Effect of Cardiac, Pulmonary, and Vascular Disease on One-Minute Oxygen Uptake*

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A simplified method for estimation of one-minute oxygen uptake (Vo2-1) during treadmill grade walking at vertical power requirements of 250, 750, and 1,000 kg-meters/min was devised, where power = weight (kg) × grade (fractional) × walking speed. All subjects were men. There were 29 controls, 34 subjects with coronary arterial disease (of whom 18 had had myocardial infarction), nine subjects with diffuse pulmonary disease, and four subjects with ischemic vascular disease. Abnormally reduced values for Vo2-1 were related to these diseases and, more specifically, to a history of myocardial infarction and (in pulmonary subjects) to reduced single-breath diffusing capacity. Lowest values of Vo2-1 for a group were found in ischemic vascular disease. Reduced response of Vo2-1 may therefore be caused by central defects of oxygen transport.

Previous work from this laboratory demonstrated that during treadmill grade walking, the oxygen uptake one minute after the onset of walking (Vo2-1) was reduced in 21 of 40 subjects with coronary arterial disease. Estimates of cardiac output using a single-breath expired-air method in a subgroup of seven subjects with coronary arterial disease were made at the same settings and time and were correlated with Vo2-1 (r = 0.95; P < 0.001). We concluded that reduced values of Vo2-1 in subjects with coronary arterial disease were caused by a defective response of cardiac output. In that study, administration of digitalis and long-acting nitrate preparations appeared to have no effect on Vo2-1, and subjects receiving propranolol were excluded. Also, in this earlier study, efforts at physical training in normal subjects had no consistent effect on Vo2-1.

A second study, to be presented in this report, was then undertaken. Its three aims were (1) to make the determination of Vo2-1 simpler and more accurate, (2) to confirm or refute the original observation that Vo2-1 was often reduced in subjects with coronary arterial disease, and (3) to compare the response in patients with coronary arterial disease with that in patients having either of two other diseases known to impair the transport of oxygen to exercising muscles. The additional two diseases studied were diffuse nonobstructive pulmonary disease and ischemic vascular disease of the lower extremities. These two diseases provided an opportunity to correlate Vo2-1 with diffusing capacity and with the effects of aortofemoral bypass.

Subjects
All subjects were men. They were divided into the following five groups: (1) normal, (2) normal findings on catheterization and coronary arteriographic studies, (3) coronary arterial disease, (4) diffuse pulmonary disease, and (5) ischemic vascular disease. Data on the numbers of subjects, age, height, weight, and Vo2-1 are given in Table 1.

The normal subjects were apparently healthy in that physical activity was normal and sympotoms of cardiovascular or pulmonary disease were absent. Procedures designed to detect disease of oxygen transport not causing symptoms were limited to a brief interview before the test, spirometric studies, and the monitoring electrocardiogram. Only five subjects engaged in regular efforts to improve physical fitness.

The subjects with normal findings on catheterization complained of chest pain, and all had undergone left cardiac catheterization with selective coronary arteriographic studies, left ventriculographic studies, and measurement of left cardiac pressures. Normal results were obtained. The subjects believed, therefore, to be free of cardiac disease, and no other disease of oxygen transport was known to exist.

In the group with coronary arterial disease, the presence of that disease had been established by coronary arteriographic studies. The number of vessels involved was scored according to the method of Saltups and associates.

The subjects with diffuse pulmonary disease initially had diffuse densities on chest radiographs, and spirometric testing excluded obstructive airway disease. Analysis of the diffusing capacity (Dsb) in liters by the single-breath technique was performed on all subjects in this group.

All of the subjects with ischemic vascular disease complained of intermittent claudication, and angiograms in three of the four confirmed the presence of disease affecting the arteries to the lower extremities. Two of the subjects were restudied following successful aortofemoral bypass.

In addition to these groups, an additional group was studied in order to define the reproducibility of Vo2-1 as determined in laboratory sessions on different days. Six subjects (two normal, one with normal findings on catheterization, one with coronary arterial disease, one subject with...
hypertension, and one with unexplained chest pain) underwent second studies at intervals of one to ten days after the first study. The inclusion of subjects in categories other than those of the main portion of the study was considered to be justified because no other mechanism affecting the value of VO2-1 was known to exist which might not have been present in the other subjects.

