

25

Asthma: Multisystem Implications

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Nearly one in ten children in the United States has a diagnosis of asthma (CDC 2010). This frequency has been increasing for decades both in the United States and abroad for reasons that are not yet clearly understood.^{21a,22,23,47,63,110,123} According to the U.S. Centers for Disease Control and Prevention (CDC), from 1979 to 1995 the incidence of asthma increased over 160% for children ages 0 to 4 years and 74% for children ages 5 to 14 years. Similarly, the morbidity rate increased 63% for children ages 0 to 4 years and 20% for children ages 5 to 14 years, whereas the mortality rate increased 12% for children ages 0 to 4 years and 146% for children ages 5 to 14 years.²² Follow-up data in a CDC report on the state of childhood Asthma in the United States indicated that asthma morbidity and mortality rates may have peaked in the mid/late 1990s.²

Pragmatically, the incidence figures mean that nearly 10% of all children seen by pediatric physical therapists may have asthma. Does this disease impact a child's motor performance? If so, what kind of impact does it have and what clinical implications does the presence of asthma have for the physical therapist treating pediatric patients?

The purpose of this chapter is to achieve the following:

1. Define asthma and discuss the medical ramifications of the disease.
2. Demonstrate the process of a differential physical therapy diagnosis for potential physical and activity limitations secondary to asthma through the illustration of a clinical case.
3. Identify the types of cardiopulmonary, neuromuscular, musculoskeletal, integumentary, and gastrointestinal impairments that may be associated with this diagnosis.
4. Present possible treatment strategies and specific PT procedural interventions.
5. Present potential long-term outcomes of physical therapy procedural interventions on the maturation and physical performance of a child with asthma.
6. Through a case study, illustrate participation benefits when a child's health is well supported.

PATHOPHYSIOLOGY

Asthma is a pulmonary disease with three significant characteristics: (1) airway inflammation, (2) airway obstruction

that is often reversible either spontaneously or with pharmacologic intervention, and (3) bronchial hyperresponsiveness to stimuli that are classified as either extrinsic or intrinsic.^{92,95,131} It is a disease of both the large and the small airways with recurrent episodes of shortness of breath, wheezing, chest tightness, and coughing. Bronchial hyperresponsiveness to a variety of extrinsic and intrinsic stimuli is increased. Extrinsic or allergic stimuli include but are not limited to pollen, mold, animal dander, cigarette smoke, foods, drugs, and dust. Intrinsic or nonallergic stimuli include but are not limited to viral infections, inhalation of irritating substances, exercise, emotional stress, and environmental factors such as the weather or climate changes. An individual may be sensitive to either type of stimuli or to both types.

Researchers have found genetic causes for the development of asthma, but genetics alone does not account for all types and severities of the expression of the disease.^{7,15,46} The physical, environmental, neurogenic, chemical, and pharmacologic factors that are associated with asthma are specific to each individual. They stimulate or trigger the immune system to release chemical mediators, which in turn cause constriction of the bronchial muscles, increased mucus production, and swelling of the mucous membranes. Mucus accumulation, which has been shown to be abnormal in asthma, may cause blockage of the airways, resulting in further air trapping and hyperinflation, and is a primary cause of death associated with asthma.⁷⁰

Over the decades, inflammation has been identified as a central component of asthma and may be a primary contributor to airway remodeling leading to chronic inflammation. This structural change may make the airways less responsive to medications.⁹⁵

In addition to the pulmonary manifestation of asthma, numerous studies have shown that a diagnosis of asthma in childhood results in frequent hospitalizations, poorer growth and development than peers, sleep disorders, and a reduction in overall quality of life.^{1,13,69,64,86,100,104,132,134} These factors result in an increased number of missed school/work days and limitations on the child's participation in typical childhood activities.^{17,29,35}

Asthma and other chronic respiratory diseases have been shown in recent years to be associated with the presence of

gastroesophageal reflux disease (GERD).^{67,69,89,96,121,135} Children with asthma should be screened for the possibility of underlying reflux. In some cases, chronic cough can be misdiagnosed as asthma, when in fact the primary cause is GERD.^{102,124} This has obvious implications for the medical management of cough symptoms.

PRIMARY IMPAIRMENT

DIAGNOSIS

The diagnosis of asthma is made on the basis of history, physical examination, auscultation and palpation, and pulmonary function tests (PFTs), especially in response to a methacholine challenge.⁶¹ Wheezing and rhonchi may be detected by auscultation even when the child does not show difficulty breathing. Breathing is often reported as worse at night or early in the morning. Hyperexpansion of the thorax, increased accessory muscle breathing, postural changes, increased nasal secretions, mucosal swelling, nasal polyps, "allergic shiners" (darkened areas under the eyes), and evidence of an allergic skin condition may be noted on physical examination. During an acute asthma attack, the child may show an increased respiratory rate, expiratory grunting, intercostal muscle retractions and nasal flaring, an alteration in the inspiration-expiration ratio, and coughing. In severe cases, a bluish color of the lips and nails may be noted (oxygen desaturation).

Attempts have been made to produce a national classification system for the severity of the disease based on clinical findings, but follow-up studies found those systems to inconsistently reflect the severity of the disease.^{10,16,104} In spite of the shortcomings, one of the most common severity classification systems was published by the U.S. National Institutes of Health (NIH) Heart, Lung and Blood Institute in 1997 and the details are listed in Table 25-1.⁹⁴ The NIH classification system lists asthma by clinical symptoms as (1) intermittent, (2) mild persistent, (3) moderate persistent, or (4) severe persistent. A recently updated Expert Panel Report #3 emphasizes initially diagnosing asthma by the severity of the disease, but then emphasizes controlling asthma rather than continuing to classify the severity⁹⁵ (www.nhlbi.nih.gov/guidelines/asthma/asthgdln.pdf).

Pulmonary Function Tests (PFT)

PFTs are performed to determine the location and degree of the respiratory impairment as well as the reversibility of bronchoconstriction following administration of a bronchodilator (methacholine challenge). Test values are compared with predicted values based on age, sex, and height.²⁴ PFT measurements may reveal (1) decreased forced vital capacity (FVC), (2) decreased forced expiration during the first second of FVC (FEV₁) as well as FEV₆ (6th second of exhalation), (3) decreased forced expiratory volume

TABLE 25-1 Clinical Classification of the Disease Severity of Asthma

Classification	Indications and Behaviors
Step 1 Intermittent	Intermittent symptoms occurring less than once a week Brief exacerbations Nocturnal symptoms occurring less than twice a month Asymptomatic with normal lung function between exacerbations FEV ₁ or PEFr rate greater than 80%, with less than 20% variability
Step 2 Mild persistent	Symptoms occurring more than once a week but less than once a day Exacerbations affect activity and sleep Nocturnal symptoms occurring more than twice a month FEV ₁ or PEFr rate greater than 80% predicted, with variability of 20–30%
Step 3 Moderate persistent	Daily symptoms Exacerbations affect activity and sleep Nocturnal symptoms occurring more than once a week FEV ₁ or PEFr rate 60–80% of predicted, with variability greater than 30%
Step 4 Severe persistent	Continuous symptoms Frequent exacerbations Frequent nocturnal asthma symptoms Physical activities limited by asthma symptoms FEV ₁ or PEFr rate less than 60%, with variability greater than 30%

Practical Guide for the Diagnosis and Management of Asthma Based on Expert Panel Report 2. NIH Publication No. 97-4053. Bethesda, MD: National Institute of Health, National Heart, Lung and Blood Institute, 1997, p. 10; and Morris, M. & Perkins, P. Asthma. e-Medicine, available at <http://www.emedicine.com/med/topic177.htm>, last updated 5/9/04.

compared with forced vital capacity (FEV/FVC), (4) decreased peak expiratory flow rate (PEFR) because of airway obstruction in large or small airways, (5) forced expiratory flow (FEF) during 25% to 75% of FVC (FEF_{25%} to 75%) because of airway obstruction in the small airways, (6) increased residual volume (RV), and (7) increased functional residual capacity (FRC) because of air trapping.⁹⁵ Generally, patients with asthma are instructed to monitor their daily pulmonary fluctuations and adjust their medication levels by testing their PEFR with a peak flow meter. However, recent studies have shown that FEV₁ and midexpiratory FEF_{25%} to 75% are better indicators of disease status than PEFR.^{45a} Peak flow meters are cheaper and more readily available in a home environment, so they will probably

continue as the home equipment of choice until FEV₁ and FEF25% to 75% can be readily tested at home. The child under the age of 5 or 6 years, however, cannot perform pulmonary function tests and needs to be monitored on the basis of clinical signs and symptoms.¹⁹

IMPAIRMENT IN INFANCY TO ADULTHOOD

A diagnosis of asthma is not typically made until the child is 3 to 6 years of age when numerous episodes of pulmonary problems have been demonstrated and are consistent with asthma.⁶¹ In the meantime, children may be diagnosed with “reactive airway disease.” More sophisticated tests such as PFTs are not possible until children are around 6 years of age when they are capable of cooperating and performing the tests.¹⁹ The young child diagnosed with asthma will typically present with a family history of asthma or atopic predispositions, prematurity, lung abnormalities, exposure to second hand smoke, history of episodes of wheezy bronchitis, croup, recurrent upper respiratory tract infections, chronic bronchitis, recurrent pneumonia, respiratory distress syndrome, difficulty sleeping, bronchopulmonary dysplasia, or respiratory syncytial virus (RSV) infection.¹²⁷

Severe RSV infection in infancy is highly associated with a later diagnosis of asthma. Currently, it is not known if children with asthma have a more severe reaction to the virus or if a severe infection with RSV actually causes asthma to develop later in childhood.^{40,97,117} In addition to normal childhood illness, complications associated with prematurity and very low birth weight also have a high correlation with a later diagnosis of asthma. Like RSV infection, it is not known if prematurity causes asthma or simply makes infants more predisposed to asthma.^{65,66} Respiratory problems following preterm births were also associated with eczema and GERD.⁶⁵

By adolescence, asthmatic symptoms often decrease. Even when free of clinical symptoms, however, the adolescent may have significant impairment revealed by PFT measures. In several long-term studies, children who were preterm babies with respiratory problems, such as bronchopulmonary dysplasia (BPD) or respiratory syncytial virus (RSV) infection, were more likely to have lung and airway restrictions later in childhood, especially noted in reduced expiratory flows.^{33,42,88,118} Preterm babies with chronic lung conditions are more likely to have asthma, and it is speculated that they may be more likely to develop emphysema-like conditions in middle age.¹¹ This remains an hypothesis, however, as survivors of extreme prematurity have not yet reached middle age.

Pediatric physical therapists should pay careful attention to a child’s medical history to note evidence of a risk for asthma and consider all the ramifications on that child’s health, growth, and development when planning procedural interventions.

MEDICAL MANAGEMENT

Episodes of asthma attacks are usually reversible and can be prevented or modified to some degree when the individual-specific triggers have been identified. The frequency, duration, and severity of attacks are highly variable even for the same individual.^{4,101} Acute treatment is aimed at reversing the bronchoconstriction. Bronchodilator medications are administered by inhalation or injection. If the asthma attack is severe and does not respond to bronchodilator medications, the diagnosis of status asthmaticus may be made. This is considered a life-threatening medical emergency.⁹⁹ Hospitalization will be required to administer medications intravenously, to monitor blood gases, and to administer oxygen.

The goals of long-term management are to prevent chronic and troublesome symptoms, to maintain pulmonary function and physical activity level, to prevent recurrent exacerbations, to minimize the need for emergency room visits or hospitalizations, to provide optimal pharmacotherapy, and to meet the patient’s and family’s expectations of and satisfaction with asthma care.⁹⁵ This is accomplished through pharmacologic support as well as periodic examination, ongoing monitoring, and education. The patient should be taught to self-monitor asthma symptoms and patterns, response to medications, quality of life, and functional status and to perform and record peak flow readings. A written action plan should be developed and reviewed and revised periodically. This action plan should be shared with school and other personnel who are involved with the child. Some allergens such as cigarette smoke, animal dander, and dust can be handled by environmental control. Desensitization (“allergy shots”) may be used for triggers such as pollen or mold. Triggers such as emotional stress may be handled by relaxation exercises and education.²⁵

There is no cure for asthma. The pharmacologic management is intended to stop, control or prevent symptoms through short-term relief or long-term management of the condition. Most patients take more than one medication to control their symptoms.

The current National Institutes of Health (NIH) Publication on the Guidelines for Asthma describes a terraced approach to pharmacologic management of asthma⁹⁵ (www.nhlbi.nih.gov/guidelines/asthma/asthgdln.pdf). The key recommendations of the NIH publication describe dosing medication according to the severity of the disease in three distinct age groups: 0 to 4 years, 5 to 11 years, and 12 years and older, as recent evidence indicates that children respond differently to asthma medications than their adult counterparts. Inhaled corticosteroids continue to be the drug of choice across all age groups for long-term management of asthmatic symptoms. Inhaled medications deliver a concentrated dose most effectively with fewer systemic side effects and a quicker onset of action than other means of administration, but children with persistent asthma need

both long-term and short-acting agents to manage their disease. A press release accompanying the NIH report on August 29, 2007, summarizes the recommendations for persistent asthma as follows:

Expert Panel Report 3 includes new recommendations on treatment options such as leukotriene receptor antagonists and cromolyn for long term control; long acting beta agonists as adjunct therapy with inhaled corticosteroids; omalizumab for severe asthma; and albuterol, levalbuterol, and corticosteroids for acute exacerbations. <http://public.nhlbi.nih.gov/newsroom/home/GetPressRelease.aspx?id=2442>

Newer drugs are constantly being researched and brought on the market; thus, any listing of medications is relevant only within a limited time frame.^{44,58} The overall goal of medication research is to find drugs that will stop the inflammatory process at an earlier point or prevent the presentation of asthma altogether. As the understanding of the pathophysiology and genetics of asthma increases, new medications with more specific but fewer side effects will probably be developed. Medications to address coexisting conditions such as allergies and GERD will also contribute to the effective management of the child's asthma.^{32,41,58,121} Physical therapists should check with the child's physician about current medications. A consumer-friendly website from the Mayo Clinic describes asthma medications for children and adults (www.mayoclinic.com/health/asthma-medications/ap00008).

SECONDARY IMPAIRMENTS

QUALITY OF LIFE

Recurrent asthma attacks or poorly controlled asthma may result in the child or family deciding to restrict normal childhood activities out of fear of asthma exacerbations or social retributions.⁷²⁸ In fact, it is not only the child who suffers. Adult caregivers are forced to miss days of work just like the child is forced to miss days of school; taken together they reduce the quality of life for the entire family.¹¹⁴ Growing up with this chronic disease is bound to play a role in the child's choice of an adult vocation and living situation.

MEDICATION SIDE EFFECTS

Although the medications used in the management of asthma are necessary, the side effects of these medications also may have an impact on daily life. For example, oral corticosteroids may cause an increased appetite and weight gain, fluid retention, increased bruising, and mild elevation of blood pressure. Other side effects reported from a variety of asthma medications are nervousness, headache, trembling, heart palpitations, dizziness or light-headedness, dryness or irritation of the mouth and throat, heartburn,

nausea, bad taste in the mouth, restlessness, difficulty concentrating, and insomnia, to mention a few.⁹² To determine if motor, cognitive, or emotional behaviors are related to the medication, consult with the child's physician.

