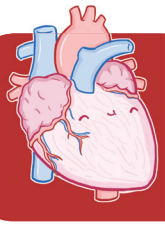

NOTES
HEMODYNAMICS

BLOOD PRESSURE, BLOOD FLOW, & RESISTANCE

osms.it/blood-pressure-blood-flow-resistance

PRESSURE (P)

- Force over area → blood pressure is force of blood over blood vessel surface area

BLOOD FLOW (Q)

- Volume (cm^3) blood flow through vessel over period of seconds (s)
- E.g. Q = $83cm^3/s$

Determined by two factors

- ΔP = Pressure gradient (mmHg); difference in pressure between two blood vessel ends
- R = Resistance (mmHg/mL per min)
  - $Q=\Delta P/R$
- Q directly proportional to pressure gradient
  - Increased pressure gradient → increased blood flow
- Q inversely proportional to resistance
  - Increased resistance → decreased blood flow

BLOOD FLOW VELOCITY (v)

- Major mechanism for changing blood flow is changing resistance
- Blood flow velocity (v) is distance (cm) traveled in certain amount of time (s)
- Using the equation for area (A) of a circle, $(d/2)^2 \times \pi$, we get $(2 / 2)^2 \times \pi = 3.14cm^2$
- Since cardiac output = blood flow → convert L/min to cm^3/s → $1000cm^3$ in a L, 60 seconds in a minute, multiplying those equals $83cm^3/sec$
- Rearranging formula, velocity equals flow rate divided by area, equals about 26cm/s, about 1km/hr

TOTAL PERIPHERAL RESISTANCE (TPR)

- Resistance of entire systemic vasculature
  - Can be measured by substituting cardiac output for flow (Q), pressure difference between aorta, vena cava for ΔP
- Resistance within an organ
  - Can be measured by substituting organ blood flow for flow (Q), pressure difference in pressure between organ artery, vein for ΔP

PRESSURES IN THE CARDIOVASCULAR SYSTEM

osms.it/cardiovascular-system-pressures

- Blood pressure highest in large arteries (e.g. brachial artery), about 120/80mmHg

SYSTOLIC BLOOD PRESSURE

- First/top number
- Pressure in aorta caused by ventricular contraction
- During systole, heart contracts → transfers kinetic energy (140mmHg) to blood → aortic elastic walls stretched, where some kinetic energy stored as elastic energy of walls (form of potential energy) → blood pressure drops to 120mmHg (systolic pressure)

DIASTOLIC BLOOD PRESSURE

- Second/bottom number
- Pressure caused by recoil of arteries during diastole
- During diastole, heart relaxes, aortic valves close → kinetic energy drops to 50mmHg → potential energy of stretched aortic walls adds to kinetic energy again when walls recoil → pressure rises to 60mmHg (diastolic pressure) → allows blood to move forward
- *Pulse pressure:* difference between systolic, diastolic pressure

Mean arterial pressure (MAP/P_a)

- Average blood pressure during cardiac cycle including systolic, diastolic blood pressure
- MAP, pulse pressure decline with distance from heart

MAP measured in two ways

- Diastole lasts longer than systole, therefore MAP is equal to one third systolic pressure plus two thirds diastolic pressure
  - *MAP = ⅓ systolic pressure + ⅔ diastolic pressure*
- For person with normal blood pressure of 120/80mmHg
  - *MAP= ⅓ 120 + ⅔ 80 = 93mmHg*
- Diastole lasts longer than systole; roughly equal to diastolic pressure plus one-third pulse pressure

$$MAP = Diastolic\ pressure + \frac{pulse\ pressure}{3}$$

- For person with normal blood pressure of 120/80mmHg

$$MAP = 80mmHg + \frac{120mmHg}{3} = 93mmHg$$

- MAP demonstrated using relationship of blood flow, blood pressure, resistance, applying the following equation
- $Q=\Delta P/R \rightarrow P_i - P_f = Q \times R$
  - P_i = mean arterial pressure (MAP)
  - P_f = central venous pressure (CVP)
  - Q = blood flow, equals cardiac output (CO)
  - R = resistance; combined resistance of all of blood vessels of systemic circulation equals systemic vascular resistance (SVR)
- Applying this equals the following
  - *MAP - CVP = CO x SVR*
- CVP is a small number, usually ignored; equation simplified
  - *MAP = CO x SVR*
- Based on this relationship → increased resistance will cause increased blood pressure

