

Cardiac excitation contraction coupling

MEDICAL PHYSIOLOGY DEPARTMENT

ILOs:

1. Define cardiac contractility.
2. Describe the excitation-contraction coupling.
3. Identify the regulation of cardiac contractility (frank-starling law, staircase phenomenon, homeometric regulation).
4. Factors affecting cardiac contractility.

Cardiac Contractility

Definition:

It is the ability of the cardiac muscle to contract to pump blood.

Contraction starts after excitation of cardiac wave

Heart

In systole:

Cardiac contraction to eject blood

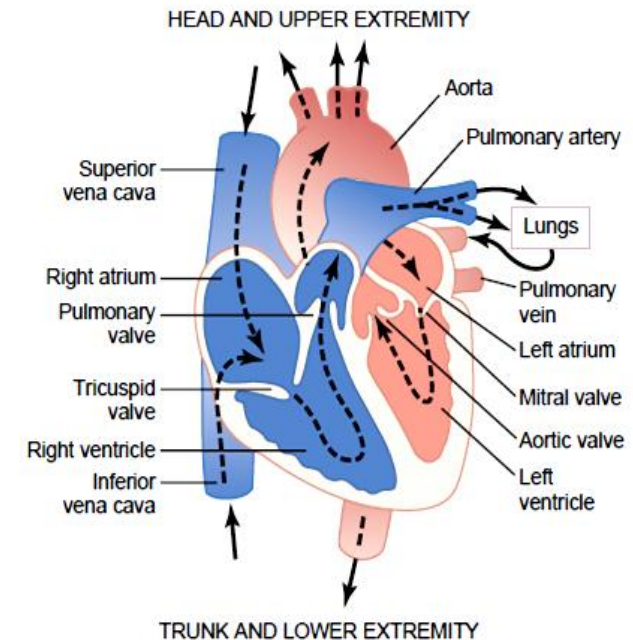
In diastole:

Cardiac relaxation to be filled with blood

The heart is formed of: 2 Separate pumps:

1]Right side pump (volume pump)

2]Left side pump (pressure pump)



Important notes about the structure of the cardiac muscle cell:

Sarcomere

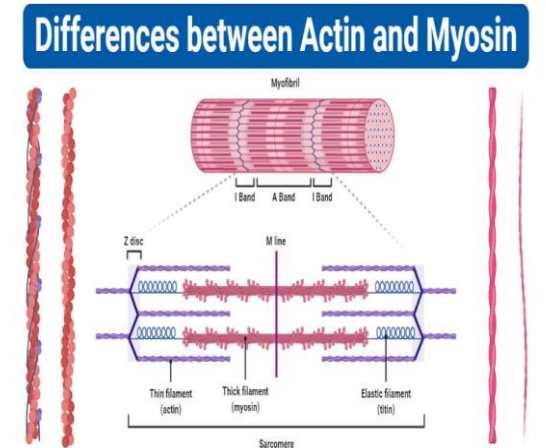
- is the contractile unit and has the same structure of actin and myosin filament as in skeletal muscle.

Shortening

- occurs according to the sliding filament theory, which states that thin filaments slide along adjacent thick filaments by forming and breaking cross-bridges between actin and myosin which also the same as in skeletal muscle.

Intercalated disks

- occur at the ends of the cells. Its function is to maintain cell-to-cell connection.



Important notes about the structure of the cardiac muscle cell:

Gap junctions:

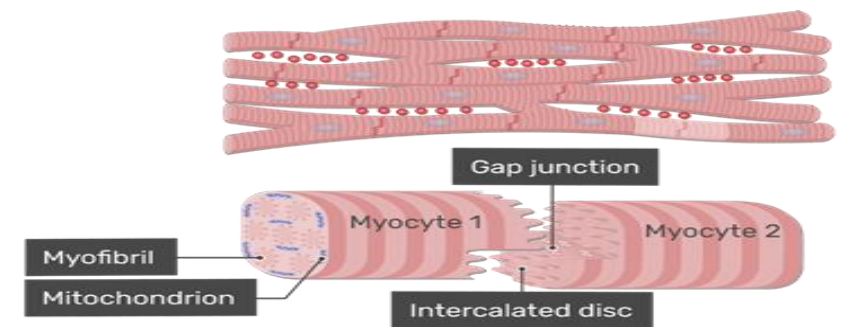
- are present at the intercalated disks.
- o Allow rapid spread of ions from cell to cell during action potentials.
- o the rapid spread of ions between cells make the heart act as **an electrical syncytium**.

