Limitations of Exercise Reconditioning in COLD*

Richard L. Hughes, M.D., F.C.C.P.; and Richard Davison, M.D.

The progressive improvement in ability to walk without dyspnea suggested that a physiologic response similar to a training program in athletes may have been produced.1

This comment by Alvan Barach in 1951 emphasizes the relative ignorance that existed only three decades ago concerning the "reconditioning" of patients with chronic obstructive lung disease (COLD).

Dr. Barach's proposal to exercise patients with disorders of oxygen transport met with appropriate skepticism in the clinical community. Until well into the 1960s, the standard therapy for patients with COLD was rest and avoidance of stress. The first study to question this dogma directly was published in 1962, when Pierce et al demonstrated what has since become dogma in 1982; reconditioning patients with COLD permits them to perform the same exercise with lower heart rate, respiratory rate, minute ventilation, and CO2 production. These benefits were obtained without change in pulmonary function and the authors wondered whether they were due to increased efficiency of motion, or to enhanced oxygen utilization within exercising muscles. Two years later, Paez et al concluded that efficiency and oxygen utilization were both improved. Christie then demonstrated that these changes could be achieved on an out-patient basis with relatively little supervision.

The past 15 years have witnessed a virtual explosion in the investigation and popularity of exercise therapy. Many patients with COLD are now automatically placed in a pulmonary rehabilitation program, or encouraged to recondition themselves with judicious activity. A number of recent reviews have addressed the specific goals and techniques of exercise reconditioning and its proposed benefits.4-9 This review will concentrate on the limitations and uncertainties of this mode of therapy.

PROPOSED BENEFITS

Rehabilitate (L. habilitare: to make suitable); to restore
Recondition (L. condicio: to speak together); to put back together.

The modern use of "recondition" implies a limited aspect (usually functional) of a more comprehensive effort to "rehabilitate" patients. There is no doubt that exercise reconditioning (EXRE) improves exercise performance. What remains largely unknown is how this is accomplished. Despite two decades of study (and a wealth of equipment), the critical adjustments that permit COLD patients to exercise longer following reconditioning are not known. One problem is the inability of most patients to achieve a work rate much different from rest. COLD patients, especially those unfamiliar with exercise laboratories, usually stop exercising before they reach their limits of oxygen transport. A second problem is the almost universal selection of patients by degree of airway obstruction. The natural history of COLD is much too varied to expect that pulmonary functions characterize uniform subsets of patients. Duration of illness, lean body mass, broncho-reactivity, ventricular function, previous athletic experiences, familiarity with the laboratory and a host of other factors, impinge upon an individual's exercise performance. These variables make it unlikely that exercise laboratories will ever achieve comparable results unless a more appropriate index of effort tolerance is found. A good example of this problem is provided by two studies of the A-V oxygen difference during EXRE in patients with COLD.10,11

In the first, which concluded that the A-V difference widened with training, one third of the patients contributed two thirds of the increase in the mean A-V difference.10 In the second study, which concluded just the opposite, the size of the standard deviation indicates that a similar range of responses was present.11 Both groups trained their patients on a
Finally, the pulmonary pump itself, which also serves to remove carbon dioxide and deliver oxygen, is the last element of the cardiovascular system. It is well known that the lung function is critical to the maintenance of life, and any impairment of this process can result in serious health problems. Therefore, understanding the factors that affect lung function is crucial for improving patient outcomes.

In conclusion, the respiratory and cardiovascular systems are interconnected and work together to maintain homeostasis. Any disturbance in one system can affect the other, highlighting the importance of a holistic approach to respiratory care.

