Department of Anesthesia Techniques
Title of the lecture:-Cardiovascular Effects of Anesthesia
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Cardiovascular Effects of Anesthesia

Aim of Anaesthetic Management

1. Maintain an optimal circulation.
2. Protect the cardiovascular system during the perioperative period.
3. Understand the cardiovascular actions of the anaesthetic agents.

Effects of inhalational and intravenous anesthetics

- Both can affect cardiovascular performance.
- Choice of anesthetic agent is entirely dependent upon:

1. patient's cardiovascular :

a. The presence of heart failure.

b. Hypovolemia.

2. Maintaining blood gases (oxygen saturation, pH and pCO 2) within physiologic levels during light anesthesia.

❖ **In general, anesthesia may provide:**

1. Analgesia (pain killer)
2. Amnesia (loss of memory)
3. Hypnosis
4. Muscle relaxation.

Effects of light & deep anesthesia on Heart

- There is very little evidence that cardiac function is depressed during light and moderate levels of anesthesia in the normal human subject.
- However, myocardial depression may occur during the deeper levels of anesthesia or when associated with other stresses such as anoxia, hypercarbia, hypotension and impaired adrenal function,
- The ability of a cardiac patient to withstand anesthesia is directly related to his cardiac reserve (ratio rate to capacity) and other disease conditions

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Cardiovascular Effects of Anesthesia

➡ Anesthetic agents can cause significant cardiac depression and hemodynamic instability .

They can affect:

1. Cardiac output
2. Heart rate
3. Systemic vascular resistance
4. Cardiac conduction system
5. Myocardial contractility
6. Coronary blood flow
7. Blood pressures

Coronary Circulation

The heart has its own nourishing circulatory system

1. Coronary arteries
2. Cardiac veins
   - Blood empties into the right atrium via the coronary sinus
   - Blood Flow Rate (Q) : is the volume flow rate of blood

   $Q = \Delta P/R$ (ml/min).

• ΔP: change in pressure on two ends of a vessel.

• R: Vessel resistance

• flow through a vessel will be **INVERSELY** proportional to its resistance

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1. **Cardiac output** – the volume of blood pumped from each ventricle per minute:

CO	=	SV	x	HR
cardiac output (ml/minute)	=	stroke volume (ml/beat)	X	heart rate (beats/min)

a. Average heart rate = 70 bpm
b. Average stroke volume = 70–80 ml/beat
c. Average cardiac output = 5,500 ml/minute

2- Stroke volume

Volume of blood pumped by each ventricle in one contraction.

- Stroke volume remains relatively constant
- Starling's law of the heart – the more that the cardiac muscle is stretched, the stronger the contraction

3- Heart Rate

Changing heart rate is the most common way to change cardiac output

❖ **Increased heart rate**

A. Sympathetic nervous system a. Activated in a Crisis b. Low blood pressure
B. Hormones :-
   i. Epinephrine
   ii. Thyroxin
C. Exercise
D. Decreased blood volume

❖ **Decreased heart rate**

A. Parasympathetic nervous system
B. High blood pressure or blood volume
C. Decreased venous return

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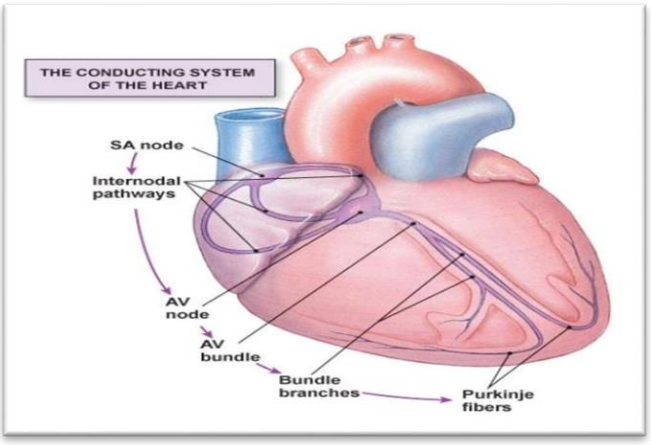
Intrinsic conduction system (nodal system)

Heart muscle cells contract, without nerve impulses, in a regular, continuous way by pacemaker cells:

1. Sinoatrial node (SA)
2. Atrioventricular node (AV)
3. Atrioventricular bundle
4. Bundle branches
5. Purkinje fibers



The electrical system gives rise to electrical changes (depolarization/ repolarization) that is transmitted through isotonic body fluids and is recordable The ECG (Electrocardiograph) A recording of electrical activity Can be mapped to the cardiac cycle.

The Heart's Cardiac Cycle

• Cardiac cycle is the sequence of events as blood enters the atria, leaves the ventricles and then starts over.

• Synchronizing this is the Intrinsic Electrical Conduction System.

• Atria contract simultaneously - Atria relax, then ventricles contract.

