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 decreased 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

Right CFV PERIPH

Vein

Right CFV PERIPH Comp
Noncompressible Vein: Causes

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

- Soft, deformable with compression
- Enlarges vein
- Smooth
Popliteal vein recurrent thrombosis
Development of DVT depends on baseline risk and risk events.

Normal Thrombophilia
Atherosclerosis timeline

Endothelial dysfunction

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

Adapted from Pepine CJ. *Am J Cardiol.* 1996;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
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 eventually 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 aneurysm

• 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
Atherosclerosis timeline

- Foam cells
- Fatty streak
- Intermediate lesion
- Atheroma
- Fibrous plaque
- Complicated lesion/rupture

Endothelial dysfunction

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

Adapted from Pepine CJ. Am J Cardiol. 1996;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

Decades

- Normal
- Asymptomatic Atherosclerosis

Symptomatic Atherosclerosis:
- Angina
- Myocardial Infarction
- Stroke

Modified from Stary HC et al, Circulation 92:1355, 1995
Coronary artery at lesion-prone location

Type II lesion

Adaptive thickening (smooth muscle)

Intima

Media

Macrophage foam cells

Type III (preatheroma)

Small pools of extracellular lipid

Type IV (atheroma)

Core of extracellular lipid

Type V (fibroatheroma)

Fibrous thickening

Type VI (complicated lesion)

Thrombus

Tissue and hematoma


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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 in one (11.1%).

**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 30 asymptomatic patients and two with only bruises 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 brain 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

Range of Wall Shear Stress Magnitude

- Normal Vein
- Normal Artery
- Atherosclerosis-Prone Arterial Regions
- High-Shear Thrombosis (Complex Plaque, Carotid Wakes, Stents)
Hemodynamic Shear Stress and Its Role in Atherosclerosis

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

For more than a century, hemodynamic forces have been proposed as factors regulating blood vessel structure and influencing development of vascular pathology such as atherosclerosis, aneurysms, poststenotic dilations, and arteriovenous malformations. 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. Shear stress has thus only been shown to be a critical determinant of vessel caliber, but also implicated in vascular remodeling and pathology.

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 geometrical 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.
Plaque
Progress and challenges in translating the biology of atherosclerosis

Peter Libby¹, Paul M Ridker¹,² & Göran K. Hansson³

19 May 2011 | Vol 473 | Nature | 317
Atherosclerosis Timeline

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

Cholesterol Accumulation - Inflammation → Thrombosis

Decades

- Normal
- Asymptomatic Atherosclerosis

Symptomatic Atherosclerosis:
- Angina
- Myocardial Infarction
- Stroke

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

Range of Wall Shear Stress Magnitude

- Normal Vein
- Normal Artery
- Atherosclerosis-Prone Arterial Regions
- High-Shear Thrombosis (Complex Plaque, Cardiac Valves, Stents)
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

Intraplaque Hemorrhage and Blood Flow Velocities


In the plaques that we studied, the regions of hemorrhage were not of uniform “age” according to the pathological analysis. Therefore, more than one hemorrhage event probably occurred.

The results of this study are preliminary, the

where \( \rho = \text{density of fluid} \), \( g = \text{acceleration due to gravity} \), \( h = \text{height of fluid column} \), and \( v = \text{fluid velocity} \). Mechanical energy can be freely exchanged between the two forms, kinetic and potential. In a steady flow, energy is conserved. Pressure at the distal end of the tube is lower than that at the proximal end. The transmural pressure exerted on the wall in the stenosis is lower than at the proximal or the distal pressure. This is because the stenosis is a critical region, where velocity is low, must be converted into kinetic energy density (velocity energy), while past the stenosis, where velocity is high.

If, distal to the stenosis, the tube is shape streamlined, then kinetic energy can be converted back to potential energy in the poststenosis.
MRI of the vulnerable carotid plaque
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, these cells increase 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 occlusive disruption of the microvessels. The ensuing intraplaque hemorrhage tends to weaken the plaque and predisposes 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

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. Lesion echogenicity (class I to IV), degree of stenosis, and...
Inflammation within Carotid Atherosclerotic Plaque: Assessment with Late-Phase Contrast-enhanced US

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

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

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
Before calcification

- R Prox ICA
  - PSV: -79.5 cm/s
  - EDV: -20.1 cm/s

- PW
  - 46%
  - WF: 60Hz
  - SV: 1.5mm
  - M: 3.5MHz
  - L: 1.5cm
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
Angle errors worse for higher angles

