Hemodynamics
Why Do Waveforms Look the Way They Do?

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No disclosures
Doppler Spectrum

- Graphical representation of Doppler

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

“Describe, then diagnose”

- Site of signal
  - What is normal and abnormal?
- Shape (edge, envelope) of spectrum
  - Velocity of blood flow
  - Pulsatility
- Structure (contents) of spectrum
  - Spectral broadening
- Diagnosis
The vascular network

http://www.cvphysiology.com/Blood%20Pressure/BP019.htm
Aortic pressure stays high despite closing of aortic valve
Lots of shapes in the pressure pulse. Lots of squiggles in the Doppler.
Some principles

• Pressure gradient drives flow
  – Actually energy but generally the same
• Waveform shape varies along arterial tree
  – Pressure pulse takes time to go down arteries
• Reflected waves occur at arterioles
Why do parts of Doppler go backward?

• Normal
  – Reflected wave from arterioles reverse flow
  – Eddy currents

• Abnormal
  – Turbulence

• They don’t
  – Machine issue
  – Aliasing
Diastolic Component

- Flow reversal
  - Created by reflected waves from arteriolar branching
  - More resistance ➟ stronger reverse component
Pulsatility
High “resistance” versus “low resistance”
AV Fistula

- Increased diastolic flow
- Turbulence
  - Color or spectral bruit
- +/- Arterial pulsations on draining vein
Arteriovenous Fistula

• Usually traumatic, iatrogenic
• Low pulsatility waveform into AVF (bruit by color or spectrum)
• Arterialized vein (more variable, compare to other side if question)
Vein of Galen “Aneurysm”
Characterization of Spectral Doppler
“Describe, then diagnose”
Pressure Waveforms

- Pulse shape varies through the circulation
- Note
  - Femoral peak later, less diastolic pressure
Which is the correct PSV? Left image or right image
Weird Doppler from the bulb

Original Contributions

Diagnostic Significance of Flow Separation in the Carotid Bulb

Stephen C. Nicholls, ME, David J. Phillips, PhD,
Jean F. Primoizich, RS, Ramona L. Lawrence, BS, Ted R. Kessler, MD,
Thomas G. Rudd, MD, and E. 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 posteroslateral aspect exhibits transient blood flow reversal. It is now possible to document these flow velocity components using pulsed Doppler ultrasound methods. When atheroembolism develops, it preferentially involves the posteroslateral 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 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 crum in two. Flow separation was bilateral in 13 patients (59%). There were 15 patients with symptoms in the territory of a carotid...
Flow separation

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Carotid Bifurcation Atherosclerosis

Quantitative Correlation of Plaque Localization with Flow Velocity Profiles and Wall Shear Stress

Christopher K. Zarins, Don P. Giddens, B. K. Bharadwaj, Vikrom S. Sottilrai, Robert F. Mabon, and Seymour Glagov

From the Departments of Surgery and Pathology, The University of Chicago, Chicago, Illinois, and The School of Aerospace Engineering, Georgia Institute of Technology, Atlanta, Georgia

SUMMARY. The distribution of nonstenosing, asymptomatic intimal plaques in 12 adult human carotid bifurcations obtained at autopsy was compared with the distribution of flow streamline patterns, flow velocity profiles, and shear stresses in corresponding scale models. The postmortem specimens were fixed while distended to restore normal in vivo length, diameter, and configuration. Angiograms were used to measure branch angles and diameters, and transverse histological sections were studied at five standard sampling levels. Intimal thickness was determined at 15° intervals around the circumference of the vessel sections from contour tracings of images projected onto a digitizing plate. In the models, laser-Doppler anemometry was used to determine flow velocity profiles and shear stresses at levels corresponding to the standard specimen sampling sites under conditions of steady flow at Reynolds numbers of 400, 800, and 1200, and flow patterns were visualized by hydrogen bubble and dye-washout techniques. Intimal thickening was greatest and consistently eccentric in the carotid sinus. With the center of the flow divider as the 0° index point, mid sinus sections showed minimum intimal thickness (0.05 ± 0.02 mm) within 15° of the index point, while maximum thickness (0.9 ± 0.1 mm) occurred at 161 ± 16°, i.e., on the outer wall opposite the flow divider. Where the intima was thinnest, along the inner wall, flow streamlines in the model remain axially aligned and unidirectional, with velocity maxima shifted toward the flow divider apex. Wall shear stress along the inner wall ranged from 31 to 600 dynes/cm² depending on the Reynolds number. Where the intima was thickest, along the outer wall opposite the flow divider apex, the pattern of flow was complex and included a region of separation and reversal of axial flow as well as the development of counter-rotating helical trajectories. Wall shear stress along the outer wall ranged from 0 to −6 dynes/cm². Intimal thickening at the common carotid and distal internal carotid levels of section was minimal and was distributed uniformly about the circumference. We conclude that in the human carotid bifurcation, regions of moderate to high shear stress, where flow remains unidirectional and axially aligned, are relatively spared of intimal thickening. Intimal thickening and atherosclerosis develop largely in regions of relatively low wall shear stress, flow separation, and departure from axially aligned, unidirectional flow. Similar quantitative evaluations of other atherosclerosis-prone locations and corresponding flow profile studies in geometrically accurate models may reveal which of these hemodynamic conditions are most consistently associated with the development of intimal disease. (Circ Res 53: 502-514, 1983)
Hemodynamic Shear Stress and Its Role in Atherosclerosis

