Inotropic Stimulation And Myocardial Energetics
Inotropic stimulation of the myocardium, as well as vasodilation and diuresis as essential principles in the treatment of congestive heart failure have recently met with considerable criticism and reevaluation. It is generally agreed that unloading of the heart, either through vasodilation and/or diuresis, improves the working conditions of the dilated, failing heart. It reduces myocardial oxygen consumption through reduction of chamber radius and, thereby, wall tension as the major determinants of myocardial oxygen consumption. Inotropic stimulation, quite in contrast, does not conserve oxygen. It rather consumes energy and that may be disadvantageous in situations of compromised oxygen supply and energy metabolism of the working myocardium. However, under conditions of sufficient oxygen supply and metabolic support inotropic stimulation may bring about increased pumping and subsequent improvement of myocardial failure. In recent years it could convincingly be demonstrated that vasodilation leads to symptomatic improvement of congestive heart failure, improvement of exercise tolerance, and it prolongs life - especially in the case of ACE-inhibitors and the combination of hydralazine with long-acting nitrates. Quite in contrast, equally beneficial effects could not be demonstrated for inotropic agents in congestive heart failure. Only for the cardiac glycosides has it been shown that beneficial effects can be achieved, especially if atrial fibrillation with absolute arrhythmia is present. The influence of the cardiac glycosides on the latter represents an effect which is independent of the inotropic action.

--This text refers to an out of print or unavailable edition of this title.
Inotropic Stimulation and Myocardial Energetics Myocardial Protection by Calcium Antagonists

Dmca