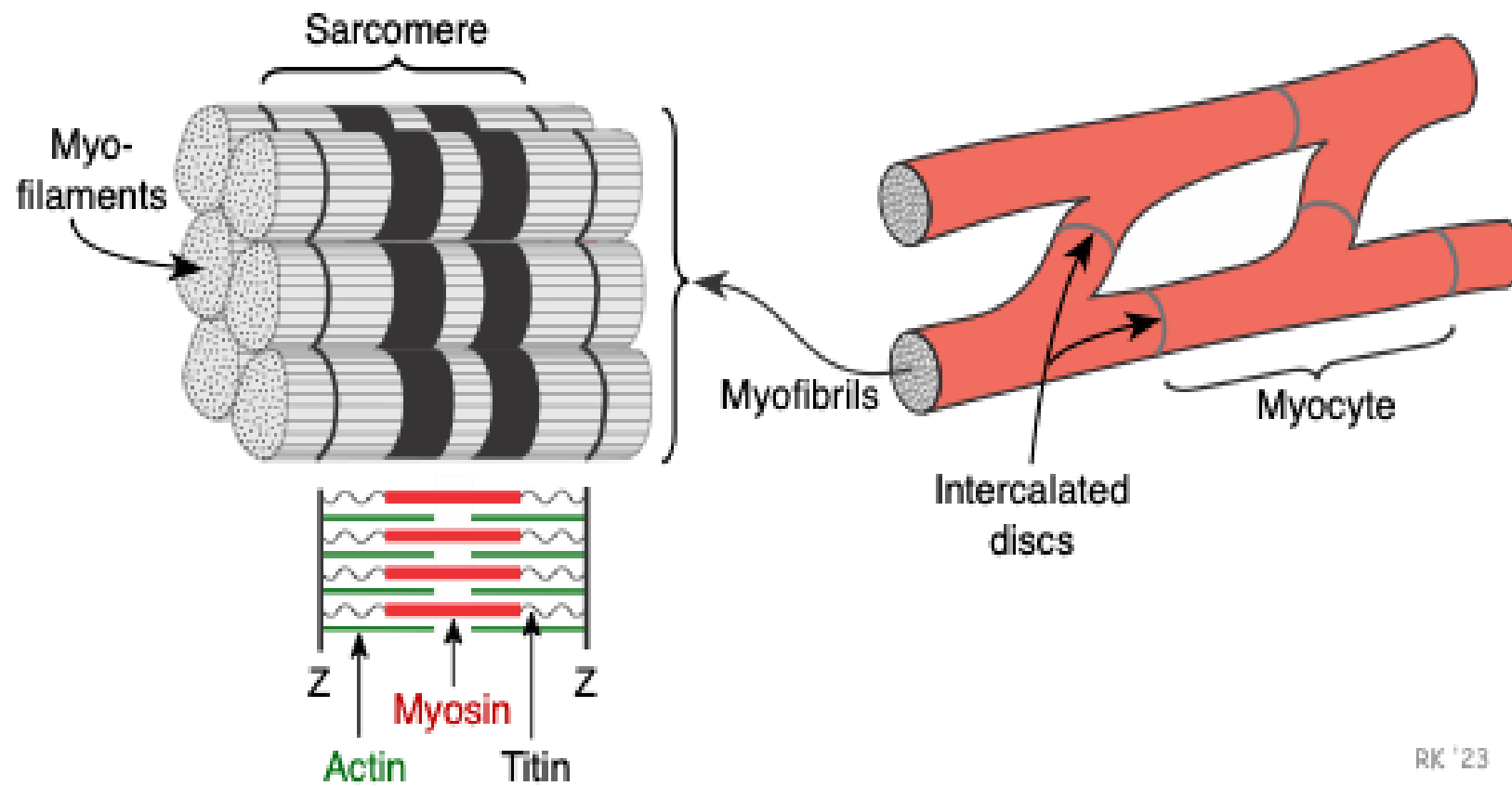
Sarcoplasmic reticulum (SR):

- they are the sites of storage and release of Ca^{2+} for excitation–contraction coupling.

