Beta blockers have become a prescription drug staple for recovering heart attack patients. However, these blood pressure-reducing medications cannot be tolerated by many patients who are at higher risk for developing cardiovascular disease, including those with chronic obstructive pulmonary disease (COPD) and asthma, the elderly, and diabetics. As seen in the March 26 issue of *Thyroid*, researchers at New York Institute of Technology College of Osteopathic Medicine (NYITCOM) now pose a new treatment for patients with beta blocker intolerance: thyroid hormone therapy.

Formally known as "beta-adrenergic blocking agents," beta blockers came to prominence in the 1960s, when deaths from myocardial infarction (MI), the clinical term for heart attack, were very common. The drugs work by blocking the neurotransmitters norepinephrine and epinephrine, also known as adrenaline, from binding to receptors in the heart. Consequently, when the effects of the neurotransmitters are impeded, heart rate and blood pressure are lowered, allowing the heart to beat with less force and more easily deliver circulation to the body.

During MI, increased adrenaline raises pressure in the arteries and increases heart rate to compensate for the sudden loss of contractile tissue. Unfortunately, this places added stress on surviving myocardium, the heart's muscular tissue. Muscular damage to the heart sustained during infarction may cause the organ to be less effective in pumping blood to the rest of the body, a condition that can eventually lead to heart failure and death.

Since beta blockers are known to improve chance of survival, patients unable to tolerate beta blockers may then be at greater risk for heart failure than those able to withstand the drugs. However, the NYITCOM researchers suggest that the thyroid hormone triiodothyronine (T3), which controls many aspects of cardiovascular function and is also a powerful regulator of beta receptor function, may offer an alternative therapy.

"While beta blockers have been viewed as the gold standard in MI treatment for years, a significant population at risk for heart failure is unable to tolerate these drugs. If given beta blockers, these patients' conditions can, in fact, worsen—heart rate may fall too low and heart function could deteriorate," said Martin Gerdes, Ph.D., chair, Biomedical Sciences, NYITCOM, and senior investigator in the study. "Preclinical studies have shown thyroid hormone treatment to be a safe and effective method for managing cardiovascular disorders, and may offer a better option for these patients."

To investigate this option, Gerdes' team, which included experts from China's top cardiovascular center, FuWai Heart Hospital, compared the effectiveness of T3 and metoprolol, a commonly prescribed beta blocker, in female laboratory rats. Immediately following MI, the rats were provided either a low dose of T3 or the beta blocker in their drinking water for a total of eight weeks. At the end of that period, thyroid hormone proved to be as good, if not better, than metoprolol at improving heart function and reversing expression of detrimental genes linked to heart failure, providing all the benefits of the beta blocker plus some additional benefits unique to thyroid hormones, such as improved expression of genes related to better contraction and relaxation of the heart.

"Both treatments provide comparable results and similar long-term benefits, including improved function in the left ventricle, an area often damaged during heart attack, as well as reduced infarction size and improved vessel function," said Gerdes, who has studied the cardiovascular benefits and effects of thyroid hormone treatment for more than a decade. "Overall, these results suggest that T3 is capable of providing a safe alternative for beta
blocker intolerant patients following MI."

The researchers will continue studying the effectiveness of thyroid hormone after MI and encourage clinical researchers to consider examining low dose T3 treatment of MI patients who cannot tolerate beta blockers.


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