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Special Report

Flow Dynamics for Radiologists
II. Practical Considerations in the Live Human

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In reviewing original work on circulation, one finds that many early fluid dynamicists were also physicians. For example, the Greek physician Galen, writing in the second century, was the first to correlate pulse changes with patient health (1). In 1628, another physician, William Harvey, made the important observation that blood actually circulates rather than ebbs and flows (2). Young, in 1808, derived the velocity of propagation of the pulse wave in blood and also made remarkably precise estimates of the pressure drop caused by viscous losses in the arteries (3, 4). Gradually, as science became more specialized, and medicine and the physical sciences moved apart, physicians became less involved in the study of fluids.

During the past three decades the field now called biological fluid dynamics (or biorheology) has expanded logarithmically, but that expansion of knowledge has been primarily generated by engineers, physicists, and physiologists (5–10). Given the current interest in angiographic interventional techniques and the ability of magnetic resonance (MR) scanning to show dynamic flow, it seems timely and prudent for physicians, especially radiologists, to reenter the field.

But the world of the scientific laboratory, with its ability to control experiments and measure precisely, bears little resemblance to the hectic and sometimes messy angiography suite. In addition, human body fluids are more complex than the inert materials used in most scientific studies.

When we compare our analytic problems with those that engineers face, significant differences become immediately apparent. First, flow in the body is not constant, but rather is pulsatile and moves with a complex waveform. Second, the fluid that we deal with contains particles and is viscoelastic rather than Newtonian. Third, the arteries are viscoelastic, too. Moreover, arterial walls are not strictly parallel; the walls taper. Arteries bifurcate frequently, and vessels course through the body in compound, three-dimensional curves. To complicate matters even more, arteries are lined by relatively rough endothelia. They become diseased, develop plaques, and lessen their elasticity, thus altering the shapes and positions and diameters of their lumens. Furthermore, the plaques may ulcerate. Finally, the flow of blood in the body occurs in the range in which flow characteristics are neither completely smooth nor completely turbulent.

We can begin to sort out this complexity in patients by studying the dynamic interaction of the system's three fundamental components: the pump, the fluid, and the fluid's container.

The Pump: The Heart and Great Vessels

For our purposes we need not be cardiologists; it is the heart's velocity and volume profile that is important to us. The heart is not the only engine that causes the forward flow of blood; the heart and proximal great vessels act as a unit. The heart's output is a rapid gush that stops abruptly at the end of systole. At the root of the aorta some true turbulence probably exists (11–13). (This seems to be the only place in the body where turbulence is found in the absence of disease.) Central stream velocities at the normal aortic root are a remarkable 100 to 200 cm/s (13).

When the left ventricle ejects an aliquot of blood, part of the energy is transferred to the elastic arterial walls, which expand radially and store energy. When systole ends, the aortic valve closes, and there is some backward flow of blood toward the heart. This biphasic flow wave can be

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seen as far away as the femoral arteries (Fig 1A). When the aortic valve closes and the blood rebounds against it, the stored energy in the great vessels acts on the blood column, and as the great vessels contract, their energy continues to propel the column of blood forward.

If the circulatory system were a simple series of tubes, the pressure wave recorded at the aortic root would be measurable in the veins, although at diminished amplitude because of the resistances it has encountered. Such is not the case. Specific vascular beds have their own acceptance characteristics. In the femoral artery we see persistence of the retrograde flow pattern in diastole. The external carotid artery, however, has no retrograde (or biphasic) flow; flow into its branches falls to zero during diastole (Fig 1B). The internal carotid artery continues to carry a large volume of blood antegrade during diastole, and ultrasonography routinely shows that diastolic velocities are 30% to 40% of the peak flows of systole (Fig 1C). This flow differential is important if a catheter tip delivering particles into the external carotid artery lies near the bifurcation.

Flow and Its Indirect Indicators

This is a good point at which to consider the differences between *pulse*, *pressure*, *velocity*, and *flow volume*. Especially when dealing with the brain, it is flow volume per time that relates most directly to neural function. The problem is obvious; it is difficult to get real-time flow information in the live human, so we have historically depended on indirect methods to assess blood flow.

The *pulse* is a high-speed wave that originates with systolic ejection. The wave is modified by many influences, but principally by the arteries' elasticity and tethering, and by the wave's reflection retrograde from junctions and bifurcations.

Flow information *cannot* be obtained from a pulse wave. For example, ocean waves still run in toward the shore at 1 to 2 m/s—even though the tide flow is going out. In other words, flow may be toward or away from the direction of a wave. So, too in an artery. An artery may be totally occluded distally, but the palpating finger may feel a pulse.

Historically, after palpating the pulse, measuring the *pressure* of the blood was the next means of assessing flow indirectly. Determining the height of a column of fluid was the first method of blood pressure measurement (14), and a mercury column remains a convenient way to find a patient's blood pressure today. However, pressure is not flow. Certainly, if there is no pressure in the arteries, there is usually little or no flow, but again, the converse is not necessarily true. We can measure a positive pressure in completely occluded arteries (at least until clotting occurs) or in arteries with scant distal runoff. For flow to occur, there must be a difference in pressure from the proximal end of the artery to the distal. Surprisingly little change in pressure is needed to make blood flow. For example, there is a pressure difference of only 6 to 8 mm Hg driving the blood from the origin of the internal carotid artery to the middle cerebral artery (15). Our arterial tree is indeed an efficient system.

Studying *velocities* brings us closer to our goal of finding flow volume. The recent development of Doppler ultrasonography has given clinicians

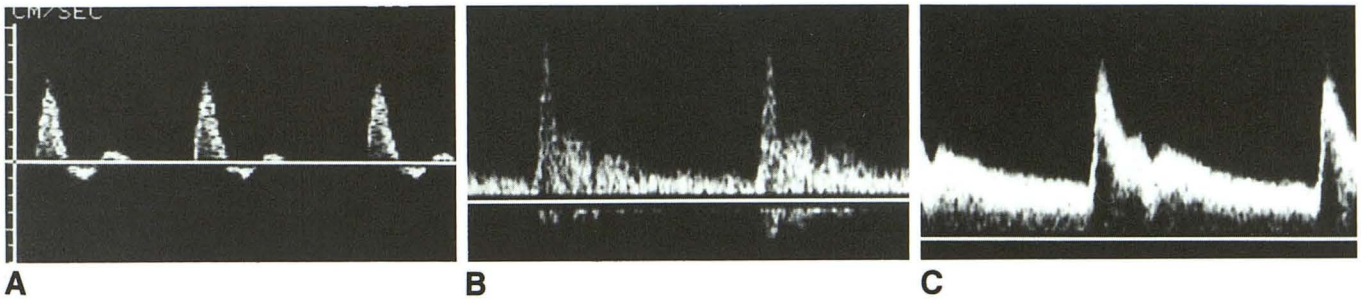


Fig. 1. Ultrasound gray-scale Doppler images of a healthy subject.