T tubules

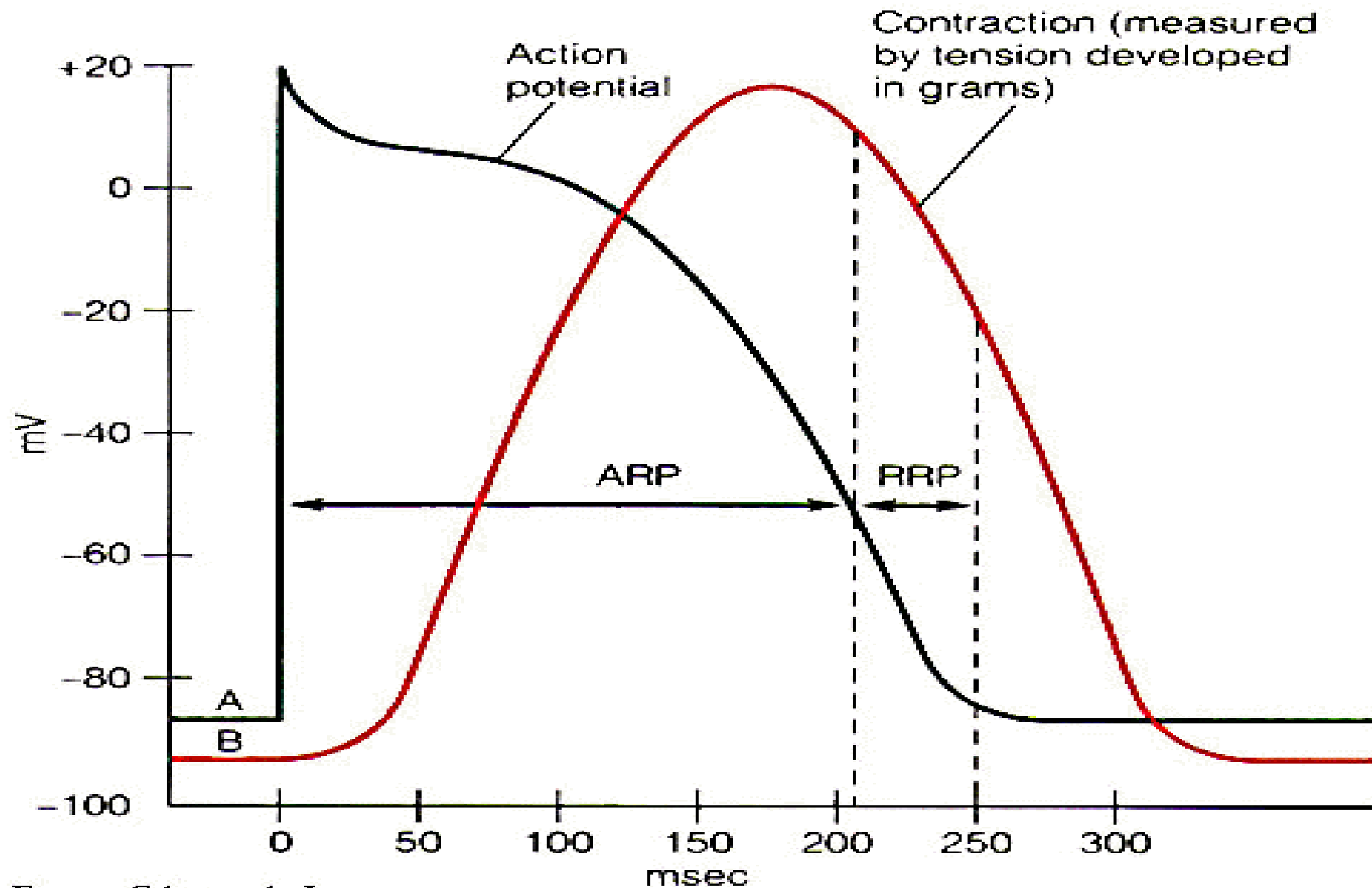
- are **well developed (How?)** and carry the action potential to the interior of the cells.





