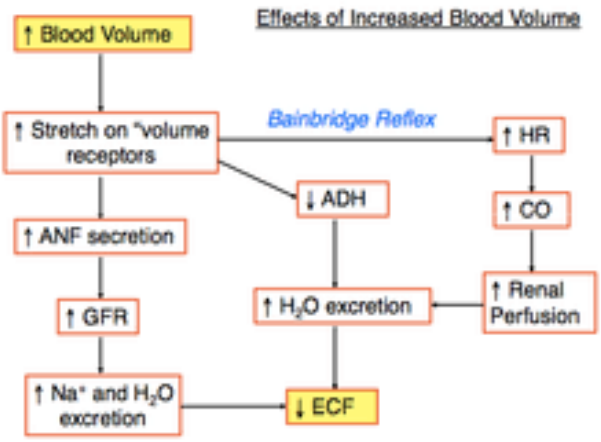
CARDIAC OUTPUT AND VENOUS RETURN

- CARDIAC OUPUT:
  - Quantity of blood pumped into the aorta each minute by the heart
- VENOUS RETURN:
  - Quantity of blood flowing from the veins into the right atrium each minute
  - **Must equal cardiac output**
- NORMAL VALUES:
  - Varies widely
  - Many factors affect cardiac output:
    - **Age**
    - Body **size**
    - Level of body **metabolism**
    - **Exercise**
  - CARDIAC INDEX:
    - Cardiac output increases approximately in proportion to the surface area of the body
    - Cardiac index is the cardiac output per square metre of BSA
      - Reaches a peak age 10 and then gradually declines thereafter

FRANK-STARLING MECHANISM:

- Cardiac output is controlled by venous return primarily
- FRANK-STARLING LAW OF THE HEART:
  - Increased quantities of blood flow into the heart, the increased blood stretches the walls of the heart chambers
  - As a result of this stretch, the cardiac muscle contracts with increased force and empties from the expanded chambers the blood that has entered from the systemic circulation
- INCREASE STRETCH INCREASES HEART RATE:
  - **Stretching sinus node has direct effect on the rhythmicity of heart, increasing HR**
  - Stretched RA initiates a nervous reflex called the **Bainbridge reflex** ->vasomotor centre -> sympathetic nerves/vagus ->increase HR
- VENOUS RETURN TO THE HEART IS THE SUM OF ALL THE LOCAL BLOOD FLOWS THROUGH ALL THE INDIVIDUAL TISSUE SEGMENTS
  - **Tissue metabolism regulates most local blood flow**
  - Cardiac output and oxygen consumption increase in a parallel
  - All the local blood flows summate to form the venous return, and the heart automatically pumps this returning blood back into the arteries



EFFECT OF TOTAL PERIPHERAL RESISTANCE:

- Long term cardiac output varies reciprocally with the changes in TPR
- **MAP = CO x TPR**
- **Flow = pressure / resistance**
- **Pressure = Flow x resistance**
- Hence when TPR increases and MAP remains constant, CO decreases (afterload)

LIMITS OF CARDIAC OUTPUT:

- The normal heart, functioning without any special stimulation, can pump an amount of venous return up to 2.5 normal
- HYPER-EFFECTIVE HEART:
    - Two types of factors usually make the heart pump better than normal:
        - Nervous stimulation:
            - Sympathetic stimulation and parasympathetic inhibition
            - Greatly increases heart rate and contractility
        - Heart hypertrophy:
            - Increased pump effectiveness
            - Increase in muscle mass and contractile strength with increased workload
            - Seen in athletes
- HYPO-EFFECTIVE HEART:
    - Any factor that decreases the heart's ability to pump:
        - Inhibition of nervous excitation
        - Abnormal rhythm or rate
        - Valvular heart disease
        - Increased arterial pressure to pump against

        - Congenital heart disease
        - Myocarditis
        - Cardiac anoxia
        - Myocardial damage

ROLE OF THE NERVOUS SYSTEM IN CONTROLLING CARDIAC OUTPUT:

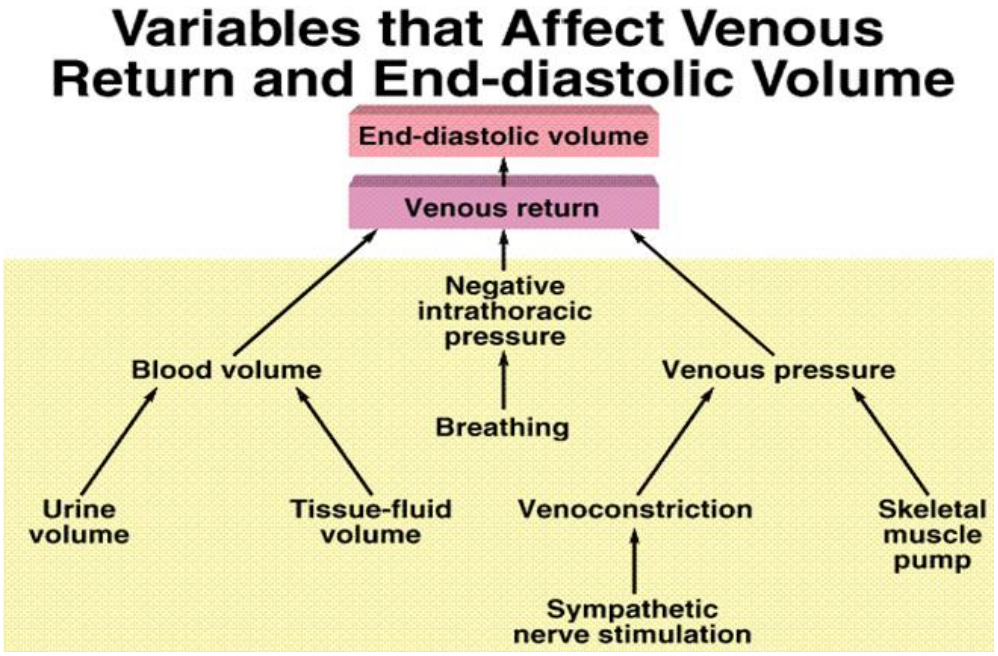
- Crucially important in maintaining arterial pressure when the venous return and cardiac output increase
- Maintenance of normal arterial pressure by nervous reflexes, is essential to achieve high cardiac outputs when peripheral tissues dilate their vessels to increase venous return
- During exercise:
    - Same brain activity that sends motor signals to peripheral muscles, also sends simultaneous signals into the autonomic nervous centres to excite circulatory activity:
        - Increased vasoconstriction
        - Increased heart rate
        - Increased contractility
    - All these changes increase BP above normal, thereby forcing more blood through active muscles

QUANTITATIVE ANALYSIS OF CARDIAC OUTPUT REGULATION

RELATE CARDIAC OUTPUT TO RIGHT ATRIAL PRESSURE

EFFECTS OF EXTERNAL PRESSURE OUTSIDE THE HEART:

- Some of the factors that can alter the intrapleural pressure and thereby shift the cardiac output curve are the following:
    - Cyclical changes during respiration (normally +/- 2mmHg, but can be as great as +/- 50mmHg during exercise and strenuous breathing)
    - Breathing against a negative pressure shifts curve to more negative RA pressure (shifts curve to the left)
    - **Positive pressure breathing (shifts curve to the right i.e decreased cardiac output for any given right atrial pressure)**
    - Opening thoracic cage converts intrapleural pressure to atmospheric from subatmospheric, shifting curve to the right
    - **Tamponade insults heart with increased external pressure, shifting curve to the right**



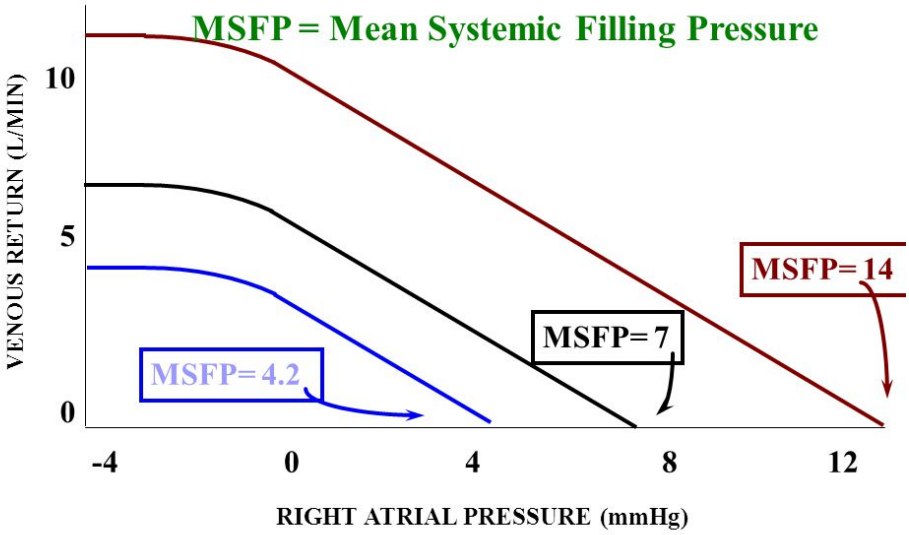
VENOUS RETURN CURVES:

