Veins

- Venous system transports blood back to heart (VENOUS RETURN)
- Capillaries drain into venules
- Venules converge to form small veins that exit organs
- Smaller veins merge to form larger vessels
- Veins
  - Large radius offers little resistance to blood flow – high capacitance i.e. large change in volume with small change in pressure
  - Also serve as blood reservoir (fill up with blood)

Venous return

Cardiac output

Mean arterial blood pressure

Total

Heart rate

Stroke volume

Parasympathetic activity

Sympathetic activity and epinephrine

Blood volume

Respiratory activity

Passive bulk-flow fluid shifts between vascular and interstitial fluid compartments

Salt and water balance

Vasopressin, renin–angiotensin–aldosterone system

(Chapters 14 and 15)
Factors which enhance venous return

1. Driving pressure from cardiac contraction
2. Sympathetically induced venous venoconstriction
   ◦ Decrease venous COMPLIANCE ($\Delta V \Delta P$)
3. Skeletal muscle mechanical compression
   ◦ Effect of venous valves
4. Respiratory activity
5. Effect of cardiac suction
6. Blood Volume: Can’t pump out blood that doesn’t exist
At rest, >60% of blood is located in systemic veins, there decreasing capacity of veins has big effect on venous return and EDV

Mechanisms to Support Venous Return

- **Venous valves**
  - Mechanically prevent backflow of blood
- **Pressure imparted to blood by cardiac contraction**
  - Venous pressure gradient
- **Sympathetic vasoconstrictor activity**
  - Venous pressure gradient; venous capacity
- **Respiratory pump**
  - Pressure in chest veins; pressure gradient
- **Skeletal muscle pump**
  - Venous pressure gradient
- **Blood volume**
  - Venous pressure gradient
- **Passive bulk-flow shift of fluid from interstitial fluid into plasma**
- **Salt and water retention**

**KEY**

- = Short-term control measures
- = Long-term control measures

Fig. 10-23, p. 284
Pressure = 100 mm Hg

90 mm Hg caused by gravitational effect
10 mm Hg caused by pressure imparted by cardiac contraction

Pressure = 0 mm Hg

Pressure = 90 mm Hg
Supine position relieves much of gravitational effect on volume distribution – increases venous return and EDV

Elevating feet augments VR

Less than atmospheric pressure
Atmospheric pressure

Fig. 10-26, p. 286
Fig. 10-28, p. 287
Mean Arterial Pressure is regulated to support homeostasis –
MEAN ARTERIAL PRESSURE = CARDIAC OUTPUT X TOTAL PERIPHERAL RESISTANCE

- MAP is monitored by PRESSURE (stretch) RECEPTORS
  - Aortic arch (aortic baroreceptors)
  - Carotid arch (carotid baroreceptors)
  - Right Atria (atrial stretch receptors)
  - Kidney (juxtaglomerular cells)
- BARORECEPTORS send action potentials up afferent neurons that are integrated in the CARDIOVASCULAR CONTROL CENTER in the brain’s MEDULLA
- CARDIOVASCULAR CONTROL CENTER integration IMMEDIATELY alters efferent output to systemic effectors i.e. heart and vessels:
  - CARDIAC OUTPUT
    - HEART RATE
    - STROKE VOLUME
      - CONTRACTILITY (ALTER CONTRACTION POWER TO + OR - END SYSTOLIC VOLUME
  - TOTAL PERIPHERAL RESISTANCE
    - SYMPATHETIC EFFECT ON ARTERIOLE DIAMETER TO + OR - RESISTANCE
  - VENOUS RETURN
    - SYMPATHETIC EFFECT ON VENOUS COMPLIANCE TO + OR - VENOUS RETURN AND END DIASTOLIC VOLUME
- ATRIAL AND KIDNEY RECEPTORS SLOWLY SUPPORT BLOOD VOLUME AND indirectly effect TOTAL PERIPHERIAL RESISTANCE

Fig. 10-29, p. 288

Mean arterial blood pressure
Cardiac output
Heart rate
Stroke volume
Parasympathetic activity
Sympathetic activity and epinephrine
Venous Return
Local metabolic control
Extrinsic vasoconstrictor control
Number of red blood cells
Blood volume
Respiratory Activity
Skeletal Muscle Activity
Sympathetic activity and epinephrine
Vasopressin (ADH) and angiotensin II
Passive bulk-flow fluid shifts between vascular and interstitial fluid compartments
Salt and water balance
Vasopressin, renin–angiotensin–aldosterone system (Chapters 14 and 15)
Fig. 10-30, p. 290