Table 1 — Potential Benefits from Exercise Reconditioning

<table>
<thead>
<tr>
<th>Accepted benefits for COLD patients²⁴,²⁵,²⁶,²⁷,²⁸,²⁹,³⁰,³¹,³²,³³</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased endurance</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Increased maximum oxygen consumption</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Increased skill in performance, decreased ventilation, oxygen consumption, heart rate and increased anaerobic threshold</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Possible benefits (usual in COLD patients)</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Increased sense of well-being</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Increased mucociliary clearance</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Increased hypoxic drive</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Increased left ventricular function</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Unlikely, debated or unknown benefits</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Improved survival</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Improved pulmonary function tests</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Lowered pulmonary artery pressure</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Improved blood gases</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Improved blood lipids</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Change in muscle O₂ extraction</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Change in step desaturation or apnea</td>
<td>Yes</td>
<td>No</td>
</tr>
</tbody>
</table>

Table 2 — Possible Effects of COLD on Cardiac Function

<table>
<thead>
<tr>
<th>Description</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decreased RV function</td>
<td>32-36</td>
<td>—</td>
</tr>
<tr>
<td>Abnormal RV response to stress/exercise</td>
<td>33,37,38</td>
<td>—</td>
</tr>
<tr>
<td>LV hypertrophy</td>
<td>39,40</td>
<td>41,42</td>
</tr>
<tr>
<td>Decreased LV function at rest</td>
<td>35,39,43</td>
<td>33-36,42,44</td>
</tr>
<tr>
<td>Abnormal LV response to stress/exercise</td>
<td>37,38,43</td>
<td>33,34,44</td>
</tr>
<tr>
<td>Improved function with O₂</td>
<td>33</td>
<td>47</td>
</tr>
<tr>
<td>Normal coronaries</td>
<td>39</td>
<td>35,42</td>
</tr>
<tr>
<td>Increased arrhythmia</td>
<td>48-50</td>
<td>—</td>
</tr>
</tbody>
</table>

*Conclusions based on separate studies. Numbers refer to references which concluded that the described condition was present (YES) or absent (NO). RV, LV = right and left ventricular. In reference 33, decreased LV function present in severe, but not mild COLD.

Limitations of Exercise Reconditioning in COLD (Hughes, Davisson)
techniques, several investigators\textsuperscript{30,31} have demonstrated that cardiac output increases appropriately during submaximal exercise, but that this is accomplished via an increase in heart rate with little or no change in stroke volume. This limited cardiac reserve is only exceptionally due to "primary" failure of the left ventricle.\textsuperscript{30,31,32,33} More recently, increased interest has developed in the effects that large changes in negative and positive intrapleural pressures have on ventricular function.\textsuperscript{33,34} A shift of the interventricular septum with a resulting reduction in the end-diastolic volume of the left ventricle could play an important role in the observed impairment of cardiac function.\textsuperscript{33,34} Accurate, serial measurements of right and left ventricular function can now be obtained during exercise with noninvasive techniques in patients with severe airway obstruction.\textsuperscript{35} The little-remembered observation of Nahkjian et al\textsuperscript{35} that inferior vena cava blood flow can cease completely during inspiration in COLD patients suggests that not all of the factors affecting cardiac output in this disease have been identified.

The best recent review of the factors influencing normal cardiac function during exercise is the Second Harry Moss Symposium held in 1979.\textsuperscript{36} The major contribution of this symposium was to define lucidly the similarities and differences between static (isometric) and dynamic (isotonic) exercise in normal subjects. The concluding chapter should be read by everyone interested in improving exercise tolerance in patients or normal subjects. With certain reconditioning programs, normal subjects will increase their cardiac mass, the number of capillaries perfusing muscle, and the percentage of high density lipoproteins in their serum.\textsuperscript{37,38,39,40} However, the intensity requirements of these programs make it highly unlikely that COLD patients will achieve any of these benefits.