- THREE FACTORS AFFECT VENOUS RETURN TO THE HEART:
  - Right atrial pressure:
    - Backward force that impedes flow of blood from the veins into the right atrium
  - Degree of filling of the systemic circulation:
    - Measured by the **MEAN SYSTEMIC FILLING PRESSURE**
      - Forces the blood toward the heart
      - **This is the pressure measured everywhere in the systemic circulation when all flow of blood is stopped**
  - Resistance to blood flow between the peripheral vessels and the right atrium

Venous return dependent on:

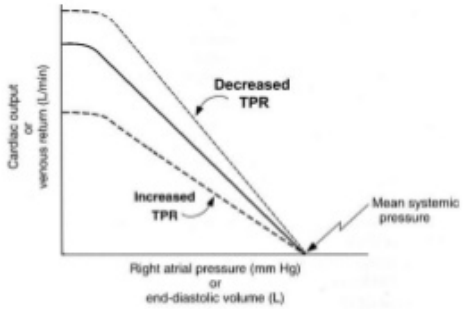
1. **RV pressure**
   a. **Intrathoracic pressure**
   b. **PA pressure**
2. **Mean systemic filling pressure (volume in circulation)**
3. **Resistance between peripheral tissues → RA**

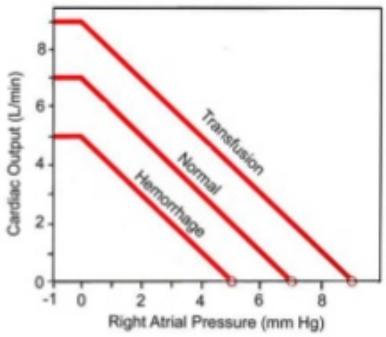
The Venous Return Curve

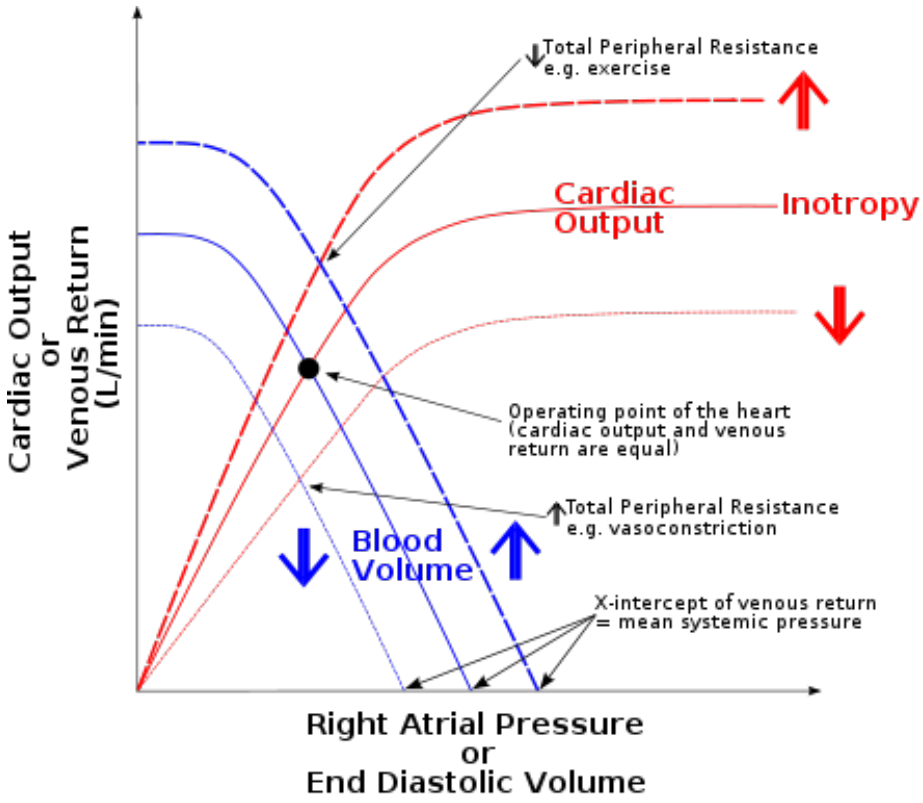


Changes in Vascular Function Curves

- **Total Peripheral Resistance**
  - ↑ TPR causes counter-clockwise shift in curve
  - ↓TPR causes clockwise shift in curve
- **Blood Volume or shift in flow to** organs and tissues
  - ↑ blood volume or ↑ restriction of flow to organs or tissues shifts curve upward
  - ↓ blood volume or ↑ flow to organs or tissues previously restricted shifts curve downward







