Chapter 17 – Blood Vessels, Blood Flow, Vascular Resistance, and Blood Pressure

Objectives

Given the synopsis in this chapter, competence in each objective will be demonstrated by writing short essays, drawing diagrams, and responding to multiple choices or matching questions, at the level of 85% or greater proficiency for each student.

A. To describe the anatomical features of the blood vessels and to explain how their organization and size affect the movement of blood through the systemic and pulmonary vessels.
B. To explain the change in the blood pressure that occurs as the blood moves through the blood vessels, and to describe the calculation of mean arterial pressure.
C. To define blood flow, to explain the flow of blood through vessels in series and in parallel, and to describe the calculation of blood flow.
D. To define vascular resistance, explain how blood vessel size influences vascular resistance, and describe the calculation of vascular resistance.
E. To explain how the size of blood vessels influences the distribution of cardiac output and the flow of blood to different organs.
F. To explain the effects of changes in cardiac output and total peripheral resistance on mean arterial pressure.
G. To explain the effects of changes in blood volume and vascular compliance on mean arterial pressure.
H. To explain how local factors control the size of blood vessels.
I. To explain how the sympathetic nervous system, the posterior pituitary, and the renin-angiotensin system controls the size of blood vessels.
J. To explain how baroreceptor reflexes control blood vessel size and regulate arterial pressure.
K. To name representative drugs acting on the blood vessels and to explain their actions.

As we saw in the previous chapter, the primary function of the cardiovascular system is to transport oxygen and nutrients to the tissues of the body, and to transport carbon dioxide and other metabolic byproducts away from these tissues. Cardiac pumping moves blood out of the heart, to the lungs, back to the heart, to the rest of the body, and back to the heart. Blood is transported, of course, through blood vessels.

Blood Vessels

Just like we saw for the pumping of blood through the heart, the flow of blood through the blood vessels can be viewed simply as the movement of blood from an area of higher pressure to an area of lower pressure.

- Blood flows from an area of higher pressure to an area of lower pressure.
- Blood flows most through the path of least resistance.

Anatomical features of blood vessels

The flow of blood through the circulatory system is shown in Figure 17.1.

- Arteries carry blood away from the heart.
- Arteries branch to form more numerous but smaller arterioles.
- Arterioles branch to form more numerous but smaller capillaries.
- Capillaries come together to form venules.
- Venules come together to form veins.
- Veins carry blood back to the heart.

The general organization of an artery, capillary and vein is shown in Figure 17.2. Arteries and arterioles have a relatively thick wall with a thick layer of smooth muscle (tunica media). Arteries generally have more elastic connective tissue than arterioles. Capillaries are composed of just endothelium. Veins have a relatively thin wall with a thin layer of smooth muscle (tunica media) and venules have little if any smooth muscle.

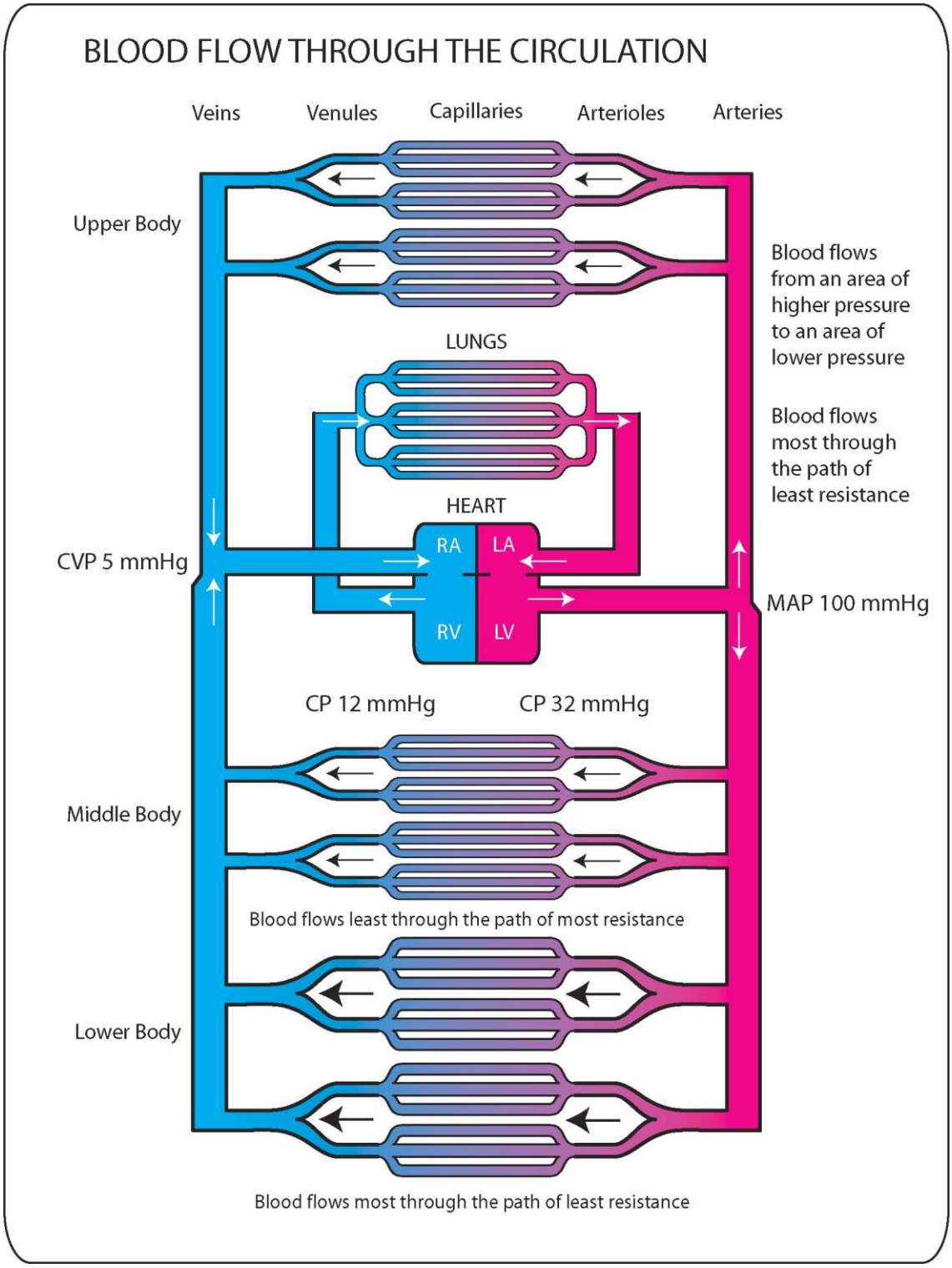


Figure 17.1 © 2007 David G. Ward, Ph.D.

