Functional Respiratory Disorders in Children

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KEYWORDS

• Functional respiratory disease • Habit cough • Vocal cord dysfunction
• Hyperventilation • Dysfunctional breathing

KEY POINTS

- Disorders without medical explanation are functional.
- Functional disorders can create as much disability as those with medical explanation.
- Functional disorders often suffer from iatrogenesis because of being treated with a disorder the physician knows rather than recognizing a disorder as functional.

INTRODUCTION

Functional respiratory disorders (FRDs) are those that are characterized by medically unexplained symptoms (MUSs).[1] That is, they constitute symptoms that have no anatomic or organic etiology. Clinicians caring for children encounter these disorders. Providing appropriate diagnosis and treatment tests the mettle of pediatric health care providers. Failure to diagnose the functional disorder often results in iatrogenesis from unnecessary testing and medication. MUSs in children occur in all systems—gastrointestinal, neurologic, musculoskeletal, and respiratory. This review addresses respiratory MUSs.

The most common MUS involving the respiratory system is the habit cough syndrome. Some less common related variations include habit throat clearing and habit sneezing. Other functional disorders present as dyspnea. These include the various vocal cord dysfunction (VCD) disorders, hyperventilation disorders, functional dyspnea in the absence of any abnormal physiology, and sighing syndrome. The challenge of correctly diagnosing a functional disorder can be complicated by the coexistence of organic disease, such as asthma, which also can cause cough and dyspnea.

Patients with an FRD present to a health care provider with persistent, frequent respiratory symptoms that can be mistaken for organic disease, such as asthma.

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Physicians are prone to diagnose and treat organic disorders they know, such as asthma or infection rather than consider a functional disorder. Failure to consider a functional disorder may result in inappropriate evaluation procedures and delay in effective treatment. Even if a functional disorder is suspected by a physician, reluctance to confirm a functional diagnosis may be the result of discomfort in treating the disorder. Writing a prescription is easier and less time-consuming than providing the behavioral strategies required to treat a functional disorder. Delay of diagnosis contributes, however, to emotional, physical, and social distress for the patient and family in addition to monetary costs from consultations, testing, and medication. The aim of this article is to review the clinical presentation, manifestation, and treatment of FRDs. How health care providers can successfully identify and treat these reversible conditions in the clinical setting is illustrated.

HABIT COUGH AND RELATED DISORDERS

What is a cough? It is readily recognized when heard. The sound of a cough can vary somewhat between individuals and in pitch, volume, and shrillness. What causes that sound? A cough starts with inspiration to fill the lungs, then the glottis closes, subglottic air is compressed, and the sudden opening of the glottis lets out the gust of compressed air. Two physiologic mechanisms of cough can occur. A reflex cough occurs when laryngeal receptors are stimulated by aspiration of foreign material; the inspiratory component then may be more limited. The more usual cough is tracheobronchial, initiated distal to the larynx and can be volitional.[2] The sound occurs from vibration of the large airways and laryngeal structures during turbulent flow of the rapidly expired air from release of the compressed air. Cough is a common symptom that results in a child being brought to a doctor. When cough continues daily for more than 4 weeks, it is considered chronic[3] (8 weeks is used in the adult literature to define a cough as chronic).[4]

Cough is a natural response to various stimuli. It is an important part of the defense mechanism for the lungs. Cough prevents pulmonary aspiration and clears airway debris. The cough reflex readily responds to physical or inflammatory stimuli. A foreign substance anywhere in the airway can stimulate cough. A viral respiratory infection causes inflammation of respiratory mucosa and secretion of mucus, both of which act as a stimulus to cough. Most causes of cough are self-limited. Even the chronic cough of *Bordetella pertussis*, whooping cough, generally runs its course within a hundred days.

There are many causes of chronic cough in children. An algorithmic approach to diagnosis begins with history.[5] That can identify a specific chronic cough, habit cough. The history of habit cough is unique in several ways. Although commonly beginning after an initial ordinary cause of cough, such as an asthma exacerbation or viral bronchitis (chest cold), the cough morphs into having a dry, barking, or honking character. Parents sometimes describe this transition by saying that the child had an ordinary cough for 2 weeks, “and then it changed.” The cough becomes repetitive up to several times per minute or repetitive every few minutes for many hours on end. Nonetheless, there is no cough once the child is asleep. The absence once asleep is a sine qua non for diagnosis of the habit cough syndrome.