Muscle Sump

With current data, it is not possible to answer the critical question of whether EXRE affects the extraction of oxygen by muscle during activity. The limited working capacity of most COLD patients probably prevents them from ever exercising long enough to achieve a "training effect" in skeletal muscles (increased capillary density, muscle fiber size, metabolites and mitochondria).\textsuperscript{9,12,20} Belman et al\textsuperscript{9} have demonstrated that training patients with COLD did not increase three of the many skeletal muscle enzymes responsible for delivering ATP to the myofibrils. Although the patients in this study worked at rates approximately 70 percent of maximum, their absolute work rates were almost certainly inadequate to expect any metabolic training effect.\textsuperscript{9} The major problems with attempting to measure the effects of EXRE on muscle fuels are the rapid changes that occur before complete freezing of biopsy specimen\textsuperscript{42} and the sequestration of muscle metabolites within subcellular compartments.\textsuperscript{50,51} These features limit the usefulness of needle biopsies of muscle, which require 3-10 seconds for complete freezing, and homogenization for analysis of metabolites. The advent of topical nuclear magnetic resonance should reduce this problem somewhat.\textsuperscript{43} One fuel which is probably not compartmentalized, and which almost certainly plays a role in muscle fatigue and reconditioning, is glycogen.\textsuperscript{53,54} In normal subjects, muscle glycogen is virtually absent at exhaustion, and in animals cannot be replenished without adequate refeeding.\textsuperscript{43} Unpublished data from our laboratory suggest that skeletal and respiratory muscle glycogen are not affected by the nutritional status of patients with mild or moderate COLD. Techniques have now been developed that will permit the analysis of various EXRE programs on single fiber fuel depletion and restoration.\textsuperscript{55} At the present time, it does not appear that the efficiency with which normal subjects utilize energy in their contracting muscles (thereby decreasing oxygen "consumption"), changes significantly with training. It is unlikely that COLD patients differ in this respect. It should be remembered that a short period of inactivity, such as an acute illness or hospitalization, may completely reverse whatever training effects are accomplished in COLD patients.\textsuperscript{56}

Downstream from exercising muscles, very little is known concerning the responses of COLD patients. Although mixed venous Po$_2$ (PvO$_2$) has often been considered a sensitive barometer of the adequacy of tissue oxygenation, it is apparent from the work of Mitchofer\textsuperscript{56,57} and others\textsuperscript{56,73} that resting PvO$_2$ does not completely describe the utilization of oxygen in patients with lung disease. At least three factors which vary widely in COLD patients (hemoglobin, capillary density and oxygen disassociation) affect oxygen utilization by the tissues, independently of driving pressure. Also, the level of PvO$_2$ does not always determine which patient with COLD will develop arterial desaturation with exercise,\textsuperscript{74} but does sometimes reflect the amount of oxygen still available for extraction.\textsuperscript{71}

Potential Hazards

A major purpose of this review is to emphasize the uncertainties that exist regarding the longterm hazards of EXRE in COLD patients, and indeed all older subjects. Although it is agreed that "man was made for exercise,"	extsuperscript{18} it is also apparent that aging man must temper his activity to avoid functional or structural injury to a variety of tissues, particularly the cardiac and respiratory muscles of patients who happen to also have COLD.

Cardiac arrhythmias have been found in a majority of COLD patients receiving longterm electrocardio-
graphic monitoring, yet their relationship with sudden death in this population remains inferential. The development of life-threatening arrhythmias during EXRE in these patients has not been documented in the medical literature. This could be related to the common practice of providing supplemental oxygen during exercise, since oxygen therapy has been shown to decrease ectopic activity. 

Less appreciated is the potential for significant systemic hypotension which may appear in patients with more severe disease. Arterial desaturation is a common occurrence in some types of activity, both in normals and patients. Somewhat paradoxically, desaturation during the early phases of exercise may be diminished in COLD patients, probably because of a slower rate of increase in oxygen utilization by exercising muscles. Mild to moderate increases in Pco₂ during exercise are possibly more common than hypoxemia, perhaps because exercise cannot overcome the CO₂ hyporesponsiveness that exists in patients with airway obstruction. The longterm effect of these transient blood gas abnormalities during exercise on the control of breathing, pulmonary pressure and muscle function is unknown. Whether the mechanical stress placed on the lungs by exercise worsens morphologic emphysema is debated and remains unstudied in humans. Exercise-induced asthma, a common problem in younger patients, is a rare complication of EXRE in COLD patients, probably because they cannot generate a large enough ventilation to cool the trachea. Similarly, it is unlikely that such patients will be at risk for hypoglycemia or exercise-induced muscle contracture.