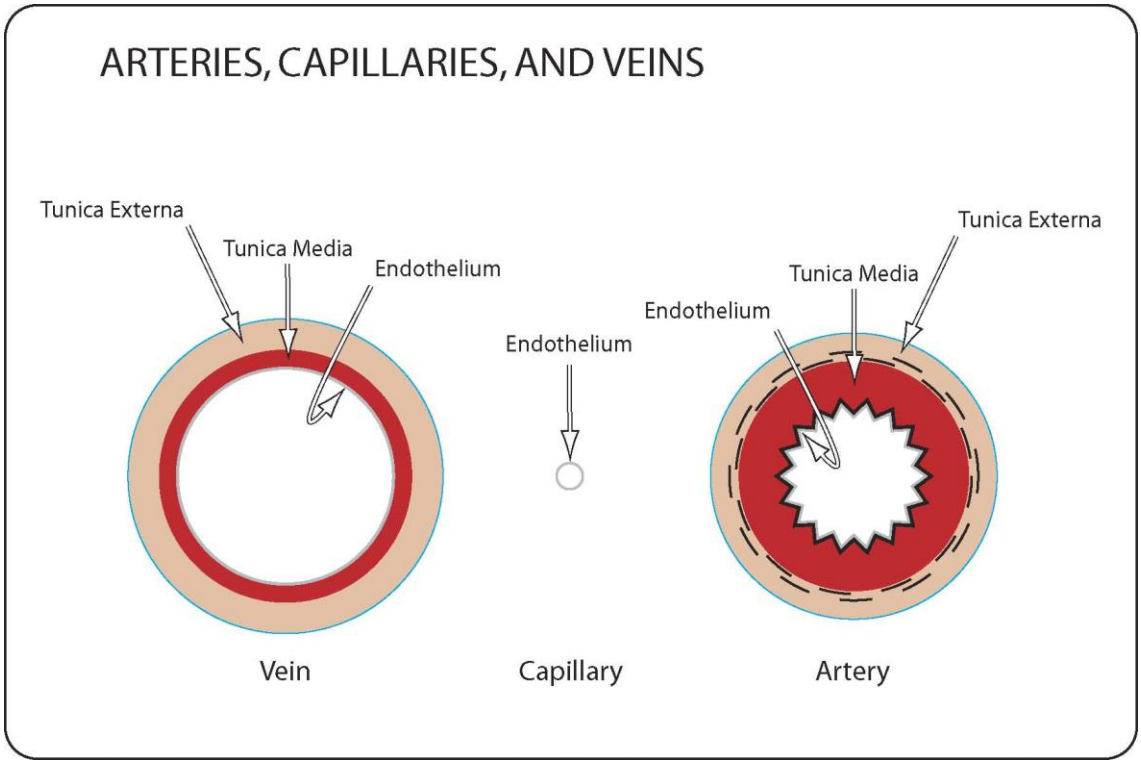


Figure 17.2 © 2007 David G. Ward, Ph.D.

Movement of blood through the vessels

Although the flow of blood out of the ventricles is intermittent, movement through the blood vessel is relatively continuous. This is largely due to the fact that the aorta and arteries distend during ventricular contraction (systole) and the elastic tissue recoils during ventricular relaxation (diastole). There is relatively little frictional resistance in the aorta and its arterial branches and blood moves rapidly through with little drop in pressure.

However, in the small arteries and arterioles, resistance to blood flow is large and the drop in pressure as blood moves through these vessels is also large. Blood will flow preferentially to the tissues and organs with the least resistance. The smooth muscle in the arterioles plays a critical role in controlling tissue blood flow and in controlling arterial pressure. The arteriolar smooth muscle just prior to a group of capillaries is sometimes referred to as a pre-capillary sphincter.

As the blood vessels branch going from the arteries to the capillaries, the cross sectional area of the blood vessels increases. This increase becomes very dramatic in the capillaries as shown in Figure 17.3. Due to the large cross sectional area blood moves very slowly through the capillaries which provide an ideal environment for the exchange of oxygen, nutrients, carbon dioxide, and metabolic byproducts between the blood and the tissues. As the blood vessels come together going from the capillaries to the veins,

the cross sectional area of the blood vessels decreases, and blood moves more rapidly back to the heart.

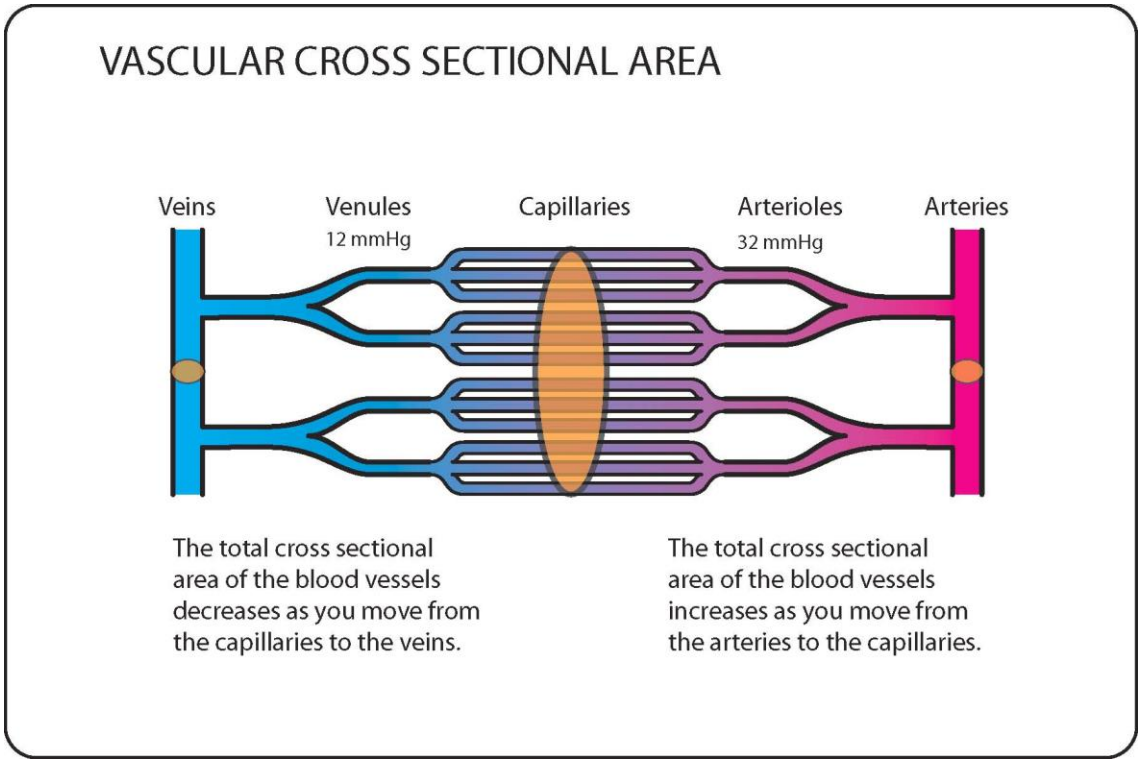


Figure 17.3 © 2007 David G. Ward, Ph.D.