The pathognomonic nature of this disorder can be seen in a sequence of publications over a 50-year period.[6–13] Each of these 8 articles describes the same type of clinical presentation, a repetitive barking or honking cough that is absent once asleep. With more than 200 children described with these characteristics in multiple clinical reports over many years, this constitutes a well-defined specific syndrome. The child

with a repetitive cough, often of a barking or honking character, that is absent once asleep constitutes this syndrome called the habit cough. Although the barking nature of the cough is the classic description, there are variations of the coughing sound. Habit throat clearing essentially is a variation of habit cough.

The name for this syndrome has varied over the years. Habit cough as a diagnostic term for this syndrome began with the first report in 1966 of 6 children seen by a Boston allergist Dr Bernard Berman.[6] Other terms used have included psychogenic cough and tic cough.[7,8] Habit cough was retained as the diagnostic terminology in a 1991 report of 9 children successfully treated by a specific form of suggestion therapy.[9] In 2016, a retrospective study at the University of Iowa from 1995 to 2014 identified 140 children, an average of 7 per year, diagnosed as having habit cough based on the syndromic criteria.[12] Using the same diagnostic criteria, 51 patients were diagnosed over a 6-year period at the Brompton Hospital in London, an average of 9 per year.

Ages of the 140 children with habit cough seen at the University of Iowa ranged from 4 years to 18 years, with a median age of 10 years (**Fig. 1**)[12]; 58% of those children were boys. Duration of cough before the initial visit was a median of 4 months and ranged from less than 1 month to periods of more than 1 year (**Fig. 2**)[12]. The median age of the 51 patients at the Brompton Hospital in London also was 10 years, with the same age range of 4 years to 18 years. The median prior cough duration was 3 months, with a range from 2 months to 3 years.[13]

Children eventually diagnosed with habit cough frequently were subjected to diagnostic procedures that included radiology and tests of lung function. Treatment prior to diagnosis frequently included inhaled albuterol, oral corticosteroids, montelukast, inhaled corticosteroids, various antibiotics, gastric acid suppressants, and cough suppressants, all without benefit. Frequent unscheduled medical care visits were common, and some had been hospitalized for the cough.

Effective treatment of habit cough was first described by Dr Bernard Berman.[6] He reported that cessation of cough was accomplished within a few days or weeks. He stated that "treatment relied on the skills of the physician in being able to convey to the patient the true nature of the disease, providing support and comfort during the period of treatment, and utilizing the art of suggestion."[6] Others subsequently referred to Berman's report and used reassurance and suggestion successfully in 9 children and 3 children, respectively, in 2 publications.[7,8]

Nine patients at the University of Iowa with habit cough were treated by a specific form of suggestion therapy.[9] Symptoms previously had been present for up to 2 years

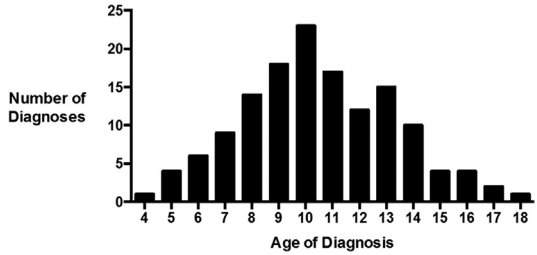

Fig. 1. Distribution of ages among 140 children diagnosed with habit cough from mid-1995 to mid-2014 at the University of Iowa Pediatric Allergy and Pulmonary Clinic. (*From* Weinberger M, Hoegger M. The cough without a cause: the habit cough syndrome. J Allergy Clin Immunol 2016;137:930; with permission.)