**Methods**

**Measurement of VO2-1**

All subjects were studied employing an open circuit with timed collection and electronic analysis of mixed expired air (Fig 1). In the early part of the series, two stopcocks of the three-way type (Collins P-321) were employed, an upper one for switching the subject into the circuit and one beneath it and directly connected to it for evacuation of the 30-L neoprene bag which was used for gas collection (Collins P-342-30). A three-way directional valve (dead space, 60 ml) connected the subject to the circuit, and the expiratory part of this valve was in turn connected to the double-stopcock assembly by a 110-cm length of neoprene tubing whose internal diameter was 2.54 cm. The three-way stopcocks were replaced at a later stage of the study by two pneumatically powered, electronically controlled solenoid valves. Attempts to minimize the volume of tubing resulted in a higher resistance than desired (19 cm H2O at a flow of 200 L/min), but the present study did not require the high ventilations encountered during maximal exercise in normal subjects. Use of the electronic valves facilitated switching but did not alter values of VO2-1 obtained. Since accuracy required that switching of the valves be done at the same stage of the respiratory cycle on each run, a pneumotachometer which furnished a signal visible to the valve operator was placed in the inspiratory circuit.

Figure 1 presents a circuit diagram of the system in its final form. Gas collections were started on the first inspiration after 48 seconds of exercise and were terminated on the first inspiration after 12 seconds had elapsed. Gas volumes were measured on a dry gas meter (Warren Collins) whose accuracy had been validated against a 120-L gasometer (Tissot). A spirometer-emptying pump facilitated this process. The method of gas analysis has been described and consisted of meters for carbon dioxide (Godart infrared) and for oxygen (Westinghouse electrolytic cell) linked to a circuit designed to eliminate errors resulting from changes in water vapor concentration. The VO2-1 was calculated by a form of the conventional equation for calculations of VO2 in open circuit estimates, which assumes no uptake or output of nitrogen. There was no provision for changes in gas stores. A previous study of the effects of gas storage had shown that these were of negligible importance by the end of the first minute of exercise.

The heart rate and the electrocardiographic wave form were monitored for purposes of safety.

**Diffusing Capacity**

The Dsb was determined by the single-breath method of Ogilvie et al, as modified by Mitchell and Benetti. The percent predicted Dsb was calculated from a formula shown below (determined by John F. Keighley, M.B., of the Syracuse Veteran's Administration Hospital, written communication, August 1975):

\[
\% \text{ predicted } \text{Dsb} = 0.205 \times \text{height} - 0.255 \times \text{age} + 6.51
\]

where height is measured in centimeters and age in years.
Experimental Protocols and Calculations

For the standard determinations of \( \dot{V}O_2 \)-1, the subject gave consent after explanation of the nature and purpose of the experiment. The postabsorptive state was not required. In a preliminary interview conducted by a technician, the medication and state of physical activity in usual life were assessed. Spirometric testing was performed, and chest electrodes were applied for electrocardiographic monitoring. The subject practiced walking on the treadmill without holding the rails. He then performed three to six runs at 53.6 meters/min (2 mph) with the incline set so that the vertical power requirement was 250 kg-meters/min, where

\[
\text{power} = \text{walking speed} \times \text{treadmill grade} \times \text{body weight}
\]

with walking speed measured in meters per minute, treadmill grade in fractional units, and body weight in kilograms.

In both repetition of the test and in progression to more strenuous tests, there was no arbitrary period of time between tests. In deciding when to start the next test, the condition of the subject, his willingness to perform the next test, and the fall of the heart rate to a stable or slowly changing value were taken into account. Subjects were allowed to stop a test by grasping the rails, to delay the start of the next test, or to stop the entire series on request. All calculations were averaged from two satisfactory determinations at any specific vertical power requirement, and incomplete runs were not used. The estimate of \( \dot{V}O_2 \)-1 for a vertical power requirement of 250 kg-meters/min employed the following conventions: (1) six runs were performed if an apparent upward or downward trend occurred in the series; and (2) the two lowest values were averaged, but the first value was discarded when it was the lowest, in order to exclude a “warm-up” effect, which was frequently observed. As few as three runs were performed if there were no upward or downward trends. These decisions were made possible by immediate calculation of \( \dot{V}O_2 \)-1 by hand or, later (Fig 1), by a locally constructed analog computer which provided accuracy equivalent to calculation by hand. Following completion of the runs at a vertical power requirement of 250 kg-meters/min, two runs were performed at a power requirement of 750 kg-meters/min and two runs at 1,000 kg-meters/min. Walking speed was increased to 67 meters/min (2.5 mph) or 80.5 meters/min (3 mph), if necessary, to avoid using treadmill grades higher than 30 percent, a procedure used in the previous study.1 In nine subjects, three runs were performed at a vertical power requirement of 750 kg-meters/min, and in nine subjects, three runs were performed at a vertical power requirement of 1,000 kg-meters/min when the first two determinations failed to agree within 0.2 L/min. In this event the two closest runs were averaged.