Blood Pressure

As just noted there is little drop in pressure as blood moves through the aorta and its arterial branches. However, as the blood moves through the small arteries and arterioles there is a large drop in pressure. The pressure of the blood as it moves from the aorta to the vena cava is illustrated in Figures 17.1 and 17.4. The mean arterial pressure (MAP) in the aorta is about 100 mmHg and the central venous pressure (CVP) in the vena cava is about 5 mmHg. The capillary pressure (CP) starts at about 32 mmHg and ends at about 12 mmHg. Although not shown, the mean pulmonary arterial pressure is about 15 mmHg and the pulmonary venous pressure is about 5 mmHg.

Because the flow of blood out of the heart is intermittent, the pressure in the aorta and large arteries is pulsatile (refer to chapter 16, Figure 16.11). As a result, there is a systolic pressure that corresponds to ventricular contraction (systole), and a diastolic pressure that corresponds to ventricular relaxation (diastole). As the blood moves into the smaller arteries and arterioles, the pressure becomes progressively less pulsatile as the blood vessels absorb the systolic energy. At this point only single pressures are evident. The pressure in the veins is not pulsatile.

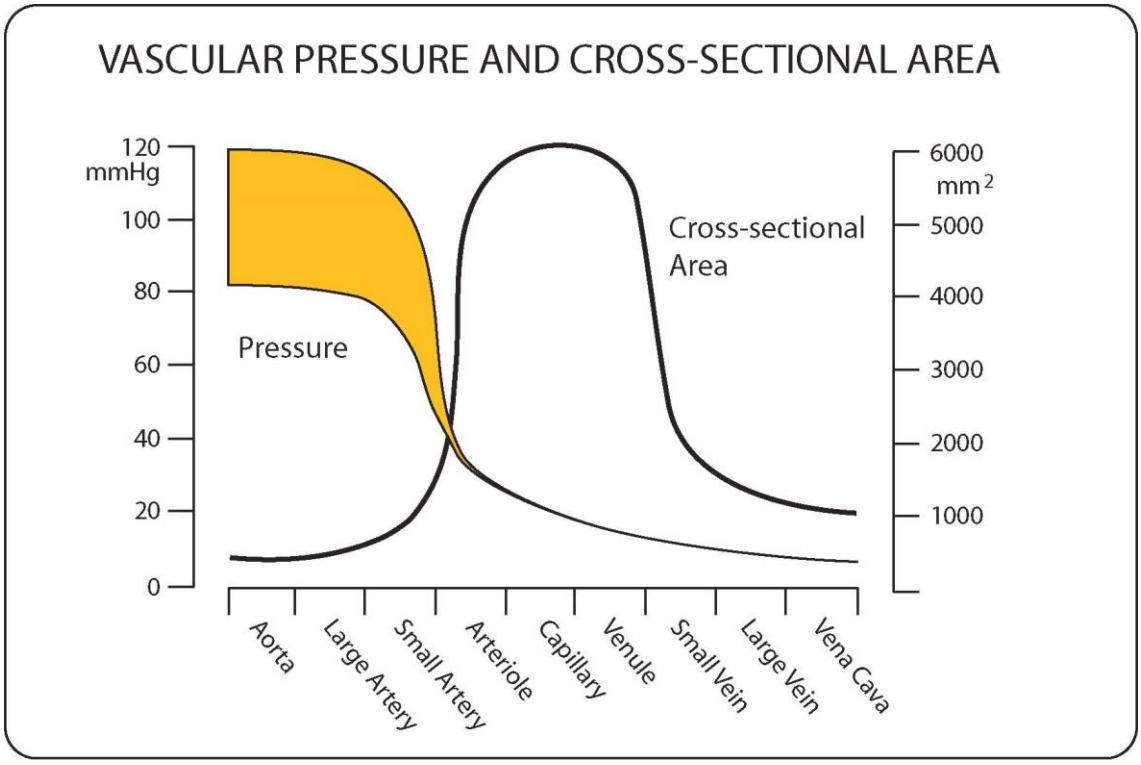


Figure 17.4 © 2007 David G. Ward, Ph.D.

The pulsatile pressure in the aorta and large arteries can be reduced to a single pressure by averaging the pressures over time. This is called the mean arterial pressure (MAP). When monitoring arterial pressure directly, mean arterial pressure is usually determined electronically by integration. When measuring arterial pressure indirectly (using a blood pressure cuff), mean arterial pressure is usually estimated from the systolic and diastolic readings. We need to examine a pressure tracing again to understand the procedure involved. As shown in chapter 16, Figure 16.11, only about 1/3 of each cardiac cycle corresponds to systole and the remaining 2/3 corresponds to diastole. Accordingly, mean arterial pressure is estimated as:

$$MAP = \frac{SYSTOLIC + (2 \cdot DIASTOLIC)}{3}$$

In an average person the systolic pressure will be about 120 mmHg and the diastolic pressure will be about 80 mmHg. From these values an estimate of MAP can be calculated.

$$MAP = \frac{120 + (2 \cdot 80)}{3}$$

$$MAP = 93 \text{ mmHg}$$

Mean arterial pressure is a good measure of the pressure that drives the blood through the blood vessels, and of the stress that the pressure exerts on the cardiovascular system.

