

## 6<sup>th</sup> International Conference on

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### What you should know about cardiac remodeling

The "Age of Cardiac Remodeling" began in the mid-1990s with the realization that drugs leading to improved ventricular remodeling were doing something remarkable in cardiac patients. This created an experimental need for high quality assessment of changes in cardiac tissue composition, including myocyte shape, myocardial fibrosis/collagen, and vascular remodeling. Many working in the field today have little or no training related to recognition of fixation artifacts or common errors associated with quantitative morphology. Unfortunately, such skills had become somewhat of a lost art during the ages of cardiac physiology in the mid-20th century and molecular biology, gaining prominence by the mid-1970s. Consequently, cardiac remodeling studies today are often seriously flawed to the point where data are not reproducible and subsequent researchers may be chasing the molecular basis of a non-existent or erroneous phenotype. The current unacceptably high incidence of irreproducible data is a serious waste of time and resources as noted recently in comments by the NIH Director. The goal of this talk is to share some lessons I have learned during nearly 40 years of assessing morphological changes in the heart. It is possible for any lab to routinely publish highly reproducible morphologic data that stand the test of time and contribute to our fundamental knowledge of cardiac remodeling and the molecular mechanisms that drive it.

#### 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. 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-till present. His publication includes 120 peer reviewed journal articles. His research since 2013 Graduate School of Biomedical Sciences, UTMB he 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).

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