### Angle effects: errors

<table>
<thead>
<tr>
<th>Angle Degrees</th>
<th>&gt; angle cosine</th>
<th>&lt; angle cosine</th>
<th>% velocity over</th>
<th>% velocity under</th>
<th>% pressure over</th>
<th>% pressure under</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0.99619</td>
<td>0.99619</td>
<td>0.38%</td>
<td>0.38%</td>
<td>0.77%</td>
<td>0.77%</td>
</tr>
<tr>
<td>10</td>
<td>0.96593</td>
<td>0.99619</td>
<td>1.95%</td>
<td>-1.14%</td>
<td>3.95%</td>
<td>-2.27%</td>
</tr>
<tr>
<td>20</td>
<td>0.90631</td>
<td>0.96593</td>
<td>3.68%</td>
<td>-2.72%</td>
<td>7.50%</td>
<td>-5.36%</td>
</tr>
<tr>
<td>30</td>
<td>0.81915</td>
<td>0.90631</td>
<td>5.72%</td>
<td>-4.44%</td>
<td>11.77%</td>
<td>-8.69%</td>
</tr>
<tr>
<td>40</td>
<td>0.70711</td>
<td>0.81915</td>
<td>8.34%</td>
<td>-6.48%</td>
<td>17.36%</td>
<td>-12.55%</td>
</tr>
<tr>
<td>50</td>
<td>0.57358</td>
<td>0.70711</td>
<td>12.07%</td>
<td>-9.10%</td>
<td>25.59%</td>
<td>-17.36%</td>
</tr>
<tr>
<td>60</td>
<td>0.42262</td>
<td>0.57358</td>
<td>18.31%</td>
<td>-12.83%</td>
<td>39.97%</td>
<td>-24.01%</td>
</tr>
<tr>
<td>70</td>
<td>0.25882</td>
<td>0.42262</td>
<td>32.15%</td>
<td>-19.07%</td>
<td>74.63%</td>
<td>-34.51%</td>
</tr>
<tr>
<td>80</td>
<td>0.08716</td>
<td>0.25882</td>
<td>99.24%</td>
<td>-32.91%</td>
<td>296.96%</td>
<td>-64.99%</td>
</tr>
</tbody>
</table>

Error associated with incorrect angle specification by ± 5 degrees.

Table courtesy of Frank Miele
Angle Correction

- 60°: 92.6 cm/s
- 50°: 90.9 cm/s
- 70°: 122 cm/s
- 88°: 4 cm/s
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

<table>
<thead>
<tr>
<th>Degree of Stenosis (%)</th>
<th>Primary Parameters</th>
<th>Additional Parameters</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ICA PSV (cm/sec)</td>
<td>Plaque Estimate (%)</td>
</tr>
<tr>
<td>Normal</td>
<td>&lt;125</td>
<td>None</td>
</tr>
<tr>
<td>&lt;50</td>
<td>&lt;125</td>
<td>&lt;50</td>
</tr>
<tr>
<td>50–69</td>
<td>125–230</td>
<td>≥50</td>
</tr>
<tr>
<td>≥70 but less than near occlusion</td>
<td>&gt;230</td>
<td>≥50</td>
</tr>
<tr>
<td>Near occlusion</td>
<td>High, low, or undetectable</td>
<td>Visible</td>
</tr>
<tr>
<td>Total occlusion</td>
<td>Undetectable</td>
<td>Visible, no detectable lumen</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Additional Parameters</th>
<th>ICA/CCA PSV Ratio</th>
<th>ICA EDV (cm/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;2.0</td>
<td>&lt;40</td>
</tr>
<tr>
<td></td>
<td>&lt;2.0</td>
<td>&lt;40</td>
</tr>
<tr>
<td></td>
<td>2.0–4.0</td>
<td>40–100</td>
</tr>
<tr>
<td></td>
<td>&gt;4.0</td>
<td>&gt;100</td>
</tr>
</tbody>
</table>

### Combined Criteria for Grading Internal Carotid Stenosis

<table>
<thead>
<tr>
<th>Degree of Stenosis as Defined by NASCET (%)</th>
<th>10−40</th>
<th>50</th>
<th>60</th>
<th>70</th>
<th>80</th>
<th>90</th>
<th>Occlusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Main criteria</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. B-mode image, diameter</td>
<td>Applicable</td>
<td></td>
<td>Possibly applicable</td>
<td></td>
<td></td>
<td></td>
<td>Imaging of occluded artery</td>
</tr>
<tr>
<td>3. PSV threshold (cm/s)</td>
<td>≤160</td>
<td>125</td>
<td>230</td>
<td>NA</td>
<td>NA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4a. PSV average (cm/s)</td>
<td>≥50</td>
<td>&lt;50</td>
<td>&lt;30</td>
<td>NA</td>
<td>NA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4b. PSV poststenotic (cm/s)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Collateral flow (periorbital arteries or circle of Willis)</td>
<td>Possible</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Additional criteria</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Prestenotic flow (diastole) (CCA)</td>
<td>Possibly reduced</td>
<td>Reduced</td>
<td>Reduced</td>
<td>Reduced</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Poststenotic flow disturbances (severity and length)</td>
<td>Moderate</td>
<td>Pronounced</td>
<td>Pronounced</td>
<td>Pronounced</td>
<td>Variable</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>8. End-diastolic flow velocity in the stenosis (cm/s)</td>
<td>&lt;2</td>
<td>≥2</td>
<td>&gt;4</td>
<td>&gt;4</td>
<td>Variable</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>9. Carotid ratio ICA/CCA</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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

Future directions
# Trifurcation of Ultrasound

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

**Future trends**

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