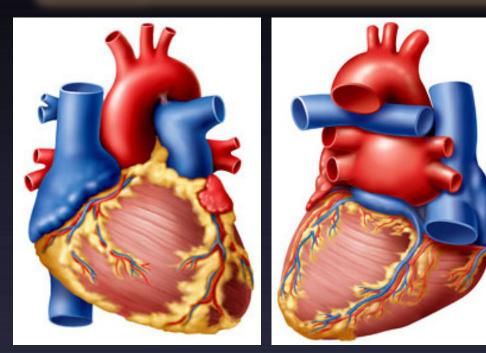


CARDIOVASCULAR SYSTEM

VENOUS RETURN & CARDIAC OUTPUT



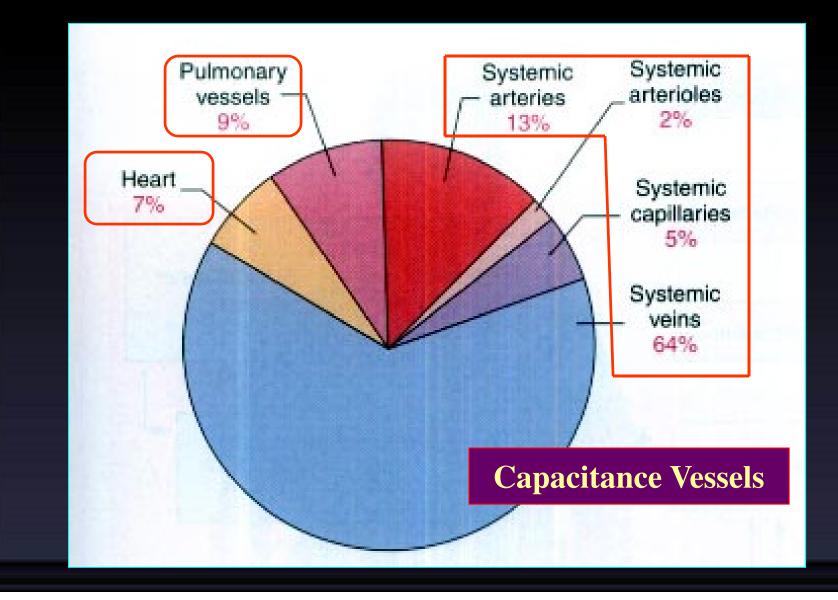
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