**THYROID HORMONE TREATMENT OF HEART FAILURE: IS THERE A THERAPEUTIC WINDOW?**

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In 1950, a study showed that Thyroid Hormone (TH) treatment significantly reduced cardiovascular mortality and rates of myocardial infarction in three patient groups. Rather than extend these findings, subsequent poorly designed larger clinical studies using toxic doses of TH analogs convinced the medical community that TH treatment of heart diseases was too risky, primarily due to increased risk of inducing arrhythmias. Due to a steady stream of positive new information, however, this issue has not gone away. Over the years, we have learned many things about low thyroid function and heart diseases. In many studies, low TH function has been linked to increased mortality in patients with various heart diseases. Many short term clinical studies also show improvement in cardiac patients treated with THs. A key animal study clearly demonstrated that hypothyroidism alone can eventually cause heart failure with maladaptive myocyte remodeling and impaired coronary blood flow. Other rat studies showed that low TH function promotes arrhythmias and TH treatment of myocardial infarction protects from arrhythmia induction. Cumulatively, animal studies suggest that all types of heart disease lead to low cardiac tissue T3 levels. One has to ask the question, why is there so much opposition to a drug that improves systolic/diastolic function, improves coronary blood flow, inhibits myocardial fibrosis, reverses fetal gene expression, and reduces arrhythmias? There are good reasons to be apprehensive. But, is fear of overtreatment unreasonable? Is there a safe, therapeutic window for TH treatment of heart diseases, including heart failure? Over the past few years, animal research in our lab has focused on answering the critical questions that have blocked progress to translation in this field. Results provide considerable optimism that TH treatment of heart disease can be done safely and with remarkable benefits.