Myocardial Infarction?

By Edgar Haber, M.D.

patients after digitalization to treat left ventricular failure following myocardial infarction. Others have found evidence of significant improvement from ouabain therapy in some indexes of left ventricular performance even though cardiac output was not affected. Nevertheless, it was felt that more study was needed of how digitalis acts on ventricle size and myocardial oxygen consumption.

Indication for Atrial Fibrillation

The clearest indication for digitalis after acute myocardial infarction is in the treatment of atrial fibrillation with a rapid ventricular rate. Electrical cardioversion may be preferred in the treatment of other supraventricular tachyarrhythmias. Animal experiments suggest that digitalis may induce arrhythmias at lower doses in cases of acute myocardial infarction, and that the toxic dose of digitalis glycosides is reduced as a result of rhythm disturbances. Clinical evidence on this is not clear.

The questions raised will certainly be studied intensively. Since 1912, when Herrick advocated use of digitalis in all patients with acute myocardial infaretion, practice and techniques have changed. The magnitude of effort being expended by biomedical investigators in the study of digitalis can be judged from the listing of more than 600 citations in Index Medicus in 1972 alone. More than 300 compounds in the digitalis classification are known to exist, and the sources and structure-activity relations have been known in depth for more than ten years. But no compound has as yet been indentified with an improved therapeutic-toxic ratio.

It is now considered that the major action of digitalis is the augmentation of the force of myocardial contraction, although this appeared to have been generally overlooked by 19th century investigators, who stressed slowing of the heart as its major effect. Another myth that investigation has dispelled in the past 25 years has been the belief that the inotropic action of digitalis was confined to the decompensated heart. It is now clear that the drug has this

action in the normal, as well as failing, heart muscle.

Yet the administration of cardiac glycosides results in no change, or a slight decline, in cardiac output in normal subjects. Cardiac output is determined not only by the cardiac contractile state, but also by ventricular filling pressure, peripheral arterial resistance, and heart rate. It now appears that digitalis augments the contractile state of the normal myocardium in intact man, but that reflex adjustments in the other determinants of cardiac output prevent a ready appreciation of this inotropic effect.

Is there such a thing as too much myocardial contractility? Experiments have shown that the inotropic action of digitalis increases progressively until toxic arrhythmias appear. The clinician's task is to determine the maximum dose consistent with an adequate margin of safety.

Practice Procedures

Debate Over Digitalis

The Questions:

- Is the drug useful for cardiogenic shock?
- Does it increase the size of a myocardial infarct?
- Should digitalis be used routinely when congestive heart failure complicates myocardial infarction?
- Should it be used for left ventricular failure following myocardial infarction?
- Does digitalis induce arrhythmias in acute myocardial infarction?
- Is there a possibility of too much myocardial contractility?

There are few, if any, clear-cut answers at present, but intensive investigation is continuing. In the meantime, the physician must exert extreme care to determine the maximum dose of digitalis that is consistent with an adequate margin of safety.

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