RESULTS

In order to evaluate the reproducibility of \( \dot{V}O_2 \)-1 within laboratory sessions, the difference between consecutive determinations in the 29 subjects classified as normal or having normal findings on catheterization was compared with a similar estimate for 25 normal subjects studied using the previously described method.1 The variance of the paired differences in the present study was 0.016 L/min. This was significantly lower than the value of 0.043 L/min found in the previous study (\( F = 2.67; P < 0.01 \)).

The reproducibility of the \( \dot{V}O_2 \)-1 determination
Figures 2. Mean values of VO2-1 for all groups at vertical power requirements of 250, 750 and 1,000 kg-meters/min. Value for VO2-1 at vertical power requirement of 250 kg-meters/min is similar for all groups (values not significantly different). For each group, there is linear relationship between VO2-1 and vertical power requirement. This relationship is most abnormal for group with ischemic vascular disease (POD). N, Normal group; NC, group with normal findings on catheterization; DPD, diffuse pulmonary disease; D1, single-breath diffusing capacity; CAD, coronary arterial disease; and MI, myocardial infarction.

(average of two determinations as described in the methods section) between two sessions was evaluated in the six subjects studied on two occasions. The mean of the differences was 0.03 L/min and was not significant. The standard deviation of the paired differences was 0.095 L/min.

Figure 2 shows mean values of VO2-1 for all of the groups studied at vertical power requirements of 250, 750, and 1,000 kg-meters/min. It will be noted that the groups with coronary arterial disease or diffuse pulmonary disease have each been subdivided based upon other information. In the case of the group with coronary arterial disease, the subdivision was based upon the history of myocardial infarction, so that the 31 subjects were divided into a subgroup with coronary arterial disease but no previous myocardial infarction (16 subjects) and a subgroup with coronary arterial disease and a previous myocardial infarction (12 subjects). This subdivision was done retrospectively after a search for other correlations was nonrevealing. There was no correlation of test scores with the number of vessels involved, the left ventricular end-diastolic pressure, or the ejection fraction. For the group with diffuse pulmonary disease, the separation was based on percent predicted Dsb. Of the nine subjects, four had a percent predicted Dsb greater than 60 percent, and five had values less than 60 percent.

In Figure 2, each group or subgroup is displayed as a line which reflects the increase in VO2-1 caused by increasing values for the vertical power requirement. All of the lines are essentially straight, and a separation between various groups emerges as the vertical power requirement increases. The group with ischemic vascular disease had the line with the lowest slope. The subgroup with coronary arterial disease and a previous myocardial infarction and the subgroup with diffuse pulmonary disease and a percent predicted Dsb less than 60 percent were similar and were intermediate between normal subjects (the normal group and the group with normal findings on catheterization) and the group with ischemic vascular disease. The subgroup with coronary arterial disease and no previous myocardial infarction and the subgroup with diffuse pulmonary disease and a percent predicted Dsb more than 60 percent had similar lines slightly below the normal group and the group with normal findings on catheterization.

The purpose of Figure 2 is to show these differences employing the unmodified VO2-1 data. Subsequent analysis suggested that correction of VO2-1 scores for differences in age and height was desirable prior to any estimate of the range of variation. It is for this reason that Figure 2 does not contain indicators of variation within the individual groups.

In order to define a lower limit of normal for VO2-1 or a calculation derived from it, it was necessary to reach a decision on whether to combine the normal group and the group with normal findings on catheterization into a single control group. Figure 2 suggests that these two groups had similar performance, but they were selected for study using completely different criteria. The normal group was asymptomatic but may well have included subjects with occult coronary arterial disease. The group with normal findings on catheterization was symptomatic but presented no evidence of coronary arterial disease after intensive study. The development of correlations for age and height and the reason for combining the two groups is, therefore, presented in some detail.

