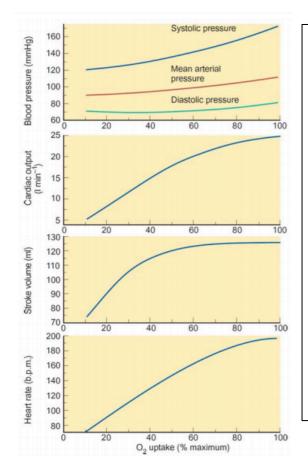
# **Cardiovascular Responses to Exercise**

#### 1) Meeting the Increased Energy Demand of Exercise - Cardiac Output, Heart Rate, Stroke Volume

Changes that occur during exercise that allow the cardiovascular system to increase blood flow to muscles:

- Arterial pressure INCREASES.
- Cardiac output (volume of blood, in L, pumped by the heart per minute) INCREASES.
- Cardiac output (CO) = heart rate (HR) x stroke volume (SV).
  - o Heart rate (number of heart beats per minute) INCREASES.
  - o Stroke volume (amount of blood pumped by the left ventricle per minute) INCREASES.



- Heart rate increases linearly.
- Stroke volume increases exponentially.
- However, eventually, at approximately 60% of VO2 max, stroke volume plateaus and therefore any further increases in cardiac output are due to increases in the heart rate.
- The increase in metabolic demand during exercise is thus met by an increase in cardiac output.
- Cardiac output (CO) is equal to the volume of blood pumped by the heart per minute, in L/min.
- In untrained and trained individuals, resting CO is the same.
- In untrained individuals at maximal exercise, CO can reach 20L/min.
- In trained individuals at maximal exercise, CO can reach 40L/min.
- 4-fold and 8-fold increase in cardiac output following exercise.
- The capacity to exercise is LIMITED by CARDIAC OUTPUT.

# **Autonomic Control of Heart Rate**

- Heart rate is under tonic parasympathetic (vagal) control at rest.
- Heart rate increases with exercise or stress.
- Parasympathetic vagal tone is reduced and sympathetic tone is increased.
- The parasympathetic nervous system neurotransmitter is acetylcholine, binds cholinergic receptors.
- The sympathetic nervous system neurotransmitter is noradrenaline, binds adrenergic receptors.



#### **Heart Rate and Training**

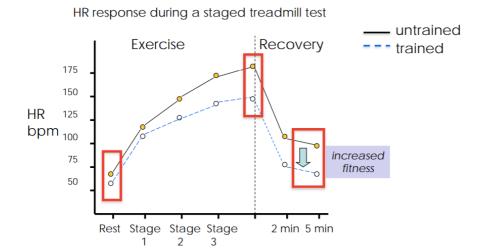
- With training, there is improved efficiency of cardiovascular system to pump blood (increased stroke volume).
- Heart needs less beats to provide oxygen with chronic exercise training.
- The **resting heart rate is reduced in individuals after training**, less beats needed per minute.
- Thus, there is a greater filling time (end-diastolic volume is increased) → more blood pumped with each beat.
- End-diastolic volume: the volume of blood in the heart at the end of diastole (relaxation) and just before systole.
- Note that resting CO is the same in trained and untrained individuals, but heart rate is lower in trained.

## **Resting Heart Rate**

untrained person 60-80 bpmtrained person 35-45 bpmelite athlete 25-40 bpm

Heart rate can be used to monitor improvements in fitness during CV training

- Note that *maximal heart rate does not change* with chronic exercise training.
- Maximal heart rate depends on an individual's AGE. Maximal heart rate = 200 bpm age.
- Sub-maximal heart rate changes with training however trained individuals have a lower heart rate for the same workload compared to untrained individuals.
- Trained individuals have increased vagal input + decreased sympathetic input -> sub-maximal heart rate lower.
- Heart rate increases with exercise nonetheless both in untrained and trained individuals in order to increase the
  oxygen supplied to working skeletal muscles and tissues.



Sub-maximal heart rate decreases
with chronic exercise training.
Heart rate increases nonetheless
with an acute exercise bout.
Maximal heart rate is relatively
unchanged with heart rate.

#### **Stroke Volume** and Training

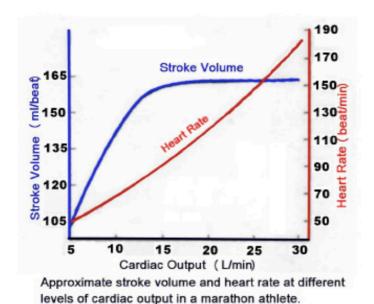
- Stroke volume: the volume of blood ejected from the left ventricle per beat.
- **Systole**: contraction or pumping of the heart.
- **Diastole**: relaxation and refilling of the heart with blood.
- End-systolic volume: the volume of blood in the heart following systole.
- End-diastolic volume: the volume of blood in the heart following diastole.
- Stroke volume therefore is equal to end-diastolic volume end-systolic volume.
- Thus, when end-systolic volume is higher (greater volume of blood in the heart after the heart contracts), the stroke volume will be smaller (as ESV is a larger value).

## SV = EDV - ESV

At rest, untrained individuals have a lower SV than trained individuals, but higher HR.

Therefore, CO = same.

- Stroke volume increases proportionately with increasing exercise <u>until around 60% of maximal exercise</u> (VO2 max), where it then plateaus.
- Increases in CO are therefore due to increases in heart rate after 60% VO2 max.



Stroke volume is influenced by:

- 1) Afterload
- 2) Preload
- 3) Venous return
  - a. Skeletal muscle pump
  - b. Respiratory pump\*
  - c. Venous dilation
- 4) Contractility
  - a. Positive inotropic agents
  - b. Negative inotropic agents

<u>STROKE VOLUME</u> is regulated by FOUR main factors, including: afterload, preload, contractility and venous return.

#### **Afterload Influences Stroke Volume**

MAP = CO x TPR

- Afterload: the amount of resistance/load the heart must pump against to eject blood.
- If the load is higher due to increased mean arterial blood pressure (MAP), then the end-systolic blood volume will be increased, as more blood will remain in the heart after systole. As stroke volume = EDV ESV, then the stroke volume will be reduced. But this reduction is only slight; due to the increased MAP → greater venous ret.
- Therefore, stroke volume is inversely proportionate to afterload (when afterload is high, stroke volume is lower).
- Thus, increased afterload is concomitant with reduced stroke volume and therefore, reduced cardiac output.
- However, this is dampened in exercise due to vasodilation of the vessels.
- MAP decreases with chronic training (still higher than rest), therefore afterload is decreased → slight fall in SV.
- At maximal exercise there is no change in mean arterial pressure.
- There is increased vascular conductance and reduced vascular resistance of the vessels during exercise.