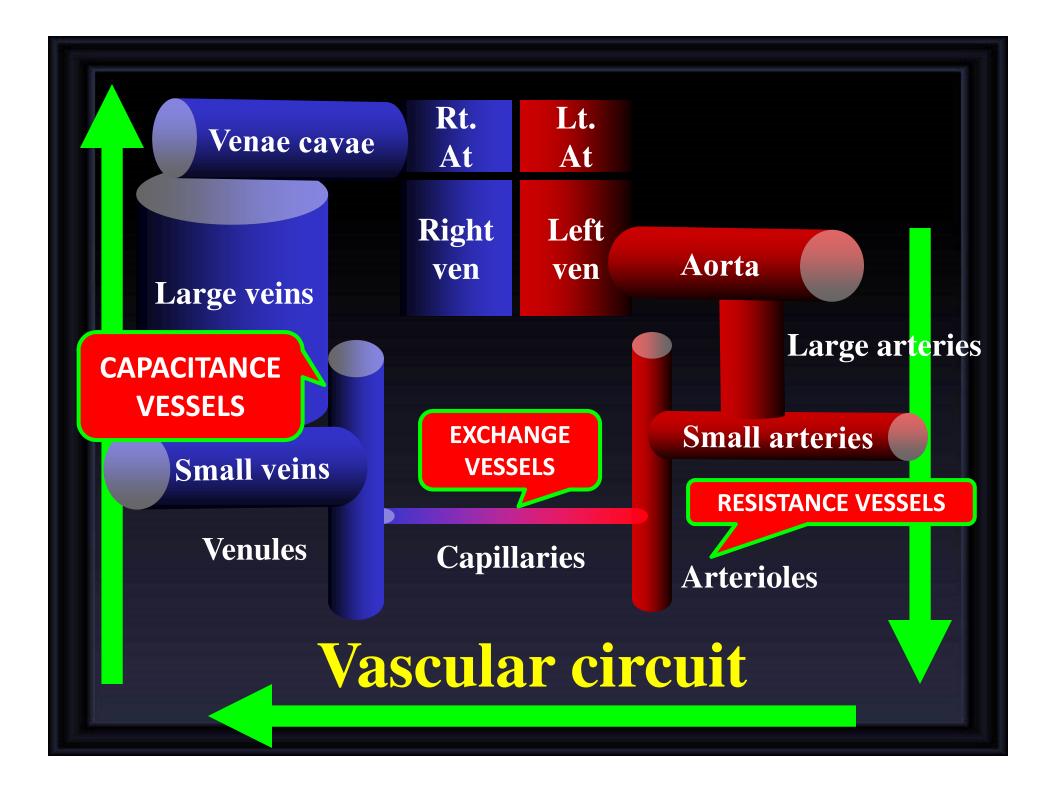
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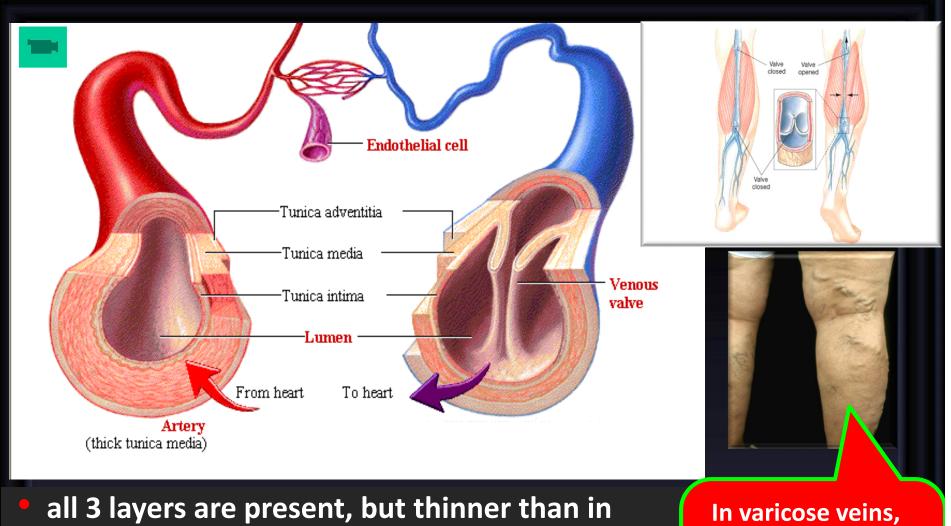
OBJECTIVES

