JSPCCS AHA Joint session

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[II-AHAJS-4]Mitochondrial transplantation for Cardioprotection

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The importance of the mitochondrion in the maintenance and preservation of cellular homeostasis and function is well established and there is a sufficient body of evidence to show that mitochondrial injury or loss of function is deleterious. In our research, we have focused on the myocardium, a highly aerobic organ in which mitochondria comprise 30% of cellular volume. The mitochondria supply the energy requirements of the myocardium, derived through oxidative phosphorylation and is dependent upon oxygen delivery through the coronary circulation. Our studies and those of others have demonstrated that attenuation or cessation of oxygen delivery to the myocardium (ischemia) damages mitochondrial structure, volume, calcium accumulation, complex activity, oxygen consumption, high energy synthesis, and the mitochondrial-mediated intrinsic apoptotic pathway, and causes mtDNA deletion. These events occur during ischemia and extend into reperfusion to severely compromise post-ischemic functional recovery and cellular viability.
Instead of targeting a single step or one mediator of a complex, or the interconnected reactions of mitochondrial injury, we have pioneered a novel therapy based on mitochondrial transplantation. This approach uses replacement of native mitochondria with viable, respiration-competent mitochondria isolated from non-ischemic autologous tissue to overcome the many deleterious effects of ischemia/reperfusion injury on native mitochondria.
In this presentation we will provide evidence for the safety and efficacy of mitochondrial transplantation and show methods for the rescue of myocardial cellular viability and function.