GROWTH AND DEVELOPMENT

Another aspect of asthma that is particularly important for self-esteem in adolescence is growth and development. There is conflicting data on whether children with asthma eventually catch up to their peers in terms of skeletal maturation.^{3,12,79} For example, Baum¹² found that children with severe asthma have a significantly shorter stature, skeletal retardation, and delayed puberty. Researchers have questioned whether asthma itself or the prolonged use of steroids is responsible for such findings. In 2009, however, Mainz showed that adherence to current inhaled corticosteroid (ICS) dosing recommendations was more beneficial to the growth and development of a child with severe persistent asthma than not taking corticosteroids. In fact, the 9-year-old child who was the subject of the study had an additional skeletal impairment (thoracic deformity) caused by the pulmonary hyperinflation that resulted from his poor asthma management. Thus, proper use of ICS and other asthma medication may actually promote normal growth.

IMPACT ON FINANCIAL COSTS TO THE FAMILY AND SOCIETY

Asthma is associated with the highest related costs of routine pediatric care, reportedly topping \$3 billion a year in the United States.⁸⁷ A study of 71,818 children ages 1 to 17 years who were enrolled in a health maintenance organization,^{78a} was conducted to measure the impact of asthma on the use and cost of health care. The children with asthma incurred 88% more costs than children without asthma. Thus, having a child with asthma not only increases the family's focus on its medical needs but also consumes a family's financial resources. For some families, this cost may be at the expense of other needs, placing a financial burden on the family and the community.

SUMMARY OF THE MEDICAL ASPECT OF ASTHMA

Asthma is a common childhood disease that can result in severe functional limitations and restrictions in childhood activities. There are three primary components of asthma: often reversible airway obstruction, airway inflammation, and airway hyperresponsiveness that can affect the large and the small airways causing shortness of breath. The disease itself is complex with multiple system interactions such that each child's presentation of asthma is unique. The physical therapist needs to know how this disease affects

that particular child's ability to participate in physical activities and what role the therapist can play in optimizing the child's potential for normal development, participation, and health.

PHYSICAL THERAPY EXAMINATION, EVALUATION, AND INTERVENTIONS

Physical therapists are traditionally involved in exercise programs for children with asthma, and studies have shown the efficacy of such programs in improving endurance and decreasing asthmatic symptoms.^{93,103,129} The specifics of exercise testing and the development of a fitness program are covered in Chapter 6 and will not be covered here. Endurance programs such as treadmill training, which are also common, will not be covered either, as this author prefers to find ways to improve fitness and endurance through participation in typical childhood activities rather than in contrived activities, circumstances permitting. If physical fitness is seen as an "exercise duty," it is this author's experience that the child and family are less likely to follow through, seeing physical exercise as a chore rather than an opportunity for growth. Thus, the intent for the physical therapy section of this chapter is to (1) help the clinician understand the process of a differential diagnosis for the potential physical and activity limitations that may occur secondary to the interaction of asthma with growing and maturing bodies and (2) to present strategies and interventions that endeavor to get these children back among their peers, playing and competing in age-appropriate physical activities, rather than participating in adult-supervised exercise programs.

Nevertheless, the child with asthma may need more than a nudge and emotional support to engage in age-appropriate physical activities. Few studies address possible secondary physical impairments, such as adverse musculoskeletal changes/alignments, and neuromuscular recruitment problems that could limit the child's functional potential.^{27,79,125} In the *Guide to Physical Therapist Practice*, physical therapy is defined as a "profession with ... widespread clinical applications in the restoration, maintenance and promotion of optimal physical function."⁶ Thus, if physical and functional limitations were identified as occurring secondary to asthma, then physical therapy would be the appropriate service to restore, maintain, and promote optimal physical functioning. Physical therapy examinations

Box 25-1 MOTOR IMPAIRMENT CATEGORIES

1. Neuromuscular system
2. Musculoskeletal system
3. Integumentary system
4. Cardiovascular/pulmonary system
5. Internal organs, especially gastrointestinal system*

Adapted from American Physical Therapy Association. *Guide to Physical Therapist Practice*, 2nd ed., *Physical Therapy*, 81(1), 29, 2001.

*The APTA's impairment categories do not have a category for dysfunction of internal organ systems other than the cardiovascular/pulmonary system; thus, "internal organs" was added by this author to correct for this deficit.

and evaluation and considerations for physical therapy interventions will be discussed within the context of a single case to illustrate how to perform a differential diagnosis through a multisystem review and how to appropriately plan procedural interventions to address both the medical and physical deficits. Impairment categories listed in the *Guide*, plus an additional category of "internal organs," will be specifically evaluated for their impact on movement potential for the child with asthma (Box 25-1). Long-term outcomes from these interventions are also presented.

SUMMARY

This chapter presented the pathophysiology and current medical management strategies associated with childhood asthma. In addition, through the use of a single case, ideas for the physical therapy diagnosis and management of physical limitations associated with asthma and its resultant functional limitations were presented through a multisystem and multidiscipline perspective. Impairments in the cardiopulmonary, neuromuscular, musculoskeletal, integumentary, and gastrointestinal systems were assessed for their contribution to the activity and participation limitations that could not be fully explained by asthma alone. An individualized physical therapy program was then presented as a template for other pediatric physical therapy programs. Short-term and long-term results from the physical therapy procedural interventions used with this single case were presented to give the reader an indication of the potential success of such interventions. Obviously, each individual case is unique and must be developed within the context of that particular patient's situation.



CASE STUDY

"Jonathan"

"Jonathan" was referred to physical therapy by his pediatric pulmonologist at 9 years of age. He was in fourth grade and lived with both parents and two older siblings in a large metropolitan area with access to excellent pediatric care. He had two significant diagnoses: exercise-induced asthma (EIA) and a pectus excavatum. Figure 25-1 shows Jonathan at 10 years old.

A pectus excavatum is an anterior chest wall deformity, particularly of the body of the sternum and the surrounding costal cartilage. The cartilage is collapsed inward giving the visual presentation of a hollowing out of the chest, otherwise called a "cavus," "caving-in," or a "funnel" deformity of the lower sternum⁴⁶ (Figure 25-2). Jonathan's mother reported that his chest "always looked that way" from birth (Figure 25-3). The thoracic surgeon recommended surgery to correct the deformity, but the family refused a surgical intervention.

Jonathan's mother reported a history of frequent bouts of recurring bronchitis from 3 to 6 years old before the eventual diagnosis of asthma at age 6 by a pediatric pulmonologist. He had no history of pneumonia or hospitalizations. Jonathan's asthma has been managed with medications since then, including Flovent twice a day (two puffs), and Intal and Ventolin as necessary before participation in soccer. In spite of the medications, the patient and his mother

reported frequent episodes of extreme EIA symptoms, including chest tightness, wheezing, and shortness of breath after 5 to 10 minutes of soccer, resulting in a termination of the activity. The pulmonary physician reported that Jonathan's pulmonary function tests (PFTs) indicated that his pulmonary limitations were minor (i.e., minor peripheral airway resistance). No other significant deficits were found on four different testing dates over a year's time. Cardiac testing was negative. Even on an exercise challenge test by the pulmonologist, Jonathan showed no significant change in lung function, nor a positive response to a bronchodilator challenge. The diagnosis of EIA was made primarily on the basis of the child's clinical presentation rather than pulmonary tests.

If his lung function tests did not show significant impairment from his EIA, and his chest deformity was not causing lung or heart impairments, then what could explain his level of functional limitations? The pulmonologist believed that the medical status of his EIA alone could not have caused such a severe activity limitation. She knew that the patient and his family were motivated to follow his asthma management program, especially because Jonathan wanted to qualify for the travel soccer team. As a result, she referred Jonathan to physical therapy to rule out physical impairments that might account for some of the severity of his disease presentation.



Figure 25-1 Jonathan at age 10 years. Note pectus excavatum (cavus deformity of the lower chest and sternum).



Figure 25-2 **A**, This 16-year-old male has asthma and a more severe congenital pectus excavatum deformity. **B**, Note lower sternal depression (or funnel), bilateral rib flares, and elevated and protracted shoulders.



CASE STUDY—cont'd

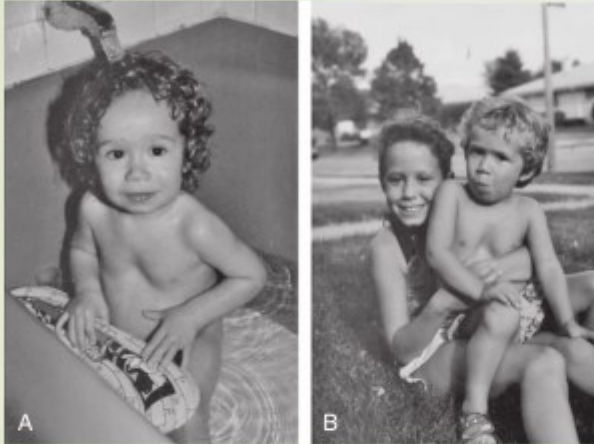


Figure 25-3 Comparison picture of Jonathan at ages 2 and 3 years old. Note the pectus excavatum is more severe at 3 years of age.



EVIDENCE TO PRACTICE 25-1

CASE STUDY "JONATHAN"

EXAMINATION DECISION

The decision to look at Jonathan's musculoskeletal and neuromotor systems was based on years of clinical experience of the therapist following long-term, complicated pediatric cardiopulmonary cases, and the referring physician's impression that Jonathan's endurance and lifestyle limitations could not be fully accounted for by the asthma alone. At the time of the referral, there was no evidence published on the consequential physical impairments for kids with pulmonary or chest wall deformities, but today we have two case studies that show improved postural alignment (musculoskeletal changes) and other physical improvements for such children following physical therapy interventions (Canavan & Cahalin, 2008; Massery, 2005). A Cochrane systematic review in 2001 by Hondras and colleagues (2001) showed insufficient evidence yet to support manual therapy for asthma. That finding does not mean that manual therapy is not effective. It means more research needs to be done in this area before broader conclusions can be drawn. Holloway and Ram (2004) reported similar findings for neuromotor retraining for breathing.

PLAN OF CARE DECISION

The decision to treat Jonathan's secondary physical problems, primarily musculoskeletal and neuromotor control issues, was based on Jonathan's personal goals and motivation, his mother's motivation, the clinical experience of his physician

and therapist, and limited research on chronic diseases at the time that physical therapy was initiated. Now, however, we have multiple research studies confirming this decision to treat. Asthma negatively impacts the quality of life for children with asthma and other chronic health issues, and these effects carry over into adulthood. Children with asthma are (1) less likely than their peers to participate in ongoing physical activities without additional support by the family or medical community, (2) more likely to report poorer quality of life across the life span, (3) more likely to miss days of school or work, and (4) more likely to be obese in adulthood, to name a few (Fletcher et al., 2010; Philpott et al., 2010; van den Bemt et al., 2010). The good news is that these trends can be reversed when quality of life measures are included in the child's asthma management (Bravata et al., 2009).

PATIENT/FAMILY PREFERENCES

Jonathan's family was motivated to try a nonsurgical (pectus excavatum), minimal medication approach to managing his asthma and chest wall deformity with the long-term goal of maximizing his participation in childhood activities. Jonathan himself had a specific goal: to make the travel soccer team. This goal was made the epicenter of his physical therapy plan. Every intervention was introduced as a way to improve Jonathan's odds of making the team, demonstrating relevance to Jonathan's goals, not just the therapist's desired outcome. Studies, such as that by Robinson and colleagues (2008), have shown patient-centered programs result in better long-term

Continued



CASE STUDY—cont'd



EVIDENCE TO PRACTICE 25-1—cont'd

adherence to medical management programs and improve health and quality-of-life outcomes.

Canavan, P. K., & Cahalin, L. (2008). Integrated physical therapy intervention for a person with pectus excavatum and bilateral shoulder pain: A single-case study. *Archives of Physical Medicine and Rehabilitation*, 89(11), 2195–2204.

Holloway, E., & Ram, F. S. (2004). Breathing exercises for asthma. [Update of *Cochrane Database Systematic Reviews*, 2000, (3), CD001277; PMID: 10908489.] *Cochrane Database of Systematic Reviews*, 1, CD001277.

Hondras, M. A., Linde, K., et al. (2001). Manual therapy for asthma. [Update of *Cochrane Database Systematic Reviews*, 2000, (2), CD001002; 10796578.] *Cochrane Database of Systematic Reviews*, 1, CD001002.

Massery, M. (2005). Musculoskeletal and neuromuscular interventions: A physical approach to cystic fibrosis. *Journal of the Royal Society of Medicine*, 98(Suppl 45), 55–66.

Bravata, D. M., Gienget, A. L., et al. (2009). Quality improvement strategies for children with asthma: a systematic review. *Archives of Pediatrics & Adolescent Medicine*, 163(6), 572–581.

Fletcher, J. M., Green, J. C., et al. (2010). Long term effects of childhood asthma on adult health. *Journal of Health Economics* 29(3), 377–387.

Philpott, J. F., Houghton, K., et al. (2010). Physical activity recommendations for children with specific chronic health conditions: juvenile idiopathic arthritis, hemophilia, asthma, and cystic fibrosis. *Clinical Journal of Sport Medicine*, 20(3), 167–172.

van den Bemt, L., Kooijman, S., et al. (2010). How does asthma influence the daily life of children? Results of focus group interviews. *Health Quality of Life Outcomes*, 8, 5.

Robinson, J. H., Callister, L. C., et al. (2008). Patient-centered care and adherence: Definitions and applications to improve outcomes. *Journal of the American Academy of Nurse Practitioners* 20(12), 600–607.

Medical History and Multisystem Screening of the Neuromuscular, Musculoskeletal, Integumentary, Cardiovascular/Pulmonary, and Gastrointestinal Systems

A multisystem approach to screening medical and physical deficits was performed starting with an extensive medical history, followed by identifying the child's limitations in activities and participation, and then working "backward" with this information to try to uncover the primary impairment(s) that might explain the presenting signs and symptoms. In this case, the pulmonologist had already done an extensive medical history and pertinent tests to rule out other underlying medical pathologies that could account for his participation limitations. Other medical reasons for an increase in asthmatic symptoms could have included gastroesophageal disease (GERD), sleep disordered breathing, pulmonary ciliary dysfunction, or vocal fold dysfunction to name a few.^{34,104,124,150} Jonathan never had any overt clinical symptoms of reflux or nocturnal dysfunction; thus, no tests were done. His mother did not recall any testing for ciliary dysfunction (which results in impaired airway secretion motility because of dysfunction of the beating cilia), and the lack of any recurrent respiratory infection, such as repeat pneumonias, made this diagnosis unlikely.²⁶ At the time of the physical therapy examination, vocal fold dysfunction and supra-esophageal manifestations of GERD were not commonly understood to be a possible cause of asthmatic symptoms, and this possibility was thus not explored. However, physical therapists currently assessing children with asthma should try to rule out gastric and vocal fold disorders as a routine part of the asthma medical screening.^{6,34,59,62,133,135}

Screening Assessment of Functional Limitations Related to Asthma

Following a medical history review, the physical therapy examination and evaluation focused on assessment of Jonathan's breath support

throughout everyday activities to determine if there was a specific area of impairment or a pattern of limitation that could explain his endurance limitations (Table 25-2).

