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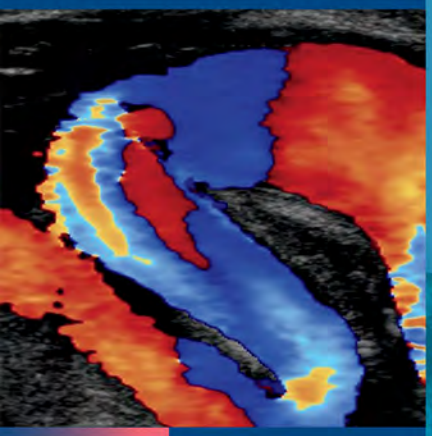

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CLAUDIA RUMWELL | MICHALENE McPHARLIN

Vascular Technology

An Illustrated Review

5th Edition

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DAVIES

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CHAPTER 2

Physiology and Fluid Dynamics

Arterial System

Energy

Blood Flow Characteristics

Poiseuille's Law

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Pressure/Velocity Relationships (Bernoulli Principle)

Steady versus Pulsatile Flow

Peripheral Resistance

Cardiac Effects

Collateral Effects

Effects of Exercise

Effects of Stenosis on Flow

ARTERIAL SYSTEM

The arterial system is a multibranched elastic conduit that carries blood away from the heart and outward to the most distant tissues.

- The arterial tree oscillates with every beat of the heart, each one of which pumps approximately 70 ml of blood into the aorta and causes a blood pressure pulse.
- At the beginning of the cardiac contraction, the pressure in the left ventricle rises rapidly, quickly exceeding that in the aorta so that the aortic valve opens, blood is ejected, and the blood pressure rises. The amount of blood ejected is called the *stroke volume*.
- Increased heart rate delivers an increased blood volume that supplies more nutrients. Conversely, the lower the heart rate, the smaller the volume of pumped blood.
- The patient's cardiac status plays an important role in the movement of blood throughout the vascular system.

The heart pump generates the pressure (*potential energy*) to move the blood. The stroke volume of blood produced by each heartbeat creates a pressure (or *energy*) wave that travels rapidly throughout the arterial system (Figure 2-1):

- The propagation speed, shape, and strength of the pressure wave change as the wave moves through the arterial system.

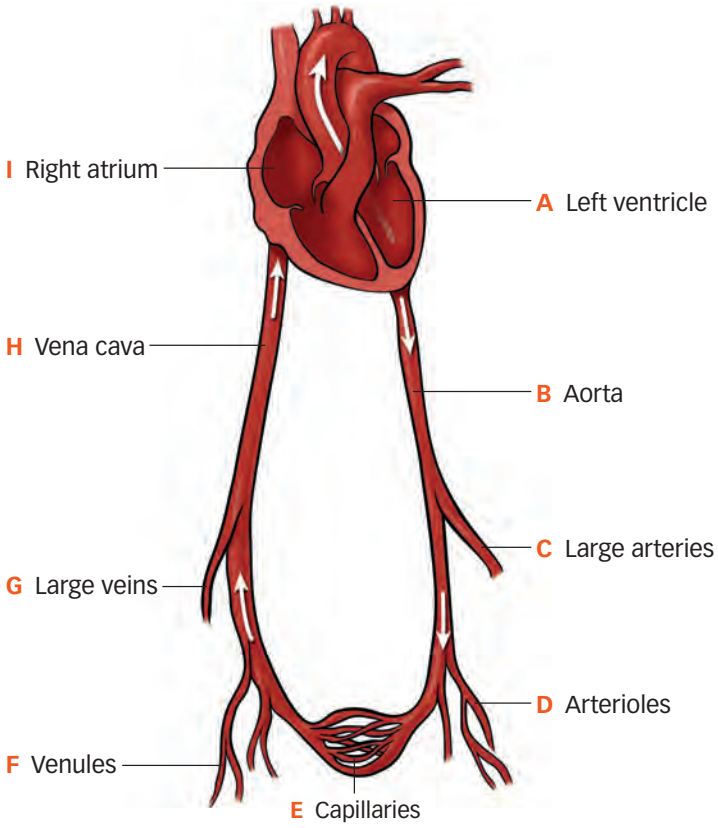


Figure 2-1. Once the heart generates the pressure to move the blood, the energy wave produced travels rapidly throughout the system beginning with the left ventricle (**A**) to the capillary bed (**E**) back to the heart via the venous system (**F–I**).

Example: As the arterial pressure wave moves distally, away from the heart and out toward the periphery, the propagation speed—the pulse wave velocity—increases with the growing stiffness of the arterial walls.

- Variations in the characteristics of the vessels influence these alterations in blood flow. Velocity and flow direction also vary with each heartbeat.
- As the pressure wave moves from the large arteries through the high-resistance vessels, capillaries, and then into the venous side, the mean pressure gradually declines because of losses in total fluid energy.

The pumping action of the heart maintains a high volume of blood in the arterial side of the system that in turn sustains a high pressure gradient between the arterial and venous sides of the circulation. This pressure gradient is necessary to maintain flow.

Cardiac output governs the amount of blood that enters the arterial system, while arterial pressure and total peripheral resistance (which is controlled by the level of vasoconstriction in the microcirculation) determine the volume of blood that leaves it.

A large portion of the energy created with each left ventricular contraction results in distention of the arteries, producing an arterial "reservoir" that stores some of the blood volume and the potential energy supplied to the system.

It is this store of energy and volume that promotes the flow of blood into the tissue during diastole. That is, potential energy is stored in the distended arterial wall and is released when the wall recoils.

Pressure is greatest at the heart and gradually decreases as the blood moves distally. This pressure difference (or *gradient*) is necessary to maintain blood flow.

ENERGY

The movement of any fluid medium between two points requires two things: (1) a route along which the fluid can flow and (2) a difference in energy (pressure) levels between the two points. The volume of flow depends on the net energy difference between these two points, a factor that is affected by losses resulting from the movement of the fluid—i.e., friction—and any resistance within the pathway that opposes such movement.

The greater the energy difference (or the lower the resistance), the greater the flow, as illustrated in Figure 2-2.

The greater the pathway resistance and/or energy losses, the lower the flow.