A, Femoral artery. Flow velocity rises rapidly during systole and then almost as rapidly falls below the baseline as blood rebounds back toward the heart. As the cycle progresses, the flow returns to baseline.

B, External carotid artery. After a sharp rapid rise to about 60 cm/s, the velocity falls smoothly essentially to baseline at the end of diastole, with no biphasic or retrograde flow wave back toward the heart.

C, Internal carotid artery. Just centimeters away from the external carotid artery, this vessel feeds a different vascular bed. The brain's arteries' resistance accepts blood throughout diastole. Even late in the cycle, the velocity remains 30% to 40% of peak systolic velocities.

an accurate and noninvasive method to see real-time velocity profiles in arteries accessible to the probe. This technique has been especially valuable in the cervical carotid arteries, in which the effects of atherosclerosis on velocity have been well correlated (16–19). Ultrasound correlates velocity to degree of stenosis well.

Extrapolating flow volumes from velocity profiles is difficult and is calculated in only a few ultrasound laboratories. Still, ultrasonography of arteries in the ultrasound laboratory is a valuable procedure. Unfortunately, those with little ultrasound experience may not appreciate the richness of information that can be gained by moving an ultrasound probe across an artery and seeing in real time the variations in velocity profiles throughout the cardiac cycle.

Direct *flow* measurements can be obtained, but the devices are invasive and require insertion of a catheter into the vessel. Flow-measurement catheters depend on some variation of the Fick principle and are, by neuroradiologic standards, large and stiff. Some researchers (20) have attempted to measure flow during angiography by video densitometry, but again, the techniques have not been adopted widely by radiologists.

MR offers promise in being able to show flow profiles not only in vessels in general, but also during a single cardiac cycle. MR now can show individual slipstreams (Fig 2A), and determining flow volumes from MR data is an area of active and important research (21–27). Similar slipstreams are recognized during conventional angiography, too (Fig 2B).

Blood is composed of a fluid—plasma—and particles—the blood cells. The plasma itself is a colloidal fluid containing proteins, chiefly albumin, which gives the plasma some of its viscous properties; and solutes, chiefly sodium, chlorine, and bicarbonate ions. The other plasma constituents contribute little to blood's rheologic properties, except the clotting component, which has the desirable property of changing liquid blood to a solid within the context of certain injuries. The concentration of red cells is highly important, because it is the prime determiner of blood's viscosity.

The portion of blood that gives it its peculiar flow properties is the mass of red cells. Individual cells are able to deform to pass through capillaries under the influence of pressure or shearing stresses. However, the majority of the complex rheologic properties of blood come from its ability to form and break up large aggregates of stacked cells called *rouleaux*.

The viscosity of a Newtonian fluid such as water or isotonic saline solution remains constant even if it has a deforming or accelerating force placed on it, or, more accurately stated, when it has a shear force applied to it. Blood, however, is not Newtonian. At higher shear rates, as when it passes a partial obstruction or when the vessel makes a bend, blood decreases its viscosity because the shear breaks up the rouleaux. The smaller the rouleaux, the lower the viscosity. It thus behaves as a *thixotropic* liquid.

Blood flowing in large vessels is generally considered to behave in a Newtonian fashion. Certainly the majority of flow in large vessels occurs under conditions in which inertial forces predominate. Blood in the carotid artery has an average Reynolds number of about 200. But that Reynolds number is an *average* calculation. Looking at the velocity profile (see Part I of this special report), we see areas near vessel walls, regions adjacent to bifurcations, and flow near plaques in which velocities are low. There, viscous forces predominate. As we will see below when we study bifur-

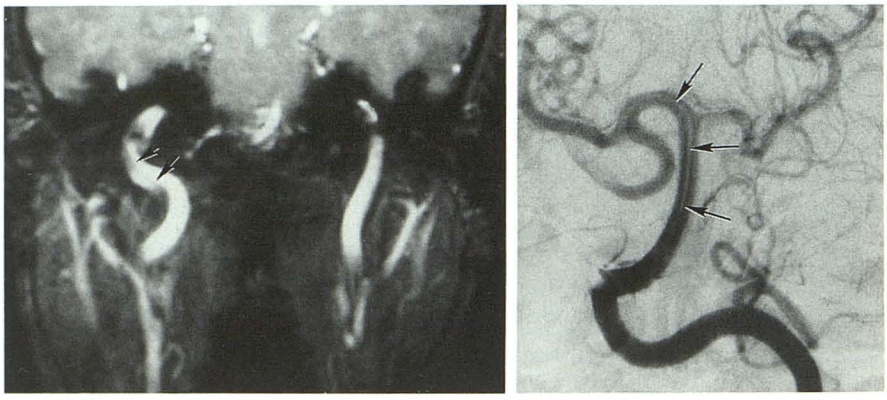


Fig. 2. *A*, This gradient-echo (33/12/4 [repetition time/echo time/excitations], 60° flip angle) frontal image shows the swirling pattern of individual slipstreams (*arrows*) in both internal carotid arteries as the blood passes through the distal cervical internal carotid artery into the petrous internal carotid artery.

B, Slipstreams are occasionally seen during angiography, too. This late arterial phase of the angiogram shows some unopacified blood passing through the contrast agent outlining one slipstream (*arrows*). However, this is not physiologic information, because contrast agent is hyperbaric. Using angiography to get physiologic data must wait until the invention of isobaric contrast agents.

cations, there are areas of actual flow reversal, or slow flow in certain regions of a bifurcation. Liepsch has shown that blood's non-Newtonian properties become important in such regions (28).

Blood's Kinetic Energy

Although blood's primary function is the transfer of oxygen, metabolites, and carbon dioxide, we must consider its action with a broader perspective. As a flowing fluid with mass, it does have energy, and that energy can do work. Whether the work is useful or damaging to the organism depends on how the energy of the slipstreams is channeled. Stehbens has shown that pathologic states that increase flow volumes and velocities lead to degeneration of vessels and to the formation of berry aneurysms (29–31). Flow dynamics has long been considered a prime factor in atherogenesis (32–37), but the causes of the disease are not yet known. At our present state of knowledge, even respected researchers cannot agree whether increased shear rates (38, 39) or low shear rates (40) cause atherosclerosis.

The Container: Arteries

The composition and location of the arteries make them difficult to study. Arteries can be seen clearly only on the retina. Otherwise, they are contained within tissues that are generally opaque. Problems with directly observing arteries in the cranial vault are especially severe, because they change course frequently and pass through dense bone. Moreover, arteries are not optically clear. Only with the smaller arteries can we visualize the path of the flowing fluid within them (41). Arteries have properties completely opposite those of the pipes the engineers study; arteries are viscoelastic, and as they deform, angles at bifurcations change (42–44). The situation is made even more complex because arteries taper in diameter about 2% as they pass distally, bifurcate frequently, are composed of compound curves, and are lined by relatively rough endothelium that, with time and disease, becomes even rougher and even may ulcerate.