Soda-Pop Can Model of Postural Control and Respiration

Before describing Jonathan's examination results, the dual nature of the respiratory and postural muscles needs to be understood. External and internal forces affect respiration and postural control strategies. This concept is illustrated in the following model^{93,41} (Figure 25-4).

The aluminum shell of a can of soda pop is thin, flimsy, and inherently weak. Yet when unopened, it is functionally strong. It is almost impossible to compress or deform the unopened can unless the exterior shell is punctured. However, when it is opened, it loses its "strength" and is easily crushed. It is the internal pressure from the carbonation, not the aluminum exterior, that gives the can strength. The trunk of the body embodies a similar concept, using its muscular contractions to prevent its flimsy casing, the skeleton, from being "crushed" by external forces such as gravity. The trunk is composed of two chambers (thoracic and abdominal cavities), which are completely separated by the diaphragm, thus rendering each chamber capable of creating different internal pressures. The diaphragm is the trunk's primary pressure regulator. The chambers are functionally sealed at the top by the vocal folds and at the bottom by the pelvic floor muscles. The entire system is dynamically supported by muscles that generate, regulate, and maintain internal pressures in both chambers. As a result, these muscles must simultaneously meet the respiratory and postural needs of that person. In other words, the trunk muscles act as one continuous functional unit, providing the core support for pressure regulation that allows the individual to multitask, enabling "walking, talking, and chewing gum" to occur simultaneously and effortlessly.^{34,38,51,53,57,120}



CASE STUDY—cont'd

TABLE 25-2 Assessing Functional Limitations Associated with Asthma or Other Ventilatory Dysfunction*

Functional Activity	Secondary Problems [†]
Breathing	Inadequate breath support and inefficient trunk muscle recruitment at rest or with activities such that breathing or postural control are compromised Asthmatic triggers such as rapid airflow caused by sudden increase in physical activity, dry air or extreme air temperatures, or other triggers that trip an asthmatic reaction
Coughing	Ineffective mobilization and expectoration strategies
Sleeping	Breathing difficulties, signs of obstructive or central sleep disorders Nocturnal reflux (GERD)
Eating	Swallowing dysfunction Reflux (GERD) Dehydration Poor nutrition
Talking	Inadequate lung volume and/or inadequate motor control for eccentric and concentric expiratory patterns of speech Poor coordination between talking (refined breath support) and moving (postural control)
Moving	Inadequate balance between ventilation and postural demands Breath holding with more demanding postures: use of the diaphragm as a primary postural muscle for trunk stabilization Inadequate lung volume to support movement Inadequate and/or inefficient muscle recruitment patterns for trunk/respiratory muscles causing endurance problems or poor motor performance Ineffective pairing of breathing with movement, especially with higher level activities

*The following activities require adequate lung volumes and coordination of breathing with movement for optimal performance.
[†]These typical secondary problems associated with asthma should be screened for to determine their possible contribution to the child's motor impairment or motor dysfunction.

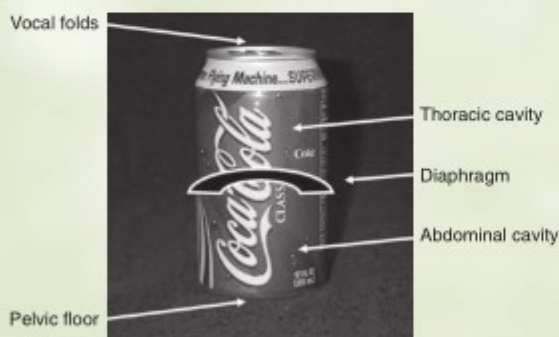


Figure 25-4 A postural control model using a soda pop can.

The functions of the internal organs are supported by these pressures as well, especially the lungs, heart, vascular structures, gastrointestinal system, and lymphatic systems. For example, following a lower cervical spinal cord injury, the diaphragm is still functioning, but the intercostals and abdominal muscles are paralyzed. As a result, normal pressures cannot be generated. Without the other muscles' support, the diaphragm is mechanically compromised resulting in

impaired inspiratory lung volumes, impaired expiratory force (weak cough), low blood pressure, and frequent constipation.^{63,15,19} In Jonathan's case, his pectus excavatum became his "weak spot." The postural/respiratory implications will be discussed in the next section.

Breathing

Increased effort was noted with Jonathan's quiet breathing pattern, including (1) occasional paradoxical breathing (i.e., inward

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CASE STUDY—cont'd

movement of the chest or abdomen during inhalation) and (2) frequent forced exhalations. Paradoxical breathing is thought to be due to the significant negative inspiratory pressures that the child with asthma must exert to overcome inspiratory resistance in the airways.^{21,45,81}

The paradoxical movements of his chest wall indicated a muscle imbalance between the respiratory muscles, usually associated with weaker intercostals and abdominal muscles in relation to the diaphragm.⁹ This weakness in the chest muscles, combined with the unbalanced descent of the diaphragm, may be the result of the pectus excavatum or it may have contributed to the further development of the pectus (the “chicken and the egg” syndrome). On the other hand, Jonathan’s forced exhalations were probably secondary to the obstructive lung component of asthma, which constricts the conducting airways during exhalation. This forces the child to recruit expiratory muscles (primarily the abdominals and internal intercostals) to push the air out of the chest, even during quiet exhalation, causing increased work of breathing even at rest.

These patterns indicated that Jonathan’s motor planning for ventilation muscle recruitment did not appear to be optimal for activities that required greater oxygen consumption because he was already overusing his diaphragm and recruiting his upper accessory muscles at rest, all the while underutilizing his intercostal muscles. All these observations led me to believe that his respiratory muscle imbalance may be significantly contributing to his decreased endurance and poor musculoskeletal alignment of his chest and overall postural alignment, and it may account for the endurance limitations not attributed to asthma itself by the pulmonologist.

Coughing

The patient demonstrated an effective cough. The only reports from the family of ineffective coughing or impaired airway clearance strategies during respiratory episodes came from his mother, noting that sometimes when he is sick, his secretions are so thick that they get “stuck” in his chest. Jonathan reported that he rarely drank water at school. This would indicate a need for increased hydration and a possible screening for ciliary dysfunction to rule out the possibility that the cilia themselves were dysfunctional rather than that the mucus was simply thicker because of dehydration.¹³⁰ He did not report vomiting associated with forceful coughing as many children with asthma report. Gagging or vomiting is a common occurrence following a hard cough, likely because of the lower esophageal sphincter (LES) succumbing to the high abdominal pressures associated with a series of coughs. When the LES fails, the intraabdominal pressure of cough will force the abdominal contents up into the thoracic esophagus. This is clinically noted as a severe reflux presentation: a dry gag, or a full vomit at the end of the cough.¹¹⁶

Sleeping

The patient reported that he sleeps on his back with his arms by his side, and occasionally he sleeps on his side. No breathing difficulties (including apnea, snoring, or irregularities), coughing, or drooling at night were reported that could indicate upper airway obstruction or

GERD.¹²⁶ A preference for the supine position at night, however, may indicate a recruitment of upper accessory muscles even while sleeping owing to the optimal length-tension relationship of those muscles in supine along with increased posterior stabilization.¹⁸ Jonathan reports that he does not “curl up” to sleep. It is my clinical observation that children who are primarily upper chest breathers instead of diaphragmatic breathers will often choose to sleep supine with their arms up over their head rather than prone or curled up on their side, probably because of the improved length-tension relationship of all the anterior and superior chest muscles in supine. They may also report that they start out on their side or stomach, but find themselves on their backs in the morning. Depending on the rest of the findings, one may want to recommend a change in sleep postures for Jonathan, but only if that still allows him to sleep through the night as sleep-disordered breathing is a common consequence for children with asthma. Increased negative pressure during inhalation because of asthma’s restricted airways, combined with a recumbent position, may predispose patients to nocturnal GERD and disrupted sleep.^{108,152}

Eating

Jonathan did not report problems with chewing or swallowing any foods or textures, nor did he report any difficulties with drinking any type of liquid at any speed. In addition, there was no history of aspiration, choking, or gagging episodes. He did not present with any clinical signs of GERD, which is a common association with asthma and should be ruled out as a contributor to the motor or health restrictions.¹²⁴ Asthma is typically associated with a higher sensitivity or reactivity to dry air in the airway; thus, adequate hydration to keep the airway moist (humidity) is necessary to decrease external triggers to asthmatic reactions.⁵⁶ Hydration is also necessary to keep secretions thin and mobile.⁵ Jonathan did not have a “feeding problem,” but he did have a hydration problem, which most likely exacerbated his EIA symptoms.

Talking

Jonathan demonstrated a normal number of syllables per breath (at least 8 to 10) as noted during conversational speech.^{30,49} He was capable of excellent sustained vocalization: 20 seconds (twice the expected length).²⁰ He could also talk in all postures at multiple volume levels with good postural control and controlled eccentric breath support. This was clearly the patient’s strongest demonstration of breath control within a functional task. I anticipated using this strength to reinforce eccentric trunk control and pacing activities with soccer. Speech breathing is primarily eccentric control of the inspiratory muscles; thus, I could use his excellent eccentric motor planning for the trunk muscles during speech to recruit the same muscles for eccentric control during other eccentric trunk and postural maneuvers.²⁷

Moving

Jonathan reported episodes of extreme shortness of breath (dyspnea) and asthmatic episodes within 5 to 10 minutes of participating in strenuous activities such as soccer. He reported that he “warms up for a minute” before starting to run in soccer. This quick change from



CASE STUDY—cont'd

rest to running would cause a rapid acceleration in inspiratory volume and flow rates and could possibly trigger his EIA response secondary to upper airway hyperresponsiveness or increased airway resistance.^{5,91} He also reported that he used his bronchodilator inhaler immediately before team practice, which doesn't allow for maximal benefit of the drug; thus, incorrect use of medications may also be contributing to his EIA. It was interesting that Jonathan did not report breathing problems with quiet activities, in spite of the fact that his breathing demonstrated inconsistent recruitment patterns and an increased work of breathing at rest. No breath holding was noted with any developmental posture or transitional movement. Poor coordination between breathing and movement appeared to be contributing to his limitations in higher-level activities such as sport participation but not during quiet activities.

Summary of Functional Screening

The functional screening indicated impairment at the level of muscle recruitment for breath support at rest and during strenuous

exercise, with resultant endurance impairments. Activities that demanded greater oxygen consumption and faster inspiratory flow rates, such as soccer, immediately used up his pulmonary reserves, causing Jonathan to hit an early "ceiling" effect, forcing him to terminate the activity because of dyspnea and asthmatic symptoms. It also caused a rapid influx of dry air, which most likely triggered the EIA response. No significant problems were noted with functional tasks requiring less oxygen demand and slower inspiratory flow rates such as sleeping, coughing, eating, or talking. In fact, breath support for talking was extremely well developed and was noted as his strongest asset on the functional assessment. Inadequate daily hydration, which would decrease his secretion mobility and produce heightened airway hyperresponsiveness (bronchospasm), was also a significant finding. Jonathan's functional screening results are summarized along with his other examination and evaluation findings in Table 25-3.

TABLE 25-3 Synopsis of Jonathan's Initial Physical Therapy Examination and Evaluation

Evaluation	Jonathan's Results
Medical diagnoses (pathology)	Asthma, primarily exercise induced (EIA) Pectus excavatum
Impairment (summary of body functions and structure)	<p>Cardiopulmonary: Inflammation and hyperresponsiveness of airways particularly after initiation of exercise with PFTs indicating mild peripheral airway resistance Marked endurance limitations (5–10 minute tolerance) especially with higher level activities (particularly soccer) Occasional dehydration and decreased secretion mobility Increased work of breathing even at rest, RR 20 breaths/min (high end of normal) Auscultation clear in all lung fields No cardiac deficits per cardiologist</p> <p>Musculoskeletal: Marked pectus excavatum and elevated sternal angle Rib flares, L > R, with weakness noted in oblique abdominal muscles L > R (patient is right-handed) Functional midthoracic kyphosis of the spine particularly at the level opposite the pectus Decreased lateral side bending, indicating chest wall and quadratus lumborum restrictions Rib cage mobility restrictions greatest in mid chest nearest the pectus Mid trunk "fold" in sitting (rib cage collapsing onto the abdomen in sitting) "Slouched" sitting and standing postures: shoulders protracted and internally rotated Shortened neck musculature, hypertrophy No shoulder range-of-motion limitations</p> <p>Neuromuscular: Muscle imbalances in trunk muscles with significantly weaker/underutilized intercostal muscles, oblique abdominal muscles, and scapular adductors Inefficient neuromuscular recruitment patterns for inspiratory and expiratory efforts as well as for postural demands</p> <p>Integumentary: No restrictions noted</p> <p>Internal organs, especially gastrointestinal system: No reflux, constipation, or other gastrointestinal dysfunction</p>

Continued



CASE STUDY—cont'd

TABLE 25-3 Synopsis of Jonathan's Initial Physical Therapy Examination and Evaluation—cont'd

Evaluation	Jonathan's Results
Functional limitations (breathing, coughing, sleeping, eating, talking, moving)	Breathing pattern was inefficient showing muscle imbalance among the diaphragm, abdominals, intercostals, and upper accessory muscles Movement and participation limitations secondary to medical impairments, endurance impairments, postural impairments, and breath support impairments In addition to movement limitations due to the medical component of asthma, his movements were limited by the simultaneous postural and respiratory demands presented during higher level activities such as soccer and the ventilatory needs to support such tasks No functional breath support limitations noted in sleeping, eating, coughing, or talking activities
Activity and participation limitations	According to mother, Jonathan was beginning to withdraw from participation in physical activities, especially organized athletics, secondary to his "deformed chest" and fear of asthmatic episodes EIA caused him to stop playing soccer after typically 5 to 10 minutes Patient had already stopped swimming to avoid taking off his shirt among his friends
Diagnosis	9-year-old boy, with history of severe EIA and marked pectus excavatum Significant restrictions in chest wall mobility and posture, as well as motor planning deficits, contributed to limitations in adequate breath support, postural control and endurance for desired functional activities and contributed to the continued development of the pectus and other postural deformities Dehydration also appeared to play a significant role in triggering a bronchospasm (EIA) during the rapid change in inhalation volume and negative force associated with participation in sports such as soccer
Prognosis	Excellent Capable of developing new motor plans Musculoskeletal deformities were functional, not fixed; still prepubescent Motivated by his desire to "make" the traveling soccer team, and be "normal" Supportive family Good medical care

Assessing the Impairments Related to Functional Limitations

When limitations are noted during the functional limitation screening assessment, further impairment testing should be done (age appropriately) to assess the extent of the initial limitations and as a baseline for assessing future progress. A baby or young child would not be capable of performing or cooperating with some tests, such as PFTs; thus, the physical therapist must assess the appropriateness of any impairment test for each specific patient.

