Heart Failure’s Dark Secret
Does Anyone Really Care About Optimal Medical Therapy?

Today, most heart failure physicians focus on devices and transplantation; hospital-based management teams devoted only to achieving optimal medical therapy are scarce. The financial demands on heart failure specialists are enormous. A viable business plan can no longer be based on the misguided hope that payers will reimburse generously for prescriptions of digitalis and diuretics; in contrast, cardiac procedures generate meaningful revenues. A growing advocacy now encourages the use of ventricular assist devices in ambulatory patients on the basis of the dual misconceptions that the hazards are readily managed and that the clinical responses to medical therapy are poor. The biases in favor of performing procedures are so great that a National Institutes of Health–sponsored randomized trial comparing left ventricular assistance and optimal medical therapy in ambulatory patients was closed because of slow recruitment.

In its place, a nonrandomized study of 200 patients (ROADMAP [Risk Assessment and Comparative Effectiveness of Left Ventricular Assist Device and Medical Management]) was carried out and reported major advantages in patients who chose to receive a left ventricular assist device compared with those who chose to receive optimal medical management.1 The numerous limitations of the study are beyond the scope of this article, but it is intriguing that the authors failed to identify optimal medical therapy. Patients not electing a device were prescribed guideline-based treatments, but strangely, the authors do not tell us about the doses of renin-angiotensin inhibitors or β-blockers, the use (if any) of aldosterone antagonists or digoxin, or efforts to titrate diuretics to achieve euvoemia. We are not informed of the patients’ systolic blood pressures, but we are told that their cardiac filling pressures and natriuretic peptide levels were increased. However, larger doses of diuretics were not used, although the patients had preserved renal function. We know that the patients who received a left ventricular assist device were treated by heart failure specialists, but we do not know who directed medical therapy in the patients who did not undergo a procedure.

What is optimal medical therapy for heart failure in 2016? Some think that the answer lies in guidelines documents, but guidelines do not define to what degree a patient’s medical therapy can vary from the target doses used in clinical trials and still be considered adequate.2 How should we view the use of low doses of valsartan or carvedilol once daily if a physician fears (but has not demonstrated) that the patient might not tolerate more intense regimens? What asymptomatic level of systolic blood pressure or azotemia should lead a physician to stop uptitration of a diuretic or an inhibitor of the renin-angiotensin system? What are the valid reasons for not prescribing a β-blocker at the target doses3 shown to be effective in clinical trials? Why do physicians avoid the use of digoxin,4 although this inexpensive drug safely reduces the risk of hospitalizations? Perhaps the patients in the ROADMAP study received optimal medical therapy, but how would we know? We do know that a large number received implantable defibrillators and cardiac resynchronization devices.

The Table provides my personal view of pharmacological therapy for heart failure in 2016. I define inadequate as commonly prescribed but without supporting evidence...
from clinical trials, and I consider adequate to be identical to optimal. After all, how can the use of an angiotensin-converting enzyme inhibitor (even at target doses) be considered adequate if we know that patients live longer on an angiotensin receptor neprilysin inhibitor? Many readers might challenge specific suggestions, but doing so would miss my point entirely. If people want to test or propose a new treatment or device as superior to optimal medical therapy, we should expect them to document how assiduous the attempts were to achieve the targets described in the Table (or some alternative targets).

Thirty years ago, cardiologists were expected to be experts in the use of antianginal drugs. Patients who had exertional angina were treated with maximally tolerated combinations of nitrates, β-blockers, and calcium channel blockers. Interventional coronary revascularization procedures were offered primarily to those who remained disabled on optimal medical therapy. The battle between the use of drugs and procedures raged for years because we had no reliable data that either intervention did anything except relieve symptoms. However, in 2016, everyone with obstructive coronary artery disease is considered for an interventional procedure, whether he or she has angina or not. This enthusiasm has been prompted by outcomes data and potentially by revenue considerations. Today, there seems to be little interest in the molecular basis of disease progression. However, will our discoveries ever amount to anything? We now live in a world in which, when something is broken, we prefer to replace or circumvent it rather than to understand what went wrong and to direct therapies to the underlying pathophysiological processes. That is what has happened to our patients with coronary artery disease and serious cardiac arrhythmias. Is that what will happen to our patients with heart failure, although we can save their lives with drugs?

**DISCLOSURES**

Dr Packer has consulted for Admittance, Amgen, AstraZeneca, Bayer, BioControl, Boehringer Ingelheim, Boston Scientific, Celldyad, Cardiorentis, Daiichi Sankyo, GlaxoSmithKline, Novartis, NovoNordisk, Takeda, and ZS Pharma.

**AFFILIATION**

From Baylor Heart and Vascular Institute, Baylor University Medical Center, Dallas TX.

**FOOTNOTES**

*Circulation* is available at http://circ.ahajournals.org.
REFERENCES


Heart Failure's Dark Secret: Does Anyone Really Care About Optimal Medical Therapy?
Milton Packer

_Circulation_. 2016;134:629-631
doi: 10.1161/CIRCULATIONAHA.116.024498
_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2016 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/134/9/629

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation_ is online at:
http://circ.ahajournals.org/subscriptions/