A Brief History of Pulmonary Rehabilitation

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Pulmonary rehabilitation is widely accepted as effective therapy for patients with chronic obstructive pulmonary disease (COPD). This paper presents a brief (and somewhat subjective) history of pulmonary rehabilitation, and stresses the development of the exercise component. Until the middle of the 20th century, patients with COPD were advised to avoid the dyspnea that activity brings. Barach can be credited with positing that patients with COPD should strive to be more active. In the 1960s Petty created the multi-disciplinary team that was found to be effective in delivering pulmonary rehabilitation. In the 1980s doubts surfaced as to the ability of rehabilitative exercise to improve muscle function in COPD, but in the 1990s studies showed that well-designed exercise programs caused beneficial physiologic adaptations. The current decade has yielded studies that exploited those insights to design interventions that boost the effectiveness of rehabilitative exercise.
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Introduction

Pulmonary rehabilitation is a therapy with well-documented benefits that might reasonably be considered a standard of care for patients with debilitating chronic obstructive pulmonary disease (COPD). A good number of guideline and review documents have been published in recent years.[1-3] It is a bit surprising that little attention has been paid to remembering the roots of pulmonary rehabilitation and tracking the course by which we arrived at this point. The history I present below is not intended to be exhaustive; it is highly personal and focuses mainly on the exercise component of pulmonary rehabilitation (arguably its most important component). I encourage others to compose a more detailed and less subjective review.

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History of Pulmonary Rehabilitation

Alvan L Barach MD

Though COPD was not nearly as common as it is today, by the middle of the last century it was clearly a major health problem. The common wisdom was that, since dys-

Fig. 1. Alvan L Barach MD, a pioneer in pulmonary medicine. (From Reference 4, with permission.)

pnea on exertion was a major troubling symptom, avoiding dyspnea was appropriate disease management. Patients were advised to avoid activities that led to dyspnea, much as we would today advise patients with coronary artery disease to avoid activities that lead to angina. A contrary opinion was offered by Barach, who, starting in the 1950s, offered important insights (Fig. 1). Barach was a physician affiliated with Columbia University in New York. He had a long career, spanning the 1920s through the 1970s. He published prolifically; the PubMed database lists 128 publications whose indexing survives to our time. We must stand in awe of his earlier work: in 1926 he published a description of the oxygen tent that reduced respiratory distress of those critically ill with pneumonia.[5] In 1936 he described the use of heliox for relief of dyspnea in asthma and emphysema.[6] In 1945 he published an early description of the benefits of penicillin in treating pneumonia.[7] In the 1950s he worked on developing portable oxygen systems for patients with emphysema,[8] including, remarkably, a 1-kg cane that contained an oxygen cylinder. He was active late in life and, amazingly, published 6 papers in the year of his death (at 82 years of age).

Barach had wide-ranging interests. He was a confidant, if not a member, of the Algonquin Round Table, the group that was the intellectual focus of Manhattan society in the 1920s. His obituary in *The New York Times* gives a quotation ascribed to him: "Remember to cure the patient as well as the disease." I particularly enjoy his quotation, "An alcoholic has been lightly defined as a man who drinks more than his own doctor."

To the subject of this review, Barach appreciated the plight of his patients suffering from emphysema and worked to lessen their burden. To quote from a paper he wrote in 1952:

> In 2 patients with pulmonary emphysema in whom dyspnea on exertion was relieved during inhalation of oxygen, an exercise program was instituted with subsequent marked improvement of capacity to exercise without oxygen. . . The progressive improvement in ability to walk without dyspnea suggested that a physiological response similar to a training program in athletes may have been produced.[9]

It would be 40 years before a physiological training effect would be demonstrated conclusively in COPD; but Barach clearly understood the concept in 1952. Another lovely quotation taken from a paper a decade later demonstrates his maturing approach:

> When I see a patient, then, whose pulse on walking back and forth 2 or 3 times in the hallway in my office increases to 140, it is evident he hasn't walked enough to maintain cardiovascular efficiency. It may seem unusual perhaps to suggest exercise to these breathless people, but in fact it is one of the ways by which they can restore physical fitness. I am unhappy about patients who always use an elevator to go up stairs. I will say that from now on you can practice walking up the stairs breathing oxygen. The muscles in the legs of these people are very often atrophied. The point is that these patients can begin walking 50 to 100 steps the first day and gradually extend the oxygen walking distance to half a mile twice a day. More recently the portable oxygen cylinders have been utilized. In some older people, the old method of using a tank and a 50 foot length of rubber tubing is employed to enable them to walk back and forth.[10]

Barach clearly understood the interventions necessary for the rehabilitation of patients with COPD, but it would await the arrival of another giant in the field to organize what we today recognize as the multidisciplinary, well-organized program of pulmonary rehabilitation.

Thomas L Petty MD

Petty (Fig. 2) might well be identified as the most accomplished pulmonologist of his generation (or, in fact, any generation). Besides his contributions to pulmonary rehabilitation, we can skim the cream of his accomplishments and credit him with establishing the scientific basis for long-term oxygen therapy[11] and with naming and describing what was at first called the "adult respiratory distress syndrome" and which we now call "acute respiratory distress syndrome."[12]

Fig. 2. Thomas L Petty MD, a pioneer in pulmonary medicine. (Photo courtesy of the American Association for Respiratory Care.)

In the late 1960s, Petty, working at the University of Colorado, brought together a team and established a standardized out-patient program of pulmonary rehabilitation. This program offered patients individualized instruction about their disease; taught about bronchial hygiene; utilized breathing retraining, physical reconditioning, and individualized pharmacologic therapy; and employed supplemental oxygen (as indicated, mostly for right-heart failure). By 1969 he had accumulated enough experience to publish the landmark paper "A Comprehensive Care Program for Chronic Airway Obstruction," in which he and his group reported their experience with their first 124 patients.[13] Petty et al judged 94 patients as "better" as a result of the program and documented improved exercise tolerance, reduced hospitalization, and return to gainful employment as specific program benefits. In the years following this demonstration, rehabilitation programs began to be set up on Petty's model. In 1974, the American College of Chest Physicians formulated a definition of pulmonary rehabilitation, and in 1980 the American Thoracic Society issued an official statement in which pulmonary rehabilitation components were described and benefits specified. Importantly, exercise conditioning was defined as an "essential" component.

The Dark Ages

In my view, the 1980s might be viewed as the Dark Ages of pulmonary rehabilitation. There were those who doubted the value of rehabilitative exercise; for a time that view predominated. The argument was that the exercise tolerance of patients with COPD is limited by their lungs and it was pointed out that exercise conditioning does not improve lung function. Further, it was doubted that patients with COPD could exercise to a sufficient intensity to exceed their "critical training threshold," above which improvements in muscle function could be expected. This view was supported by the 1981 publication of a study very much ahead of its time, titled "Exercise Training Fails to Increase Skeletal Muscle Enzymes in Patients With Chronic Obstructive Pulmonary Disease."[14] In that study, Bellman and Kendregan treated patients with an exercise training program but found no biochemical markers of a training adaptation in muscle biopsy specimens obtained before and after the intervention. Interestingly, shortly after its publication the paper was criticized in a letter to the editor,[15] for selecting an exercise intensity too low to expect biochemical adaptations. Nevertheless, the paper was very influential. Later in the 1980s, Bellman posited that mechanisms of improvement in exercise tolerance associated with a rehabilitation program included increased motivation, desensitization to dyspnea, and improved mechanical skill, but did not include improved physiological ability to exercise.[16] In the view of some, this tagged rehabilitation with a "touchy-feely" aura. Clearly, if no improvement in muscle function was expected (or important!), then the way in which the program was conducted mattered little.

