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, homometric 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

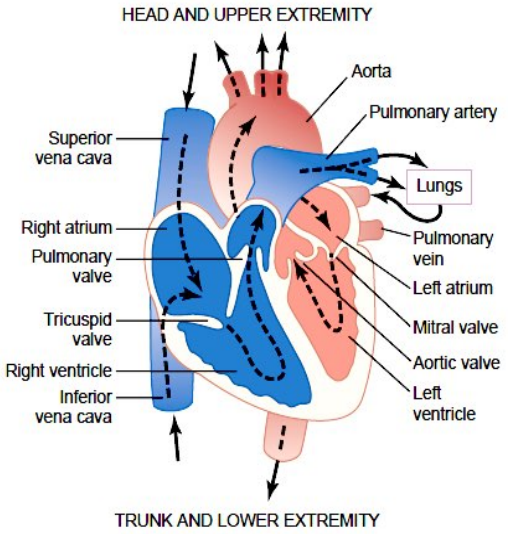
Heart

In systole:

Cardiac contraction to eject blood

In diastole:

Cardiac relaxation to be filled with blood



The heart is formed of: 2 Separa

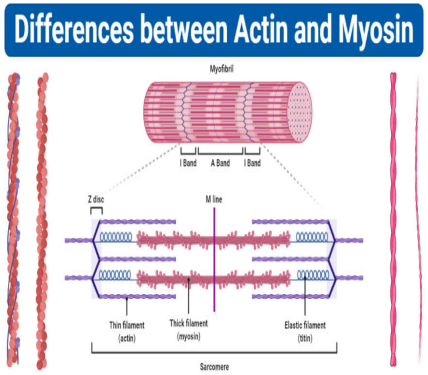
1]Right side pump (volume pump)

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.

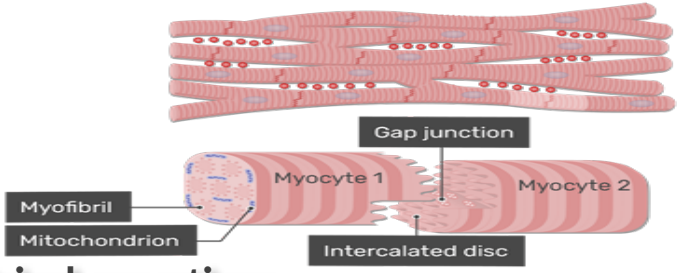
Important notes about the structure of the cardiac muscle cell:

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.**

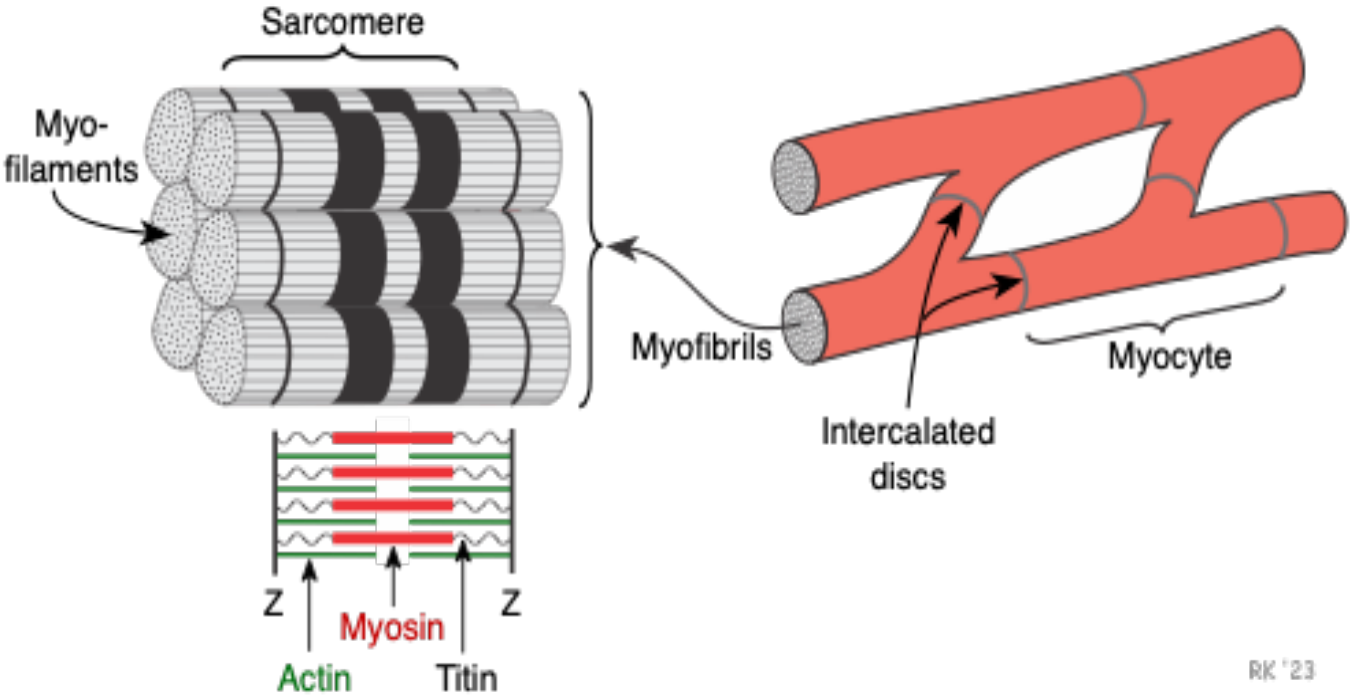


Sarcoplasmic reticulum (SR):

- they are the sites of storage and release of Ca2+ for excitation–contraction coupling.