Before combining the normal subjects and those with normal findings on catheterization into a single control group, the data on VO2-1 at a vertical power requirement of 1,000 kg-meters/min were analyzed using either the normal subjects, or those with normal findings on catheterization, or the single combined normal group as the control group for the various diseased groups (coronary arterial disease,
diffuse pulmonary disease, and ischemic vascular disease). For this purpose a multiple regression formula was devised for each of the three possible control groups. Age and height were found to correlate significantly and independently in the normal group and in the single combined normal group by calculating partial correlation coefficients, holding age and height, each in turn, constant. Failure of partial correlation to reach significance for the group with normal findings on catheterization was attributed to the small size of this group. Weight was not used because the partial correlation coefficient was not significant. These calculations provided us with three multiple regression equations, each equation based on age and height. Each equation was then used to calculate the percent predicted \( \text{Vo}_2 \) for every case in all 76 subjects, and the percentage of reduced values for the percent predicted \( \text{Vo}_2 \) was determined. Only three of the 76 subjects (one with normal findings on catheterization, one with diffuse pulmonary disease, and one with ischemic vascular disease) would have had to be placed in a different classification (normal percent predicted \( \text{Vo}_2 \) vs reduced percent predicted \( \text{Vo}_2 \)) as a result of any difference in the selection of the control group. Therefore, the regression equation used was that derived from the combined group (normal subjects plus those with normal findings on catheterization). This equation was

\[
\text{Predicted } \text{Vo}_2 = -0.2375 - 0.0084 \text{ age} + 0.0135 \text{ height}
\]

where age is measured in years and height in centimeters (\( r = 0.74; P < 0.001 \)).

Since only reduced values were considered to be abnormal, the lower limit of normal was taken as the mean minus 1.64 SD. Table 1 presents the data for percent predicted \( \text{Vo}_2 \) for all groups and subgroups employing this calculation, together with a listing of the numbers of subjects in each category with reduced values for the percent predicted \( \text{Vo}_2 \). There were no reduced values observed in the control series (normal subjects and those with normal findings on catheterization). The incidence of reduced values was as follows: 19 percent (3/16) for the subgroup with coronary arterial disease and no previous myocardial infarction; 56 percent (10/18) for the subgroup with coronary arterial disease and a previous myocardial infarction; 25 percent (2) for the subgroup with diffuse pulmonary disease and a percent predicted \( \text{D}_{SB} \) greater than 60 percent; 100 percent (5/5) for the subgroup with diffuse pulmonary disease and a percent predicted \( \text{D}_{SB} \) less than 60 percent; and 100 percent (4/4) for the group with ischemic vascular disease.

Figure 3 demonstrates that in the group with diffuse pulmonary disease, there was a significant positive correlation between percent predicted \( \text{Vo}_2 \) and the \( \text{D}_{SB} \) expressed as a percent of predicted (\( r = 0.73; P = 0.027 \)).

Marked improvement in the percent predicted \( \text{Vo}_2 \) occurred in two subjects with ischemic vascular disease following aortofemoral bypass. In one subject the value rose from 77 percent to 100 percent nine months after surgery. In the other, it rose from 54 percent to 88 percent four weeks after surgery.

**DISCUSSION**

The method for measuring \( \text{Vo}_2 \)-1 used in this study is simple enough for use in any laboratory with experience in estimating \( \text{Vo}_2 \) by gas collecting techniques and has been found to be more reproducible on repetition during the same laboratory session than the more complicated methods used by us in the past. The automation is a convenience but not necessary for accuracy. Averaging of two determinations is desirable in order to reduce the errors inherent in short periods of gas collection. The inclusion of runs at lower levels of stress (vertical power requirements of 250 and 750 kg-meters/min) does much to eliminate spurious low values (referred to by us as the "warmup" effect) and high values due to lack of familiarity. Initiation of the testing procedure with mild exercise is also necessary for safety in the subjects with coronary arterial disease, where electrocardiographic abnormalities may appear at very low levels of stress and may contraindicate more severe testing. The test does not routinely depend on reaching the limit of endurance or any other index, such as 90 percent of the predicted maximum heart rate. The measurement is sufficiently reproducible so that it can be used to monitor therapeutic efforts and to assess the natural history of the disease process both in individual subjects and in groups.

