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Cardiac arrhythmia and heart failure: From bench to bedside

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Cardiac arrhythmia is an abnormal rate and/or rhythm of a heart due to its abnormal electrical impulse origination and/or propagation. Various etiologies can cause arrhythmias. Heart failure (HF) is a clinical syndrome due to an impaired heart that can not pump sufficient blood to meet the systemic metabolic needs. The common causes of HF include myocardial infarction, hypertension, valvular heart disease, and cardiomyopathy.[1] The morbidity and mortality of HF and cardiac arrhythmia are high in the aging population aged ≥ 65 years. This geriatric population in the world is predicted to be 973 million by the year 2030.[2] In USA, elderly patients account for more than 85% of all cardiovascular disease deaths, 65% of cardiovascular disease hospitalizations, 62% of myocardial infarction hospitalizations, and 77% of HF hospitalizations.[3–5] Therefore, the *Journal of Geriatric Cardiology* organized this special issue, Cardiac Arrhythmia and Heart Failure: from Bench to Bedside. This issue contains several reviews and research articles contributed by a group of scientists from different institutions. These articles include the authors own and others' research from bench at the molecular and cellular levels to bedside of the treatment and management of cardiac arrhythmia and heart failure.

Mitochondria are organelles found in almost all cells in our body and generate 90% or more of the energy for cell use.[6] Aging can cause cellular and sub-cellular structural alteration and dysfunction. In early 1970s, Dr. Harman proposed the mitochondrial theory of aging.[7] As myocardium heavily depends on oxidative metabolism and consumes a lot of energy, mitochondrial dysfunction can cause heart failure, or vice versa. In this issue, Chaudhary *et al.*[8] discussed cellular aging of the heart with special emphasis on mitochondrial dysfunction. Cardiomyopathy is generally clarified into three major categories (hypertrophic, dilated and restrictive). Current treatment of restrictive cardiomyopathy (RCM) is hardly effective and its prognosis is very poor. Some RCM patients die within two years after diagnosis or in their early childhood. Recently, Dr. Huang and his colleagues had investigated the potential mechanisms underlying RCM development and progression in a transgenic RCM animal model.[9,10] In this specific issue, they reviewed the progress in RCM research from basic to clinical studies, especially from RCM transgenic animals.[11] As the conventional therapies for cardiac arrhythmias still face great challenges and some shortfalls, biological alternatives to supplement or to replace the current anti-arrhythmic treatments have been investigated in recent years.[12-16] Research highlights of cell and gene therapy for repairing cardiac conduction damage or defect had been discussed in this issue.[17]

Atrial fibrillation (AF) is one of the most common arrhythmias. AF and congestive heart failure (CHF) are frequent co-morbidity in the geriatric population. The probability that AF precedes CHF or vice versa is similar.[18] They can deteriorate each other and make the therapy more difficult. AF and CHF animal models, especially in large animals, can help to understand the pathophysiological mechanisms and provide critical information on the safety and efficacy of drugs and/or medical devices prior to human clinical trials. One article as translational research written by Dr. Urban and his colleagues introduced the methods of induction, management, and characterization of several AF and CHF models in dogs.[19] These animal models can be highly valuable for basic and preclinical research. A continuum of atrial anatomical remodeling occurs during AF. Rolfes *et al.*[20] used their library of perfusion-fixed human hearts to investigate AF-induced remodeling. Human AF specimens were compared to non-AF human

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controls. They found that compared to controls, AF hearts typically had larger atrial volumes, more variation in volume, and other structural alterations. If a reader is interested to know more about the structure and function of the human heart, here is an excellent website (www.vhlab.umn.edu) to visit.

Population aging of the world is a global issue. Clinically, the morbidity and mortality of cardiac arrhythmia and HF are much high in the geriatric population. The medical costs are on the exponential increase as this population grows fast. Proper and prompt therapy and management of elderly patients become increasingly important. In this specific issue, two reviews discussed some therapeutical issues about the use of medical devices and antiarrhythmic drugs in geriatric patients. Current guidelines for implantable cardioverter-defibrillator (ICD) therapy in HF patients were based on device multi-trials in which very few geriatric patients were recruited. Drs. Revenco, Morgan, and Tsao thus put together an article for this specific issue to address the determining indications for ICD implantation in elderly patients.[21] The article also discussed the controversies of ICD implantation and therapy in geriatric patients, as well as the withdrawal of ICD therapy near the end of life. In addition, Dr. Lee and his colleagues raised the important issue to distinguish the pathological alterations of cardiac electrophysiology from the normal and physiological progression in aging populations.[22] They discussed the changes of the pharmacokinetics and pharmacodynamics of antiarrhythmic drugs in elderly patients due to the changes of their absorption, distribution, metabolism, and elimination. Therefore, the use of antiarrhythmic drugs in geriatric patients needs to be individualized according to each patient's physiology and disease stage. Closely monitoring liver and kidney function is necessary for prevention of serious side effects in elderly patients.

Finally, the journal greatly appreciates all of the contributors for their hard work to make this specific issue unique and tight to geriatric cardiovascular research.

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