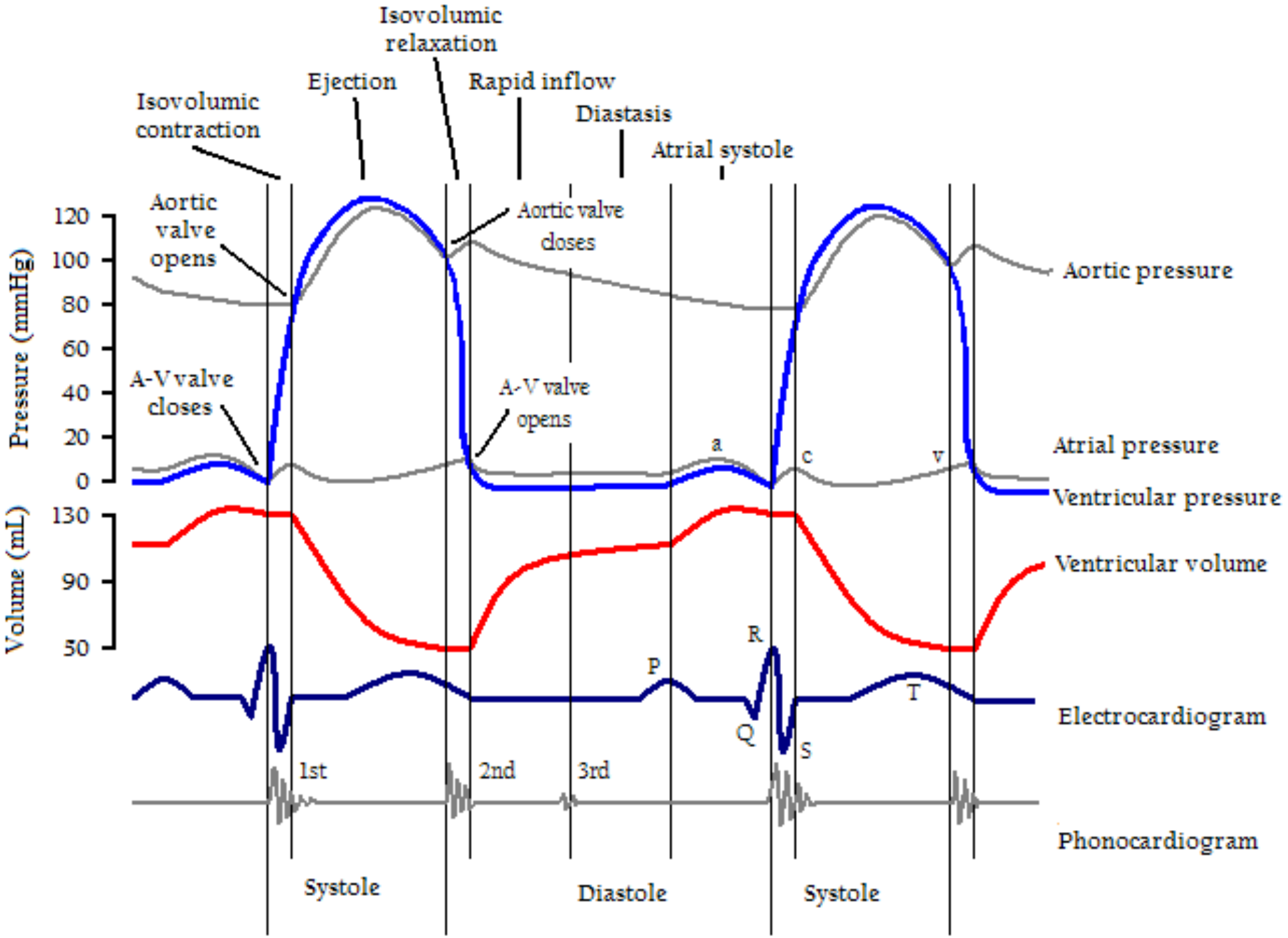