According to Jonathan's pulmonologist, his lung pathology alone could not have caused his marked functional limitations noted during athletics such as soccer. Results of our functional screening concur with that opinion; thus, further impairment tests and measures were taken. A few key findings from his examination will be interpreted here to explain their relevance to his functional limitations.

Jonathan demonstrated a muscle imbalance between his three primary respiratory muscles (diaphragm, abdominals, and intercostals) and his upper accessory muscles of respiration. As previously described, all play a dual role in simultaneously meeting his breathing needs and his postural needs. Because of his asthma, Jonathan had to overcome increased inspiratory resistance even at rest, which likely led him to over-recruit the upper accessory muscles from a

very young age, setting up a pattern of overuse, which leads to fatigue (endurance factor). When he needed more oxygen during exercise, he recruited those same accessory muscles even more so, reaching a ceiling on his respiratory reserves. There were no muscles left to recruit when he needed more oxygen (again with an impact on endurance). Thus, when his postural demands increased, such as during soccer, his oxygen requirements limited the activity. As Hodges described in 2001, when faced with increasing oxygen demands, the diaphragm will decrease its active role in postural control in order to concentrate on its survival role as a respiratory muscle.⁵⁵

Typical of many patients who have an increased work of breathing, Jonathan used his accessory muscles of respiration at the expense of his diaphragm and external intercostals, seen clinically as occasional paradoxical breathing and forced expiratory maneuvers at rest. I suspect this pattern contributed to the sternal abnormalities (elevated sternal angle and pectus excavatum) that formed early in his life. In my clinical observations, children with an early onset of asthma or other chronic respiratory conditions who overuse their sternocleidomastoid (SCM), scalenes, and trapezius muscles, cause a greater force on the anterior-superior pull on the sternal angle, resulting in an elevated sternal angle. The manubrium (the top portion of



CASE STUDY—cont'd

the sternum) is calcified at birth, whereas the body of the sternum is primarily cartilaginous.⁷¹ Perhaps that is why the solid manubrium tilts superiorly with the pull of the SCM muscles, whereas the less stable sternal body is less likely to be drawn upward. This in turn causes greater superior expansion of the chest at a loss of anterior chest excursion (decreased circumferential chest wall excursion), leading to chest wall restrictions. In addition, children like Jonathan tend to initiate inspiration with a greater effort to overcome the increased airway resistance from asthma, creating a larger negative inspiratory force (NIF) and more collapsing forces on the chest wall.^{21,88} Clinically, this is observed as an excessive inferior descent of the diaphragm (low abdominal excursion) with flat or paradoxical intercostal movement (inward movement of the middle or lower chest wall). I believe that, over time, the repeated excessive NIF contributed to a decreased developmental stimulus for the activation of the intercostal muscles, thus setting up a pattern of muscle imbalance along Jonathan's chest wall and contributing to the further development of his pectus excavatum and associated rib cage and thoracic spine restrictions.

This is a pattern that I see repeated in numerous other cases in which asthma limits the child's participation in normal activities from infancy through puberty. I believe that the neuromuscular recruitment patterns developed early in life because of the child's ventilatory needs result in musculoskeletal abnormalities and neuromuscular imbalance of the respiratory/postural trunk muscles for movement. This is unique to childhood asthma because of the maturation and development of their systems versus adult-onset asthma where the motor systems have already completed typical development.

Evaluation of Examination Results: Impairments of the Neuromuscular, Musculoskeletal, Integumentary, Cardiovascular/Pulmonary, and Gastrointestinal Systems

1. From a medical perspective, Jonathan's asthma was well managed, but it was still limiting participation in typical childhood activities. Thus, his cardiopulmonary system was not the only impaired system. Typical secondary medical impairments such as GERD were not overtly present, but daily underhydration was likely a significant contributor to his EIA response.
2. Jonathan demonstrated muscle imbalance in quiet and strenuous breathing. It appeared that he could benefit from learning new motor strategies to breathe effectively and efficiently (neuromuscular retraining) in order to better support ventilatory needs simultaneously with the postural demands of the task.
3. Jonathan demonstrated numerous chest wall and spine restrictions, but no integumentary restrictions. He needed more musculoskeletal mobility to support adequate internal lung expansion at low energy cost and decrease the triggers that caused his EIA response, such as rapid inspiratory airflows. This mobility was necessary before neuromuscular retraining could be effectively undertaken, and before adaptive cardiopulmonary strategies could be optimized. Thus, with his asthma well managed from a

medical perspective, the musculoskeletal system presented the first obstacle to his optimal physical function and endurance.

Therefore, in spite of the fact that his primary diagnosis was cardiopulmonary, this examination pointed to significant musculoskeletal and neuromuscular impairments associated with Jonathan's medical diagnosis. Large-scale literature reviews of breathing retraining such as the Cochrane Reviews have been more plentiful in the past few years, but they generally apply to adult populations. Although authors of these reviews continue to conclude that the evidence for strengthening respiratory muscles or neuromuscular retraining of breathing patterns is inconclusive based on a lack of controlled studies or the small number of available controlled studies, they specifically express the opinion that the current state of the evidence is such that one cannot conclude that breathing retraining doesn't work, just that there is not enough hard evidence to make a decision either way.^{39,55,92} The result is similar for manual therapy musculoskeletal interventions.⁵⁶

Diagnosis

Jonathan is a 9-year-old boy with a history of severe EIA and marked pectus excavatum. Significant restrictions in his chest wall mobility and posture, as well as motor planning deficits and underhydration, appear to contribute to limitations in breath support and endurance for his desired functional activities and contribute to the continued development of the pectus excavatum and other postural deformities by perpetuating trunk muscle imbalance and an increased work of breathing.

Prognosis

Jonathan's parents have rejected a surgical option to reduce his pectus, and thus his prognosis was related to the potential success of a noninvasive physical therapy program. I believed that Jonathan had an excellent prognosis for the following reasons: (1) he was closely followed from a medical perspective, (2) he was neurologically intact and capable of developing new motor strategies, (3) his musculoskeletal deformities were functional, not fixed, and he was still prepubescent, and (4) just as important, Jonathan was extremely motivated by his desire to "make" the traveling soccer team and his desire to be able to take his shirt off without embarrassment because of the pectus. His mother was completely committed to helping her son maximize any opportunity to improve his health and well-being, including doing daily exercises at home under her supervision, if necessary. With this high level of support from the patient and his family, I anticipated making maximal progress with about 6 to 12 visits over a 1-year time frame.

Physical Therapy Procedural Interventions and Outcomes

The goals of Jonathan's physical therapy program are listed in detail in Tables 25-4 and 25-5, and the physical therapy interventions are summarized in Tables 25-5 and 25-6. These represent typical goals and intervention strategies for many children with

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CASE STUDY—cont'd

TABLE 25-4 Goals of Physical Therapy Program

Physical Therapy Goals	Jonathan's Goals
Long-term goal	Reduce secondary impairments that limit Jonathan's ability to achieve his desired level of physical activity performance and participation (soccer, baseball, swimming etc.) and health (missed days of school, ER visits, sicknesses).
Short-term goals	<p>Increase joint mobility of rib cage and thoracic spine to promote full ROM for optimal breath support, full trunk movements to optimize skilled movements of the trunk musculature, decrease forces promoting developing kyphosis, as well as decrease forces promoting developing pectus excavatum.</p> <p>Improve muscle strength and muscle balance between diaphragm, intercostals, abdominals, paraspinals, scapular retractors, and neck muscles to normalize forces on the developing spine (decrease kyphosis), ribs (increase individual rib movement potential), sternum (decrease pectus forces), and shoulder (decrease anterior humeral head positioning and potential shoulder ROM losses).</p> <p>Improve motor planning of trunk muscle recruitment for respiration and posture by: Changing the sequence of activation of respiratory muscles to promote sooner activation of intercostal muscles, thus preventing paradoxical chest wall movement, which increases pectus forces (greater negative inspiratory forces reinforce development of a pectus if intercostals are weak, paralyzed, or delayed).</p> <p>Refining the respiratory pattern during quiet and stressful breathing to improve endurance by teaching Jonathan to utilize his diaphragm (endurance muscle) for a greater percentage of the ventilatory workload, and to decrease his over-recruitment of accessory muscles (short burst supporters) during quiet breathing.</p> <p>Refining recruitment pattern of postural muscle to:</p> <ul style="list-style-type: none"> Increase recruitment of intercostals, oblique abdominal and transverse abdominal muscles, scapular retractors, and paraspinals. Decrease over-recruitment of rectus abdominus and sternocleidomastoid (SCM). Improve core trunk movements so that the intercostals, oblique abdominals, and transverse abdominal muscles become the primary stabilizers of the mid trunk, thus avoiding the SCM being overutilized as the primary trunk flexor, which can cause rib elevation, forward head, and eventually rib flares from underuse of oblique abdominals. <p>Improve coordination of breathing with movement to improve oxygen transport during an activity (improving endurance) and to optimize the coordination between the respiration and postural demands of any physical task in order to improve overall physical performance from simple tasks such as activities of daily living to demanding tasks such as soccer.</p> <p>Improve patient and family's understanding of how they can more effectively manage the adverse effects of asthma on Jonathan's posture and movement patterns in order to reduce external triggers that precipitate his asthma attacks. This includes improving his overall hydration levels especially during athletic activities, decreasing activities that result in rapid changes in inspiratory airflow demands (slower warm-ups), and improving the timing of his asthma medications with strenuous activities.</p>

TABLE 25-5 Physical Therapy Interventions

Impairment Category	Interventions for Jonathan
Asthma (cardiopulmonary) management strategies	<ul style="list-style-type: none"> Increased hydration to decrease extrinsic EIA triggers Improved timing of medications with activity level to get maximal benefit of medication Developed and implemented a new warm-up protocol for soccer practices and games that slowly increased his respiratory work load to avoid dramatic changes in inspiratory lung volumes and speed to avoid EIA trigger such as initiating a walk/run warm-up rather than running only, with gradual increase in running time and speed and stretching all trunk musculature prior to soccer Coordinated ventilatory strategies with movement and stretching to decrease respiratory workload and EIA trigger Improve efficiency of movement with resultant improved endurance Implement breath control techniques to prevent or minimize EIA attacks Improve awareness of oncoming EIA symptoms Use controlled breathing techniques to ward off EIA attack when possible



CASE STUDY—cont'd

TABLE 25-5 Physical Therapy Interventions—cont'd

Impairment Category	Interventions for Jonathan
Musculoskeletal interventions	Rib cage mobilization to increase chest wall and thoracic spine mobility in order to reduce respiratory workload and increase likelihood of recruiting intercostal muscles for more efficient respiration and support for developing thorax (reducing pectus excavatum forces) Intercostal muscle release to optimize length-tension relationship Quadratus lumborum muscle release to promote activation of oblique and transverse abdominis muscles for lower trunk stabilization instead of quadratus Active assistive anterior and axial glides to thoracic spine Home program to maintain newly gained trunk mobility
Neuromuscular interventions	Specific diaphragmatic training from recumbent to upright positions, and eventually to sporting conditions Emphasis on slow, easy effort during initiation of inhalation to prevent overpowering developing intercostal muscles Increased recruitment and strength of intercostals for all breathing patterns, postural control, and skeletal development (reducing pectus, paradoxical breathing, and thoracic kyphosis) Specific coordination of inhalation/exhalation patterns with all activities (ventilatory strategies) Increased recruitment and strength of scapular adductor, shoulder external rotators, and paraspinals for increased posterior stabilization Lengthening of neck accessory muscles through active stretching Midtrunk stabilization exercises (reducing rib flares and improving midtrunk interfacing between intercostals abdominals)
Integumentary interventions	None needed at this time
Internal organs (gastrointestinal) interventions	Increase hydration, especially during sporting activities

TABLE 25-6 Lateral Trunk Flexion Mobility Test for Rib Cage and Quadratus Lumborum

Test	Initial Date	Discharge Date 11 Months Later	Reevaluation 4 Years After Discharge
Lateral side bend toward L: mobility of right rib cage	2¼"	4½"	3⅝"
Lateral side bend toward L: mobility of right quadratus lumborum	1"	3"	2⅝"
Lateral side bend toward R: mobility of left rib cage	1½"	3¾"	3"
Lateral side bend toward R: mobility of left quadratus lumborum	1¼"	2½"	2⅝"

Note: From initial evaluation to discharge 11 months later, Jonathan's rib cage mobility doubled on the right, and more than doubled on the left. His quadratus lumborum length tripled on the right and doubled on the left. At the 4-year follow-up examination, he had lost some mobility at all levels except the left quadratus lumborum.

asthma and can be adapted for other cases or age ranges. All goals are developed in the context of a patient-centered care plan as research and clinical experience shows better long-term outcomes and carryover.^{107,109}

Asthma (Cardiovascular/Pulmonary and Gastrointestinal) Procedural Interventions

Jonathan was instructed in immediate changes that he could implement at school, home, and on the soccer field to decrease the triggers

that set off his EIA response. He was extremely sensitive to a sudden increase in inspiratory volumes and flow rates that occurred secondary to soccer warm-ups, which started with laps around the soccer field. It was likely that the combination of (1) the dryness in his airway caused by the change from nose breathing to mouth breathing because of the sudden need for increased inspired air during the running activity and (2) the large, fast moving volume of air required to perform this high level of exercise played a significant role in

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CASE STUDY—cont'd

triggering an acute attack.^{5,50} Within 5 to 10 minutes of soccer, he would typically experience such extreme shortness of breath that he was forced to stop playing. Often he did not recover in time to rejoin his teammates.

Jonathan's management program included several steps:

1. He was instructed in ways to increase his hydration overall, and specifically to use hydration before and throughout the games and practices in order to keep his upper airway moist.
2. He began to take his medications sooner, at least 15 to 30 minutes before the start of soccer, to receive the maximum benefit from the drugs.
3. He started a new warm-up that slowly increased his activity level so that the oxygen demand gradually increased, allowing him to breathe through his nose for a longer period of time and allowing the necessary inspired air volume to also increase slowly.
4. He stretched his trunk, spine, rib cage, and shoulders before the game to maximize mobility (compliance) of his chest wall movements, thus decreasing his work of breathing and reducing the negative pressures (NIF) that he needed to generate to inhale adequate volumes of air, which in turn reduced the collapsing forces on his chest wall.
5. He coordinated his breathing specifically with the relationship of the trunk movement and rib cage during each stretching exercise and movement in general to reinforce normal pattern combinations of movement and breathing (ventilatory strategies).^{37,72}
6. He was taught two particular breath control techniques to help him regain control of breathing during the early stages of an asthmatic attack: (a) repatterning controlled breathing technique and (b) an enhanced Jacobsen's progressive relaxation technique. These techniques were developed by Frownfelter.³⁷

Repatterning controlled breathing technique

- 6a. The patient is asked to start with exhalation. "Try to blow out easily with your lips pursed. Don't force it just let it come out." Suggesting that the patient visualize a candle with a flame which their exhalation makes flicker but not go out will help to produce a prolonged, easy exhalation. Doing this allows the respiratory rate to decrease automatically. When the patient feels some control of this step, then ask him or her to "hold your breath at the top of inspiration just for a second or two." Make sure the patient does not hold his or her breath and bear down as in a Valsalva maneuver. Last, ask the patient to take a slow breath in, hold it, and let it go out through pursed lips. Patients learn that when they are short of breath, this technique often helps them to gain control, making them feel less panicky.³⁷ Jacobsen's progressive relaxation technique.
- 6b. Utilizes ventilatory strategies to help the patient experience the difference between inhaling and contracting the upper trapezius versus exhaling and relaxing the trapezius in order to develop new motor strategies to keep the trapezius from being over recruited.³⁷

Using a mirror for feedback, the patient is instructed to lift his shoulders while inhaling and is then asked to hold his breath at the peak of shoulder elevation. The therapist applies maximal downward resistance while telling the patient to "hold." The patient is then instructed to "let go" slowly and to exhale with gentle pursed lip breathing while the therapist slowly applies axial rotation to the spine through the shoulders. The purpose of the activity is to help the patient feel and see the difference between excessive recruitment of the trapezius and relaxation of the trapezius.