lower resistance = higher flow rate
higher resistance = lower flow rate

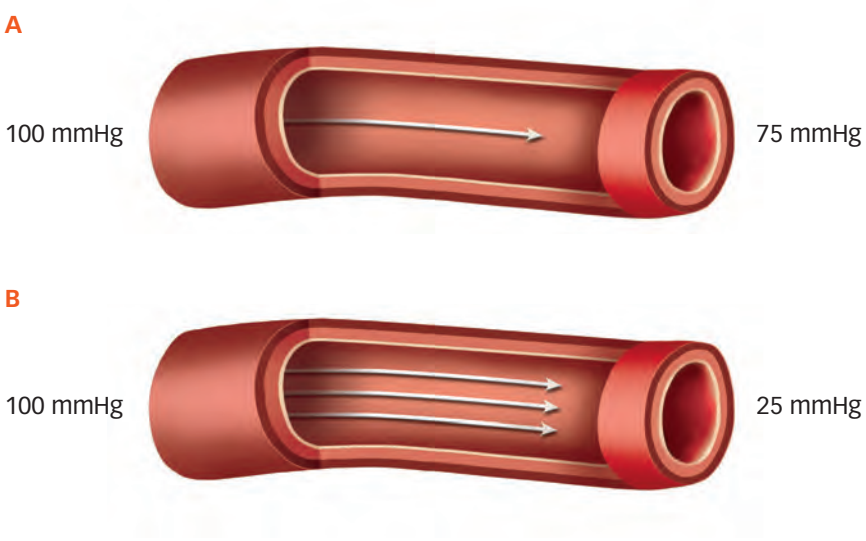
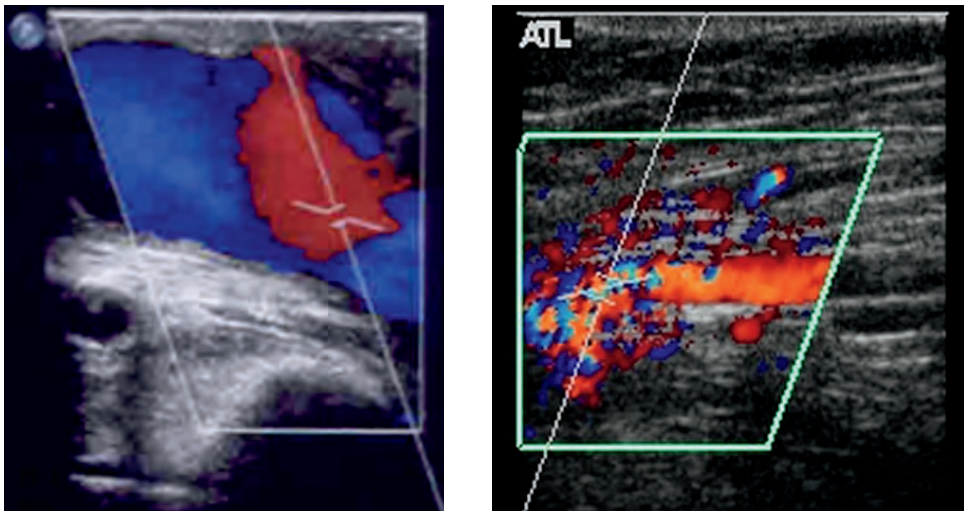


Figure 2-2. The greater the pressure gradient, the greater the flow. Example **A** has less pressure difference and therefore less flow than example **B**, which has a larger pressure gradient and greater flow.

The total energy contained in moving fluid is the sum of pressure (potential) energy, kinetic (movement) energy, and gravitational energy, as explained below:

- *Pressure energy* is the main form of energy present in flowing blood (see Figure 2-3A). It is created by the pumping action of the heart, which subsequently distends the arterial vessels. This distention occurs because of elastin, which allows for a rapid increase in capacity. Arteries are more elastic proximally and less distally as they become smaller. Elastin quickly converts kinetic energy to pressure energy. The pressure energy is then converted back to kinetic energy in diastole. Pressure energy is also referred to as *potential energy*. Potential energy has several components. The dominant source is the pumping action of the heart muscle, as well as distention of the arterial wall.
- *Kinetic energy* is the ability of flowing blood to do work as a result of its velocity; it is the energy of something in motion (see Figure 2-3B). With regard to blood flow, the kinetic energy portion is small compared to the pressure energy. Kinetic energy is also proportional to the density of blood (which is normally stable) and to the square of its velocity. The everyday example of a dam illustrates the difference between potential and kinetic energy: The water behind the dam has *potential* energy (with the height of the dam providing a form of *gravitational* energy), while the water flowing through the dam has *kinetic* energy.
- *Gravitational energy* (*hydrostatic pressure*) is expressed in millimeters of mercury (mmHg). Changes in the height of the fluid column introduce the element of gravitational energy, which is hydrostatic pressure. In the circulatory system, hydrostatic pressure is equivalent to the weight of the column of blood extending from the heart, where the right atrium is considered the 0 pressure reference point (i.e., atmospheric pressure), to the level where the pressure is being measured.



A B

Figure 2-7. **A** In a popliteal artery aneurysm, resistance to flow is reduced due to the larger-than-normal diameter. Although the volume of blood flow through this vessel remains constant, velocities are decreased compared to normal. **B** In this superficial femoral artery (SFA) stenosis, resistance to flow is increased because of the stenotic lumen. Although the volume flow remains constant, velocities are elevated as a result of the stenosis.

POISEUILLE'S LAW

Poiseuille's law defines the relationship between volume flow (Q), pressure (P), and resistance (R) and may be written as $Q = P/R$. It helps to answer the question of how much fluid is moving through a vessel. When combined with the resistance equation (see page 21), Poiseuille's law may be stated as follows:

$$Q = \frac{(P_1 - P_2)\,\pi r^4}{8\eta L}$$

where Q = volume flow, P_1 = pressure at the proximal end of the vessel, P_2 = pressure at the distal end of the vessel, r = radius of the vessel, L = length of the vessel, $\pi = 3.1416$, and η = viscosity of the fluid.

- A change in the diameter (hence, radius) of a vessel affects resistance more dramatically than viscosity or vessel length (see Figures 2-7A and 2-7B).
- The radius of a vessel is directly proportional to the volume flow. Small changes in radius may result in large changes in flow, as demonstrated in Figure 2-8.

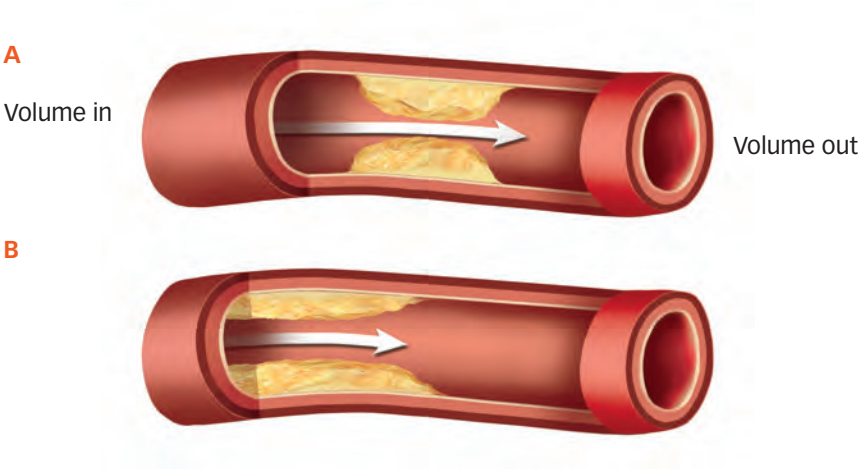


Figure 2-8. Parts **A** and **B** both illustrate narrowed segments. The reduction in volume flow through segment A will be greater than that through segment B, where the radius of the stenotic segment is somewhat larger.

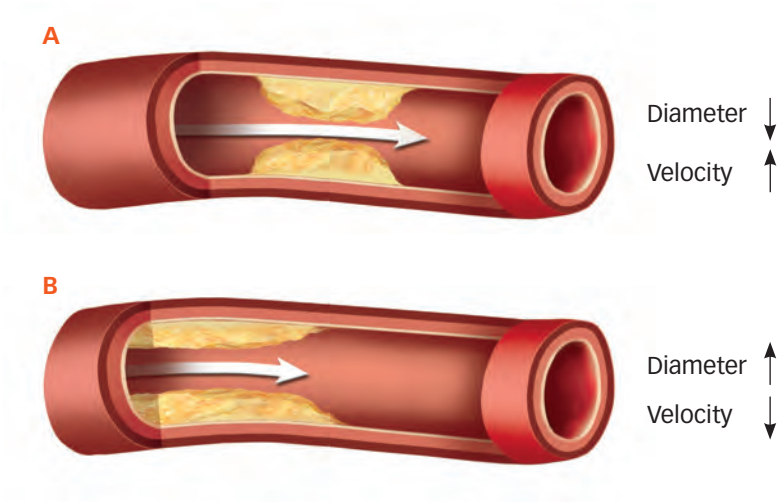


Figure 2-9. A As the diameter decreases, the velocities increase. **B** As the diameter increases, the velocities decrease.