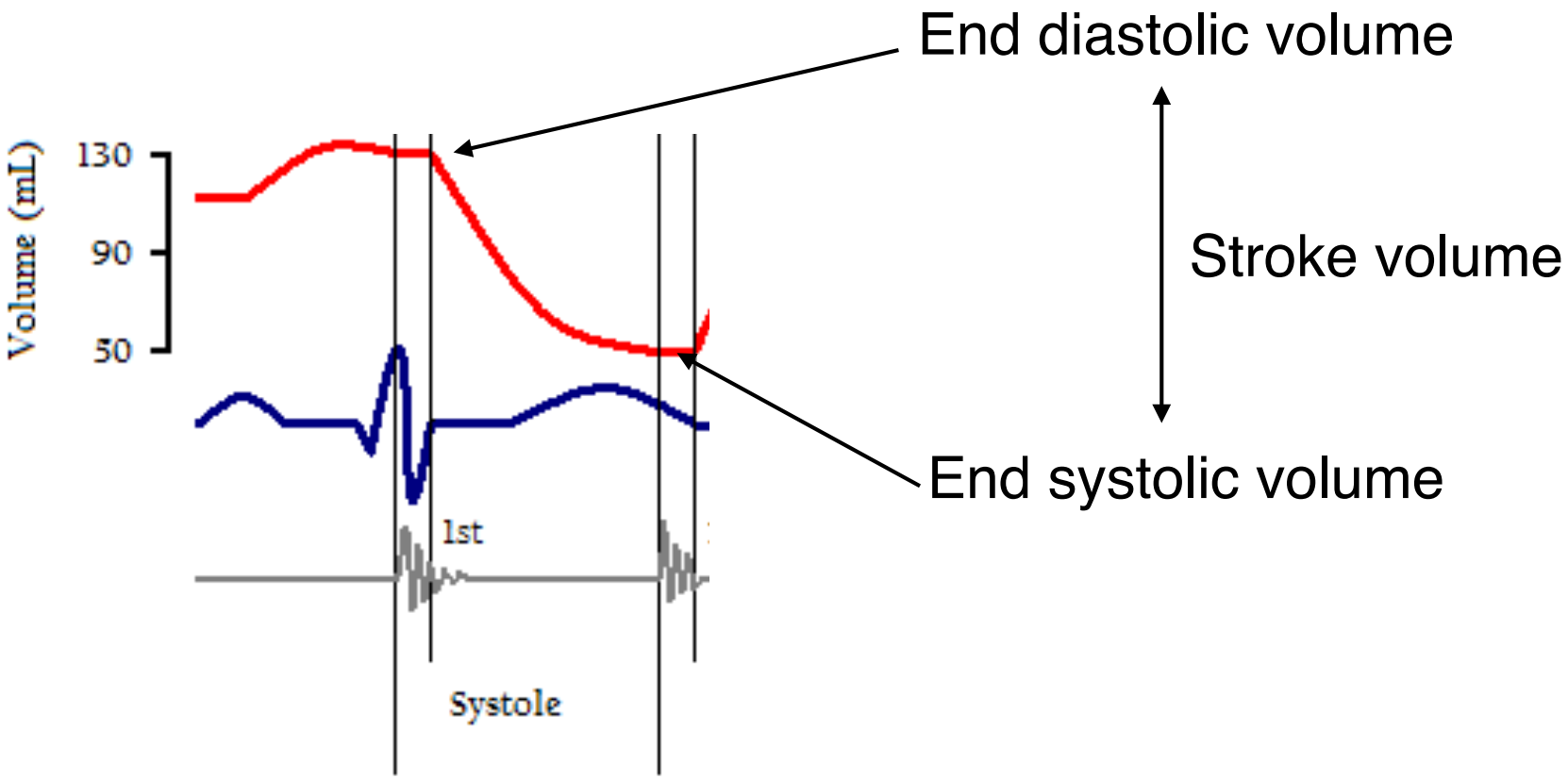
Module outline

