Ninety-eight percent of events occurred on Days 0–3. All 3 pts with later events also had events on Day 0. In a multivariate model, only EF was a significant predictor of events, p = 0.04. Of pts with EF < 40%, 16% had events vs 8% with EF ≥ 40%, p < 0.005. The 3 pts with events after Day 3 had EF 40–43%. Conclusion: It is safe to discharge AMI pts after hospital Day 3, if they had successful primary PCI and have not experienced complications during Days 0–3. While EF predicts overall events, it is an imperfect tool for discharge planning.


Purpose: To study the indications, complications and course of patients admitted to a community hospital and needed temporary pacemaker. Method: We studied 181 consecutive patients who needed a temporary pacemaker between 04/2001 and 04/2003. Results: A total of 181 patients were studied. The average age was 76 ± 12.3, 100 males, 81 females. The commonest site of insertion was right subclavian (45%) followed by right femoral (27%), and internal jugular vein (18%), left femoral (3%) and left subclavian (1%). The commonest indication for temporary pacemaker was sick sinus syndrome and symptomatic bradycardia (56%) as a bridge for permanent pacemaker. 47 (23.9%) needing temporary pacemaker had an acute coronary syndrome as their cause, 14 (28%) of which died of cardiac complications, 9 had persistent conduction defect needing permanent pacemaker. 25 of these patients had infarction involving the inferior wall. Non essential medications causing symptomatic bradycardia needing temporary pacemaker was found in 29 (16%) of patients. 20 (11%) patients had hyperkalemia (medication induced or renal failure) and 3 patients had digoxin toxicity as the cause of conduction disturbance. 6 patients needed temporary pacemaker as their permanent pacemaker generator had reached end of life.

Conclusion: Temporary support of conduction system prior to placement of permanent pacemaker was the commonest cause of temporary pacemaker in our study. Myocardial infarction leading to high risk pacemaker patient with a high mortality.

8 EFFECTS OF FRUCTOSE-1,6-BISPHOSPHATE AND OTHER COMPOUNDS ON HYPERPERMEABILITY INDUCED BY VASCULAR ENDOTHELIAL GROWTH FACTOR (VEGF) IN TERTIARY HUMAN ENDOTHELIAL CELLS. J. E. Wheeler,1 Departments of Biochemistry and Molecular Biology and Surgery, University of Louisville School of Medicine, Louisville, KY.

Purpose: Limitation of heart transplantation is the small time period during which a heart remains viable while it is being transported on ice. Some studies have shown that fructose-1,6-bisphosphate (FBP), a glycolytic intermediate, can help preserve heart tissue while it is in this ischemic state. The purpose of this project was to better understand the mechanism of this enhanced preservation and to explore other treatments that may extend heart viability during hypothermia. FBP’s protective effect may come from its calcium-chelating property, and its ability to phosphorylate and thus is readily used to produce ATP. Methods: Using myocytes prepared from rat heart, we measured cytosolic Ca2+ fluorometrically using fura-2 AM. The leakage of the plasma membrane, an indication of necrosis, was examined by measuring extracellular lactate dehydrogenase (LDH). To assess mitochondrial membrane leakage, we measured the penetration of nitroblue tetrazolium and its reduction via succinate dehydrogenase. Using an experimental model in which cell morphology was observed as a measure of cell survival during hypothermic incubation, three possible preservatives were tested. Results: For freshly-prepared myocytes, Ca2+ was 33 ± 7% (n = 5) lower for FBP-treated than for controls, after 24 h of hypothermic incubation it was 70 ± 11% (n = 4) lower in FBP-treated samples. LDH release after 24 h was 25% lower for FBP-treated cells compared with untreated cells; after 24 h of hypothermic incubation it was 70% lower in FBP-treated samples. LDH release after 24 h was 25% lower for FBP-treated cells treated than for control cells; after 24 h of hypothermic incubation it was 70% lower in FBP-treated samples.

Conclusion: These results indicate that FBP exerts at least some of its effects via lowering cytosolic calcium, and that it helps preserve plasma membrane but not mitochondrial membrane integrity. The survival studies indicate that FBP, but not NBTI or adenine, may be a useful addition to FBP in enhancing heart preservation. Supported by NIH HL-61486 and T32HL071486.

9 MECHANISMS OF ENDOTHELIAL BARRIER ENHANCEMENT INDUCED BY ADENOSINE TRIPHOSPHATE, 1,6-bisphosphoglycerate, and Myosin Light Chain Phosphatase Activation. T. Kogouli, T. Mirzapoiazova, D. Akhvedy, P. Usatyuk, V. Natourajan, A. Verin, Johns Hopkins School of Medicine, Division of Pulmonary and Critical Care Medicine, Baltimore, MD.

Purpose: Endothelial barrier dysfunction is often the underlying cause of vascular leakage and edema. It is important therefore to find ways to preserve barrier properties. Extracellular adenine triphosphate (ATP) has been known to protect endothelial barrier. In this study we measured the mechanisms of endothelial barrier enhancement caused by extracellular ATP. Methods: Combination of pharmacological and molecular approaches is used in this study. Summary of Results: ATP and its non-hydrolyzed analogues enhanced barrier properties of cultured endothelial cell monolayers, caused remodeling of cell-cell junctions, and significantly attenuated thrombin-induced barrier disruption. Intracellular Ca2+ increase and Erk activation observed by ATP were irrelevant to barrier enhancement. Inhibitory analysis and silencing RNA revealed the involvement of G proteins (specifically Gß and Go) as well as protein kinase A and its substrate VASP in ATP-induced barrier enhancement. Contractile state of endothelial cells governed by myosin light chain (MLC) phosphorylation underlies barrier properties. ATP treatment decreased MLC phosphorylation and specifically activated myosin-associated phosphatase. Dephosphorylation of Gß with sodium vanadate and Cyclic AMP activated PKA. Conclusions: We conclude that ATP-induced barrier-improving mechanism is independent from intracellular Ca2+, but involves activation of myosin phosphatase via novel G protein coupled mechanism and PKA.