- * At the end of the lecture you should be able to
- Discuss functions of the veins as blood reservoirs.
- Describe measurement of central venous pressure (CVP) and state its physiological and clinical significance.
- State determinants of venous return and explain how they influence venous return.
- Define mean systemic filling pressure, give its normal value and describe the factors which affect it.
- Explain the effect of gravity on venous pressure and explain pathophysiology of varicose veins.
- Describe vascular and cardiac function curves under physiological and pathophysiological conditions.

DISTRIBUTION OF BLOOD







- all 3 layers are present, but thinner than in arteries of corresponding size (external diameter).
- Veins have paired semilunar, bicuspid valves to restrict backflow in lower extremities:

In varicose veins, blood pools because valves fail causing venous walls to expand.

VASCULAR COMPLIANCE (VASCULAR CAPACITANCE)

TOTAL QUANTITY OF BLOOD THAT CAN BE STORED IN A GIVEN PORTION OF THE CIRCULATION FOR EACH MMHG PRESSURE RISE

Vascular compliance =

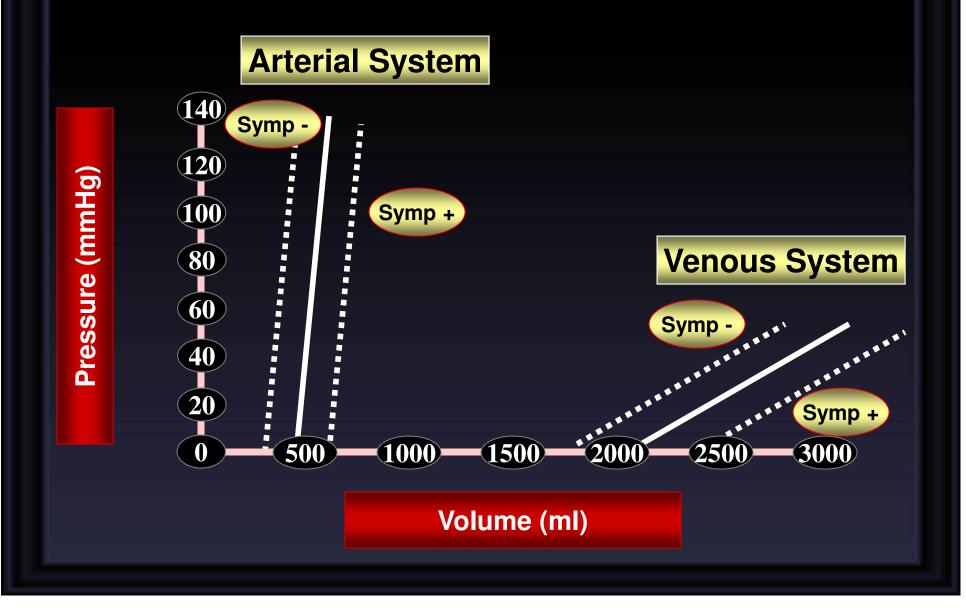
Increase in volume

Increase in pressure

Compliance is equal to distensibility times volume

The compliance of a systemic vein is about 24 times that of its corresponding artery because it is about 8 times as distensible and it has a volume about 3 times as great $(8 \ge 3 = 24)$





Veins are blood reservoirs

When the body is at rest and many of the capillaries are closed, the capacity of the venous reservoir is increased as extra blood bypasses the capillaries and enters the veins.

When this extra volume of blood stretches the veins, the blood moves forward through the veins more slowly because the total cross sectional area of the veins has increased as a result of the stretching. Therefore, blood spends more time in the veins.

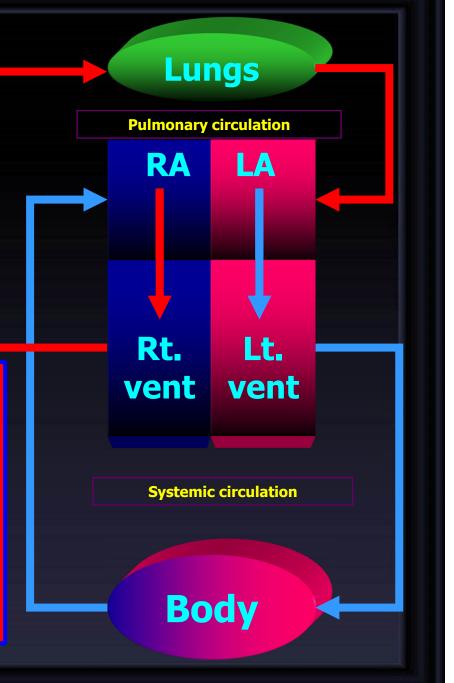
• When the stored blood is needed, such as during exercise, extrinsic factors reduce the capacity of the venous reservoir and drive the extra blood from the veins to the heart so that it can be pumped to the tissues.