Asthma Management Outcomes

Jonathan rigorously followed the regimen including carrying a water bottle with him everywhere, even in the classroom. He noticed an immediate decrease in chest tightness and dyspnea during soccer practice and games. Of particular note, before using the repatterning controlled breathing technique, Jonathan said he had no way to stop the progression of his asthma attack once it started. Now he said that if the attack was mild he was able to "work through it" with the repatterning, and it did not develop into a full-blown attack. He could now play a whole game of soccer without EIA preventing his participation. In fact, he made the travel soccer team and could play four consecutive games of soccer in 1 day without EIA symptoms. As a consequence of decreasing EIA triggers, Jonathan began having fewer and fewer asthmatic attacks, such that all asthma medications were discontinued 2 months after starting physical therapy. This was not an intended consequence of physical therapy, but a welcomed one. Jonathan reported only one incident of bronchitis in the following year and no asthma attacks after 2 months of physical therapy.

Musculoskeletal Interventions

Jonathan needed increased chest wall and spine mobility before attempting neuromuscular training of muscles along that tight rib cage. Jonathan was positioned in side lying with a large towel roll placed under his lower ribs to maximize rib expansion on the uppermost side (Figure 25-5, A). Manual rib mobilization was performed to all 10 ribs bilaterally to increase individual rib movement potentials, to increase rib cage compliance, and to increase the potential for axial rotation of his thoracic spine (a tight rib cage makes lateral or axial movements of the thoracic spine less possible).⁸⁰ From the results of my testing, the intervention was focused more on the left side than the right, and more in the midchest than the upper or lower chest (Figure 25-5, B). This was followed by intercostal muscle release techniques to maximize intercostal spacing and optimize their length-tension relationship for neuromuscular retraining (Figure 25-5, C). Finally, his quadratus lumborum was released bilaterally to allow for more separation between the rib cage and the pelvis (Figure 25-5, D). Posteriorly, the thoracic spine was only mildly restricted in anterior glides (extension of spine) and axial rotation, so active assisted mobilizations were incorporated into his home program. Jonathan worked on maintaining his newfound trunk mobility with a home stretching program.



CASE STUDY—cont'd



Figure 25-5 PT musculoskeletal interventions to increase chest wall mobility (demonstrated with a female, not with Jonathan). **A**, Start position. Note towel roll under the patient's lower ribs to maximize chest expansion. **B**, Rib mobilization technique for the midribs. Similar positioning would be used for other ribs. **C**, Intercostal muscle release (lengthening technique) on lower ribs. In this picture, the therapist's thumb is between ribs 8 and 9 in the intercostal spacing. The release movement proceeds from posterior to anterior on the chest wall. **D**, Quadratus lumborum (QL) muscle release (lengthening technique). The therapist's right hand is lengthening the QL while the left hand is stabilizing the pelvis.

Musculoskeletal Outcomes

Jonathan made tremendous progress in trunk mobility as measured by range of motion in lateral trunk flexion (Figure 25-6). His rib cage mobility doubled on the right and more than doubled on the left. His quadratus lumborum length tripled on the right and doubled on the left (see Table 25-6). His anterior glides and axial rotation glides of thoracic spine were now normal.

His pectus excavatum volume, which was measured using a water displacement method, was 34 mL H₂O when measured 4 months into treatment (Figure 25-7). The volume was reduced by half to 18 mL H₂O at discharge 7 months later (Table 25-7). (Pectus volume was not measured on initial evaluation.)

Postural assessment showed elimination of functional kyphosis in sitting and standing postures. Jonathan no longer showed a midtrunk

"fold" in a sitting posture. Mother and son reported that his teachers no longer continually reminded him to sit up straight in school. Inferior rib flares were no longer apparent as his abdominal muscles now adequately stabilized the rib cage at the midtrunk and his primary neuromuscular recruitment pattern now utilized his abdominal muscles instead of his sternocleidomastoid muscles as his primary trunk flexor. His sternal angle elevation appeared slightly reduced but was not objectively measured.

Neuromuscular Interventions

The priorities of Jonathan's physical therapy were to address his medical needs first, then his musculoskeletal restrictions, and finally his neuromuscular impairments. Jonathan needed to balance the strength and recruitment patterns of his respiratory and postural muscles to optimize breath control at a low energy cost, while

Continued



CASE STUDY—cont'd



Figure 25-6 Lateral trunk flexion measurement sites. **A**, Lateral trunk flexion movement. Total excursion was measured in two segments. **B**, Rib cage mobility was measured from a full upright position to the end lateral trunk flexion position. The starting points for the tape measure were the midaxillary line (head of the humerus) superiorly to rib 10 (lowest palpable rib) inferiorly. Total movement in inches was recorded. **C**, The mobility of the quadratus lumborum, and to a lesser extent the gluteus medius, was measured likewise from rib 10 superiorly to the greater trochanter inferiorly.

TABLE 25-7 Other Tests and Measures

Test	Initial Date	Discharge Date 11 Months Later	Reevaluation 4 Years after Discharge
Pectus volume displacement (typical: zero or minimal volume)	34 mL (taken 4 months after initial evaluation)	18 mL	17 mL
Respiratory rate (typical 10–20)	20	11	–
Auscultation	Clear	Clear	Clear
Phonation (typical 10 seconds)	20 sec	25.5 sec	28.6 sec
PFTs (pulmonary function tests)	Normal lung volumes and flow rates	Not taken	Normal lung volumes and flow rates

Note that Jonathan's pectus excavatum, which was 34 mL H₂O when measured 4 months into treatment, was reduced by half to 18 mL H₂O at discharge and was maintained relatively at the same level when remeasured 4 years later.



CASE STUDY—cont'd



Figure 25-7 Pectus excavatum volume displacement measurement. The patient is positioned supine with the pectus deformity parallel to the floor (in this picture, the patient is slightly tilted superiorly and would need to have his upper trunk raised slightly with pillows to make the pectus parallel to the surface). A volume displacement measurement is used as a crude estimate of the extent of the volume of the pectus. Water from a measured syringe fills the pectus until spilling over the deformity. The test is repeated three times or more until consistent. If a consistent measurement cannot be made, the measurement is discarded.

simultaneously providing appropriate muscle force to his developing spine and rib cage—quite a balancing act.^{28,43,52,54,75,76,111,112,115}

The neuromuscular retraining of Jonathan's respiratory muscles started with specific diaphragmatic training in a sidelying posture to facilitate a more optimal length-tension relationship of the diaphragm while simultaneously facilitating a less optimal length-tension relationship of the upper accessory muscles to minimize their

recruitment during quiet breathing. Jonathan did not respond with increased diaphragmatic recruitment and excursion with positioning and verbal cues alone, so manual facilitation techniques were added.

Several techniques were used, but the one that produced the greatest consistency, reproducibility, and appropriate timing of activity in the diaphragm was the "diaphragm scoop" technique, which is an indirect facilitation technique to the diaphragm's central tendon

Continued



CASE STUDY—cont'd

area.⁷⁷ This technique provided specific quick stretch input to the central tendon of the diaphragm via the patient's abdominal viscera at the end of the expiratory cycle in an effort to recruit the central tendon as the initiator of the next inspiratory effort. Continued manual cueing was provided throughout the entire inspiratory phase to facilitate greater inferior excursion of the diaphragm. An emphasis was placed on initiating inspiration with an "easy, slow onset" to avoid recruitment of the upper accessory muscles and an overpowering of his intercostal muscles (paradoxical breathing). Once the patient could consistently succeed in recruiting his diaphragm in sidelying, he was challenged by decreasing manual input and increasing postural demands by using positions such as sitting and standing. At this point Jonathan was instructed to practice this technique using visualization at home just before sleeping to take advantage of a relaxed state. Eventually, he was trained to use the diaphragm breathing technique in sports as well as static postural holds. Auditory cues for the rate, rhythm, and depth of inspiration were included in all breathing retraining techniques. Objective measures of his success were taken with assessment of chest wall excursion (CWE).^{73,74,85}

Jonathan demonstrated poor recruitment of his external intercostal muscles, which are needed to stabilize the chest wall during inspiration to prevent paradoxical breathing and the potential development of a pectus excavatum secondary to this inward force.⁸² Jonathan demonstrated this paradoxical chest wall movement even at rest in his mid-rib cage. Thus, weak intercostals could be, in part, responsible for the development of his pectus. I used manual facilitation techniques with (1) upper extremity flexion, abduction, and external rotation activities (D2) diagonals from proprioceptive neuromuscular facilitation (PNF)⁶⁸ combined with (2) thoracic extension and rotation; intentionally paired with (3) large inspiratory efforts, to utilize optimal length-tension relationships and function of the external intercostals; and (4) a maximal inspiratory effort followed by a

peak inspiratory hold to increase positive outward pressure on the anterior chest wall (Figure 25-8). Jonathan was instructed to visually follow his arm motions to maximize the trunk rotation because thoracic rotation produces greater intercostal muscle recruitment than straight plane motions.^{57,86} Jonathan was instructed to continue the exercises at home once he could demonstrate the proper recruitment pattern.

Another chest wall exercise was added. Jonathan was positioned supine lying on a vertical thoracic towel roll to maximize his thoracic spine extension and to stabilize the costotransverse junctions (junction of the ribs to the transverse process of the thoracic spine). Jonathan was then instructed to externally rotate his shoulders while pinching his shoulder blades back to the towel roll to maximize anterior chest expansion by recruiting the external intercostals and the pectoralis muscle (using the pectoralis muscles to act as a chest wall expander rather than an upper extremity adductor). The position also stretched his neck flexors. During this activity, he was instructed to take in a deep breath and hold it during a PNF hold-relax technique to maximize the response from his scapular retractors.⁸² This provided maximal positive pressure from within his chest cavity, which provided a significant counterforce to the pectus, thus lifting his anterior chest.^{9,78} This concept can be achieved independently by substituting a resistive band instead of the therapist's hands (Figure 25-9).

Jonathan's abdominal muscles were often recruited concentrically for exhalation. To retrain the abdominals for quiet breathing, Jonathan was given eccentric trunk exercises to be done during his warm-up for soccer where he had a history of asthma exacerbations. He was instructed to pair eccentric exhalation (quiet speech) with eccentric trunk movements to reinforce the natural coupling of breathing and postural control responses.^{74,84} For example, during knee lunge exercises, Jonathan would count out loud while going into a controlled lunge (eccentric task) and then purposely inhale as he

Figure 25-8 Home exercise program. Prone on elbows. This is one activity that Jonathan did at home to promote intercostal muscle activation and strengthening as well as to promote spinal axial rotation and chest wall expansion.





CASE STUDY—cont'd

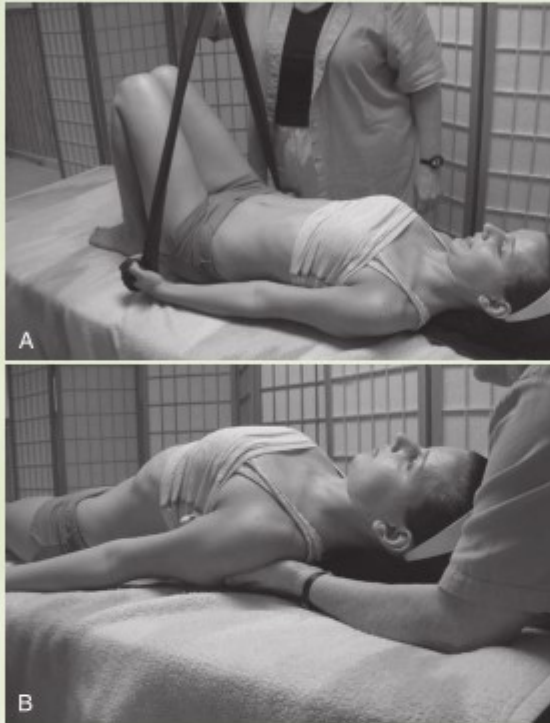


Figure 25-9 Home exercise program. Supine over a vertical towel roll. **A**, This exercise is a variation of the one used for Jonathan in the clinic. It is used to increase thoracic extension and to elevate the anterior rib cage. Resistance is applied to trunk extension, rib cage expansion, and shoulder external rotation via a resistive band such as Thera-Band as the patient pulls the band downward toward the floor. **B**, In Jonathan's case, resistance was applied via the therapist's hands in the clinic (demonstrated here with a female, not with Jonathan).

came back up to stance. Jonathan would adapt this concept to the variety of soccer warm up exercises.

Lastly, Jonathan was instructed in specific recruitment of internal intercostals and oblique abdominal muscles as the primary stabilizers of the inferior rib cage to (1) decrease the rib flare deformity, (2) improve midtrunk stabilization to offer the diaphragm better mechanical support, (3) reduce his overdependence on the rectus muscle for stabilization, which again reinforced the development of the pectus, and (4) provide stability of the rib cage during activation of the SCM muscles to prevent the chest from being lifted toward the head when Jonathan's intended movement was to bring his head to the chest. Once again, a PNF D2 upper extremity pattern was used.⁶⁸ This time, the patient was positioned supine with his arm positioned in flexion, abduction, and external rotation while lying over a vertical towel roll. The patient's arm was stabilized distally. The patient was asked to "try to lift his arm up in the diagonal pattern" but was not allowed any movement. The result was a strong isometric contraction of the midtrunk muscles (oblique and transverse abdominis and internal intercostals), which are required for stabilization of the trunk before the distal extremity could be moved off the ground. This allowed him

to perform small concentric contractions of his internal intercostals and external obliques without being overpowered by the rectus. When the patient successfully demonstrated consistency in recruiting these muscles, which was observed by a flattening of the rib flares during the active contractions of the intercostals and obliques, he was instructed to carry over the training independently with higher level postures and longer periods of dynamic stabilizations such as holding his trunk while continuing to breathe through longer segments of activities like running.