- As vessel radius decreases, resistance increases. The volume of blood flow through the vessel nevertheless remains constant. To maintain volume flow as vessel size decreases, velocity must increase. As illustrated in Figures 2-9 and 2-7A and B, the size of a vessel is inversely proportional to the velocity of blood flow. According to the basic laws of fluid dynamics, most notably the law of conservation of mass (i.e., what goes in must come out), the relationship among velocity, volume flow, and cross-sectional area of the vessel is

$$V = \frac{Q}{A}$$

  where V = velocity (cm/sec), Q = volume flow (cm^3), and A = cross-sectional area (cm^2).
- In the cardiovascular system, the length of the vessels and the viscosity of the blood usually do not change much. This means that changes in blood flow occur mainly as a result of changes in the radius of a vessel and in the pressure energy gradient that makes flow possible.

The abbreviated pressure/volume flow relationship is quite similar to one used in electronics to explain the flow of electricity—Ohm's law—commonly expressed as I (flow of electrons) = E/R. Please note that it may be written as I = V/R in some references. Here is how the two equations compare:

Q = P/R		**I = E/R**
Flow volume (Q)	similar to	Current (I), flow volume of electrons
Pressure (P)	similar to	Voltage (E)
Flow resistance (R)	similar to	Electrical resistance (R)

REYNOLDS NUMBER

After the initial acceleration in systole, blood movement continues and develops into distinct streamline formations. Where P = pressure, note in Figure 2-10A that the streamlines are evenly distributed. When the flow pattern becomes unstable, these continuous streamlines break up and form small circular currents called eddy currents and vortices (swirling patterns of rotational flow) (Figure 2-10B).

Osborne Reynolds sought to determine how viscosity, vessel radius, and the pressure/volume relationship influence the stability of flow through a vessel. Although most of his work applied to straight, rigid tubes, it still provides insight into the physics of blood flow.

Flow volume increases as pressure increases, but only to a point. As flow changes from stable to disturbed, Reynolds found that an increase in pressure no longer increased flow volume. Instead, it increased flow disturbance, contributing to the formation of eddy currents.

The elements that affect the development of turbulent flow are expressed by a "dimensionless" number called the *Reynolds number* (Re). The factors that affect the development of turbulence are expressed by this number according to the following equation:

$$Re = \frac{V\rho 2r}{\eta}$$

where Re = Reynolds number, V = velocity, ρ = the density of the fluid, r = the radius of the tube, and η = the viscosity of the fluid. Because the density and viscosity of the blood are fairly constant, the development of turbulence depends mainly on the size of the vessels and the velocity of flow. When the Reynolds number exceeds 2000, laminar flow tends to become disturbed. Flow disturbances also can occur at lower values because of other factors, such as body movement, pulsatility of blood flow, and irregularities of the vessel wall and plaque.

Turbulent flow may cause vessel walls to vibrate. The harmonics of this vibration produce vascular bruits.

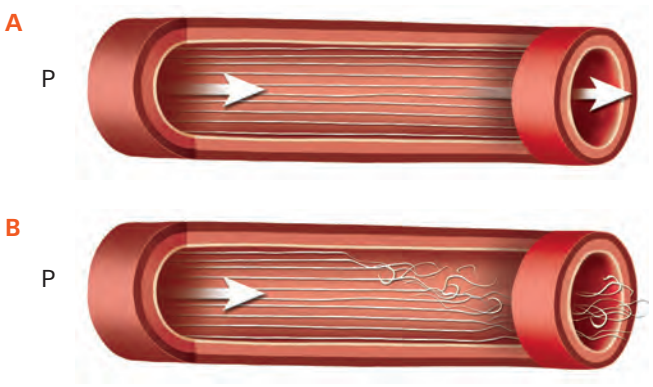


Figure 2-10. Arterial flow streamlines. **A** Evenly distributed. **B** Disrupted (turbulent). P = pressure.

PRESSURE/VELOCITY RELATIONSHIPS (BERNOULLI PRINCIPLE)

As previously described in the section on energy, the total energy contained in moving fluid is the sum of potential (i.e., pressure), kinetic, and gravitational energies. If one of these variables changes, the others also must change to maintain total fluid energy at the same level.

Example: If gravitational energy remains unchanged (that is, there is no change in the height of the fluid) but kinetic energy (velocity) increases, then potential (pressure) energy must decrease to maintain the same total fluid energy.

The Bernoulli equation shows that velocity and pressure are inversely related. Where there is high velocity, there is low pressure; where there is low velocity, there is high pressure. This inverse relationship between pressure and velocity explains why pressure decreases where fluid velocity increases (within the stenotic segment of an artery, for example) and why pressure distal to a stenosis (the region of poststenotic turbulence, where velocity decreases) is higher than that within the lesion itself. (See Figure 2-11.)

In other words, this is the law of conservation of energy:

1. In the region proximal to the stenosis (prestenosis), the pressure energy is higher and the kinetic energy lower. This region has the highest total energy sum.
2. As blood flows into the area of the stenosis, the pressure energy decreases and the kinetic energy increases (higher). However, total energy in this stenotic segment is less than that in the prestenotic segment because energy is lost (i.e., converted into heat) as it moves through the narrowing.
3. Lastly, distal to the stenosis (poststenosis), the kinetic energy decreases and the pressure energy increases. At this point the total energy sum is lowest.

Pressure gradients—the difference in pressure between two points in a vessel—are described as *flow separations*. Flow separations within a vessel may be

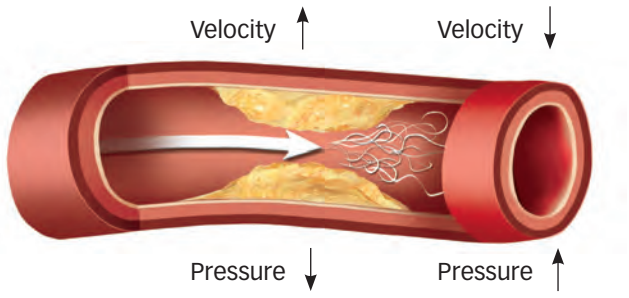


Figure 2-11. Velocity energy is elevated and pressure energy diminished within a stenosis. Relative velocity energy decreases and pressure energy increases distal to a stenosis. However, overall energy decreases.

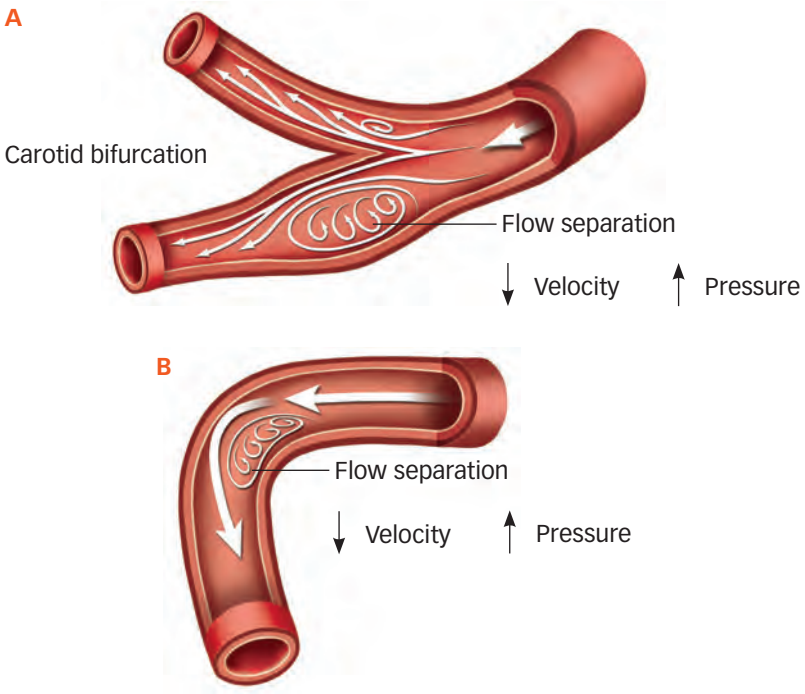


Figure 2-12. Flow separation patterns. In both examples **A** and **B**, pressure energy is higher and velocity energy lower in the area of the pressure gradient (flow separation). This causes flow direction to move to the area of lower pressure energy. **A** Flow separation in the carotid bulb. **B** Flow separation at a curve (note that it is located on the inside of the vessel curve).

