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**Lecturer:** Simon Green

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## Lecture 2

1. Efferent / motor pathway: **VENTRAL HORN**  
Brain via spinal cord and peripheral nerves → muscles  
Feed-forward control
2. Afferent / sensory pathway: **DORSAL HORN**  
Muscles → brain via peripheral nerves and spinal cord  
Feedback control

### TWO TYPES OF SENSORY NEURONS:

1. MUSCLE SPINDLE → SENSE LENGTH → PROPRIOCEPTOR
2. GOLGI TENDON → SENSE FORCE/TENSION → PROPRIOCEPTOR

STRETCH REFLEX INVOLVES MUSCLE SPINDLES.

## Lecture 3

1. Concentric = shortening (towards the centre)  
Eccentric = lengthening  
Isometric = no change in length
2. **Motor unit = a single  $\alpha$ -motor neuron and the muscle fibres it innervates.**
3. A motor neuron has multiple branches ~~that attach to many muscle fibres~~
4. A muscle fibre is only innervated by one branch of a motor neuron.
5. The cell body (soma) of the  **$\alpha$ -motor neuron** is located in the spinal cord.
6. Smaller muscles tend to have more motor units, but less muscle fibres. *Small motor units*  
Smaller muscles require more **precision and control**, <sup>with</sup> thus more motor units.
7. Muscle fibres of a motor unit are widely dispersed → fibres of a single motor unit do not lie directly next to each other.
8. Force is developed by muscle fibres.  
 **$\alpha$ -motor neurons** do not generate the force.  
Motor neurons control the muscle fibres that generate the force.
9. The level of force output from a single muscle can be varied in two ways: by varying  
1) the number of **motor units 'recruited/activated'** and 2) the **firing rate of a motor neurons** (frequency of depolarisation, but not speed).

## Lecture 6

### 1. Anatomy of skeletal muscle:

**EPI** → Muscle  
**PERI** → Muscle fascicle  
**ENDO** → Muscle fibre  
Myofibril

Sarcomere → contractile unit of muscle

Myofilaments → myosin (thick) and actin (thin)

### 2. Muscle fibres contract towards the centre

When muscle fibres shorten they apply tension to tendons

### 3. Muscle is able to apply higher force when it lengthens than when it shortens (concentric) or acts isometrically (same length).

### 4. The increase of calcium is an important trigger for attachment of myosin to actin (muscle contraction).

### 5. Relaxation of skeletal muscle requires energy / ATP → active process.

### 6. Muscle strength is the highest force developed during a maximum voluntary effort and it actually occurs during **eccentric** actions.

### 7. A muscle's **strength** is a function of the number of sarcomeres in **parallel**.

### 8. Series → speed of shortening

Parallel → force of shortening

### 9. Increase the **width** to increase the strength → hypertrophy

Increasing the number of muscle fibres → **hyperplasia** does not lead to strength increases.

### 10. Muscle length influences maximum force:

→ more cross-bridge attachment sites for myosin and actin.

→ greater distance travelled means greater ability to generate force.

### 11. Major site of ATP use = myosin.

### 12. Maximum amount of force generated by muscle depends on how quickly it shortens: the quicker it shortens, the less force it generates.

### 13. Pennate muscles have more parallel fibres → generates more force.

A LONGER MUSCLE HAS THE ABILITY TO GENERATE MORE FORCE.

HOWEVER, THE "QUICKER" (HIGHER VELOCITY) A MUSCLE SHORTENS, THE LESS FORCE IT CAN GENERATE.

22. Blood flow and pressure increase due to exercise.

23. Resistance to blood flow reduces as exercise intensity increases.

24. Training = enlarged heart, enlarged chamber volume (capacity to hold blood), heart wall thickness doesn't change.

### Aerobic training and the heart:

25. Maximum cardiac output increases

26. Resting cardiac output does **not** change

27. Maximum heart rate does **not** change via training

28. Resting heart rate is reduced by training

29. Maximum stroke volume rises

30. Resting stroke volume rises

31. At rest a fall in HR is countered by a rise in stroke volume, thus cardiac output is unchanged

### Stroke volume is MECHANICALLY influenced by:

#### Preload:

The amount of blood in the ventricles after diastole (period of relaxation).

The greater the rate of filling, the greater the rate of blood the chambers can hold → increases pressure, which **increases** contraction strength and therefore **stroke volume**.

#### Afterload:

The force opposing the flow of blood → resistance.

An increase in afterload **lowers stroke volume**.

#### Contractility: (neural control)

Relates to the activity of the cardiac sympathetic nerves → releases more noradrenaline.

Increase **contractility** equals **more power** of the hearts contraction, which **increases stroke volume**.

## Lectures 15 + 16

1. Normal alveolar ventilation = 4-5L/min
2. Normal minute ventilation = 8-10L/min
3. Alveolar ventilation = total amount of air that reaches the LUNGS. per minute
4. Minute ventilation = total amount of air in and out per minute.
5.  $AV = (TV - \text{dead space}) \times f_b$ .

★ 6. Minute ventilation (MV) = tidal volume (TV) x frequency of breathing per minute ( $f_b$ ).

7. Breathing frequency at rest ~~10-15~~ → 12-15 breaths/minute

8. Alveolar ventilation is always less than minute ventilation → not all the air that is inhaled reaches the lungs → dead space.

9. Normal  $PCO_2$  in the blood = 40mmHg

10. Normal  $PO_2$  in the blood ~~100~~ → 100 mmHg

11. Dead space = the amount of inhaled air that doesn't reach the respiration zone (i.e. never leaves the conduction zone) = 150ml per breath.

12. Respiratory zone = where gas exchange occurs → contains alveoli

13. Gas exchange occurs between the alveolar air and the blood in the pulmonary capillaries → respiratory membrane via diffusion.

Total area and thickness of the interface affects the rate of gas diffusion.

Larger surface area = more gas flow.

Influenced by the size of the surface area of the alveoli and the thickness of the respiratory membrane.

~~Carbon dioxide diffuses quicker through the respiratory membrane than oxygen~~  
~~As CO<sub>2</sub> is more soluble in water.~~

15. Untrained individuals reach ventilation limit earlier than trained individuals.

16. Diaphragm innervated by the phrenic nerves.

17. Diaphragm contracts flat (increases volume of lungs)

18. Major breathing muscle at rest = diaphragm.

19. Tidal volume increase with graded exercise intensity, but plateaus towards max.

20. Breathing frequency rises with graded exercise and doesn't plateau.

21. During normal breathing at rest, lung volumes vary within a small operating range between the end-inspiratory lung volume and end-expiratory lung volume.

INSPIRATION REQUIRES THE DIAPHRAGM TO CONTRACT.