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Wrong about β Blockers! Wrong about positive inotropes! Wrong about thyroid hormone treatment of heart failure?

A. Martin Gerdes
New York Institute of Technology-College of Osteopathic Medicine, USA

Medical opinion currently opposes thyroid hormone (TH) treatment of heart failure (HF), largely due to fear that overdosing may lead to increased arrhythmias and death. Yet, overwhelming evidence suggests that TH treatment of HF offers great promise. Importantly, recent clinical studies have shown increased HF mortality as TH function declines. A rat study showed that hypothyroidism eventually evolves into dilated HF with chamber dilatation from myocyte lengthening and impaired coronary blood flow. Cardiac diseases typically lead to re-expression of fetal genes, which also occurs in hypothyroidism. New studies show that heart diseases may trigger cardiac tissue hypothyroidism by upregulation of the D3 deiodinase, which converts T4 to inactive rT3. We showed that dilated HF results in excessive myocyte lengthening from series addition of new sarcomeres in the absence of a compensatory change in cell diameter. Indeed, this cellular change may largely account for the hallmark change in HF, increased chamber diameter/wall thickness ratio. THs not only induce a physiological, proportional growth of myocyte length and width, they promote a beneficial change in myocyte shape in the background of diseases leading to dilated HF. A new study shows that both hypo- and hyperthyroidism promotes arrhythmias. A major impediment to clinical trials is lack of a protocol that restores cardiac tissue T3 levels safely and improves cardiac function without inducing hyperthyroidism. We will present new data documenting a safe treatment/monitoring protocol in various animal models of HF that should easily translate to patients.

Biography

A. Martin Gerdes completed his Ph.D. at the University of Texas Medical Branch at Galveston in 1978 and was recently recognized as the 2013 Distinguished Alumnus of the Graduate School of Biomedical Sciences. Throughout his career, work has focused on understanding the cellular and molecular mechanisms of maladaptive ventricular remodeling in heart failure and potential treatments. He has published over 100 peer reviewed journal articles and has been the PI on ~$30M in NIH funding during his career. He has identified key cellular mechanisms involved in dilated heart failure, including a maladaptive change in myocyte shape.

agerdes@nyit.edu