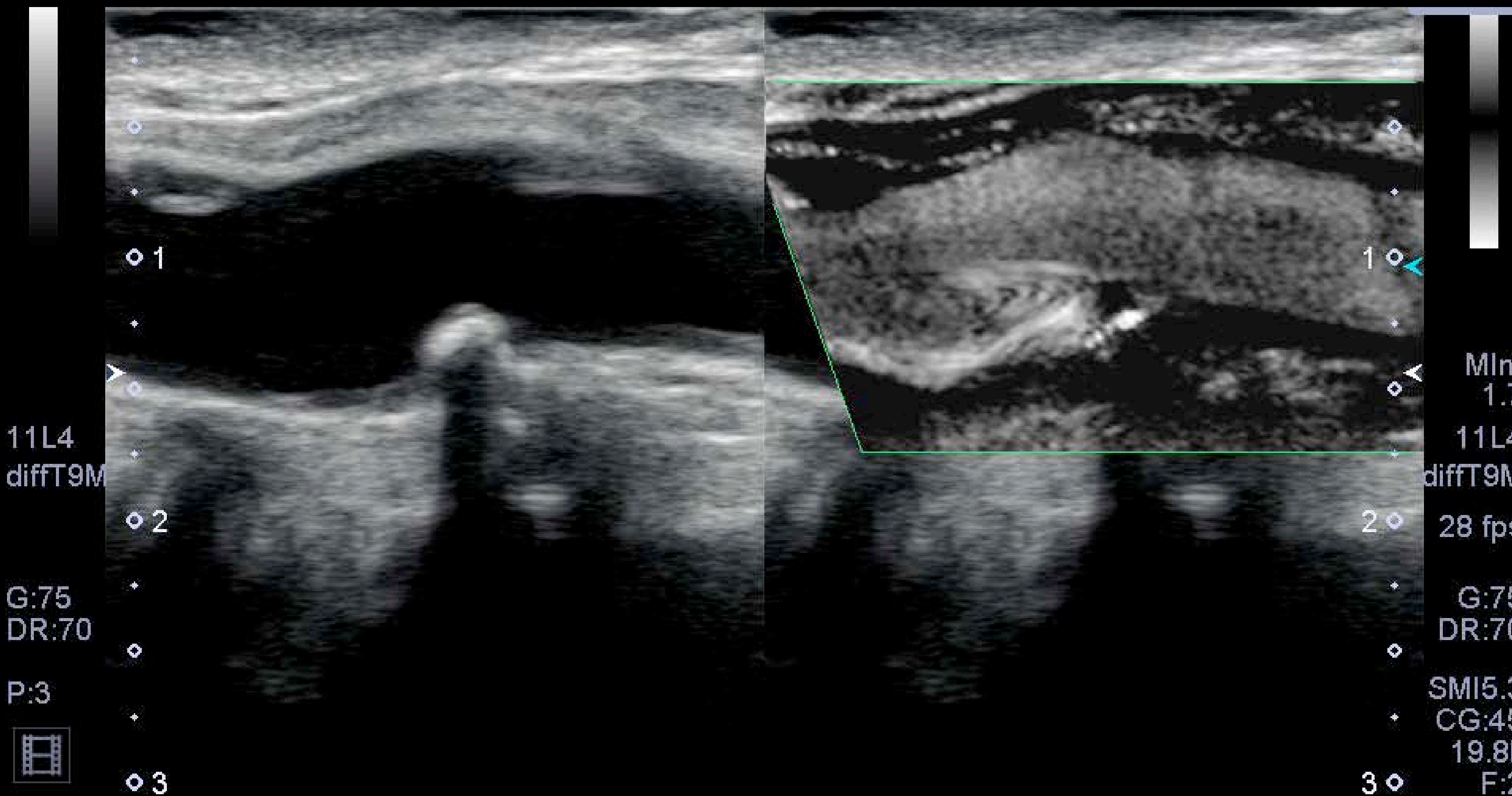
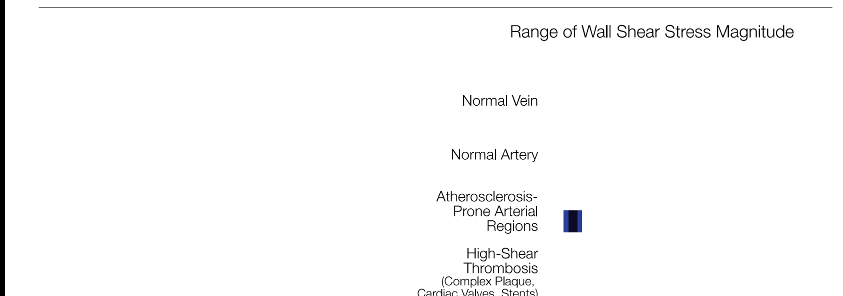
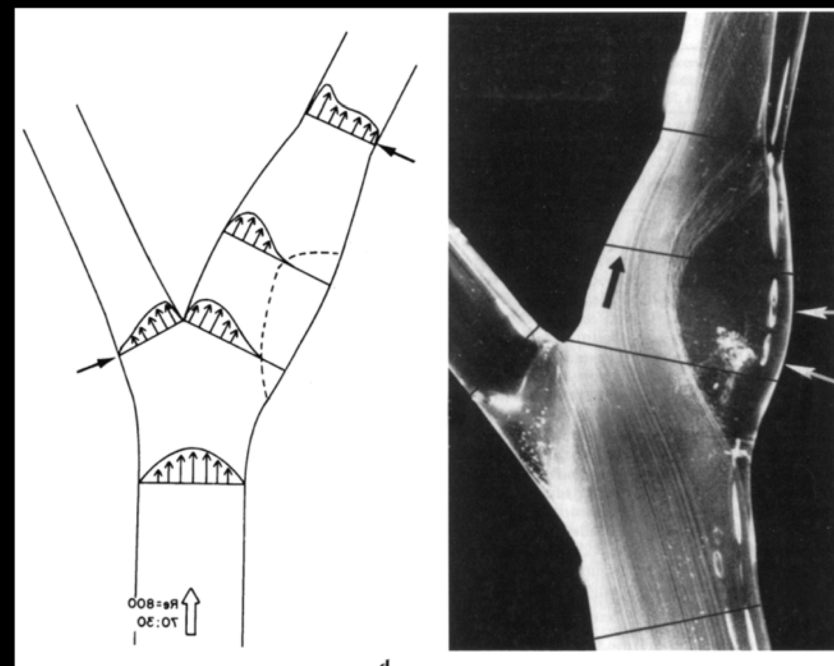
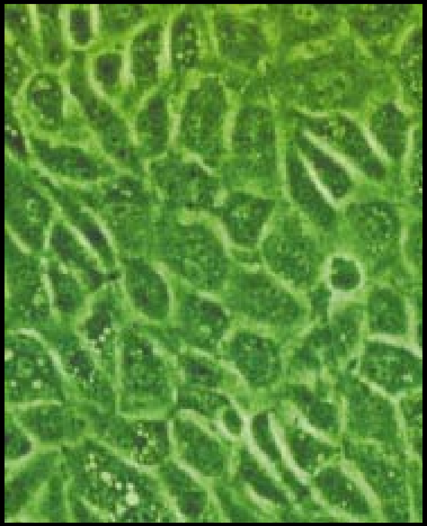


Figure 1. Hemodynamic Shear Stress





Hemodynamic Shear Stress and Its Role in Atherosclerosis

Adel M. Malek, MD, PhD
Seth L. Alper, MD, PhD
Seigo Izumo, MD

Atherosclerosis, the leading cause of death in the developed world and nearly the leading cause in the developing world, is associated with systemic risk factors including hypertension, smoking, hyperlipidemia, and diabetes mellitus, among others. Nonetheless, atherosclerosis remains a geometrically focal disease, preferentially affecting the outer edges of vessel bifurcations. In these predisposed areas, hemodynamic shear stress, the frictional force acting on the endothelial cell surface as a result of blood flow, is weaker than in protected regions. Studies have identified hemodynamic shear stress as an important determinant of endothelial function and phenotype. Arterial-level shear stress (>15 dyne/cm²) induces endothelial quiescence and an atheroprotective gene expression profile, while low shear stress (<4 dyne/cm²) which is prevalent at atherosclerosis-prone sites, stimulates an atherogenic phenotype. The functional regulation of the endothelium by local hemodynamic shear stress provides a model for understanding the focal propensity of atherosclerosis in the setting of systemic factors and may help guide future therapeutic strategies.

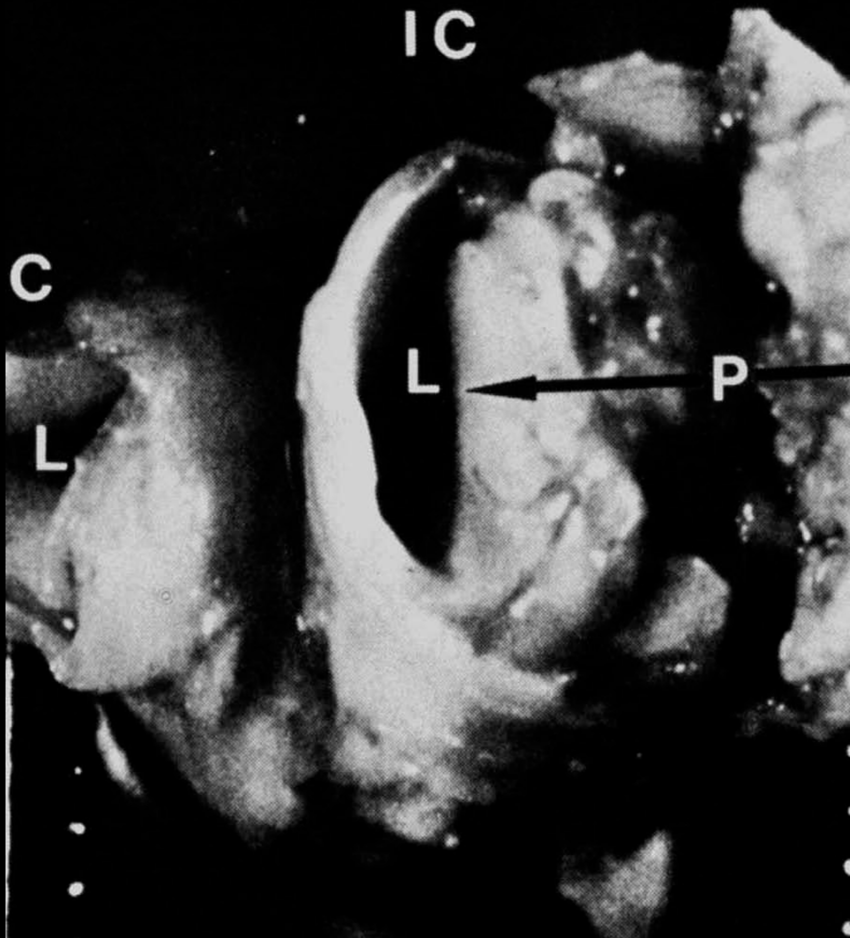
JAMA. 1999;282:2035-2042 www.jama.com

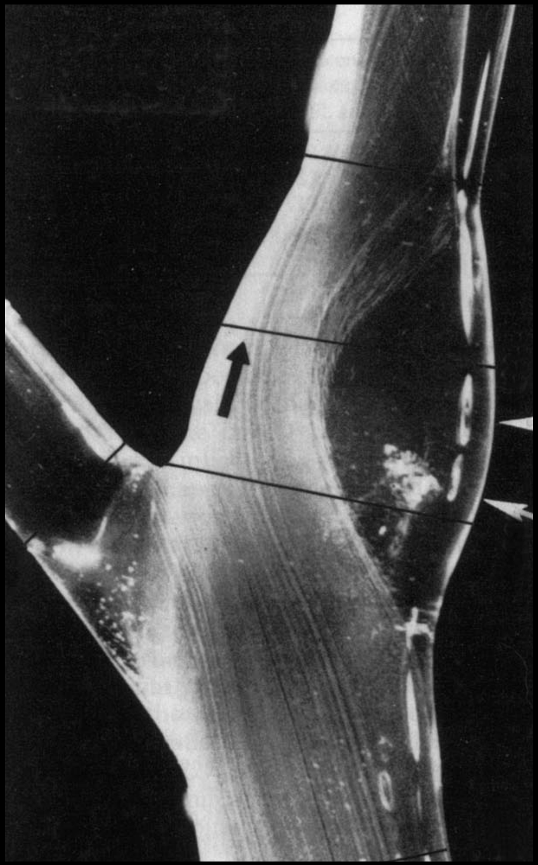
FOR MORE THAN A CENTURY, HEMODYNAMIC forces have been proposed as factors regulating blood vessel structure[1,2] and influencing development of vascular pathology such as atherosclerosis,[3-5] aneurysms,[6] poststenotic dilatations,[7] and arteriovenous malformations.[8] The flow of blood, by virtue of viscosity, engenders on the luminal vessel wall and endothelial surface a frictional force per unit area known as hemodynamic shear stress.[9-11] Shear stress has not only been shown to be a critical determinant of vessel caliber,[2,11,12] but has also been implicated in vascular remodeling[13,14] and pathobiology.[5]

sponse to hemodynamic shear stress have provided new insights into its possible contribution to the pathogenesis

to blood flow viscosity, and inversely proportional to the third power of the internal radius.[11,12,31,32] Measurements us-

