

THE ROLE OF PERICARDIAL RESTRAINT IN EXPERIMENTAL RIGHT VENTRICULAR INFARCTION.

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To determine the potential role of pericardial restraint during right ventricular infarction (RVI), 6 closed pericardium (peri), open chest dogs were studied before and after RVI had been produced by ligating the right coronary artery and injecting 0.15 ml of mercury distally. The animals were preinstrumented with RV and left ventricular (LV) Millar pressure transducers. Regional ventricular size at end diastole was measured by segments lengths (L) of the infarct (I) and non infarct (NI) territories of the RV and the LV. The animals were volume loaded after RVI sufficiently to increase RV end diastolic pressure above 12 mmHg. Opening the peri partially to allow release of restraint upon the atria decreased left ventricular end diastolic pressure (LVP_a) slightly from 21±3 to 18±3 mmHg, P.N.S. Cardiac output (CO) was unaffected (+6% P.N.S.). L-NI and L-LV increased by 21%, p <.01 and 5% p <.05, respectively. Completely opening the peri to release restraint on the ventricles reduced LVP_a further to 16±3 mmHg, p <.01 and increased CO by 33%, p <.001 and L-LV by 24%, p <.01. We confirm in RVI that pericardial restraint upon the ventricles mediates changes in LV compliance, filling and CO. Furthermore, pericardial restraint upon the atria can also mediate changes in LV compliance.

CHAGAS DISEASE IN THE UNITED STATES: A FIFTEEN-YEAR EXPERIENCE

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Chagas disease of the heart has been considered rare in the USA. Since 1974 the diagnosis has been confirmed in 32 patients at our institution; only 4 were from South America, the remainder from Central American countries. There were 25 women and 7 men with a mean age of 51 at diagnosis.

Presenting findings were congestive heart failure (CHF) in 8 (25%), aborted sudden death in 3 (9%), sustained ventricular tachycardia (VT) in 2, anginal chest pain in 5 (16%), symptomatic atrioventricular block in 6 (19%), conduction abnormality on ECG in 5, and other in 3. Right bundle branch block, left axis deviation, or both, was present in 80% and left bundle branch block in 3 (10%). Findings suggesting coronary artery disease were frequent, with pathologic Q waves in 13 (41%), primary T wave inversions in 11 (34%), and ST elevation in 4 (13%). Exercise thallium scans suggestive of ischemia and infarction were found in 4 cases without coronary disease. At angiography, ventricular aneurysm was found in 11 (34%), focal areas of akinesia in 5, and diffuse dysfunction in 5.

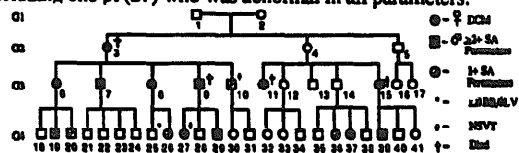
An aneurysm was associated with malignant arrhythmias at presentation (4 of 5 patients, P=.01) and late sudden death (P=.03). Sustained VT was inducible in 7 of 10 patients studied by programmed stimulation. There have been 7 cardiac deaths at follow-up averaging 58 months (3 sudden, 2 CHF, 2 unclassified); 48 month actuarial survival for the group was 68%. Survival in those with LV ejection fraction <.40 was 18% at 48 months vs. 86% with ejection fraction >.40 (P=.004); with aneurysm it was 54% vs 87% without it (P=.15). Death occurred only in those with ventricular dysfunction or aneurysm.

We conclude that Chagas disease of the heart is not infrequent in the USA, presents as one of several well-defined syndromes, and often mimics coronary artery disease. Prognosis is extremely poor in patients with ventricular dysfunction, and those with aneurysms are at high risk for arrhythmic events; both these groups frequently have inducible VT, and should be treated aggressively.

FAMILIAL DILATED CARDIOMYOPATHY: REPORT OF A LARGE KINDRED AND THE USE OF SIGNAL AVERAGING ON ASYMPTOMATIC INDIVIDUALS

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We identified a kindred with a high frequency of dilated cardiomyopathy (DCM) and a large number of clinically unaffected individuals. We speculated that these unaffected pts had occult cardiac disease and that signal averaging (SA) may be abnormal. All available information on affected individuals was reviewed. Unaffected pts were prospectively evaluated with SA, examination, ECG, 24 hour Holter and echocardiography. Six pts had DCM. The mean age of symptom onset was 46.2 years (range 43-54). Clinical events in this group included heart failure, cardiac dysrhythmias and sudden cardiac death. Twenty-one pts were clinically well and available for evaluation. SA was performed in 20 pts. At least 1 abnormal parameter was noted in 10/20 pts (50%) and 6/20 pts (30%) had 2 or more positive values, including one pt (27) who was abnormal in all parameters.



Left bundle branch block and LV dysfunction was seen in 1 pt (25). Non-sustained ventricular tachycardia (NSVT) was present in 2 pts (10,27), both of whom had positive SA ECG's. We conclude that DCM may occur as a familial disease with a primary expression in adulthood. In addition, SA was frequently abnormal in the individuals tested. Although of unclear significance, a positive SA ECG in this setting may be an early marker of familial DCM and warrants further longitudinal investigation.

NATURAL HISTORY OF MIDDLE-AGED ASYMPTOMATIC PATIENTS WITH HYPERTROPHIC CARDIOMYOPATHY

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Patients with hypertrophic cardiomyopathy (HCM) may present a wide spectrum of clinical and morphologic manifestations. Although some characteristics of the natural history of these pts are well known, the initial presentation and subsequent clinical course of certain specific subgroups of pts are not yet well defined. To further the understanding of the natural history of HCM, we analyzed the initial findings and longitudinal evaluation of 25 middle-aged pts (35-55 years) who presented to our institution with no or minimal symptoms. Follow-up ranged from 5 to 10 years (mean 8). Mean age was 41±5 years; 21 pts (84%) were male. The ECG was abnormal in 21 pts (84%). Maximal septal thickness was 18±4mm (range 13-30). Nineteen pts showed mild left ventricular hypertrophy, largely localized to the anterior septum, whereas only 6 pts had more diffuse involvement of 2 or more ventricular segments. Subaortic obstruction was evident in only 7 pts. During follow-up, 22 pts remained without significant symptoms and 2 showed slight deterioration. Of note, 1 pt who died suddenly at age 51, had shown diffuse hypertrophy and significant obstruction at initial evaluation. Thus, pts with HCM who achieve middle age without developing important symptoms usually show mild and localized left ventricular hypertrophy. Importantly, this specific subgroup of pts, with occasional exception, appears to have a benign long-term clinical course.