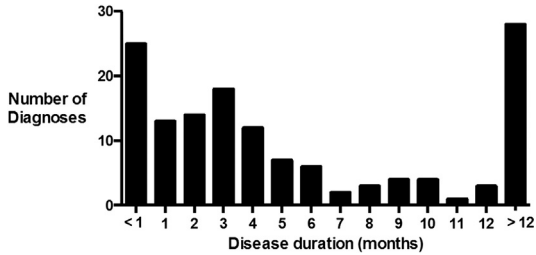

Fig. 2. Duration of repetitive cough prior to a diagnosis of habit cough among 140 children seen from mid-1995 to mid-2014 at the University of Iowa Pediatric Allergy and Pulmonary Clinic. (*From* Weinberger M, Hoegger M. The cough without a cause: the habit cough syndrome. J Allergy Clin Immunol 2016;137:930; with permission.)

(median 2 months). Five of the 9 had been hospitalized for the cough. Evaluation revealed no physiologic or radiologic abnormality. All patients became symptom-free during a single session of suggestion therapy that usually took approximately 15 minutes. During the subsequent week, 1 remained completely asymptomatic and 8 had transient minor relapses that were readily self-controlled by autosuggestion recommendations provided at the initial clinic visit. Seven of the 9 could be contacted for determination of long-term outcome at periods up to 9 years (median 2.2 years) after the session. Six were totally asymptomatic; 1 had occasional minor self-controlled symptoms. A standardized questionnaire assessing psychological symptoms at the time of follow-up revealed no somatization or emotional distress.[12] These data suggested the classic habit cough syndrome is amenable to immediate relief and long-term cure in most cases with a single session of appropriate suggestion therapy.

This conclusion was supported further by the results of 85 children seen during the period 1995 to 2014 who were actively coughing when seen at the University of Iowa[12]; 81 of the 85 had cessation of cough by the end of the clinic visit as a result of a 15-minute to 30-minute session of suggestion therapy performed by the various pediatric pulmonologists seeing patients in the clinic that day.

The principle of suggestion therapy was to ask the child to focus on the examiner and concentrate on being aware of a forthcoming cough. Holding back the cough, if even briefly, was encouraged. During continuous verbal patter, the patient was told that the cough began with some ordinary cough and then a vicious cycle occurred where the original cause of the airway irritation was gone, but now it was the cough causing the cough. The patient was told that each time the cough could be prevented from occurring that it would become easier the next time. An approximation of the script used for suggestion therapy is in **Box 1**. A video providing suggestion therapy to a 12-year-old girl with chronic cough for the previous 3 months is illustrated at www.habitcough.com/ and is reproduced on YouTube (https://www.youtube.com/watch?v=jnQUvD8Qdj0&t=670s).

In performing suggestion therapy, the parents generally were not informed of the diagnosis prior to the suggestion therapy session. Instead, the parents simply were told that the physician would show their child how to stop the coughing. Parents were asked to sit quietly with cell phones silenced and distractions minimized. When the repetitive coughing stops by the end of approximately 15 minutes, the parents were told of the diagnosis, which by then was self-evident.

The subsequent discussion with the patient and parents emphasized that it was not the physician who stopped the cough; it was the child. The child also was told they

Box 1
Major elements of suggestion therapy as a text guide

- Approach the patient with confidence that the coughing will be stopped.
- Explain the cough as a vicious cycle that started with an initial irritant that is now gone.
- Tell the patient that it is the cough itself that is causing irritation and more cough.
- Instruct the patient to concentrate solely on holding back the urge to cough.
- Select an initially brief timed period (eg, 1 min).
- Progressively increase this time period and utilize an alternative behavior, such as sipping lukewarm water to "ease the irritation."
- Tell the patient that each second the cough is delayed makes it easier to suppress further coughing.
- Repeat expressions of confidence that the patient is developing the ability to resist the urge to cough.
- "It's becoming easier to hold back the cough, isn't it" (nodding affirmatively generally results in a similar affirmation movement by the patient).
- When ability to suppress cough is observed (usually by approximately 10 min), ask in a rhetorical manner, "You're beginning to feel that you can resist the urge to cough, aren't you?" (said with an affirmative head nod).
- Discontinue the session when the patient can repeatedly respond positively to the question, "Do you feel that you can now resist the urge to cough on your own?" This question is asked only after the patient has gone 5 minutes without coughing.
- Express confidence that if the urge to cough recurs that the patient can do the same thing at home (autosuggestion[a]).