Plaque

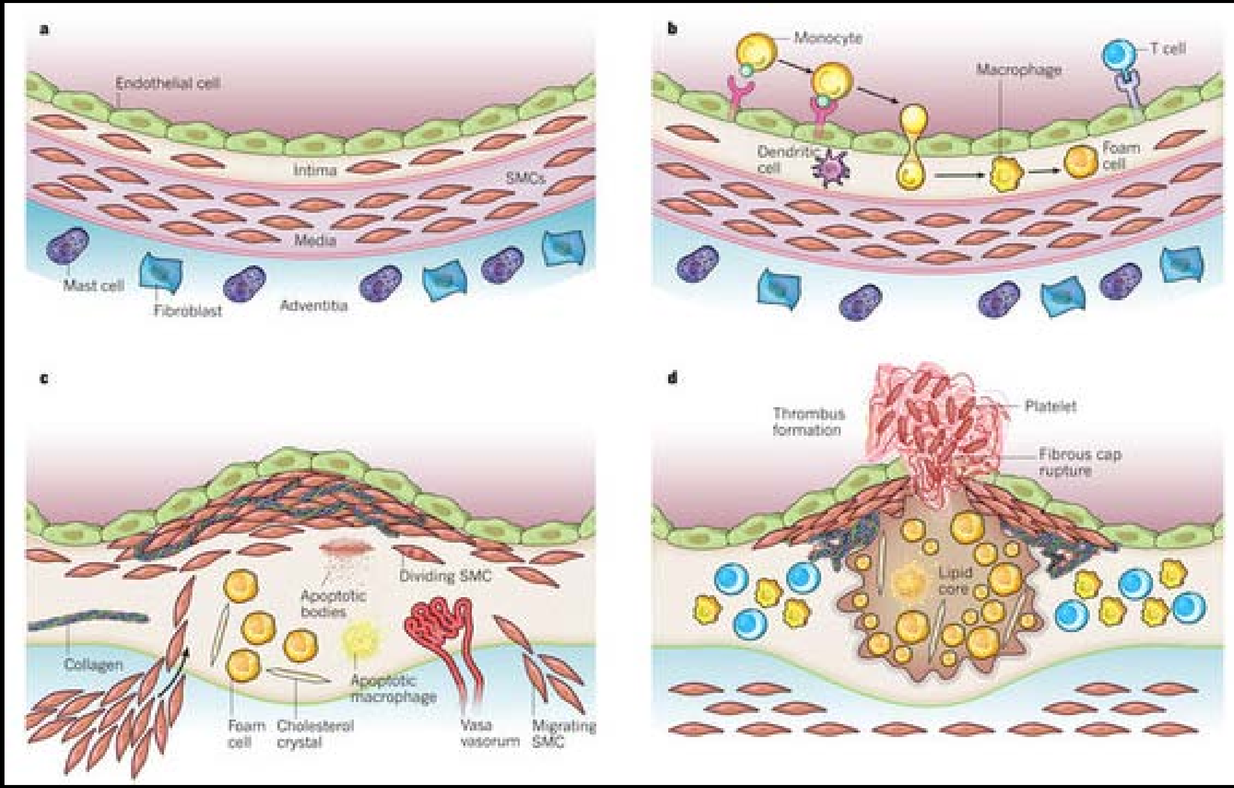




Progress and challenges in translating the biology of atherosclerosis

Peter Libby[1], Paul M Ridker[1,2] & Göran K. Hansson[3]

19 MAY 2011 | VOL 473 | NATURE | 317



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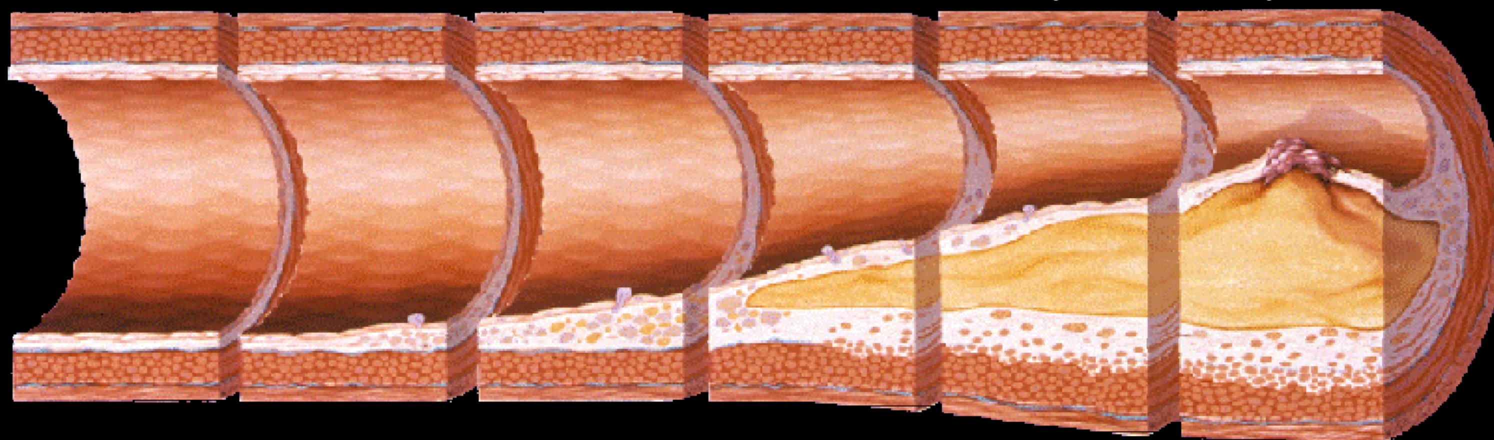

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Modified from Stary HC et al, Circulation 92:1355, 1995

e British Journal of Radiology, 83 (2010), 729–735

REVIEW ARTICLE

Introduction to the biomechanics of carotid plaque pathogenesis and rupture: review of the clinical evidence

[1,2]G C MAKRIS, MD, [2]A N NICOLAIDES, MD, MS, FRCS, [3]X Y XU, PhD and [1,2]G GEROULAKOS, MD, DIC, PhD

[1]*Vascular Surgery Department, Ealing Hospital, NHS Trust, London, UK,* [2]*Imperial College London, UK, and* [3]*Department of Chemical Engineering, Imperial College London, UK*

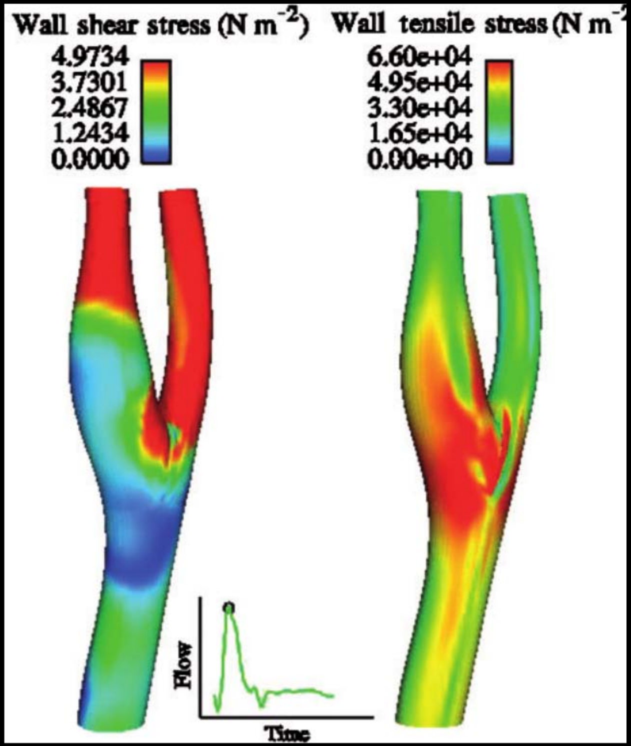
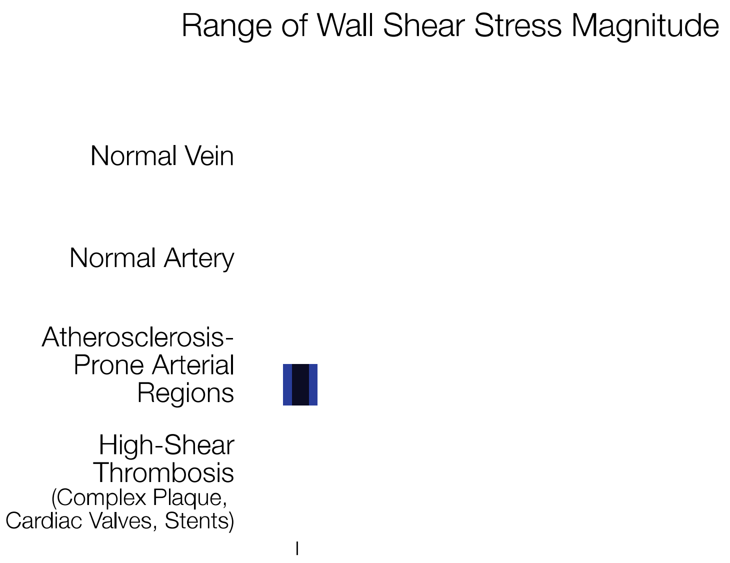


Figure 1. Hemodynamic Shear Stress



Carotid artery intraplaque hemorrhage and stenotic velocity

KW Beach, T Hatsukami, PR Detmer, JF Primozich, MS Ferguson, D Gordon, CE Alpers, DH Burns, BD Thackray and DE Strandness, Jr

Stroke 1993, 24:314-319

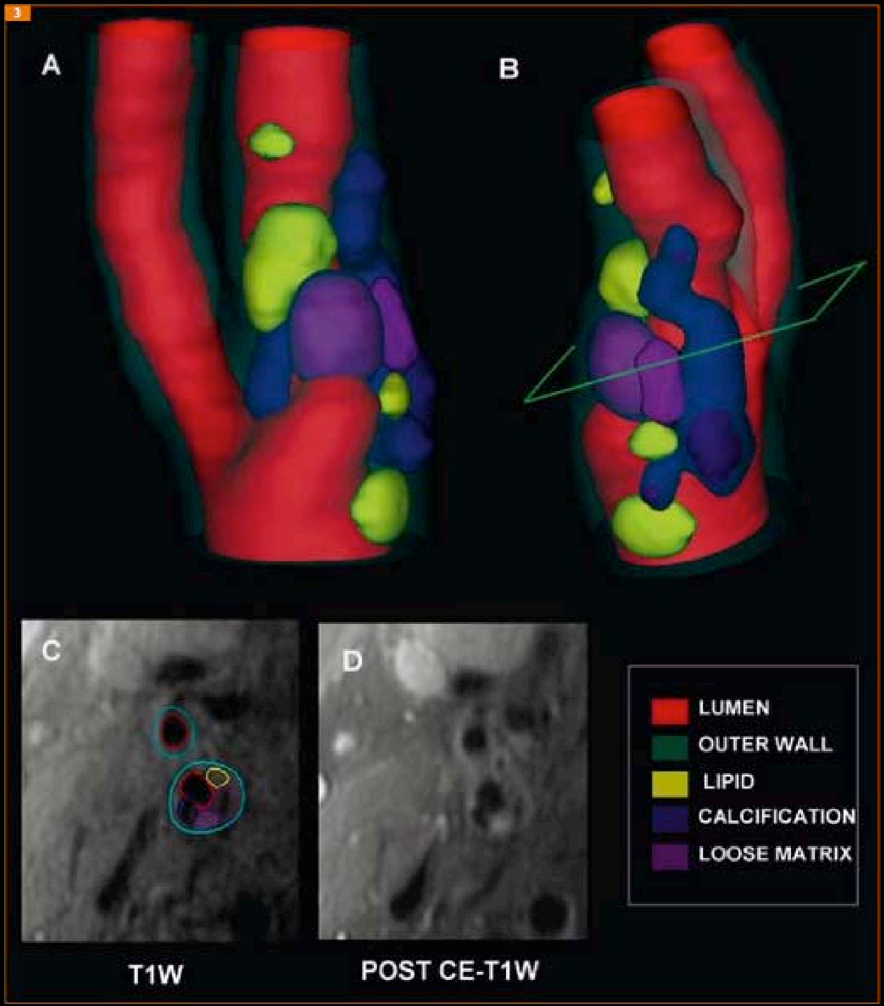
'hotomicrographs illustrating classifications of
morrhage. Top left: H1, perioperative hemor-
s formed erythrocytes. Upper left: H2, recent
ontains erythrocyte ghosts. Lower left: H3, older
ontains coalesced erythrocytes. Bottom left: H4,
ge; contains solid thrombus. Top right: H5,
hage; "glassy" appearance. Upper right: NV,
ation; contains endothelialized channels. Lower
Bottom right: drawing of section showing
I1 to NV.