To improve recruitment of thoracic paraspinal muscles, rather than primarily lumbar extensors (to decrease kyphotic forces), Jonathan was instructed in (1) full upper extremity swings in standing during soccer warm-up routine, (2) coordinating slow inhalation with shoulder abduction and scapular adduction, and (3) coordinating eccentric exhalation (counting out loud) when he returned his arms down to his side. He was instructed to focus on recruiting diaphragm and intercostal muscles during inhalation (which should recruit more thoracic extensors) and to concentrate on controlling the eccentric component of the arm and trunk muscles during exhalation. If Jonathan was my new patient today, I would include more specific

Continued



CASE STUDY—cont'd

TABLE 25-8 Chest Wall Excursion (CWE) in Sitting and Supine Positions

Tidal Volume Sitting (Quiet Spontaneous Breathing)	Initial Date	Discharge Date 11 Months Later	Reexamination 4 Years after Discharge
Upper chest (level of 3rd rib) upper accessory muscles	$\frac{1}{2}$ "	$\frac{1}{2}$ "	—
Mid chest (level of xiphoid) intercostals	$\frac{1}{4}$ "	$\frac{3}{4}$ "	—
Lower chest (half the distance from xiphoid process to navel) lower intercostals and diaphragm	$\frac{1}{8}$ "	$\frac{3}{4}$ "	—

Tidal Volume Supine (Quiet Spontaneous Breathing)	Initial Date	Discharge Date 11 Months Later	Reexamination 4 Years after Discharge
Upper chest (level of 3rd rib) upper accessory muscles	$\frac{1}{8}$ "	$\frac{1}{2}$ "	$\frac{1}{4}$ "
Mid chest (level of xiphoid) intercostals	0"	$\frac{1}{4}$ "	0"
Lower chest (half the distance from xiphoid process to navel) lower intercostals and diaphragm	$\frac{1}{4}$ – $\frac{1}{2}$ "	$\frac{1}{4}$ "	$\frac{1}{4}$ "

Note that, in sitting, improvements were noted in mid and lower chest expansion. No 4-year follow-up measurements.

In supine, all levels increased by discharge, but at the 4-year follow-up examination, the gains in the mid and upper chest had disappeared. Only the lower chest expansion continued to show similar levels to the discharge values.

training for the transverse abdominis which has been shown to be associated with diaphragmatic movements.^{38,52,58}

Neuromuscular Outcomes

Jonathan now demonstrated an effective balance between the primary respiratory muscles (diaphragm, intercostals, and abdominals) during volitional and spontaneous breathing in both quiet breathing and maximal inspiratory maneuvers in multiple postures and activities. Paradoxical movement of the chest wall was no longer noted (improved functional strength of intercostal muscles). No functional thoracic kyphosis was noted during quiet stance or during active recruitment of trunk extensors. Quiet breathing now demonstrated a normal recruitment pattern: (1) initiation of inhalation with the diaphragm and simultaneous chest wall movement, (2) easy inspiratory onset, no apparent effort (low work of breathing, low negative inspiratory force which reduces pectus forces), and (3) smooth continuous movements throughout the inspiratory cycle. Objectively, this was seen with (1) significant increases in mid-chest wall excursion measurements (intercostal recruitment) during quiet breathing (tidal volume) in both supine and standing (Table 25-8), (2) a respiratory rate that decreased from 20 to 11 breaths/minute, and (3) phonation support in syllables/breath that increased by 28% (see Table 25-7). Midtrunk stabilization showed marked improvement in strength of oblique abdominal muscles, right still stronger than left. Posturally, this was noted by the minimization of his rib flares and appropriate timing recruitment of the abdominals during trunk stabilization activities both in therapy and, as reported by the patient, during sports activities. Functionally, Jonathan reported that he could now run the mile at school without excessive dyspnea or asthmatic symptoms.

Jonathan needed maximal sensory and motor input to change his motor strategies for respiration. Verbal cues alone did not produce satisfactory results. Manual, visual, auditory, and positional input in each activity was specifically applied to assist Jonathan in developing new motor plans to improve breathing efficiency and appropriate skeletal forces that promoted normal development of his rib cage and spine.

Integumentary Interventions: None needed.

Functional Outcomes and Quality of Life Issues

Following his physical therapy program, Jonathan and his mother noted important functional improvements (Table 25-9). He made the travel soccer team and could play four consecutive games without EIA attacks. His last EIA episode occurred 2 months after starting physical therapy. Before physical therapy, he had an EIA episode almost every time he played soccer. At discharge, he could also run the mile in gym class at school without EIA or excessive dyspnea.

He did not miss any days of school for EIA after initiating physical therapy. His mother said that before the physical therapy program, "he would miss 5 to 8 days a year because of sickness related to EIA, but those sick days don't take into account the weekends, holidays, and summer days that Jonathan was incapacitated with asthma-related problems." He had two severe EIA episodes before physical therapy that resulted in emergency room (ER) visits. During his physical therapy interval, he did not have any ER visits.

His mother said that in addition to making it possible for him to rejoin his classmates in regular physical activity such as soccer and baseball, following the year of physical therapy Jonathan began to go



CASE STUDY—cont'd

TABLE 25-9 Functional Outcomes

Functional Outcomes	Initial Date	Discharge Date 11 Months Later	Reexamination 4 Years after Discharge
EIA attacks or symptoms during sports activities	Frequent	None	None
Length of participation in a sporting activity	5 to 10 minutes before EIA symptoms forced him to stop	Full participation: up to four soccer games per day	Full participation: plays baseball in high school
Complete the "mile test" in gym class	No	Yes	Yes
Average number of days absent from school due to asthma-related complications	5 to 8	0	0
Emergency room visits	2	0	1
Hospitalizations	0	0	0
Daily asthma medications	Yes	No	No

Note that Jonathan's greatest improvements are in activity and health gains.

swimming again. He had all but given up swimming the year before because of "his deformed chest" and the derogatory comments that were directed at him by other children.

When asked for a general statement about how the physical therapy program affected her son's quality of life, Jonathan's mom said: "It was a miracle. Before we began to see you, Jonathan and I had to focus on his medical condition rather than focusing on being a kid. It completely changed his life." Jonathan and his mother no longer saw him as "disabled" by his pulmonary disease.

Discussion

Jonathan was seen for eight visits over 11 months. The family's motivation to follow through diligently on home programs, and the child's excellent ability to learn new motor strategies, resulted in a minimal number of visits to accomplish the goals of treatment. Under different circumstances, achieving the procedural intervention goals in a similar case may take longer or goals may be less attainable.

The results of this particular case were marked, but not unrepeatable. Jonathan's physical therapy program was developed from a multisystem perspective to develop better "external support" for his "internal" asthma. I believe the keys to his success were threefold:

1. A team approach to his condition: recognition by his pulmonologist that his functional limitations were more severe than his medical condition alone indicated, her belief that physical interventions are an integral part of effective management of pulmonary diseases, and her belief that a surgical intervention for his pectus should be the last, not his first, option.
2. A detailed physical therapy examination that focused on identifying the underlying impairments outside of his "asthma and the pectus diagnoses alone," examining both medical and physical impairments to determine which system(s) could account for the severity of his functional limitations.

3. A specific intervention program targeted to reverse or minimize those impairments with a major emphasis on the patient's responsibility in the program (education), and on applying new strategies directly into his daily life (functional).

Although it is possible that his changes were due to maturation, it is unlikely according to his mother, who noted that all of his improvements came after the initiation of physical therapy compared to the previous school year without physical therapy.

Following physical therapy, Jonathan's pulmonary symptoms went into complete remission, which neither this author nor his pulmonologist had anticipated. Physical therapy does not "cure" asthma. Could it be that the EIA diagnosis was not completely accurate? Jonathan had all the symptoms of EIA, but his PFTs did not confirm the diagnosis. Recently, doctors have begun to explore other possible explanations for EIA symptoms that do not fit the classic picture of asthma, such as vocal fold dysfunction or supraesophageal manifestations of GERD, which present with similar symptoms: high sensitivity to fast inspiratory flow rates, a lack of typical asthmatic responses on PFTs, and a lack of significant improvement with asthma medications. Because of Jonathan's dramatic improvement with physical interventions, his pulmonologist is now reconsidering his original diagnosis.

The tests and measures used in this case have varied levels of reliability and validity. The medical tests, such as PFTs and respiratory rates, have long-established reliability and validity.^{24,27} Tests for the physical impairments are not as well established. Tests for phonation length were established in the speech therapy field.²⁹ Inter- and intratester reliability for chest wall excursion (CWE) measurements have been established, but normative standards were not found to be predictable by age and sex. The patient served as his own control.^{73,74,85} Lateral trunk flexion and the pectus volume measurement have not been validated by research.

Continued



CASE STUDY—cont'd

Four-Year Follow-up at Age 14 Years

Jonathan participated in physical therapy for 1 year. Four years after discharge (5 years after initiating physical therapy), Jonathan was contacted, interviewed, and reexamined to assess the long-term effects of this program on his pathology (asthma), his impairments, activity limitations, and participation. Jonathan was 14 years old and a freshman in high school (Figures 25-10, 25-11, and 25-12).

Medical Update

An examination by his pulmonologist showed no limitations noted in PFT volumes or flow rates. He was also reevaluated by his cardiologist who diagnosed an asymptomatic mitral valve prolapse, which is not uncommon with a pectus excavatum.²⁶ No cardiac intervention was needed. He had had only one respiratory episode in the last 5 years: a croup-type virus that resulted in a severe bronchitis and his only trip to the emergency department. He did not have any EIA episodes during the 4-year interval. He did not use daily asthma medication. He did, however, report use of his bronchodilator prophylactically when he had a cold, “just in case.”

Test and Measures

See Tables 25-6, 25-7, and 25-8 for results of the tests.

Functional Outcomes and Quality of Life Update

See Table 25-9 for Jonathan's functional outcomes.

Jonathan received a “perfect attendance award” in eighth grade, which his mother commented was a complete reversal of his school years before physical therapy. Endurance is no longer a limitation according to both Jonathan and his mother.

Jonathan's mother reported that he continues to gain confidence both socially and athletically following the physical therapy intervention. She no longer sees any signs of self-consciousness regarding his

chest wall deformity. This may be a result of maturation, but she thought it was worth noting because it changed so significantly during and following the intervention period.

Even with 4 to 5 years' reflection since the onset of physical therapy, Jonathan's mother still says that the physical, medical, and emotional benefits to her son were incredible. She said that they kept up the home exercises for approximately 4 months after his discharge from physical therapy, but slowly drifted away from them, which may explain some of the minor loss of chest wall mobility upon reexamination. Jonathan did keep using the strategies that he learned in physical therapy such as maintaining adequate hydration levels and proper warm-up before exercise.

Impression

Jonathan has maintained his pulmonary health since discharge 4 years ago with no apparent signs of EIA or its impairments, especially as it affected his endurance and participation in activities and his overall health. At this point, it appears that his asthma or other undiagnosed pulmonary disease is resolved or benign. His spinal alignment is now completely normal, avoiding what appeared to be the likely development of a true thoracic kyphosis. His chest wall deformities are still minimally present and more localized, less noticeable, and do not cause any activity limitations. Recent medical tests also show that his chest wall deformities do not have any measurable impact on his cardiac or pulmonary function. His remarkable gains in individual rib cage mobility from the initial visit to discharge (lateral side bending test) have been nearly retained. Jonathan and his mother stated that they wished they had continued with periodic physical therapy rechecks to maintain all the gains he made during that first year.

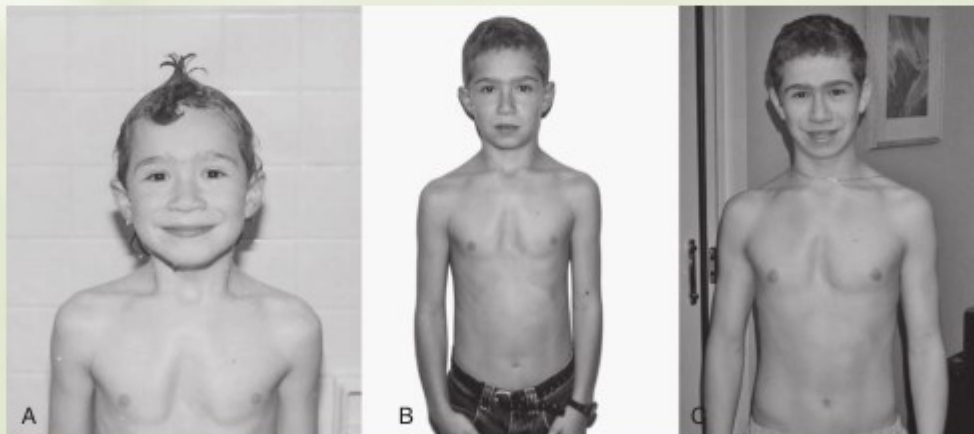


Figure 25-10 Comparison of pectus excavatum and postural alignments. **A**, Jonathan at 6 years old. **B**, Jonathan at 10 years old. **C**, Jonathan at 14 years old. By age 14, Jonathan's pectus has become narrower and more localized. Shoulders are less protracted, resulting in a more neutral resting position. His trapezius is less elevated, and although no rib flare is noted in either standing posture, adequate abdominal stability is more apparent at age 14.



CASE STUDY—cont'd

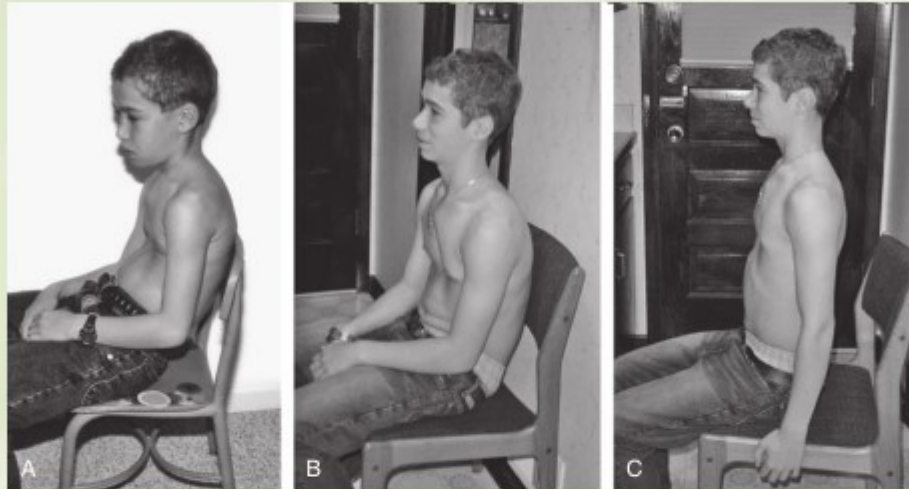


Figure 25-11 Comparison of sitting postures. **A**, Typical sitting posture in school per his mother: age 9 and younger. This is a reenactment picture taken at age 10. Jonathan was too embarrassed to have his picture taken of his “deformed chest” when he was initially evaluated at age 9. Note slouched posture (functional kyphosis) with midtrunk fold, pectus, elevated sternal angle. By age 10, patient no longer regularly postured himself like this in sitting. **B**, Jonathan at 14 years old. When asked to slouch in sitting, the midtrunk fold and kyphosis are barely noticeable. Prominent sternal angle is still noticeable. **C**, Straight sitting posture at age 14 years old. Note normal back posture. Mild pectus and mild rib flare still present at base of sternum.

