

because of central, but mostly because of peripheral causes, despite recovery of a normal left ventricular function. As substantial improvements have only been reported by long term retraining, we investigated the effects of a short term, square wave, modified interval training (SWEET) on muscular energetics and ultrastructural composition in 13 HTR ( $42 \pm 9$  yrs,  $74 \pm 12$  kg,  $10 \pm 3$  months after the operation) during a 3 sessions/week — 6 weeks program. After defining the maximal tolerated power (MTP) and the ventilatory threshold (VT) during an incremental test (IT), the 45 minutes SWEET sessions consisted in 9 crenels, each of 4 minutes at the power corresponding to the VT (approximately 50–60% of the MTP) followed by 1 minute at 90–100% of the MTP.  $O_2$  uptake ( $VO_2$ ),  $CO_2$  output ( $VCO_2$ ), ventilation (VE), heart rate (HR) were compared between the pre and post training IT and SWEET, blood lactates (LA), norepinephrine (NE) during the pre and post training SWEET. The ultrastructural capillary density (CD) and mitochondrial density (MD) on vastus lateralis biopsies were compared before and after training. **Effects of training:** (1) On maximal exercise: there were significant changes in MTP (+16%), peak  $VO_2$  (+9%), and VT (occurring at  $66 \pm 7^*$  vs  $59 \pm 6\%$  of the peak  $VO_2$ ). (2) On submaximal exercise: at the same power (approximately 50% of the initial MTP) there were significant decreases of  $VO_2$  (–12%),  $VCO_2$  (–12%), VE (–11%), HR (–7%), LA (–27%), NE (–35%). (3) On ultrastructural aspects: although CD did not change, the MD tended to increase (+17%), mostly because of a significant subsarcolemmal MD increase (+60%), without a central MD change. The intracellular lipid droplets volumetric density tended to increase (+43%). \*  $p < 0.05$ . **Conclusion:** a short term (6 weeks) endurance retraining program in HTR leads to an enhancement of the muscular oxidative capacity, that resembles the one seen in normal subjects, emphasizing the necessity of an endurance retraining in these patients.

3:00

#### 758-5 Influence of Exercise Training on Skeletal Muscle Vasodilatory Capacity in Chronic Heart Failure

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**Background.** Patients with chronic heart failure (CHF) are known to exhibit abnormalities in redistribution of blood flow and vasodilatory capacity during exercise. Exercise training has recently been recognized as an important therapeutic modality for patients with CHF. Training has been shown to improve vasodilatory capacity among normals in training-specific muscle.

**Methods.** Seventeen patients with first-diagnosis of CHF due to coronary artery disease ( $56 \pm 5$  years, mean EF =  $32 \pm 6\%$ ) were randomized to an exercise training group (N = 8), or a control group (N = 9). At randomization, patients in both groups initiated ace-inhibition therapy. Patients in the training group performed one hour of daily walking along with four sessions per week of high intensity monitored stationary cycling (40 minutes at 70 to 80% peak capacity) at a residential rehabilitation center for a period of two months. Control patients received usual community care. Calf baseline and post-ischemic flow rates were measured by strain gauge plethysmography before and after the study period.

**Results.** Training resulted in a 25% increase in maximal oxygen uptake ( $19.6 \pm 3$  vs.  $24.9 \pm 3$  ml/kg/min,  $p < 0.01$ ), whereas control patients did not change significantly ( $19.6 \pm 4$  vs.  $19.3 \pm 5$  ml/kg/min). Leg baseline flow was similar in both groups initially and did not change with training ( $1.3 \pm 0.6$  vs.  $1.6 \pm 0.6$  ml/100 ml in trained patients and  $1.8 \pm 0.6$  vs.  $1.9 \pm 0.6$  ml/100 ml in controls). Post-ischemic leg flow however, was significantly higher in both groups ( $14.4 \pm 8.0$  vs  $19.9 \pm 8.1$  ml/100 ml in trained patients and  $15.0 \pm 8.1$  vs.  $20.3 \pm 8.1$  in controls,  $p < 0.05$  for both groups).

**Conclusion.** The trained group improved post-ischemic leg flow, suggesting an improved vasodilatory capacity. However, the control group improved to a similar degree, suggesting that ACE inhibition therapy, not exercise training, causes the improvement. These data underscore the importance of treating these patients with ACE inhibitors.