- Anatomy
- Myocytes
- **Cardiac cycle**
- Haemodynamics & blood pressure
- Coronary circulation
- Capillaries

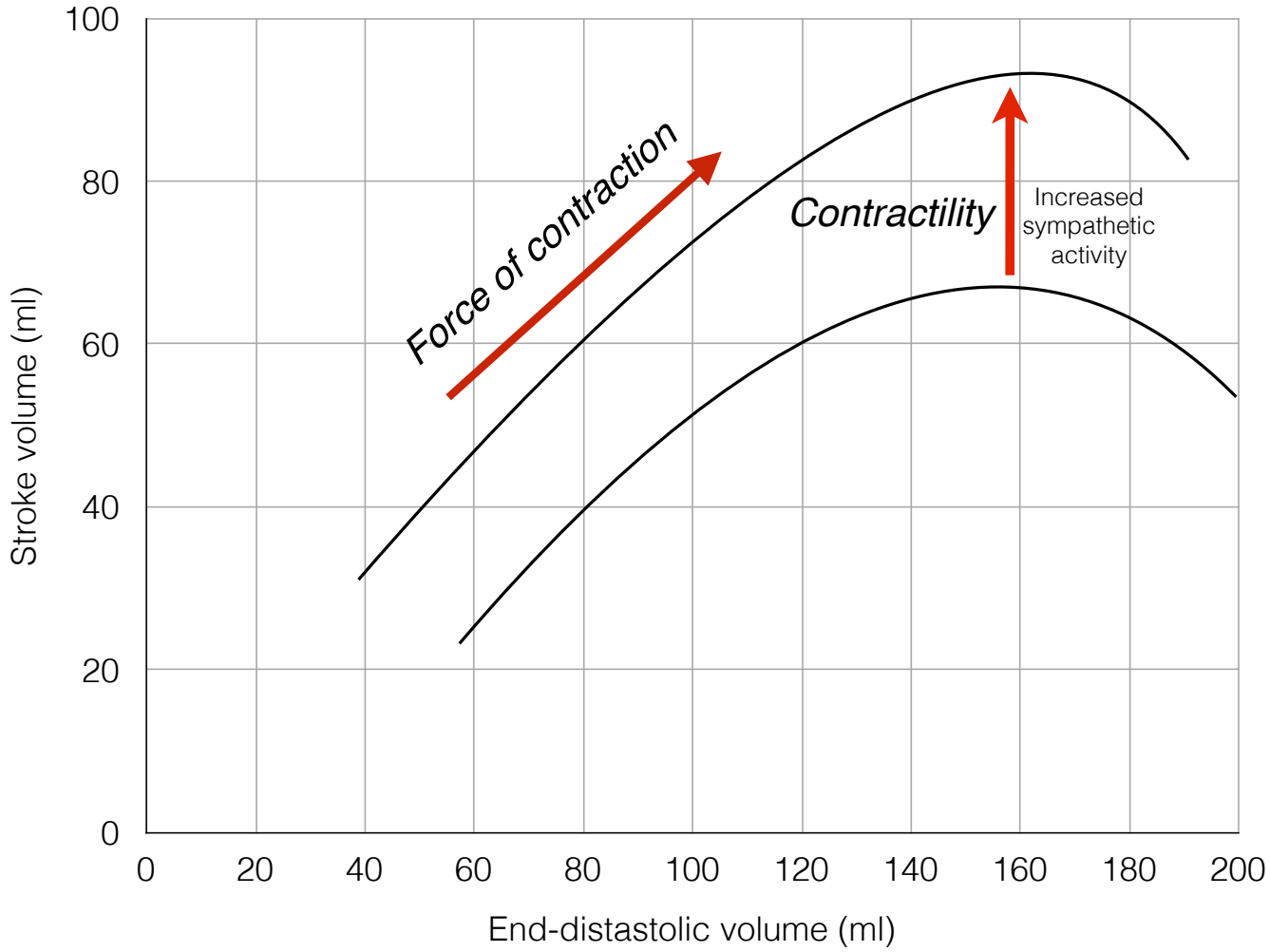

Isovolumic relaxation
Ejection
Rapid inflow
Isovolumic contraction
Diastasis
Atrial systole
Aortic valve opens
Aortic valve closes
A-V valve closes
A-V valve opens
Pressure (mmHg)
120
100
80
60
40
20
0
Volume (mL)
130
90
50
a
c
v
R
P
T
Q
S
1st
2nd
3rd
Aortic pressure
Atrial pressure
Ventricular pressure
Ventricular volume
Electrocardiogram
Phonocardiogram
Systole
Diastole
Systole