Arterial blood pressure Blood pressure is the force the blood exerts against the walls of the blood vessels			
Systolic pressure	Maximum pressure during systole 120mmHg		
Diastolic pressure	Minimum pressure during diastole 80 mmHg		
Pulse pressure	Systolic pressure – diastolic pressure 40 mmHg		
Mean pressure	Diastolic pressure + (1/3 pulse pressure) 93 mmHg		
Mean arterial pressure is the main driving force for blood flow			

VENOUS RETURN

Normally venous return must equal cardiac output (CO) when averaged over time because the cardiovascular system is essentially a closed loop. Otherwise, blood would accumulate in either the systemic or pulmonary circulations.

Venous return is determined by the difference in pressure between the venous pressure nearest to the tissues (mean systemic filling pressure; mean circulatory pressure; MCP) and the venous pressure nearest to the heart (CVP).



 CVP: is the venous pressure in the right atrium and the big veins of the thorax (right atrial pressure (RAP) = jugular venous pressure).

Venous pressure is measured with a catheter inserted in the central venous system, usually SVC.

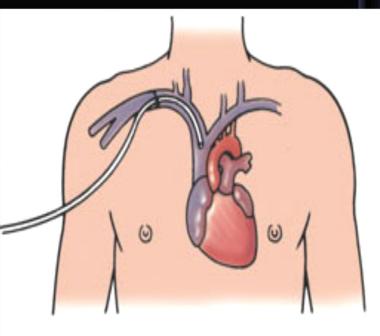
The normal range of the CVP = 0 - 4 mmHg.

It is the force responsible for cardiac filling.

CVP is used clinically to assess
 hypovolaemia and during IV transfusion
 to avoid volume overloading.

CVP is raised in right-sided failure.

Central venous pressure (CVP)



CATHETER (LINE) ENTERING VEIN

Mean systemic filling pressure Mean circulatory pressure; MCP

It is the pressure nearest to the tissues and is about / mm Hg.

The value for right atrial pressure at which venous return is zero is called the mean systemic filling pressure. It is the point at which the vascular function curve intersects the X-axis (i.e., where venous return is zero and right atrial pressure is at its highest value).

It is affected by:

Blood volume (it is directly proportional to blood volume).
 Venous capacity (it is inversely proportional to the venous capacity).