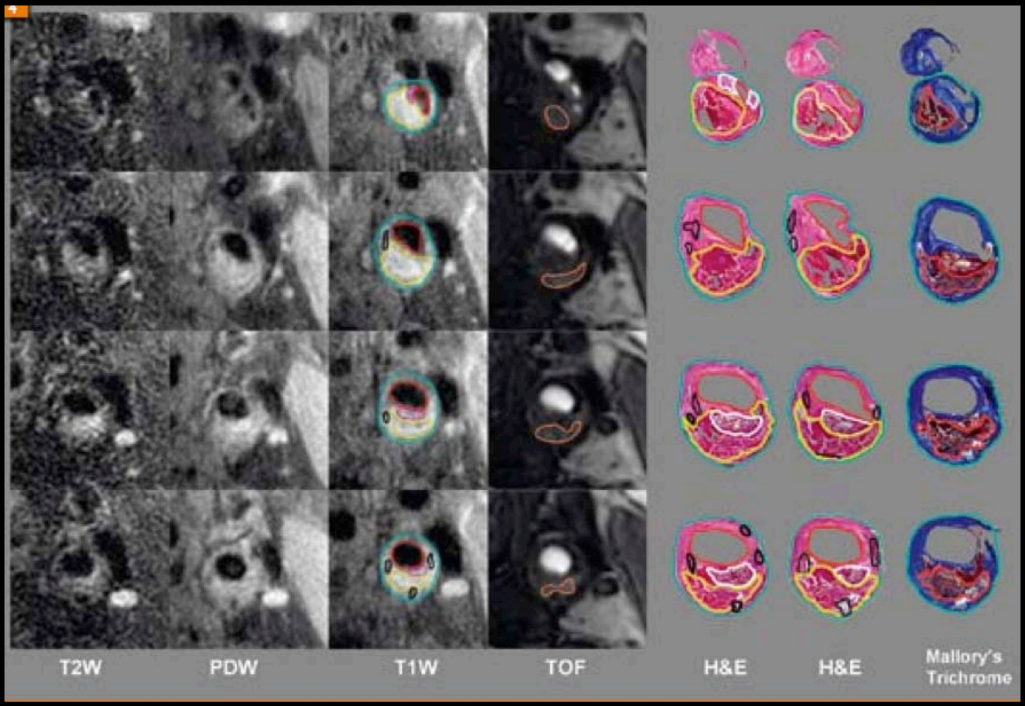
the plaques that we studied, the regions of
emorrhage were not of uniform "age" ac-
tological analysis. Therefore, more than one
event probably occurred.
results of this study are preliminary, the

where ρ=density of fluid, g=acceleratio
h=height of fluid column, and v=fluid v
chanical energy can be freely exchanged
two forms, kinetic and potential. In a Ve
flow is always laminar and no boundary lay
occurs in the poststenotic region (Figure
Pressure at the distal end of the tube is ne
that at the proximal end. The transmu
exerted on the wall in the stenosis is lowe
the proximal or the distal pressure. This is
potential energy density (pressure energy)
imal region, where velocity is low, must b
into kinetic energy density (velocity en
stenosis, where velocity is high.

If, distal to the stenosis, the tube is shape
flow is streamlined, then kinetic energy

MRI of the vulnerable carotid plaque





Annals of Medicine. 2008; 40: 606–621

informa healthcare

REVIEW ARTICLE

Inflammatory angiogenesis in atherogenesis – a double-edged sword

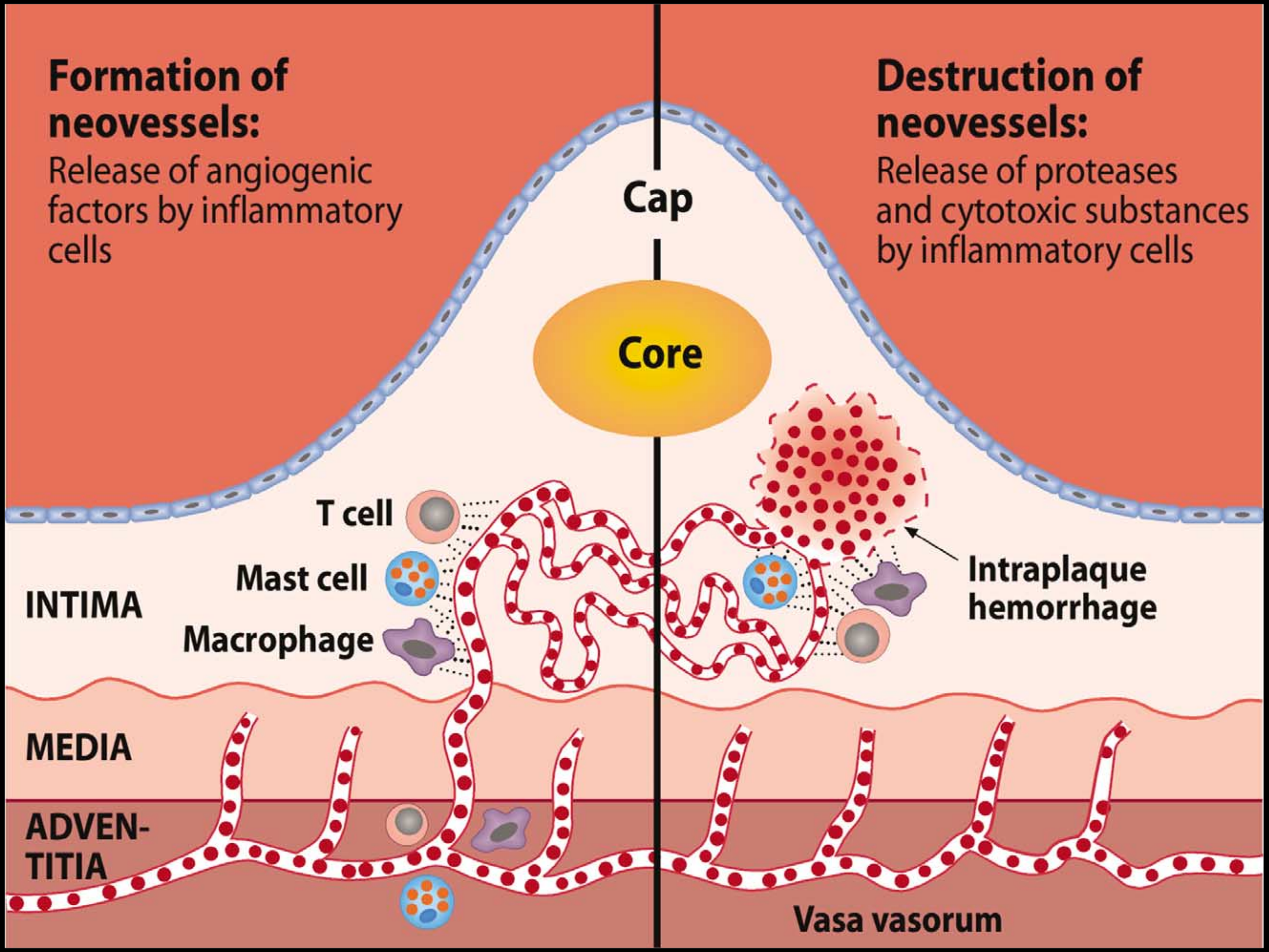


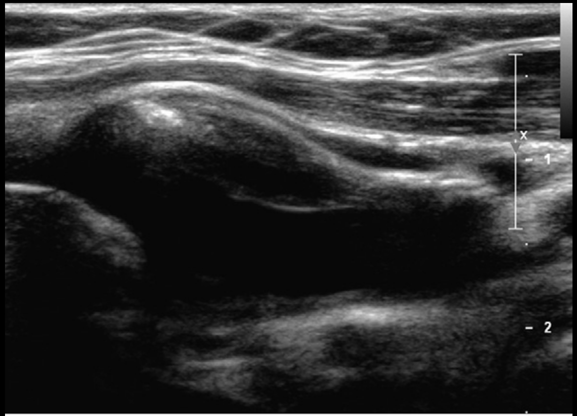
Figure 2. Schematic presentation of the two roles of an infiltrate of proinflammatory cells in an advanced atherosclerotic lesion: left angiogenic and right angiolytic effects. The infiltrate consists of macrophages, T cells, and mast cells. Left: by releasing a variety of angiogenic factors, the cells induce the growth of neovessels which originate from the vasa vasorum in the outer layer of the arterial wall. Right: by releasing a variety of proteases and cytotoxic substances, the cells induce death of endothelial cells and so create local disruption of the microvessels. The ensuing intraplaque hemorrhage tends to weaken the plaque and predispose to plaque rupture with ensuing clinical sequelae, such as myocardial infarction and stroke.

Correlation of Carotid Artery Atherosclerotic Lesion Echogenicity and Severity at Standard US with Intraplaque Neovascularization Detected at Contrast-enhanced US[1]

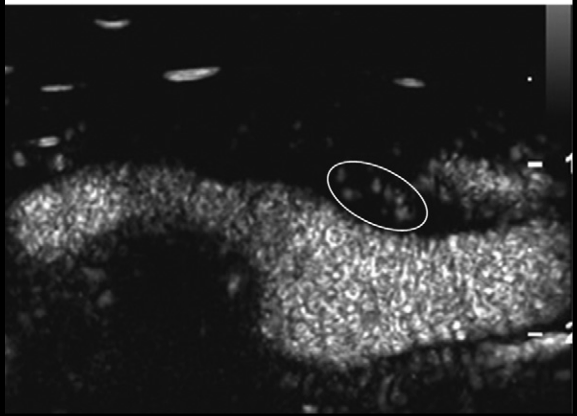
Daniel Staub, MD
Sasan Partovi, BS
Arend F. L. Schinkel, MD, PhD
Blai Coll, MD, PhD
Heiko Uthoff, MD
Markus Aschwanden, MD
Kurt A. Jaeger, MD
Steven B. Feinstein, MD

Purpose: To correlate echogenicity and severity of atherosclerotic carotid artery lesions at standard ultrasonography (US) with the degree of intraplaque neovascularization at contrast material–enhanced (CE) US.

Materials and Methods: This HIPAA-compliant study was approved by the local ethics committee, and all patients provided informed consent. A total of 175 patients (113 [65%] men, 62 [35%] women; mean age, 67 years ± 10 [standard deviation]) underwent standard and CE US of the carotid artery. Le-



a.



Inflammation within Carotid Atherosclerotic Plaque: Assessment with Late-Phase Contrast-enhanced US[1]

David R. Owen, MA, MBBS, MRCP
Joseph Shalhoub, BSc, MBBS, MRCS
Sam Miller, MSc
Thomas Gauthier, MSc
Ortansia Doryforou, MBBS
Alun H. Davies, MA, DM, FRCS, FHEA
Edward L. S. Leen, MB, MCh, BAO, MD, FRCR

