**WHAT HAPPENS TO THE BRAIN AFTER MYOCARDIAL INFARCTION?**

**J.L. Mehta**, X. Wang

University of Arkansas for Medical Sciences and VA Medical Center, Little Rock, AR, USA

It is assumed, but not proven, that acute myocardial infarction affects function of remote organs- such as kidneys and brain. We examined brain morphology in wild type (WT) mice subjected to left coronary artery (LCA) ligation. Coronary ligation resulted in extensive myocardial infarction (MI) and diminished cardiac contractile function. Brain morphology showed a large number of neuronal cells undergoing apoptosis (TUNEL staining and caspase-3 expression) and necrosis (cresyl violet staining) mainly in the temporal area. These changes appeared at 1 week post-MI and persisted for at least 4 weeks. Brain and cardiac tissues revealed intense inflammation (elevated IL-6 and TNF-α, and LOX-1 [a receptor for ox-LDL] expression). Plasma levels of IL-6 and TNF-α were increased over the 4 weeks, maximally at 1 week. To determine the role of LOX-1 in brain inflammation and neuronal injury following LCA occlusion, LOX-1 knockout mice were subjected to total LCA ligation; these mice showed much less increase in systemic, cardiac and brain pro-inflammatory cytokines, and much less neuronal injury than the WT mice (all P<0.05). Cardiac functional deterioration was much less in the LOX-1 KO mice than in the WT mice (P<0.05). This study shows that MI results in significant neuronal injury (apoptosis and necrosis) lasting for at least 4 weeks (equivalent to 3-5 years in humans). This study also suggests that systemic inflammation mediated by LOX-1 is a key modulator among multiple mechanisms underlying brain following MI.