The Anatomic and Functional Makeup of Arteries

The pathologist analyzes arteries anatomically. Under the microscope, arteries have three layers: an adventitia, a muscularis, and an intima. The rheologist sees them in a different light, asking how they hold their shape under different flow velocities and under increasing pressures. Roach and Burton (45) have shown two functional passive components of arteries. At relatively low pressures the arteries expand in response to their elastic component. Above a certain pressure elastin's limit is overcome. Then the resistance to expansion comes from the much stiffer collagen. Beyond these two intrinsic and passive properties, the overall vascular tone is modified by the autonomically controlled muscle fibers within the walls. The three functional components acting in concert give arteries the property *viscoelasticity*. The elastic element returns the artery to baseline size when the stress is removed; the viscous element results in a property called *creep*, which allows the material to deform over time.

The Function of Arteries

An artery's rheologic function is to resist the radial pressure of the blood, to be the conduit for the delivery of blood to the periphery, and to do so with as little energy loss as possible.

Control of blood pressure is the function of the smallest arterial vessels. These controlling vessels are in the 200-μm range and smaller, about the size of striate vessels. It is a remarkably energy-efficient delivery system, and it is a rapidly compliant one, too. Just imagine the pressure change in a giraffe's head as she stops drinking and raises her head to browse on treetop leaves, an excursion of 17 feet, or a hydrostatic change of some 380 mm Hg, with homeostatic control in seconds.

Aging

As the individual ages, the arteries stiffen, often dilate, and become tortuous. More elastic (younger) vessels damp the pulse wave and pressure wave more effectively than stiffer older vessels. Aging increases the pulse wave velocity, widens the systolic-to-diastolic pressure ratio, and causes even more reverse flow at bends and bifurcations (28).

Cell Damage and Blood's Velocity

In normal arteries there is little shear stress (or drag) on the endothelium, because the blood near the wall moves little. If a plaque significantly narrows the vessel lumen, however, the higher velocity of the stream increases the shear stress

to a point at which actual endothelial cell damage may occur (38, 46, 47). Rapid velocities usually also damage platelets and activate certain chemical reactions (48–53). When combined, the effects on both often lead to local and downstream clots.

It is less appreciated that too slow a flow in an artery causes clotting even in the absence of injury. A common example is a lenticulostriate artery which supplies a deep arteriovenous malformation. Normally a 50- to 100-μm vessel, this artery may have enlarged tenfold. Then, when the distal arteriovenous malformation nidus is occluded by embolization, the large artery carries only the required scant blood to the capsule. Commonly, the entire vessel clots down to the point of rapid runoff, the middle cerebral artery, resulting in a capsular stroke.

Practical Applications

With these basic concepts in mind we now can explore three questions critical to our understanding of human arterial flow dynamics. First, what happens to flow when vessels curve; second, what happens when they branch; and third, what happens when they become narrowed by disease or are modified by the insertion of our catheter? With this knowledge, we should be able to manipulate the dynamics to keep our devices where they belong, to maximize patient safety, and perhaps even to understand why blood vessel diseases occur.

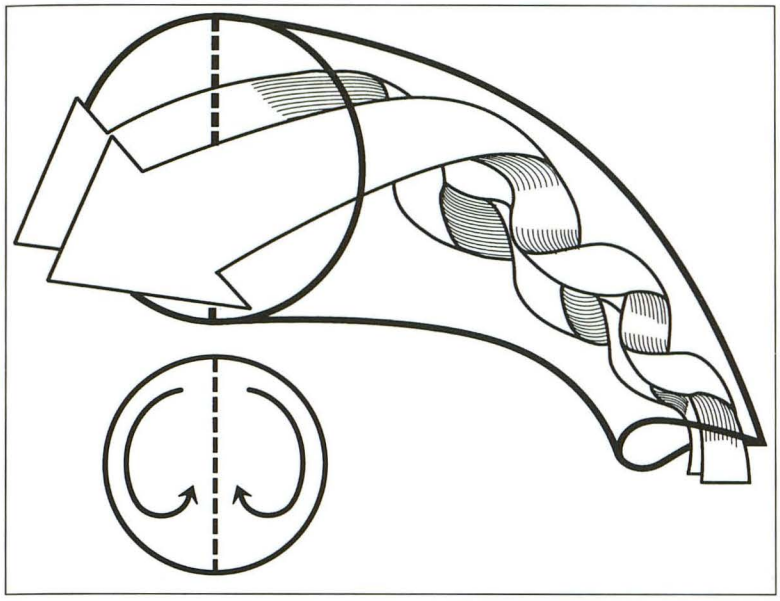
Flows through Curves and Bifurcations

Let us begin with a laboratory model, a perfectly symmetrical curved tube with perfect laminar flow through it. The central faster slipstreams, having inertia, attempt to continue in a straight line until they reach the greater curvature wall. There, they strike it, split, and rotate, forming dual internal helices (54) (Fig 3). In arteries, however, we rarely deal with perfect flows, and generally, a single swirling helix is created. Along the lesser curve (the concavity of the tube) one may see areas of stagnant or slow flow, and when flow velocities are high, regions of separation and actual flow reversal are seen.

Flow separation is a term used frequently by rheologists and is important for us to understand. Ordinarily, slipstreams progress along an artery in an orderly fashion. Their velocity profile is determined by the fluid's intrinsic properties and by the proximity to the artery's origin or inlet, as explained in detail in Part I. Curves present a problem to flowing fluid particles (54). The fluid slipstreams have inertia and tend to continue in a straight line as the bend is entered. If the velocity is high enough or the radius of curvature small enough, the slipstreams break away or separate from the lesser curvature wall. This region along the lesser curve is called a *separation zone* and contains fluid that is moving sluggishly, swirling, or even sometimes moving retrograde (Fig 3) (55–57).

Bifurcations are similar but more complex (58–64). The prototypical bifurcation begins as a straight tube, which then divides to form two daughter limbs (Fig 4). If flow velocities are to remain relatively constant in the daughter limbs, the combined cross-sectional areas of the daughter limbs must be about 30% greater than the area of the parent vessel. Under most flow conditions, the central, highest energy slipstream strikes the carina (or apex of the bifurcation) with some force and then splits to pass downstream into each daughter limb. This force can be measured by inserting a small probe into the carina and is called the *dynamic pressure* ($[\rho/2]w^2$, where ρ is fluid density and w velocity). In straight tubes, the central slipstream has the most energy because it is the fastest. If the flow energy level is high enough, vessel wall damage and subsequent degeneration may occur. Because arteries are generally curved, the highest energy slipstream entering the bifurcation may not strike exactly at the carina, which could explain in part why berry aneurysms are not always located at a bifurcation's carina. Varying the angle of the bifurcation causes important changes in the slipstreams next to the lateral walls. As we have seen, the slipstreams have mass and energy. Their inertia makes them continue along in a relatively straight line, and they do not make the change in direction abruptly into the daughter limbs (55). The slipstreams pass toward the wall adjacent to the carina, and because fluid is incompressible, velocity increases. The region of low shear at the wall often has a separation zone or even some reversed flow in it. With time, this low shear area fills in with endothelium and is called an *endothelial cushion*.

