An overview of cardiac rehabilitation and exercise rehabilitation

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I can understand why many people, both physicians and patients, do not feel that exercise rehabilitation with telemetry monitoring (CPT 93798) is necessary. But, as emphasized in my previous BUMC Proceedings articles (1-3), I cannot understand why anyone would doubt the necessity of cardiac rehabilitation (also CPT 93798). Therein lie the problems that I have dealt with for 20 years—cardiac rehabilitation is equated with exercise rehabilitation. Even though exercise rehabilitation is a portion of the discipline required to complete the goal of restoring normal living for heart patients, the exercise part is perceived by third parties and most paying parties as the mainstay of a cardiac patient’s rehabilitation. Why can’t heart patients do exercise on their own, unmonitored and unsupervised? What is the medical necessity of telemetry-monitored exercise?

Exercise rehabilitation, whether supervised or not, and whether monitored or not, requires at least an elementary understanding of exercise physiology. With denial of exercise rehabilitation (CPT 93798) often occurring, such an understanding seems even more appropriate. It is, after all, the heart’s responsibility to serve as a major link in the transport of oxygen, not only to itself and other vital organs, but also to the organ system responsible for most of our ability to enjoy the physical aspects of life—the skeletal muscle.

I think of exercise as any type of physical activity that requires one to perform more physical work than complete bed rest. Arising from a supine position, playing golf or other recreational activities, standing and performing angioplasty, going out to dinner or to a movie, downhill skiing, and jogging are all forms of exercise. They all require the use of the skeletal-muscle system. For this muscle system to work, it must have oxygen. For oxygen delivery, one must have at least 1 lung, a heart, and skeletal muscle that is capable of extracting and using the oxygen. Thus, to live and enjoy life, we must use skeletal muscle and we must exercise, at least to some degree.

Many of my most memorable patients are physicians. The following case history illustrates this point. Early in my rehabilitation career, a physician on our staff (well-known to all of us) had become aware some 9 years earlier that he had cardiovascular disease. At that time, he required hospitalization for an acute chest pain syndrome. During the next 5 years, he was evaluated occasionally for symptoms of shortness of breath and intermittent chest pains. Three years before he presented to us, cardiac catheterization used to evaluate his symptoms revealed a large, old, well-healed aortic dissection that precluded study of his coronary anatomy. The study also revealed that he had aortic and mitral insufficiency and left ventricular dysfunction. Medical treatment was initiated and, because of the “bad heart syndrome,” he was advised to “take it easy.”

When we first visited with this physician, his cardiac status was stable at rest. His findings were aortic and mitral insufficiency, a “scary” chest x-ray with evidence of the old aortic dissection, and significant cardiomegaly. The short walk from the hospital parking lot exhausted him—let
alone making rounds to see his patients. He no longer performed surgical procedures. The trips he and his wife had previously enjoyed were now fraught with hazard because of the danger of his becoming depressed due to his diminished ability to keep pace with her on such previously enjoyable occasions. He had heard of our exercise successes and was willing to take a chance with us. After a lot of discussion and coercion on his part, I became willing to turn my head to the obvious concerns and began experimenting with the process of exercise in this perceived very-high-risk physician patient.

The treadmill peak metabolic equivalent (MET) work and symptoms versus weeks in cardiac rehabilitation (Figure 1) show serial exercise tests administered during this patient’s 12 weeks of exercise rehabilitation in our cardiac rehabilitation program. The y-axis measures physical work in METs, which will be discussed more thoroughly later in this paper. For now, it is only important to realize that 1 MET of work is all that is required to do nothing. Just prior to beginning his rehabilitation, this physician’s maximum MET working capacity, as measured by treadmill stress testing, was almost 4. By the way, 4 is the maximum MET work required to perform all our usual daily living activities. Notice that his symptomatology, including shortness of breath and chest tightness, began to occur at about 75% of his maximum MET working capacity. These data fit his described symptomatology—it took much less physical work for him to become symptomatic than it would for someone with a healthy heart who performed the same activities.

