

Heart Rate Regulation — High-Yield Summary

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I. Definition

- Heart Rate (HR): number of heart beats per minute.
- Normal adult HR: 60–100 bpm.
- Cardiac Output (CO) = Stroke Volume (SV) × Heart Rate (HR). Ensures adequate tissue perfusion.

II. Determinants of HR

- 1. Intrinsic regulation: depends on SA node automaticity and pacemaker ionic currents (basal rhythm). Intrinsic rate = 100–110 bpm.
- 2. Extrinsic modulation: controlled by autonomic nervous system and hormones.

III. Cardiac Conduction System

- SA node is the pacemaker of the heart, located in the posterior wall of the right atrium near SVC.
- Intrinsic rhythm: 100–110 bpm but reduced by vagal tone to 60–70 bpm at rest.
- Normal sinus rhythm: 60–100 bpm (below 60 = sinus bradycardia, above 100 = sinus tachycardia).

IV. Autonomic Control

1. Parasympathetic (Vagal) Regulation

- Origin: dorsal vagal nucleus (DVN) and nucleus ambiguus (NA) in the medulla.
- Effects: ↓ SA nodal firing (negative chronotropy), ↓ AV nodal conduction (negative dromotropy).
- Tonic vagal activity at rest keeps HR below intrinsic rate.

2. Sympathetic Regulation

- Origin: rostral ventrolateral medulla (RVLM).
- Postganglionic fibers innervate SA node, AV node, myocardium, and coronary vessels.
- Effects: ↑ chronotropy, ↑ dromotropy, ↑ inotropy, ↑ vascular resistance → ↑ BP.

V. Reflex Control of HR

1. Baroreceptor Reflex

- Receptors in carotid sinus and aortic arch.
- \uparrow BP \rightarrow \uparrow baroreceptor firing \rightarrow \uparrow vagal tone \rightarrow \downarrow HR.
- \downarrow BP \rightarrow \downarrow firing \rightarrow \uparrow sympathetic tone \rightarrow \uparrow HR.
- Important for short-term BP control. Seen in orthostatic hypotension, carotid massage, Valsalva, Cushing reflex.

2. Bainbridge Reflex

- Stretch receptors in venoatrial junctions.
- \uparrow Venous return \rightarrow \uparrow atrial stretch \rightarrow \uparrow HR via sympathetic activation.
- Opposite to baroreceptor reflex.

3. Chemoreceptor Reflex

- Peripheral chemoreceptors: carotid and aortic bodies.
- Stimulated by \downarrow O \blacksquare , \uparrow CO \blacksquare , \uparrow H \blacksquare , \downarrow flow to carotid bodies.
- Central chemoreceptors: in medulla, stimulated by \uparrow CO \blacksquare and H \blacksquare in CSF.
- Effect: \uparrow ventilation and sympathetic tone \rightarrow \uparrow HR and vasoconstriction.

VI. Hormonal Regulation

1. Catecholamines (epinephrine, norepinephrine): \uparrow HR and contractility.
2. Thyroid hormones: \uparrow HR via SA node stimulation and \uparrow adrenergic sensitivity.
3. Atropine: blocks vagal tone \rightarrow \uparrow HR.
4. Histamine: vasodilator \rightarrow compensatory \uparrow HR.
5. Bile salts: inhibit SA node \rightarrow \downarrow HR.

VII. Integration — Reciprocal Control

- \uparrow Sympathetic \rightarrow \downarrow Vagal; \uparrow Vagal \rightarrow \downarrow Sympathetic.
- Allows fine, rapid control of HR depending on body needs.

High-Yield Takeaways

- **Normal HR:** 60–100 bpm

- **Intrinsic SA node rate:** 100–110 bpm
- **Resting HR lowered by:** Vagal tone
- **Main control centers:** Medulla oblongata (DVN, NA, RVLM)
- **Reflexes:** Baroreceptor ↓HR when BP↑; Bainbridge ↑HR when venous return↑
- **Chemoreceptors:** Respond to ↓O■, ↑CO■, ↑H■
- **Hormones ↑HR:** Epinephrine, Thyroid hormones, Atropine, Histamine
- **Hormones ↓HR:** Bile salts