VENOCONSTRICTION T MCP

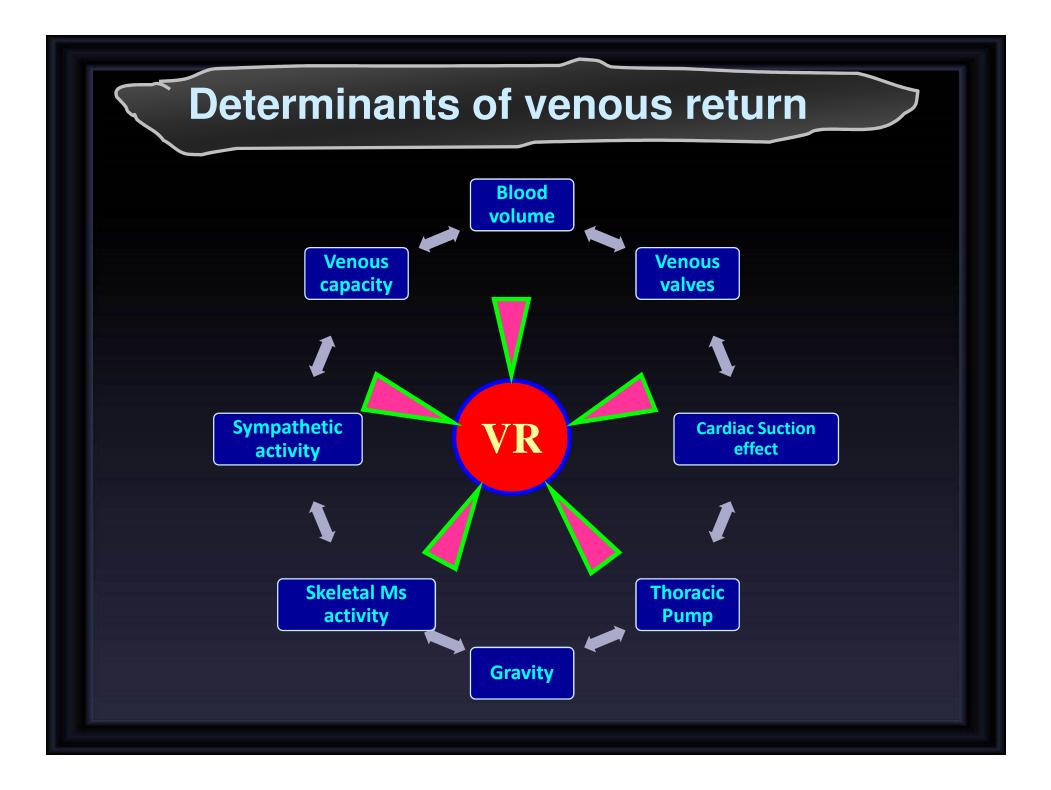
Pulmonary

Systemic

LV

RV

VENODILATION \downarrow MCP



1. Blood volume:

At constant venous capacity, as the blood volume $\uparrow \rightarrow$ the MCP $\uparrow \rightarrow \uparrow$ VR.

At constant venous capacity, as the blood volume $\downarrow \rightarrow$ the MCP $\downarrow \rightarrow \downarrow$ VR.

Venous capacity: is the volume of the blood that the veins can accommodate.

• At a constant blood volume, as the venous capacity $\uparrow \rightarrow$ the MCP $\downarrow \rightarrow \downarrow$ VR.

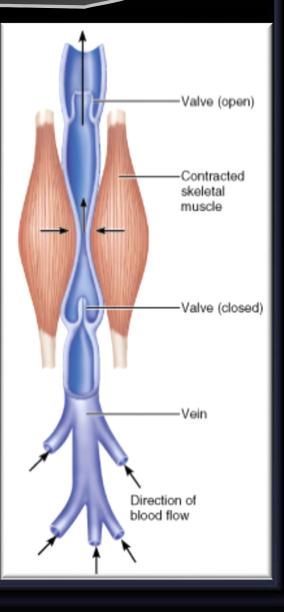
As the venous capacity $\downarrow \rightarrow \uparrow$ VR.

3. Sympathetic activity:

Venous smooth muscle is profusely supplied with sympathetic nerve fibers.

Sympathetic stimulation → venous vasoconstriction → modest ↑ in mean systemic filling pressure (MCP) → ↑ VR.
 Sympathetic stimulation → ↓ venous capacity → ↑ VR.
 The veins normally have such a large diameter that the moderate vasoconstriction accompanying sympathetic stimulation has little effect on resistance to flow.

4. Skeletal muscle activity:
Skeletal muscle contraction → external venous compression → ↓ venous capacity → ↑ VR (This is known as skeletal muscle pump).
Skeletal muscle activity also counter the effects of gravity on the venous system.