In our cast models, even the slightest asymmetry of the parent vessel causes the fluid entering the bifurcation to rotate. The observer sees a



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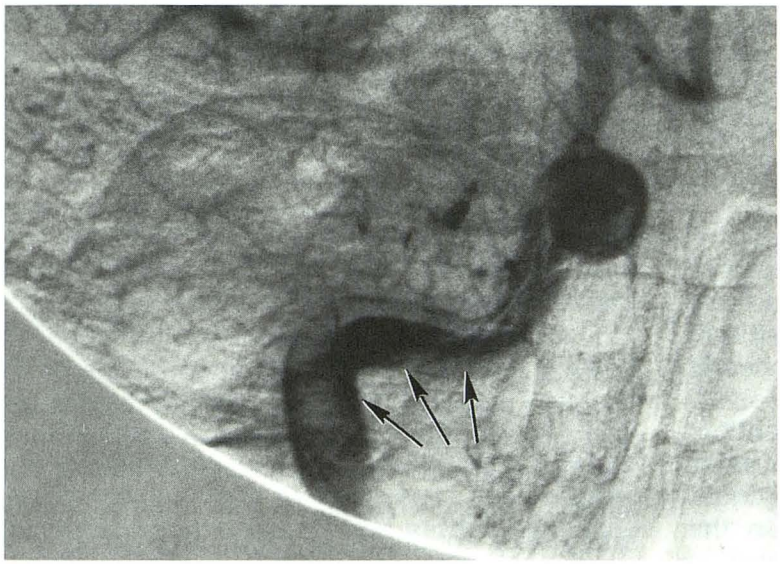

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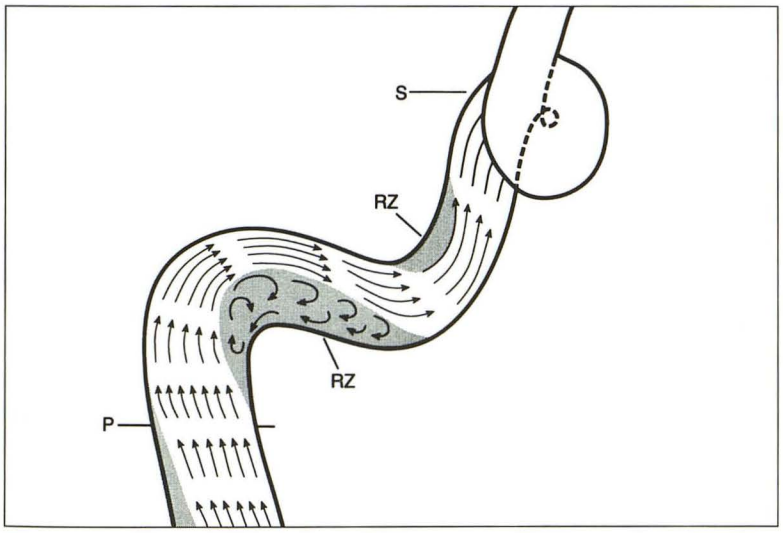

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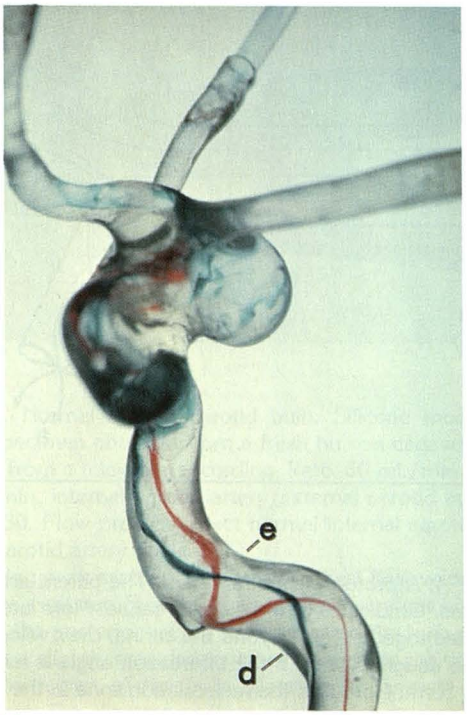


B

Fig. 3. *A*, Flows in a rigid, parallel wall, perfectly curved tube. Assume that flow entering the tube is laminar with a typical parabolic velocity profile. The central fastest slipstreams, having inertia, continue in a straight line until they reach the far wall, forcing the fluid there in toward the lesser curve, causing dual internal helixes.

B, Left internal carotid artery. In our human artery castings, the slipstreams tend to continue along a straight line, passing toward the wall of each greater curvature. Being incompressible, they must respond by increasing their velocities. When the slipstreams separate from the lesser curvature, areas of low shear or recirculation form at points *d* and *e*. Because most arterial flow is not perfect, a single helical flow (rather than the double helix shown in *A*) usually results as the fluid exits from the curve.

C, Late arterial phase, frontal view, right internal carotid angiogram. Note the contrast agent remaining in the recirculation zone along the lesser curvature of the intrapetrous internal carotid artery (*arrows*). Unopacified rapidly flowing blood has washed away the contrast agent in the stream. The hyperbaric contrast agent accentuates the size of the recirculation zone.

D, Diagramatic representation of slipstreams as revealed in *B* and *C*. *Arrow lengths* represent vector velocities. *RZ* indicates recirculation zones; *S*, siphon.