Figure 1. Hemodynamic Shear Stress

A. Cross-sectional schematic diagram of a blood vessel illustrating hemodynamic shear stress, i.e., the frictional force per unit area acting on the inner vessel wall and on the luminal surface of the endothelium as a result of the flow of viscous blood. B. Tabular diagram illustrating the range of shear stress magnitudes encountered in veins, arteries, and in low-, shear- and high-shear peripheral shears.

Range of Vessel Shear Stress Magnitude

- Normal Shear
- Low Shear
- High Shear

Shear Stress, \( \tau \) in dyn/cm²

Re = 800

70:30
Hemodynamic Shear Stress and Its Role in Atherosclerosis

**Figure 3. Transformation of Endothelial Cell Morphology by Fluid Shear Stress**

**Physiologic Arterial Hemodynamic Shear Stress**

**Pathologic Arterial Hemodynamic Shear Stress**

Bowman's arterial endothelial cells exposed to physiologic shear stress (>15 dyn/cm²) left panel; for 36 hours align in the direction of blood flow while those exposed to low shear stress (right panel) do not (please compare to original magnification × 25). See "Biologic Response of the Endothelium to Shear Stress" section.

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**Hemodynamic Shear Stress and Its Role in Atherosclerosis**

Adel M. Malek, MD, PhD
Seth L. Alper, MD, PhD
Søge Lauritzen, 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. Recent advances in understanding the role of shear stress in the formation of atheromatous plaques have led to the hypothesis that low shear stress (<4 dyn/cm²), which is prevalent in atherosclerosis-prone areas of the arterial system, may play a significant role in the development of atherosclerosis.

Atherosclerosis, the leading cause of death in the developed world and nearly the leading cause of death in the developing world, is associated with systemic risk factors including hypertension, smoking, hyperlipidemia, and diabetes mellitus, among others. Nonetheless, atherosclerosis remains a genetically determined 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 dyn/cm²) induces endothelial quiescence and an anti-inflammatory gene expression profile, while low shear stress (<4 dyn/cm²), which is prevalent in atherosclerosis-prone sites, stimulates an atherogenic phenotype. The functional regulation of the endothelium by local hemodynamic shear stress provides a model for understanding the local propensity of atherosclerosis in the setting of systemic factors and may help guide future therapeutic strategies.
Plaque
Laminar flow: Parabolic vs. Plug flow
Plug versus parabolic flow

Peripheral Artery

Renal Artery
Interior of the waveform

• “Single“ velocity
  – Pathological or physiological

• Spectral broadening
  – Pathological or physiological
<table>
<thead>
<tr>
<th></th>
<th>Single velocity</th>
<th>Many velocities</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Physiologic</strong></td>
<td>Plug flow (large straight vessels)</td>
<td>Parabolic flow (smaller vessels) Curving vessels, vessel origins</td>
</tr>
<tr>
<td><strong>Pathologic</strong></td>
<td>Jet inside a stenosis</td>
<td>Post stenotic disturbed flow and turbulence</td>
</tr>
</tbody>
</table>
Normal Spectral Broadening