Blood Flow

Blood flow (F) is usually expressed as mL/min or L/min. Blood flow through a blood vessel is dependent on the pressure driving the blood and the resistance of the vessel (R), as shown in Figure 17.5. The pressure driving the blood (ΔP) is the difference between the higher pressure (P_{in}) at one end of the vessel and the lower pressure (P_{out}) at the other end of the vessel. Formally, blood flow (F) equals the pressure driving the blood (ΔP) divided by the resistance of the blood vessel (R).

$$F = \frac{P_{in} - P_{out}}{R} = \frac{\Delta P}{R}$$

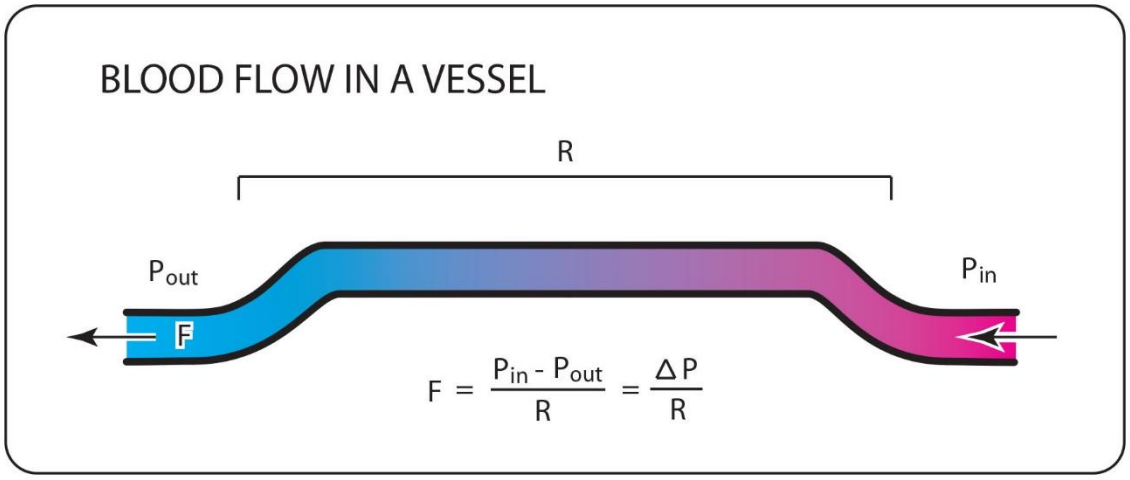


Figure 17.5 © 2014 David G. Ward, Ph.D.

Regional blood flow

We can apply this general equation to the calculation of blood flow in any organ, vascular bed, or blood vessel, or to the calculation of cardiac output. Let us look at the effect of constriction in a blood vessel on flow, as shown in Figure 17.6

The blood has to flow through the compressed segment before it can flow through the remaining segment that is in series. Therefore, the resistances add together. The total resistance (R_{total}) of the blood vessel simply equals the resistance of the compressed segment (R_1) plus the resistance of the remaining segment (R_2). The driving pressure equals the incoming pressure (P_{in}) minus the outgoing pressure (P_{out}). Therefore the flow out of the blood vessel is:

$$F_{out} = \frac{P_{in} - P_{out}}{R_1 + R_2}$$

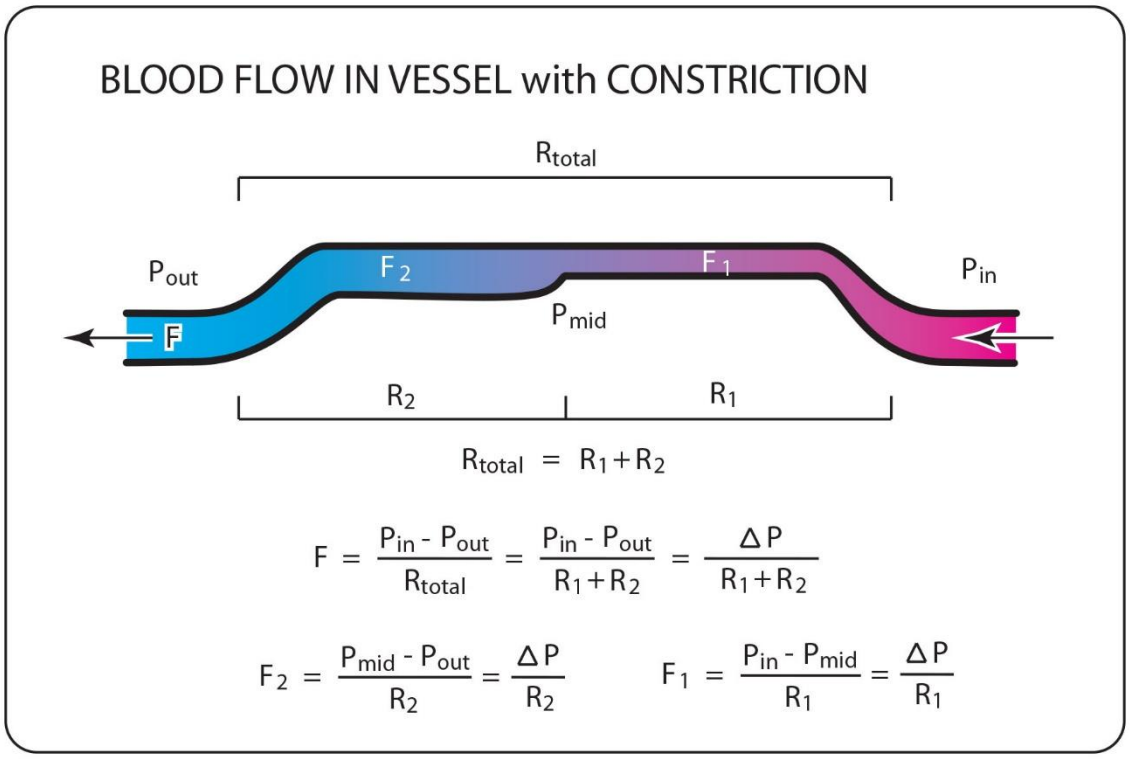


Figure 17.6 © 2014 David G. Ward, Ph.D.

If desired, the flow through the first segment and through the second segment could be calculated independently. Such a calculation would require knowing the pressure at the transition between the compressed and the remaining segment.

Vascular resistance

The general equation for the calculation of blood flow is easily rearranged to obtain a better appreciation of the concept of resistance.

$$F = \frac{\Delta P}{R} \quad ; \quad R = \frac{\Delta P}{F}$$

Resistance is simply measured as the change in pressure for a given change in flow. Pressure is usually measured in mmHg, and flow is usually measured in mL/min or L/min. Therefore, resistance is expressed as mmHg/ mL/min or mmHg/ L/min.

As we have shown in our discussions of blood vessel anatomy, resistance increases as blood vessels decrease in size. Conversely, resistance decreases as blood vessels increase in size. Vascular resistance (R) is affected by the viscosity of the blood (η), the length (L) of the blood vessel, and the radius (r) of the blood vessel. From the following relationship, we see that resistance (R) is inversely proportional ($\propto$) to the 4th power of the radius (r^4):

$$R = \frac{8\eta L}{\pi r^4} \quad ; \text{assuming } \eta L \text{ is constant:} \quad R \propto \frac{1}{r^4}$$

Accordingly, very small changes in the size of blood vessels will markedly change the vascular resistance. The radius of blood vessels, especially small arteries and arterioles, is affected by contraction or relaxation of the smooth muscle of the tunica media.