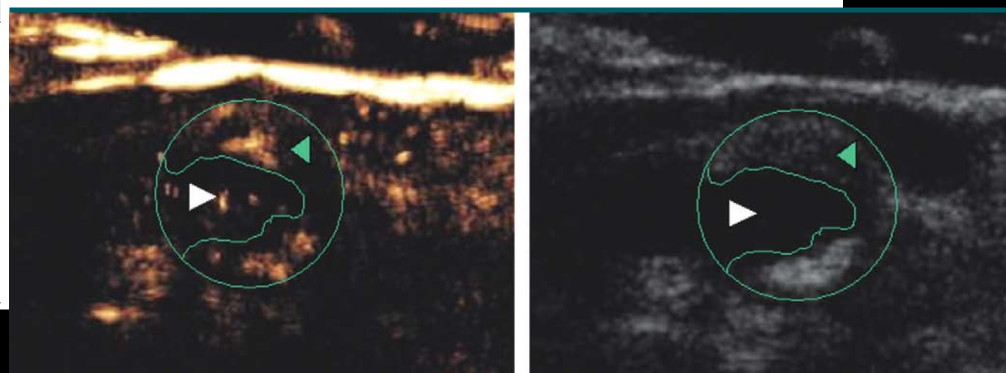


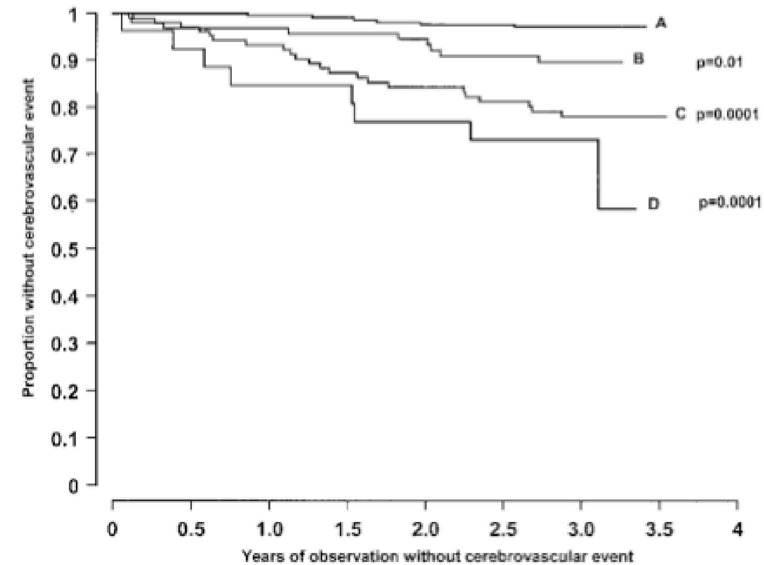
Table 2

US Features of Carotid Plaque in Patients with and Those without Symptoms

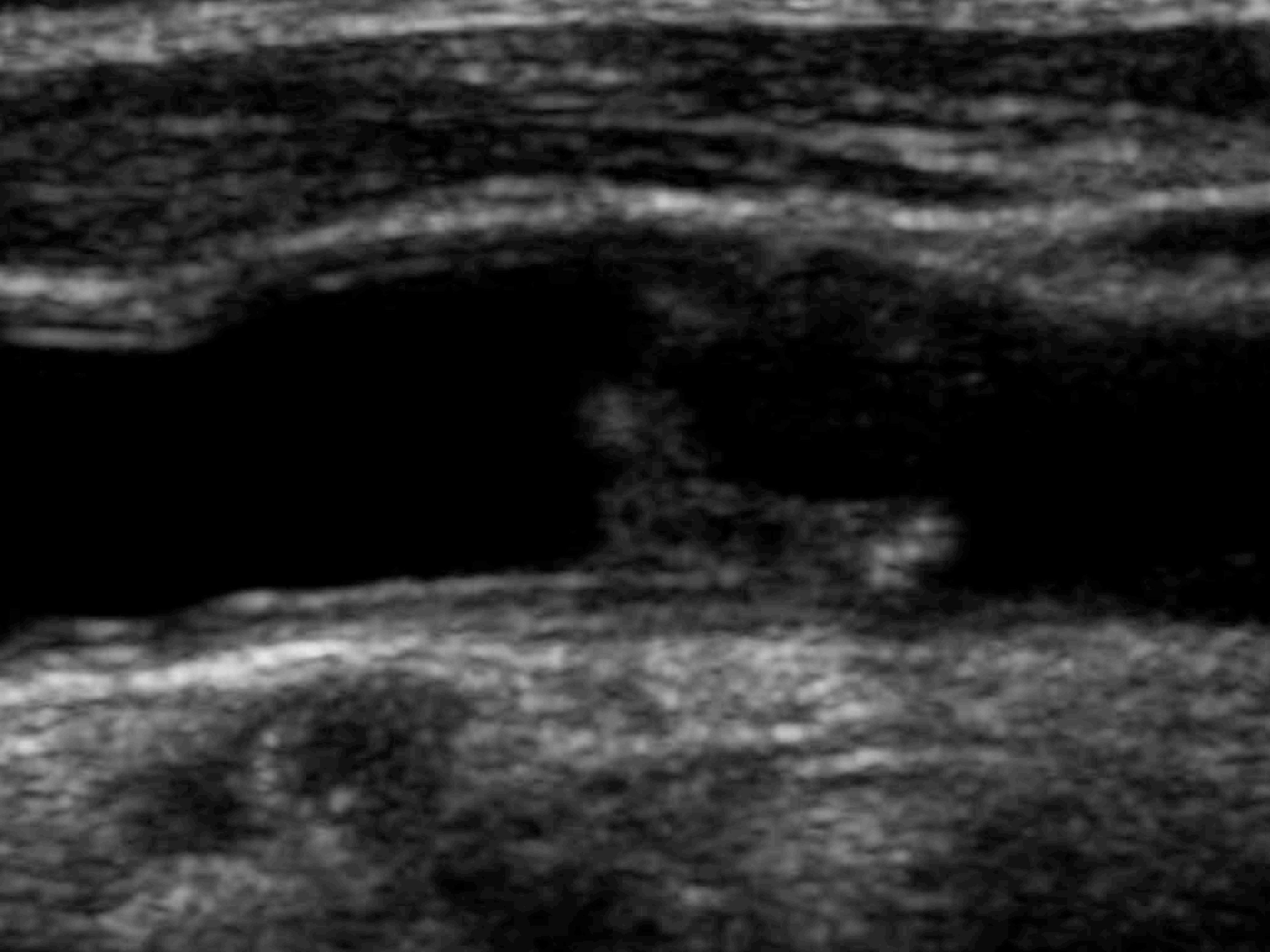
Variable	Symptomatic Group (*n* = 16)	Asymptomatic Group (*n* = 21)	Difference between Symptomatic and Asymptomatic Groups	*P* Value
Luminal stenosis (%)	79 (69, 89)	67 (58, 75)	12 (−0.4, −25)	.06
LP contrast-enhanced US	0.39 (−0.11, 0.89)	−0.69 (−1.04, −0.34)	1.08 (0.49, 1.66)	.0008
Gray-scale median score	17 (13, 20)	29 (21, 37)	−12 (−3, −21)	.009

Note.—Unless otherwise indicated, data are means, and data in parentheses are 95% confidence intervals.

This plaque is different



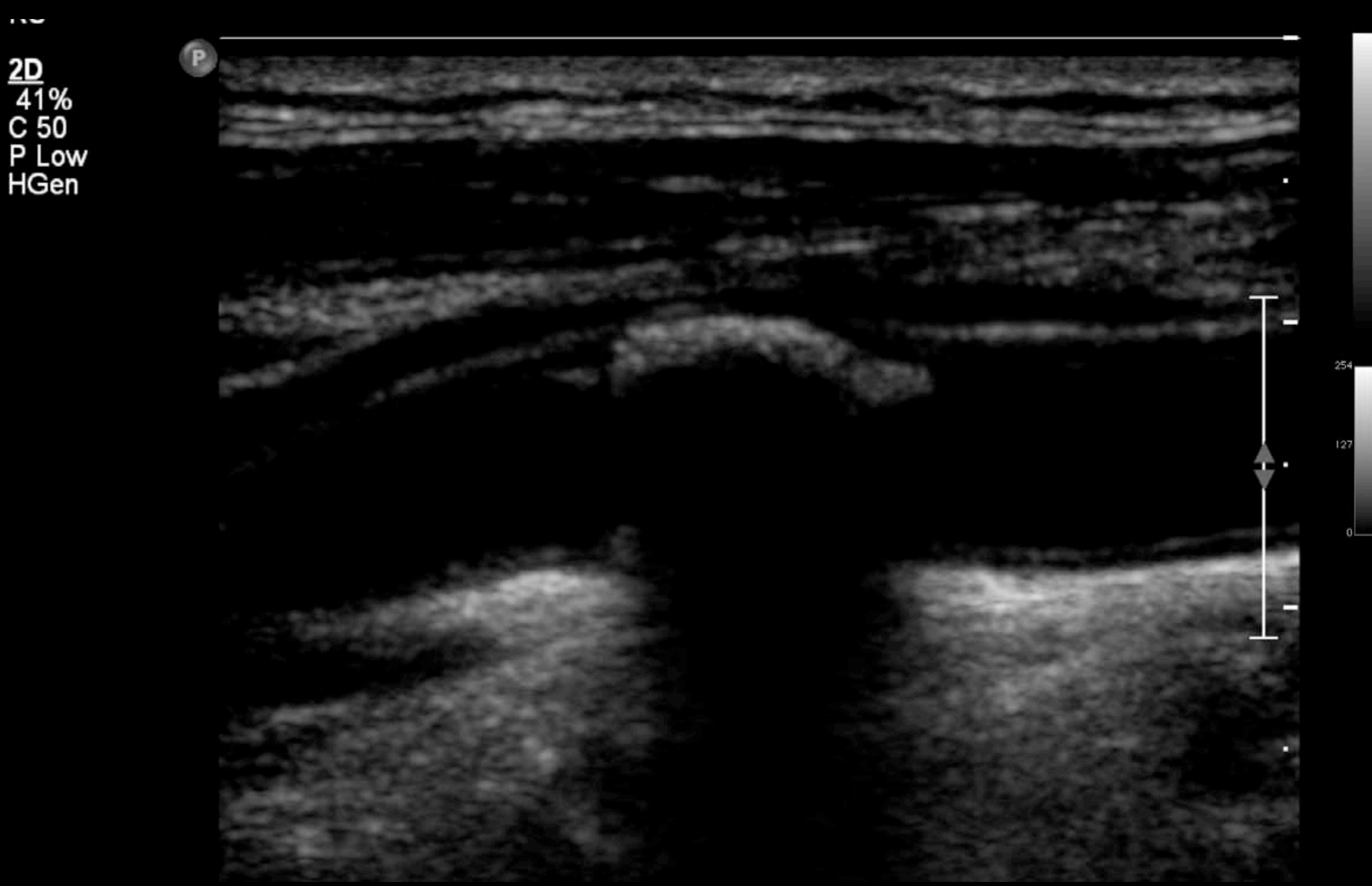
Graph of event-free survival for subjects without stenosis and subjects with stenosis according to plaque echogenicity. A, Subjects without stenosis; B, subjects with echogenic and predominantly echogenic plaques; C, subjects with predominantly echolucent plaques; and D, subjects with echolucent plaques. Probability values refer to comparison between group B, C, or D vs control subjects (A).