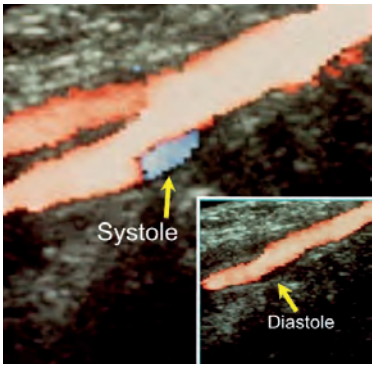


Figure 2-13. Longitudinal image of the carotid bifurcation in systole. The inset shows the same image in diastole. Note that flow separation (blue) is present during systole, absent in diastole.

caused by changes in the geometry of the vessel (with or without intraluminal disease) or the direction of the vessel, as depicted in Figures 2-12A and B. See also Figure 2-13.

Flow separations leave behind regions of flow reversal, stagnant or little movement. Figure 2-14 demonstrates two different regions within a bypass graft where these flow separations can occur.

Because flow moves from high to low pressure (described as a *pressure gradient*), the direction of flow in the region of flow separation (e.g., carotid bulb, bypass graft anastomosis) changes with respect to the transducer, causing a visible color change in the color flow image at systole.

During diastole, when flow at the vessel wall is stagnant, there is no movement of blood and therefore no color in the color flow image. The flow separation pattern is an ideal one to use to help define whether an image is in systole or diastole.

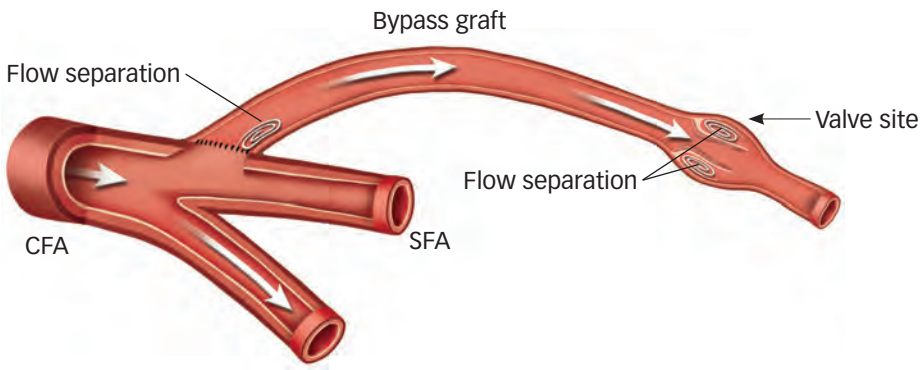


Figure 2-14. In this reversed saphenous vein graft at the end-to-side proximal anastomosis there is an area of flow stagnation (flow separation) on the inside wall. Distally, there are areas of flow separation at the site of a valve cusp. Note the variability of vessel dimension in the bypass graft. CFA = common femoral artery, SFA = superficial femoral artery.

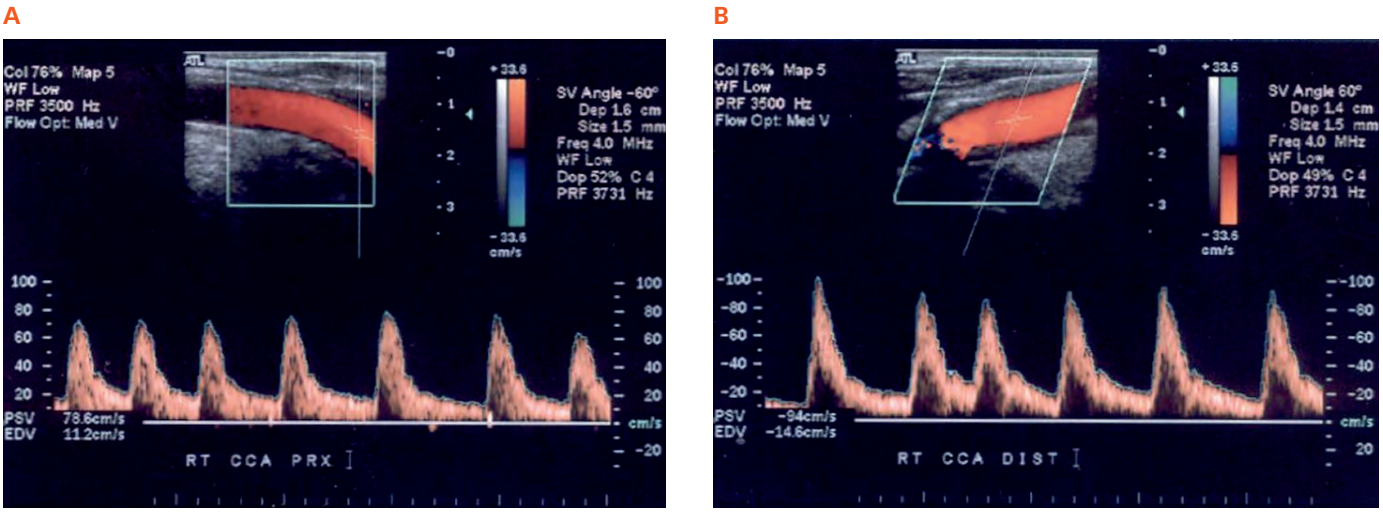


Figure 2-22. An irregular heart rhythm is evident in this spectral analysis of a common carotid artery.

- A severely irregular heart rhythm (Figure 2-23), on the other hand, represents a challenge in obtaining reliable peak systolic velocity (PSV) measurements. Some protocols call for averaging a few cycles to be the most reliable; others consider the averaging of 10 cycles to be the best method. When determining the significance of a stenosis, calculating a velocity ratio may be helpful; e.g., the highest internal carotid artery PSV is divided by the PSV of the more distal common carotid artery.

Stenosis of the Aortic Valve

- A delay in the systolic upstroke is evident (Figure 2-24).
- Decreased peak systolic velocities (PSVs) are also usually seen. Therefore, PSVs may underestimate a stenosis.