What may prove to be the most controversial aspect of EXRE is the question of how much stress to place on the muscles themselves. Can a disadvantaged diaphragm be exercised without tipping it into progressive fatigue and potential respiratory failure? Does exercise cause the right ventricle to fail early, as suggested by animal studies? Although the reports of improved effort tolerance that result from protocols designed to improve respiratory muscle strength and endurance are encouraging, the limits to this approach have not yet been defined. It should be remembered that one type of dyspnea results from a failing diaphragm. When the diaphragm has failed "too much," or its function is complicated by cardiac failure, the patient may require mechanical ventilation and rest to return his respiratory muscles to some semblance of normal function. It behooves us to consider these factors when prescribing EXRE programs for the spectrum of patients with COLD. In summary, although the long-term consequences of exercise in COLD patients remain undefined, properly supervised EXRE appears to carry little immediate risk when tailored to the patient's impairments.

**Adjuncts**

EXRE can be considered a major treatment modality in the management of COLD patients. Currently, there are five adjuncts that can improve the usefulness of EXRE: oxygen, other drugs, psychologic defenses, nutrition, and specific respiratory muscle training. All of these adjuncts, including the use of oxygen, remain experimental in some aspects of their use.

**Oxygen**

Oxygen is an expensive drug. Its use in COLD is completely analogous to dialysis for end-stage renal disease. Oxygen unequivocally prolongs survival in many patients, especially women who are eucapnic, or eucythemic. What is not defined is when oxygen should be started, either in COLD management or in EXRE. One pragmatic approach would be to use oxygen for all patients who retain CO₂ during acute exercise. It seems clear that in some patients there is a late period in the natural history of COLD in which oxygen helps symptoms and oxygen delivery, but does not prolong survival. It is reasonable to assume that this difference in mortality between patient groups has something to do with the sequence of responses to hypoxemia in COLD.

Oxygen during exercise relieves dyspnea and improves endurance. This benefit is frequent, but not universal, and it cannot be predicted on the basis of symptoms, hypoxemia, hypercarbia or acidosis during exercise. It is probably not due to improvement in pulmonary hypertension, but this may be a function of how oxygen is administered, since intermittent hypoxia-normoxia may be as deleterious as persistent hypoxia.

It should be remembered that oxygen alone can improve exercise tolerance without formal EXRE, and that predosing with oxygen before exercise can produce the same improvement in effort tolerance as oxygen during exercise. How oxygen improves endurance, whether it affects maximum oxygen consumption, and whether it should be used in normoxic patients continues to be debated. It appears to relieve large airway bronchoconstriction, but probably does not alter the hypoxic drive, at least during rest. In the face of inspiratory resistance, short-term hypoxia will significantly reduce the endurance of respiratory muscles.

**Other Drugs**

A number of drugs affect exercise tolerance. Which will prove to be genuinely useful adjuncts to EXRE awaits further study. The ubiquitous effects of theophylline make it a prime candidate for improving effort tolerance. Beta, bronchodilators before exercise are a widely used and simple means for the short-term improvement of effort tolerance. It now appears that
man (at least normal man) has resting vagal tone throughout his airways, including terminal lung units, and that some patients will respond to vagal blockade, but not to beta agonists. Most clinicians recognize that the relief of dyspnea does not always require improved air flow. The reverse is also true; decreased air flow may not influence effort tolerance. In fact, opiates and alcohol, which probably do not influence airway resistance, have profound effects on effort dyspnea. Finally, the pharmacologic manipulation of pulmonary hypertension and breathing pattern may prove to be of substantial benefit during EXRE.

Psychological Defenses

The best and virtually only review of this poorly understood aspect of COLD is Dudley's three-part series published in 1980. In the relatively few studies available, there is unanimity that improving the patient's ability to cope with his illness is vital to a successful rehabilitation program. Baum's data that improvement in effort tolerance may be more closely related to psychosocial health than to the degree of airway obstruction need confirmation by other groups. Whether formal psychotherapy will prove to be an effective addition to an EXRE program is quite unknown. It is likely that exercise itself improves psychological attitudes in patients, perhaps even more so than direct psychotherapy.