[a] Autosuggestion involves expressing confidence that 15-minute sessions at home concentrating on holding back the cough using sips of lukewarm water to "ease the irritation causing cough."

From Weinberger M, Lockshin B. When is cough functional, and how should it be treated?; *Reproduced* with permission of the © ERS 2020: Breathe 2017 13: 22-30; DOI: 10.1183/20734735.015216; with permission.

could stop the cough in the future by doing what they had done during the suggestion therapy session with the physician. That comment was important because the stimulus to cough, some children called it their "tickle," would persist for a day or more. The child, therefore, was empowered to control any return of cough. The authors called this autosuggestion, and it was reported by parents that their child utilized the autosuggestion recommendations.

Failure to provide some form of suggestion therapy or self-hypnosis is associated with continued coughing and the disability associated with that disorder, including missed school and decreased quality of life. The course of habit cough diagnosed but not treated beyond informing family of the diagnosis and provision of counseling was examined in a series of 60 children at the Mayo Clinic in Rochester Minnesota, mean age of 10 years. Those children were identified as having no physical cause for their cough; no specific treatment was given.[14] Mean duration of symptoms prior to being diagnosed was 7.6 months. Mean telephone follow-up was 7.9 years. Complete resolution of cough occurred in 44 after a mean duration of 6.1 months; 2 of those subsequently relapsed. Sixteen of the patients were still coughing a mean of 5.9 years after the Mayo Clinic diagnosis.

At the Brompton Hospital in London, treatment was limited to diagnosis and reassurance. Follow-up was possible in 39/55 (71%) children after a median duration of 1.9 years. In 32/39 (82%), the cough had resolved completely, 59% within 4 weeks, including 12% on the day of the clinic visit. Improvement occurred eventually in another 6/39 (15%). In the 29 children of parents who said they believed the diagnosis, there was eventual resolution of the cough in 96%. In the 13 children of parents skeptical or disbelieving of the diagnosis, however, only 54% experienced resolution of the chronic cough during the follow-up period.[13] These experiences at Mayo clinic and the Brompton provide the natural history of habit cough in the absence of specific suggestion therapy.

Habit throat clearing essentially is a variation of the habit cough syndrome. Of 140 children diagnosed with the habit cough syndrome over a 20-year period at the University of Iowa, a repetitive softer throat-clearing sound rather than the more typical barking cough was the presenting symptom in 10% of the patients; 11% exhibited both the barking cough and the softer throat-clearing patterns of coughing.[12] Response to suggestion therapy was as effective in these patients as in those with the harsh barking cough.

Habit sneezing is a much less common disorder than the habit cough syndrome, but there have been several case reports.[15] In the oldest reported case, a 40-year-old lady had repetitive sneezing that eventually was stopped by suggestion therapy.[16] One of the authors of this review (MW) treated an 8-year-old girl who was sneezing several times per minute for a week during waking hours. She had no symptoms when sleeping. The history indicated that the initial trigger was a nasal foreign body, which caused irritation with persistent sneezing that continued after expulsion of the foreign body. Suggestion therapy as used for habit cough stopped the sneezing.

In summary, habit cough is a specific form of chronic cough readily diagnosed by the description of a repetitive persistent cough, often with a barking or honking quality that is totally absent once asleep. Treatment with suggestion therapy has been repeatedly successful for the habit cough and its variations, habit throat clearing and habit sneezing. Diagnosis and treatment with suggestion therapy avoid the morbidity and dysfunction that results from this disorder.

FUNCTIONAL DYSPNEA DISORDERS

Although dyspnea can occur from many physical etiologies, including cardiac, pulmonary, chest wall abnormalities, and muscle disorders, the subjective feeling of dyspnea can be present in the absence of any of these organic or anatomic abnormalities. Functional dyspnea includes VCD and hyperventilation. Hyperventilation often is associated with panic attacks and anxiety. Exercise may be a trigger for both VCD[17] and hyperventilation,[18] both of which also can occur spontaneously or from other stimuli. Functional dyspnea is the feeling of shortness of breath in the absence of physical disease. The dyspnea can be from upper airway obstruction when vocal cords close paradoxically during inspiration and/or expiration. Dyspnea also can occur, however, without any physiologic abnormality of the lungs or physical obstruction to airflow.

