PROSPECTIVE MULTICENTER ASSESSMENT OF CRITICAL FACTORS DELAYING HOSPITAL THROMBOLYTIC THERAPY

Edgar R. Gonzalez, Joseph P. Ornato, Lori A. Jones, Giselle C. Bleecker, Michael J. Strauss, and the Virginia Thrombolytic Study Group, Medical College of Virginia, Richmond, Virginia

Purpose To assess the reasons for thrombolytic treatment delay in pts with AMI who present to the emergency department (ED) in a wide range of community hospitals. Study population AMI pts treated with thrombolytics in the ED and pts with presumed AMI who initially had no contraindications to therapy, but who did not receive a thrombolytic. Methods The following variables were monitored prospectively during a 6-month study period on all pts: ECG-time (time from ED arrival to initial ECG), decide-time (time from initial ECG to the decision to administer thrombolytic), process-time (time from decision to treat to administration of thrombolytic), and hosp-time (time from ED arrival to administration of thrombolytic). Results 230 pts from 13 Virginia hospitals were enrolled. 12M 8F (mean age 61.1±14 yrs) did not receive thrombolytic therapy. 210 pts (mean age 57±14.1 yrs) received thrombolytic therapy. tPA was used in 95% of cases. An average of 177±108 minutes elapsed between the onset of pain and treatment with thrombolytic therapy, with 61% of the delay occurring prior to reaching the hospital. Mean time delays were: ECG-time 11±15 min; decide-time 31±35 min; process-time 23±16 min; hosp-time 64±42. Variables that significantly correlated ($p < 0.05$) with hosp-time delay were: urban hospital location, teaching hospital status, high AMI case volume (>16 during study), stocking the thrombolytic drug in the ED instead of the pharmacy, initiating the drug in the ED rather than the CCU, and having the ED physician make the decision to treat without involving other physicians. Process-time and hosp-time were significantly faster with APSAC than with tPA (23±16 vs 9±4 min; 66±43 vs 33±16 min). Conclusions 1) thrombolytics should be given in the ED rather than the CCU; 2) thrombolytics should be stocked in the ED; 3) the decision to treat AMI with thrombolytics in the ED should generally be made by ED physicians; and 4) further study is needed to confirm that total hosp-time to treatment is faster with APSAC than with tPA and whether the difference is clinically significant.

INCIDENCE AND SIGNIFICANCE OF MITRAL REGURGITATION FOLLOWING FIRST MYOCARDIAL INFARCTION TREATED WITH rt-PA: RESULTS FROM TIMI II.

Peter B. Berger, Nicholas A. Ruocco, Jr., Thomas J. Ryan, Daniel J. Diver, Margaret Frederick, Barry Sharaf, Frederick Feit, Michael J. Attubato, David P. Faxon. Boston University Medical Center, Boston, MA.

Noninvasive data from the pre-thrombolytic era suggest that mitral regurgitation (MR) is present in over 30% of pts following acute myocardial infarction (MI) and is severe in up to 5% of pts. In TIMI I, contrast ventriculography within 7 hours of the onset of symptoms and immediately prior to thrombolytic therapy revealed MR in 13% (2% moderate/severe), associated with increased mortality. To assess the incidence and significance of MR following MI treated with thrombolytic therapy, we studied contrast ventriculograms from 196 consecutive pts at 4 sites in TIMI II undergoing protocol catheterization 18 to 48 hours following a first MI treated with rt-PA within 4 hours. Ventriculograms were analyzed retrospectively in a blinded manner and MR was graded on a 1+ to 4+ scale. 119 pts (60%) had no MR; 70 pts (36%) had 1+; 7 pts (4%) had 2+; no pt had 3+ or 4+ (0%). Comparison of pts with and without MR revealed no significant differences in infarct location (anterior 56% vs 48%), history of angina (29% vs 30%), multivessel disease (27% vs 25%), LV ejection fraction (48% vs 47%), or the 21 day incidence of congestive heart failure (CHF) (22% vs 26%) or mortality (2% vs 3%).

We conclude that within this cohort of pts who survived and underwent 18 to 48-hour protocol angiography following first MI treated with rt-PA, mild MR: 1) is common; 2) is similarly frequent following anterior as well as inferior infarction; and, 3) is not predictive of early CHF or death. Severe MR was not observed among these pts.

