



**BRIEF ANSWERS
TO SPECIFIC
CLINICAL
QUESTIONS**

Q: Should beta-blockers be discontinued when a patient is admitted to the hospital with acutely decompensated heart failure?

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A: Data on this topic are scant. However, most experts recommend discontinuing beta-blockers in patients with shock and lowering the dose for those with bradycardia or hypotension.¹ All others should continue to receive a beta-blocker at the same dose they had been receiving.

Furthermore, the evidence clearly supports resuming (or starting) a beta-blocker after the patient attains a euvolemic state before he or she leaves the hospital unless a clear contraindication exists.^{2–4}

■ **BETA-BLOCKERS: THE STANDARD FOR CHRONIC HEART FAILURE**

Beta-adrenergic antagonists—specifically, metoprolol succinate (extended release) and carvedilol, the only agents of this class approved for this indication—are the standard of care for chronic heart failure because they have been proven to decrease mortality rates.^{5–7} They should also be beneficial in acutely decompensated heart failure because they help break the neurohormonal vicious circle that occurs in both conditions (**FIGURE 1**).

Crude mortality rates of patients after a first hospitalization for acutely decompensated heart failure are 11.6% at 30 days and 33% at 1 year.⁸ Brief periods of treatment with medication during the initial presentation of acutely decompensated heart failure may significantly affect long-term survival.⁹

However, there are very few data on the efficacy and safety of continuing beta-blockers

in patients admitted to the hospital with acutely decompensated heart failure.

The question has not been directly tested: an appropriate clinical trial would be difficult to design, and it would be difficult to recruit patients in acute decompensation to such a study.¹⁰ Consequently, the balance of risks (negative inotropic effects) and benefits (inhibiting the adverse effects of the sympathetic nervous system) of beta-blocker therapy has not been established in acute decompensation.

The uncertainty has led to inconsistent management: some physicians discontinue these drugs until the patient is close to euvolemic status, some reduce dosage, and others continue the patient's outpatient regimen.

Patients with more severe symptoms of heart failure have more side effects at the