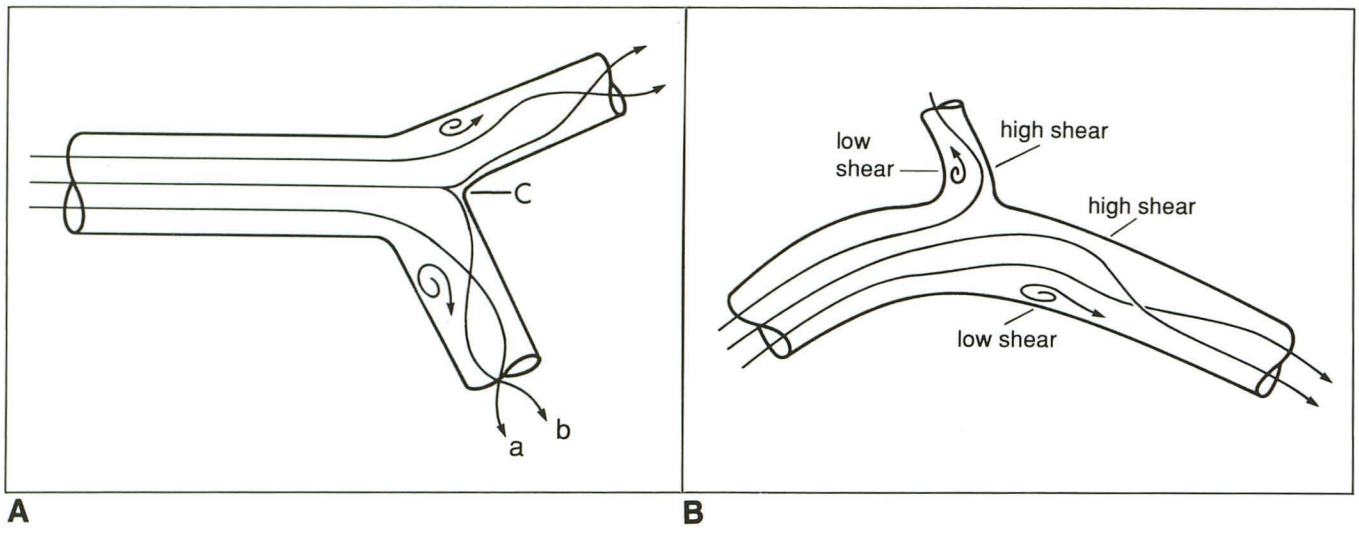


Fig. 4. *A*, A prototypical bifurcation. The parent artery divides to form two daughter limbs (or branches). The branch angles must be specified. *C* indicates flow divider or carina. More lateral slipstreams have inertia and tend to continue in a straight line. As slipstreams are crowded together just beyond the carina, their velocities increase. A lower pressure (or lower shear) region then forms at the lateral wall of the daughter limb. If the bifurcation angle is large enough or the velocity is high enough, an actual separation or recirculation zone may form. Note that the recirculation zone in the lower, more sharply angled daughter limb is larger than the upper limb's zone.

B, If the daughter limb is small in relation to the parent vessel and exits at or near a right angle, the bifurcation is called a *T junction*. Examples are the lenticulostriates, posterior communicating, and intercostal arteries. Fluid slipstreams must change direction abruptly, producing low-shear recirculation zones. In these regions, blood's non-Newtonian properties become important (28). As the fluid passes distally into the daughter limb, helical flow usually occurs.

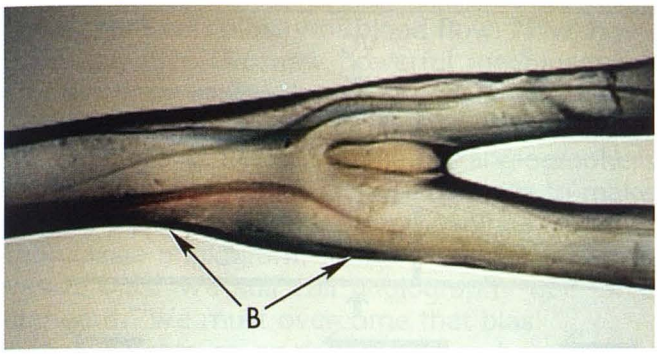
swirling helical flow in the daughter limbs. It seems that the angles of the bifurcation are important in the production of or protection from atherosclerosis. For example, atherosclerosis in the arm is rare, whereas leg arteries frequently are affected (65).

One special kind of bifurcation bears mention: that of the T junction. In a T junction a small artery exits from the parent vessel at nearly right angles and is usually small in relation to the luminal diameter of the parent vessel. Examples are the intercostal and lumbar arteries exiting from the aorta, the anterior choroidal and posterior cerebral arteries, and, in most patients, the anterior cerebral artery. As a small percentage of parent artery flow enters them, and as the flow exits from the parent artery at right angles to the major flow vector, blood's viscous properties become more important (28).

In humans, we rarely find idealized anatomy. Because most arteries are curved, almost all bifurcations begin with a curve. And because blood has a rotating helical pattern in curved vessels, most human bifurcations are presented with a complex disturbed flow. All arteries are subject to the general rules of hemodynamics, but to understand flow in a particular vessel (eg, the A1 segment or the cisternal segment of the anterior choroidal artery), it may be best to study each with accurately cast models, non-Newtonian fluid, and physiologic flow profiles.

The carotid bulb is a peculiar bifurcation because it contains a focal dilatation of one of the daughter branches. Furthermore, the velocity and flow profiles are not the same in each branch. The external carotid artery has essentially no end-diastolic flow, whereas in the internal carotid artery flow continues at about 30% to 40% of the rate measured at peak systole. This difference causes an exceedingly complex flow pattern, with areas of flow reversal in the posterior portion of the bulb during diastole and some siphoning of blood from the external carotid artery into the bulb (Fig 5). Flow dynamics in this important bifurcation have been studied by Ku in an elegant doctoral dissertation (Ku DN, Hemodynamics and Atherogenesis at the Human Carotid Bifurcation, Georgia Institute of Technology, May 1993) and by numerous other respected researchers (66–68, 59, 60).

When an atherosclerotic plaque forms within the bulb, it fills in the dilatation, and the complex flows nearly disappear. Then the bulb acts hydrodynamically similar to other bifurcations. Why there is such a striking area of flow reversal in a



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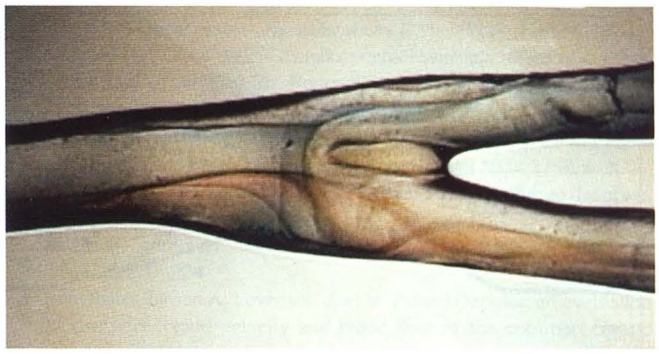

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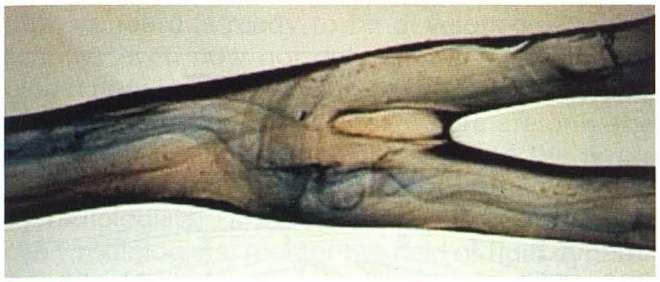


C

Fig. 5. Normal human carotid bulb. Silicone model of an injected specimen obtained from a fresh human cadaver. Images are taken from a television recording. Rate, 60 mL/min. Volume, 480 mL/min. Internal carotid artery/external carotid artery flow ratio, 70/30. Flow profiles reflect normal internal carotid artery/external carotid artery flows.

A, During peak systole, the time of highest flow velocities, the slipstreams continue forward in a relatively straight line but do not follow the posterior wall of the artery into the bulb dilatation. They appear slightly crowded together adjacent to the carina. The fluid in the region of the bulb dilatation (*B arrows*) has little forward flow. The slipstreams from prior cycles swirl gently there and act as an internal lozenge-shaped buffer helping force towards the faster slipstreams toward the carina.

B, During early diastole, flow becomes exceedingly complex. There is no net diastolic flow into the external carotid artery, and the anterior slipstreams pass from the orifice of the external carotid artery posteriorly into the lateral aspect of the bulb, striking the lateral wall with some force. They then rebound, flow distally, swirl, and continue onward toward the head, eventually rejoining the more medial and faster slipstreams in a helical stream.