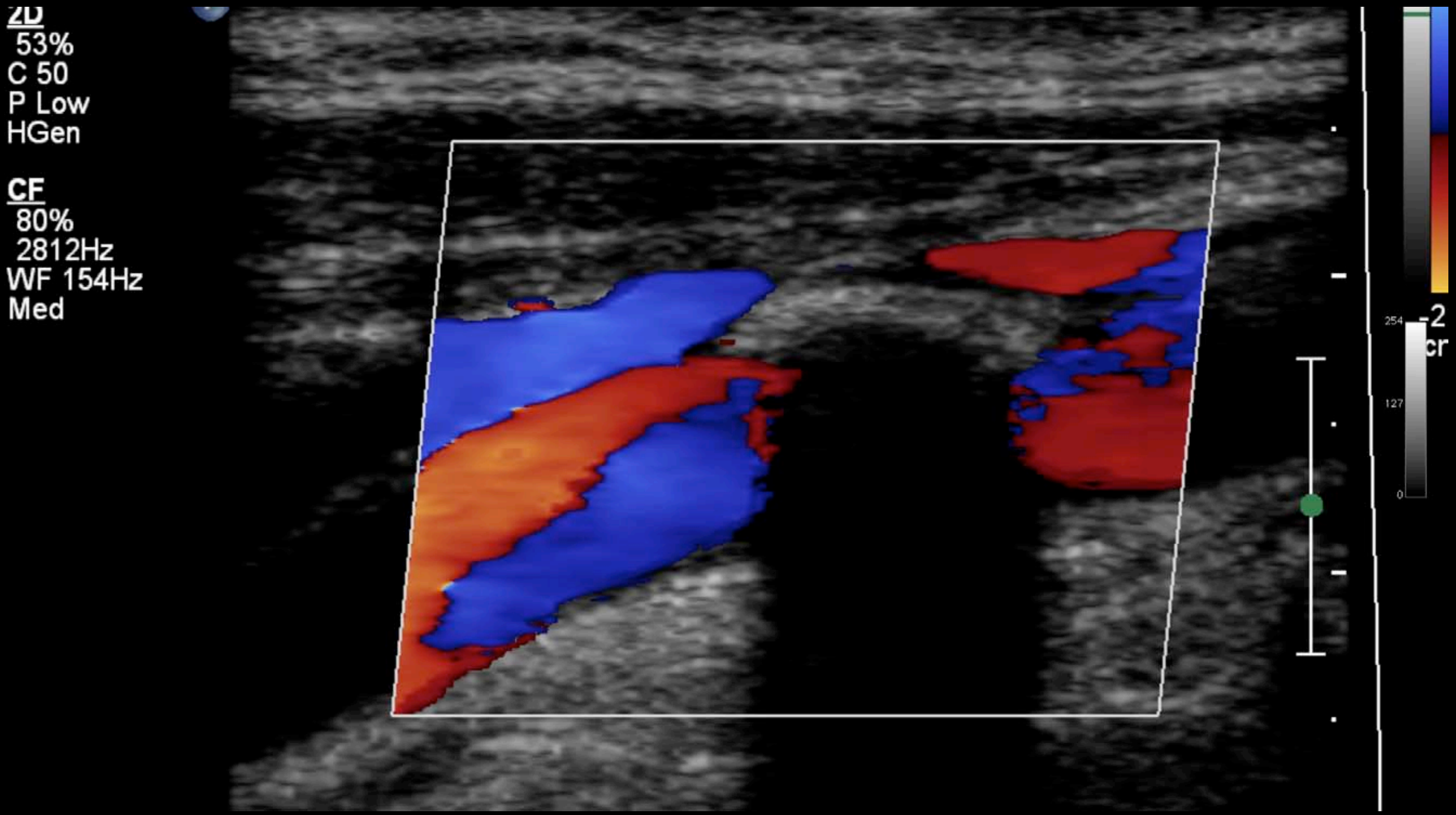
Current needs and problems

Calcifications and depth

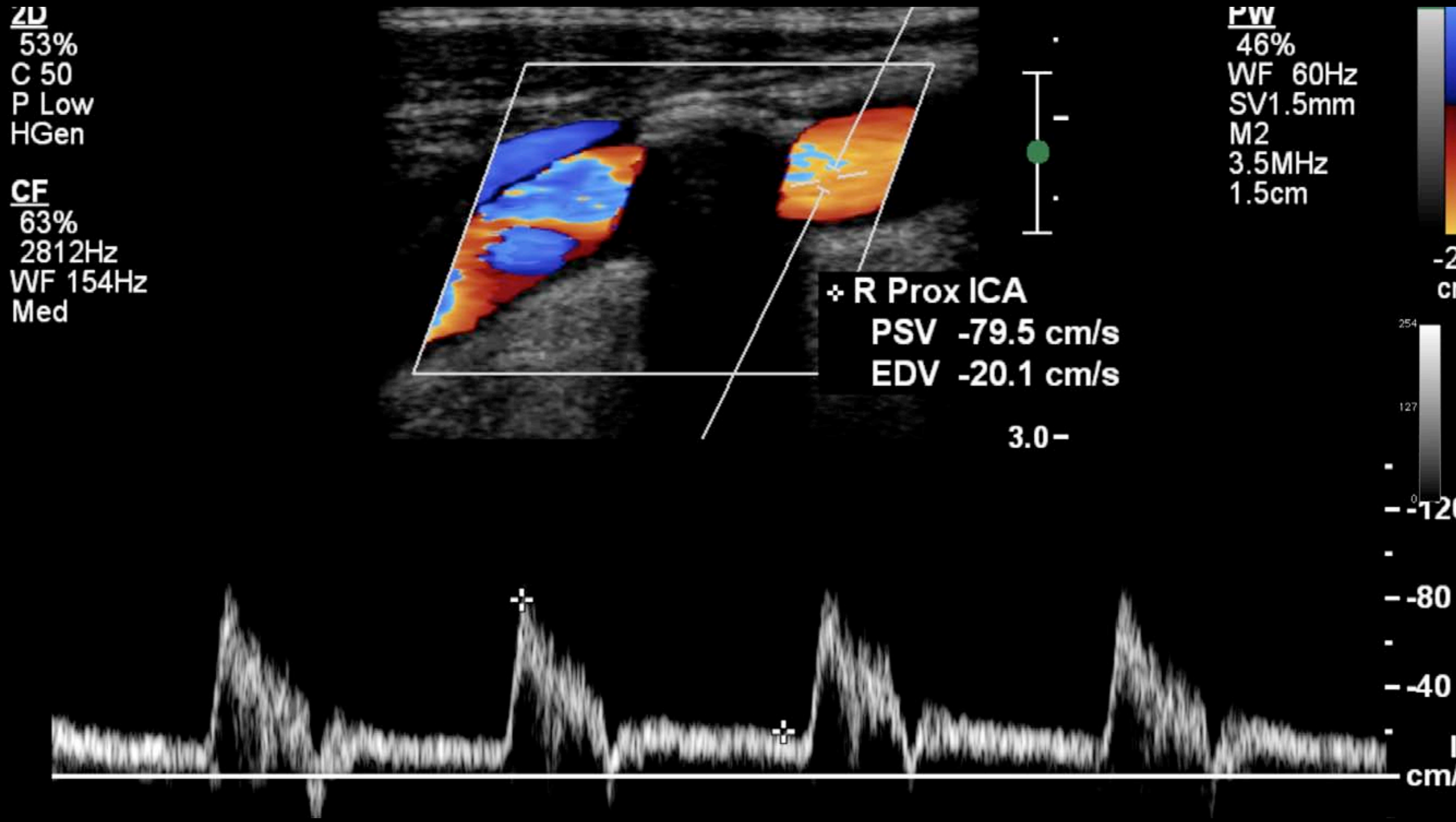
PICA



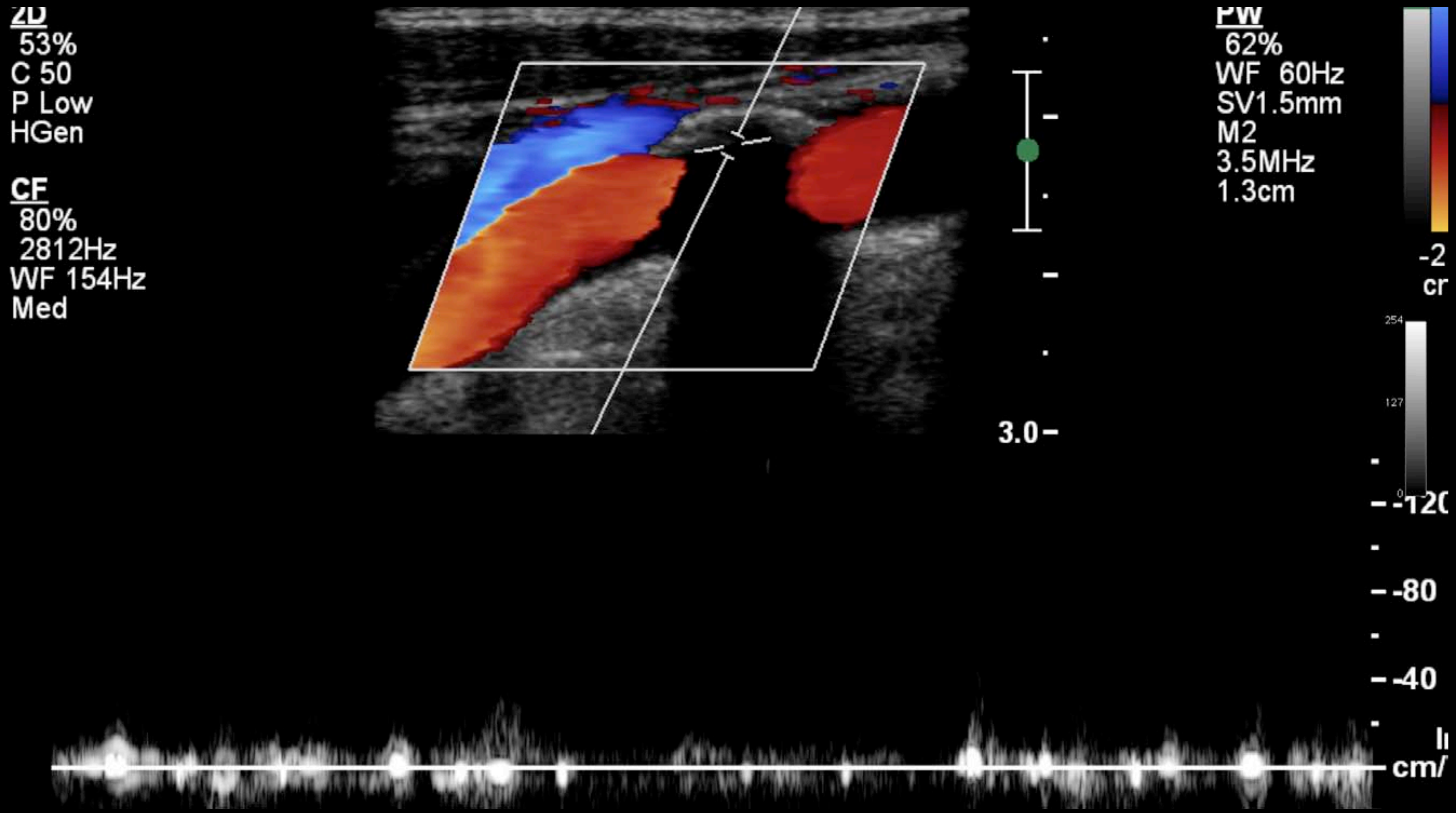
PICA color



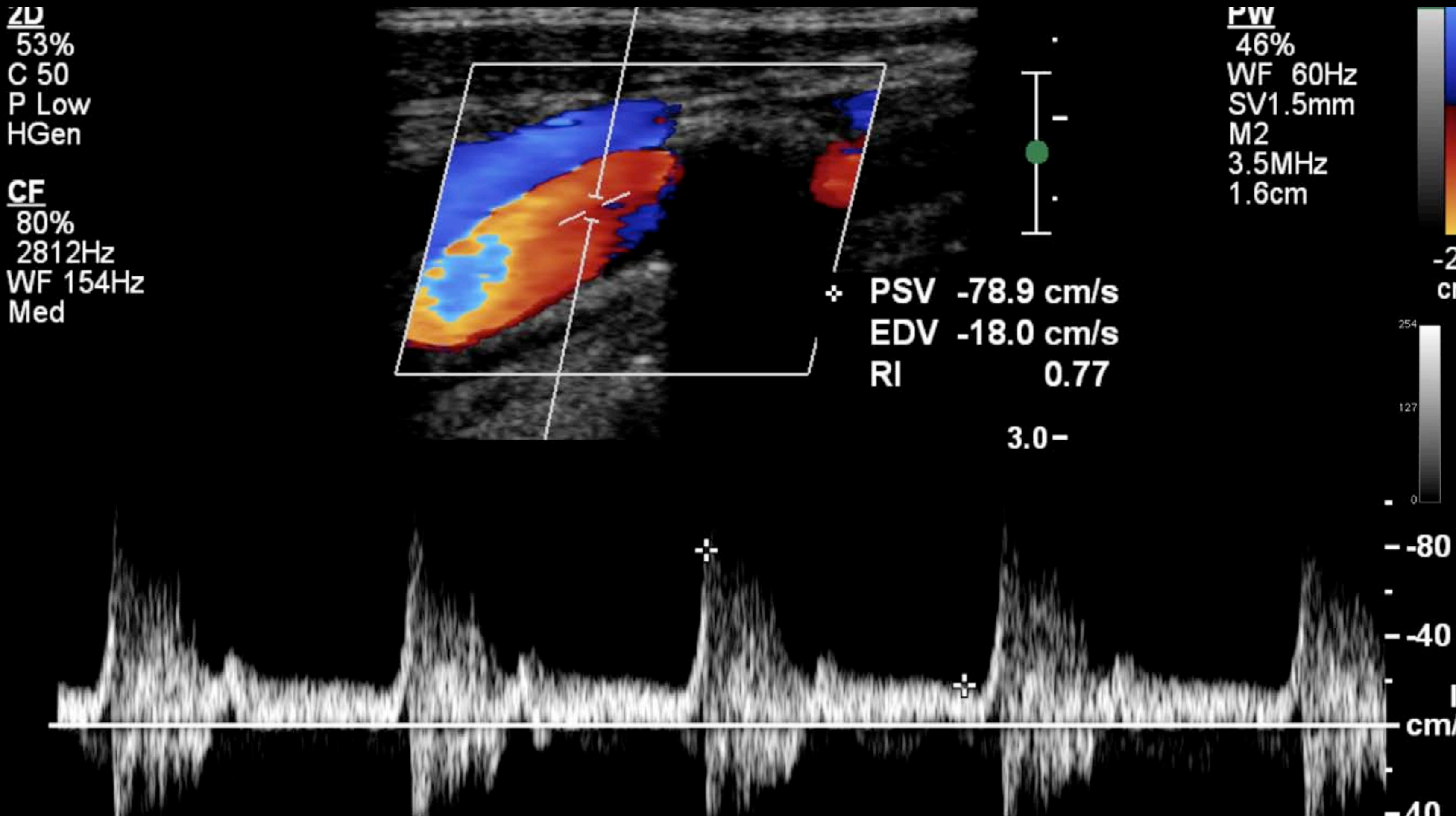
Before calcification



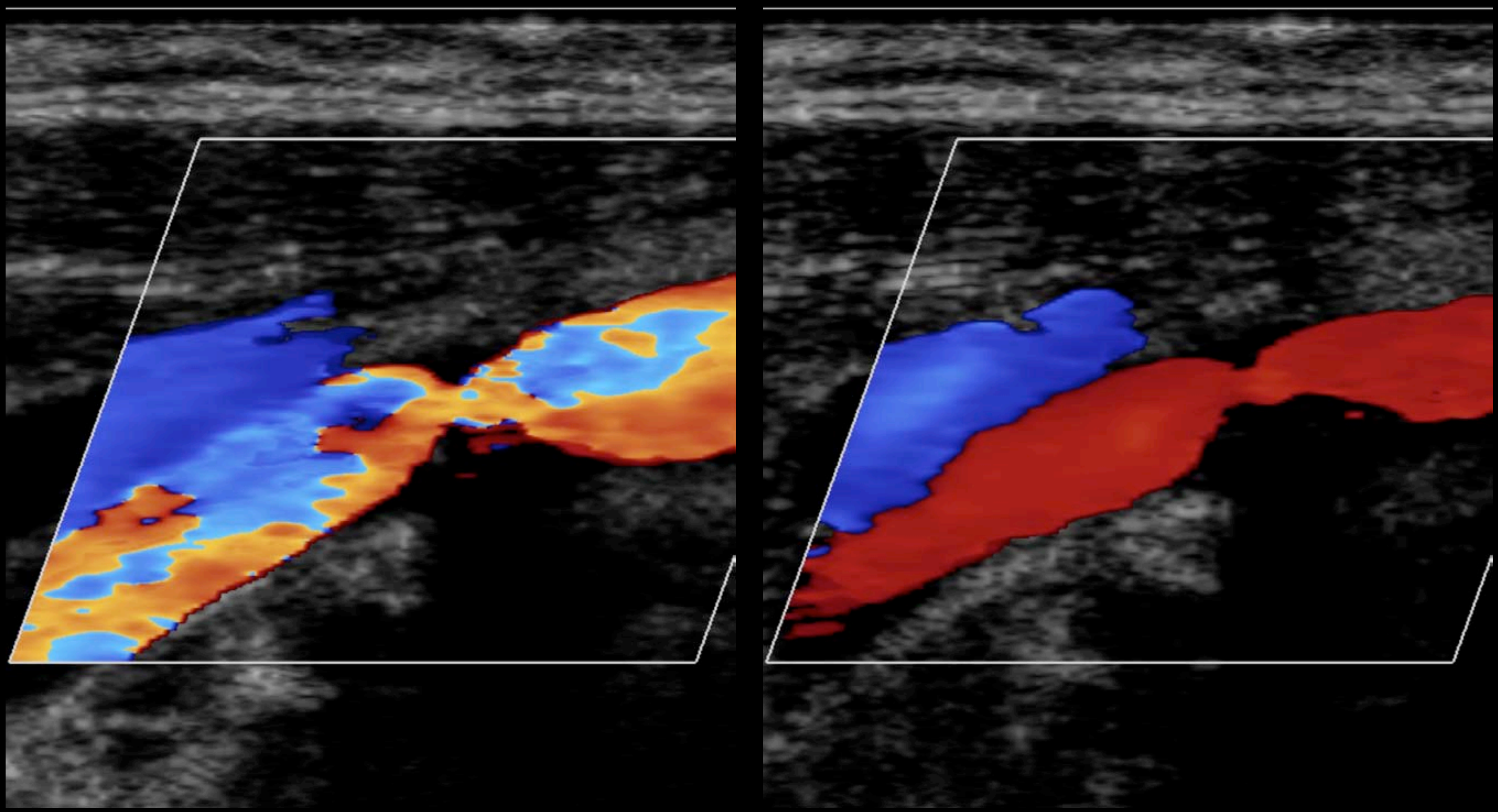
In


2D
53%
C 50
P Low
HGen
CF
80%
2812Hz
WF 154Hz
Med
PW
62%
WF 60Hz
SV1.5mm
M2
3.5MHz
1.3cm
3.0
-120
-80
-40

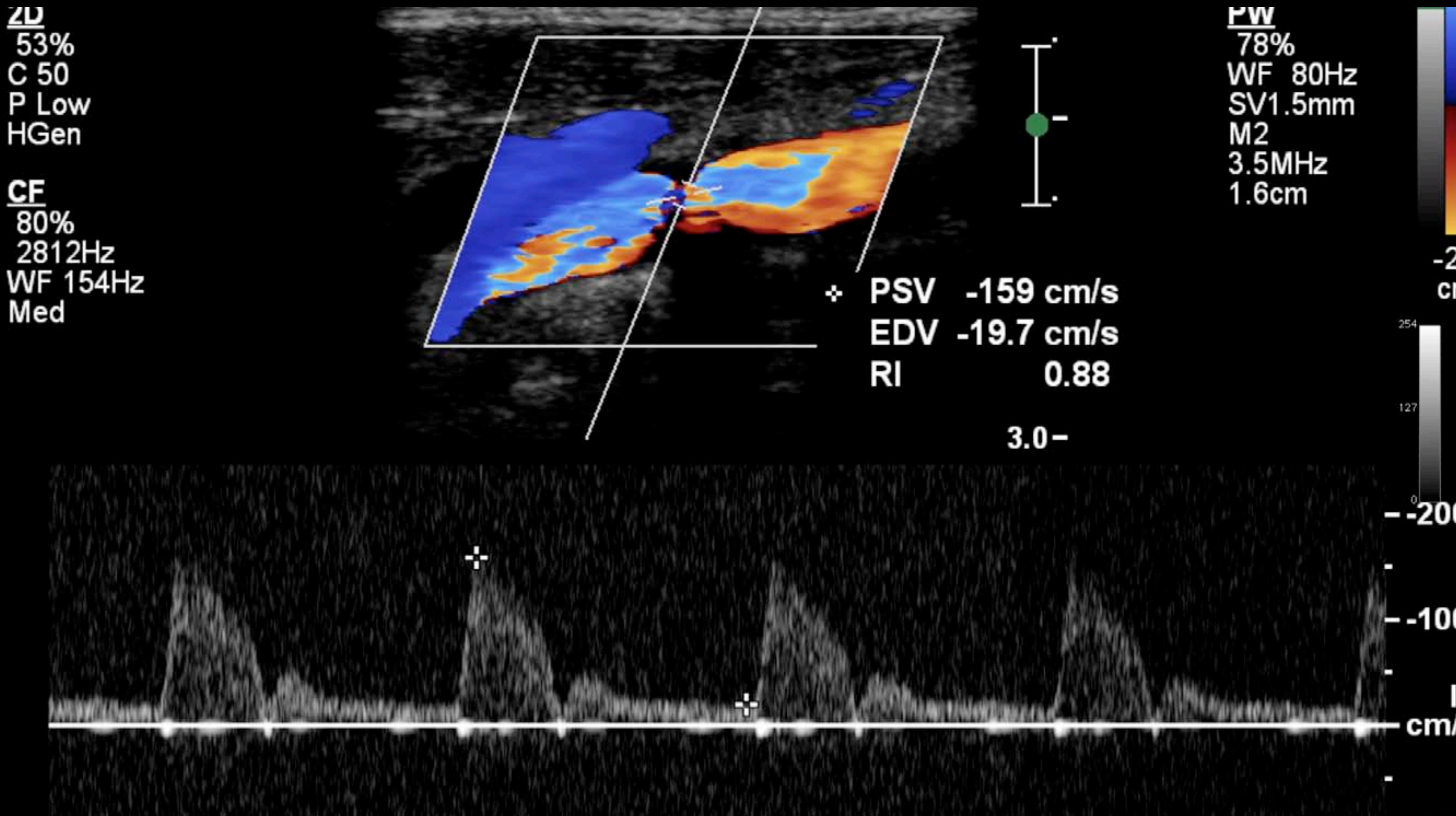