Excitation contraction coupling

- Excitation of cardiac muscle (depolarization of AP) leads to Ca^{++} entry inside cardiac muscle.
- Ca^{++} enters sarcoplasm triggers release of large amount of Ca^{++} from sarcoplasm reticulum (through Ca^{++} release channel).
- Ca^{++} binds to **troponin-C** thus tropomyosin uncovers active site. Interaction between actin and myosin followed by sliding of actin over myosin leads to contraction.
- Removal of Ca^{++} from sarcoplasm leads to relaxation.
- **Relaxation:** when action potential ends, Ca^{2+} is pumped back into the SR via a Ca^{2+} ATPase pump and expelled outside the cell with the help of a $\text{Ca}^{2+}/\text{Na}^{+}$ pump. This reduces intracellular $[\text{Ca}^{2+}]$ and removes Ca^{2+} from troponin, which terminates contraction of the sarcomere



Factor affecting contractility

- 1-All or none Rule
- 2- tetanus
- 3- Length – Tension relationship
- 4- Force - velocity relationship
- 5- Staircase phenomena
- 6- Inotropic state(Contractile state)

1- All or none Rule

- Cardiac muscle contract maximally or does not contract at all
- Increase stimulus intensity more than threshold will **not change the strength of contraction**
- Stimulation by stimulus below threshold will **produce No response**

2-Tetanus

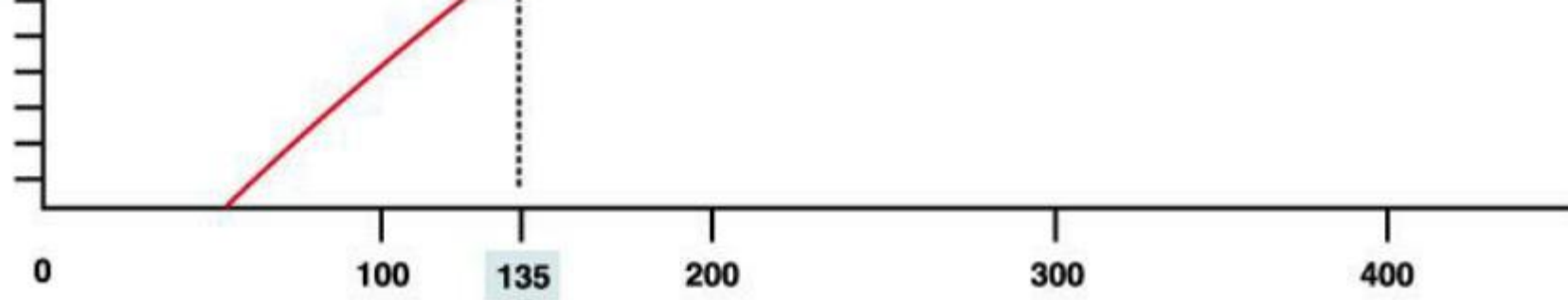
- Tetanus means sustained contraction (successive contractions fuse & cannot be distinguished from one another)

Tetanus cannot occur in cardiac muscle due to: Long ARP which coincide with systole

3-Length – Tension relationship:

- Force of contraction of heart is directly proportional to initial length of cardiac muscle fiber within limit (starling's law):
 - Increase initial length of cardiac muscle fiber will increase force of contraction to reach maximum at certain length which called L max
 - At L max all cross bridge of myosin bind to active site of actin
 - Increase length above L max will decrease force of contraction

Force
stroke



Stretch: indicated by ventricular
end-diastolic volume (mL)

4-Force - velocity

relationship:

- Initial velocity of shortening of cardiac muscle is inversely related to load moved

during contraction:

- Increase load carried by muscle will decrease initial velocity of shortening
- Decrease load will increase initial velocity of shortening