Nutrition

The physiognomy of emphysema is characteristic. . . when emaciation has occurred, the appearance alters. The lines in the forehead are now deep, the brows knit, the nasolabial folds distinct, the expression careworn.

Progressive weight loss was recognized as a terminal phase of COLD, even in the 19th century. The reasons for weight loss are complex and not a simple function of airway obstruction, dyspnea or gastrointestinal symptoms. The most comprehensive review of this subject is provided by Askana et al. Chronic undernourishment has a number of direct adverse effects on lung and respiratory muscle function, including vital capacity, surfactant metabolism and lung defense mechanisms. There is good evidence that starvation accelerates emphysema in both man and animals, but it is not known to what degree this feature influences the natural history of COLD. The most obvious effects of chronic undernourishment are loss of muscle protein and strength. Activity with weakened limb muscles produces greater demands on ventilation at a time when the respiratory muscles are probably equally weakened, resulting in a cycle of increased symptoms and decreased activity. However, in some severely symptomatic patients, this adverse cycle between nutrition and muscle function may be reversible. It should be remembered that exercise in inadequately nourished patients may deplete muscle proteins rather than increase them.

There has been a recent flurry of interest in the possible adverse effects of carbohydrates on patients with COLD. Glucose metabolism produces more CO\textsubscript{2} per mol than fat. If patients have a fixed ventilation (eg, controlled mechanical ventilation), acute hypercarbia can result from parenteral nutrition. Similar changes would be expected in patients who have reduced responses to CO\textsubscript{2}, a feature of some malnourished patients, but not others. However, if the CO\textsubscript{2} drive is intact, carbohydrate loads should increase ventilation and alveolar Po\textsubscript{2}, while reducing the demand for oxygen by approximately 10 percent. Whether these changes apply to exercising patients is not known—most will probably tolerate carbohydrate loads without difficulty. Both adrenal and testicular hormonal changes occur in COLD, and may explain why some patients lose weight despite overeating. The current state of knowledge, which is limited, suggests that nutrition and exercise tolerance have complementary effects on each other. However, it is premature to recommend specific nutritional supplements in an attempt to manipulate EXRE.

Respiratory Muscle Exercise

The adjunct that promises to bring the greatest benefit to patients is attempting to improve respiratory muscle function. The best reviews of this area are by Rochester et al., Edwards and Macklem and his group. Edwards has beautifully condensed the various hypotheses regarding muscle fatigue into a three-dimensional model that relates the loss of muscle force to energy stores and excitation/activation. Although one might expect respiratory muscles to hypertrophy with increasing work of breathing (as they appear to do in selected patients and animals), the majority of patients demonstrate increased fatigability and atrophy. Inspiratory muscle fatigue appears to be one factor that limits exercise tolerance in COLD. These abnormalities may be present in patients with mild-to-moderate disease. The reasons for this seemingly paradoxical response are not completely defined, but are probably related in part to hyperinflation, malnutrition and loss of sarcomeres within the muscle itself. Under normal conditions, the diaphragm is quite resistant to fatigue, and can use other inspiratory muscles to "rest itself" during prolonged stress. However, the inspiratory muscles of patients with COLD appear unusually compromised, perhaps because of working under chronic hypoxia. Although the traditional explanation for CO\textsubscript{2} retention in these patients has been increased airway obstruction, this view is probably too simplistic. Eucapnic and hypercapnic patients can also be distin-
guished by their tidal volumes, duration of inspiration and ventilatory response to exercise. A better index of the stress placed on inspiratory muscles during EXRE may be the percentage of maximum transdiaphragmatic pressure used to perform each tidal breath, and the fraction of time spent on each inspiration.