At about 7 weeks into the rehabilitation process, another exercise test was performed. In just 6?2 weeks, his maximum MET working capacity increased significantly. Now, and most importantly, considerably more physical work was required by him to provoke his symptoms, which again occurred at about 75% of his maximum capacity. His improvement trend continued throughout his 12-week participation in the cardiac rehabilitation program.

The typical exercise prescription designed from the first graded exercise test (GXT) (Figure 2) demonstrates what is required to effect improvement during the first 6 weeks. Notice that following a 2-MET warm-up for several minutes, we used interval work in the 3-MET range for a total of almost 40 minutes. This work was just below the threshold that would have provoked his symptomatology. Notice also that the heart work or heart rate (HR) response was about 75% of the maximum achieved HR on the exercise test.

Thus, the exercise portion of rehabilitation allowed 2 important changes to occur for this patient (see Figure 2). First, it allowed the training required to improve functional capacity at a nearly normal, everyday-living range (up to 4 METs of work) to be performed without symptoms. Secondly, it provided documentation of the performance safety of such levels of activity and clear countering of the words “take it easy.” Furthermore, the group therapy portion of cardiac rehabilitation provided him reassurance, and this restored his confidence as well as assured us that, with a dimension of reasonable certainty, he could return to normal living some 6 years after his being diagnosed with serious heart illness. Although no change occurred in his cardiac status, he experienced waning symptoms of depression, returned to active medical practice including surgery, and again enjoyed worldwide travel with his wife. In turn, his wife was reassured and became more confident that it was safe for him to assume this new level of activity.

The first step in exercise prescription is determining the safety of the activity that requires more than the minimum physical work. No work requires only “basal metabolism,” or about 3.5 mL
O\textsubscript{2}/kg/minute, also known as 1 MET. Any work more intense than that requires multiples of this basal unit. For instance, to complete the first stage of a modified Bruce treadmill protocol requires use of about 7 mL O\textsubscript{2}/kg/minute, or about 2 METs of work. To complete the first stage of a Bruce treadmill protocol requires one to use about 17.7 mL O\textsubscript{2}/kg/minute, or perform about 5 METs of work. To complete stage V of a Bruce treadmill protocol requires about 52 mL O\textsubscript{2}/kg/minute, or about 15 METs of work.

Tables are available that allow for the prediction of the MET work required for any job, leisure, recreational, or sporting physical activity. Taking a shower, mowing the lawn, downhill skiing, sexual intercourse, tennis, driving—they all require physical work and oxygen uptake, delivery, and use. And, they all have a defined MET work.

To determine safety, we have to assure that the heart’s response to exercise is as normal as we can make it. By this time in the patients’ illness, using medication, surgery, or tools used in interventional cardiology, we have provided and are satisfied with the control we and the patients have over their “resting” hearts, i.e., the myocardial demands required for the patient to do nothing, or maybe even to “take it easy.” During exercise (enjoyment of life), we have to be sure that the heart is free from advanced arrhythmia and free of ischemia and thus can provide enough cardiac output to accomplish the task. Then, we have to assure the exercise does not provoke adverse symptoms or signs. An objective method to determine this safety is exercise stress testing.

So, now the questions focus on the types of tests one should order—a submaximal exercise test, a postmyocardial infarction (MI) exercise test, a submaximal Bruce test, a maximal Bruce test, a submaximal modified Bruce test, or perhaps a Naughton protocol exercise test? Is swimming maximal or submaximal exercise? Is mowing the lawn maximal or submaximal? What about driving a car—is that maximal or submaximal? Is the “take it easy” caveat a definition of submaximal exercise, or does a further subset of submaximal exercise exist that allows one to use that term when talking with a patient? I have always felt that the answer to these questions requires determining the patient’s maximal or symptom-limited exercise capabilities in order to define submaximal body work.