- NORMAL VENOUS RETURN CURVES:
  - In the same way that cardiac output curves relate pumping of blood by the heart to the right atrial pressure, the venous return curve relates venous return also to right atrial pressure
  - Shows that when heart pumping fails and right atrial pressure rises, the backward force of rising right atrial pressure on the veins decreases venous return to the heart
  - IF ALL CIRCULATORY REFLEXES ARE PREVENTED FROM ACTING:
    - **Venous return decreases to zero when the right atrial pressure rises to about 7mmHg**
    - Such a small increase causes drastic decrease in venous return as the venous circulation is a distensible bag
    - At the same time that the right atrial pressure is rising and causing venous stasis, the pumping by the failing heart also approaches zero because of failing venous return
  - **Both the arterial and the venous pressures come to equilibrium when all flow in the systemic circulation ceases**
    - **MEAN SYSTEMIC FILLING PRESSURE**
- PLATEAU AT NEGATIVE ATRIAL PRESSURES:
  - Caused by collapse of the large veins
  - When right atrial pressure falls below zero, further increase in venous return ceases

- Caused by collapse of the veins entering the chest as right atrial pressure sucks the walls of the veins together

MEAN CIRCULATORY FILLING PRESSURE:

- When the heart is stopped from pumping, the flow of blood everywhere in the circulation ceases and pressure becomes equal after a minute or so
    - This equilibrated pressure level is called the **mean circulatory filling pressure**
- The greater the blood volume, the greater the mean circulatory filling pressure because extra blood volume stretches the walls of the vasculature
- Strong sympathetic stimulation constricts all the systemic blood vessels as well as the larger pulmonary vessels:
    - Capacity of the circulation decreases
    - Thus, the mean circulatory filling pressure is increased at any given blood volume
- Even slight changes in blood volume or slight changes in the capacity of the system caused by various levels of sympathetic activity can have large effects on the mean circulatory filling pressure

Mean circulatory filling pressure = systemic + pulmonary circulations
Mean systemic filling pressure = systemic circulation only

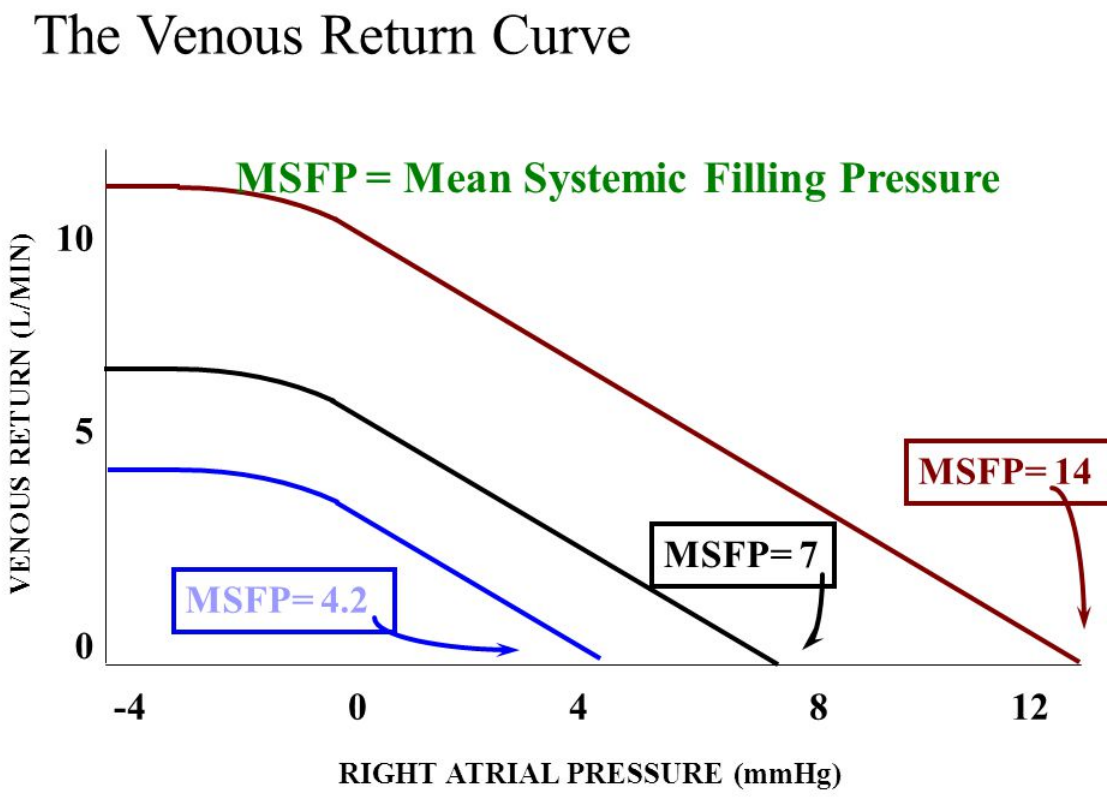
MEAN CIRCULATORY AND MEAN SYSTEMIC FILLING PRESSURES:

- Systemic doesn't include pulmonary circulation
- Mean systemic filling pressure (Psf) is slightly different from mean circulatory filling pressure:
    - Psf is the pressure measured everywhere in the systemic circulation after blood flow has been stopped by clamping the large blood vessels at the heart, so that pressures in the systemic circulation can be measured independently from those in the pulmonary circulation
    - Psf is the critical element in determining venous return
    - MSFP is almost always identical to mean circulatory filling pressure as pulmonary circulation has less than one eighth as much capacitance and one tenth the volume of the systemic circulation

EFFECTS ON VENOUS RETURN CURVE OF CHANGES IN PSF:

- The greater the Psf, which also means the greater the "tightness" with which the circulatory system is filled with blood, the more the curve shifts UPWARD and to the RIGHT (i.e. higher venous return at any given right atrial pressure)

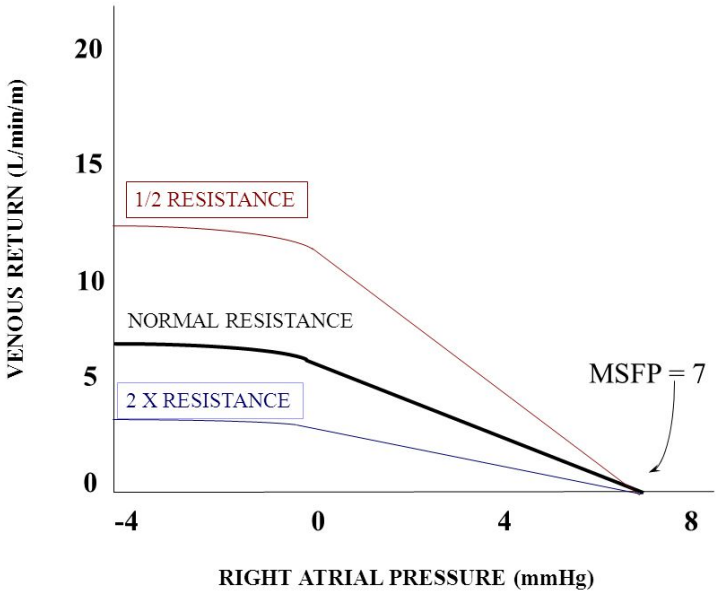
- Conversely, lower Psf results in lower venous return at any given right atrial pressure
    - IN OTHER WORDS, THE GREATER THE SYSTEM IS FILLED, THE EASIER IT IS FOR BLOOD TO FLOW TO THE HEART
- **When the right atrial pressure rises to equal the mean systemic filling pressure, there is no longer any pressure difference between the peripheral vessels, hence no flow**
    - Greater the pressure gradient, the greater the venous return



RESISTANCE TO VENOUS RETURN:

- Mean systemic filling pressure forces blood towards the heart
- There is also resistance to this flow
- Most resistance occurs in the veins, but also some in the arteriole and small arteries
- As resistance in the veins increases, blood begins to be dammed up in all parts of the systemic circulation, but the venous pressure rises very little as they are very distensible
    - Hence an upstream increase in venous pressure does little to overcome resistance

- **VR = MSFP - P(right atrium) / resistance to venous return**
    - Hence it can be seen that by decreasing resistance to one half, venous return is doubled and if resistance DOUBLED, venous return is HALVED