Resurgence of Pulmonary Rehabilitation

Ries at the University of California at San Diego initiated an important line of research. He discovered that patients could exercise at "high targets" (ie, higher fractions of their maximum exercise intensity than could healthy subjects).[17] This led to the realization that using the exercise-intensity prescription guidelines used for healthy subjects resulted in training programs that were doomed to failure: the exercise intensity was too low. A second insight, stemming from work at my institution, found that, in healthy subjects, exercise training yielded markedly reduced ventilatory response to a high exercise level.[18] This was traced to a lower level of lactic acid production (a ventilatory stimulus) at a given level of exercise after the training program, as a result of improved aerobic function of the leg muscles.

My research group wondered whether similar results would be possible in patients with COPD, who, it was reasoned, would have better exercise tolerance if their ventilatory demand could be reduced.[19] This concept was tested and reported in a 1991 paper titled "Reduction in Exercise Lactic Acidosis and Ventilation as a Result of Exercise Training in Obstructive Lung Disease."[20] Though Wasserman and I designed this study, the work was (expertly) carried out by Donner and his team in Veruno, Italy—a marvelous collaboration. It was shown that high-intensity rehabilitative exercise training of patients with COPD yielded a lower lactate level at a given level of exercise. Ventilatory response was lower in proportion to the lactate-lowering, and this yielded better exercise tolerance.

This work received some criticism because patients with predominantly moderate disease were studied (average forced expiratory volume in the first second [FEV_1] 56% predicted).[21] A study published several years later examined patients with more severe COPD (average FEV_1 36% predicted) and found physiologic evidence of training adaptations.[22] In 1996, Maltais and his group found increases in aerobic enzyme concentrations in the muscles of ambulation as a result of a high-intensity exercise program.[23] That finding directly challenged Bellman's concept that patients with COPD were unable to improve muscle function through exercise training.[16]

In 1999 a group of 16 experts in muscle biology, COPD, and rehabilitative science were brought together under the auspices of the American Thoracic Society and European Respiratory Society. They produced a 40-page document[24] that reviewed the state of the art in muscle biology, presented evidence that indicated that the muscles of ambulation of patients with COPD do not function well, and summarized available evidence regarding approaches to improve muscle function in COPD. The intention was to provide a road map for future research into strategies to improve muscle function in COPD. The new millennium has, indeed, seen refinements of our understanding of mechanisms of benefit of rehabilitative exercise training. Saey et al, of the Quebec City group, presented convincing evidence that in a substantial fraction of patients with COPD fatigue of the leg muscles rather than inability to increase pulmonary ventilation is the primary limitation in their exercise tolerance.[25] This implies that, in some patients with COPD, improving muscle function through rehabilitation *directly* improves exercise tolerance, rather than through a secondary impact on modulating ventilatory requirement for exercise. Porszasz et al clarified the mechanism by which rehabilitative training reduces dyspnea; at a given level of exercise, the respiratory rate is reduced, which allows more time to exhale, which decreases dynamic hyperinflation.[26]

Because exercise training programs now have a physiologic focus, rational approaches to improve their effectiveness can be examined. Supplemental oxygen,[27] anabolic drugs,[28] and bronchodilator[29] are attractive choices for boosting the effectiveness of rehabilitative exercise training.[30] Other strategies, such as heliox,[31] interval training,[32] and proportional-assist ventilation,[33] have not yet been shown to improve rehabilitation outcomes, but further study seems warranted. Combining these individual components would be expected to yield additive benefits.

Summary

We can see that pulmonary rehabilitation has strong roots; its founders deserve our thanks and our respect. Pulmonary rehabilitation experienced a period during which its benefits were doubted, but it survived and prospered, at least in part because a strong physiologic rationale was developed to support its effectiveness. Pulmonary rehabilitation can be argued to improve dyspnea, exercise tolerance, and health-related quality of life to a greater degree than any other COPD therapy. Best of all, there seems to be plenty of work yet to do, with scientific studies that promise to yield additional advances that will benefit our patients.

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