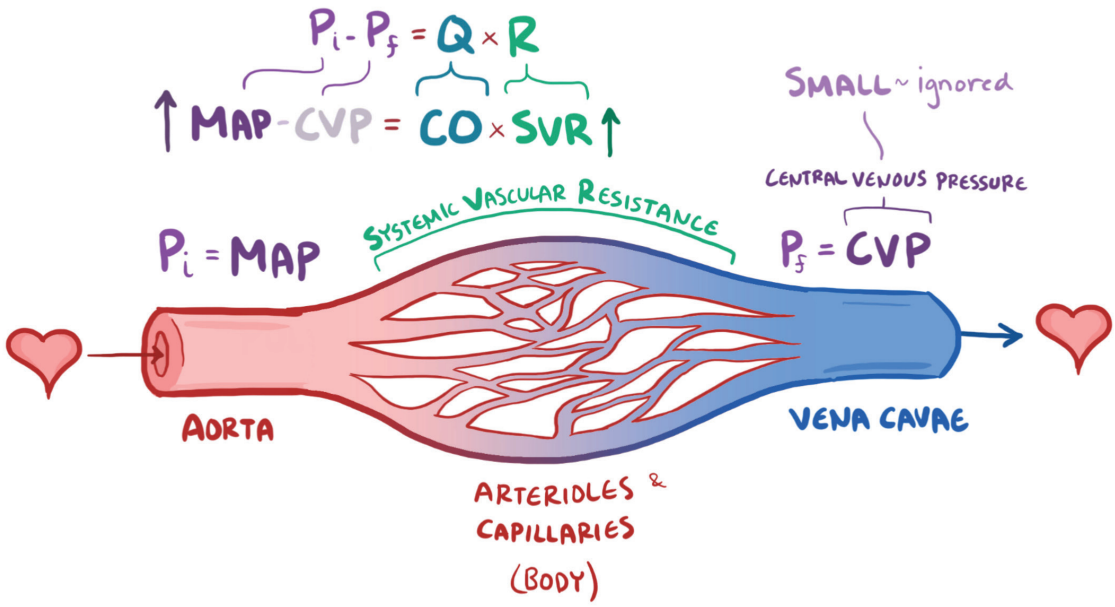

Figure 19.1 Visualization of MAP equation components.

PRESSURE GRADIENT

- Pressure gradient pressure difference between two ends of blood vessel
  - Gradient from aorta to arteriole ends
- Pressures in different parts of cardiovascular system not equal, keeps blood moving
- Blood flow generated by heart pumping action, moves along pressure gradient from high pressure areas (arteries) to low pressure areas (veins)
- Fluctuations on arterial side
  1. Blood ejected into aorta → pressure rises
  2. Small amount of blood backflows into ventricles
  3. Valves close → pressure drops
  4. Dicrotic notch/incisura pressure drop followed by small pressure increase as a result of valve recoiling
  5. Aorta settles, heart relaxes → pressure drops
- Pulse pressure lower in aorta than in large arteries → because pressure from blood travels faster than blood itself; pressure waves bounce off branch points in arteries which increases pressure even more
- Systolic pressure higher in large arteries than aorta, blood keeps moving forward
- Diastolic pressure is lower than in large arteries → mean arterial pressure mostly affected by diastolic pressure → mean arterial pressure is higher in aorta → driving force for blood flow
  - *For example:* aortic systolic pressure is 115mmHg; diastolic pressure is 85mmHg → Mean arterial pressure is 95mmHg; large artery systolic pressure is 120mmHg; diastolic pressure is 80mmHg → mean arterial pressure is 93mmHg