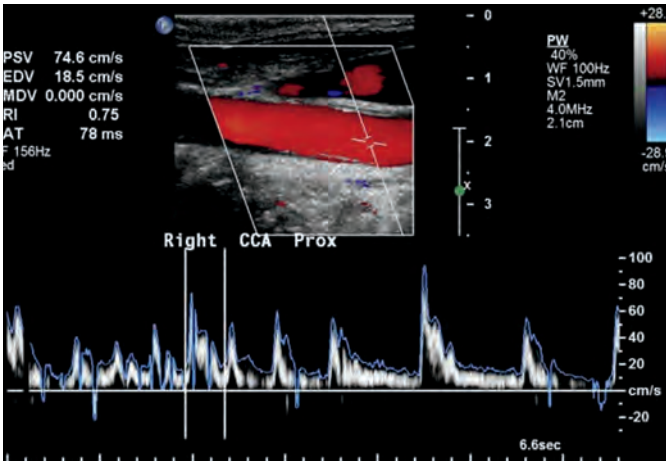


Figure 2-23. Severely irregular heart rhythm provides significant challenges in determining a reliable peak systolic velocity measurement.

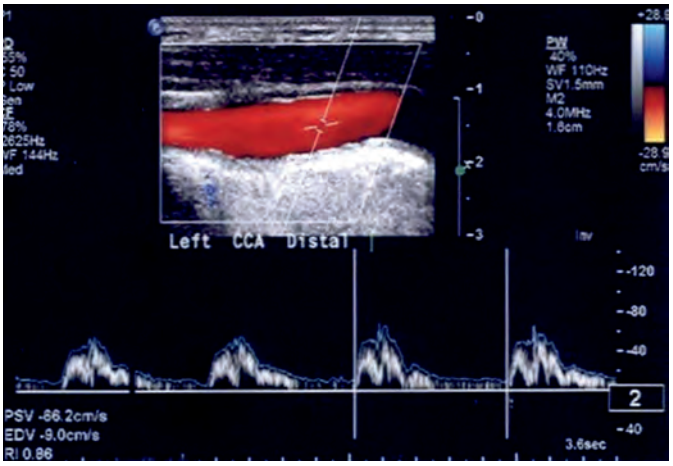


Figure 2-24. A delay in the systolic upstroke is evident with a stenosis of the aortic valve. A decrease in peak systolic velocities is also usually seen.

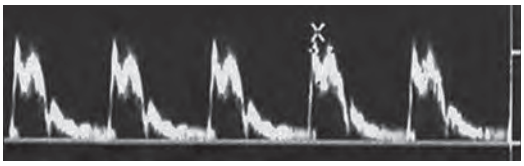


Figure 2-25. Double systolic peak (pulsus bisferiens) in the common carotid artery of a patient with aortic regurgitation/insufficiency.

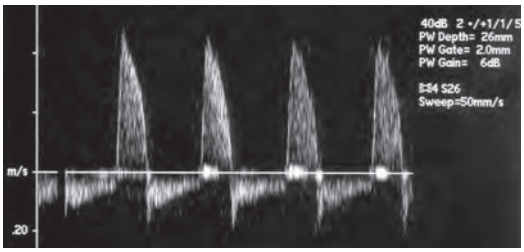


Figure 2-26. Pandiastolic flow reversal in the subclavian artery of a patient with aortic regurgitation/insufficiency.

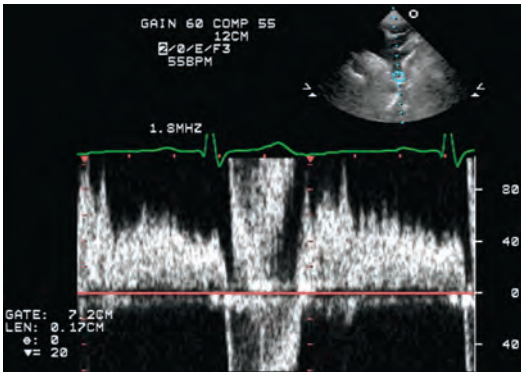


Figure 2-27. Significant flow reversal in the proximal aorta of a patient with severe aortic regurgitation/insufficiency.

Regurgitation/Insufficiency of the Aortic Valve

- A double systolic peak, sometimes referred to as *pulsus bisferiens*, may be evident (Figure 2-25). However, a similar finding, i.e., a second systolic peak, may be a normal finding in a young patient secondary to vessel wall compliance. In addition, one author* suggests that there may be a relationship between a double systolic peak and aortic dissection.
- Diminished diastolic flow or even reversed flow throughout diastole may also be documented on spectral analysis (Figures 2-26 and 2-27).

High Cardiac Output

- A systemic increase in peak systolic velocities may normally be evident in younger, athletic individuals or physically fit adults (Figure 2-28). A decrease in end-diastolic velocities may also be evident in a low-resistance vessel, e.g., the internal carotid artery. As previously mentioned, a double systolic peak may also be a normal finding in a young patient secondary to vessel wall compliance (Figure 2-25).

*Burgess W: Recommended protocol for duplex ultrasound in common carotid artery dissection extending from the aortic arch. Paper presented at the Annual Conference of the Society for Vascular Ultrasound, San Francisco, California, May 2013.

chronic obstructive pulmonary disease, primary pulmonary hypertension, and renal failure. It should be noted that elevated right heart pressure may also be related to a pulmonary embolism. In addition, it may reflect a fluid overload condition not necessarily related to a disease process, such as overhydration.

COLLATERAL EFFECTS

In an extremity at rest, total blood flow may be fairly normal even in the presence of severe stenosis or complete occlusion of the main artery because of the development of a collateral network, as well as the aforementioned compensatory decrease in peripheral resistance.

To evaluate the approximate location of the obstructed artery, Doppler segmental pressures may be helpful.

Arterial obstruction may alter flow in nearby or more distant collateral channels, increasing volume flow, reversing flow direction, increasing velocity, and/or altering the pulsatility of the waveform. Note in Figure 2-38 the low-resistance quality of the spectral waveform.

The location of collateral vessels helps to provide a tentative indication of the obstruction level, as demonstrated in Figure 2-39.

Secondary collateral changes (such as evidence of tissue healing or granulation, increased capillary refill, and decreased symptomatology) also provide some limited information regarding the adequacy of a collateral system that has evolved in response to arterial obstruction.

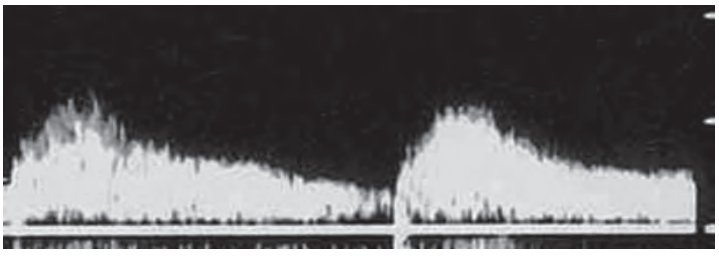


Figure 2-38. With a proximal superficial femoral artery (SFA) occlusion, flow in this popliteal artery is reconstituted via collaterals. Because flow is collateral-based and moving into a vasodilated vascular bed, its quality is of low resistance.

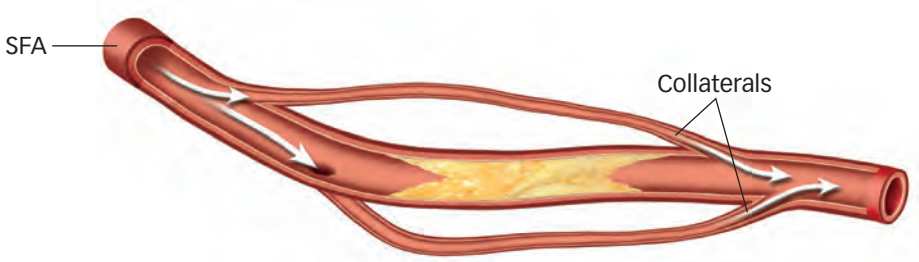


Figure 2-39. Representation of an occluded superficial femoral artery (SFA) with reconstituted flow distally due to collateralization. Arterial branches that were small are now taking a larger portion of the arterial flow, actually bypassing the obstruction in order to provide needed arterial flow to the foot.