Pathophysiologic changes in acutely decompensated heart failure

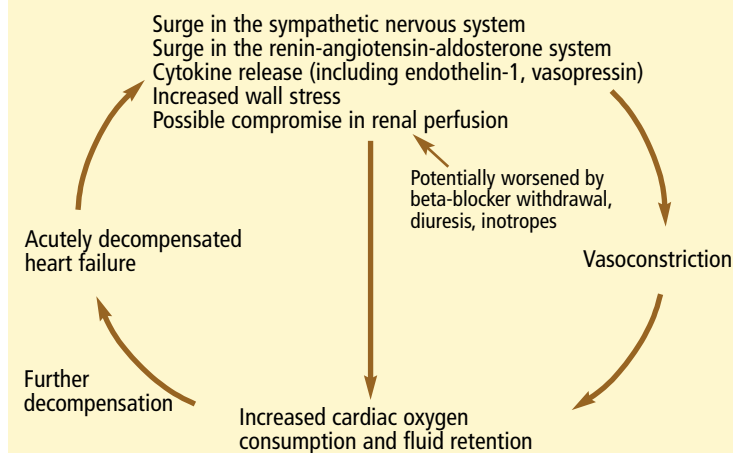


FIGURE 1

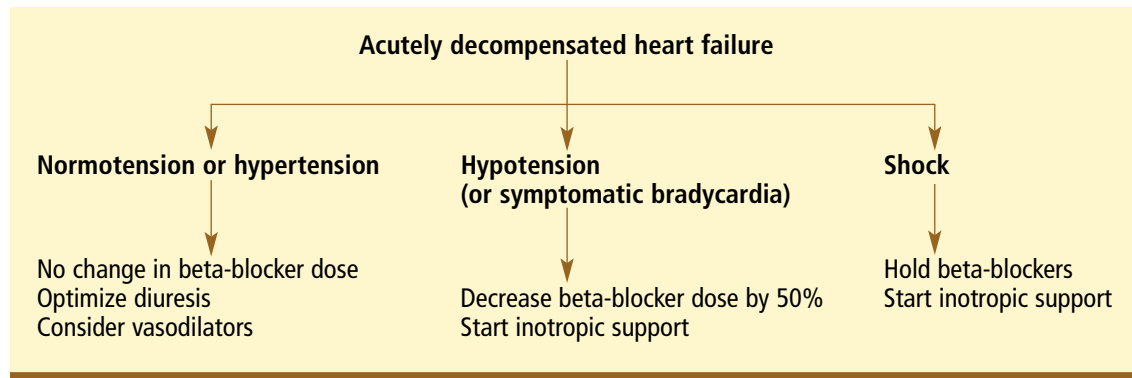


FIGURE 2. Beta-blocker dosage changes in acutely decompensated heart failure.

start of beta-blocker therapy than those with milder disease.¹¹ But one cannot assume that patients sick enough to be admitted to the hospital will not tolerate beta-blocker therapy: the hemodynamics of acutely decompensated heart failure differ from those of chronic severe heart failure. In fact, patients with severe symptoms (New York Heart Association [NYHA] class IV) on beta-blockers have a significantly lower mortality rate than comparable patients not taking beta-blockers.^{5,6} In addition, abruptly withdrawing beta-blockers in patients with heart failure results in clinical deterioration and should be avoided.^{12–14}

RECOMMENDATIONS

FIGURE 2 is an algorithm to help guide decisions regarding beta-blockers for patients with acutely decompensated heart failure, based on vital signs and perfusion status. It assumes that the patient has been on a stable dose of a beta-blocker for at least a few weeks before being hospitalized. Again, we emphasize that a patient who is not already taking a beta-blocker should be started on one once he or she is in a compensated, euvolemic state, before leaving the hospital.^{2–4}

A comparable and practical approach for the overall pharmacologic management of patients with acutely decompensated heart failure is based on the patient's congestion and perfusion status (**TABLE 1**).

In patients with adequate perfusion, every effort should be made to avoid reducing or stopping beta-blockers. Evidence indicates

that beta-blockers can be continued in patients with worsening heart failure without compromising safety.^{15,16}

Patients with shock, bradycardia, or hypotension. Most experts recommend discontinuing beta-blockers in patients with shock and reducing the dosage for those who have bradycardia or hypotension.¹ Treatment with diuretics should be optimized, and inotropes should be considered. Hemodynamic monitoring is recommended.

In patients with recurrent decompensation, briefly holding beta-blockers or significantly lowering the dose may be warranted (unless inappropriate therapy is to blame).

Patients with documented significant worsening of left ventricular ejection fraction may benefit from a reduction of the beta-blocker dose, although the evidence to support this practice is weak,¹⁴ and it could deprive patients of the full benefit of the drug, especially if they are doing well clinically.^{5–7}

Inotropic agents. The routine use of inotropic agents is not recommended. However, for patients receiving a beta-blocker who need an inotropic agent because of low cardiac output, hypotension, or cardiogenic shock, a phosphodiesterase inhibitor such as milrinone is preferable to a beta-adrenergic agonist such as dobutamine or dopamine because phosphodiesterase inhibitors work at a site distal to beta-adrenergic receptors.^{1,17–19}

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Start or resume a beta-blocker after a euvolemic state is attained, before hospital discharge



TABLE 1

Managing medications on the basis of congestion and perfusion status

PERFUSION AND VOLUME STATUS	BETA-BLOCKER DOSE	DIURETIC DOSE	INOTROPIC MEDICATION
"Wet and warm" (congested, well perfused)	Don't change	Increase	Usually not required
"Wet and cold" (congested, poorly perfused)	Hold or significantly reduce dose	Increase or don't change	Usually required
"Dry and warm" (euvolemic or hypovolemic, well perfused)	Don't change	Don't change	Usually not required
"Dry and cold" (euvolemic or hypovolemic, poorly perfused)	Hold or significantly reduce dose	Hold or significantly reduce dose	Usually required

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