**AGING AND HEART FAILURE 2016. UPDATE**

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Concurrent worldwide increases in the aging population and prevalence of heart failure (HF) are accompanied by a parallel increase in the elderly (age ≥ 65 years) with two leading causes of HF, hypertension (HTN) and myocardial infarction (MI). Aging results in progressive cardiovascular remodeling with an “aging phenotype” that negatively impacts disease expression and response to therapy. Aging-related changes contribute to adverse cardiac remodeling with HF and preserved ejection fraction (HFpEF) and result in increased risk for HTN which exacerbates HFpEF, and MI which leads to HF with reduced EF (HFrEF).The remodeling involves changes in structure, physiological and pathophysiological pathways and responses to stress/injury. Optimal healing is critical for a favorable outcome and defective post-MI healing with aging contributes to adverse remodeling with poor outcome. The cardiac extracellular matrix (ECM) is critical for maintaining cardiac shape/function and progression of HF due to MI and HTN involves adverse ECM remodeling with disruption of the ECM network and dysregulation of ECM homeostasis and metabolism resulting in shape deformation and dysfunction. While better post-MI therapies improve survival, therapy for optimizing post-MI healing is needed to further improve outcome. While early reperfusion reduces MI size, delayed reperfusion results in reperfusion damage, impaired healing and adverse remodeling and progression to HF in the elderly. Therapy for the young may not be optimal for the old. Several recommended post-MI therapies can impact early and late phases of healing in positive or negative directions. Preclinical studies suggest that pathways during early and late phases can be targeted for optimizing post-infarct healing and the march to HF. Progressive remodeling and progression to HFpEF or HFrEF are persistent problems in older patients and have important therapeutic implications. Studies suggest that in the elderly, novel pathways can be targeted for optimizing therapy in HFrEF post-MI and HFpEF post-HTN.