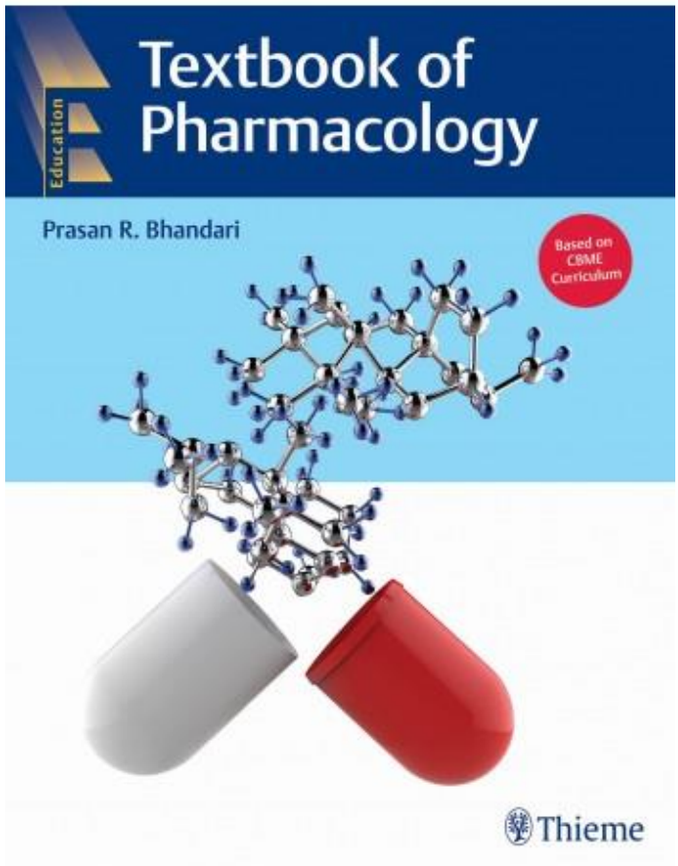
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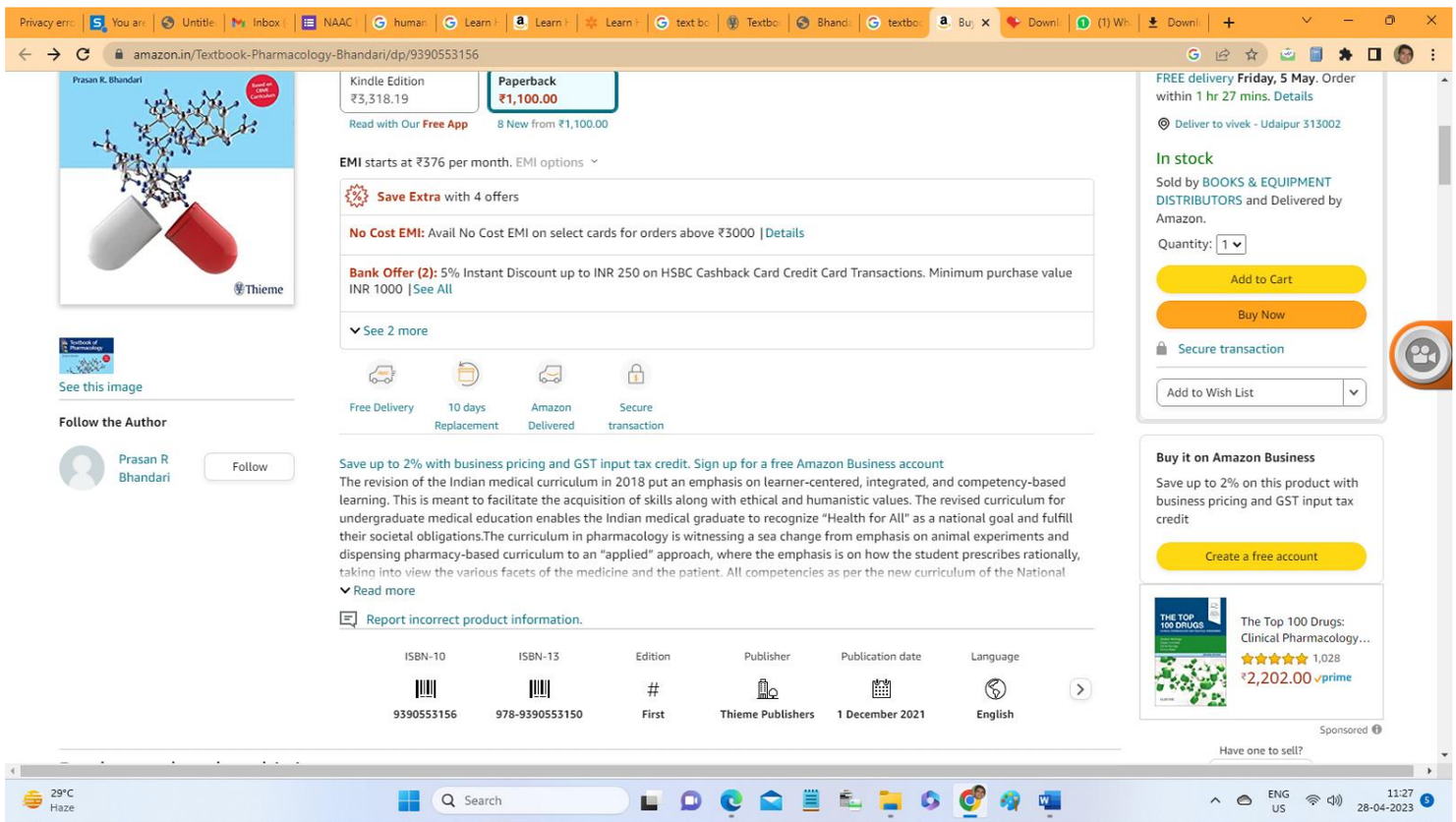
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The revision of the Indian medical curriculum in 2018 put an emphasis on learner-centered, integrated, and competency-based learning. This is meant to facilitate the acquisition of skills along with ethical and humanistic values. The revised curriculum for undergraduate medical education enables the Indian medical graduate to recognize "Health for All" as a national goal and fulfill their societal obligations.The curriculum in pharmacology is witnessing a sea change from emphasis on animal experiments and dispensing pharmacy-based curriculum to an "applied" approach, where the emphasis is on how the student prescribes rationally, taking into view the various facets of the medicine and the patient. All competencies as per the new curriculum of the National
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Part VIII: Drugs Acting on Blood and Blood-forming Organs