Cardiac cycle



Frank-Starling law of the heart

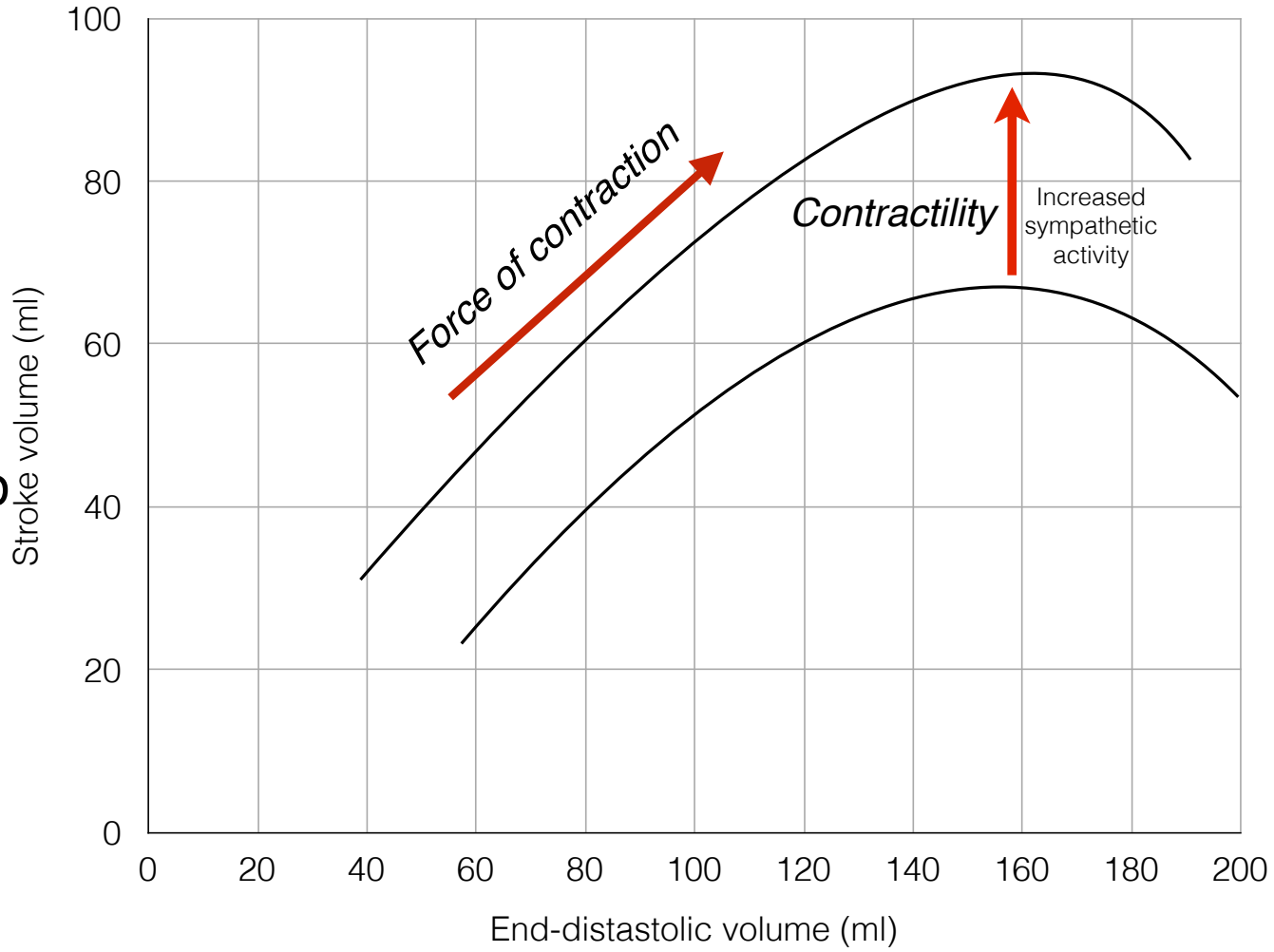
- Preload/EDV determines SV
- Length tension relationship = EDV
- Filament overlap
- Symp ↑ SV for any given EDV



Frank-Starling law of the heart

Factors affecting EDV:

- Venous tone
- Blood volume
- Skeletal muscle pump
- Respiratory pump
- Posture



Heart failure

- = Cannot meet metabolic demand (at normal CVP)