To begin understanding maximal versus submaximal exercise, I use data from our experiences with the treadmill. A tremendous amount of emotional energy is expended when either a physician or a heart patient considers using a treadmill for diagnostic testing, exercise training, or recreational walking. I have calculated the average walking speed of the typical, hospitalized, post-MI, postoperative, or postinterventional stroller to be about 1.5 miles per hour (mph). While making rounds, I am very comfortable observing unattended patients strolling at 1.5 mph in the halls of Baylor University Medical Center’s acute care “heart” floors. But when they walk on the treadmill at 1.5 mph in the noninvasive laboratory in the Heart Center, it seems to be a totally different story. In that setting, the exact same physical work (1.5 mph on a treadmill) is perceived as a potential danger, and thus the patient is surrounded by medical personnel and the equipment necessary to offer advanced life support if required.

The modified Bruce treadmill protocol (Table) shows the zero stage during which no elevation is applied to the treadmill, and its speed is 1.5 mph. I used this protocol when another physician friend agreed to an evaluation by me. During the test, he elevated his heart rate to 162, with a normal systolic blood pressure response, at which time we stopped his test due to fatigue. We continuously monitored his expired air and measured oxygen uptake during this evaluation, and
we were able to determine in terms of oxygen required the precise amount of physical work he could perform—14 times the work required to read this paper, or 14 METs of work. We could have measured the work in terms of horsepower, watts, foot pounds, or calories expended, but MET work has become the more standard comparable unit in exercise physiology.

<table>
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<th>Table. Modified Bruce treadmill protocol, volunteer physician</th>
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<td>Time (min)</td>
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METs = metabolic equivalents; HR = heart rate; BP = blood pressure

**Figure 3** shows the physician subject’s gas exchange during exercise. Remembering that more physical work is required by further increasing the treadmill speed and elevation every 3 minutes, notice his oxygen use (uptake) that is measured by sampling his expired oxygen. A steady, gradual increase in oxygen requirement is necessary because the muscles providing the work, namely the skeletal muscles of his legs, are primarily dependent on aerobic mechanisms (using oxygen to generate adenosine triphosphate [ATP]). A point in time occurs, however, when no increase in oxygen use can occur no matter how much the speed and elevation of the treadmill are increased. At this point, the subject has reached his VO₂ max (maximal oxygen consumption), and a leveling off of his oxygen use occurs—his heart can offer no higher flow and the skeletal muscle is at its maximum capability of oxygen extraction.

Now, let’s focus attention on CO₂ production that is also readily measured by sampling his expired air. On the way to reaching VO₂ max, products of the Krebs cycle, which are predominantly CO₂ and water, are released locally in the muscle. As long as enough oxygen is provided by an increase in cardiac output and in muscle extraction of oxygen, the ratio of these gases, CO₂ and O₂, known as the respiratory quotient (RQ), is balanced, with an RQ of <1. Somewhere around 60% to 80% of the VO₂ max in both physician subjects (the well physician and the earlier-cited sick one), leg skeletal muscle demand for energy could not be met fully by oxygen delivery. Thus, local anaerobic metabolism began providing supplemental ATP for muscle work, and by-products of lactate and lactic acid accumulated locally and required further buffering, thus producing more CO₂ and water. At this point, more CO₂ was recognized in the measurement of expired gas, the RQ exceeded 1, and local discomfort in the legs and general fatigue began. In other words, both CO₂ production and CO₂ removal (ventilation) increased out of proportion to oxygen use.

Now we have defined maximal exercise in 2 different ways: 1) we have noticed that leveling off of VO₂ occurs when maximal exercise is performed, and 2) we have also noticed that the RQ becomes >1.
What is the physician’s heart doing during this activity? Two readily available parameters allow us to observe cardiac work: HR and systolic blood pressure. They help define myocardial oxygen demand or consumption. Notice that in Figure 4, the blood pressure response is appropriate. The systolic blood pressure increases and the diastolic blood pressure remains the same or decreases somewhat. The HR also increases gradually during exercise. And when (as described above) maximal exercise is reached and the leveling off of VO2 occurs, leveling off of HR also is observed. When the skeletal muscles’ requirement for oxygen increases, the demand of the heart increases. In response, the demand for an increased cardiac output is met in 2 ways: HR and stroke volume increase. Yet, here also, a point is reached when no matter how much more work is required, a limit to the increase in the heart’s rate exists and a leveling off of the HR occurs—constituting a third observation in defining maximal exercise.