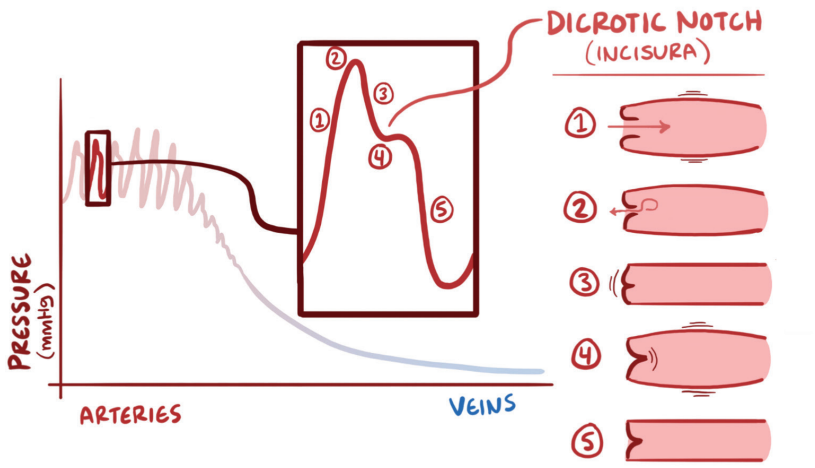

Figure 19.2 The five stages of fluctuation in arterial pulse pressure.

SYSTEMIC CIRCULATION

- Mean pressure in aorta results from two factors
  - Blood volume (cardiac output)
  - Compliance (low compliance → high pressure)
- Pressure remains high in large arteries because of high elastic recoil

Small arteries

- Pressure decreases; biggest pressure drop is in arterioles (30mmHg)
  - Occurs because arterioles develop high resistance to flow

Capillaries

- Pressure drops for 30mmHg to 10mmHg
- Two causes for pressure drop
  - Fluid filtration in capillaries
  - Increase frictional resistance
- Pressure drop is less than in arterioles
  - Many capillaries running in parallel → reduces total resistance (total resistance for vessels in parallel is less than resistance in any individual vessel)

Veins

- Systolic pressure drops even further → 4mmHg in vena cava, 2mmHg in right atrium
  - Venous pressure too low to promote venous return to heart
- Factors that facilitate venous return
  - *Muscular pump:* as muscles contract, relax they compress surrounding veins, force blood towards heart
  - *Respiratory pump:* during inhalation, abdominal pressure increases, forces blood in local veins forward
  - *Sympathetic vasoconstriction:* as smooth muscle in veins contracts, blood pushed towards heart

PULMONARY CIRCULATION

- Right ventricle → lungs → left atrium
- *Pulmonary arteries:* systolic pressure 25mmHg; diastolic pressure 8mmHg
  - Mean arterial pressure → 25 (⅓) + 8 (⅔) = 14mmHg
- *Capillaries:* pressure drops to 10mmHg
- *Pulmonary vein:* pressure drops to 8mmHg
- *Left atrium:* pressure drops to 2–5mmHg

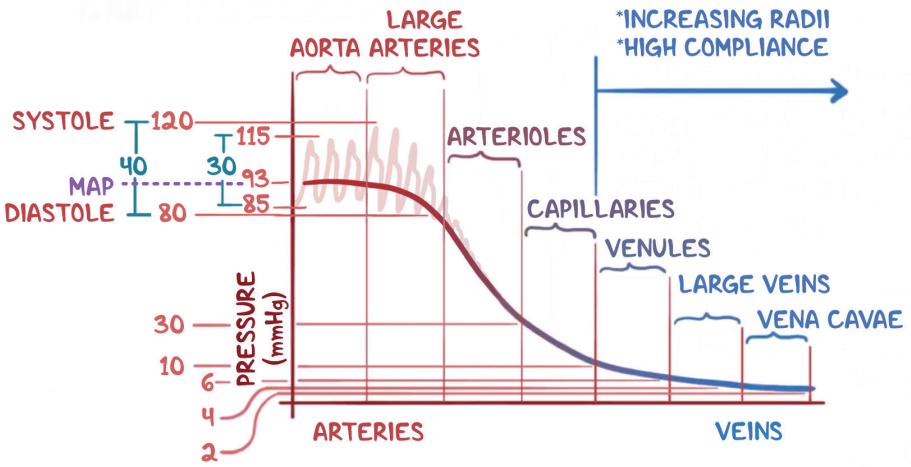

Figure 19.3 Visualizing pressures throughout the systemic cardiovascular system.