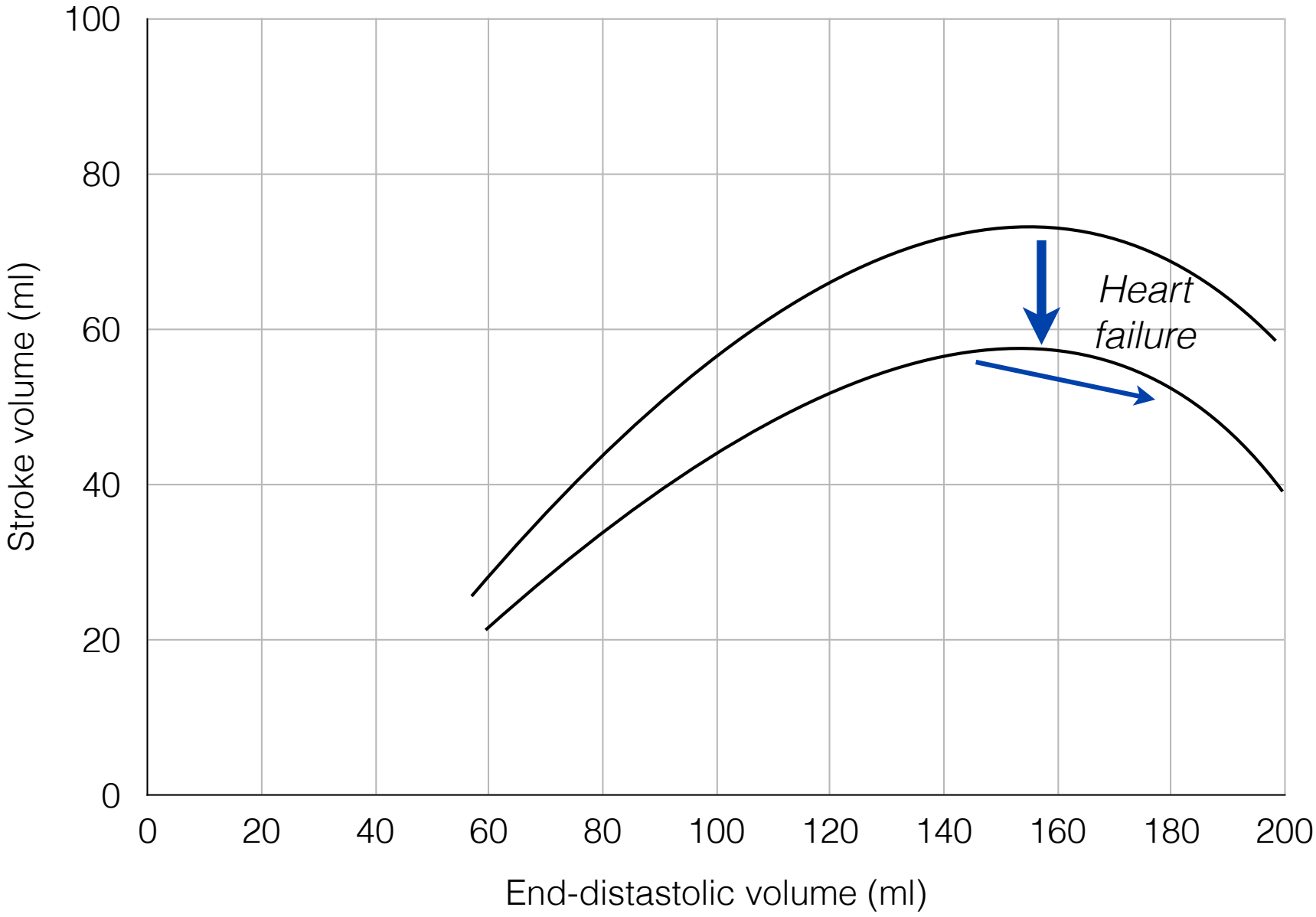
- Causes of heart failure:

