Thus, despite the proscription of propranolol for established myocardial infarction, its use seems to be hopeful in acute coronary insufficiency and invites further investigation.

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References

The author replies:

To the Editor:

These favorable results from treatment of preinfarction angina with propranolol are encouraging. However, a much larger series is needed to establish the efficacy of the drug in this unpredictable situation. It would be interesting to know whether decreases in heart rate or blood pressure, which would diminish myocardial oxygen requirement, were obtained with the modest doses of propranolol used in six of the seven cases.

It is also very important in such therapeutic trials to document carefully radiographic changes in heart size, central venous pressure, body weight, and other objective measurements which might indicate development of cardiac failure. Increases in cardiac size should be avoided, since increased wall stress and myocardial oxygen requirement would result.

It is interesting that the multicenter trial quoted by Dr. Papazoglou did show an increased incidence of cardiac failure and hypotension in patients with established myocardial infarction receiving propranolol, compared to a group receiving placebo. This is not surprising, since clinically unsuspected left ventricular failure is present in a high percentage of patients with uncomplicated myocardial infarction (Hamosh P, Cohn JN: Left ventricular function in acute myocardial infarction. J Clin Invest 50: 523, 1971). Quite probably we must continue our trials of propranolol with great caution not only in patients with established infarction, but also in patients with preinfarction angina who show any evidence of cardiac decompensation.

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Fine Structure of the His Bundle

To the Editor:

I have read with interest the fine paper by Drs. James and Sherf on the fine structure of the His bundle (Circulation 44: 9, 1971). The authors are to be congratulated on their informative work. I would like to voice some misgivings on some of the terminology and concepts offered. The authors use the term Purkinje-like cells for some of the cells of the bundle. I do not think that this is wise. The term Purkinje cell has been used so indiscriminately in the literature that it has lost its meaning. It seems to me, if we are to be semantically pure, in man, we should apply it only to the large cells of the left bundle branch and the third part of the right bundle branch.

Then, I should like to raise the question of types of cells seen in the A-V bundle. Are the Purkinje-like cells, the slender transitional cells, and the broad transitional cells and P cells variations of the same kind of cell? The sampling in electron microscopy is so tenuous that the same type of cell may look somewhat different from micrograph to micrograph.

The third point I should like to make is the question of crossover connections in the A-V bundle. Can we tell with certainty these crossover connections without three-dimensional wax-plate reconstruction? I realize that this is a tedious process, but to the authors this is very important in their theoretical discussion.

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The author replies:

To the Editor:

In the world of research on the cardiac conduction system, an accolade from Maurice Lev is a jewel to be treasured. Dr. Sherf and I are grateful for his kind remarks about our study on the His bundle. The questions that he raises deserve a reply. There are three of these: about the term Purkinje cell, about the variety of cells in the His bundle and its branches, and about waxplate reconstruction of the crossovers.

The first two points can be considered together. In this paper and on previous occasions Dr. Sherf and I have defined what we mean by Purkinje cell, transitional cells, and other cell types in the heart. We look on these simply as working definitions and do not feel wedded to our terminology, if a better one is proposed. There is a spectrum of cell types included in our term of...
Fine Structure of the His Bundle
MAURICE LEV

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