Distal – 79 cm/s

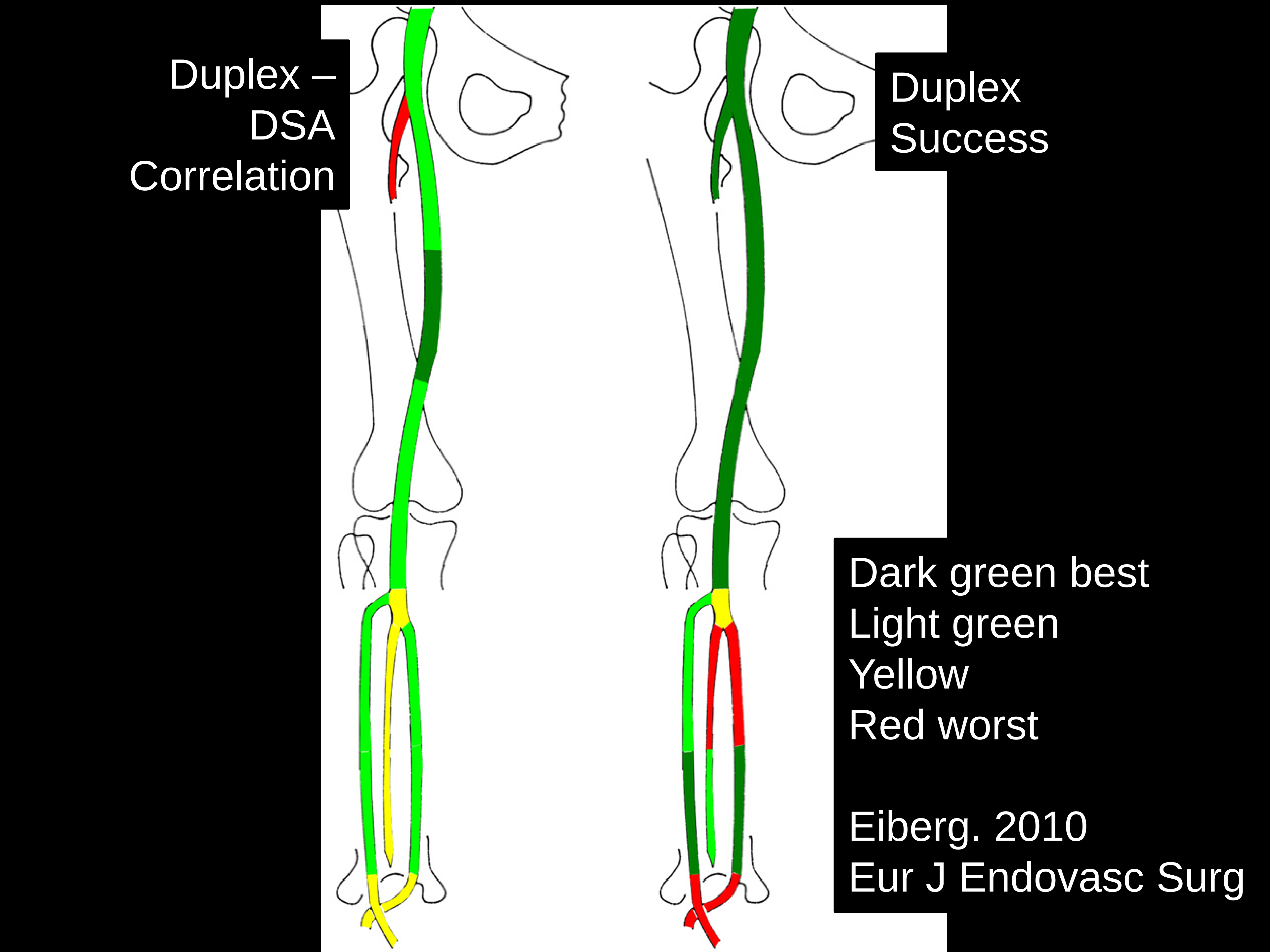


Additional scanning



PSV 159, EDV 20, IC:CC 2




Duplex – DSA Correlation
Duplex Success
Dark green best
Light green
Yellow
Red worst
Eiberg. 2010
Eur J Endovasc Surg

Duplex Arteriography

- 1020 scans
  - Not well visualized
    - Iliac 73
    - Femoral 26
    - Popliteal 17
    - Infrapopliteal 221
  - **Arterial wall calcifications 64**
  - **Poor runoff 18**

