RIGHT VENTRICULAR DYSFUNCTION ATTENUATES THE SENSITIVITY OF ECHOCARDIOGRAPHIC SIGNS OF CARDIAC TAMPONADE

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To determine the influence of right ventricular dysfunction (RVD) on cardiac chamber collapse during cardiac tamponade (TAMP), we studied collapse of the RA (RAC) and RV (RVC) using transesophageal echocardiography in nine closed chest dogs. TAMP was produced by the stepwise instillation of saline into the pericardial space to achieve a pericardial pressure (Peri P) of 15 mmHg before and after RVD. RVD was produced by injection of 50μ spheres into the right coronary artery, resulting in an increase in mean RA pressure (RAP, 1.6 ± 1.0 vs. 7.6 ± 1.4 mmHg (control vs. RVD, mean ± SD, p<.05) and an upward shift of the pericardial pressure-volume relation. RAC and RVC occurred with larger effusions (EFF) at higher Peri P and RAP and greater pulsus paradoxus (PP) after than before RVD:

	RA COLLAPSE		RV COLLAPSE	
	Before RVD	After RVD	Before RVD	After RVD
EFF (cc)	48±28	136±66 *	92±51	188±63*
Peri P (mmHg)	2.5±1.0	8.1±1.8 *	5.3±1.8	11.6±1.9*
RAP (mmHg)	2.9±1.4	9.5±2.8 *	5.1±2.0	12. ±2.0*
PP (mmHg)	7.1±2.4	11.8±4.0 *	10.2±4.6	15±3.5*

*p<.05 after vs. before RVD (mean ± SD)

Mean aortic pressure had fallen 1.9 ± 2.0% and 6.5 ± 6.9% from baseline at the time of RAC (p=NS) and 3.0 ± 4.1 and 20.1 ± 20.8% at the time of RVC (p<0.03), before and after RVD, respectively). Thus in the presence of RV dysfunction, right heart chamber collapse is a less sensitive sign of cardiac tamponade and occurs later in the hemodynamic progression of pericardial effusions.

HUMAN CHAGASIC CARDIOPATHY AND MYOCARDIAL REJECTION: A SIMILAR PATTERN OF MYOCARDITIS.

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A cellular auto-immune phenomenon involved in the pathogenesis of cardiac lesions in Chagas' myocarditis has been widely postulated. To assess this proposal we compare the inflammatory infiltrate present in Chagas' myocarditis. CD8+ (cytotoxic/suppressor) and CD4+ (helper / inducer) T cells were analyzed and quantified by the immunoperoxidase technique in frozen sections of endomyocardial biopsies (EMB) from 13 chronic symptomatic chagasic patients and from 13 cardiac allograft patients with moderate rejection.

The mean number of cells/400 microscopic field in the chagasic group were CD8+=12.5 and CD4+=3.3, and CD8+=14.4 and CD4+=11.2 in the myocardial rejection group. There was a predominance of CD8+ relative to CD4+ T cells in all of the chagasic EMB and in 77% of the EMB with rejection. The CD8+/CD4+ ratio was significantly lower in EMB of the chagasic group than in the rejection group (p 0.003, Mann-Whitney test).

Conclusions: Chronic chagasic myocarditis in humans is constituted by mainly cytotoxic T cells , similar pattern to that of "myocarditis" in the rejection process, favoring the idea of a cellular , autoimmune process in Chagas' disease. However, there is a lower number of helper/inducer T cells in the chagasic group, suggesting that there is a different immunologic modulation in the chronic myocarditis induced by Trypanosoma cruzi.

ADAPTATION TO CHRONIC ALCOHOL INGESTION IN HAMSTERS

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Hamsters tolerated 50% alcohol in their drinking water for 42 weeks, with no deaths. All hamsters expressed depressed cardiac performance during the 42 weeks while the serum level of alcohol remained 0.10 g/dl or higher. When alcohol was withdrawn for 48 hours and the serum level of alcohol fell to 0, cardiac dysfunction reversed to control levels. Similar cardiac dysfunction, to that obtained with chronic alcohol ingestion, was obtained when hamsters hearts were perfused with 1% alcohol. Perfusion of hamster hearts with 1% alcohol, as well as chronic treatment of hamsters with 50% alcohol, caused an increase in diastolic pressure during the first hour of alcohol withdrawal. The latter may be due to the increase in diastolic $[Ca^{2+}]_i$ which results with early reperfusion of hearts following 1% alcohol perfusion. Treatment of hamsters with verapamil prevents the rise in diastolic $[Ca^{2+}]_i$ and the increase in diastolic pressure during early reperfusion. The PCr/Pi ratios, as determined by ^{31}P-NMR, were significantly depressed during the first 14 weeks of chronic alcohol ingestion, but returned to control values by 28 weeks [serum alcohol >0.10 g/dl]. Cardiac dysfunction in hamsters caused by chronic alcohol ingestion is reversible when alcohol is withdrawn for 48 hours and is preventable by treatment with verapamil.

NEW TWO-DIMENSIONAL ECHOCARDIOGRAPHIC METHOD FOR QUANTITATIVE ESTIMATION OF LARGE PERICARDIAL EFFUSIONS

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On echocardiography, pericardial effusions (PE) have been hitherto graded as small, moderate or large, with no further attempt to quantify the amount of PE. We devised a simple method, based on 2-D echo measurements in apical 4-chamber and parasternal views, for estimating the volume (vol) of moderate to large PE. Method: PE vol = Pericardial sac vol minus cardiac vol. Pericardial sac vol as well as cardiac vol were calculated by formula for ellipsoidal shape, $V=\frac{\pi}{6} \times L/2 \times D1/2 \times D2/2 = 0.523 \times L \times D1 \times D2$, where L = major axis, and D1 = minor axis, in apical 4 chamber view (Fig); D2 = minor axis in parasternal long or short axis view (Averages of 3 to 5 measurements, at onset of QRS on ECG).

Echo-estimated vol of PE correlated excellently with actual vol of PE drained in 15 patients (r=0.97) all of whom had large PE and tamponade. Arithmetic mean of differences between echo-estimated PV vol and actual PE vol drained was 73 ml; mean difference as percentage of actual PE vol drained was 9%. Our echo method underestimated PE vol in 3 patients with conspicuous atrial compression. PE vol drained varied from 400 to 2100 ml.

We conclude that a reliable approximation of moderate to large PE vol can be made by our simple 2-D echo method.