3:15

#### 758-6 Does Moderate to High Intensity Exercise Affect Left Ventricular Remodeling in Cardiac Rehabilitation Patients After Myocardial Infarction?

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LV remodeling (LVR) can occur after MI and may adversely affect prognosis. The effect of regular moderate to high intensity exercise on LVR after MI is debated and needs further study. LVR was evaluated in 44 patients using serial echocardiographic measures including % abnormal wall motion (%AWM) and endocardial surface area/body surface area index (ESAI) taken initially  $8 \pm 5$  wks post MI (echo 1) and 12 weeks later (echo 2). Data were

compared between 22 consecutive pts who exercised  $3 \times /wk$  for 30 minutes at 60–85% HR reserve vs 22 control pts without exercise training, who were matched for age, sex, medications, type and site of MI, revascularization, and %AWM. Exercise patients improved peak MET levels from  $5.7 \pm 2.3$  to  $7.7 \pm 2.0$  ( $p < 0.001$ ). Serial echo data in the two groups follows:

	Exercise (n = 22)	Control (n = 22)
%AWM		
echo1	$18.68 \pm 13.72$	$18.26 \pm 14.11$
echo2	$18.00 \pm 13.42$	$17.33 \pm 14.77$
ESAI ( $cm^2/m^2$ )		
echo1	$56.89 \pm 9.30$	$64.26 \pm 10.33$
echo2	$54.57 \pm 7.40$	$56.89 \pm 9.37$

No significant change in %AWM and ESAI occurred in either group from echo 1 to echo 2. Thus, in this group of consecutive patients carefully matched for size and location of infarct, moderate to high intensity exercise does not appear to adversely affect LVR early after MI.

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#### Computerization of Cardiology: Expert Systems, Neural Networks, and Decision Analysis

Tuesday, March 21, 1995, 2:00 p.m.–3:30 p.m.  
Ernest N. Morial Convention Center, Room 21

2:00

759-1

#### In Vivo Characterization of Vascular Pathology Using Texture Analysis of Intravascular Ultrasound and a Neural Network Classifier

Evelin Lieback, Isabelle Hardouin, Michael Schartl, Jürgen Boksch, Roland Hetzer. *German Heart Institute, Berlin, Germany*

Intravascular ultrasound has received acceptance for accurate diagnosis of coronary disease. Conventional image formation provides limited information about tissue characteristics.

We have investigated the use of tissue characterization techniques to differentiate different pathologies in plaque tissue and thrombus. Using a PC-based data acquisition system interfaced to an interventional ultrasound system (IVUS) whose catheter probe operates at 30 MHz, we recorded the intracoronary B-scan images from 10 patients. We analyzed the gray level distribution by means of texture analysis in the plaques or in the thrombus. All in all we calculated 51 texture parameters. The parameters were implemented in a knowledge base. Different classification methods were investigated to determine their ability to differentiate between different plaque types and thrombus. A neural network architecture was developed and implemented for tissue characterization. Learning was achieved through a backpropagation algorithm. Using this neural network it was possible to differentiate between different types of plaques and thrombus. The echocardiographic texture of a thrombus was darker and had a lower heterogeneity than an atherosclerotic plaque. The results indicate that neural network can have an important role in the extraction of maximum information from the ultrasound signal. On the other site the incorporation of tissue characterization into intravascular ultrasound scanners could improve the efficacy of such procedures.

2:15

759-2

#### Use of Artificial Neural Networks within Deterministic Logic for Computer ECG Diagnosis of Myocardial Infarction

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The study was aimed at assessing the effect of incorporating neural networks (NN) inside an existing deterministic computer ECG analysis program in order to enhance the diagnosis of myocardial infarction. Separate neural networks were trained for inferior and anterior myocardial infarction using 200 normals, 100 IMI, 80 AMI and 42 left ventricular hypertrophy cases, all clinically validated. All the networks had a single output to discriminate between MI and non-MI. A variable number of inputs to the networks was used consisting of QRS  $\pm$  ST-T measurements. Separate test sets including 200 normals, 42 LVH, 101 AMI and 80 IMI cases were then utilised to find the best performing neural networks for IMI and AMI. The best neural network for each of IMI and AMI was then selected and inserted into the existing Glasgow Program (GP) for ECG analysis together with some modifications (M) to the diagnostic logic. The modified and original GP were then assessed using a completely new test set composed of 74 AMI, 52 IMI, 60 LVH and 230 normals.