T tubules

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

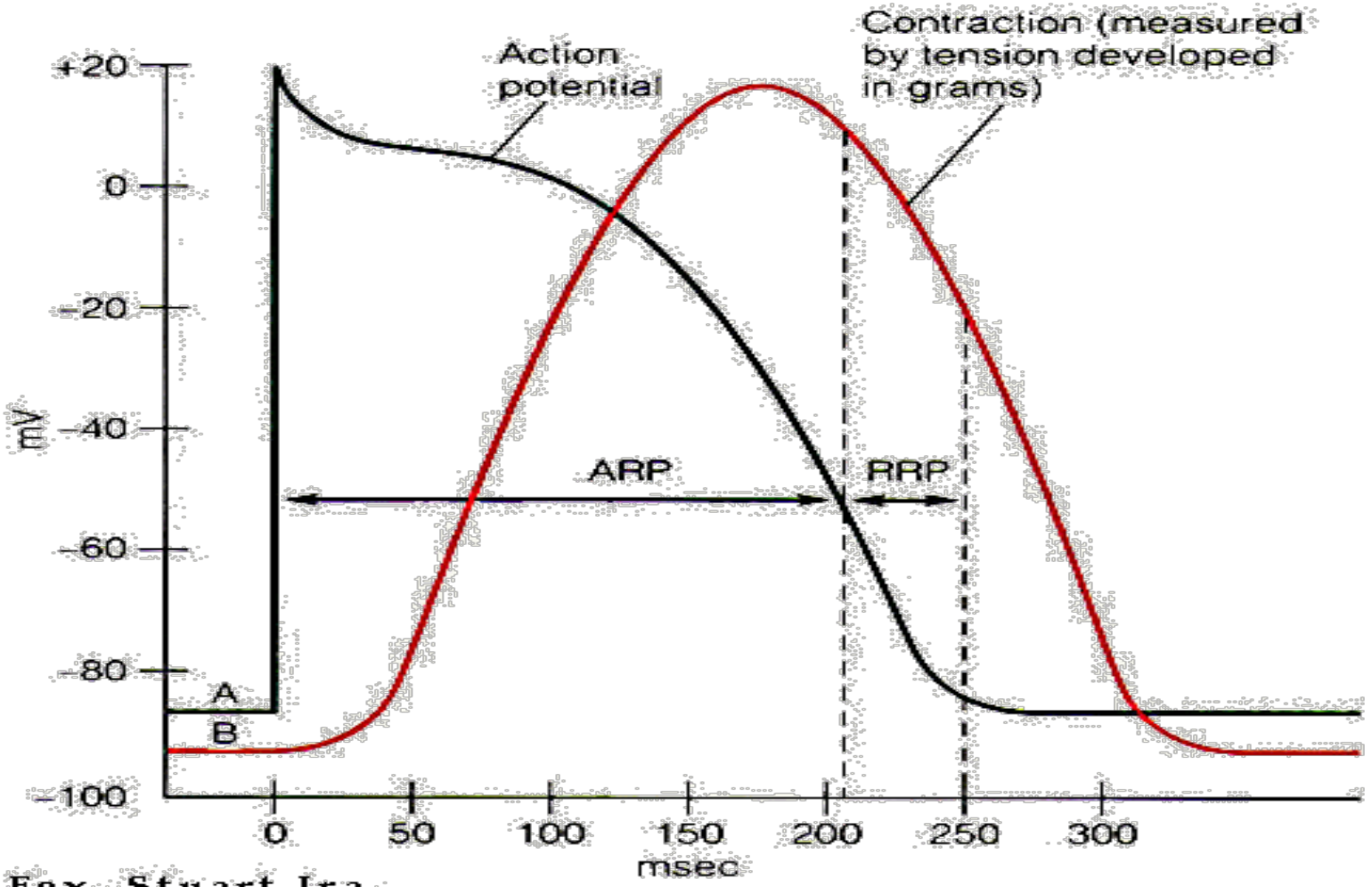

Sarcomere
Myo-
filaments
Myofibrils
Myocyte
Intercalated
discs
Z
Myosin
Z
Actin
Titin
RK '23

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, Ca2+ is pumped back into the SR via a Ca2+ATPase pump and expelled outside the cell with the help of a Ca2+/Na+ pump. This reduces intracellular [Ca2+] and removes Ca2+ from troponin, which terminates contraction of the sarcomere



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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