10 AN ANALYSIS OF PATIENTS’ INTEREST IN RESEARCH FROM A MEMORY SPECIALTY CLINIC. N. J. Lane, T. Ott, S. A. Meade, A. M. Slattery, C. E. Gleason, S. Asthana, University of Minnesota, Minneapolis VA Health Care Center, Minneapolis, MN. and The Methodist Clinic of Minnesota, Minneapolis, MN and Methodist University Hospital, Memphis, TN.

Purpose: To determine the interest in participating in research of patients who attended a successful recruitment session. Methods: Research recruitment sessions are held at a memory clinic twice a month. Potential subjects are actively recruited by a staff member and asked to complete a basic screening questionnaire that includes their interest in participating in research. The questionnaire allows patients to indicate their interest level and preferred mode of research participation (phone, mail, face to face). Results: The majority of the patients that attended the session were interested in research (74%), and 57% of these patients expressed interest in phone or mail participation. However, only 16% expressed interest in face to face participation. Conclusions: The majority of clinic patients are interested in research participation. As the research enterprise continues to grow and become more diverse, there will be an increase in the need to attract patients from different populations. Currently, dementia clinics are the commonest source of research participants, but this may change as clinical trials of new pharmaceuticals, including Alzheimer’s disease and Parkinson’s disease, are developed.

11 THE RISK OF NEPHROLITHIASIS IN SPOON-LIKE ARTHRITIS. S. A. Leonard, J. A. Singh, H. M. Fellow of Rheumatology, University of Minnesota, Minneapolis, MN.

Purpose: Inflammatory arthritis is not generally considered a risk factor for nephrolithiasis; however previous studies have found a higher point prevalence of nephrolithiasis in rheumatoid arthritis than in the general population. The aim of our study was to examine the incidence of renal stones in patients with spondyloarthropathy (SPA) as compared with rheumatoid arthritis (RA) controls. Methods: This study was designed as a VA retrospective cohort study. The study group was obtained from patients followed in theopholate treatment with a successful recruitment comparing the prevalence of nephrolithiasis in patients with SPA (RA) and RA controls. This study was performed as per chart review. All patients met ACR criteria for either RA or SPA. Results: Forty-nine SPA patients (72%) and 48 RA patients have been enrolled. Over our goal to enroll 295 patients (84 SPA patients and 111 RA patients). These results will be discussed at the time of presentation. To date 16/49 (32.7%) SPA patients reported a history of renal stones compared with 4/27 (14.8%) RA patients. Chi square equals 3.95 (p = 0.047). Four patients with kidney stones in the SPA group and 2/4 patients in the RA group had stones or kidney stones at the time of diagnosis. The prevalence of renal stones in SPA was compared with RA and the prevalence of nephrolithiasis in RA was compared with SPA. Conclusion: These results show an increase in the prevalence of kidney stones in SPA compared with RA. This is similar to the findings of Miadne et al. These results suggest that SPA patients should be done to ascertain the cause of increased nephrolithiasis in SPA.

12 CONCENTRATION-DEPENDENT EFFECTS OF VASCULAR ENDOTHELIAL GROWTH FACTOR ON ENDOTHELIAL PERMEABILITY. T. Mirzapoiazova, I. Kolovou, P. Usatyuk, V. Natourajan, A. Verin, Johns Hopkins University School of Medicine, Baltimore, MD.

Increased endothelial permeability is involved in the pathogenesis of many cardiovascular and pulmonary diseases. Vascular endothelial growth factor (VEGF) is considered to be a major permeability increasing cytokine. On the other hand, VEGF is known to have beneficial effect on endothelial cells (EC), increasing their survival. In most cases barrier-disruptive properties of VEGF were observed at rather high concentrations, while lower concentrations induce cell migration and proliferation. We found that 10 ng/mL VEGF significantly improved barrier properties of cultured human pulmonary artery EC (HPAEC), as indicated by decreased transendothelial resistance measurement (20 ± 2.6% increase, p < 0.01), while 100 ng/mL VEGF had barrier-disruptive effect (48 ± 3.2% decrease, p < 0.01). Consistent with these data treatment with 10 ng/mL VEGF enhanced VE-cadherin staining at the endothelial monolayer, suggesting stabilization of cell-cell contacts. Level of myosin light chain phosphorylation (an index of EC contraction and barrier disruption) was dramatically increased after treatment with 100 ng/mL, but not with 10 ng/mL VEGF. In contrast, 100 ng/mL VEGF, but not 10 ng/mL, increased phosphorylation of G proteins (specifically Gß and Go), as well as protein kinase A and its substrate VASP in ATP-induced barrier disruption. Contractile state of endothelial cells governed by myosin light chain phosphorylation underlies barrier properties. ATP treatment decreased MLC phosphorylation and specifically activated myosin-associated phosphatase. Dephosphorylation of Gß with sodium vanadate and Cyclic AMP activated PKA. Conclusions: We conclude that ATP-induced barrier-improving mechanism is independent from intracellular Ca2+, but involves activation of myosin phosphatase via novel G protein coupled mechanism and PKA.