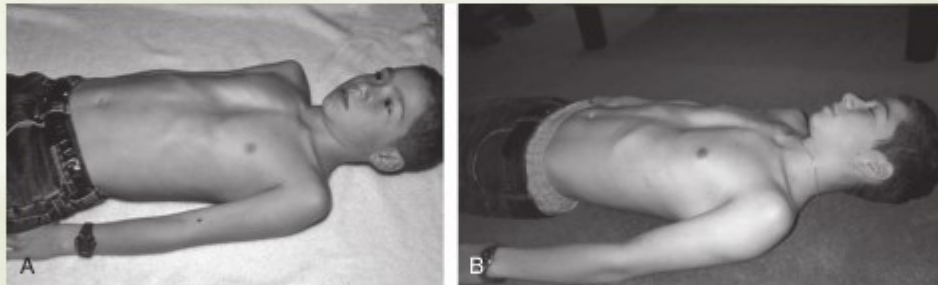


Figure 25-12 Comparison of supine postures. **A**, Discharge picture at age 10. Pectus was reduced almost in half from 34 to 18 mL H₂O displacement measurement during the 11 months of physical therapy. Lower rib flares functionally integrated with abdominal muscles. Neck muscles more elongated. Slight shoulder protraction still noted. **B**, Four years later at age 14. Pectus slightly deeper, but narrower (volume unchanged from discharge at 17 mL). Rib flares more prominent than at discharge. Patient stated that he stopped doing his trunk exercises about 4 months after discharge because he was doing so well. Neck muscles more elongated. Shoulders less protracted.

I believe Jonathan’s physical therapy program worked so well because it was tailored to address his specific EIA pattern and chest wall deformities from a multisystem perspective and included educational, medical, psychologic, and physical perspectives. Interventions by physical therapists can have a tremendous positive impact on the impairments, activity limitations, and resultant disabilities that occur as a result of a primary pulmonary pathology, especially in a maturing system. If the patient cannot breathe efficiently and effectively, then that patient cannot function at his or her highest level. The concepts

presented here for Jonathan can certainly be adapted to infants and toddlers as well as older children. The key is to develop a program that keeps the patient, his family, and his resources in mind while developing a targeted intervention strategy.^{20,22}

Following the reexamination, Jonathan’s home program was updated and reinitiated with an emphasis on maintaining his musculoskeletal alignment and trunk control. I recommended quarterly check-ups throughout puberty to modify the program as necessary.

REFERENCES

- Abrams, S. A. (2001). Chronic pulmonary insufficiency in children and its effects on growth and development. *Journal of Nutrition*, 131(3), 938S–941S.
- Akinbami, L. (2006). *The state of childhood asthma, United States, 1980–2005*. Washington, DC: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, 1–24.
- Allen, D. B. (2002). Safety of inhaled corticosteroids in children. *Pediatric Pulmonology*, 33(3), 208–220.
- Amdekar, Y. K. (2001). Natural history of asthma in children. *Indian Journal of Pediatrics*, 68(Suppl 4), S3–S6.
- Anderson, S. D., & Holzer, K. (2000). Exercise-induced asthma: Is it the right diagnosis in elite athletes? *Journal of Allergy & Clinical Immunology*, 106(3), 419–428.
- APTA (2001). Guide to physical therapist practice, ed. 2. *Physical Therapy*, 81(1).
- Apter, A. J., & Szeffler, S. J. (2004). Advances in adult and pediatric asthma. *Journal of Allergy & Clinical Immunology*, 113(3), 407–414.
- Arguin, A. L., & Swartz, M. K. (2004). Gastroesophageal reflux in infants: A primary care perspective. *Pediatric Nursing*, 30(1), 45–51.
- Bach, J. R., & Bianchi, C. (2003). Prevention of pectus excavatum for children with spinal muscular atrophy type 1. *American Journal of Physical Medicine & Rehabilitation*, 82(10), 815–819.
- Baker, K. M., Brand, D. A., & Hen, J., Jr. (2003). Classifying asthma: Disagreement among specialists. [see comment]. *Chest*, 124(6), 2156–2163.
- Baraldi, E., Carraro, S., & Filippone, M. (2009). Bronchopulmonary dysplasia: Definitions and long-term respiratory outcome. *Early Human Development*, 85(Suppl 10), S1–S3.
- Baum, W. F., Schneyer, U., & Kloditz, E. (2002). Delay of growth and development in children with bronchial asthma, atopic dermatitis and allergic rhinitis. *Experimental & Clinical Endocrinology & Diabetes*, 110(2), 53–59.
- Berhane, K., McConnell, R., Gilliland, F., et al. (2000). Sex-specific effects of asthma on pulmonary function in children. *American Journal of Respiratory & Critical Care Medicine*, 162(5), 1723–1730.
- Bhatia, J., & Parish, A. (2009). GERD or not GERD: The fussy infant. *Journal of Perinatology*, 29(Suppl 2), S7–S11.
- Birkisson, I. F., Halapi, E., Bjornsdottir, U. S., et al. (2004). Genetic approaches to assessing evidence for a T helper type 1 cytokine defect in adult asthma. *American Journal of Respiratory & Critical Care Medicine*, 169(9), 1007–1013.
- Braganza, S., Sharif, I., & Ozuah, P. O. (2003). Documenting asthma severity: Do we get it right? *Journal of Asthma*, 40(6), 661–665.
- Bravata, D. M., Gienger, A. L., Holty, J. E., et al. (2009). Quality improvement strategies for children with asthma: A systematic review. *Archives of Pediatrics & Adolescent Medicine*, 163(6), 572–581.
- Butler, J. E., & Gandevia, S. C. (2008). The output from human inspiratory motoneuron pools. *Journal of Physiology*, 586(5), 1257–1264.
- Callahan, K. A., Panter, T. M., Hall, T. M., et al. (2010). Peak flow monitoring in pediatric asthma management: A clinical practice column submission. *Journal of Pediatric Nursing*, 25(1), 12–17.
- Canavan, P. K., & Cahalin, L. (2008). Integrated physical therapy intervention for a person with pectus excavatum and bilateral shoulder pain: A single-case study. *Archives of Physical Medicine and Rehabilitation*, 89(11), 2195–2204.
- Cappello, M., Legrand, A., & De Troyer, A. (1999). Determinants of rib motion in flail chest. *American Journal of Respiratory & Critical Care Medicine*, 159(3), 886–891.
- 21a. CDC. (2010, May 15, 2009). "Asthma Fast Facts." Retrieved May 8, 2010, 2010, from <http://www.cdc.gov/nchs/fastats/asthma.htm>.
- Centers for Disease Control and Prevention. (1998, April 24). Surveillance for Asthma—United States, 1960–1995. Retrieved May 30, 2004, from www.cdc.gov/mmwr/preview/mmwrhtml/00052262.htm.
- Centers for Disease Control and Prevention. (2009, May 15). Asthma Fast Facts. Retrieved May 8, 2010, from www.cdc.gov/nchs/fastats/asthma.htm.
- Cherniack, R. M., & Cherniack, L. (1983). *Respiration in health and disease* (3rd ed.). Philadelphia: WB Saunders Co.
- Chiang, L. C., Ma, W. F., Huang, J. L., et al. (2009). Effect of relaxation-breathing training on anxiety and asthma signs/symptoms of children with moderate-to-severe asthma: A randomized controlled trial. *International Journal of Nursing Studies*, 46(8), 1061–1070.
- Cole, P. (2001). Pathophysiology and treatment of airway mucociliary clearance: A moving tale. *Minerva Anesthesiology*, 67(4), 206–209.
- Cserhati, E. F., Gegesi Kiss, A., Poder, G., et al. (1984). Thorax deformity and asthma bronchial. *Allergologia et Immunopathologia*, 12(1), 7–10.
- De Troyer, A., Kirkwood, P. A., & Wilson, T. A. (2005). Respiratory action of the intercostal muscles. *Physiological Reviews*, 85(2), 717–756.
- Dean, B. B., Calimlim, B. M., Kindermann, S. L., et al. (2009). The impact of uncontrolled asthma on absenteeism and health-related quality of life. *Journal of Asthma*, 46(9), 861–866.
- Deem, J. F., & Miller, L. (2000). *Manual of voice therapy* (2nd ed.). Austin, TX: PRO-ED.
- Doskocil, M. (1993). Contribution to the study of the development and ossification of human sternum. *Functional and Developmental Morphology*, 3(4), 251–257.
- Dowdee, A., & Ossege, J. (2007). Assessment of childhood allergy for the primary care practitioner. *Journal of the American Academy of Nurse Practitioners*, 19(2), 53–62.

33. Doyle, L. W., Cheung, M. M., Ford, G. W., et al. (2001). Birth weight <1501 g and respiratory health at age 14. *Archives of Diseases in Childhood*, 84(1), 40–44.
34. Eid, N. S., & Morton, R. L. (2004). Rational approach to the wheezy infant. *Paediatric Respiratory Reviews* 5(Suppl A), S77–S79.
35. Fletcher, J. M., Green, J. C., & Neidell, M. J. (2010). Long term effects of childhood asthma on adult health. *Journal of Health Economics*, 29(3), 377–387.
36. Fonkalsrud, E. W. (2003). Current management of pectus excavatum. *World Journal of Surgery*, 27(5), 502–508.
37. Frownfelter, D., & Massery, M. (2006). Facilitating ventilation patterns and breathing strategies. In D. Frownfelter & E. Dean (Eds), *Cardiovascular and pulmonary physical therapy evidence and practice* (4th ed., chap. 23). St. Louis, MO, Elsevier Health Sciences.
38. Gandevia, S. C., Butler, J. E., Hodges, P. W., et al. (2002). Balancing acts: Respiratory sensations, motor control and human posture. *Clinical & Experimental Pharmacology & Physiology*, 29(1–2), 118–121.
39. Garrod, R., & Lasserson, T. (2007). Role of physiotherapy in the management of chronic lung diseases: An overview of systematic reviews. *Respiratory Medicine*, 101(12), 2429–2436.
40. Gern, J. E. (2004). Viral respiratory infection and the link to asthma. *Pediatric Infectious Disease Journal*, 23(Suppl 1), S78–S86.
41. Gerson, L. B., & Fass, R. (2009). A systematic review of the definitions, prevalence, and response to treatment of nocturnal gastroesophageal reflux disease. *Clinical Gastroenterology and Hepatology*, 7(4), 372–378; quiz, 367.
42. Greenough, A., Alexander, J., Boit, P., et al. (2009). School age outcome of hospitalisation with respiratory syncytial virus infection of prematurely born infants. *Thorax*, 64(6), 490–495.
43. Grimstone, S. K., & Hodges, P. W. (2003). Impaired postural compensation for respiration in people with recurrent low back pain. *Experimental Brain Research*, 151(2), 218–224.
44. Hallstrand, T. S., & W. R. Henderson, Jr. (2010). An update on the role of leukotrienes in asthma. *Current Opinion in Allergy and Clinical Immunology*, 10(1), 60–66.
45. Han, J. N., Gayan-Ramirez, G., Dekhuijzen, R., et al. (1993). Respiratory function of the rib cage muscles. *European Respiratory Journal*, 6(5), 722–728.
- 45a. Hansen, E. F., J. Vestbo, et al. (2001). "Peak flow as predictor of overall mortality in asthma and chronic obstructive pulmonary disease." *American Journal of Respiratory & Critical Care Medicine* 163(3 Pt 1): 690–693.
46. Harik-Khan, R. I., Muller, D. C., & Wise, R. A. (2004). Serum vitamin levels and the risk of asthma in children. *American Journal of Epidemiology*, 159(4), 351–357.
47. Hartert, T. V., & R. S. Peebles, Jr. (2000). Epidemiology of asthma: The year in review. *Current Opinion in Pulmonary Medicine*, 6(1), 4–9.
48. Hebra, A. (2004, February 25). Pectus Excavatum at e-Medicine. Retrieved June 2, 2004, from www.emedicine.com/ped/topic2558.htm.
49. Hixon, T. J. (1991). *Respiratory function in speech and song*. San Diego, CA: Singular Publishing Group.
50. Hodges, P., Kaigle Holm, A., Holm, S., et al. (2003). Intervertebral stiffness of the spine is increased by evoked contraction of transversus abdominis and the diaphragm: In vivo porcine studies. *Spine*, 28(23), 2594–2601.
51. Hodges, P. W., & Gandevia, S. C. (2000). Activation of the human diaphragm during a repetitive postural task. *Journal of Physiology*, 522(Pt 1), 165–175.
52. Hodges, P. W., Gurfinkel, V. S., Brumagne, S., et al. (2002). Coexistence of stability and mobility in postural control: Evidence from postural compensation for respiration. *Experimental Brain Research*, 144(3), 293–302.
53. Hodges, P. W., Heijnen, I., & Gandevia, S. C. (2001). Postural activity of the diaphragm is reduced in humans when respiratory demand increases. *Journal of Physiology*, 537(Pt 3), 999–1008.
54. Hodges, P. W., Sapsford, R., & Pengel, L. H. (2007). Postural and respiratory functions of the pelvic floor muscles. *Neurology & Urodynamics*, 26(3), 362–371.
55. Holloway, E., & Ram, F. S. (2004). Breathing exercises for asthma. [Update of Cochrane Database Systematic Reviews, 2000;(3), CD001277; PMID: 10908489.] *Cochrane Database of Systematic Reviews*, 1, CD001277.
56. Hondras, M. A., Linde, K., & Jones, A. P. (2001). Manual therapy for asthma. [Update of Cochrane Database Systematic Reviews, 2000;(2), CD001002; 10796578.] *Cochrane Database of Systematic Reviews*, 1, CD001002.
57. Hudson, A. L., Butler, J. E., Gandevia, S. C., et al. (2010). Interplay between the inspiratory and postural functions of the human parasternal intercostal muscles. *Journal of Neurophysiology*, 103(3), 1622–1629.
58. Indinnimeo, L., Bertuola, F., Cutrera, R., et al. (2009). Clinical evaluation and treatment of acute asthma exacerbations in children. *International Journal of Immunopathology and Pharmacology*, 22(4), 867–878.
59. Jádcherla, S. R., & Shaker, R. (2001). Esophageal and upper esophageal sphincter motor function in babies. *American Journal of Medicine*, 111(Suppl 8A), 64S–68S.
60. Janse, A. J., Sinnema, G., Uiterwaal, C. S., et al. (2008). Quality of life in chronic illness: Children, parents and paediatricians have different, but stable perceptions. *Acta Paediatrica*, 97(8), 1118–1124.
61. Joseph-Bowen, J., de Klerk, N. H., Firth, M. J., et al. (2004). Lung function, bronchial responsiveness, and asthma in a community cohort of 6-year-old children. *American Journal of Respiratory & Critical Care Medicine*, 169(7), 850–854.
62. Kabakus, N., & Kurt, A. (2006). Sandifer Syndrome: A continuing problem of misdiagnosis. *Pediatrics International*, 48(6), 622–625.

