

INCREASING HEMOLYSIS AND PREDICTABILITY OF ANEMIA IN THE CLOTH COVERED STARR-EDWARDS PROSTHETIC AORTIC VALVE
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This is an evaluation of the frequency and severity of increasing hemolysis in patients with hemolytic anemia from prosthetic valve replacement. In addition the serum lactic dehydrogenase (LDH) levels were evaluated in an effort to predict future anemia. Patients attended the postoperative clinic and had regular screening studies for hemolysis including serum LDH levels (normal 23-65). Patients were placed into anemic and non-anemic groups. LDH values for the anemic group include all yearly values for that patient including pre-anemia levels. The median LDH values for the groups are in the table and the numbers in parentheses are the patients studied that year.

Postop Year	1	2	3	4
Anemic	383 (16)	478 (19)	560 (12)	653 (11)
Non-anemic	181 (20)	150 (18)	187 (11)	147 (5)

The yearly increase in the anemic group is significantly different from the non-anemic group ($p < 0.005$). A subset of these patients showed a one-year increase of 3 LDH units in 15 non-anemic patients and 242 units in 17 anemic patients. The predictive value of the LDH level was assessed from values obtained prior to diagnosis. A value of 250 units predicted anemia on the next yearly visit with a 28% false positive and 4% false negative accuracy. The increasing hemolysis in the anemic patients may represent cloth wear (found in 5 of our 6 reoperated patients). Our current prevalence of anemia in the totally cloth covered aortic valve is 36/90 patients (40%). Therefore, monitoring of the LDH level is suggested. Values in excess of 250 units (4 times the upper limit of normal by other LDH methods) and/or increasing levels would suggest future anemia.

MAIN LEFT CORONARY ARTERY DISEASE. Patrick J. Scanlon, M.D., James V. Talano, M.D., John F. Moran, M.D., and Rolf M. Gunnar, M.D., FACC. Loyola University Stritch School of Medicine, Maywood, Illinois 60153

In a 3 1/2 year period, 76 patients (pts) were found to have greater than 50% obstruction of the main left coronary artery (LCA) by coronary arteriography (CA). Clinical presentation was one of pre-infarction angina (PIA) in 30 pts (39%). Total occlusion of the LCA was found in two pts. Severe obstruction of other coronary vessels was present in most pts. Total occlusion was present in the right coronary artery in 32 pts (42%) and in the left anterior descending artery in 18 pts (24%). Isolated obstruction of the LCA was seen in four pts. Calcification of the LCA was seen fluoroscopically in 18 (24%). Intercoronary collaterals were visualized in 51 (67%). Of 70 pts undergoing left ventriculography, 49 (70%) had obvious contraction abnormalities. One pt (1%) became hypotensive and died at the termination of the procedure. Another pt developed myocardial infarction six hours after study and died within 24 hours. 66 pts (87%) were considered operable; 51 underwent surgery with an operative mortality rate (MR) of 12% and a total long term MR of 16%. 24 pts who survived CA were not revascularized; 6 (25%) eventually died. Of 30 pts with PIA, surgical MR was 13% (3 of 23), and nonsurgical MR was 57% (4 of 7) ($p < 0.05$). It is concluded that LCA disease, especially when presenting as PIA, is a lethal condition which can be cautiously studied with an acceptable MR, and which usually can be treated surgically at an acceptable risk.

NON-TRAUMATIC DETERMINATION OF CARDIAC OUTPUT AND LEFT VENTRICULAR EJECTION FRACTION BY RADIONUCLIDE ANGIOGRAPHY. Heinrich R. Schelbert, MD; John W. Verba, PhD; Gary W. Brock; Allen D. Johnson, MD; Frank J. Rose, MD; Naomi P. Alazraki MD; William L. Ashburn, MD; University of California, San Diego, School of Medicine-VA Hospital, San Diego, CA

Previous reports indicated that cardiac output (CO) and left ventricular (LV) ejection fraction (EF) can be determined non-traumatically by radionuclide angiography (RCG). By recording the passage of a radioactive bolus through the heart a time-activity curve corresponding to LV volume changes can be derived. The purpose of the study was to develop a computer program for calculating CO and EF and to examine effects of background activity, scattered radiation and assignment of regions-of-interest (ROI) on these determinations. ^{99m}Tc human serum albumin was injected in 8 patients into the sup. vena cava during cardiac catheterization and the precordial activity recorded by an Anger camera interfaced with a small digital computer. Only that period of precordial activity, during which the bolus passage through the LV, was analyzed. After assigning LV and background ROI, EF was calculated automatically in time segments for the entire LV time-activity curve. The correlation coefficient between EF obtained from biplane cineangiograms (area-length-method) and EF calculated only during the early downslope of the LV time-activity curve and corrected for background activity was 0.92. EF was overestimated when the background ROI was too close to the LV and underestimated when the LV ROI encompassed other cardiac structures. There was good agreement between CO determined by RCG and dye-dilution ($r = 0.96$). It is concluded that although CO and EF can be measured by this non-hazardous technique with great accuracy, proper selection on the time-activity curve and proper assignment of the LV and background ROI are important.

BUNDLE BRANCH BLOCK DUE TO HIS BUNDLE LESIONS

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We have previously reported that ischemic lesions in the His bundle (Hb) could result in bundle branch block patterns or even delta wave formation (Circulation, In Press). To more definitively study this problem, 10 in vivo and in vitro dog hearts were studied with extra- or intracellular recordings from the proximal and distal Hb as well as the right (Rb) and left (Lb) bundle branches (BB). Vagal slowing or atrial pacing showed normal intraventricular conduction within a wide range of heart rates, 20-300/min. Lesions were induced by ischemia (ligation of the anterior septal artery) or by trauma (needle puncture). In vivo, Hb damage was manifested as fragmented or split Hb potentials (H_1, H_2). Atrial pacing or proximal Hb pacing induced $H_1 - H_2$ delay with concomitant Rb or Lb block. However, distal Hb pacing at comparable or even higher rates produced a normal QRS. In 3 dogs traumatic His lesions induced a split Hb potential which showed bradycardia-dependent right bundle branch block with concomitant delay and temporal alternation between H_1 and H_2 . In vitro, using a modified Elizari preparation containing the Hb, proximal and distal Rb and Lb, normal conduction from Hb to proximal BB (10-15 msec) and Hb to earliest ventricular muscle activation (26-33 msec) was observed at pacing rates from 30-220/min. Resting potentials of Hb cells ranged from 65-85 mV. Punctate lesions placed above the branching Hb caused bradycardia-dependent (10-30 beats/min) delays to the distal Hb, 2:1 block as well as complete Rb or Lb block. In addition, some portions of the distal Hb were activated after the bundle branch potentials. We conclude that lesions in the Hb can cause bundle branch block probably due to dissociation of normally synchronous conducting pathways in the common bundle.