Cardiac output (blood flow out of the heart)

We can also apply the general equation for blood flow to the calculation of cardiac output. Cardiac output (CO) is defined as the amount of blood pumped out of the heart each minute. The amount of blood pumped out each minute is the flow (F); therefore, cardiac output is the flow of blood out of the heart. Accordingly, cardiac output can be expressed as the pressure between the aorta (mean arterial pressure (MAP)) and the vena cava (central venous pressure (CVP)) divided by the resistance of the systemic blood vessels. The expression total peripheral resistance (TPR) is often used to describe the resistance of the systemic blood vessels. Alternatively, the term total vascular resistance (TVR) is used.

$$CO = F = \frac{MAP - CVP}{TPR}$$

In an average 70 kg person the CO will be about 4.9 L / min, MAP will be about 100 mmHg and CVP will be about 5 mmHg. From these data TPR can be calculated and expressed in units of mmHg/L/min.

$$4.9 \text{ L / min} = \frac{100 \text{ mmHg} - 5 \text{ mmHg}}{TPR}$$

$$TPR = \frac{100 \text{ mmHg} - 5 \text{ mmHg}}{4.9 \text{ L / min}} = \frac{95 \text{ mmHg}}{4.9 \text{ L / min}} = 19.4 \text{ mmHg / L / min}$$

Vascular size and the distribution of cardiac output

Once the blood flows out of the left ventricle it will basically follow the path of least resistance. As illustrated in Figure 17.1, blood flows from an area of higher pressure to

an area of lower pressure, blood flows most through the path of least resistance, and blood flows least through the path of most resistance. As we have just recognized, small changes in the size of blood vessels can markedly change the vascular resistance and the flow of blood. As shown in the Table 17.1, exercise markedly affects the distribution of cardiac output to the various organs. Based on a relatively constant MAP of 100 mmHg we can calculate the vascular resistance (VR) of each organ.

In the resting state cardiac output is about 5 L/min. With strenuous exercise the cardiac output increased to about 17.5 L/min. Although the table is self-explanatory, it is remarkable to note that the skeletal muscle received 20% of the CO during rest and 73% during exercise. The kidneys received 20% of the CO during rest and only 3% during exercise. The GI organs received 24% of the CO during rest and only 3% during exercise. In response to exercise the vascular resistance of the heart, skeletal muscle and skin markedly decreased; and the vascular resistance of the kidney, GI organs, and other organs markedly increased. We therefore see vasodilation in the heart, skeletal muscle and skin; and vasoconstriction in the kidney, GI organs and other organs during exercise.

Table 17.1. Distribution of cardiac output during rest and strenuous exercise

Organ	Brain	Heart	Muscle	Skin	Kidney	GI organs	Other
Rest	13%	4%	20%	9%	20%	24%	10%
BF(L)	0.65	0.22	1.03	0.43	0.95	1.20	0.53
VR(mmHg/L/min)	154	454	97	232	105	83	189
Exercise	4%	4%	73%	11%	3%	3%	2%
BF(L)	0.75	0.75	12.50	1.90	0.60	0.60	0.40
VR(mmHg/L/min)	133	133	8	52	167	167	250

Blood Pressure Revisited

The importance of our general equation for blood flow becomes more apparent when we rearrange it and find that mean arterial pressure is equal to cardiac output multiplied by total peripheral resistance. This shows us that increases in either cardiac output or total peripheral resistance will increase arterial pressure.

$$CO = \frac{MAP - CVP}{TPR} \quad ; \quad MAP = (CO \times TPR) + CVP$$

We saw in chapter 16 that increases in heart rate and/or stroke volume can increase cardiac output. Such increases can raise mean arterial pressure. This is one of the major reasons why drugs that decrease heart rate and the force of cardiac contraction can lower arterial pressure.

More importantly, we now see that vasoconstriction or vascular obstruction can increase total peripheral resistance. Such increases increase mean arterial pressure. This is one of the major reasons why drugs that decrease vasoconstriction lower arterial pressure.

- Drugs that decrease vasoconstriction and/or blood volume are first-line treatments for high blood pressure.

Vascular Compliance and Blood Volume

Blood pressure is also affected by blood volume. In a way this is rather obvious, because without blood there would be no blood pressure. At any given vascular compliance (C), as blood volume (BV) increases, mean arterial pressure increases. Compliance is a description of the flexibility of the blood vessels measured as the change in volume for a given change in pressure. Compliance is usually expressed as mL/mmHg or as L/mmHg.

$$MAP = \frac{BV}{C} \quad ; \quad C = \frac{BV}{MAP}$$

About 80% of the blood volume is held in the veins. Contraction of the smooth muscle of the blood vessels, especially the veins, decreases the compliance. As the compliance decreases, mean arterial pressure will increase unless the blood volume is reduced.

We now see another reason why vasoconstriction can increase mean arterial pressure. Furthermore, we can see how drugs that cause diuresis and lower blood volume decrease arterial pressure.

Local, Neural, and Hormonal Control of Blood Vessels

Control of blood vessel size is critical for the delivery of blood to the tissues according to the needs of the tissues.

Local control of blood vessels

As shown in Figure 17.7, arteriolar vasodilation occurs in response to increased metabolic activity (this response is often called hyperemia). Arteriolar vasodilation occurs in response to decreased pressure or flow (this response is often called flow auto-regulation or reactive hyperemia). In either case

- Decreased oxygen and increased carbon dioxide and, H^+ leads locally to vasodilation
- Increases in many paracrines such as adenosine, nitric oxide, eicosanoids, bradykinin and histamine lead locally to vasodilation

In addition, blood vessels exhibit myogenic responses where arteriolar vasoconstriction occurs in response to vessel stretch or vessel damage.

