

*Commentary:* T. E. CUDDY, *Winnipeg, Manitoba*

I WOULD like to contribute a brief comment on some measurements of the oxygen cost of breathing during exercise. These observations were made jointly with H. Levison and R. M. Cherniack. In a previous report from our laboratory, it was predicted, from extrapolation of incomplete data in the literature, that the oxygen cost of breathing might well play a significant role in limiting the oxygen available for non-respiratory work, and so limit exercise.

It was known that the oxygen cost of breathing was increased in disease states, but the effect of such high breathing costs during exercise was unknown.

Since that time, the increase in oxygen consumption during CO<sub>2</sub>-induced added ventilation has been measured in 11 normal subjects and 17 patients with emphysema. The normal subjects showed a mean cost of  $1.96 \pm 0.81$  ml. of oxygen for each litre of ventilation, compared to  $6.30 \text{ ml.} \pm 1.01/\text{l.}$  ventilation in the emphysematous subjects\* In addition, the total  $\dot{V}O_2$  was lower in the patient group, so that significantly less oxygen was left for the non-respiratory cost of exercise. This may impose a definite limitation on the performance of exercise in these patients, and suggests that the oxygen cost of breathing is an important measurement that should be made during their assessment.

\*Editor's note: The significance of this difference is increased by the fact that the cost was measured over a much smaller ventilatory span in the emphysematous patients.

## SESSION III: Paper 1

### The Influence of Active Conditioning Upon Subjects with Coronary Artery Disease:

#### Cardiorespiratory Changes During Training in 67 Patients

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SEVERAL parameters tend to differentiate physically fit from physically unfit men. With physical training, an individual may move from the unfit to the fit group.

One differentiating parameter, which has been referred to in previous papers, is that of physical work capacity. We express our results as work load 150 (WL 150)\*—the work load necessary to reach a heart rate of 150/min. The reason for our selection of 150 instead of 170, as used by many other workers (particularly T. Sjöstrand),

is that it is often difficult or inadvisable for a patient with coronary disease to reach a heart rate of 170. Fit or trained subjects are able to do more work per kilogram of body weight before their heart rates reach a level of 150 than people who are untrained. As did Dr. Yuhasz in his comparison of muscle strength in coronary patients and normal sedentary subjects (p. 714), we found no difference between the performance of the two groups (untrained cardiac patients and untrained normals) before training.

Another important parameter is systolic tension time or systolic tension time index (STTI), which we now call "the heart rate-systolic blood pressure product". Katz, Sarnoff and Braunwald<sup>1, 2</sup> have shown that there is a definite relationship between myocardial oxygen uptake and certain physiological parameters such as heart rate and systolic blood pressure, and perhaps a

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\*This corresponds to the PWC 150 referred to by other authors in this symposium.

closer relationship between the product of the two. Multiplication of these two factors yields a unit of mm. Hg beats/min. The work load corresponding to a fixed value of this index (25,000 mm. Hg. beats/min) can be determined. This in effect is a statement of approximately how much work a subject must perform before his myocardial oxygen consumption reaches a certain level. Trained normals and untrained normals fall on two different distribution curves with respect to this index, but again the untrained cardiac patients follow a distribution curve similar to that of the untrained normals.

It is one thing to say that unfit individuals can be characterized differently from fit individuals. It is another to say that, with physical training, a coronary subject can move from the physically unfit to the physically fit category. In view of this, we studied the effects of training on 67 patients with documented coronary heart disease, part of the group of 485 subjects described elsewhere (p. 901). These subjects participated in a physical training program, the details of which are described on p. 902. Of these 67, 50 adhered well to the training regimen, 10 adhered to it partially, and seven did not adhere at all. The WL 150 of those who adhered to the program (the adherent group) increased from 615 kpm/min to 764 kpm/min. The work load of those who partially trained changed from 586 to 639 kpm/min; this was a statistically significant change, but less so than in the completely adherent group.

The heart rate-systolic blood pressure product of the adherent group likewise showed a significant increase, while the partial and non-adherent groups did not.

Another parameter that tends to change with exercise is the electrocardiogram. Physically unfit and physically fit people have somewhat different electrocardiograms, and more important, in men with coronary disease certain ST-T changes and abnormalities of rhythm commonly develop during exercise.

Many patients have been encountered in whom arrhythmias were present in the exercise electrocardiogram before training, but not after training. Even more striking changes may be seen in the ST-T segment: there may be a large ST depression during the initial exercise test, and a much smaller depression after training, even at the higher workload that the subject is then able to perform.

Having looked at these specific examples of apparent improvement in certain physical parameters, the question arises: what are we doing basically, physically, that produces these changes? The biochemical changes produced by

training have been discussed by other participants. We may examine as a possible cause of this improvement, changes in the anatomy of the heart and coronary circulation. One method of studying this would be through coronary arteriography. Is an increase in the amount of collateral circulation to the myocardium produced by training? Some previous work suggests that this might be true. Richard Eckstein showed that if the coronary arteries of the dog were narrowed, and the animal was then exercised, coronary collateral circulation increased. But does this happen in man? It is a difficult question to answer, because it is difficult to gain consent for one arteriogram, and it is even more difficult to gain consent for two. However, in a few of the subjects in this study, coronary arteriograms were done before and after training.

One man, for example, in his initial untrained state had an ST displacement of 2 mm. on exercise. Incidentally, this could be abolished by nitroglycerin given before exercise. After training, he had no ST displacement at the corresponding level of exercise. As he went on to a higher level of exercise, the ST displacement appeared; again it could be abolished with nitroglycerin before exercise and finally would appear again at a still higher level even in the presence of nitroglycerin.

Coronary arteriograms were made before and after training. Initially he had a well-marked blockage of a major coronary artery; following the training program, there was clear evidence of an increase in collateral circulation.

A second patient also had definite improvement in WL 150 and in heart rate—systolic blood pressure index following the exercise program, and an increase in coronary circulation could reasonably be anticipated. Unfortunately, coronary arteriograms were not obtained on this man before he joined the program. After he had participated in the program and had benefited, coronary arteriograms showed that he still had significant obstructive disease and *there was no evidence whatsoever of increased collateral circulation.*

It would appear that some individuals may show an increase in coronary collateral circulation but this tendency is not present in all and the apparent benefit may be due to other changes—perhaps a decrease in circulating catecholamines. Part of our future work in Cleveland will include systematic analysis of coronary arteriograms taken before and after physical conditioning.

#### REFERENCES

1. SARNOFF, S. J. *et al.*: *Amer. J. Physiol.*, 192: 148, 1958.
2. KATZ, L. N. AND FEINBERG, H.: *Circ. Res.*, 6: 656, 1958.