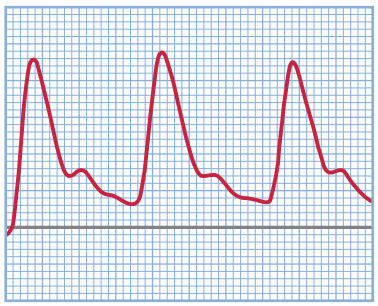


Figure 2-40. Because exercise produces a demand for blood to the muscles, a normally high-resistance arterial Doppler signal becomes low-resistance. In this analog waveform, the usual reversal below the baseline is seen as a forward reflection instead. This finding is quite normal after exercise.

EFFECTS OF EXERCISE

Exercise should induce peripheral vasodilation in the microcirculation so that distal peripheral resistance diminishes and blood flow markedly increases.

Peripheral resistance—the resistance to blood flow caused by the ever-decreasing size of the vessels, especially in the microcirculation—changes in response to a variety of stimuli such as heat, cold, tobacco use, and emotional stress.

Vasoconstriction and vasodilation of the blood vessels within skeletal muscles are also influenced by sympathetic innervation fibers that function primarily to regulate body temperature.

Exercise is probably the best single vasodilator of high-resistance vessels within skeletal muscle.

Autoregulation also controls vasoconstriction and vasodilation. Autoregulation accounts for the ability of most vascular beds to maintain a constant level of blood flow over a wide range of perfusion pressures.

- Autoregulation does not function normally when perfusion pressure drops below a critical level.
- High-resistance vessels constrict in response to increased blood pressure and dilate in response to decreased blood pressure.

By decreasing resistance in the working muscle, exercise normally decreases reflection (flow reversal) of the Doppler flow signal in the exercising extremity. ***Example:*** A low-resistance, monophasic Doppler flow signal (Figure 2-40) may be present normally in an extremity artery after vigorous exercise because the exercise causes peripheral dilatation and reduced flow resistance. This same low-resistance monophasic pattern is also seen pathologically when peripheral dilatation occurs in response to proximal arterial obstruction (see Figure 2-38).

On the other hand, a high-resistance signal (Figure 2-41) may occur from normal (physiologic) vasoconstriction at the arteriolar level or from distal arterial obstruction.

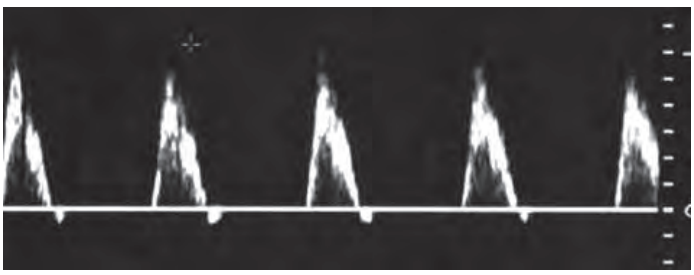


Figure 2-41. This high-resistance Doppler signal can occur with normal vasoconstriction at the arteriolar level. It can also occur proximal to distal arterial obstruction.

Additional notes:

- Proper characterization of velocity waveforms requires an understanding of both the normal flow characteristics of a particular artery and the physiologic status of the circulation supplied by the vessel.
- Questions need to be asked: Was the extremity cooled or warmed? Has it been exercised prior to the exam? Flow to a cool, vasoconstricted extremity will have pulsatile signals. Flow to a warm, vasodilated extremity will have continuous, steady signals.
- Proximal and distal pulsatility changes do not precisely differentiate between occlusion and severe stenosis.
- If good collateralization is present, proximal or distal Doppler velocity waveform qualities may not be altered.
- The distal effects of obstructive disease may be detectable in the presence of exercise or hyperemic evaluation.

EFFECTS OF STENOSIS ON FLOW

Laminar flow review:

- Laminar flow has an even distribution of frequencies at systole, with the lower frequencies distributed at the walls (the boundary layer) and the higher frequencies in center stream.
- Stable flow through a relatively straight vessel is usually laminar, the layers of fluid slipping over one another with minimal friction normally.

A hemodynamically significant stenosis causes a major reduction in volume flow and pressure. A stenosis usually becomes hemodynamically significant when the cross-sectional area of the arterial lumen is reduced 75%, which corresponds to a diameter reduction of 50%.

- Diameter reduction is a one-dimensional measurement (Figure 2-42).
- Area reduction is a two-dimensional measurement (Figure 2-43).
- In hemodynamically significant stenoses, both pressure and flow volume decrease.

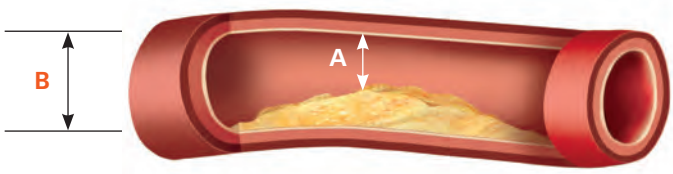


Figure 2-42. The nondiseased arterial segment is measured from wall to wall in a longitudinal approach (**B**) and then compared to the residual flow channel at the area of stenosis (**A**). A percentage diameter reduction is calculated.

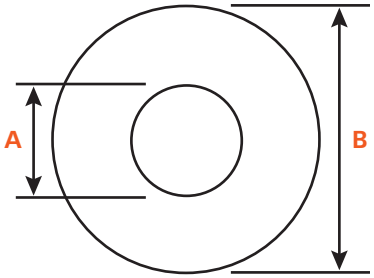


Figure 2-43. The original lumen (**B**) is measured and compared to the residual lumen (**A**) when the vessel is in a transverse approach. A percentage area reduction is calculated.

Vascular Technology

AN ILLUSTRATED REVIEW

5th EDITION

Claudia Rumwell, RN, RVT, FSVU | **Michalene McPharlin, RN, RVT, RVS, FSVU**

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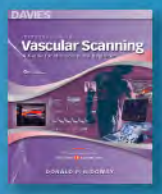

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Claudia Rumwell lectures on registry preparation, provides consulting services, and continues to write. She is a past member of the board of the Society for Vascular Ultrasound, is a recipient of the SVU Distinguished Service Award, and has been honored with the status of SVU Fellow. She previously served in the following roles for the Division of Vascular Surgery at Oregon Health & Science University: Technical Director of the Vascular Lab, Clinic Nurse Coordinator, Research Nurse Coordinator, and Instructor in Surgery.

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160 The ultrasound image below shows an internal carotid artery with:

A A calcified plaque

B An ulcerated lesion

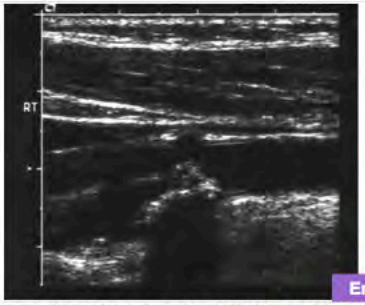
C A normal arterial wall

D An intraplaque hemorrhage

E A homogeneous plaque

Next | 160 of 665 | Prior | Bookmark/Skip | Pause | First | Last | Finish

Question Image 1



Enlarge

Question Image 2

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Exam Topic I. Cerebrovascular (25%–35% of ARDMS exam)

160 The ultrasound image below shows an internal carotid artery with:

A A calcified plaque

B An ulcerated lesion

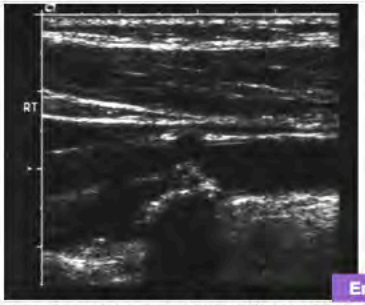
C A normal arterial wall

D An intraplaque hemorrhage

E A homogeneous plaque

Next | 160 of 665 | Prior | Bookmark/Skip | Pause | First | Last | Finish

Explanation Image 1



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Explanation Image 2

Enlarge

Exam Topic I. Cerebrovascular (25%–35% of ARDMS exam)

RIGHT!