63. Kaiser, H. B. (2004). Risk factors in allergy/asthma. *Allergy & Asthma Proceedings*, 25(1), 7–10.
64. Karkos, P. D., Leong, S. C., Benton, J., et al. (2009). Reflux and sleeping disorders: A systematic review. *Journal of Laryngology and Otology*, 123(4), 372–374.
65. Kase, J. S., Pici, M., & Visintainer, P. (2009). Risks for common medical conditions experienced by former preterm infants during toddler years. *Journal of Perinatal Medicine*, 37(2), 103–108.
66. Kennedy, J. D. (1999). Lung function outcome in children of premature birth. *Journal of Paediatrics & Child Health*, 35(6), 516–521.
67. Khoshoo, V., Haydel, R., & Saturno, E. (2006). Gastroesophageal reflux disease and asthma in children. *Current Gastroenterology Reports*, 8(3), 237–243.
68. Knott, M., & Voss, D. E. (1968). *Proprioceptive neuromuscular facilitation*. New York: Harper & Row.
69. Kobernick, A. (2009). Treating GERD in asthma improved lung function. American College of Allergy, Asthma and Immunology. Seattle, WA. *Chest Physician News*, 4, 15.
70. Kuyper, L. M., Pare, P. D., Hogg, J. C., et al. (2003). Characterization of airway plugging in fatal asthma. [see comment]. *American Journal of Medicine*, 115(1), 6–11.
71. Lamberg, E. M., & Hagins, M. (2009). *Evidence supporting the role of breath control in postural stabilization*. American Physical Therapy Association, Combined Sections Meeting 2009. Las Vegas, NV.
72. Lamberg, E. M., & Hagins, M. (2010). Breath control during manual free-style lifting of a maximally tolerated load. *Ergonomics*, 53(3), 385–392.
73. LaPier, T. K. (2002). Chest wall expansion values in supine and standing across the adult lifespan. *Physical and Occupational Therapy in Geriatrics*, 21(1), 65–81.
74. LaPier, T. K., Cook, A., Droegge, K., et al. (2000). Intertester and intratester reliability of chest excursion measurement in subjects without impairment. *Cardiopulmonary Physical Therapy*, 11(3), 94–98.
75. Lee, L. J., Chang, A. T., Coppieters, M. W., et al. (2010). Changes in sitting posture induce multiplanar changes in chest wall shape and motion with breathing. *Respiratory Physiology & Neurobiology*, 170(3), 236–245.
76. Lee, L. J., Coppieters, M. W., & Hodges, P. W. (2009). Anticipatory postural adjustments to arm movement reveal complex control of paraspinous muscles in the thorax. *Journal of Electromyography and Kinesiology*, 19(1), 46–54.
77. Leiner, G. C., Abramowitz, S., Small, M. J., et al. (1963). Expiratory peak flow rate; standard values for normal subjects. *American Review of Respiratory Disease*, 88, 644–651.
78. Lissoni, A., Aliverti, A., Tzeng, A. C., et al. (1998). Kinematic analysis of patients with spinal muscular atrophy during spontaneous breathing and mechanical ventilation. *American Journal of Physical Medicine & Rehabilitation*, 77(3), 188–192.
- 78a. Lozano, P., P. Fishman, et al. (1997). "Health care utilization and cost among children with asthma who were enrolled in a health maintenance organization." *Pediatrics* 99(6): 757–764.
79. Mainz, J. G., Kaiser, W. A., Beck, J. E., et al. (2009). Substantially reduced calcaneal bone ultrasound parameters in severe untreated asthma. *Respiration*, 78(2), 230–233.
80. Massery, M. (2005). Musculoskeletal and neuromuscular interventions: A physical approach to cystic fibrosis. *Journal of the Royal Society of Medicine*, 98(Suppl 45), 55–66.
81. Massery, M. (2006). Multisystem consequences of impaired breathing mechanics and/or postural control. In D. Frownfelter & E. Dean, *Cardiovascular and pulmonary physical therapy evidence and practice* (4th ed., chap. 39). St. Louis, MO, Elsevier Health Sciences.
82. Massery, M. (2009). The Linda Crane Memorial Lecture: The patient puzzle—piecing it together. *Cardiopulmonary Physical Therapy Journal*, 20(2), 19–27.
83. Massery, M., & Cahalin, L. (2004). Physical therapy associated with ventilatory pump dysfunction and failure. In W. DeTurk & L. Cahalin, *Cardiovascular and pulmonary physical therapy* (chap. 19). New York, McGraw-Hill.
84. Massery, M. P. (1994). What's positioning got to do with it? *Neurology Report*, 18(3), 11–14.
85. Massery, M. P., Dreyer, H. E., Bjornson, A. S., et al. (1997). Chest wall excursion and tidal volume change during passive positioning in cervical spinal cord injury. (Abstract). *Cardiopulmonary Physical Therapy*, 8(4), 27.
86. Mellinger-Birdsong, A. K., Powell, K. E., Iatridis, T., et al. (2003). Prevalence and impact of asthma in children, Georgia, 2000. *American Journal of Preventive Medicine*, 24(3), 242–248.
87. Mellon, M., & Parasuraman, B. (2004). Pediatric asthma: Improving management to reduce cost of care. *Journal of Managed Care Pharmacy*, 10(2), 130–134.
88. Metsala, J., Kilkkinen, A., Kaila, M., et al. (2008). Perinatal factors and the risk of asthma in childhood: A population-based register study in Finland. *American Journal of Epidemiology*, 168(2), 170–178.
89. Molle, L. D., Goldani, H. A., Fagondes, S. C., et al. (2009). Nocturnal reflux in children and adolescents with persistent asthma and gastroesophageal reflux. *Journal of Asthma*, 46(4), 347–350.
90. Moloney, E., O'Sullivan, S., Hogan, T., et al. (2002). Airway dehydration: A therapeutic target in asthma? *Chest*, 121(6), 1806–1811.
91. Moloney, E. D., Griffin, S., Burke, C. M., et al. (2003). Release of inflammatory mediators from eosinophils following a hyperosmolar stimulus. *Respiratory Medicine*, 97(8), 928–932.
92. Morris, M., & P. Perkins. (2004, May 9). Asthma. Retrieved June 1, 2004, from www.emedicine.com/med/topic177.htm.
93. Morris, P. J. (2008). Physical activity recommendations for children and adolescents with chronic disease. *Current Sports Medicine Reports*, 7(6), 353–358.
94. National Institutes of Health. (1997). *Practical Guide for the Diagnosis and Management of Asthma Based on Expert Panel Report 2*. NIH Publication No. 97-4053, National Institutes of Health: National Heart, Lung and Blood Institute, 60, Bethesda, Md.

95. National Institutes of Health. (2007). Expert panel report 3: Guidelines for the diagnosis and management of asthma. NIH Publication 07-4051, National Institute of Health: National Heart, Lung, and Blood Institute, Bethesda, Md.
96. Nordenstedt, H., Nilsson, M., Johansson, S., et al. (2006). The relation between gastroesophageal reflux and respiratory symptoms in a population-based study: The Nord-Trøndelag health survey. *Chest*, 129(4), 1051–1056.
97. Openshaw, P. J., Dean, G. S., & Culley, F. J. (2003). Links between respiratory syncytial virus bronchiolitis and childhood asthma: Clinical and research approaches. *Pediatric Infectious Disease Journal*, 22(Suppl 2), S58–S64; discussion S64–S65.
98. Papastamelos, C., Panitch, H. B., & Allen, J. L. (1996). Chest wall compliance in infants and children with neuromuscular disease. *American Journal of Respiratory and Critical Care Medicine*, 154, 1045–1048.
99. Papiiris, S., Kotanidou, A., Malagari, K., et al. (2002). Clinical review: Severe asthma. *Critical Care (London)* 6(1), 30–44.
100. Parish, J. M. (2009). Sleep-related problems in common medical conditions. *Chest*, 135(2), 563–572.
101. Parsons, J. P., & Mastrorarde, J. G. (2009). Exercise-induced asthma. *Current Opinion in Pulmonary Medicine*, 15(1), 25–28.
102. Peterson, K. A., Samuelson, W. M., Ryujin, D. T., et al. (2009). The role of gastroesophageal reflux in exercise-triggered asthma: A randomized controlled trial. *Digestive Diseases and Sciences*, 54(3), 564–571.
103. Philpott, J. E., Houghton, K., & Luke, A. (2010). Physical activity recommendations for children with specific chronic health conditions: Juvenile idiopathic arthritis, hemophilia, asthma, and cystic fibrosis. *Clinical Journal of Sports Medicine*, 20(3), 167–172.
104. Powell, C. V., Kelly, A. M., & Kerr, D. (2003). Lack of agreement in classification of the severity of acute asthma between emergency physician assessment and classification using the National Asthma Council Australia guidelines (1998). *Emergency Medicine (Fremantle, WA)*, 15(1), 49–53.
105. Ram, F. S., Wellington, S. R., & Barnes, N. C. (2003). Inspiratory muscle training for asthma. *Cochrane Database of Systematic Reviews* (4), CD003792.
106. Rimmer, K. P., Ford, G. T., & Whitelaw, W. A. (1995). Interaction between postural and respiratory control of human intercostal muscles. *Journal of Applied Physiology*, 79(5), 1556–1561.
107. Robinson, J. H., Callister, L. C., Berry, J. A., et al. (2008). Patient-centered care and adherence: Definitions and applications to improve outcomes. *Journal of the American Academy of Nurse Practitioners*, 20(12), 600–607.
108. Roger, G., Denoyelle, F., & Garabedian, E. N. (2001). Dysfonction laryngée épisodique. *Archives de Pédiatrie*, 8(Suppl 3), 650–654.
109. Rothman, R. L., Yin, H. S., Mulvaney, S., et al. (2009). Health literacy and quality: Focus on chronic illness care and patient safety. *Pediatrics*, 124 (Suppl 3), S315–S326.
110. Rudd, R. A., & Moorman, J. E. (2007). Asthma incidence: Data from the National Health Interview Survey, 1980–1996. *Journal of Asthma*, 44(1), 65–70.
111. Saboisky, J. P., Gorman, R. B., De Troyer, A., et al. (2007). Differential activation among five human inspiratory motoneuron pools during tidal breathing. *Journal of Applied Physiology*, 102(2), 772–780.
112. Saunders, S. W., Rath, D., & Hodges, P. W. (2004). Postural and respiratory activation of the trunk muscles changes with mode and speed of locomotion. *Gait Posture*, 20(3), 280–290.
113. Schilero, G. J., Spungen, A. M., Bauman, W. A., et al. (2009). Pulmonary function and spinal cord injury. *Respiratory Physiology & Neurobiology*, 166(3), 129–141.
114. Schmier, J. K., Manjunath, R., Halpern, M. T., et al. (2007). The impact of inadequately controlled asthma in urban children on quality of life and productivity. *Annals of Allergy, Asthma & Immunology*, 98(3), 245–251.
115. Shirley, D., Hodges, P. W., Eriksson, A. E., et al. (2003). Spinal stiffness changes throughout the respiratory cycle. *Journal of Applied Physiology*, 95(4), 1467–1475.
116. Sidhu, A. S., & Triadafilopoulos, G. (2008). Neuro-regulation of lower esophageal sphincter function as treatment for gastroesophageal reflux disease. *World Journal of Gastroenterology*, 14(7), 985–990.
117. Silvestri, M., Sabatini, F., Defilippi, A. C., et al. (2004). The wheezy infant: Immunological and molecular considerations. *Paediatric Respiratory Reviews*, 5(Suppl A), S81–S87.
118. Simoes, E. A. (2008). RSV disease in the pediatric population: Epidemiology, seasonal variability, and long-term outcomes. *Managed Care*, 17(11 Suppl 12), 3–6; discussion, 18–19.
119. Skjold, N. M., Farran, R. P., Hawes, H. G., et al. (2001). Simulation of acute spinal cord injury: Effects on respiration. *Respiratory Physiology*, 127(1), 3–11.
120. Smith, M. D., Russell, A., & Hodges, P. W. (2009). Do incontinence, breathing difficulties, and gastrointestinal symptoms increase the risk of future back pain? *Journal of Pain*, 10(8), 876–886.
121. Sopo, S. M., Radzik, D., & Calvani, M. (2009). Does treatment with proton pump inhibitors for gastroesophageal reflux disease (GERD) improve asthma symptoms in children with asthma and GERD? A systematic review. *Journal of Investigational Allergology & Clinical Immunology*, 19(1), 1–5.
122. Sullivan, P. E., Markos, P. D., & Minor, M. A. (1982). *An integrated approach to therapeutic exercise: Theory and clinical application*. Reston, VA: Reston Publishing Co.
123. Sunyer, J., Anto, J. M., & Burney, P. (1999). Generational increase of self-reported first attack of asthma in fifteen industrialized countries. European Community Respiratory Health Study (ECRHS). *European Respiratory Journal*, 14(4), 885–891.
124. Sveen, S. (2009). Symptom check: Is it GERD? *Journal of Continuing Education in Nursing*, 40(3), 103–104.

125. Temprado, J. J., Milliex, L., Grelot, L., et al. (2002). A dynamic pattern analysis of coordination between breathing and rhythmic arm movements in humans. *Neuroscience Letters*, 329(3), 314–318.
126. Teodorescu, M., Consens, F. B., Bria, W. F., et al. (2009). Predictors of habitual snoring and obstructive sleep apnea risk in patients with asthma. *Chest*, 135(5), 1125–1132.
127. Van Bever, H. P. (2009). Determinants in early life for asthma development. *Allergy, Asthma, and Clinical Immunology*, 5(1), 6.
128. van den Bemt, L., Kooijman, S., Linssen, V., et al. (2010). How does asthma influence the daily life of children? Results of focus group interviews. *Health Quality of Life Outcomes*, 8, 5.
129. van Veldhoven, N. H., Vermeer, A., Bogaard, J. M., et al. (2001). Children with asthma and physical exercise: Effects of an exercise programme. *Clinical Rehabilitation*, 15(4), 360–370.
130. Voynow, J. A., & Rubin, B. K. (2009). Mucins, mucus, and sputum. *Chest*, 135(2), 505–512.
131. Wagner, C. W. (2003). Pathophysiology and diagnosis of asthma. *Nursing Clinics of North America*, 38(4), 561–570.
132. Wasilewska, J., Kaczmarek, M., Protas, P. T., et al. (2009). [Sleep disorders in childhood and adolescence, with special reference to allergic diseases]. *Polski Merkurusz Lekarski*, 26(153), 188–193.
133. Wiener, G. J., Tsukashima, R., Kelly, C., et al. (2009). Oropharyngeal pH monitoring for the detection of liquid and aerosolized supraesophageal gastric reflux. *Journal of Voice*, 23(4), 498–504.
134. Yolton, K., Xu, Y., Khoury, J., et al. (2010). Associations between secondhand smoke exposure and sleep patterns in children. *Pediatrics*, 125(2), e261–e268.
135. Yuksel, H., Yilmaz, O., Kirmaz, C., et al. (2006). Frequency of gastroesophageal reflux disease in nonatopic children with asthma-like airway disease. *Respiratory Medicine*, 100(3), 393–398.