C, During late diastole, changes are similar but more pronounced.

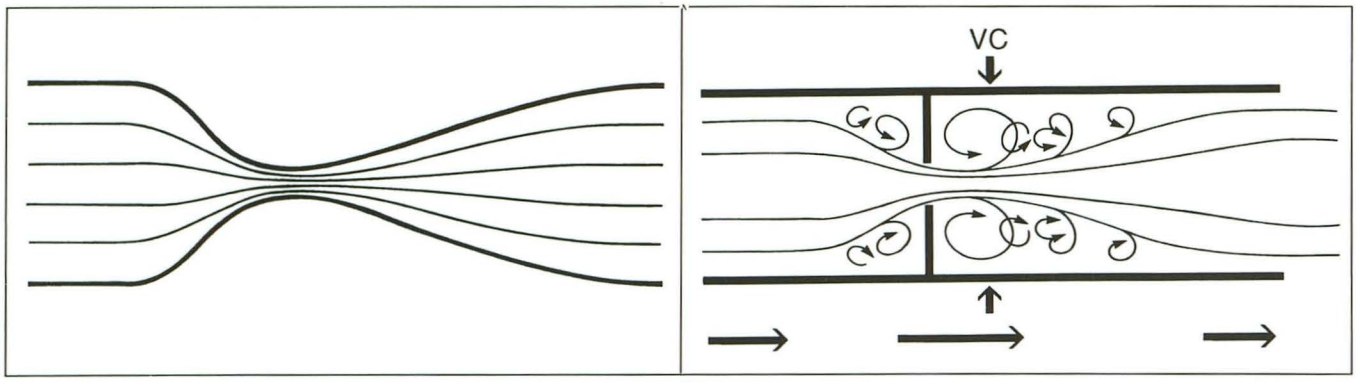
normal vessel can only be a matter of speculation now.

The Effects of Plaques and Catheters

We have already seen from Bernoulli's principle that if a vessel is narrowed, flow velocity increases, and pressure in the narrow region decreases. However, this phenomenon depends on a gradual and smooth reduction of the vessel diameter (Fig 6A). If an abrupt, short narrowing occurs in the vessel, flow becomes highly disturbed (69). Stream velocity must increase for all the fluid to get through the narrowing. Just proximal and distal to the narrowing, severe eddies are produced adjacent to the wall. The high-velocity fluid just beyond the diaphragmlike narrowing is called a *jet*. Surprisingly, the region of the lowest radial pressure and the highest velocity is always found in the jet some distance downstream from the diaphragm. This region is called the *vena contracta* (Fig 6B). The swirls and the downstream vena contracta cause difficulty in MR of abnormal vessels. If the narrowing is severe enough or long enough, a point will be reached when the traversing flow quantity will decrease. It generally takes about an 80% reduction in cross-sectional area to get a significant decrease in either flow or pressure; thereafter the decrease is exponential (70–73) (Fig 7).

When an eccentric short irregular plaque narrows a vessel, the effect is similar to half of a diaphragm-like constriction. Velocity increases across the stenosis, and proximal and distal disturbed eddies are produced. There is a low-pressure region in the stenosis, and if the plaque ulcerates and clot forms on the denuded surface, the clot may be sucked from the ulceration into the low-pressure high-velocity flow (Fig 6C). Over the ulceration the situation is analogous to the lift produced by the top surface of an aircraft wing.

Catheters can cause flow disturbances. Given the Poiseuille relationships (ie, area change being a fourth-power function) the most decrease in flow volume and increase in velocity will be found when the catheter size is large in relation to vessel lumen (74, 75). Measuring pressure from a probe facing into flowing fluid (the dynamic pressure) yields a higher value than when the pressure is measured radially. One may then reason than that measuring pressure through a catheter pointing *downstream* would yield a lower value. But



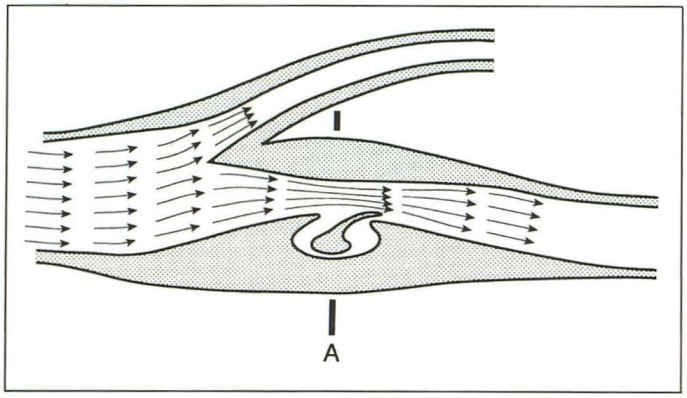


Fig. 6. *A*, A venturi tube. A gradual reduction in diameter leads to a smooth crowding of the slipstreams, allowing for their acceleration without much energy loss, and a pressure fall in the narrow region.

B, A tight short constriction. The slipstreams are unable to crowd together smoothly. Flow becomes highly disturbed. Swirls spin off from the main stream, and there is great energy loss. The disturbance extends downstream for a greater distance than upstream. The central rapidly flowing streams are called a jets. The area of lowest pressure is downstream some distance from the diaphragm, and is called the vena contracta.

C, An intravascular plaque obeys these same basic principles. As the plaque narrows the vessel the velocity increases. *Arrow lengths* represent velocity. With increased velocity, the pressure decreases, as shown in Part I. This diagram shows a small clot being drawn of the ulcer into the rapidly flowing stream by the lowered pressure.

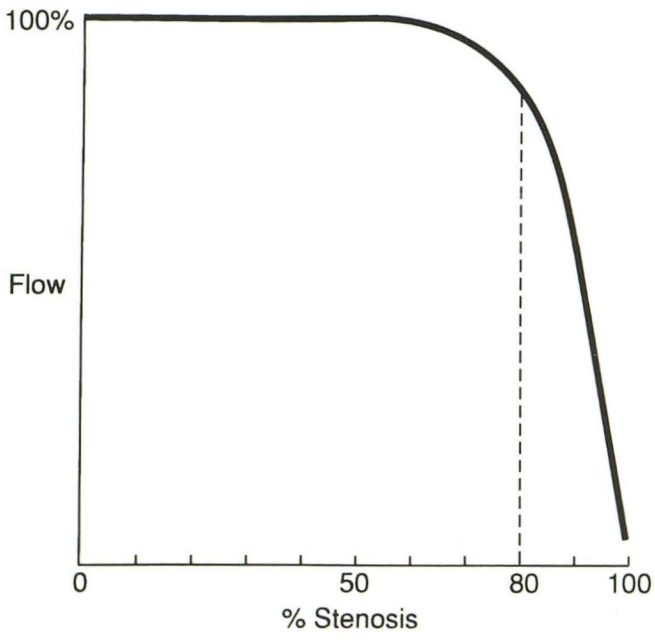


Fig. 7. Relationship of flow to luminal narrowing. At about 80% area narrowing a significant decrease of both pressure and flow occurs. Thereafter, the decrease is exponential. (Adapted from Mann et al [71].)

at physiologic flow velocities such is not the case, and we have found good correlation of radial and microcatheter measurements up to 200 cm/s (Kerber CW and coworkers, unpublished data).