Part IX: Drugs Affecting Gastrointestinal System

Part X: Chemotherapy

Chapter 28 Antiarrhythmic Drugs

Vivek Jain

PH1.30: Describe the mechanisms of action, types, doses, side effects, indications, and contraindications of the antiarrhythmics.

Learning Objectives

- Cardiac arrhythmias.
- Cardiac action potential.
- Classification of arrhythmia:
  - Class I drugs.
  - Class ID (miscellaneous).
  - Class II agents.
  - Class III agents.
  - Class IV agents.

Cardiac Arrhythmia

A healthy human heart always beats with its own rhythm, which originates from the autorhythmic fibers of the right atrium called sinoatrial (SA) node. For the patient and physician, the heart rhythm serves as an indicator of well-being and disease. When the heart rhythm is disrupted due to ischemia, sympathetic stimulation, myocardial scarring, inherited variation in ion channel or other genes, and ingestion of drugs that affect heart conduction, it may result in cardiac arrhythmia. The clinical signs of cardiac arrhythmia are strong or fast heartbeat (palpitations), fluttering, dizziness, drowsiness, shortness of breath, tiredness, lack of energy, major discomfort when exercising, near-fainting, fainting, and chest pain.

Cardiac Action Potential

The resting membrane potential of the myocardium is approximately −90 mV, which results from an unequal distribution of ions (high Na^+ outside, high K^+ inside). There are five phases of the cardiac action potential (AP) (**Fig. 28.1a, b**).

Phase 0 (Rapid Depolarization)

In this phase, rapid inward movement of Na^+ occurs due to the opening of voltage-gated sodium channels (Nav). This leads to variation in resting membrane potential from −90 to +15 mV.

Phase 1 (Initial Rapid Repolarization)

This phase leads to inactivation of sodium channels and influx of Cl^-.

Phase 2 (Plateau Phase)

In this phase, there is slow but prolonged opening of voltage-gated calcium channels; it brings about contraction.

Phase 3 (Repolarization)

In this phase, closure of calcium channels is initiated and K^+ efflux starts through potassium channels. In addition, inactivated sodium channels return to resting phase.

Phase 4 (Diastole)

This phase leads to restoration of ionic concentrations by Na^+/K^+-activated ATPase (adenosine triphosphatase) and finally, restoration of resting potential.

Important Electrocardiographic Parameters

- P wave represents atrial depolarization.
- PR interval equals the delay of conduction through the atrioventricular (AV) node.
- QRS complex represents ventricular depolarization.
- T wave represents ventricular repolarization.
- QT interval equals duration of AP in the ventricles (**Fig. 28.1c**).

Classification of Arrhythmia

Automaticity is the property of cardiac cells to generate spontaneous APs. The SA node normally displays the highest intrinsic rate. All other pacemakers are referred to as subsidiary or latent pacemakers because they take over the function of initiating excitation of the heart only when the SA node is unable to generate impulses or when these impulses fail to propagate.

Abnormal automaticity includes both reduced automaticity, which causes bradycardia, and increased automaticity, which causes tachycardia. Arrhythmias caused by abnormal automaticity can result from diverse mechanisms.

Researchers have shown many schemes to classify the mechanisms of cardiac arrhythmias (**Fig. 28.2**). Conventionally, these have been divided into nonreentrant and reentrant activity. However, mechanism of arrhythmias can also be classified on the basis of its origin and occurrence at the cellular and tissue levels. Another classification, based on dynamics and focused on the trigger-tissue substrate interactions, divided arrhythmogenic mechanisms into reduced and excess along with unstable calcium cycling.

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