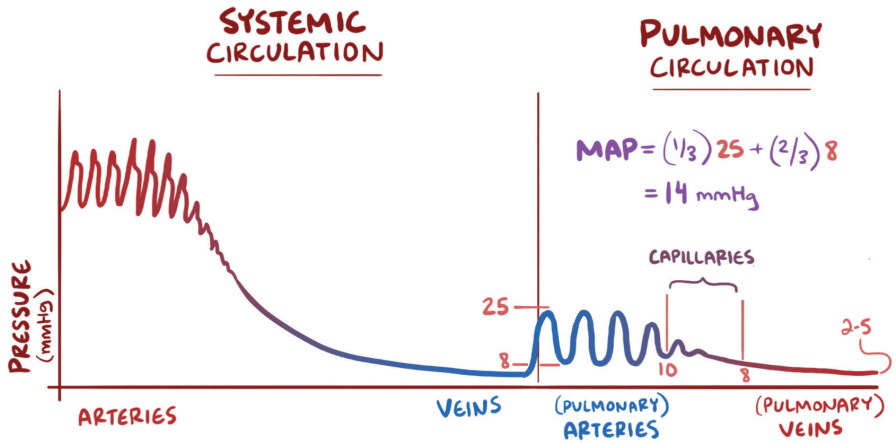

Figure 19.4 Visualizing pressures in the pulmonary circulation.

RESISTANCE TO BLOOD FLOW

osms.it/resistance-to-blood-flow

RESISTANCE

- Opposition to flow → amount friction as blood passes through blood vessels
- Determined by
  - Blood viscosity
  - Total length blood vessels
  - Diameter blood vessels

Poiseuille Equation

- Describes relationship between resistance, blood vessel diameter, blood viscosity

$$R = \frac{8\eta l}{\pi r^4}$$

  - R = resistance
  - η = blood viscosity
  - l = length of blood vessel
  - r^4 = radius (diameter) blood vessel raised to fourth power

Points expressed by Poiseuille equation

- Resistance to blood flow is directly proportional to blood viscosity, blood vessel length
- Resistance to flow is inversely proportional to radius to fourth power (r^4) → when radius decreases, resistance increases by fourth power → e.g radius decreases by one half, resistance increases 16-fold

SERIES & PARALLEL RESISTANCE

- Resistance also depends on blood vessel arrangement → series/parallel

Series resistance

- Sequential flow from one vessel to next
- Illustrated by arrangement of blood vessels within an organ
- Major artery → smaller arteries → arterioles → capillaries → venules → veins
- Total resistance of system arranged in series is equal to sum of individual resistances

$$R_{total} = R_{arteries} + R_{arterioles} + R_{capillaries} + R_{venules} + R_{veins}$$

- Blood flow at each part of system is identical but pressure decreases progressively (greatest decrease in arterioles)

Parallel resistance

- Simultaneous flow through each parallel vessel
- Illustrated by arrangement of arteries branching off aorta
- Cardiac output → aorta → branching → cerebral, coronary, renal system etc. → capillaries → venules → veins → vena cava → right atrium
- Total resistance less than any individual resistance

$$\frac{1}{R_{total}} = \frac{1}{R_1} + \frac{1}{R_2} + \frac{1}{R_3} + \frac{1}{R_4} + \frac{1}{R_5} + \ldots$$

  - Numbered subscripts represent cerebral, renal, coronary, other systems
- Blood flow in each system is only small portion of total blood flow → no pressure lost in major arteries (remains same as in aorta)

