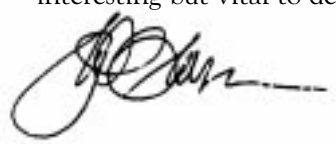
Up pops the devil

The optimal treatment of heart failure has changed dramatically since the primitive conditions of four decades ago when Mercuhydrin, digitalis leaf, and rotating tourniquets were the order of the day. Over the years, these agents have given way to better diuretics, synthetic forms of digitalis, and more recently, to adrenergic receptor blockers.

After a brief flirtation with the much maligned alpha-receptor blocker phentolamine in the 1970s, recent attention has focused on the beta-receptor blockers. Variations of these drugs have proliferated since introduction of propranolol, the prototype of the class. Furthermore, better understanding of the polymorphism of beta-adrenergic receptors and the details of selectivity of various blockers for these receptors raises the question of whether beta-adrenergic blockade by these agents is all equivalent. If not, which drug is best in treating heart failure, and why?

In this issue of the *Journal* (page 1081), Drs. Tang, Militello, and Francis review the COMET study, which compared two commonly used beta-adrenergic blocking agents, carvedilol and metaprolol tartrate, for their effectiveness in treating heart failure. Carvedilol clearly gave better survival results than metaprolol tartrate, but the question of why this was so remains difficult to answer. The authors discuss the various possible explanations and point out the need for more comparative studies.

Recognition of the role of beta-adrenergic receptor blockade in treating chronic heart failure is a great step forward in this life-threatening condition. But, as in so many complex situations, the devil is in the details. Which is the best drug, what is the best dose regimen, and what is the main determinant of success? Is it selectivity of beta blockade, drug level, or even beta blockade per se? These questions are not only interesting but vital to delivery of the best care.



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