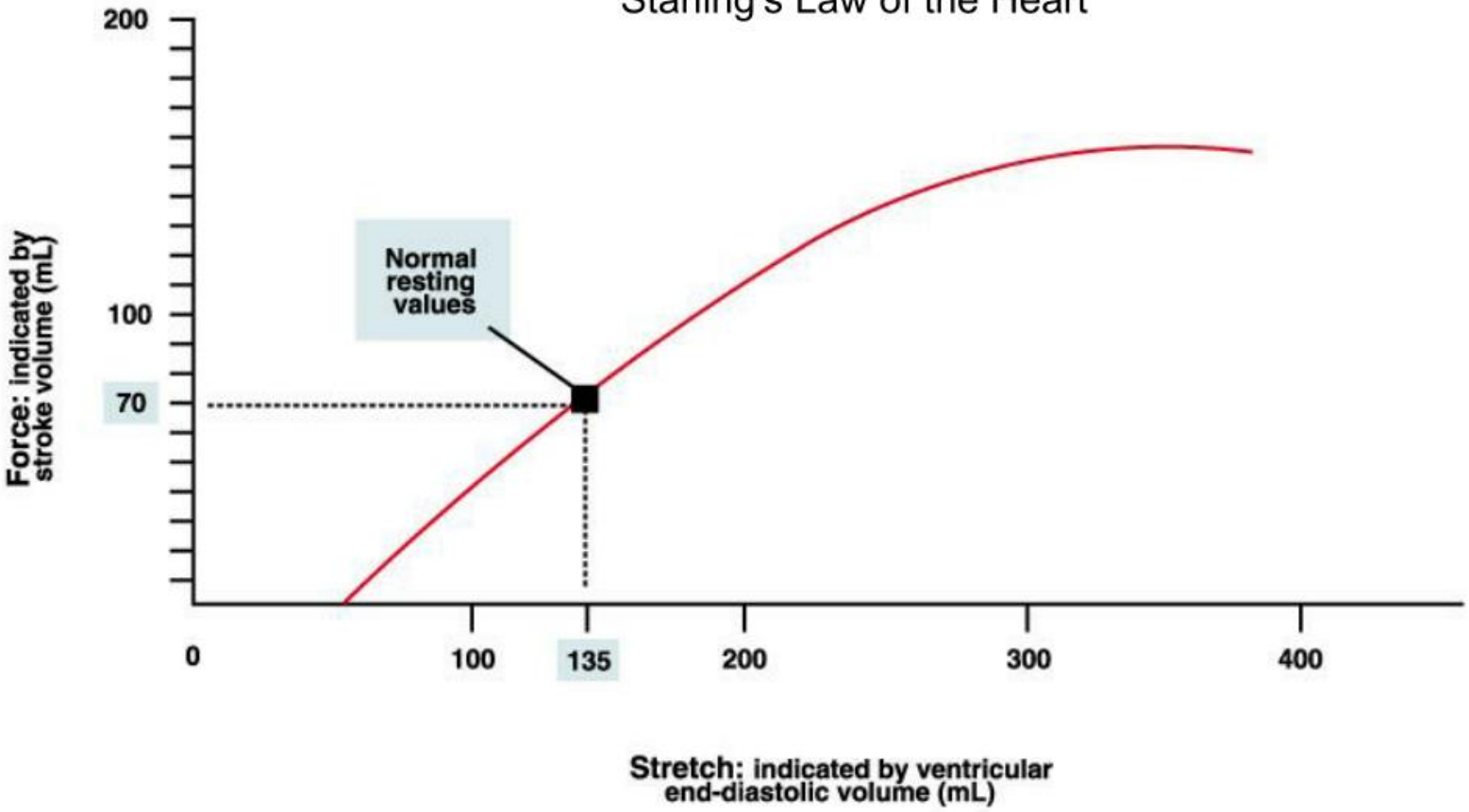
- Tetanus means sustained contraction (successive contractions fuse & cannot be distinguished from one another)

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 low):
  - 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

Starling's Law of the Heart



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

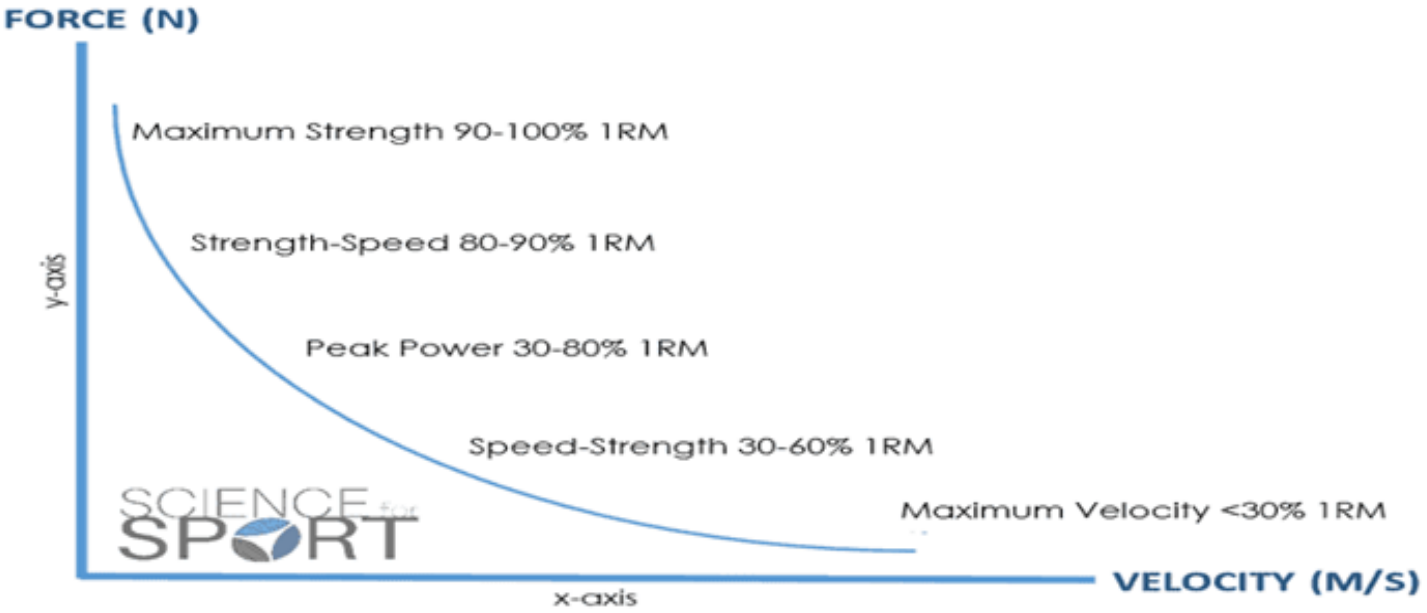
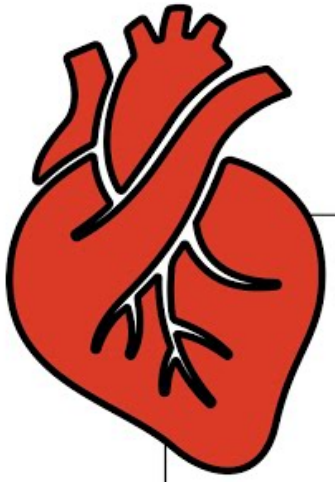