You chose the correct answer, A: A calcified plaque

Note the brightly echogenic plaque and acoustic shadowing.

References

Zwiebel WJ: *Introduction to Vascular Ultrasonography*, 5th edition. Philadelphia, Elsevier Saunders, 2005, pp 157–167.

More Q&A Information

This question belongs to the following ARDMS exam topic:

I. Cerebrovascular [25–35% of ARDMS exam]

To focus only on this or other specific exam topics:

24 The great saphenous vein:

A Originates along the medial dorsum of the foot

B Passes superiorly, anterior to the medial malleolus

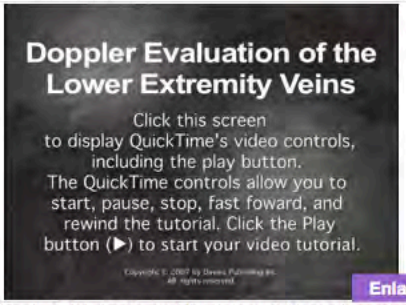
C Is accompanied by the saphenous nerve

D Receives tributaries from all surfaces of the lower extremity

E All are correct

Next | 24 of 665 | Prior | Bookmark/Skip | Pause | First | Last | Finish

Explanation Image 1



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Explanation Image 2

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Tutorial

Anatomy of the Lower Extremity Veins

by
David S. Sumner, MD

Unlike their arterial counterparts, the veins of the leg are divided into two systems: the superficial and the deep (Figures 1 and 2). [Click "More Images" on the purple navigation bar below to view figures.] The two principal veins in the superficial system are the greater (or long) saphenous vein and the lesser (or short) saphenous vein. Beginning just anteriorly and laterally to the medial malleolus, the great saphenous vein courses up the medial aspect of the calf and thigh to enter the common femoral vein at the groin (Figure 3–click "More Images"). The small saphenous vein, which is found posterior to the lateral malleolus, runs up the posterior aspect of the calf and terminates

Exam Topic I–IV. Anatomy, Physiology & Hemodynamics (4%–18% of ARDMS exam)

RESULTS FOR CURRENT TEST

Date: 5/12/2014
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Your results appear below in three categories:

(1) Overall results.
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This analysis also tells you how many questions (if any) you failed to answer and for which questions you changed your first answer. From this page you can also print your results, review wrong answers in Learn Mode, repeat the same test again, return to the Start Page for another activity, or Quit.

OVERALL RESULTS

Total Questions = 60
Total percentage correct = 18.33% / 11 of 60
Target time / actual time used = 60 minutes / 1.02 minutes
Number of unanswered questions = 0

SUBJECT AREA RESULTS

I–IV. Anatomy, Physiology & Hemodynamics = 33.33% / 1 of 3 (Unanswered 0)
I. Cerebrovascular = 15.38% / 2 of 13 (Unanswered 0)
II. Venous = 15.38% / 2 of 13 (Unanswered 0)
III. Peripheral Arterial = 18.18% / 2 of 11 (Unanswered 0)

SUBJECT AREA RESULTS

I–IV. Anatomy, Physiology & Hemodynamics = 33.33% / 1 of 3 (Unanswered 0)
I. Cerebrovascular = 15.38% / 2 of 13 (Unanswered 0)
II. Venous = 15.38% / 2 of 13 (Unanswered 0)
III. Peripheral Arterial = 18.18% / 2 of 11 (Unanswered 0)
IV. Abdomen and Visceral = 33.33% / 1 of 3 (Unanswered 0)
V. Miscellaneous Conditions & Tests = 33.33% / 1 of 3 (Unanswered 0)
VI. Quality Assurance = 0% / 0 of 2 (Unanswered 0)
VII. Image-Based Questions = 10% / 1 of 10 (Unanswered 0)
VIII. Physiology & Fluid Dynamics = 50% / 1 of 2 (Unanswered 0)

INDIVIDUAL QUESTION RESULTS

For your convenience, the following question numbers correspond to those in the book version of this mock exam. Use the CD and the book together for best results.

Question ID VTR583 = WRONG VII. Image-Based Questions
Question ID VTR426 = WRONG III. Peripheral Arterial (20%–30% of ARDMS exam)
Question ID VTR570 = WRONG VII. Image-Based Questions
Question ID VTR11 = WRONG I–IV. Anatomy, Physiology & Hemodynamics (4%–18% of ARDMS exam)
Question ID VTR446 = WRONG IV. Abdomen and Visceral (5%–15% of ARDMS exam)
Question ID VTR400 = WRONG III. Peripheral Arterial (20%–30% of ARDMS exam)
Question ID VTR403 = WRONG III. Peripheral Arterial (20%–30% of ARDMS exam)
Question ID VTR502 = WRONG VI. Quality Assurance (3%–5% of ARDMS exam)
Question ID VTR615 = WRONG VII. Image-Based Questions
Question ID VTR531 = WRONG VII. Image-Based Questions
Question ID VTR537 = WRONG VII. Image-Based Questions
Question ID VTR137 = WRONG I. Cerebrovascular (25%–35% of ARDMS exam)

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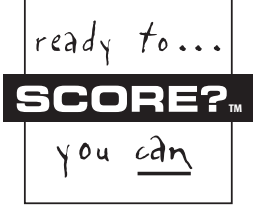

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2nd Edition

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HOW TO USE *SCORECARDS*

As part of our 1-2-3 Step Ultrasound Education and Test Preparation program, *ScoreCards for Vascular Technology* systematically prepares you to pass the Vascular Technology exam for the Registered Vascular Technologist (RVT) credential. It also helps you to master the facts, problem-solving skills, and habits of mind that form the foundation of success not only on your registry exams but also in your career as an ultrasound professional. And it's fun.

ScoreCards covers core concepts and principles topic by topic—facts you must master to pass the RVT exam. At the bottom of every question page is a "footer key," indicating the study topic's place within the exam coverage—from cerebrovascular to peripheral arterial, pathology to protocols—so you always know where you are and how you are doing. And at the end of the book you'll find a handy list of all the flashcards cross-referenced to the task-oriented ARDMS exam topics.*

In addition, all questions specifically designed to prep you for the ARDMS "Advanced Item Type" (AIT) questions are identified. This is a new class of question that tests practical sonographic skills by simulating hands-on clinical experience. (See "Examinations and Certifications" at www.ARDMS.org.)

*We use the last best ARDMS content outline for test preparation, updated to ensure complete coverage. The latest exam outlines from ARDMS provide a generalized categorical overview together with very specific clinical tasks, but they can miss key intermediate topics you must know to pass your exam—hence our hybrid approach to study outlines. Here you get it both ways: The table of contents reflects the key topics you need to know to pass the exam; at the end of the book, "*ScoreCards* Questions Cross-Referenced to the ARDMS Exam Content Outline" lists the questions under the ARDMS exam outline categories that were current as of press time.