- Blood flowing in one direction is laminar
- If many velocities are present, spectral broadening will be present
- Parabolic flow
Abnormal Spectral Broadening

- Spectral broadening
  - Flow in many directions and velocities
- Forward and reverse
- Ill defined edge
- Shift of white to baseline
Stenosis
HYDRODYNAMICA,
SIVE
DE VIRIBUS ET MOTIBUS FLUIDORUM
COMMENTARII.
OPUS ACADEMICUM
AB AUCTORE, DUM PETROPOLI AGERET,
CONGESTUM,

ARGENTORATI,
Sunt inibus JOHANNIS REINHOLDI DULSECKERI,
Anno MD CCXXXVIII.
Bournoulli and Stenosis

Pre-stenosis

Total energy = Kinetic + potential

Potential energy very decreased

Kinetic energy very increased

Stenosis

Post-stenosis

Total energy lower

Potential energy decreased

Kinetic energy very increased
Stenosis

• Focal elevation of velocity in stenosis
  – Jet is high velocity, laminar flow
  – Increased compared to prestenotic segment
  – Velocity should drop off distal to stenosis
    • (exceptions: long stenosis, near occlusive lesion)
  – Measurement of velocity quantifies stenosis
  – Gray scale and color not quantitative, used to locate stenosis
Pressure drops in stenosis
Velocity goes up in a stenosis
Jets are Laminar in a perfect world
The highest velocity may not be laminar
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)
Beyond the Stenosis
Velocity goes down, shape changes

- Disturbed flow distal to stenosis
  - Spectral broadening, loss of well defined spectral edge, forward and reverse flow
- Parvus tardus variable, further distal
Turbulence
3 parts to document stenosis
The “three musketeers”

1. Before stenosis
2. Highest velocity in stenosis
3. Disturbed flow and lower velocity beyond
There are four musketeers

The fourth musketeer is:

1. Aramis
2. Fourth guy
3. D'Artagnan
4. Porthos
Parvus Tardus
Further beyond a stenosis the shape changes and the post-stenotic flow disturbances diminish or disappear.
Parvus tardus (tardus parvus)
Parvus Tardus Waveform
The “fourth musketeer”

- Slow upstroke in systole
- Low systolic velocity (variable)
- Increased diastolic flow
  - Diminished pulsatility
Increased diastole is also seen after a stenosis.
Why high diastole after stenoses?

Vasodilatation is not so important

• Pressure gradient throughout the cardiac cycle

• Weaker poststenotic antegrade pressure ➔
  – Weaker reflected retrograde pressure pulse
  – Forward flow is created by the absence of a strong reflected wave

• Vasodilatation is a minor factor
Monophasic Signals are Abnormal in Peripheral Arteries

Monophasic Staccato
Monophasic Thump
Proximal to stenosis

Monophasic Continuous
Distal to stenosis
Complicated signal simplified

- Multiphasic to monophasic Doppler
- Continuous signals in veins distal to obstruction
  - Could be DVT
  - Also extrinsic obstruction (pelvic mass)
Stenosis = filter

- A stenosis affects the blood flow beyond it in several ways
  - Pressure beyond the stenosis reduced
  - Complicated signals simplified
  - Propagation of blood flow slower
    - Takes longer for the pulse to travel after a stenosis compared with an unobstructed artery
Left = too little variation  
Right = too much variation  

DDX of loss of phasicity and/or asymmetry:  
Venous obstruction, scarring, extrinsic compression
Hepatic vein web
Normal at IVC, blunted before web

Case courtesy of Ron Wachsberg, M.D.
Orchitis

Normal Testis

Orchitis
Nonspecificity of Neovascularity
High velocity without stenosis
Collateralization

The brain is autoregulated to get flow.
When one ICA is severely stenosed or occluded, the other compensates

Flow = area x mean velocity,
Contralateral ICA size is unchanged, flow increases, velocity increases.
Antegrade and reflected pulse make waveform

\[ P(t) \text{ reflected} \]
\[ Q(t) \text{ reflected} \]

\[ P(t) \text{ measured} \]
\[ P(t) \text{ forward} \]

\[ Q(t) \text{ measured} \]
\[ Q(t) \text{ forward} \]

\[ P_m(t) = P_f + P_r \]
\[ Q_m(t) = Q_f + Q_r \]
Without and with exercise
Model of Pressure in a Stenosis
Pressure Recovery

from Frankel and associates, Purdue university
Blood flow travels slower after a stenosis