- Vessel stretch or damage cause increased Ca^{2+} entry or release of endothelin-1 causing vasoconstriction

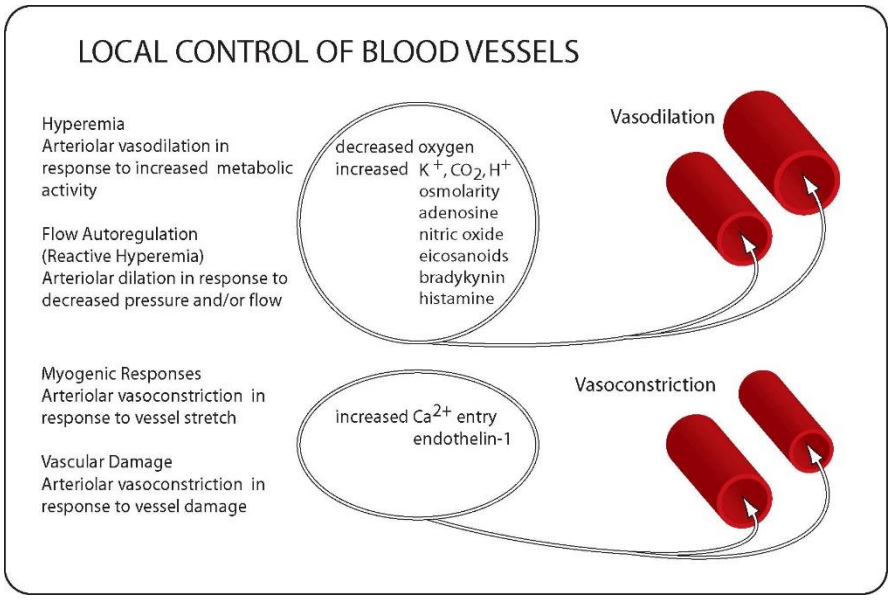


Figure 17.7 © 2007 David G. Ward, Ph.D.

Neural and hormonal factors

Postganglionic neurons of the sympathetic nervous system secrete norepinephrine and the adrenal medulla secretes epinephrine which acts on blood vessels to cause either vasoconstriction or vasodilation, as shown in Figure 17.8.

- Stimulation of alpha-1 receptors leads to vasoconstriction especially in blood vessels in abdominal organs, kidney, skin and genitals
- Stimulation of beta-2 receptors leads to vasodilation especially in blood vessels in skeletal muscle

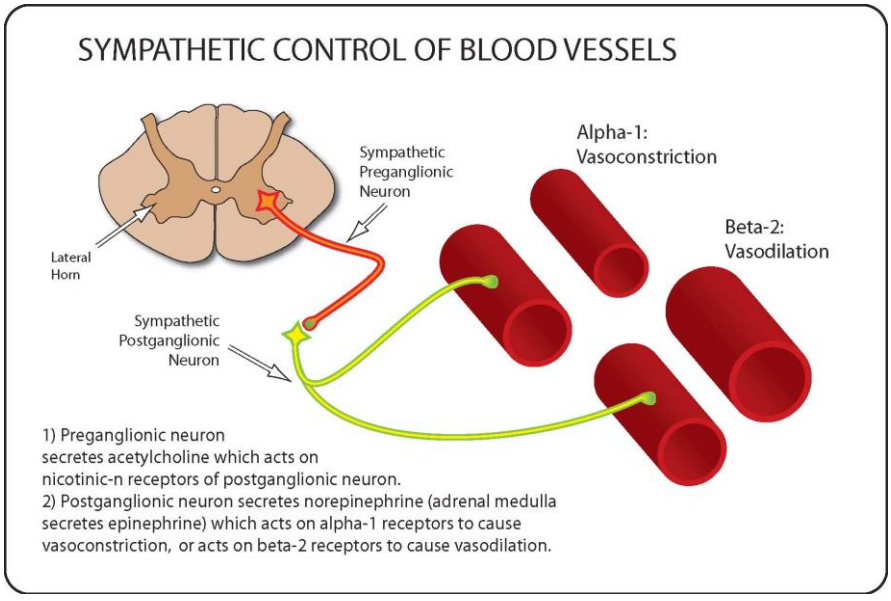


Figure 17.8 © 2007 David G. Ward, Ph.D.

The renin-angiotensin system produces angiotensin II which acts on blood vessels to cause either vasoconstriction (most common) or vasodilation, as shown in Figure 17.9. The liver produces angiotensinogen which circulates in the blood. The kidney secretes renin into the blood. The secretion of renin is controlled in part by norepinephrine acting on beta-1 receptors of the juxtaglomerular cells. Renin cleaves angiotensinogen into angiotensin I. The endothelium of blood vessels produces angiotensin converting enzyme (ACE) which converts angiotensin I to angiotensin II.

- Angiotensin II acts on AT-1 receptors (most common) to cause vasoconstriction of arterioles and pre-capillary sphincters
- Angiotensin II acts on AT-2 receptors to cause vasodilation

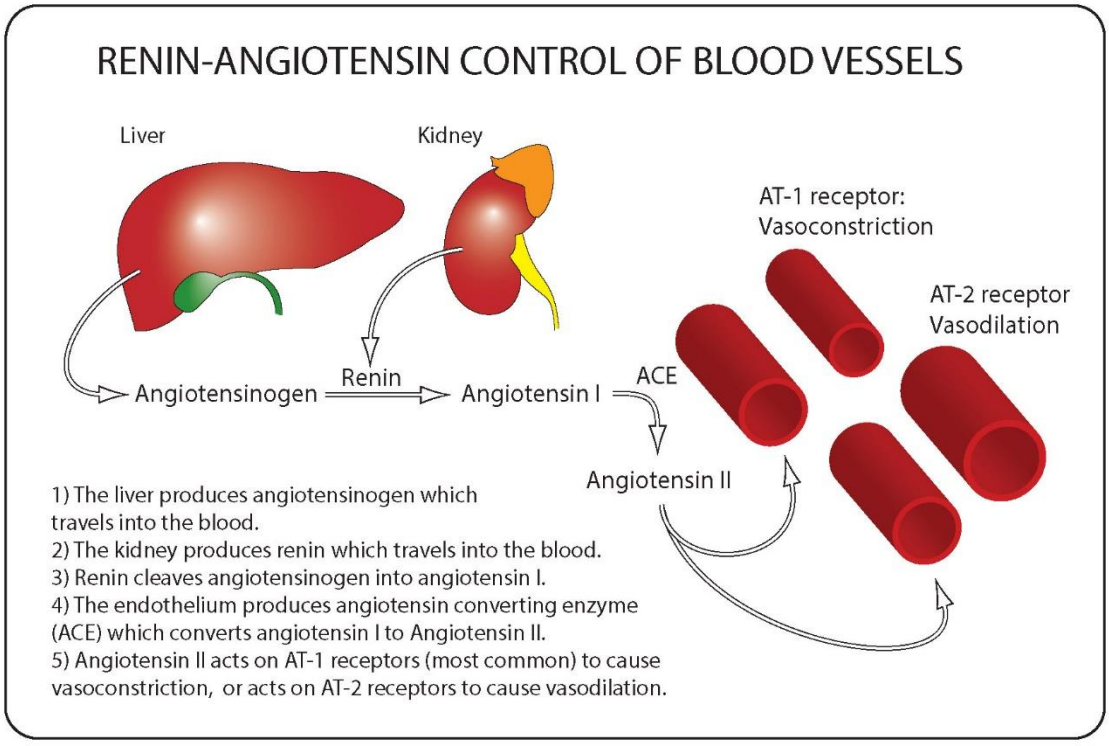


Figure 17.9 © 2018 David G. Ward, Ph.D.