  - Ischaemic heart disease

  - Hypertension

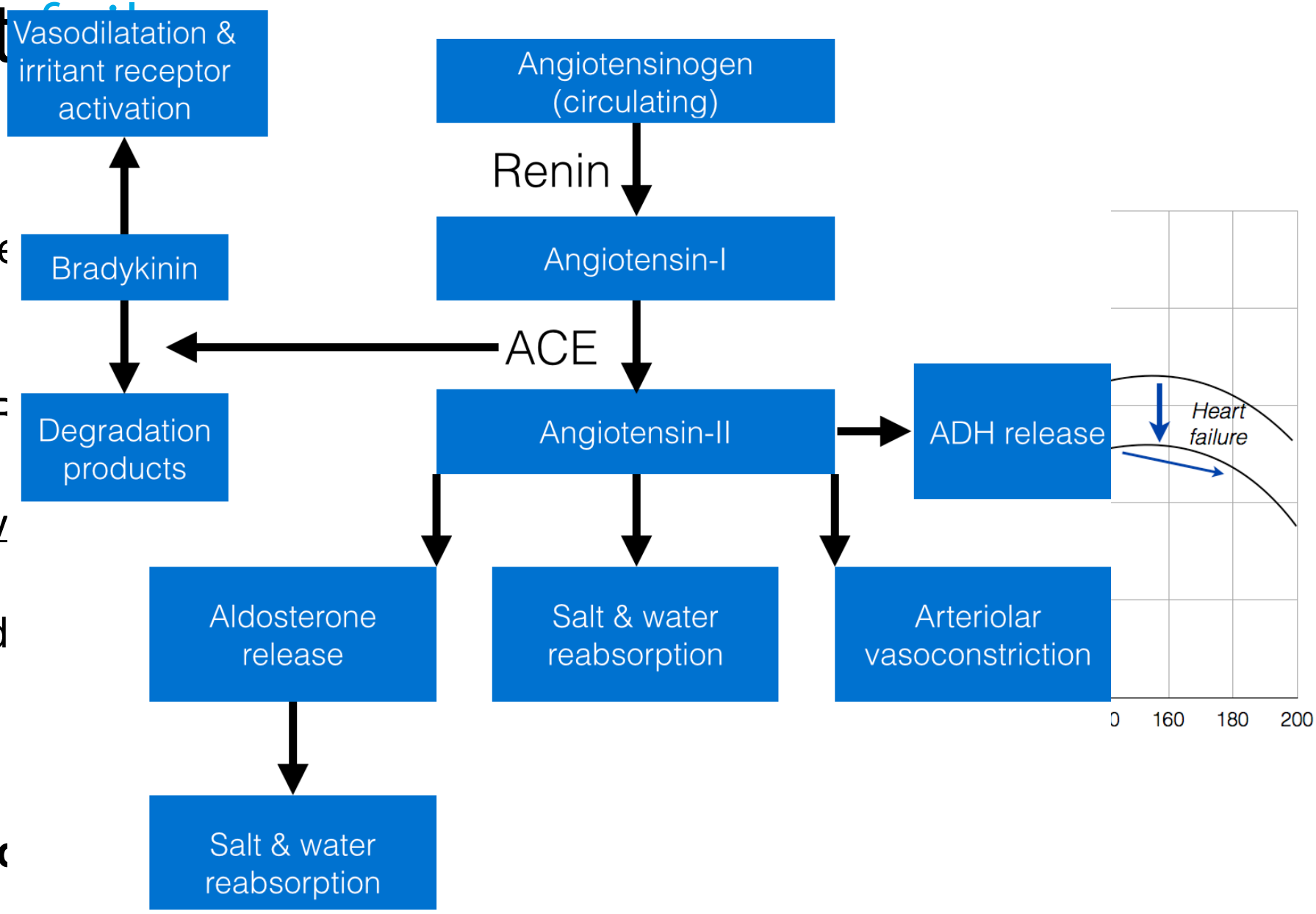
  - Valvular disease

Heart failure



Heart

- Fluid re
  CVP
  - → F
- Initially
- Late: d
- **Remod**



Heart failure

RAAS: (↓renal perfusion)

- Fluid & Na^+ retention
- Ang-II → Constriction

Sympathetic activation

- A → Renin (β1)
  - ↑ Contractility
  - ↑ HR (inc. ↓ Vagus)
- NA → Vasoconstriction

∴ ↑ AFTERLOAD* → ↑ ENERGY & ↓

*Afterload = total peripheral resistance

Heart failure

Right-sided heart failure	Left-sided cardiac failure
Raised jugular venous pressure	Pulmonary oedema
Hepatomegaly	Peripheral hypoperfusion
Peripheral oedema	Orthopnoea
Ascites	Paroxysmal nocturnal dyspnoea
Fatigue	Cough & wheeze

Key points

- **Pacemaker cells** have a depolarising pre-potential due to high sodium permeability

- **End-diastolic volume** (pre-load) determines force of contraction by controlling myocyte length

- **Sympathetics** increase SAN rate, reduce AVN delay and increase ventricular contractility

- Heart failure causes right- & down-wards shift on **Frank-Starling curve**