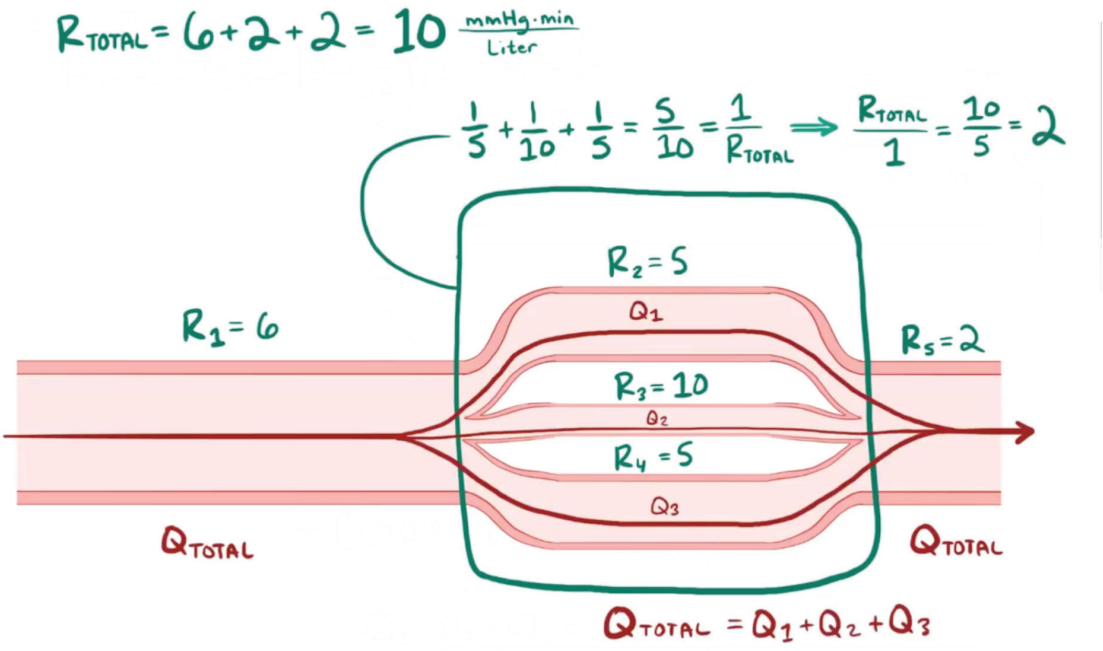

Figure 19.5 Calculating the total resistance for this system involves finding the total parallel resistance first and then adding R_1, $R_{Parallel}$, and R_5. The total blood flow in series, Q, is equal across all parts of the system. Individual vessels in the parallel system have different Qs, since the blood flow is split between each of the vessels, but they add up to Q_{Total}.

LAMINAR FLOW & REYNOLDS NUMBER

osms.it/laminar-flow-and-Reynolds-number

LAMINAR FLOW

- Smooth blood flow through blood vessels → blood velocity highest in center, lowest towards blood vessel walls → zero at walls

TURBULENT FLOW

- Laminar flow disrupted; blood flows axially, radially → kinetic energy wasted → more energy needed to drive blood

Reynolds Number

- Determines whether flow likely to be laminar/turbulent

$$N_R = \frac{\rho d v}{\eta}$$

  - N_R = Reynolds number
  - ρ = blood density
  - d = blood vessel diameter
  - v = blood flow velocity
  - η = blood viscosity
- As viscosity decreases (e.g. anemia), Reynolds number increases
- As velocity increases (e.g. increased cardiac output), Reynolds number increases
- Since velocity depends on diameter
  - $v = 4Q / \pi d^2$
  - Decrease in diameter (e.g. thrombus, atherosclerotic plaque) → velocity increases → Reynolds number increases
- Values of Reynolds number
  - If < 2000 → laminar flow
  - If > 2000 → increased likelihood of turbulent flow
  - If > 3000 → turbulent flow

SHEAR

- Friction between blood, vessel walls
  - Highest at vessel wall, lowest in center → difference in blood flow velocity
- Difference in velocity is parabolic → moving away from walls velocity increases quickly, near middle change in velocity low
- Shear inhibits red blood cell aggregation, lowers viscosity

