A “new” treatment for heart failure right under our nose for over 60 years?

Anthony Martin Gerdes
New York Institute of Technology, USA

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. 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 (yes, really)? 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. These results will be discussed.

Biography

Anthony Martin Gerdes has done PhD in Anatomy (1978), from University of Texas Medical Branch at Galveston. He was the Professor/Chair of Anatomy, University of South Dakota. Also he is the founding Scientist for Sanford Research-University of South Dakota. His Current position is Professor/Chair Biomedical Sciences, NYIT College of Osteopathic Medicine, USA 2011-present. Publications: ~120 peer reviewed journal articles. 2013 Distinguished Alumnus, Graduate School of Biomedical Sciences, UTMB at Galveston Anthony Martin Gerdes developed a precise method to determine cardiac myocyte shape. He then provided a comprehensive understanding of how cardiac myocytes remodel during growth, maturation, aging, cardiac hypertrophy, and heart failure (HF) from many etiologies. After demonstrating the low thyroid hormone function alone can cause heart failure, he showed remarkable beneficial changes in myocyte shape and vascular remodeling, reduced fibrosis, and improved LF function after thyroid hormone treatment of various models of HF (including ischemia, diabetes and hypertension).

agerdes@nyit.edu

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