Summary

Although the rules about flows seem complex and even at times suggest chaos, some general principles can be extracted and used by the radiologist. Normal arteries damp disturbances, tolerate our catheters well, and generally cause the blood's slipstreams to swirl.

It seems that the swirls and the energy that accompanies the passage of those swirls are prime determiners of the development of degenerative changes, most importantly of atherosclerosis and berry aneurysms. Knowing this, we now must direct our research to look beyond today's practical applications and this simplistic summary.

Radiologists who trained during the angiography era are often incredulous when they see the richness of information found in physiologic flow models. For years, contrast agents have hidden

the elegant complexity of blood flow. Now, however, we have two new powerful machines: the Doppler gray-scale ultrasound and the MR scanner. These machines routinely demonstrate flow data that we do not as yet use. As angiographers we have a natural and unconscious bias to make images produced by our new machines look like the classic angiogram. It is a powerful and pervasive bias. We still call angiography our "gold standard." We must overcome that bias.

As valuable as angiography has been to radiology, it may no longer be our benchmark. A new standard is ready to be developed. The MR scanner even now not only allows calculation of global flow in vessels but also analyzes individual slipstreams. The images shown here are only the beginning. Keep our old mindset, and the limits and utility of the MR scanner will not be explored by radiologists. However, if we physicians, especially radiologists, reenter the field of fluid dynamics, all of science and our patients will benefit. We have broad shoulders to stand on and see into the future. Harvey, Hales, Galen, and Poiseuille: all were physicians; they added immeasurably to the foundations of rheology and our understanding of flowing blood. We must be willing to do likewise.

References

1. Fishman A, Richards D. *Circulation of the blood: Men and ideas.* New York: Oxford University Press, 1964
2. Harvey W. *Exercitatis anantomica de motu cordis et sanguinis in animalbus*, 1628, English translation with annotations by C.D. Leake. 4th ed. Springfield, Ill: Thomas, 1958
3. Young T. Hydraulic investigations, subservient to an intended Croonian lecture on the motion of the blood. *Phil Trans R Soc Lond [Biol]* 1808;98:164–186
4. Young T. On the functions of the heart and arteries. *Phil Trans R Soc Lond [Biol]* 1809;99:164–186
5. Copley AL. *Hemorheology: Proceedings of the First International Conference.* London: Pergamon, 1968
6. Copley AL, Hartert HH. *Theoretical and clinical hemorheology: Proceedings of the Second International Conference.* Berlin-Heidelberg, New York: Springer, 1971
7. Fung YC, Perrone N, Anliker M. *Biomechanics: Its foundations and objectives.* New York: Prentice-Hall, 1972
8. Pedley T. *The fluid mechanics of large blood vessels.* New York: Cambridge University Press, 1980
9. Fung Y. *Biomechanics motion, flow, stress, and growth.* New York: Springer-Verlag, 1990
10. McDonald DA. *Blood flow in arteries.* 2nd ed. Baltimore: Williams & Wilkins, 1974
11. Stein PD, Sabbah HN. Hemorheology of turbulence. *Biorheology* 1980;17:301–319
12. Stein PD, Sabbah HN, Anbe DT. Comparison of disturbances of flow in the main pulmonary artery of man. *Biorheology* 1979;16:357–362
13. Sabbah HN, Stein PD. Turbulent blood flow in humans, its primary role in the production of ejection murmurs. *Circ Res* 1976;38:6–12
14. Hales S. *Statical essays, Haemastaticks II.* New York: Hafner, 1964
15. Langfitt TW, Obrist WD. Occlusive cerebrovascular disease: cerebral blood flow. In: Wilkins RH, Rengachery SS, eds. *Neurosurgery.* New York: McGraw Hill, 1985
16. Olson RM. Human carotid artery wall thickness, diameter, and blood flow by a noninvasive technique. *J Appl Physiol* 1974;37:955–960
17. Fitzgerald DE, O'Shaughnessy AM, Keaveny VT. Pulsed Doppler: determination of blood velocity and volume flow in normal and diseased common carotid arteries in man. *Cardiovasc Res* 1982;16:220–224
18. Benetos A, Simon A, Levenson J, et al. Pulsed Doppler: an evaluation of diameter, blood velocity and blood flow of the common carotid artery in patients with isolated unilateral stenosis of the internal carotid artery. *Stroke* 1985;16:969–972
19. Blackshear WM, Phillips DJ, Chikos PM, et al. Carotid artery velocity patterns in normal and stenotic vessels. *Stroke* 1980;11:67–71
20. Lantz B. *A methodologic investigation of roentgen videodensitometric measurement of relative flow.* Goteburg, Sweden: Goteburg Press, 1974
21. Haake EM, Smith AS, Lin W. Velocity quanitation in magnetic resonance imaging. *Top Magn Reson* 1991;3:34–49
22. O'Donnell M. Blood flow imaging using multiecho phase contrast sequences. *Med Phys* 1985;12:59–64
23. Wedeen V, Rosen B, Chesler D, et al. MR velocity imaging phase display. *J Comput Assist Tomogr* 1987;9:530–536
24. Firmin DN, Nayler GL, Klipstein RH, et al. In vivo validation of MR velocity imaging. *J Comput Assist Tomogr* 1987;11:751–756
25. Evans AJ, Fumiharu I, Grist TA, et al. Magnetic resonance imaging of blood flow with a phase subtraction technique. *Invest Radiol* 1993;28:109–115
26. Spritzer CE, Pelc NJ, Lee NJ, et al. Preliminary experience with rapid MR flow imaging using a phase sensitive limited flip angle gradient refocused pulse sequence. *Radiology* 1990;176:256–261
27. Marks MP, Pelc NJ, Ross MR, et al. Determination of cerebral blood flow with a phase contrast cine MR imaging technique: evaluation of normal subjects and patients with arteriovenous malformations. *Radiology* 1992;182:467–476
28. Liepsch DW: Flow in tubes and arteries: a comparison. *Biorheology* 1986;23:395–433
29. Stehbens WE. Etiology of intracranial berry aneurysms. *J Neurosurg* 1989;70:823–831
30. Stehbens WE. Aetiology of cerebral aneurysms. *Lancet* 1981;2:524–525
31. Stehbens WE. Cerebral aneurysm and congenital abnormalities. *Aust Ann Med* 1962;11:102–112
32. Ross R, Glomset J, Harker L. Response to injury and atherogenesis. *Am J Pathol* 1977;86:675–684
33. El Masry OA, Feuerstein IA, Round GF. Experimental evaluation of streamline patterns and separated flows in a series of branching vessels with implications for atherosclerosis and thrombosis. *Circ Res* 1978;43:608–618
34. Bergel DH, Nerem RM, Schwartz CJ. Fluid dynamic aspects of arterial disease. *Atherosclerosis* 1976;23:253–261
35. Nichols W, O'Rourke M. *McDonald's blood flow in arteries.* Philadelphia: Lea and Febiger, 1990
36. Fox JA, Hugh AE. Localization of atheroma: a theory based on boundary layer separation. *Br Heart J* 1966;28:388–399
37. Stehbens WE, Davis PF, Martin BJ. Hemodynamic induction of atherosclerosis: localization, morphology and biochemistry. *Monogr Atheroscler* 1990;15:1–12
38. Fry DL. Acute vascular endothelial changes associated with increased blood velocity gradients. *Circ Res* 1968;22:165–197
39. Houle S, Roach MR. Flow studies in a rigid model of an aorto-renal junction: a case for high shear as a cause of the localization of sudanophillic lesions in rabbits. *Atherosclerosis* 1981;40:231–244