Force velocity curve

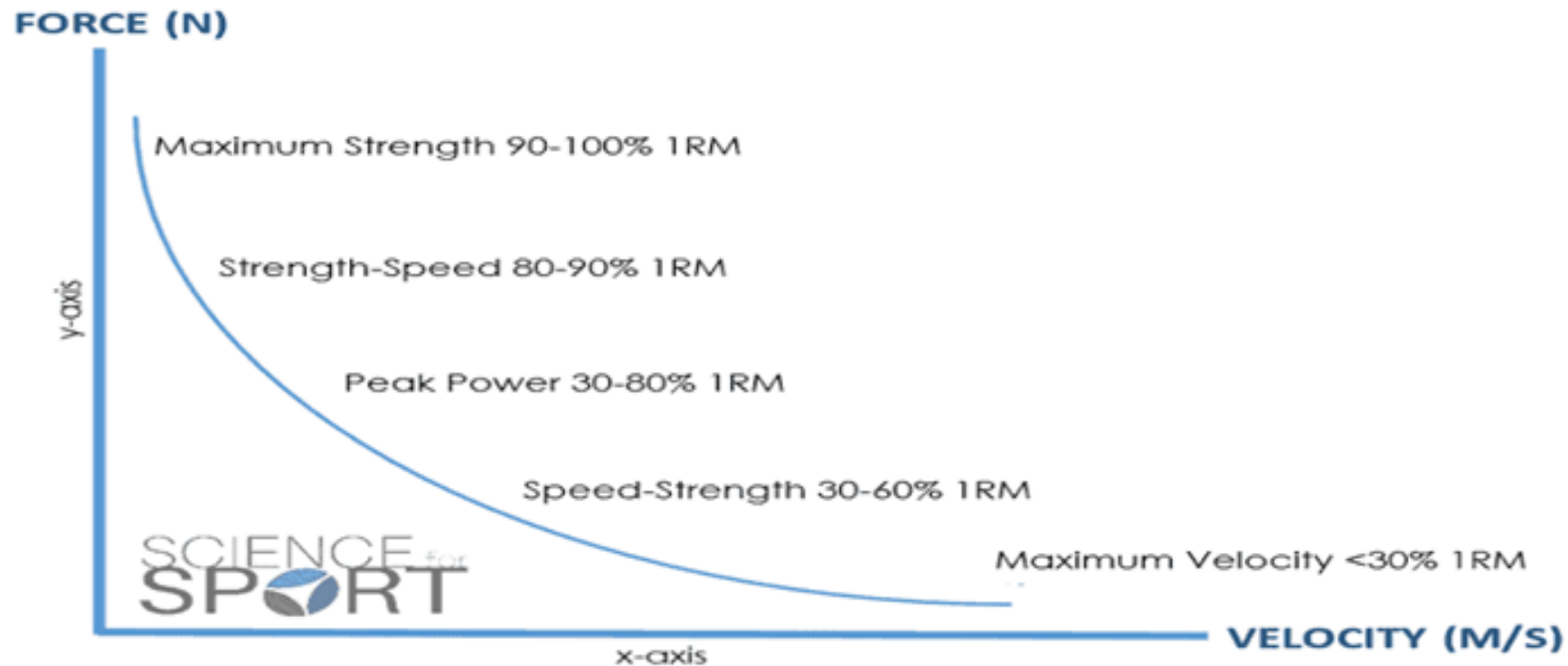


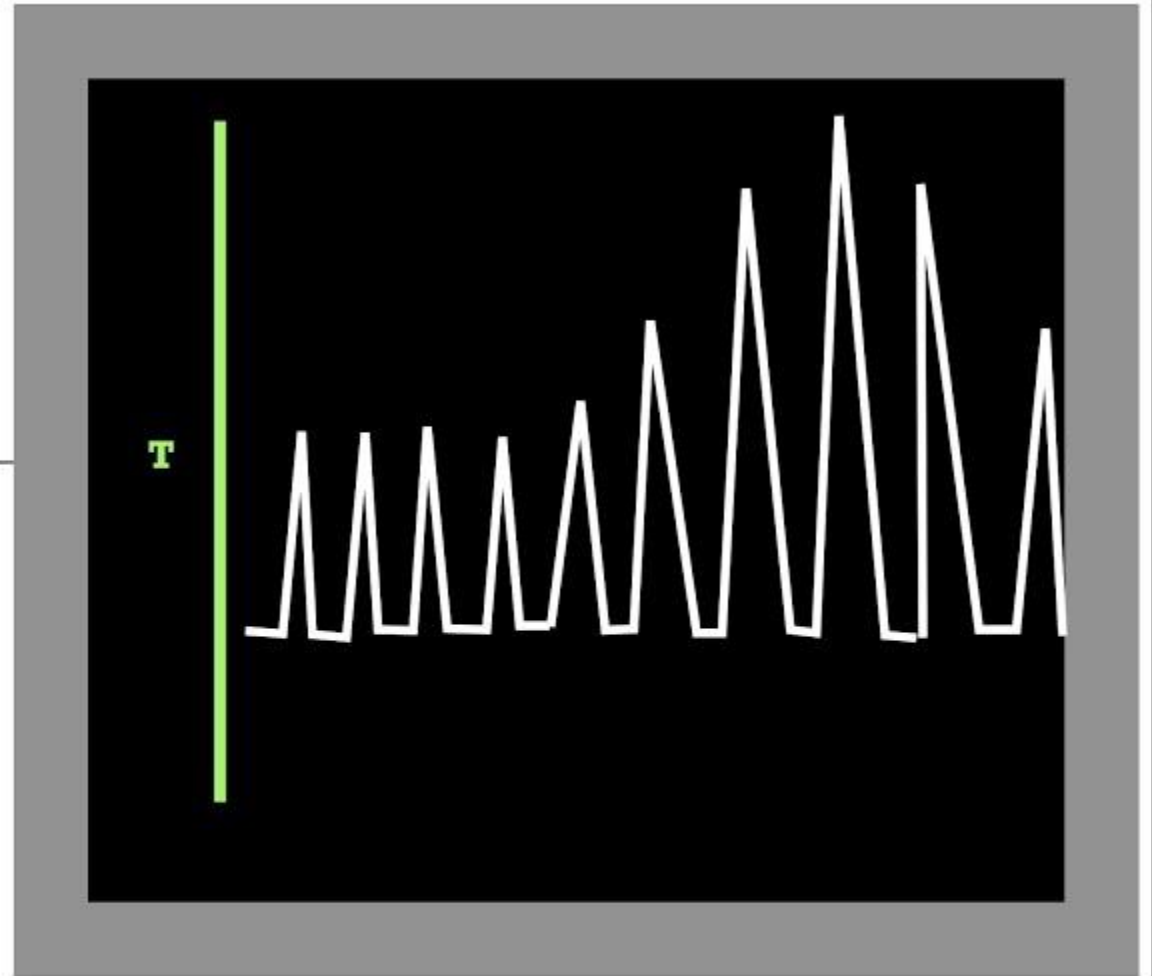
Figure 1. The Force-Velocity Curve

5-Staircase phenomena:

- Applying repeated stimuli to cardiac muscle within very short time:
 - The 2nd contraction will be higher than the first
 - And 3rd will be higher than 2nd
 - Until a new level of contraction is reached
- Due to:
 1. Increase Ca^{++} ion
 2. Failure of reuptake (due to weakness of Ca^{++} pump)



FASTER



THE STAIRCASE PHENOMENON

6-Inotropic state: (Contractility)

- The ability of cardiac muscle to develop force (contract)
- Contractile state could:
 - ① Increase (+ve inotropic)
 - ② Decrease (-ve inotropic)

[A] Positive Inotropics Factors:

① Sympathetic stimulation (β 1-adrenergic).

② Catecholamines

③ Glucagon hormone

④ Digitalis

⑤ Xanthines eg caffeine

N.B: All these factors act through increasing Ca^{++} in sarcoplasm

- [B] Negative Inotropic Factors:**
- ① Parasympathetic stimulation
 - ② Acetylcholine
 - ③ Hypoxia (due to ischemia)
 - ④ Calcium channel blockers "CCB"
 - ⑤ Anesthetics
 - ⑥ Anti-arrhythmic drugs

References

- 1- Guyton and Hall Textbook of Medical Physiology 14th edition**
- 2- Lippincott Illustrated Reviews: Integrated Systems
Sandra K. Leeper-Woodford, MS,**
- 3- Oxford Handbook of Medical Sciences ,2nd edition,
Robert Wilkins, Simon Cross, Ian Megson, David Meredith.**

The text "Thank you" is written in a dark blue, elegant cursive script. It is surrounded by several watercolor-style hearts in various colors: orange, pink, purple, and blue. The hearts are scattered around the text, with some overlapping it. The overall style is soft and artistic.

Thank
you