The successful correlation of the test scores with
disease abnormalities can be best understood from Figure 2, which shows clearly that separations of groups become clearer with increased workload, provided the duration of exercise remains fixed at one minute. This finding is, of course, consistent with general experience in stress testing and would be expected. It is somewhat less obvious, but of great importance, that the shorter the duration of the test, the greater is the workload which can be tolerated and that this fact has been used in the design of the test. The selection of a vertical power requirement of 1,000 kg-meters/min was not arbitrary or accidental. This workload represents arduous grade walking and, if carried to the steady state, yields values of \( \text{VO}_2 \) in the range of 2.5 L/min in normal subjects; however, as this study and the previous one\(^1\) indicate, the test can be performed by subjects with advanced cardiac, pulmonary, and vascular disease. This fact suggests that in subjects in whom the rise of \( \text{VO}_2 \)-1 is defective, energy is provided through nonoxidative (glycolytic) mechanisms, allowing the defect in oxygen transport to be detected. A similar phenomenon presumably occurs during the traditional demonstration of maximum oxygen uptake as a plateau of \( \text{VO}_2 \) in the presence of an increment in workload.

As part of the analysis, we examined data on ventilation and heart rate in all groups. No useful purpose would have been served by including this information. Both tachycardia and hyperventilation were frequently encountered in normal subjects, and many patients with advanced coronary arterial disease had normal responses of the heart rate with defective \( \text{VO}_2 \)-1 responses. Arterial oxygen tension was measured at a vertical power requirement of 1,000 kg-meters/min in four subjects with diffuse pulmonary disease, but the data are not presented because of the small number of observations. We have also withheld statistical correlations of any sort which were not significant, such as the correlations with data from cardiac catheterization and correlations with electrocardiographic data obtained during the test. There is no intent in this report to have the \( \text{VO}_2 \)-1 determination considered as a diagnostic procedure in competition with other tests, since reduced values were found in less than half of the subjects with coronary arterial disease.

The determination of the lower limit of normal received considerable attention because the diseased populations, especially the whole group with coronary arterial disease, were almost certain to contain subjects with normal, as well as abnormal, values. We wished to avoid bias in favor either of robust health or in favor of severely deconditioned subjects. It was therefore reassuring to observe that the subjects with normal findings on catheterization were very similar to the normal subjects. Some of these subjects were greatly concerned about their symptoms and had reduced their level of daily physical activity. It is also possible that the relatively high workload excluded extremely deconditioned subjects who simply lacked the strength and coordination necessary for treadmill walking at grades of about 20 percent. A number of subjects could have been added to all groups except the normal group if incomplete tests had been included, but the analysis of data would have become excessively complex.

When all groups of subjects are considered, it appears relatively certain that \( \text{VO}_2 \)-1 under some conditions can be reduced because oxygen transport is impaired. By "oxygen transport" in this report, we mean to include the effects of ventilation, pulmonary gas exchange, cardiac output, and the patency of major vessels supplying the exercising muscles. The initially low values in patients with ischemic vascular disease, along with the marked improvement in values after aortofemoral bypass, even to the point of achieving a normal value in one subject and a marginally normal value (88 percent of predicted) in another subject four weeks after surgery, constitute the clearest indication that reduced oxygen transport is involved. Asmussen\(^9\) has reported reduced values of \( \text{VO}_2 \) during bicycle exercise when pneumatic cuffs reduced the circulation to exercising muscles. His and our present observations may constitute an example of the "critical oxygen tension," i.e., a fall (or a failure to rise in a normal fashion) of muscular \( \text{VO}_2 \) as a result of decreased oxygen supply. This phenomenon was well described by Stainsby\(^10\) in isolated muscle preparations.

In patients with diffuse pulmonary disease, the correlation of percent predicted \( \text{VO}_2 \) (corrected \( \text{VO}_2 \)) with similarly corrected \( \text{DHb} \) also suggests a relation to oxygen transport; however, with respect to coronary arterial disease, the situation appears to be more complicated. It might be that the reduced values for percent predicted \( \text{VO}_2 \) found in 56 percent (10) of 18 subjects following myocardial infarction represented an inability to increase cardiac output in a normal fashion as a result of the infarcted muscle. Some of the reduced values in subjects with coronary arterial disease were as low as those seen in the other two diseased groups. In our previous study,\(^1\) we did find a positive correlation between \( \text{VO}_2 \)-1 and the one-minute cardiac output in seven subjects with coronary arterial disease and also found a negative correlation with values for the left ventricular end-diastolic pressure higher than 12 mm Hg; however, in the present study, cardiac output was not measured, and there was no correlation between percent
predicted VO₂, left ventricular end-diastolic pressure, or ejection fraction. In neither study was there a correlation with the number of vessels involved. The absence of these correlations suggests the possible involvement of another mechanism. Such a mechanism could be related to a decreased level of physical activity in individuals who know that they had experienced myocardial infarction and were, therefore, physically deconditioned. Such deconditioning might be obligatory if the disease was severe, or it might be iatrogenic or psychologic in origin. We are attempting to evaluate this problem with studies before and after physical training but have yet to reach a conclusion because of the small number of subjects who can be recruited for such a program. Therefore, the explanation for reduced values of percent predicted VO₂ in subjects with coronary arterial disease remains obscure at the present time, and there might well be two or more explanations.