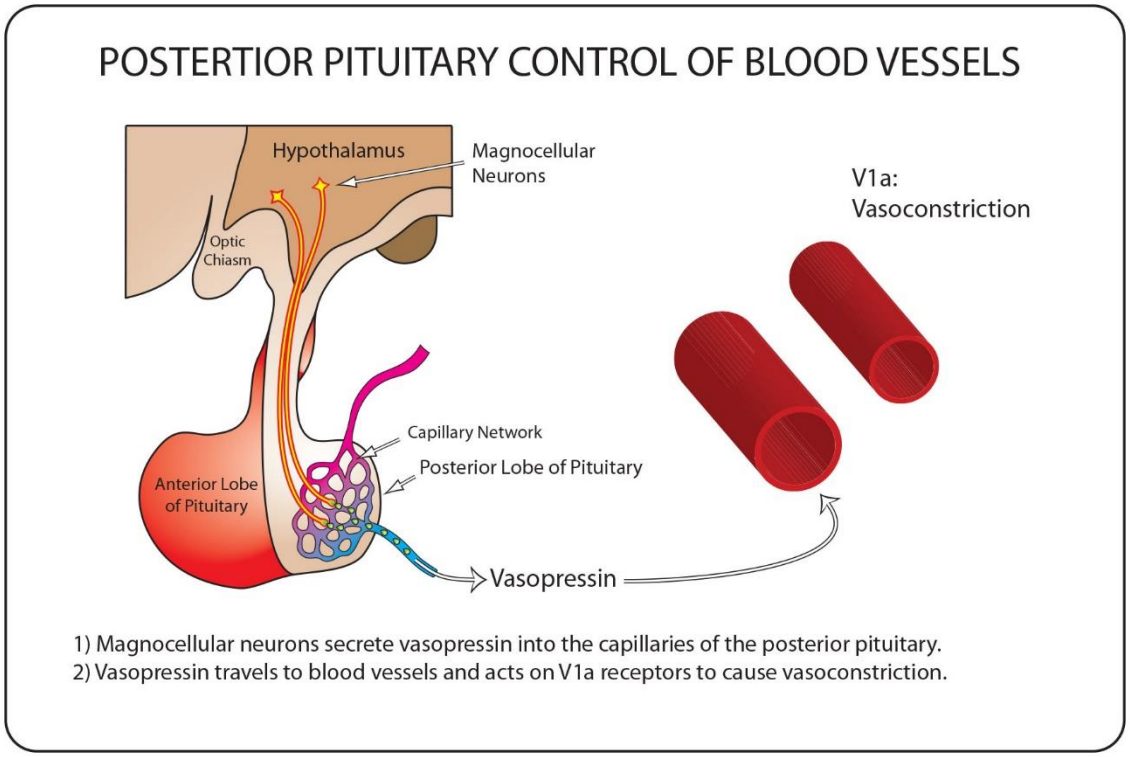
We now see how drugs that inhibit angiotensin converting enzyme or block AT-1 receptors decrease arterial pressure.

- ➢ Drugs that inhibit angiotensin converting enzyme (ACE inhibitors) or block angiotensin AT-1 receptors (ARB) are often the first-line treatments for high blood pressure.

Curiously, ACE inhibitors also inhibit the breakdown of bradykinin (enhancing vasodilation) and decrease the reabsorption of sodium (reducing blood volume).

The posterior pituitary produces vasopressin which acts on blood vessels to cause vasoconstriction, as shown in Figure 17.10.

- Vasopressin acts on V1a receptors to cause vasoconstriction of arterioles and pre-capillary sphincters



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Baroreceptor Reflexes - vascular control

The cardiovascular system contains sensory receptors that monitor blood pressures in the neck, trunk, and heart. Signals from these sensory receptor travel to the brainstem where they are compared to reference values. When necessary, cardiovascular responses are generated to normalize the pressures.

- Carotid sinus baroreceptors respond to pressure changes in the carotid arteries going to the head.
- Aortic arch baroreceptors respond to pressure changes in the aorta.
- Cardiac atrial stretch receptors respond to pressure changes in the cardiac atria.

Baroreceptor control of vasoconstriction by the sympathetic nervous system is shown in Figure 17.11. Decreases in carotid artery pressure "de-stimulate" the carotid sinus baroreceptors. The glossopharyngeal nerve carries the baroreceptor signal into the medulla of the brainstem and by way of interneurons stimulates the sympathetic nervous system which secretes norepinephrine, activates alpha-1 receptors and causes vasoconstriction. Baroreceptor control of vasoconstriction by the posterior pituitary is shown in Figure 17.12. Decreases in carotid artery pressure "de-stimulate" the carotid sinus baroreceptors. The glossopharyngeal nerve carries the baroreceptor signal into the medulla of the brainstem and by way of interneurons stimulates the hypothalamus and pituitary which secretes vasopressin, activates V1a receptors and causes vasoconstriction.