40. Caro CG, Fitzgerald JM, Schroter RC. Atheroma and arterial wall shear: observation, correlation and proposal of a shear dependent mass transfer mechanism for atherogenesis. *Proc R Soc Lond* 1971;177:109–159
41. Karino T, Motomiya M. Flow visualization in isolated transparent natural blood vessels. *Biorheology* 1983;20:119–127
42. MacFarlane TWR, Roach MR, Chan K. The geometry of human cerebral bifurcations: effect of static distending pressure. *J Biomech* 1980;13:265–277
43. Roach M. *The effects of bifurcations and stenoses on arterial disease.* Baltimore: University Park Press, 1977
44. Roach MR, Scott S, Ferguson GG. The hemodynamic importance of the geometry of bifurcations in the circle of willis (glass model studies). *Stroke* 1972;3:255–267
45. Roach MR, Burton AC. The reason for the shape of the distensibility curves of arteries. *Can J Biochem Physiol* 1957;35:681–690
46. Nerem RM. Vascular endothelial response to shear stress. *Monogr Atheroscler* 1990;15:117–124
47. Goldsmith H. The flow of model particles and blood cells and its relation to thrombogenesis. In: Spaet TH, ed. *Progress in hemostasis and thrombosis.* New York: Grune and Stratton, 1972:137–149
48. Stein PD, Sabbah HN. Measured turbulence and its effect on thrombus formation. *Circ Res* 1974;35:608–614
49. Wurzinger LJ, Blasberg P, Schmid-Schonbein H. Towards a concept of thrombosis in accelerated flow: rheology, fluid dynamics, and biochemistry. *Biorheology* 1985;22:437–449
50. Wurzinger LJ, Optiz R, Wolf M, et al. Shear induced platelet activation a critical reappraisal. *Biorheology* 1985;22:399–413
51. Parmentier EM, Morton WA, Petschek HE. Platelet aggregate formation in a region of separated blood flow. *Phys Fluids* 1977;20:2012–2021
52. Friedman MH, Bargeron CB, Mark FF. Variability of geometry: hemodynamics and intimal response of human arteries. *Monogr Atheroscler* 1990;15:109–116
53. Schmid-Schonbein H, Born GVR, Richardson PD, et al. Rheology of thrombotic processes in flow: the interaction of erythrocytes and thrombocytes subjected to high flow forces. *Biorheology* 1981;18:415–444
54. Caro CG, Pedley TJ, Schroter RG, et al. *The mechanics of the circulation.* New York: Oxford University Press, 1978
55. Hamakiotes CC, Berger SA. Flow in curved vessels, with application to flow in the aorta and other arteries. *Monogr Atheroscler* 1990;15:227–239
56. Walburn FJ, Stein PD. Flow in a symmetrically branched tube simulating the aortic bifurcation: the effects of unevenly distributed flow. *Ann Biomed Eng* 1980;8:159–173
57. Mann DE, Tarbell JM. Flow of nonnewtonian blood analog fluids in rigid curved and straight artery models. *Biorheology* 1990;27:711–733
58. Feuerstein IA, El Masry OA, Round GF. Arterial bifurcation flow: effects of flow rate and area ratio. *Can J Physiol Pharmacol* 1976;54:795–808
59. LoGerfo FW, Nowak MD, Quist WC, et al. Flow studies in a model carotid bifurcation. *Arteriosclerosis* 1981;4:235–241
60. Ku DN, Giddens DP. Pulsatile flow in a model carotid bifurcation. *Arteriosclerosis* 1983;3:31–39
61. Malcolm AD, Roach MR. Flow disturbances at the apex and lateral angles of a variety of bifurcation models and their role in development and manifestations of arterial disease. *Stroke* 1979;10:335–343
62. Brech R, Bellhouse BJ. Flow in branching vessels. *Cardiovasc Res* 1973;7:593–600
63. LoGerfo FW, Crawshaw HM, Nowak M. Effect of flow split on separation and stagnation in a model vascular bifurcation. *Stroke* 1981;12:660–665
64. Gutstein WH, Schneck DJ. In vitro boundary layer studies of blood flow in branched tubes. *J Atheroscler Res* 1967;7:295–299
65. Roach M, Brown N. What protects arm arteries from atherosclerosis? In: Liepsch D, ed. *Biofluid mechanics, blood flow in large vessels.* vol 2. New York: Springer-Verlag, 1990:7–8
66. Ku DN, Giddens DP. Pulsatile flow in a model carotid bifurcation. *Arteriosclerosis* 1983;3:31–39
67. Motomiya M, Karino T. Flow patterns in the human carotid artery bifurcation. *Stroke* 1984;15:50–56
68. LoGerfo FW, Nowak MD, Quist WC, et al. Structural details of boundary layer separation in a model carotid bifurcation under steady and pulsatile flow conditions. *J Vasc Surg* 1985;2:263–269
69. Karino T, Goldsmith HC, Motomiya M, et al. Flow patterns in vessels of simple and complex geometries. *Ann NY Acad Sci* 1987;516:441–422
71. Mann FC, Herrick JF, Essex HE, et al. The effect on the blood flow of decreasing the lumen of a blood vessel. *Surgery* 1938;4:249–252
72. Berguer R, Hwang NHC. Critical arterial stenosis: a theoretical and experimental solution. *Ann Surg* 1974;180:39–50
73. May AG, De Berg LV, DeWeese JA, et al. Critical arterial stenosis. *Ann Surg* 1963;54:250–259
74. Fei DY, Billian C, Rittgers SE. Flow dynamics in a stenosed carotid bifurcation model—part I: basic velocity measurements. *Ultrasound Med Biol* 1988;14:1:21–31
75. Bjorno L, Petersson H. Hydro- and hemodynamic effects of catheterization of vessels, I: An experimental model. *Acta Radiol Diagn* 1976;17:511–518
76. Bjorno L, Petersson H. Hydro- and hemodynamic effects of catheterization of vessels, V: Experimental and clinical catheterization of stenoses. *Acta Radiol Diagn* 1977;18:193–209