What might be the clinical usefulness of such a test? Alternative and accepted forms of evaluation are available in the disease categories of this study; however, exercise testing in practice includes patients with dyspnea, fatigue, chest pain, or leg pain on exertion who do not fall into clear-cut diagnostic categories. We have used the test in such circumstances and have found that either normal values or markedly abnormal values were helpful in reaching a decision about the need for further diagnostic evaluation. This has been the case with claudication in patients with possible ischemic vascular disease. Clinical experience would suggest that any such patient with a reduced value for percent predicted VO₂ should undergo angiographic studies. Normal values for percent predicted VO₂ have aided in the diagnosis of neurocirculatory asthenia.

If the test has clinical value in coronary arterial disease, it should be in the assessment of therapy, particularly in rehabilitative efforts. The degree of exercise is well defined; the VO₂ values are not directly related to endurance; and the heart rate, often influenced by administration of drugs, is not involved in the test score.

As the value of formal efforts to improve physical fitness in patients with coronary arterial disease who have survived myocardial infarction gains acceptability, it seems possible that there will be a need to classify subjects into two or more groups. One possible method of doing this might involve exercise performance. In addition to measurements of heart rate, ST-segment depression, and the occurrence of arrhythmias, we would propose that the percent predicted VO₂ could also be measured. If the test is performed without chest pain, exhaustion, or other adverse effects and if the percent predicted VO₂ is normal, formal rehabilitative efforts in a closely supervised program may not be critically needed. Such an individual has demonstrated that he can successfully adapt to an abrupt demand for muscular activity for a one-minute period without electrocardiographic abnormalities and with a combination of a peripheral extraction of oxygen and an increase in cardiac output which place him in a normal category. The possibility that cardiac output is reduced but that the percent predicted VO₂ remains normal may, in fact, be no great disadvantage. If cardiac output is reduced and the arteriovenous difference is elevated, the subject is responding to exercise as though he or she were physically conditioned. Several studies have shown that the arteriovenous difference (but not the cardiac output) is increased by physical training. Therefore, a subject with a normal VO₂-1 response may be said either to have a normal cardiac output or else to have demonstrated an ability to compensate for a reduced cardiac output with increased peripheral extraction of oxygen by exercising muscles. In either event the expense and effort of a cardiac rehabilitative program might be avoided. Such a subject might be advised to lead an active life (exercise in his home) in order to maintain his or her already demonstrated normal degree of fitness, or alternatively, the patient might be advised to participate in a gymnastic program designed for apparently healthy persons. Experience with the VO₂-1 test coupled with electrocardiographic monitoring suggests that at least one-third of the patients with a history of myocardial infarction could be counselled in this fashion; however, if either electrocardiographic abnormalities or a reduced value for the percent predicted VO₂ are encountered, we believe that we must assume that a patient, if known to have coronary arterial disease by reliable criteria, should involve himself in an exercise program only under careful supervision. The net effect of adding the VO₂-1 test to electrocardiographic measurement is to bias the decision in a more conservative direction. Although we presented no electrocardiographic data in this report, the occurrence of a reduced value for the percent predicted VO₂ as the sole abnormality in the testing procedure was a common finding, and there was no significant correlation between low values for percent predicted VO₂ and ST-segment depression.

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REFERENCES

Animal Sonar Systems

The first step in the discovery of bat’s sonar was made by an Italian scientist, Spallanzini. In 1793 he caught some bats in a belltower, blinded them and released them some distance away. The blind bats flew back to their roosts in the tower, and even caught insects on the way. Meanwhile a Swiss naturalist had plugged the ears of some bats and found that they were incapable of navigating. In 1938, Donald Griffin, working at Harvard, showed how bats were able to avoid obstacles by ultrasonic echolocation. Later experiments showed how insects were tracked down and caught. The sound emitted by flying bats ranges from 10,000 to 100,000 cps, but the important sounds for echolocation are usually between 30,000 and 60,000 cps. When the bat detects an insect and turns toward it in the approach phase, the rate speeds up. The high rate of pulse emissions presumably enhances the sensitivity of the sonar, so enabling the bat to judge the position of its prey so accurately that it can trap a minute fruit fly in its open mouth.

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