

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

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