Numbers of slides are from handout 1

Please study the slides very well, I've tried to mention only what's not written but you may find what's written here and there either with additional information or in another way.

#13

Veins

-When the post capillary venule reaches 50 µm in diameter and a COMPLETE layer of smooth muscles is formed (complete tunica media), it is called VIEN.

#14

-Elastic artery is ric h in elastic tissue thus it expands and recoils.

-**Medium sized Veins** (systemic veins) are rich in collagen which gives it the COMPLIANCE property.
- The graph shows: that there is a large change in the volume of blood within veins with no significant change in the pressure (15-20 mmHg).
but for arteries less than 500 ml was added and the pressure reached more than 150 mmHg.

- Veins contain more than 60% of our blood under low pressure because they are compliant. The abundance in collagen with little elastic tissue allows veins to stretch with little recoil.

- In the media about 2-3 layers of smooth muscles so there will be low resistance for blood flow.

#17 - A (very important slide)

-**The partial vasoconstriction** is due to the contraction of the circular smooth muscle layer in the wall of the arteriole under the sympathetic control.

- **Myogenic activity**:
the tone of skeletal muscles assist in posture and in bringing blood form the lower limb towards the heart; when they contract they compress the veins which lie within the muscle in order to allow for blood to flow upward against gravity.

The tone in skeletal muscles is neurogenic; if the neural signal didn't reach the muscle there will not be any contraction.

-In the arterioles the tone is mainly myogenic, in the cell membrane of smooth muscle cell of the arteriolar wall a spontaneous depolarization down to threshold could be done (explained in the next point).

- PACE MAKER ACTIVITY: without neural signal, some Na+ channels open > Na+ enters the cell > gradually the voltage reaches the threshold > firing and Action potential will take place followed by contraction.
This is a special property for smooth muscles in the arteriole wall, and it causes a partial contraction which is enhanced by the sympathetic activity.

- The Partial constriction can be increased (vasoconstriction) or decreased (vasodilation).
Adenosine and lactate cause dilation.
Angiotensin II is a very powerful vasoconstrictor

- in Generalized sympathetic activity:
*Arterioles will undergo vasoconstriction; the blood flow to the organs is limited (initially)!
*At the same time this generalized vasoconstriction elevates the blood pressure, how?
The sympathetic has a positive inotropic effect on the heart i.e. the force of contraction is increased, as the ventricular contraction is increased and the heart rate is increased > the cardiac output will be increased too.

*Sympathetic increases the peripheral resistance by narrowing the arterioles,
as a result the blood pressure will increase!
blood pressure is the main driving force for blood flow, so it will insure that the flow will reach the organ, but the arteriole is constricted, then LOCAL METABOLITES override the sympathetic effect.
*If the organ has a high metabolic activity, there will be accumulated *(adenosine, lactic acid, k+, CO2)*, which override the sympathetic effect and cause vasodilation.

So there is no contradiction; the effect is not counterproductive.

- Remember blood flow to the organs depends on pressure gradient as a result of pumping blood and the peripheral resistance.

#17 – B

-The coronary circulation is largely controlled by metabolites.

α and β receptors are found on the arterioles of coronary arteries, under the sympathetic effect > vasoconstriction. This seems illogical! But it is true because the sympathetic is activated while you are performing an activity, so heart rate is high and the metabolites will accumulate especially adenosine and cause vasodilation.
So apparently, sympathetic causes vasoconstriction but under the effect of metabolites, vasodilation.

- Norepinephrine does not cross the BBB, so it has no effect on the brain (cerebral circulation).

- In skeletal muscle, at rest **α** predominate causing vasoconstriction.

#18

-**Cardiac muscle fiber** is a group of cells (cardiac cells = cardiac myocyetes) joined end to end by the intercalated disk which contains (desmosomes, tight junction and Gap junction).

- The Cytoplasm is full of myofibrils and shows cross striations which mean that the fibrils are arranged in sarcomeres.

In skeletal muscles many nuclei are in the periphery but myocyes possess single nucleus centrally placed.

- **Gap Junction** is an area of low electrical resistance which allow rapid spread of electrical current (ion flow)
so there is a ***functional Syncetiam*** in the cardiac muscle fibers (the heart will contract as one unit) because Gap junction allows the impulse after reaching a cell to reach thousands of cells within a fraction of second causing AP.

#21

- There are two types of fibers in the heart:
1- Contracting > pump the blood.

2- Conducting > initiate and transmit action potential.

- Conducting system in the heart (SA node to the AV node to AV bundle which gives two branches: left and right, and each brand ends as PURKINJI FIBERS in the ventricular wall)

The impulse starts at the SA NODE, at the right atrium above the SVC opening, by spontaneous depolarization down the threshold and then the Action potential takes place.

- **Purkinji fibers** are large fibers that transmit the action potential very fast. They are rich in capillaries (for blood and nutrients) and rich in mitochondria (for ATP).