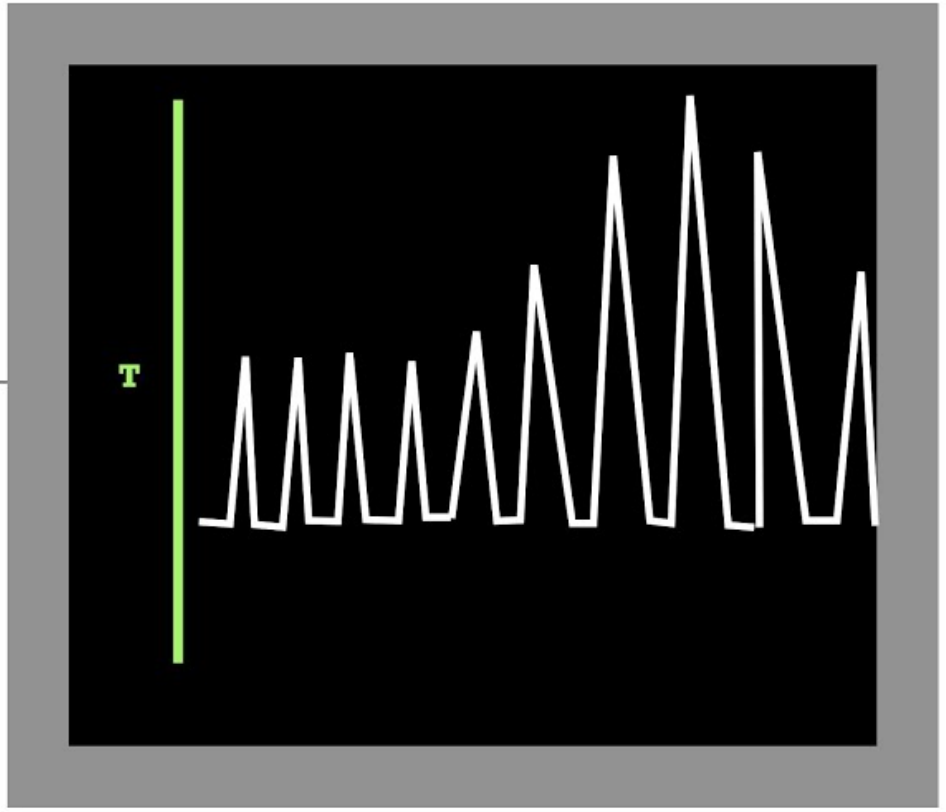
Figure 1. The Force-Velocity Curve

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 (β1-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

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thank
you