

The Myocardial Cell: New Concepts for the Clinical Cardiologist

THE GROWING BODY of research into the ultrastructure of the myocardial cell and the function of subcellular systems in the heart has reached proportions that make it both useful and pertinent to bring important new concepts in this area to the attention of the clinical cardiologist. Work on myocardial ultrastructure is no longer the province of the cellular anatomist and physiologist; increasingly, the clinical literature contains studies describing the cellular anatomy of both normal human myocardium and that of diseased hearts. Invariably, of course, such studies draw inferences about the mechanism of myocardial dysfunction on the basis of the ultrastructural changes observed.

Preservation of Tissue

Myocardium, like all contractile tissue, shortens in response to the stimulus of a sudden fall in environmental temperature. Maintenance of the resting length of the tissue prior to immersion in iced preservative, therefore, must be routine. When this is not done, the muscle shortens vigorously on making contact with the cold solution. As a consequence, all intracellular dimensions are distorted and certain ultrastructural features are even obliterated. If tissue is in contracture, no sarcomeric I bands will be visible in the section. The lumina of intracellular tubular structures will appear dilated, mitochondrial numbers will appear increased per unit area of the cell, and the cell membrane will be thrown up into folds, as will the nuclear envelope.

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If pieces of tissue placed in preservative are not sufficiently small to insure rapid and uniform penetration of the fixative, or if tissue is not preserved within minutes of the death of the experimental animal, sarcomeric Z bands will appear fuzzy and thickened and mitochondria will appear vacuolated. The nuclear contents will be largely obliterated, with peripheral clumping of chromatin just under the nuclear membrane. These features, if present, do not reflect pathology, then, but are artifacts and, clearly, cannot be interpreted to be the consequence of disease. Several workers have outlined the changes that occur in the cell simply as a consequence of postmortem changes.¹⁻²

Normal Anatomy of the Myofiber and the Function of Subcellular Systems in the Cell

The myofiber has, by virtue of the fact that it is a striated muscle cell, certain characteristic physiologic properties. It is capable of excitation and, indeed, depending on the type of cell, can spontaneously depolarize in a periodic fashion, i.e., it has pacemaking ability. The process of excitation and, if present, spontaneous depolarization depends on the properties of the cell membrane or sarcolemma and its derivatives in the cell, the transverse tubular system, and the intercalated disc.

Excitation has a definite mechanical consequence in the normal myofiber, i.e., it engenders a contractile event. In other words, excitation is coupled to contraction so that the cell shortens in response to stimulation. Contraction is achieved by the shortening of the contractile unit (or sarcomeres) that fill the cell in orderly lines called myofibrils and which make up the bulk of the volume of the myofiber. The contraction of individual sarcomeres occurs when the calcium concentration in the area of the myofilaments making

Myocardial Cell For The Clinical Cardiologist

Scott C. Dulebohn



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Table of Contents Myocardial Cell For The Clinical Cardiologist

1. Understanding the eBook Myocardial Cell For The Clinical Cardiologist
 - The Rise of Digital Reading Myocardial Cell For The Clinical Cardiologist
 - Advantages of eBooks Over Traditional Books
2. Identifying Myocardial Cell For The Clinical Cardiologist
 - Exploring Different Genres
 - Considering Fiction vs. Non-Fiction
 - Determining Your Reading Goals
3. Choosing the Right eBook Platform
 - Popular eBook Platforms
 - Features to Look for in an Myocardial Cell For The Clinical Cardiologist
 - User-Friendly Interface
4. Exploring eBook Recommendations from Myocardial Cell For The Clinical Cardiologist
 - Personalized Recommendations
 - Myocardial Cell For The Clinical Cardiologist User Reviews and Ratings
 - Myocardial Cell For The Clinical Cardiologist and Bestseller Lists
5. Accessing Myocardial Cell For The Clinical Cardiologist Free and Paid eBooks
 - Myocardial Cell For The Clinical Cardiologist Public Domain eBooks
 - Myocardial Cell For The Clinical Cardiologist eBook Subscription Services
 - Myocardial Cell For The Clinical Cardiologist Budget-Friendly Options

6. Navigating Myocardial Cell For The Clinical Cardiologist eBook Formats
 - ePub, PDF, MOBI, and More
 - Myocardial Cell For The Clinical Cardiologist Compatibility with Devices
 - Myocardial Cell For The Clinical Cardiologist Enhanced eBook Features
7. Enhancing Your Reading Experience
 - Adjustable Fonts and Text Sizes of Myocardial Cell For The Clinical Cardiologist
 - Highlighting and Note-Taking Myocardial Cell For The Clinical Cardiologist
 - Interactive Elements Myocardial Cell For The Clinical Cardiologist
8. Staying Engaged with Myocardial Cell For The Clinical Cardiologist
 - Joining Online Reading Communities
 - Participating in Virtual Book Clubs
 - Following Authors and Publishers Myocardial Cell For The Clinical Cardiologist
9. Balancing eBooks and Physical Books Myocardial Cell For The Clinical Cardiologist
 - Benefits of a Digital Library
 - Creating a Diverse Reading Collection Myocardial Cell For The Clinical Cardiologist
10. Overcoming Reading Challenges
 - Dealing with Digital Eye Strain
 - Minimizing Distractions
 - Managing Screen Time
11. Cultivating a Reading Routine Myocardial Cell For The Clinical Cardiologist
 - Setting Reading Goals Myocardial Cell For The Clinical Cardiologist
 - Carving Out Dedicated Reading Time
12. Sourcing Reliable Information of Myocardial Cell For The Clinical Cardiologist
 - Fact-Checking eBook Content of Myocardial Cell For The Clinical Cardiologist
 - Distinguishing Credible Sources
13. Promoting Lifelong Learning
 - Utilizing eBooks for Skill Development
 - Exploring Educational eBooks
14. Embracing eBook Trends
 - Integration of Multimedia Elements

- Interactive and Gamified eBooks

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