STRUCTURAL AND FUNCTIONAL PROPERTIES OF CARDIAC MUSCLE

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I INTRODUCTION / DEFINITION

- **Definition:** Cardiac muscle (myocardium) is a specialized, involuntary, striated muscle tissue found exclusively in the heart. It is designed for continuous, rhythmic contraction without fatigue, functioning as a physiological syncytium.
- **Unique Paradigm:** Unlike skeletal muscle (which requires neural recruitment) or smooth muscle (which lacks organized striation), cardiac muscle combines the high-force output of striation with the autonomous, coordinated properties of electrical conduction.

II STRUCTURAL PROPERTIES (THE ARCHITECTURE)

- **Design Principle:** Optimized for rapid electrical propagation and high mechanical durability.
- **Microscopic Anatomy:**
  - **Cardiomyocytes:** Short, branched, cylindrical cells (typically about 100 µm long). Unlike multinucleated skeletal fibers, they usually possess a **single central nucleus**.
  - **Sarcomeres:** The basic functional unit. Cardiac muscle is striated because of the highly organized arrangement of **Actin** (thin) and **Myosin** (thick) filaments.
- **Intercalated Discs (The Syncytium):** These specialized cell-to-cell junctions are the hallmark of myocardium, containing:
  - **Desmosomes:** Provide mechanical stability, “welding” cells together to prevent pulling apart during vigorous contraction.
  - **Gap Junctions:** Low-resistance electrical channels (formed by **Connexins**) that allow ions to flow freely between cells, ensuring the heart contracts as a single unit.
- **T-Tubule System & Sarcoplasmic Reticulum (SR):**
  - **T-Tubules:** Larger and more abundant than in skeletal muscle; they are located at the **Z-discs**.
  - **Diads:** In cardiac muscle, a single T-tubule associates with one terminal cisterna of SR (as opposed to triads in skeletal muscle).
- **Mitochondrial Density:** Mitochondria occupy ~**30-35%** of cell volume (compared with a few percent in skeletal muscle), reflecting a total reliance on **aerobic metabolism** and high ATP demand.

III FUNCTIONAL PROPERTIES (THE PHYSIOLOGY)

- **Excitability and Automaticity**Cardiomyocytes possess the ability to respond to electrical stimuli.
  - **Pacemaker cells** (SA Node) exhibit automaticity—the ability to spontaneously depolarize without external neural input.
- **Conductivity**The property of propagating an action potential. The speed varies (e.g., rapid in Purkinje fibers for synchronous contraction; slow in the AV node to allow for atrial kick).
- **All-or-None Law**Because of the **Gap Junctions**, a stimulus above the threshold causes the *entire* atrium or *entire* ventricle to contract as a single unit. There is no partial recruitment of fibers.
- **Long Refractory Period:**The cardiac action potential has a prolonged **Plateau Phase** (due to L-type Ca2+ channels).
  - **Clinical Significance:** This prevents **tetanization** (sustained contraction), ensuring the heart always has time to relax and fill with blood (diastole).

IV EXCITATION-CONTRACTION (E-C) COUPLING

- **Mechanism:** Cardiac muscle utilizes a specific mechanism called **Calcium-Induced Calcium Release (CICR)**:
- **Depolarization:** The action potential travels down the T-tubule.
- **Ca2+ Entry:** Small amounts of "trigger" Ca2+ enter via **L-type Calcium Channels** (Dihydropyridine receptors).
- **The Spark:** This trigger Ca2+ binds to **Ryanodine Receptors (RyR2)** on the Sarcoplasmic Reticulum.
- **Amplification:** Massive release of Ca2+ from the SR into the cytosol.
- **Contraction:** Ca2+ binds to **Troponin C**, moving tropomyosin and allowing Myosin-Actin cross-bridge formation.
- **Relaxation:** Ca2+ is pumped back into the SR by **SERCA** (regulated by **Phospholamban**) or expelled via the Na+/Ca2+ exchanger.

V BIOMECHANICAL LAWS

- **Frank-Starling Law:** Within physiological limits, the force of ventricular contraction is directly proportional to the initial length of the muscle fiber (Preload).
- **Length-Dependence of Calcium Sensitivity:** Unlike skeletal muscle, cardiac muscle increases its sensitivity to Ca2+ when stretched, which contributes to the Frank-Starling mechanism.
- **Force-Frequency Relationship (Bowditch Effect):** An increase in heart rate leads to an increase in contractile force due to the accumulation of residual intracellular Ca2+.

VI CLINICAL SIGNIFICANCE & PATHOLOGY

- **Hypertrophy:** In response to pressure overload (e.g., Aortic Stenosis), myocytes increase in width (concentric); in volume overload (e.g., AR), they increase in length (eccentric).
- **Myocardial Stun/Hibernation:** Functional impairment without cell death, often due to chronic ischemia or reperfusion injury, where E-C coupling is disrupted but metabolic viability remains.
- **Troponin Leak:** Structural disruption of the sarcolemma (as in MI or Myocarditis) releases Troponin I and T into the blood, the gold-standard biomarker for necrosis.