The systole/diastole aspect of the heart and the elastic nature of the arteries creates a pulsating pressure fluctuations.

Arterial Blood Flow and Blood Pressure

The systolic-diastolic aspect of the heart and the elastic nature of the arteries creates a pulsating pressure fluctuations.
**Arterial Blood Flow and Blood Pressure**

\[ \text{MAP} = DP + \frac{1}{3}(\text{Pulse Pressure}) \]

- Reduced elasticity  \( DP \)
- Reduced compliance  \( SP \)

Hypertension when BP > 140/90

**Pressure and the Circulation**

Pressure at capillary beds ~ = 40 mm Hg
Pressure at veins ~ = 20 mm Hg
Pressure at atrium ~ = 5 mm Hg

Flow = \( \frac{\Delta P}{R} \)

\( \Delta P = 15 \text{ mmHg} \)

That’s a low pressure gradient to drive blood back to the heart!
What promotes venous return to the heart?

- Low resistance in the veins; the low resistance conduit system allows low pressure differentials to provide adequate flow.

- Respiratory pump action of inspiration causes a decrease in pressure in the chest cavity while an increase in the abdominal cavity by the downward movement of the diaphragm. Helps to promote blood flow into the thoracic area.

Pressure and Venous Circulation

- Sympathetic stimulation of smooth muscles in veins results in vasoconstriction.

- This decreases venous compliance and hence increases venous pressure, driving more blood back to the heart.

*Figure 2.* Compliance curves for a vein showing how increasing smooth muscle contraction (arrow) decreases venous volume and increases venous pressure by decreasing compliance. Qualitatively similar changes occur in arteries.
Pressure and Venous Circulation

- Veins run between muscles;
- Contraction of muscles has a milking effect, squeezing blood upwards.
- Also, veins contain one-way valves that operate like elevator stages.

Regulation of tissue Perfusion is dependent on Blood pressure, cardiac output and resistance. Muscular Arteries & Arterioles are essential in Resistance control and are thus regulated.
If the Pressure gradient is the same, the flow is determined by the Resistance.

Thus Flow to organs is dependent on the resistance of the blood vessels (arterioles) guiding blood to that organ.
Homeostatic Regulation of BP

Short term control via regulation of resistance, CO and HR

Long term control via regulation of blood volume (venous return)

Control of Resistance

- By AutoRegulation Mechanisms
- By Neural Mechanisms
- By Endocrine Systems

All of these systems allow for CardioVascular Regulation such that tissues and organs receive proper blood perfusion without drastically changing the blood pressure driving force.
AutoRegulation

- Is a local response that results in an acceleration of blood flow through the tissue of origin
- There are three types of autoregulation.

1. Active Hyperemia
2. Re-Active Hyperemia
3. Flow auto-regulation

Resistance Regulation: AutoRegulation

1. Active Hyperemia

- Is the response to an increase in metabolism in the surrounding tissues
- If increased metabolism is not matched by an increased blood flow into those tissues and organs, the following local changes will result:
  - local decreased level in oxygen
  - an increased levels in CO₂, ADP, H⁺, K⁺ and bradykinin

- All these metabolic changes are known to cause vasodilation
Resistance Regulation: AutoRegulation

1. Active Hyperemia

- Blood Flow

- Metabolic Activity

2. Re-Active Hyperemia

- Is the response when an organ or area of an organ has been occluded for a period of time
- Release of the occlusion results in a transient but strong increase in blood flow to that region
3. Flow auto-regulation

- Is the response when an organ or area of an organ has a reduced blood flow due to for example a reduced blood pressure
- Reduced flow = reduced oxygen delivery
- Results in accumulation of metabolites such as CO\textsubscript{2}, ADP, H\textsuperscript{+}, K\textsuperscript{+} and bradykinin
- Thus results in vasodilation as well and increased blood flow

**Myogenic Response:**

When a blood vessel is stretched it causes vasoconstriction

Vasoconstriction = increase in R = reduced blood flow

---

### Resistance Regulation

#### (a) Active Hyperemia

1. ↑ Metabolic activity of organ
2. ↓ O\textsubscript{2} in organ
3. ↑ Metabolites in interstitial fluid
4. Arteriolar dilation in organ
5. ↑ Blood flow to organ

#### (b) Flow autoregulation

1. ↓ Arterial pressure in organ
2. ↓ Blood flow to organ
3. ↑ O\textsubscript{2} metabolites, vessel-wall stretch in organ
4. Arteriolar dilation in organ
5. Restoration of blood flow toward normal in organ

Active hyperemia and flow autoregulation differ in their cause, activity versus blocked flow, respectively, but both result in the production of the same local signals that provoke vasodilation (arteriolar dilation).
Neural Regulation

Occurs via the cardiovascular vasomotor centers of the medulla oblongata.