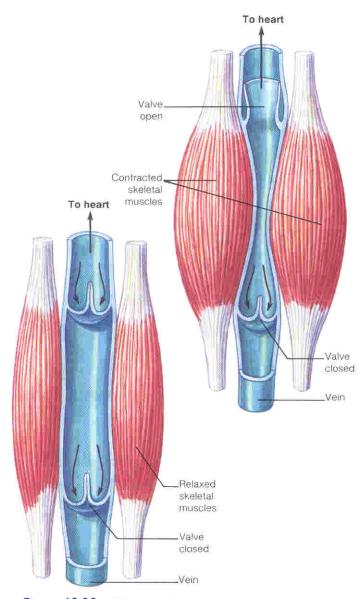
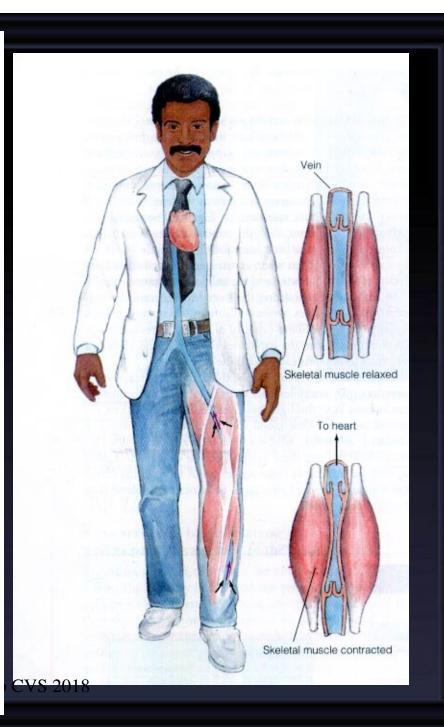


Figure 13.28 The action of the one-way venous valves. Contraction of skeletal muscles helps to pump blood toward the heart, but the flow of blood away from the heart is prevented by closure of the venous valves.

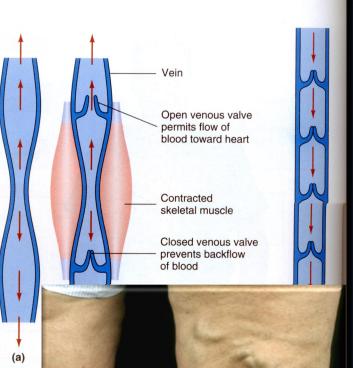


5. Venous valves:

These valves permit blood to move forward towards the heart but prevent it from moving back toward the tissues.
These valves also play a role in counteracting the gravitational effects of the upright posture.

Skeletal muscle pump is ineffective when the venous valves are incompetent.

- Chronically raised pressure in the veins leads to pathological distension of the veins (varicose veins).
- Increased capillary filtration leads to swelling (edema) with trophic skin changes and ulceration (venous ulcers).



During inhalation:

ATORY

6. Respiratory activity (respiratory pump; thoracic pump):

As the venous system returneed to demo the heart from the lower regions of the body, it travels through the chest cavity. The pressure in the chest cavity is 5 mm Hg less than atmospheric pressure.

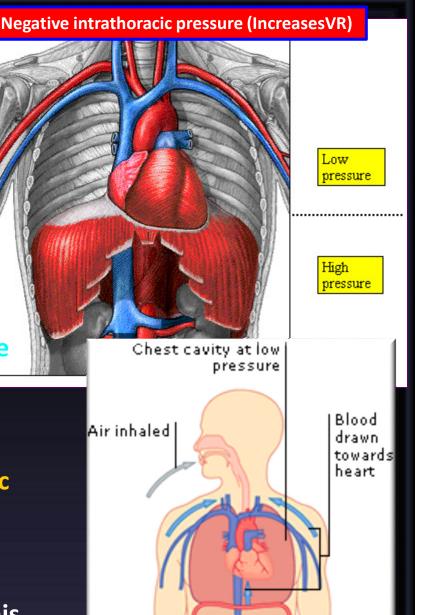
The venous system in the limbs and abdomen is subjected to normal atmospheric pressure.

Thus, an externally applied pressure gradient exists between the lower veins and the chest veins, promoting venous return (this is known as the respiratory pump).

Pressure decreases in

thoracic cavity.

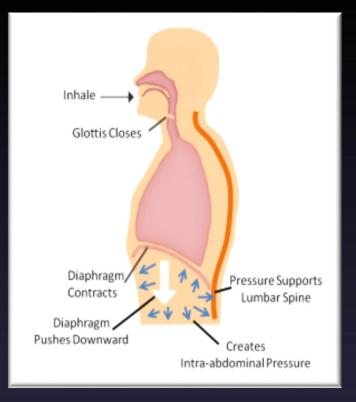
 Pressure increases in abdominal cavity.