• Cardiac cycle events of one complete heart beat:

1. Mid-to-late diastole – blood flows into ventricles
2. Ventricular systole – blood pressure builds before ventricle contracts, pushing out blood
3. Early diastole – atria finish re-filling, ventricular pressure is low

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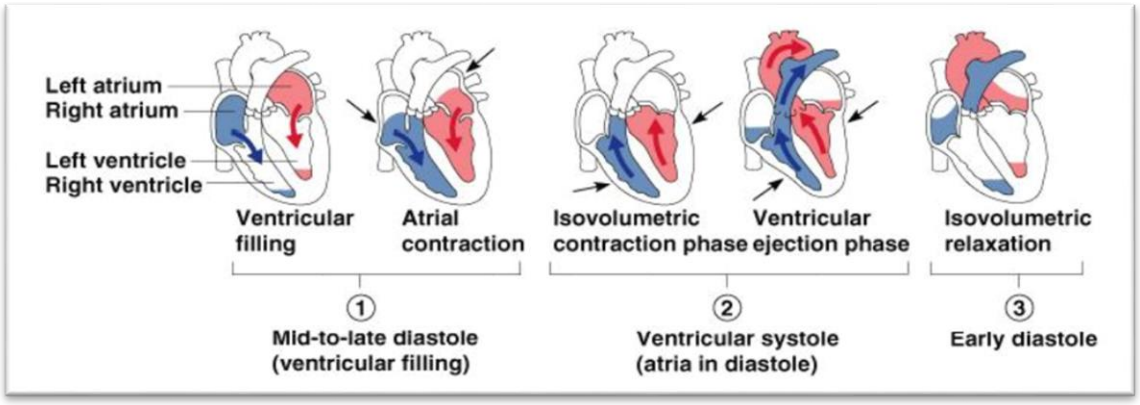
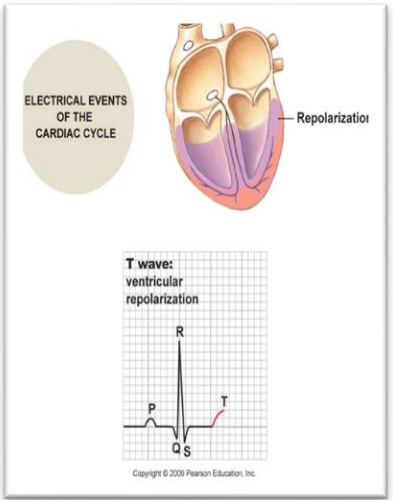


Blood pressure

- Arterial blood pressure is frequently assessed during anesthesia, either directly or indirectly
- It provides a rapid means to assess cardiac function
- Factors that determine blood pressure:

1. Heart rate and stroke volume (CO)
2. Vascular resistance
3. Arterial compliance
4. Blood volume

- All of the above factors can change dramatically during the course of anesthesia and surgery.
- Blood pressure can increase while CO decreases under the effects of several anesthetic drugs.



➡ Clinical notes

❖ Most anesthetics depress cardiovascular performance by:

1. Hypotension (low pressure)
2. Bradycardia (slow heart rate).
3. Decreased myocardial contractility

❖ Avoid these cardiovascular changes by:

1. Careful dosing
2. Balanced anesthesia
3. Adequate oxygen delivery to tissues

❖ Overcorrection of cardiac depression with tachycardia and hypertension increase myocardial O2 consumption, and is detrimental to the heart.

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Effects of Anesthesia on patient with pre-existing cardiac disease

- In the patient with pre-existing cardiac disease, these cardiovascular anesthetic effects become much more serious.

1. Vasodilatory effects of volatile agents
2. Hypotension
3. Negative intropy (weakened force of muscular contraction).

- These patients will not tolerate wide swings of hemodynamic variables,
- The stress of anesthesia and surgery frequently unmasks previously undiagnosed heart disease.
- Surgery itself provides many insults to the cardiovascular system, and these may be additive with the effects of anesthesia.

Anaesthetic Management for Patients with pre-existing cardiac disease

1. In patient with impaired cardiac performance anaesthetic should confer minimal or no cardiac depressive effects.
2. In patient with an acute illness which results in impaired vascular smooth muscle performance, then we must check the relation between blood volume, vascular tone and venous return to the heart.
3. In patient with impaired general vascular tone, and compromised venous return to the heart, then mild vasodilatory effects of anaesthetic agents can lead to an important potentially dangerous reduction in cardiac preload and hence cardiac output.

Surgical Stresses to the Cardiovascular System

Masked by Anesthesia

1. Loss of blood and other volume shifts.
2. Release of various substances into the circulation.
3. Hypothermia.
4. Sudden changes in cardiac preload and after load.
5. Myocardial ischemia (restriction in blood supply).
6. Effects of drugs or blood products given for surgical reasons.

The signs and symptoms of these surgical stresses to the cardiovascular system are often masked by anesthesia

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Nervous control of the cardiovascular system

The autonomic nervous system –significant regulator of CV function

• Sympathetic and parasympathetic outflow affect heart rate, inotropy (muscular contraction), and vascular tone to affect cardiac output, blood pressure, and distribution of blood flow

• Parasympathetic (vagus) effects

1. The vagus nerves inhibit the cardiac pacemaker, atrial myocardium and AV conduction tissue, acetylcholine serves a neurotransmitter.
2. Has negative chronotropic effects (decreased heart rate)
3. Has negative dromotropic effects (decreased conduction velocity)
4. Has negative inotropic effects (decreased contractility)

• Sympathetic effects

1. Innervation is throughout the heart, norepinephrine serves a neurotransmitter .
2. Has positive chronotropic effects (increased heart rate)
3. Has positive dromotropic effects (increased conduction velocity)
4. Has positive inotropic effects (increased contractility)

• Anesthetic agents

can and do interfere with this system at all levels:

1. Depress responsiveness of peripheral receptors
2. Depress responsiveness of central integration centers
3. Alter sympathetic and parasympathetic outflow