The AIT questions include three types, which are identified where applicable in a ScoreCard's footer key as indicated in quotation marks below:

- **"AIT–SIC" (Semi-Interactive Console) items**: These questions require the examinee to use a semi-interactive console to correct a problem with the image presented. In the *ScoreCards* system, similar questions are addressed similarly, by asking candidates what is wrong with an image or how to correct a problem. SIC questions are currently used in the SPI exam. We have included some here to provide bonus Physics coverage for Vascular.
- **"AIT–Hotspot" items** require examinees to indicate the answer to a question by pointing at or marking directly on an image. In *ScoreCards*, similar questions ask examinees to indicate the label on an image that corresponds to the correct answer. Hotspot questions appear on the RVT specialty exam.
- **"AIT–PACSim" items**: For reading physicians taking the Physician in Vascular Interpretation (PVI) exam, there are the highly interactive case-based Picture Archive and Communication Simulation (PACSim) questions. These simulate a reading station and require examinees to read a patient's clinical history, evaluate existing image(s), and complete a diagnostic ultrasound report by selecting from the options presented.

ScoreCards for Vascular Technology also contains an image gallery of challenging case-based problems and coverage of physiology and fluid dynamics—the vascular-specific physical principles that you must know to pass the Vascular Technology exam. These physical principles are key to understanding the physiologic bases of the indirect vascular tests, Doppler technology and its clinical applications, and other clinically important issues and applications.

Here are some tips for maximizing the value of the *ScoreCards* system:

Take it with you. The pocket-sized *ScoreCards* study system is designed to be portable. Use it on breaks or between patients. You can review a dozen question/answer items in five minutes.

Study, test yourself, review. As you study vascular technology, *ScoreCards* drills you on key facts and figures, it tests your knowledge of those facts in practical situations, and it provides clear explanations and references for further study. Each Q&A card is keyed to a helpful study outline so that you always know where you are, how you are doing, and how important the topic is to your overall success on the exam.

Triangulate on your target. By itself, the *ScoreCards* study system is a powerful, convenient, and fun way of learning and testing yourself. It is especially effective when used with *Vascular Technology: An Illustrated Review* (Step 1: review text) and *Vascular Technology Review* (Step 2: mock examination). Just as each ScoreCard tells you which study topic it covers, it also indicates exactly where in the Step 1 text you can find further information about the subject. So do the Davies mock examinations. This integrated, systematic strategy triangulates on your target—exam and career success!

Shuffle it! After using the flipcard format for a while, consider removing the spiral binding and mixing up the cards to vary the order in which they challenge you.

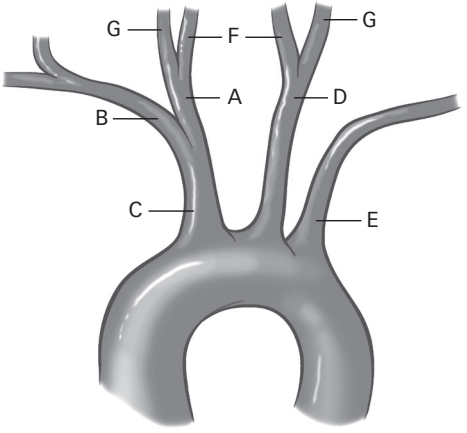
Earn CME credit. The *ScoreCards* study system is an SDMS-approved CME activity that can help meet the CME requirements necessary to maintain your registry status once you pass your exams. Use the CME application that follows the last question in this book. You may use the CME application anywhere, anytime, at your convenience.

Check our website. The latest news about your exams, continuing medical education, and diagnostic testing—as well as catalogs of additional resources and online help—is just a click away. Visit us at **DaviesPublishing.com**.

Whether you are a budding vascular technologist or a seasoned cross-training sonographer, mastery of these ScoreCards forms a solid foundation for success. For best results, we strongly urge you to combine *ScoreCards* with *Vascular Technology: An Illustrated Review* and either *Vascular Technology Review* (the book form of the mock exam) or *Vascular Technology CD-ROM Mock Exam*.

Q 1

Identify the vessels labeled A–G in this illustration of the aortic arch.



a. _____

b. _____

c. _____

d. _____

e. _____

f. _____

g. _____

DAVIES
Registry Reviews & Study Aids

A 1

A. Right common carotid artery.
B. Right subclavian artery
C. Innominate (brachiocephalic) artery.
D. Left common carotid artery.
E. Left subclavian artery.
F. External carotid arteries.
G. Internal carotid arteries.

This classic pattern of the aortic arch is seen in approximately 70% of individuals. The first of these branches is the innominate or brachiocephalic trunk, which usually courses 3–4 cm before dividing into the right common carotid and subclavian arteries. The second branch is the left common carotid artery. The common carotid arteries divide into the left and right internal and external carotid arteries. The last branch of the aortic arch is the left subclavian artery.

- Rumwell C, McPharlin M: *Vascular Technology: An Illustrated Review*, 5th edition. Pasadena, Davies Publishing, 2015, p 4.
- Kadir S: Regional anatomy of the thoracic aorta. In *Atlas of Normal and Variant Angiographic Anatomy*. Philadelphia, Saunders, 1991, pp 19–54.

The most common anatomic variant of the aortic arch is:

a. an origin of the left vertebral from the aortic arch
b. an origin of the right subclavian from the aortic arch
c. a common origin of the innominate and left common carotid arteries
d. an origin of the right common carotid from the aortic arch

A 2

C. A common origin of the innominate and left common carotid arteries.

A frequent origin of the innominate and left common carotid arteries is by far the most common variant anatomy of the aortic arch, occurring in approximately 22% of individuals.

▶ Kadir S: Regional anatomy of the thoracic aorta. In *Atlas of Normal and Variant Angiographic Anatomy*. Philadelphia, Saunders, 1991, pp 19–54.

After it crosses the lateral margin of the first rib the subclavian artery becomes known as the:

a. brachiocephalic artery

b. axillary artery

c. brachial artery

d. vertebral artery

B. Axillary artery.

The subclavian artery continues as the axillary artery after it passes the lateral margin of the first rib. The axillary artery in turn becomes the brachial artery.

- Rumwell C, McPharlin M: *Vascular Technology: An Illustrated Review*, 4th edition. Pasadena, Davies Publishing, 2009, p 4.
- Rumwell C, McPharlin M: *Vascular Technology: An Illustrated Review*, 5th edition. Pasadena, Davies Publishing, 2015, p 4.

ScoreCards for

Vascular Technology

Cindy Owen, RDMS, RVT, FSDMS | D. E. Strandness, MD

SECOND EDITION

The sophisticated ScoreCards flip- and flashcard study system yields maximum gain with minimum pain, and it's fun. Exercise your ability to think fast and recall key facts wherever you are—at lunch, on weekend outings, or between patients. Written by well-known experts, these handy ScoreCards deliver nearly 500 questions keyed to the registry's own exam outline, plus answers, explanations, and quick references. More than sixty image-based cases prepare you to tackle scans on the exam. Step 3 in Davies' CME-approved 1-2-3 Step Ultrasound Education and Test Preparation program, *ScoreCards for Vascular Technology* is very effective in combination with *Vascular Technology: An Illustrated Review* (Step 1—review text), *Vascular Technology Review,* and *Ultrasound Physics Review* (Step 2—mock exams).

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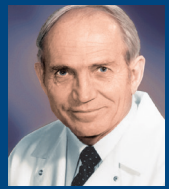

A pioneer in the development of duplex ultrasonography, the late Dr. Strandness played a seminal role in the first edition of this publication. He was professor of surgery and chair of the department of vascular surgery at the University of Washington.

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DAVIES