Carotid sinus baroreceptor

Common carotid arteries
(Blood to brain)

Aortic arch baroreceptor

Aorta
(Blood to rest of body)

Neural signals to cardiovascular control center in medulla

Fig. 10-31, p. 290

Firing rate in afferent neuron arising from carotid sinus baroreceptor

Arterial pressure (mm Hg)

Decreased
Increased
Normal

Mean pressure

Time

Fig. 10-31, p. 290
Parasympathetic stimulation

Heart rate → Cardiac output → Blood pressure

Sympathetic stimulation

Heart rate

Cardiac output → Blood pressure

Arterioles

Vasoconstriction

Stroke volume

Total peripheral resistance

Blood pressure

Veins

Vasoconstriction

Venous return

Stroke volume

Cardiac output → Blood pressure

(b) Baroreceptor reflex in response to a fall in blood pressure

When blood pressure falls below normal → Carotid sinus and aortic arch receptor potential → Rate of firing in afferent nerves → Cardiovascular center

Sympathetic cardiac nerve activity and sympathetic vasoconstrictor nerve activity and parasympathetic nerve activity

Heart rate and stroke volume and arteriolar and venous vasoconstriction

Cardiac output and total peripheral resistance

Blood pressure increased toward normal

Fig. 10-32, p. 291

Fig. 10-33, p. 291
Blood Pressure

- Additional reflexes and responses that influence blood pressure
  - Left atrial receptors and hypothalamic osmoreceptors affect long-term regulation of blood pressure by controlling plasma volume
  - Chemoreceptors in carotid and aortic arteries are sensitive to low $O_2$ or high acid levels in blood
    - reflexly increase respiratory activity
  - Associated with certain behaviors and emotions mediated through cerebral-hypothalamic pathway
  - Exercise modifies cardiac responses
  - Hypothalamus controls skin arterioles for temperature regulation
  - Vasoactive substances released from endothelial cells play role
Blood Pressure Abnormalities

• Hypertension
  – Blood pressure above 140/90 mm Hg
  – 2 broad classes
    ◦ Primary hypertension
    ◦ Secondary hypertension

• Hypotension
  – Blood pressure below 100/60 mm Hg if symptomatic

Hypertension

• Most common of blood pressure abnormalities
• Primary hypertension
  – Catchall category for blood pressure elevated by variety of unknown causes rather than by a single disease entity
  – Potential causes being investigated
    ◦ Defects in salt management by the kidneys
    ◦ Excessive salt intake
    ◦ Diets low in K+ and Ca^{2+}
    ◦ Plasma membrane abnormalities such as defective Na^+-K^+ pumps
    ◦ Variation in gene that encodes for angiotensinogen
    ◦ Endogenous digitalis-like substances
    ◦ Abnormalities in NO, endothelin, or other locally acting vasoactive chemicals
    ◦ Excess vasopressin
Hypertension

• Secondary hypertension
  – Accounts for about 10% of hypertension cases
  – Occurs secondary to another known primary problem
  – Examples of secondary hypertension
    ◊ Renal hypertension
    ◊ Endocrine hypertension
    ◊ Neurogenic hypertension

• Complication of hypertension
  – Congestive heart failure
  – Stroke
  – Heart attack
  – Spontaneous hemorrhage
  – Renal failure
  – Retinal damage
### Hypotension

- **Low blood pressure**
- **Occurs when**
  - There is too little blood to fill the vessels
  - Heart is too weak to drive the blood
- **Orthostatic (postural) hypotension**
  - Transient hypotensive condition resulting from insufficient compensatory responses to gravitational shifts in blood when person moves from horizontal to vertical position

### Hypotension

- **Circulatory shock**
  - Occurs when blood pressure falls so low that adequate blood flow to the tissues can no longer be maintained
  - 4 main types
    - Hypovolemic (“low volume”) shock
    - Cardiogenic (“heart produced”) shock
    - Vasogenic (“vessel produced”) shock
    - Neurogenic (“nerve produced”) shock