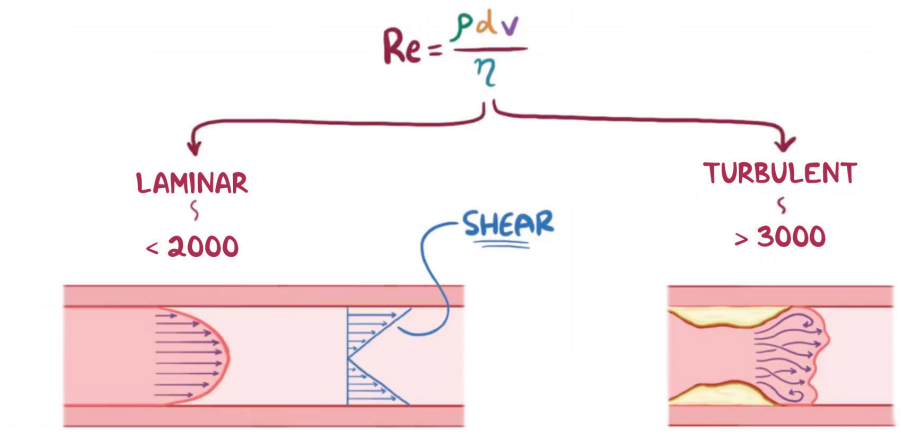

Figure 19.6 Reynolds number is a way to predict whether a fluid is going to be laminar (smooth) or turbulent. Differences in velocity across a blood vessel cause shear.

COMPLIANCE OF BLOOD VESSELS

osms.it/compliance-of-blood-vessels

COMPLIANCE (C)

- AKA capacitance/distensibility: ability of blood vessels to distend, hold an amount of blood with pressure changes
- $C = V / P$
  - C = compliance of blood vessel (mL/mmHg)
  - V = volume of blood (mL)
  - P = pressure (mmHg)
- High volume, low pressure → high compliance (veins); low volume, high pressure → low compliance (arteries)
- Arteriosclerosis → low compliance → low ability to hold an amount of blood at same pressure → blood backs up in veins
  - Arteries also become less compliant with age
  - If compliance decreases in veins (venoconstriction) → volume decreases (shift from veins to arteries)

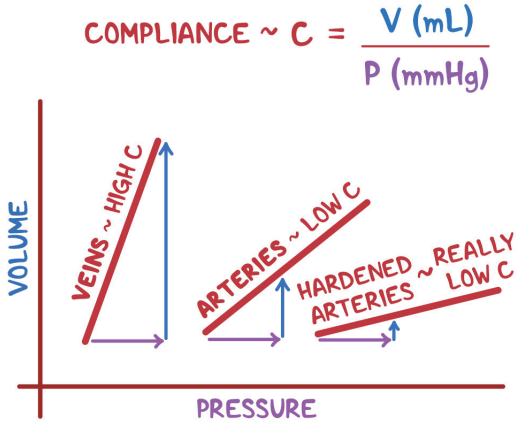

Figure 19.7 The same pressure will expand the volumes of vessels differently depending on their compliance.

ELASTANCE (E)

- Inverse of compliance
  - Blood vessel ability to recoil back after distension
- $E = P / V$
  - E = elastance of blood vessel (mmHg/mL)
  - P = pressure (mmHg)
  - V = volume of blood (mL)

During systole

- Heart contracts → transfers kinetic energy (140mmHg) to blood → stretches aortic elastic wall, where some kinetic energy stored as elastic energy of walls (form of potential energy) → blood pressure drops to 120mmHg (systolic pressure)

During diastole

- Heart relaxes, aortic valves close → kinetic energy drops to 50mmHg → potential energy of stretched aortic walls adds to kinetic energy again when walls recoil → pressure rises to 60mmHg (diastolic pressure) → allows blood to move forward during diastole
- *Pulse pressure:* 120mmHg - 60mmHg = 60mmHg
- Elastance buffers, dampens pulse pressure → Windkessel effect
- Without elastic properties, blood pressure would be 140/50mmHg with pulse pressure 90mmHg