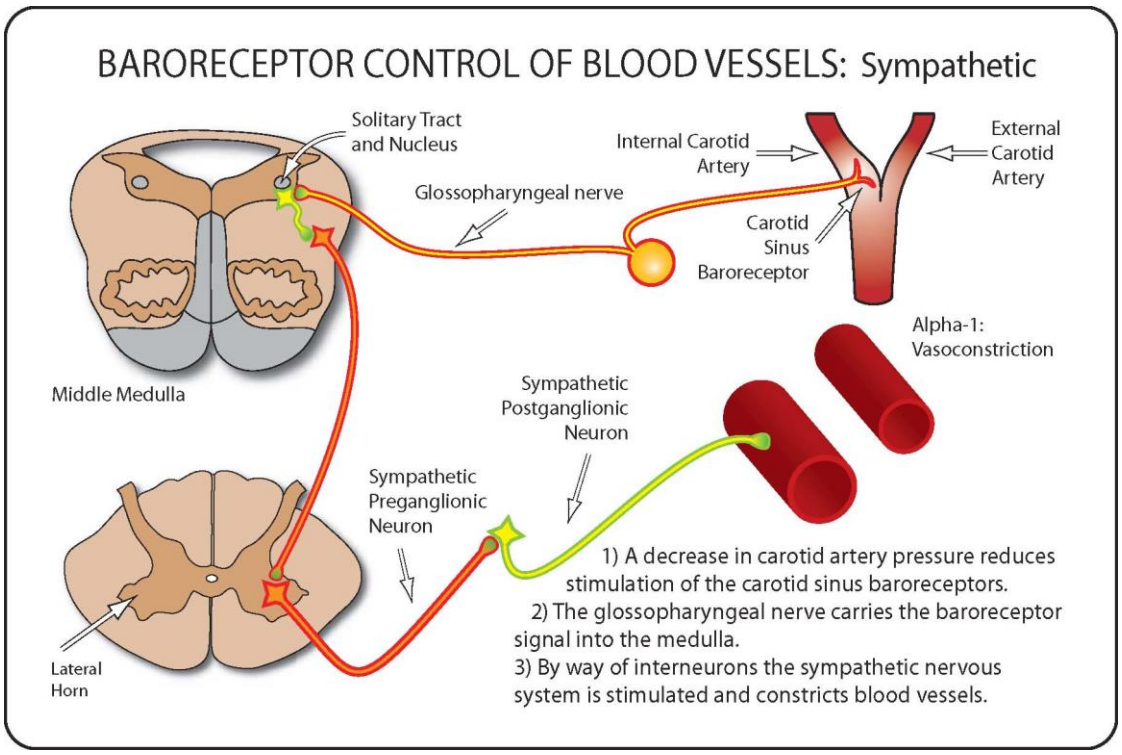

Figure 17.11 © 2007 David G. Ward, Ph.D.

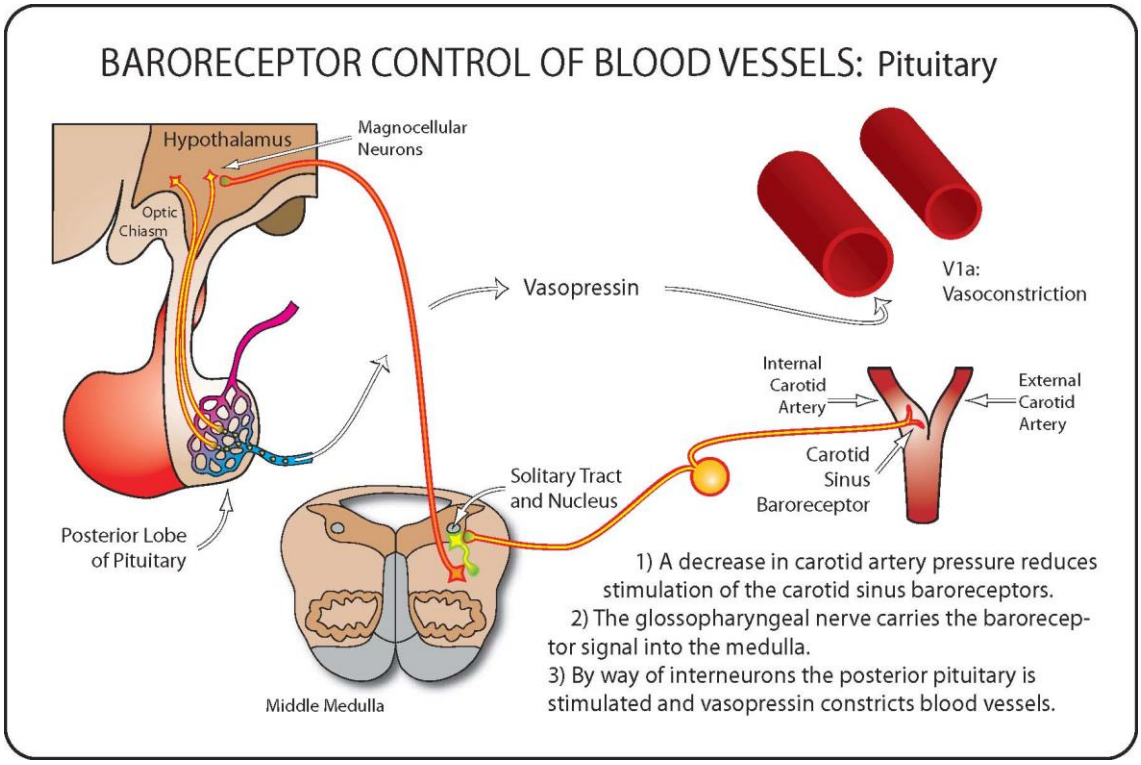

Figure 17.12 © 2007 David G. Ward, Ph.D.

Quiz Yourself

1-5. Matching (pick the nearest match)

A)	95 mmHg	Vena cava pressure	1) _____
B)	40 mmHg	Mean aortic pressure	2) _____
C)	32 mmHg	Pre-capillary pressure	3) _____
D)	12 mmHg	Post-capillary pressure	4) _____
E)	5 mmHg	Mean Ventricular pressure	5) _____

6-10. Matching

A)	MAP = 116.66	BP = 90/70	6) _____
B)	MAP = 97.50	BP = 120/80	7) _____
C)	MAP = 93.33	BP = 140/70	8) _____
D)	MAP = 80.00	BP = 110/85	9) _____
E)	MAP = 76.66	BP = 150/100	10) _____

11-15. Matching

A)	causes vasoconstriction	Adenosine and nitric oxide	11) _____
B)	causes vasodilation	Vasopressin acting on V1a receptors	12) _____
C)	none of the above	Angiotensin II acting on AT-1 receptors	13) _____
		Epinephrine acting on beta-2 receptors	14) _____
		Norepinephrine acting on alpha-1 receptors	15) _____

16-20.

A)	Total peripheral resistance (TPR)	CO/TPR	16) _____
B)	Mean arterial pressure (MAP)	COxTPR	17) _____
C)	Cardiac Output (CO)	MAP/CO	18) _____
D)	none of the above	MAP/TPR	19) _____
		MAPxTPR	20) _____

Fill in

21. Blood flow equals pressure in minus pressure out, divided by _____ _____.

22. As blood vessels get smaller the resistance of those vessels gets _____.

23. Norepinephrine leads to vasoconstriction due to activation of _____ receptors.

24. CO x TVR = _____.

25. _____ _____ _____ respond to changes in arterial pressure to the head.

Study Questions

1. Explain the role of blood vessel size in determining blood flow and the distribution of blood flow in the body.
2. Explain the role of the sympathetic nervous system and various hormones in the control of vasoconstriction.
3. Explain how the heart and blood vessels work together to influence blood pressure.
4. Explain how and why baroreceptor reflexes control heart rate, cardiac output, and vascular resistance.