Steal physiology in vertebrals
  – Also requires both vertebrals becoming basilar
Notches
Valve opens and closes versus QRST
Systole is approximately 1/3 of cardiac cycle

“Notches” occur before dicrotic notch
A Spectrum of Doppler Waveforms in the Carotid and Vertebral Arteries

Fig. 3.—78-year-old woman with severely stenotic aortic valve complicated by aortic regurgitation. Doppler sonography waveforms from arteries (right common carotid artery is shown as example) show bisferious pulse, with prominent mid systolic retraction (arrow) distinct from dicrotic notch (arrowhead). Dicrotic notch is normal finding and is because of closure of aortic valve, temporary cessation of forward flow, followed by resumption of forward flow driven by elastic rebound of aortic wall. Mechanism of pulsus bisferiens in aortic insufficiency is not well understood. One view is that first peak represents initial high-volume ejection of blood, which is followed by abrupt mid systolic flow deceleration caused by regurgitant valve, and second peak represents tidal wave reflected from distended aorta as it relaxes or from periphery of body. Others argue that rapid ejection of large volume of blood (increased preload of left ventricle) creates transient suction (Venturi) effect in aorta, which in turn produces mid systolic retraction in carotid artery waveform.
AR
A Unified Mechanism for the Water Hammer Pulse and Pulsus Biferiens in Severe Aortic Regurgitation: Insights from Wave Intensity Analysis

Julia A Chirinos, MD, Scott R. Akers, MD, PhD, Jan A. Vlietendaal, PhD, and Patrick Segers, PhD

Doppler ultrasound and arterial applanation tonometry allow for the measurement of time-resolved flow velocity and pressure waveforms, which can be obtained at the same location (such as peripheral arteries) for the assessment of forward- and backward-traveling waves via WIA. High-fidelity carotid and radial applanation tonometry and pulse-wave

We assessed the pulsatile hemodynamic phenomena underlying our patient's pulse patterns via WIA, a novel technique applied to arterial hemodynamics, in which wavefronts are assessed with simultaneous measurements of local pressure and flow (Figure 1) in order to determine the intensity, direction (forward vs. backward) and type (compression vs. suction) of waves traveling in arteries. WIA is a time-domain method in which waves are regarded as small wave incremental fronts. Measured arterial waveforms are decomposed into successive wavefronts, which act to increase or decrease pressure or flow, and travel either forward or backward. Waves can be characterized as forward or backward traveling of the compression or suction type, based on their simultaneous effect on pressure and flow.

Forward-traveling waves change pressure and flow in the same direction (increase or decrease), whereas backward-traveling waves change pressure and flow opposite directions (either increase pressure and decrease flow, or decrease pressure and increase flow). Compression waves increase pressure, whereas suction waves decrease pressure. Therefore: (a) forward-traveling compression waves increase pressure and flow; (b) forward-traveling suction waves decrease pressure and flow; (c) backward-traveling compression waves increase pressure and decrease flow; (d) backward-traveling suction waves decrease pressure and increase flow.

FIGURE 2. Applanation tonometry is performed by placing a pressure sensor over the radial artery. Shown is the SphygmoCor device (AtCor Medical, Sydney, Australia).
Wave Intensity Analysis

Figure 1. Carotid (A–C) and radial (D–F) pressure (top panels, A and D), flow (middle panels, B and E) and wave intensity (C and F). In C and F, forward-traveling waves are plotted as positive intensity, whereas backward-traveling waves are plotted as negative intensity. The vertical dashed lines in C and F mark the duration of ventricular ejection after correcting for wave transmission delays to the respective measurement sites. The carotid pulse is histotonic, due to an abnormal forward-traveling suction wave in mid-systole (green area in C), which follows the normal forward compression wave (orange area) generated by ventricular contraction. The radial pulse (D) has a “water hammer” configuration, with a sharp upstroke.
Oates: 180 ms or so for reverse wave to reach AV
Summary

• The final Doppler shape in the vessel you insonate is created by many factors
  – The heart and all vessels before it
  – Local effects such as arteriolar resistance or stenoses
  – Distal effects such as arteriovenous connections, neovascularity and vasodilatation