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Coronary Blood Flow

M1 – Cardiovascular/Respiratory Sequence
Louis D’Alecy, Ph.D.

Fall 2008
Wednesday 11/05/08, 11:00

Coronary Blood Flow
26 slides, 50 minutes

1. Vascular anatomy
2. Determinants of coronary flow
3. Flow mediated dilation
4. Ischemia
5. Coronary Flow Reserve
Surface Anatomy

Anterior view

Posterior view
Heart statistics

\[ 300\text{g}/70,000\text{g} = 0.0043 \text{ or } < 0.5\% \text{ Body Weight} \]

Coronary flow = 4\% of cardiac output = 80 mL/min/100g

“Resting “ flow \( \sim 30\times \) flow/100g tissue of skeletal muscle

Highest oxygen extraction in body
(arterial oxygen 20 vol \% to coronary sinus 8 vol \%)
(typical mixed venous oxygen higher at 17 vol \%)

Must increase flow to increase oxygen delivery

\[ \text{Vol } \% = \frac{\text{mLO}_2}{100 \text{ mL blood}} \]
Cross section of coronary artery.
Figure 6.1. Major determinants of myocardial oxygen supply and demand. 

Arterial $O_2$ content, $F_{O_2}$ & Hct (or Hb)

Coronary blood flow
- coronary perfusion pressure
- coronary vascular resistance
- external compression
- intrinsic regulation
- local metabolites
- endothelial factors
- neural innervation

Myocardial oxygen demand
- Wall stress $(P \cdot r/2h)$
- Heart rate
- Contractility

$h$, ventricular wall thickness; $P$, ventricular pressure; $r$, ventricular radius.
DETERMINANTS OF CORONARY BLOOD FLOW

1 DIASTOLIC PERFUSION PRESSURE $\Delta P$

2 SYSTOLIC COMPRESSION ("Resistance")

3 METABOLIC CONTROL (Resistance)

4 NEURAL CONTROL (Resistance)
Origin of left coronary artery

Origin of right coronary artery
Systolic compression

Left coronary flow mL/min

Right coronary flow mL/min

**Pressure X Rate Product**

If \( r \uparrow \), more tension is need for the same pressure. Thus an enlarged heart has greater demand.

Law of Laplace

\[ T = P \times r \]
Tension = Press \( X \) radius

"Cost"

\[ 2T = P \times 2r \]

If radius \( (r) \uparrow \), more tension is need for the same pressure. Thus an enlarged heart has greater demand (cost).
Tissue Blood Flow and Tissue Vascular Resistance

(**Assume Perfusion Pressure is Constant**)

- **Vasoconstriction**
  $$\Rightarrow \downarrow r \Rightarrow \uparrow R_{\text{tissue}} \Rightarrow \downarrow F_{\text{tissue}}$$

- **Vasodilation**
  $$\Rightarrow \uparrow r \Rightarrow \downarrow R_{\text{tissue}} \Rightarrow \uparrow F_{\text{tissue}}$$

$$F_{\text{tissue}} = \frac{\text{Perfusion Pressure}}{R_{\text{tissue}}}$$
Figure 6.1. Major determinants of myocardial oxygen supply and demand.

$h$, ventricular wall thickness; $P$, ventricular pressure; $r$, ventricular radius.
Isolated Vascular Effects

1. Sympathetic alpha adrenergic vasoconstriction $\alpha_1$

2. Sympathetic beta adrenergic vasodilation
   $\beta_1$ (evidence for innervated VSM)
   $\beta_2$ non-innervated VSM

3. Parasympathetic cholinergic vasodilation
BUT HOW DOES IT WORK IN VIVO ????

Parasympathetic Activation

Stimulate parasympathetic to heart >> Ach >>SA node
   >> ↓↓ HR >>↓↓ metabolism >> ↓↓ Coronary Blood flow

BUT

PACE heart (i.e. fixed heart rate) >> no change in HR >>
no change metabolism --------- Therefore
Stimulate parasympathetic to paced heart >> >>
Ach vasodilation >> ↑↑ coronary blood flow !!
BUT HOW DOES IT WORK IN VIVO ????

Sympathetic Activation

Stimulate sympathetic nerves to heart >> ↑↑ Norepi >>
>> ↑↑ inotropism >> ↑↑ metabolism ++ >> ↑↑ HR >>
↑↑ Coronary Blood flow

BUT

Block $\beta_{1&2}$ receptors and Stimulate sympathetics to heart
>> ↑↑ Norepi (stress) >> no change in HR >> >> no change
metabolism >> >> no change in inotropism >> potential for
>> ↓↓ Coronary Blood flow
by “unmasked” $\alpha_1$ adrenergic vasoconstriction

Can Metabolic control still dominate??
Flow Mediated Dilation
(FMD)
&
Coronary Stenosis
FMD = Flow Mediated Dilation

Figure 6.1. Major determinants of myocardial oxygen supply and demand.

- Wall stress \( (P \cdot r/2h) \)
- Heart rate
- Contractility

- \( O_2 \) content
- Coronary blood flow
  - coronary perfusion pressure
  - coronary vascular resistance
  - external compression
  - intrinsic regulation
  - local metabolites
  - endothelial factors
  - neural innervation

\( h \), ventricular wall thickness; \( P \), ventricular pressure; \( r \), ventricular radius.
ADMA (NOS Inhibitor)

Sheer or flow mediated Vasodilation FMD

FMD Setup of Arm, ultrasound, & cuff on fore arm

Measure here

Compress here
FMD = flow mediated dilation
NMD = nitroglycerine (Max) mediated dilation
NO mediated vasodilation

Compromised by endothelial dysfunction (?ADMA?)
lated that in normal individuals, the relaxation effect of EDRF-NO outweighs the direct $\alpha$-adrenergic constrictor effect of catecholamines on arterial smooth muscle, such that vasodilatation results. However, in patients with dysfunctional endothelium (e.g., atherosclerosis), an impaired release of endothelial vasodilators leaves the direct catecholamine effect unopposed, such that relative vasoconstriction occurs instead. The resultant decrease in coronary blood flow and myocardial oxygen supply contributes to ischemia. Of note, in patients with risk factors
Ischemia

-blood flow to a tissue or organ that is inadequate to maintain function.

- i.e. myocardial ischemia (MI)
With the same perfusion pressure, the same measured flow means the overall (series) resistance is the same regardless of a focal lesion! BUT *** You have used up vasodilator reserve !!!!!!!
Series Resistance Network

Compensatory Vasodilation here so series resistance stays the same.

Lesion here

\[ R_s = R_1 + R_2 + R_3 \]

\[ \Delta P = P_i - P_0 \]

\[ \dot{Q} = \Delta P / R_s \]
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