A cardio acceleratory center stimulates sympathetic activity.

A cardio inhibitory center acts on the vagus nerve, which in turn “tones down” the sympathetic outflow.

Resistance Regulation: Neural

1. Sympathetic Nerves

- Most arterioles have sympathetic nerve connections
- Peripheral arterioles have alpha adrenergic receptors that bind NorEpinephrine (NE) and cause vasoconstriction
- Relaxation is due to a decreased sympathetic stimulation via vagal inhibition of the sympathetic output
- There is little parasympathetic innervation of blood vessels
2. Other Nerves

- Some nerves may control the release of Nitric Oxide directly or indirectly (via ACh)
- NO is a vasodilator

These nerves are prominent in the regulation of gastrointestinal blood-vessels, certain areas of skeletal muscles and brain.

Note: a given set of arterioles will likely have either alpha- or beta-type receptors, not an equal number of each.

Alpha receptors are mostly found in skeletal muscle arterioles.

Beta receptors mostly in coronary arterioles.
Reserve Regulation: Neural

**Reflex Regulations**

Control occurs via reflex arcs involving

- baroreceptors
- afferent fibers
- vasomotor center of the medulla oblongata
- efferent fibers of the autonomic nervous system
- and the vascular smooth muscles of the arteries and arterioles

---

**Reflex Regulations**

Baroreceptors are pressure sensitive mechano-receptors. They are located in the artery walls of

- the internal carotids (called carotid sinus baroreceptors)
- aortic arch
- and most large, elastic arteries

- An increase in MAP stretches the walls, activates the receptors which then increases the frequency of action potential impulses in the afferent fibers.

- These impulses are directed towards the vasomotor center in the medulla. Decreased stretching results in a decreased number of impulses.
Resistance Regulation: Neural

**Reflex Regulations**

Increased incoming impulses from the baroreceptors (thus indicating higher BP) results in the following vasomotor action:

- decreased outgoing sympathetic activity
- this results in vasodilation
- Vasodilation of arterioles reduces peripheral resistance; vasodilation of veins results in less venous return and a drop in CO (BP = CO x R)

End result is thus a decrease in BP!

---

Resistance Regulation: Neural

**Reflex Regulations**

- Impulses from baroreceptors also reach the cardiac center where they stimulate the vagus nerve, reducing heart rate and contractile force.

- Function of such a Baroreceptor Reflex arc is to protect against acute changes in blood pressure such as changes in posture when one rises from a reclining to standing position.

  - Carotid sinus reflex protects blood supply to the brain
  - Aortic reflex maintains systemic BP
The events that happen during, for example, a sudden blood loss (resulting in a sudden drop in BP).
**Additional Reflex Regulations: Chemo receptors**

- Impulses from chemo receptors also reach the cardiac center
- Chemoreceptors are located in aortic arch and large arteries of the neck
- Most prominent ones are the **carotid and aortic bodies**
  - They sense changes in Oxygen, CO₂ and pH
  - Low O₂, low pH or high CO₂ results in impulses being directed to the vasomotor center

Results in reflex vasoconstriction, increasing BP and helps to speed blood flow to the heart and lungs!

**Higher Brain Centers in the CNS**

Thought process and emotional state of mind can influence Blood Pressure dramatically!
### Resistance Regulation: Neural

Keep in mind that neural process work quite fast. Thus, the systems discussed within neural regulation of resistance are at the basis of Blood Pressure Control on a short term (quick and fast term).

Examples of this quick acting is when going from a supine to an erect position (when waking up).
- Venous return is low when in a supine position
- This increases when we stand up (thus EDV increases)
- At same time, Symp. Branch is stimulated and HR increases, while SVR is increased.