Through subjective use of the Borg Scale, a 1 (less than “very, very light”) to 20 (greater than “very, very hard”) scale of perception of physical work, it is possible to determine a patient’s perception of maximal work. As expected from the objective measurements, it was perceived to be “very, very hard work,” with a rate of perceived exertion (RPE) of 19.

An exercise test using a modified Bruce treadmill protocol (see Table) lets us determine the maximum body work this physician could accomplish. The ability of his system to provide oxygen to working skeletal muscle was determined by measuring his VO2, and how much heart work he could perform was determined by measuring his heart rate and, as another major determinant of myocardial work, his systolic blood pressure. The exercise test along with perceived exertion allows 4 observations to determine whether patients are performing maximum exercise:

1. An RPE of 19 to 20;
2. A leveling off of VO2;
3. An RQ >1.0 during exercise; and
4. A leveling off of HR as work intensity increases.

What, then, is submaximal exercise? Well, it is less than maximum exercise. As defined by HR, it is <90% of maximum predicted HR. Whether or not you order a maximum stress test, if you send a patient to the laboratory on beta blockade, that patient will demonstrate a submaximal exercise evaluation if determined only by the heart rate definition. By using VO2 measurement, on the other hand, submaximal exercise is just not maximal exercise. By determining RQ, submaximal exercise is activity performance causing sustained use of aerobic metabolism of skeletal muscle and not necessarily causing an RQ >1. Maybe that is the definition of “take it easy.”

Now, remember the Fick formula from physiology (Figure 5) that allows us to measure the cardiac output. A simple rearrangement of the formula (Figure 6) gives us some appreciation of the interdependence of these parameters. VO2 is a measure of “body work”; HR and stroke volume, a measure of “heart work”; and the arterial-venous O2 difference, a measure of “skeletal muscle work.”

With this introduction to exercise rehabilitation, I hope it becomes easier to begin reviewing both exercise physiology and the usefulness of exercise in the treatment, care, and rehabilitation
of cardiac patients. The following are but a few of the questions that need to be answered before embarking on the development of the exercise portion of cardiac rehabilitation.

- What are the best intensity and frequency for exercising the cardiac patient?
- Can the VO2 max improve (or what is the training effect)?
- What is an anaerobic or ventilatory threshold?
- How does anaerobic threshold relate to ischemia?
- What is the relation between left ventricular ejection fraction and VO2 max?
- Are the activities I am prescribing or the activities I am allowing patients to return to safe for their enjoyment?

Finally, when people (patients, physicians, managed care organizations) struggle with “who needs cardiac rehabilitation” (or exercise rehabilitation with telemetry, both known best by CPT 93798), I try to control myself and offer the following. It has been clearly documented that exercise rehabilitation in the post-MI population reduces mortality up to 20%. Guidelines for participation and determination of risk are available from the American College of Physicians, the American College of Cardiology, the American Heart Association, the American College of Sports Medicine, and the American Association of Cardio-pulmonary Rehabilitation, and clinical practice guidelines regarding cardiac rehabilitation were published in 1995 by the US Department of Health and Human Services.

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References
Cardiac output = \( \frac{V_O_2}{\text{Arterial} - \text{Venous } O_2\text{ difference}} \)

\[ HR \times SV = \frac{V_O_2}{(A-V)} \]

**Figure 5**

\[ HR \times SV \times (A-V \text{ } O_2\text{ difference}) = V_O_2 \]

Heart work  Skeletal muscle work  Body work

**Figure 6**