INCREASED BLOOD VOLUME

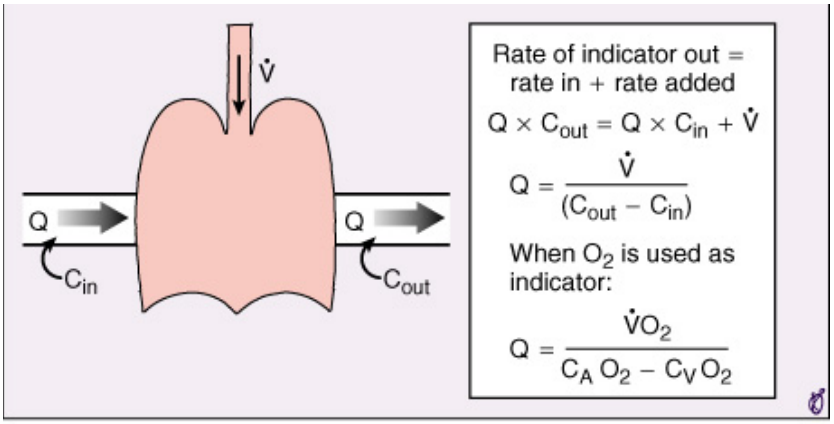
- Initiates compensatory efforts to decrease cardiac output
    - Increased cardiac output increases the capillary pressure so that fluid begins to transude out of the capillaries, returning volume to normal
    - Increased pressure in the veins causes the veins to distend by stress-relaxation, thus decreasing the pressure
    - Excess blood flow causes autoregulatory increase in the TPR

EFFECT OF SYMPATHETIC STIMULATION:

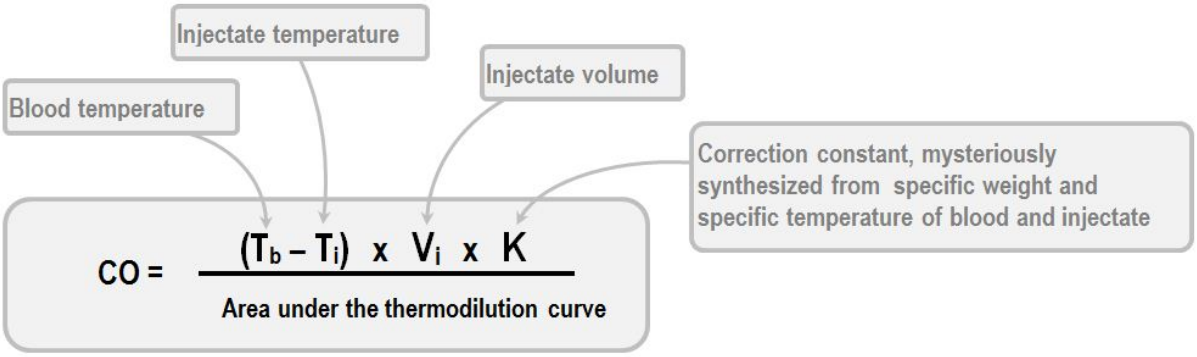
- Makes the heart a stronger pump
- Increases the mean systemic filling pressure, because of contraction of the peripheral vessels (mainly the veins)
    - Increases the pumping effectiveness of the heart by up to 100% without any appreciable change in right atrial pressure
- Increases resistance to venous return

METHODS FOR MEASURING CARDIAC OUTPUT:

- **OXYGEN FICK METHOD:**
  - 200ml/min of oxygen are being absorbed from the lungs into the pulmonary capillary blood each minute
  - Blood entering the right side of the heart has an oxygen concentration of 160mL per litre of blood
  - **Cardiac output = O2 absorbed per minute by the lungs / arteriovenous O2 difference (ml/L of blood)**
  - **Mixed venous blood is sampled via CVC in right ventricle, arterial blood from any artery**
  - **Rate of oxygen absorption is measured by the disappearance of oxygen from the respired air**



- **INDICATOR DILUTION METHOD:**
  - Cardiac output = milligrams of dye injected x60 / average concentration of dye in each mL x duration of the curve
  - A popular indicator dilution technique is thermodilution, in which the indicator used is cold saline.
  - Saline is injected into the right atrium
  - Temperature recorded in PA
  - Temperature change is inversely proportional to the amount of blood owing through the pulmonary artery; that is, to the extent that the cold saline is diluted by blood.
  - Advantages of thermodilution
    - (1) the saline is completely innocuous
    - (2) the cold is dissipated in the tissues so recirculation is not a problem, and it is easy to make repeated determinations.


Injectate temperature
Injectate volume
Blood temperature
Correction constant, mysteriously synthesized from specific weight and specific temperature of blood and injectate
CO = (Tb – Ti) x Vi x K
Area under the thermodilution curve