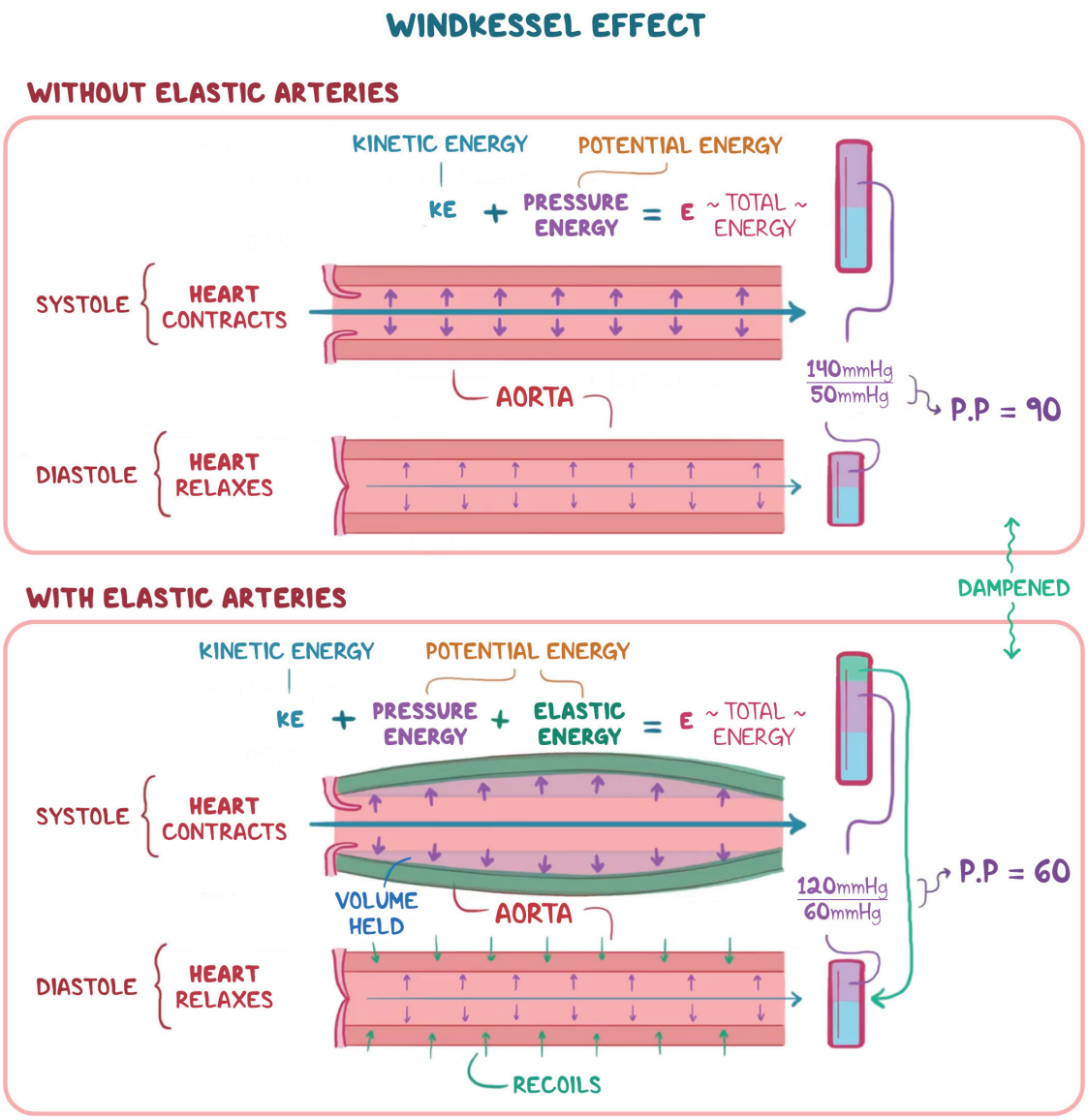

Figure 19.8 *Windkessel effect*: elastance dampens pulse pressure by lowering systolic pressure and increasing diastolic pressure.

Systole: aorta's walls stretch with high pressure contractions and store some energy as elastic energy. Since the total energy is the same as it would be without elastic arteries, there must be less kinetic energy and pressure energy to make room for the elastic energy → lower systolic blood pressure.

Diastole: elastic walls recoil, releasing the stored elastic energy and converting it to pressure energy and kinetic energy → more pressure energy.