The sentinel study of Leith and Bradley catalyzed a series of investigations which have defined some of the limits of respiratory muscle conditioning. Inspiratory resistive breathing increases tidal volume, decreases respiratory rate and improves effort tolerance. Respiratory muscle endurance training increases the aerobic capacity of respiratory muscles, but deconditioning occurs rapidly in normal subjects. The same type of training increases exercise capacity in COLD, but the reverse may not be true. Several words of caution are in order regarding respiratory muscle exercise: (1) all of these studies suffer from inadequate characterization of patients, so results cannot be extrapolated to everyone labeled with the diagnosis of COLD; (2) in some patients, simple breathing retraining may accomplish the same goals as the more stressful endurance training used by some authors; (3) some patients will deteriorate because of the increased demand on their respiratory muscles. Patients who demonstrate diaphragmatic fatigue at rest may profit far more from assisted ventilation than from attempts to strengthen respiratory muscles. Dyspnea at rest, or with minimal activity, may also be a signal of diaphragmatic fatigue. One form of muscle fatigue, the so-called low frequency or anerobic fatigue, may take several hours and perhaps as long as a day to return a muscle to normal responsiveness. Since most clinicians do not yet have access to measurements that reliably estimate respiratory muscle fatigue, it behooves us to select our patients carefully, begin respiratory muscle training slowly, and make judgments based upon symptomatic improvement. Finally, the factors influencing diaphragmatic, intercostal and abdominal muscle tone may prove to be an equally fertile field for improving respiratory muscle function.

**Summary**

EXRE is a powerful tool for improving the symptoms of patients with COLD. As clinicians, we are hampered by not knowing precisely how it works, and knowing virtually nothing of its longer term adverse effects, especially on the heart, pulmonary circulation, respiratory muscles and joints. Guidelines for the use of EXRE must therefore be governed by our clinical sensibilities, as well as the available data. It is possible that those factors listed as "adjuncts" in this review, especially drugs, nutrition and respiratory muscle training, will prove more valuable in reducing symptoms and prolonging the present "natural" history of this disease, than will specific EXRE. We still do not know when to start EXRE or its adjuncts to obtain optimal benefit. There is a point in the natural history of COLD beyond which patients should not have EXRE routinely prescribed. The almost instinctive reaction of clinicians to judge the progress of COLD by the FEV1, is probably outdated. Sleep patterns, lean body mass, transdiaphragmatic pressure and RV function during exercise may prove to be more sensitive predictors of decline, and permit more effective intervention.

For the present, we can approach EXRE prescriptions for COLD patients in the same way as for normal subjects of similar age, modifying the intensity and goals to the patient's symptoms and to the estimated impairment of oxygen transport. Except for pulmonary pressure, there are no data to suggest that patients with COLD have some unique response to exercise that separates them from sedentary, unfit normal subjects. Patients with early COLD will differ little from normal. Patients with more advanced disease will be symptom-limited, and should have EXRE which attempts to improve the performance of specific tasks. Arterial desaturation should always be measured in the laboratory in patients with more advanced disease to determine the need for supplemental oxygen. The current pessimism regarding the cardiac benefits of EXRE appears justified as long as we remain largely ignorant of the maladaptations which lead to pulmonary hypertension, and patients remain unable to engage in intense exercise. EXRE per se probably does not accelerate (or delay) the development of pulmonary hypertension in patients with early or moderate disease. Patients with cor pulmonale, failing left ventricle, lean body weight loss or dysynchronous thoracoabdominal motion should be exercised very cautiously or not at all. Data from Norma Braun's laboratory (personal communication) support resting such patients at night for three to six months by nocturnal negative pressure ventilation, before considering oxygen-supplemented, task-specific EXRE. Animal data suggest that specific attempts to restretch, re nourish and increase the endurance of the diaphragm may be beneficial.

There are other causes for optimism. A number of patients have reported improvement in dyspnea during water immersion, and swimming may prove to be an ideal mode of EXRE in this population. The behavior of isolated human muscle is being better defined, which may permit mechanical or nutritional manipulation of its energetics. Although the degree of exercise performed by most patients with moderate or advanced disease is certainly too little to produce fatigue-resistant properties in muscle fibers, there may be electro-mechanical means of influencing endurance. Until these studies evolve, the 1964 observa-
tion of Pierce et al* remains largely unqualified:

Most patients... should not only be encouraged to remain physically active to the limits of their tolerance, but should also be started on appropriate physical conditioning programs, depending on their range of disability.

REFERENCES

31. Stubbing DG, Pengelly LD, Morse JLC, Jones NL. Pulmonary mechan-