Chapter 19: Blood Vessels

I. OVERVIEW

Arteries \rightarrow\text{ arterioles} \rightarrow\text{ capillaries} \rightarrow\text{venules} \rightarrow\text{veins.}

\begin{itemize}
  \item[(A)] \textbf{Walls}: most with three layers, surround lumen.
  \begin{itemize}
    \item -1- Tunica intima = endothelium
    \item -2- Tunica media = smooth muscle & elastin. \textit{Vasoconstriction & dilation.}
    \item -3- Tunica externa = collagen with nerves, lymph vessels, sometimes elastin & vasa vasorum.
  \end{itemize}
\end{itemize}

\begin{itemize}
  \item[(B)] \textbf{Arteries}: Types, in descending size order
  \begin{itemize}
    \item -1- Elastic = Conducting. Thickest walls, nearest heart, e.g. aorta. Most elastin.
    \item -2- Muscular = Distributing. Relatively more muscle.
    \item -3- Arterioles – smooth muscle predominates.
  \end{itemize}
\end{itemize}

\begin{itemize}
  \item[(C)] \textbf{Capillaries}: exchange vessels. Microscopic. Wall only endothelium.
  \begin{itemize}
    \item Most are \textit{continuous.}
    \item Fenestrated capillaries have pores.
    \item Sinusoids have large, irregular shapes, leaky walls. e.g. in liver.
  \end{itemize}
\end{itemize}

Capillary beds have a thoroughfare channel, \textit{metarteriole}, through center.
Arterial, incoming end; Venous, outgoing, end.

\textbf{Precapillary sphincters} at branches from metarteriole regulate blood flow.

\begin{itemize}
  \item[(D)] \textbf{Veins}
  \begin{itemize}
    \item Small ones are \textit{Venules} – the tiniest, post-capillary, are porous.
    \item Others have all three tunics but with thin, distensible walls.
    \item Act as blood reservoirs; contain \sim 65\% of body’s blood.
  \end{itemize}
\end{itemize}

Some large veins with valves, prevent backflow.
May become varicose, may be due to valve damage.
(E) **Vascular Anastomoses** = interconnections
May be arterial; arteriovenous (e.g. metarterioles); venous.

**Arteriosclerosis** – most is **atherosclerosis** (fatty)
Contributing factors: damaged endothelium, high lipid levels, increased smooth muscle, Ca++ deposits, platelet activity.

Risk increases with being male, advancing age, genetics, decreased physical activity, obesity, diabetes, hypertension.

Leads to MIs and strokes.

**II. PHYSIOLOGY**

(A) **Introduction:** Blood flow related to **cardiac output**.
Opposed by peripheral resistance,
varies with blood viscosity, vessel length and vessel diameter (regulated).

\[
\text{Flow} = \frac{\text{Difference in Pressure}}{\text{Resistance}}
\]

(B) **Systemic Blood Pressure**

Pulsatile near heart. Creates distinction between **systolic** (highest P) and **diastolic** (lowest P).

Systolic P – Diastolic P = **Pulse Pressure**.

**Mean Arterial Pressure** (MAP) = Diastolic P + \(\frac{\text{Pulse P}}{3}\)

Compensation for more time spent in diastole.

BP drops as blood flows through capillaries and veins.

Venous return aided by respiratory pump, muscular pump.
Some vasoconstriction with sympathetic control, cardiac suction.

(C) **Maintaining Blood Pressure**

-1- **Short term mechanisms** include

   a. **neural controls**.

Vasomotor center of medulla maintains tone and makes necessary adjustments.
Baroreceptors in carotid sinuses and aortic arch communicate to medulla.

Chemoreceptors in aortic arch, carotid bodies, and elsewhere sensitive to CO2 & pH, communicate to cardioacceleratory & vasomotor centers.

Higher brain centers – Hypothalamus and cerebral cortex can effect BP.

b. hormones.

Angiotensin II triggered by renin release. Causes vasoconstriction, ADH & aldosterone release.

ANP decreases BP by antagonizing aldosterone.

ADH, also called vasopressin, increases BP.


Circulatory efficiency monitored with vital signs, pulse & BP.

Alterations in BP include hypotension, orthostatic and chronic;

Hypertension, 90% essential. Probably associated with atherosclerosis.

(D) Tissue Perfusion: for nourishment and waste removal.

Velocity of blood flow decreases with increasing cross-sectional area. Slowest in capillaries.

-1- Autoregulation is local. Based on requirements.

Metabolic controls e.g. H+ accumulation, NO (vasodilator).

Myogenic controls – smooth muscle contracts when stretched.

These result in reactive hyperemia.

-2- Long term reaction is angiogenesis: increased number and size of vessels.
-3- Blood flow to special areas:

   a. **Skeletal muscle** – highly variable
   
   b. **Brain** – constant
   
   c. **Skin** – also variable, involved in TB regulation
   
   d. **Lungs** – decreased resistance, arteries vein-like, low P
      Decreased O2 → vasoconstriction
   
   e. **Heart** – flow in coronary circuit influenced by HB, fairly constant.

-4- **Capillary Flow = vasomotion.**

Slow and intermittent, due to action of sphincters.

   Exchange of gases & nutrients- move down gradients.

   Mechanisms – intercellular, across membranes, through fenestrations, endocytosis.

Fluid Movements = bulk flow.

   **Filtration** at arterial end, **reabsorption** (less) at venous end.

   Forces: **hydrostatic P** - out; **COP** (Colloid Osmotic P) – in

   Lost fluid returned by lymphatic system.

(E) **Circulatory Shock** – blood not circulating sufficiently.

   Usually **hypovolemic.**

   **Vascular shock** due to extreme vasodilation. e.g. anaphylactic, septic
   
   **Cardiogenic shock** + pump failure.

CIRCULATORY PATHWAYS

Figures 19.4, p. 725  Arteries
19.9, p. 737  Veins