Hingorani AP et al. Vascular 2008;16(3) 147-153

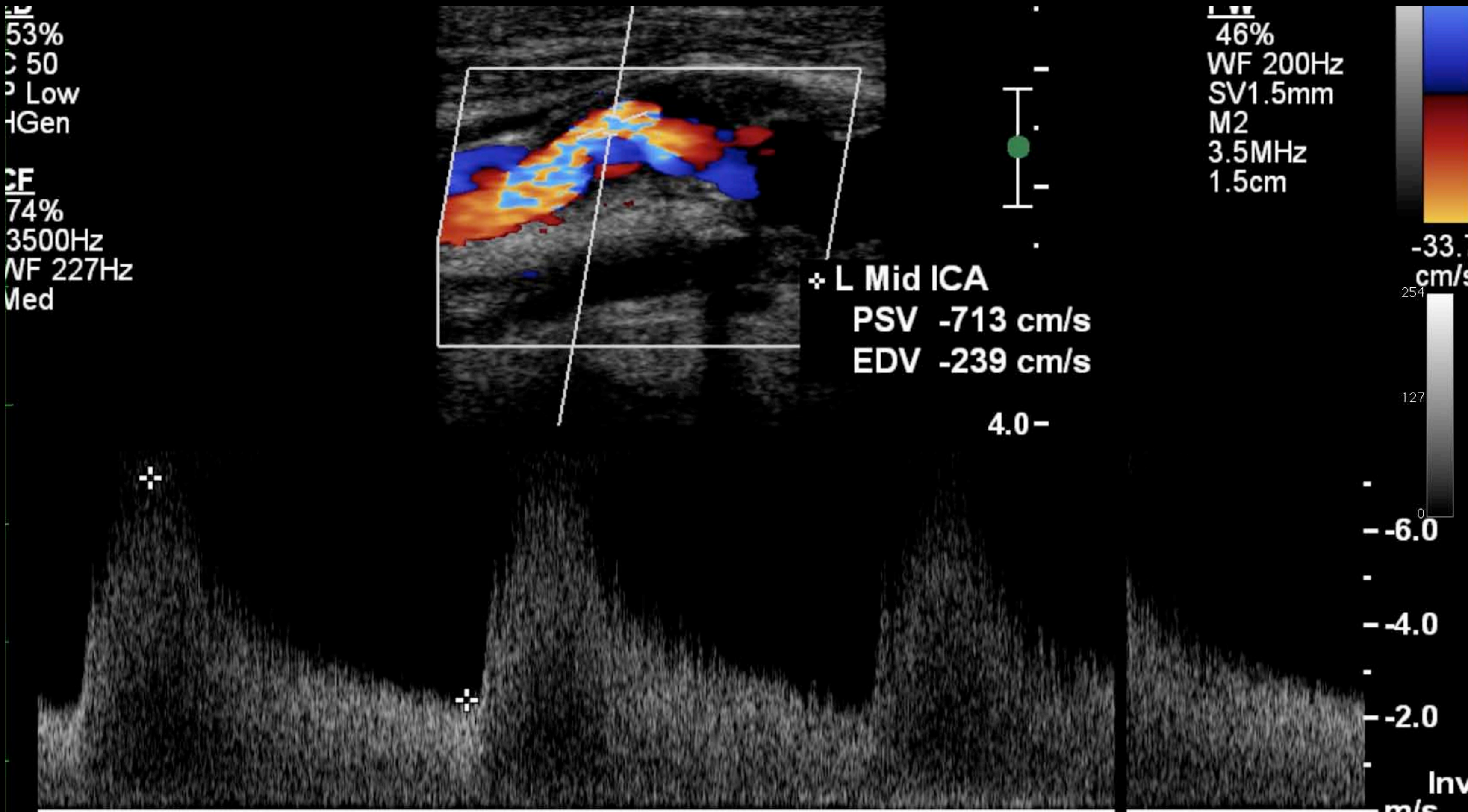
Solutions

- Better sonographers to find best direction
- CTA

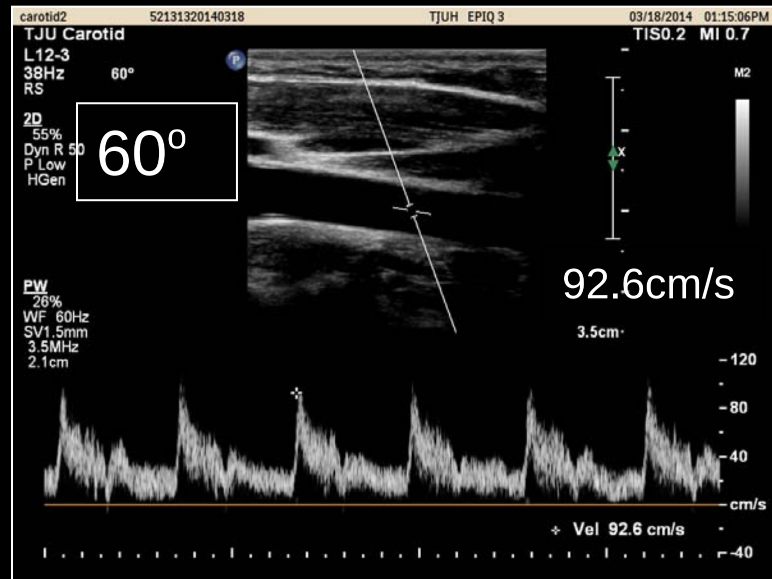
- Future
  - Multiplanar ultrasound
  - Volume acquisition
  - Volume flow (?)
  - Sensitive techniques to low flow

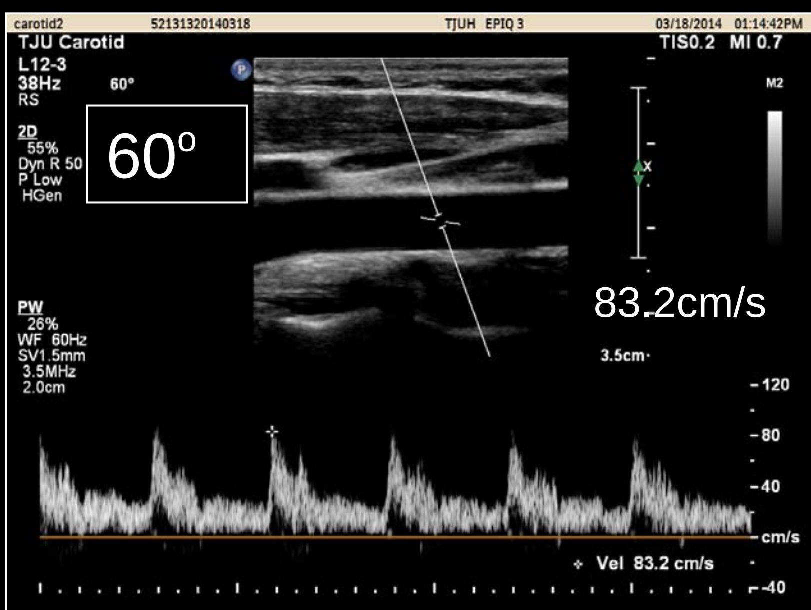
Angle

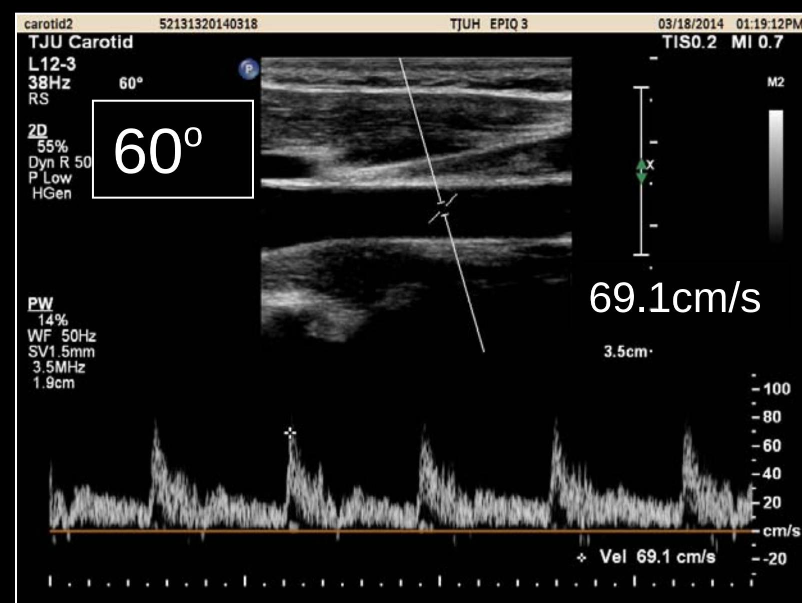
Velocity requires angle correction



60°Angle Correction







Angle errors worse for higher angles

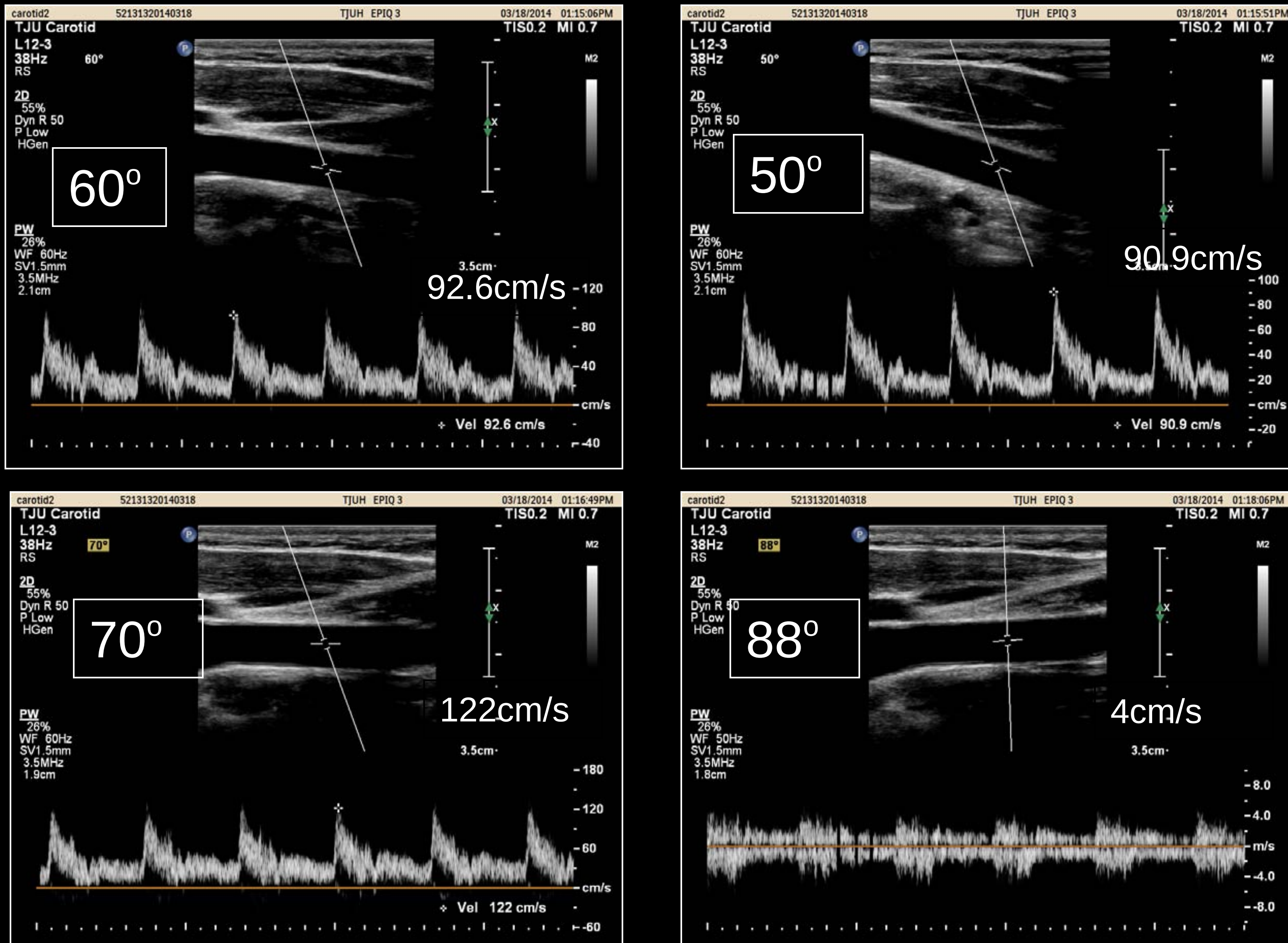
Angle effects: errors

Angle	cosine		%velocity		% pressure	
Degrees	> angle	< angle	over	under	over	under
0	0.99619	0.99619	0.38%	0.38%	0.77%	0.77%
10	0.96593	0.99619	1.95%	-1.14%	3.95%	-2.27%
20	0.90631	0.96593	3.68%	-2.72%	7.50%	-5.36%
30	0.81915	0.90631	5.72%	-4.44%	11.77%	-8.69%
40	0.70711	0.81915	8.34%	-6.48%	17.36%	-12.55%
50	0.57358	0.70711	12.07%	-9.10%	25.59%	-17.36%
60	0.42262	0.57358	18.31%	-12.83%	39.97%	-24.01%
70	0.25882	0.42262	32.15%	-19.07%	74.63%	-34.51%
80	0.08716	0.25882	99.24%	-32.91%	296.96%	-54.99%

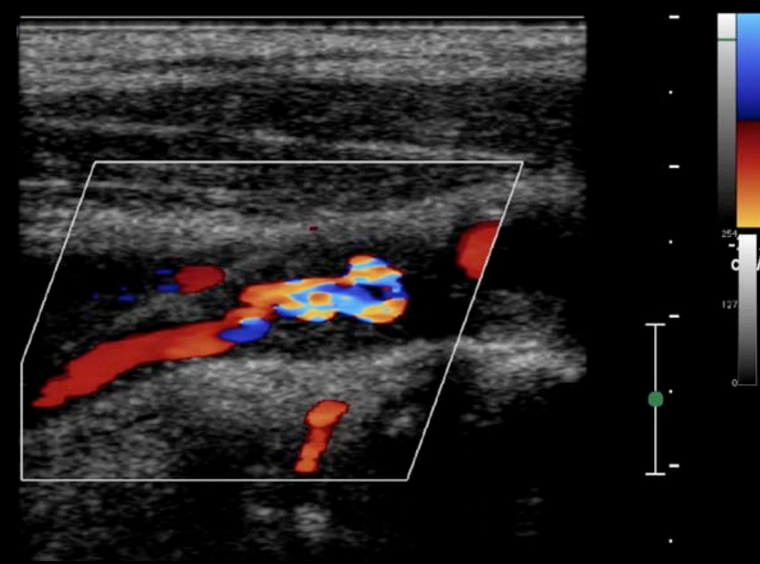
Error associated with incorrect angle specification by ± 5 degrees.