All of this increases BP \( = \text{CO} \times \text{SVR} \)

---

### Resistance Regulation: Hormonal

The endocrine system provides both short term and long term control regulation of cardiovascular performance

**Short term control**

- Epinephrine:
  - released from Adrenal medulla and acts like NE
- Angiotension II:
  - produced after RENIN is released from kidneys due to a fall in BP
  - results vasoconstriction in most arterioles

The short term control is once again due to a manipulation of the resistance (blood vessel diameter).
### Resistance Regulation: Hormonal

#### Long term control
- Long term control indicates that the effects will become effective over a longer period of time.
- In this case, it is due to a manipulation of blood volume.
- This in turn influences venous return and thus cardiac output (and hence, BP).

#### Renin Release
- Is released from kidneys when there is a chronic BP drop
- Renin produces ANG II
- ANG II induces Adrenal gland to release more Aldosterone
- Aldosterone promotes more $\text{Na}^+$ re-uptake by kidney tubules, and thus more water to be retained
- More water retention, thus, higher blood volume

### Resistance Regulation: Hormonal

#### Angiotensin II
- Also promotes thirst
- Promotes ADH release from PPG

#### ADH
- Promotes water retention
- Also released when BP drops or when osmotic concentration drops

#### Erythropoietin
- Released by kidneys when tissue don’t get enough O2
- Increases blood volume viscosity and volume
Resistance Regulation: Hormonal

Natriuretic Peptides

• Released by the atria when BP is high
• Promotes Na\textsuperscript{+} secretion by kidney tubules and thus promotes water loss by kidneys
• Blocks release of ADH, aldosterone, epinephrine
• Stimulates vasodilation

All these factors would thus reduce blood blood volume and blood pressure.

This is thus the way the body deals with acute hypertension problems.

Resistance Regulation: Paracrine

Endothelial factors

• Nitric Oxide: released by endothelial cells and causes vasodilation
• Prostacyclin: vasodilaor
• Endothelin-1: vasoconstrictor

All the factors above are considered paracrine agents
Resistance Regulation: Endocrine

Resistance Regulation: Endocrine
Diversity among signals that influence contraction/relaxation in vascular circular smooth muscle implies a diversity of receptors and transduction mechanisms.

---

**Short Term Regulation during Hemorrhage**

- Carotid and aortic reflexes increase CO and peripheral vasoconstriction
- Sympathetic nervous system elevates blood pressure
- E and NE increase cardiac output and ADH enhances vasoconstriction
Long Term Regulation during Hemorrhage

- Decline in capillary blood pressure recalls fluids from interstitial spaces
- Aldosterone and ADH promote fluid retention
- Increased thirst promotes water absorption across the digestive tract
- Erythropoietin ultimately increases blood volume and improves $O_2$ delivery
### Regulation During Exercise

<table>
<thead>
<tr>
<th></th>
<th>Heart Weight</th>
<th>Stroke Volume (mL)</th>
<th>Heart rate (bpm)</th>
<th>Cardiac Output (L/min)</th>
<th>BP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non Athlete (rest)</td>
<td>300 g</td>
<td>60</td>
<td>83</td>
<td>5.0</td>
<td>120/80</td>
</tr>
<tr>
<td>Non Athlete (active)</td>
<td></td>
<td>104</td>
<td>192</td>
<td>19.9</td>
<td>187/75</td>
</tr>
<tr>
<td>Trained Athlete (rest)</td>
<td>500 g</td>
<td>100</td>
<td>53</td>
<td>5.3</td>
<td>120/80</td>
</tr>
<tr>
<td>Trained Athlete (active)</td>
<td></td>
<td>167</td>
<td>182</td>
<td>30.4</td>
<td>200/90</td>
</tr>
</tbody>
</table>

### Regulation During Exercise

**TABLE 21-2 DISTRIBUTION OF BLOOD DURING EXERCISE**

<table>
<thead>
<tr>
<th>Organ</th>
<th>Tissue Blood Flow (mL/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
</tr>
<tr>
<td>Skeletal muscles</td>
<td>1200</td>
</tr>
<tr>
<td>Heart</td>
<td>250</td>
</tr>
<tr>
<td>Brain</td>
<td>750</td>
</tr>
<tr>
<td>Skin</td>
<td>500</td>
</tr>
<tr>
<td>Kidney</td>
<td>1100</td>
</tr>
<tr>
<td>Abdominal viscera</td>
<td>1400</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>600</td>
</tr>
<tr>
<td>Total cardiac output</td>
<td>5800</td>
</tr>
</tbody>
</table>