Wednesday, March 6, 1991
Poster Displayed: 9:00AM–12:00NOON
Author Present: 9:00AM–10:00AM
Hall F, West Concourse
Ventricular Tachycardia: Fibrillation and Defibrillation

MECHANISM OF ELECTROCARDIOGRAPHIC POLYMORPHISM DURING DIFFERENT VENTRICULAR TACHYCARDIAS IN THE SAME HEART.

Jiunn-Lee Lin, Justin Pearlman, David J. Wilber, Duke Du, Jeremy N. Ruskin, Hasan Garan. Massachusetts General Hospital, Boston, MA

The mechanism underlying multiple morphologies in surface ECG during distinct episodes of sustained monomorphic ventricular tachycardia (SMVT) was investigated in a canine model of experimental myocardial infarction. The sites of earliest activation (SEA) and global activation sequence patterns (ASP) were analyzed during 77 SMVT's induced in 10 dogs. Mapping was performed using bipolar recordings from 64 standardized endocardial and epicardial sites. ASP's of 2 SMVT's were compared quantitatively by a Euclidean metric generated by treating site-specific local activation times as unique components of 64-tuple vectors. ECG morphologies during 2 SMVT's in the same animal were classified as different if they differed in any one of their X,Y, and Z orthogonal Frank lead axes by $\geq 45^{\circ}$. This criterion resulted in 23 distinct SMVT morphologies. 123 pairwise comparisons (2 compared SMVT's always in the same animal) among morphologically similar, and 200 comparisons among morphologically different SMVT's were possible. In 81 (66%) pairs of similar SMVT's, both SMVT's had the same SEA and in 42 (34%), SEA's were observed at adjacent (<1cm) recording sites. No 2 morphologically similar SMVT's had disparate (>1cm) SEA's. The overall mean metric value for ASP comparison was 12.4 + 5.8. By contrast, in 200 pairs of different SMVT's, 131 (65%) had disparate SEA's; in 69 (35%) pairs SEA's were adjacent, although no 2 of a pair had the same SEA. Overall mean metric value for ASP was 33.91 + 7.3 (P=0.0001, vs. similar SMVT's). CONCLUSION: All morphologically similar SMVT's have closely spaced (<1cm) SEA's. Converse is not true: many morphologically different SMVT's may still have closely spaced SEA's, but can be distinguished by differences in their ventricular ASP's.

CORONARY VENOUS STASIS ABOLISHES THE ARRHYTHMIOGENIC SUBSTRATE OF ISCHEMIA

Alexandros C. Kralios, Fany A. Krallos, Fred L. Anderson, VA and University of Utah Medical Centers, Salt Lake City, Utah

Coronary sinus obstruction (CSO) increases the ventricular fibrillation (VF) threshold, and prevents or delays the early VF of acute ischemia. To clarify the electrophysiologic basis of these effects we studied changes of unipolar epicardial ventricular electrograms following double coronary artery occlusion (CAO), CSO and combined CAO + CSO in eight dogs. Multiplexed data obtained with ventricular drive from either a 32-site global flexible array (n=6) or a 64-site regional plaque array (n=2) were analyzed for activation (AT) and recovery (RT) times, A-R intervals and were used to construct isochrome maps from which longitudinal (Θ_L) and transverse (Θ_T) activation conduction velocities were derived. Within 6 minutes after CAO, sharp heterogenous changes appeared. ARI was shortened by up to 67ms ($\bar{\Delta}$ = -18.2±1.6ms) AT delayed ($\bar{\Delta}$ = 7.0±1.2ms), Θ_L and Θ_T were reduced by up to 71% and 17% respectively and conduction blocks appeared. In contrast when CAO was preceded by CSO, ARI was lengthened by $\bar{\Delta}$ = 6.1±1.6ms, AT remained unaffected and Θ_L and Θ_T reduction was only 41% and 12%. More important the sharp differences observed among closely adjacent regions during CAO, were abolished. We conclude that CSO abolishes the main characteristics of ischemic arrhythmiogenic substrate, presumably through retention of extracellular fluid which facilitates the diffusion of interstitial solutes.