Table courtesy of Frank Miele

Angle Correction



What is angle?



Solutions

- Angle dependent scanning
  - Multidirectional acquisition
- Less reliance on Doppler
  - Gray scale modes

Is carotid ultrasound a public health problem?

Same results in different laboratories give different degrees of stenosis

SRU Carotid Consensus

	Primary Parameters		Additional Parameters	
Degree of Stenosis (%)	ICA PSV (cm/sec)	Plaque Estimate (%)*	ICA/CCA PSV Ratio	ICA EDV (cm/sec)
Normal	<125	None	<2.0	<40
<50	<125	<50	<2.0	<40
50–69	125–230	≥50	2.0–4.0	40–100
≥70 but less than near occlusion	>230	≥50	>4.0	>100
Near occlusion	High, low, or undetectable	Visible	Variable	Variable
Total occlusion	Undetectable	Visible, no detectable lumen	Not applicable	Not applicable

Grant EG, et al. Radiology 2003;229:340.

World Federation Neurology

Table. Combined Criteria for Grading Internal Carotid Stenosis

Degree of Stenosis as Defined by NASCET (%)	Grading of Internal Carotid Stenosis						
	10–40	50	60	70	80	90	Occlusion
Main criteria							
1. B-mode image, diameter	Applicable	Possibly applicable					Imaging of occluded artery
2. Color Doppler image	Plaque delineation	Flow	Flow	Flow	Flow	Flow	Absence of flow
3. PSV threshold (cm/s)		125		230		NA	NA
4a. PSV average (cm/s)	≤160	210	240	330	370	Variable	NA
4b. PSV poststenotic (cm/s)				≥50	<50	<30	NA
5. Collateral flow (periorbital arteries or circle of Willis)				Possible	Present	Present	Present
Additional criteria							
6. Prestenotic flow (diastole) (CCA)				Possibly reduced	Reduced	Reduced	Reduced
7. Poststenotic flow disturbances (severity and length)		Moderate	Pronounced	Pronounced	Pronounced	Variable	NA
8. End-diastolic flow velocity in the stenosis (cm/s)			<100	>100		Variable	NA
9. Carotid ratio ICA/CCA	<2	≥2	≥2	>4	>4	Variable	NA

These criteria do not apply for external carotid stenosis. Note the hierarchical order of main and additional criteria and the change of reliability of each criterion for different degrees of stenosis. Examples how to use this Table are given in the text under "Main and Additional Criteria" and "Advantages of a Multiparametric Approach." For definition of classes of stenosis, see Figure legend.

Criterion 1: Visualization of the ICA by B-mode imaging is needed to prove no flow.

Criterion 4a: Values taken from Figure.

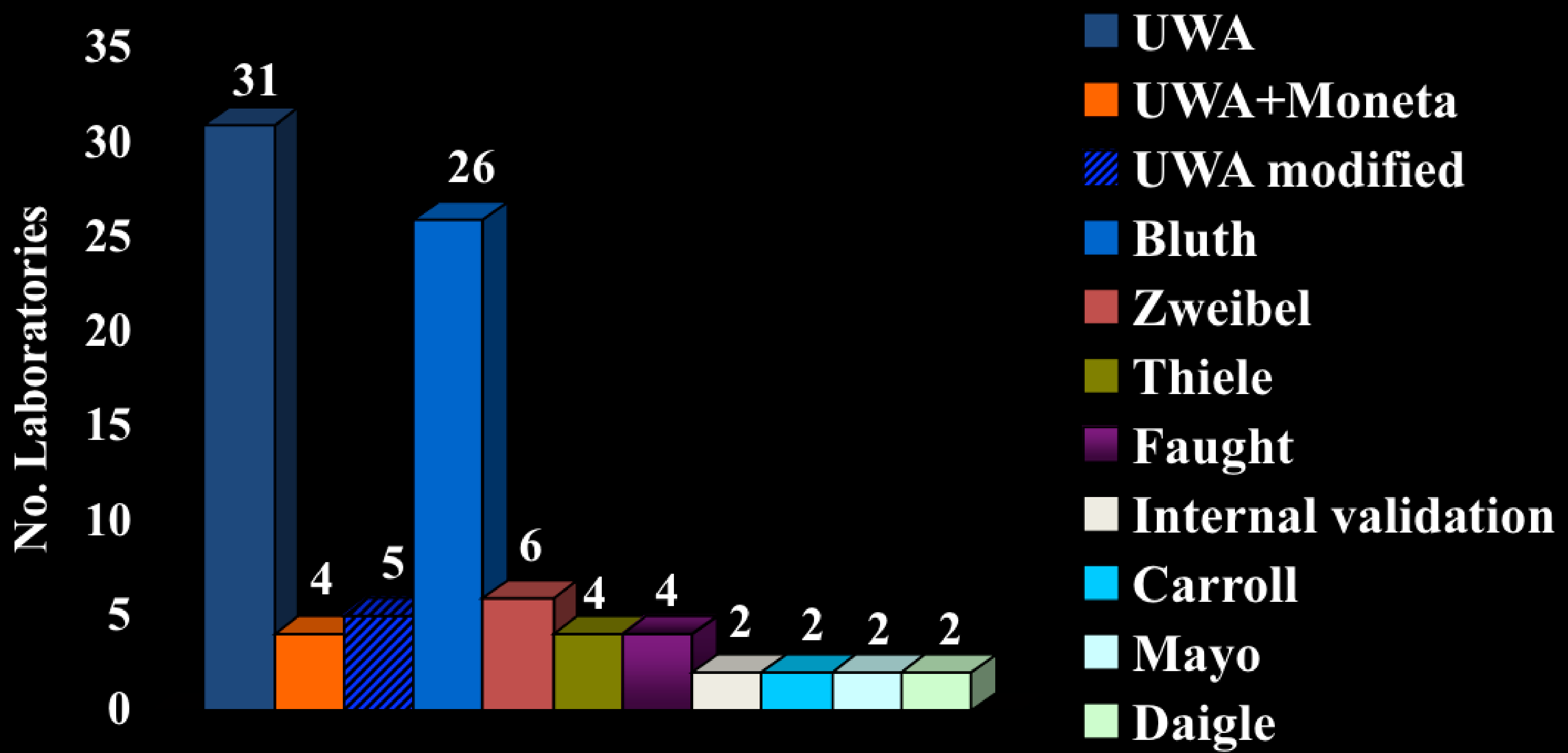
Criterion 4b: Poststenotic indicates measured distal to turbulences. In case of a short neck or a bifurcation in high position, respectively, these criteria are difficult to assess with a conventional duplex technique. Then, a lateral scan head position behind the jaw angle or a scan head with a lower frequency, as for abdominal examinations, may give access to the more distal lying segments of the ICA. Another possibility is the examination of the distal ICA with 2-MHz pulsed Doppler sonography.

Criterion 6: "Reduced" means side-to-side difference, mainly in diastolic velocity.

Modified from reference 11.

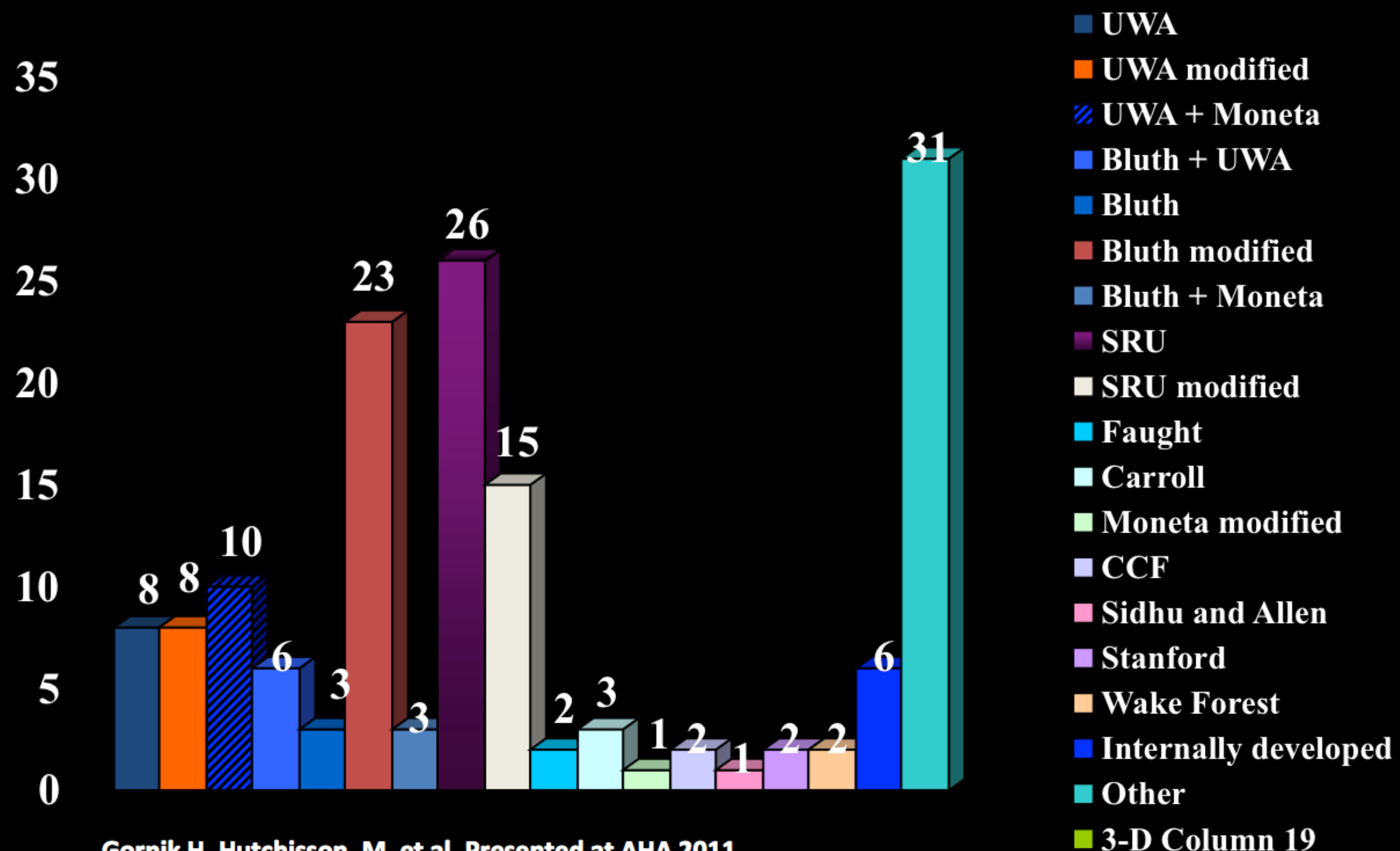
CCA indicates common carotid artery; ICA, internal carotid artery; NA, not applicable; PSV, peak systolic velocity.

2002 ICAVL Survey
100 Vascular Labs
11 Different Criteria



Unpublished data courtesy Ms. Sandra Katanick, Intersocietal Accreditation Commission

2010 ICAVL Survey
152 Vascular Labs;
>16 Diagnostic Criteria



Gornik H, Hutchisson, M, et al. Presented at AHA 2011.

Future directions

Trifurcation of Ultrasound

Point of care	Traditional Ultrasound	Personalized ultrasound (Specialized care)
ER, primary care, some specialists	Radiologists, some specialists	Ultrasound specialist and team
Problem centered, short duration	Protocol driven, more complete, Variable duration	Detailed diagnostic or therapeutic scans, time depends on study but generally long (e.g. biopsy, contrast injection, therapy)
Performed by “doctor”	Performed by sonographer/vascular technologist, presented to doctor	Performed by doctor, usually with assistance (nurse, technologist)

Trifurcation of Ultrasound
Future trends

Point of care	Traditional Ultrasound	Personalized ultrasound
Prettier pictures Few or no buttons Wireless acquisition and storage	Simpler controls Volumetric acquisition Post processing Less dependency on sonographer skill (e.g. smart Doppler, protocols, angle independence, automatic measurements) Flow and pressure	Contrast approval by FDA Drug delivery New modes with better macro and micro vasculature