Diaphragm

Determinants of venous return What is the effect of Valsalva maneuver on venous return?





Physiological changes in CO

During the first 3 hours after meals, the CO is increased by ≈ 30% to enhance blood flow in the intestinal circulation.

Later months of pregnancy are accompanied by > 30% increase in CO due to increased uterine blood flow.

At environmental temperature above 30 °C, the CO is increased due to increased skin blood flow. Also at low environmental temperature CO is increased due to shivering that increases blood flow to the muscles.

Increased sympathetic activity during anxiety and excitement enhances the CO up to 50% - 100%.

 Sitting or standing from the lying position deceases the CO by 20% -30%.

Exercise:

Effects of exercise on heart rate and SV

Moderate Exercise

- HR increases to
- SV increases to
- CO increases to

200% of resting (140 bts/min) 120% (85ml) 240% (12L)

Severe Exercise

HR increases to	300% of resting (200 bts/min)
SV increases to	175% (125ml)
CO increases to	500% - 700% (25 - 35 L)

In athletes, maximum CO may be 35L or more - can't increase maximum HR beyond 200 bts - hence - SV increases to 175 ml.

Pathological low or high cardiac output

Causes of low CO:

- Low VR (e.g., haemorrhage)
- **Reduced contractility (e.g., heart failure)**
- Tachyarrhythmias (e.g., atrial fibrillation and ventricular tachycardia)
- Marked bradycardia (e.g., complete heart block)

Causes of high CO:

- □ Hyperthyroidism: the increase in the CO is due to the high metabolic rate \rightarrow vasodilatation \rightarrow ↑ CO to 50%+ of control.
- AV fistulas.
- **G** Fever.
- Anaemia.
- Anxiety.

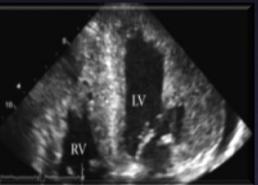
Measurement of cardiac output

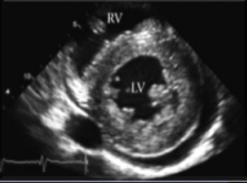
- Ultra-fast computer tomography
- Fick's principle
- 2-Dimensional Echocardiography

2-Dimensional Echocardiography

Records real-time changes in ventricular dimensions during systole and diastole. It thus computes stroke volume, which when multiplied by heart rate, gives the cardiac output.

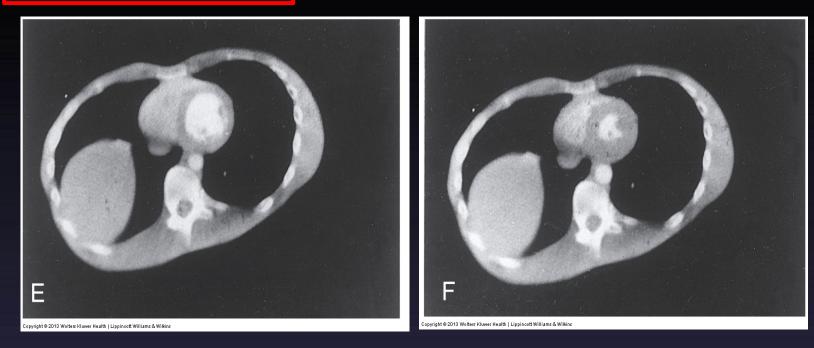






Pathological low or high cardiac output

Ultra-fast computer tomography



Can measure changes in ventricular diameter at several depths to estimate changes in ventricular volume.

Measurement of cardiac output

Fick's principle

Fick's principle assumes that the amount of oxygen consumed = the amount of oxygen delivered by the arterial blood <u>minus</u> the amount of oxygen e venous blood of the organ.

