10:45

DETERMINANTS OF REDUCED LEFT VENTRICULAR FUNCTION FOLLOWING PROLONGED EXERCISE

Pamela S. Douglas, Mary O'Toole, William B. Malarkey, Jean Woolard, Beth Israel Hospital, Boston MA

Prolonged exhaustive exercise causes transient reductions in systolic and diastolic LV function, termed cardiac fatigue. To evaluate possible mechanisms, 23 men (age 40, 19-71) were studied using 2-D guided M-mode and Doppler echo and catecholamine levels at baseline and immediately following completion of the Hawaii Ironman Triathlon (2.4mi swim, 112mi bike, 26.2mi run). Mean exercise duration was 12:21hr(9:16 to 16:28). Seven men also had continuous heart rates recorded during exercise. HR was expressed as absolute (race HR) and relative workload (% max HR determined by prerace exercise test).

At race finish, LV fractional shortening (FS) was reduced from 39 to 37% ($p<.05$) and LV filling pattern, or the ratio of early to atrial velocities (E/A), fell from 1.59 to 1.15 ($p<.001$). Epinephrine, norepinephrine and dopamine all rose (259 to 1188 pmol/L, 1.98 to 9.95 nmol/L, 29.3 to 152.1 pg/ml respectively; all $p<.001$) but were not correlated with functional changes. Reduced FS was closely correlated with higher race HR ($r=.93; p<.005$) but not % max HR ($r=.40$). Reduced E/A was also more closely correlated with higher race HR ($r=.76; p<.05$) than with % max HR ($r=.65; p=.12$). Smaller reductions in FS and E/A were related to increasing age ($r=.52, .51$; both $p<.02$) and to longer exercise duration ($r=.36$; $r=.49; p<.03$).

Thus, cardiac fatigue appears unrelated to prolonged exposure to elevated circulating catecholamines or to relative cardiac workload. Instead, reduced LV function was most prominent in younger athletes who performed higher intensity exercise. Cardiac fatigue was most closely associated with exercise at a higher absolute workload, as measured by both higher race HR and shorter race duration.

11:00

REDUCED EXERCISE CAPACITY POST CARDIAC TRANSPLANTATION: IS THE MECHANISM REDUCED REDISTRIBUTION OF REGIONAL BLOOD FLOW?

Guy A Haywood, Peter J Counihan, James F Snedddon, Stephen H Jennison, Yaver Bashir, Sonia Bent, John R Pepper, William J McKenna. St. George's Hospital, London, England.

Redistribution of blood flow from non-exercising regions to working muscle is an integral part of the hemodynamic response to exercise. In animal studies, cardaic vagal afferent reflexes have been shown to modulate this response . Peak oxygen uptake (VO_2 max) remains limited post cardiac transplantation (Tx). This study tested whether cardiac denervation causes failure of redistribution of regional flow, and contributes to limitation of VO_2 max post Tx. A continuous thermodilution technique for measurement of renal blood flow (RBF) was validated in animal and human studies. RBF, oxygen consumption (VO_2 ml/kg/min), and forearm blood flow (FBF) by forearm plethysmography, were measured simultaneously during maximal,short duration (10 min), supine bicycle exercise in 20 subjects: 5 normals, 10 patients with NYHA class II-III heart failure (CCF), 5 patients in the first year post Tx (NYHA class I).

Results: (Mean ± Standard Error)

(* = p <.01)	Peak VO_2	Forearm BF % Reduction	Renal BF %Reduction
Normals	30±2.4	32±7.8	28±3.9
CCF	16±3.2*	22±7.1	37±5.2
Tx	14±0.4*	39±14.5	41±5.9 (NS)

Conclusions: Cardiac denervation in humans does not result in a decreased redistribution of blood flow from non-exercising regions to working muscle at peak exercise. This mechanism does not contribute to limitation of VO_2 max post Tx.

11:15

IBOPAMINE LOWERS CATECHOLAMINES DURING EXERCISE IN PATIENTS WITH HEART FAILURE

Dirk J. Van Veldhuisen, Armand R.J. Girbes, Stan A.J. Van Den Broek, K.I. Lie. Thoraxcenter, Groningen, The Netherlands.

Ibopamine (IB) is a new orally active inodilator used in patients (pts) with congestive heart failure (CHF). IB may lower plasma catecholamines [norepinephrine (NOR) and epinephrine (EPI)] by improving hemodynamics, but also directly by activating dopamine-2 receptors (NOR). To examine this possible effect of IB, NOR and EPI were determined both at rest and during peak exercise in 12 pts with moderate CHF (mean age 59±13 years (SD); ejection fraction 23±8%) before and after 7±2 weeks of treatment with IB 100 mg tid. All pts were familiar with this form of exercise testing.

Results:	Baseline	Ibopamine	p value (*)
VO2 max (ml/min/kg)	13.6	15.0	0.07
exercise time (sec)	570	636	0.15
resting EPI (pg/ml)	105	64	0.04 (#)
resting NOR (pg/ml)	652	592	0.39
exercise EPI (pg/ml)	463	415	0.03 (#)
exercise NOR (pg/ml)	5387	3351	0.02 (#)

Values are expressed as median; (*) Wilcoxon's test was used for analysis; p values <0.05 were considered significant (#).

Conclusion: These results suggest that IB may blunt the rise in plasma catecholamines during peak exercise in pts with moderate CHF. Moreover, since the increase in exercise capacity with IB cannot be attributed to increased sympathetic activity, it may be the result of improved hemodynamics.

11:30

LACTATE RESPONSE TO EXERCISE IN CARDIAC TRANSPLANT PATIENTS. TRANSITION FROM HEART FAILURE?

James D. Walter, Susan C. Brozena, Peter H. Brubaker, Deborah L. Morley, Mariell Jessup, Jeffrey Alpern, Alfred A. Bove.
Temple University Hospital, Philadelphia, PA.

In order to determine metabolic response to exercise in transplant patients (TP), we used symptom-limited maximal treadmill testing and compared physiological responses and peripheral lactate [LA] at an equivalent target workload in 5 males, 12-44 weeks post-transplant to 5 age, size and sex matched controls (NL). The target workload, equivalent to consumption of 2 L oxygen/min, was derived from standard formulas adjusted for body weight. Results: (* $p<.05$)

	REST		TARGET WORKLOAD		
	HR* (bpm)	[LA]* (mmol/L)	HR (bpm)	[LA]* (mmol/L)	VO2 (ml/kg/min)
TP	105±14	1.6±.4	135±16	3.5±1.6	17±3
NL	67±5	0.9±.4	129±12	1.3±0.7	20±5

Conclusions: Transplant patients compared to normals have higher lactates at rest and at a given workload. Early onset of lactate accumulation may contribute to limited functional capacity and may indicate persistence of peripheral abnormalities secondary to pre-transplant heart failure.