Exercise-induced Dyspnea in the Absence of Respiratory, Cardiac, or Neuromuscular Disease

The most common cause of exercise-induced dyspnea in the absence of physical disorders is bronchospasm associated with asthma. Exercise-induced asthma readily is recognized by response to and prevention by an inhaled bronchodilator, such as

albuterol. Children with exercise-induced asthma generally have a history of symptoms of asthma beyond simple exercise-induced bronchospasm. When children and adolescents with exercise-induced dyspnea were evaluated systematically with cardiopulmonary exercise testing that reproduced the exercise-induced symptoms, various etiologies, both functional and organic, were identified (Fig. 3).[19] The functional disorders included VCDs, exercise-induced hyperventilation, and normal physiologic limitation perceived as pathologic dyspnea. Normal physiological dyspnea occurs after lactic acid is formed during anaerobic metabolism. The metabolic acidosis stimulates respiration at a time when the individual already has reached maximal attainable ventilation. The patient then perceives that they are not getting enough air. Although this is true, it is a function of having reached maximal ventilation.

Vocal Cord Dysfunction

Normally, the vocal cords actively abduct (come apart) to maximize air flow into the lungs during breathing. If the vocal cords adduct (come together), this creates obstruction to air flow. VCD is the abnormal closing (adduction) of vocal cords during inspiration and/or expiration. Patients describe air hunger, dyspnea, and chest or throat tightness. Clinical symptoms include stridor and/or an expiratory stertorious sound, depending on whether the vocal cords are closing on inspiration, or both inspiration and expiration. Videos of those 2 dysfunctional vocal cord movements can be seen at https://www.milesweinberger.com/copy-of-exercise-induced-dyspnea Spirometry corresponding to those videos demonstrates an abnormal inspiratory component of the flow-volume loop for adduction limited to inspiration (Fig. 4A) and marked decrease in both components for adduction persisting on both inspiration and expiration (see Fig. 4B). The latter is more clinically distressing.

There are 2 distinct phenotypes of VCD. Exercise-induced VCD (EIVCD) is the most common. Spontaneous VCD (SVCD) is more troublesome because it occurs without a specific predictable stimulus like exercise.[17,19] Although exercise, usually vigorous, is

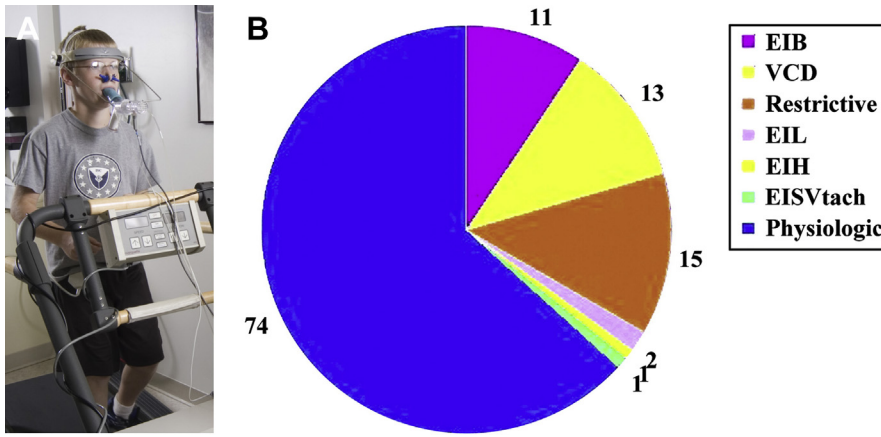

Fig. 3. Treadmill exercise with adolescent equipped for cardiopulmonary measures (*A*). Diagnoses determined by testing with cardiopulmonary physiologic monitoring (*B*). EIB, exercise-induced bronchospasm; EIH, exercise-induced hyperventilation; EISVTach, exercise-induced supraventricular tachycardia; Physiologic, physiologic limitation without other abnormality; Restrictive, apparent restriction of chest wall movement. (*From* Weinberger M, Abu-Hasan M. Pseudo-asthma: when cough, wheezing, and dyspnea are not asthma. Pediatrics 2007;120(4):862; with permission.)

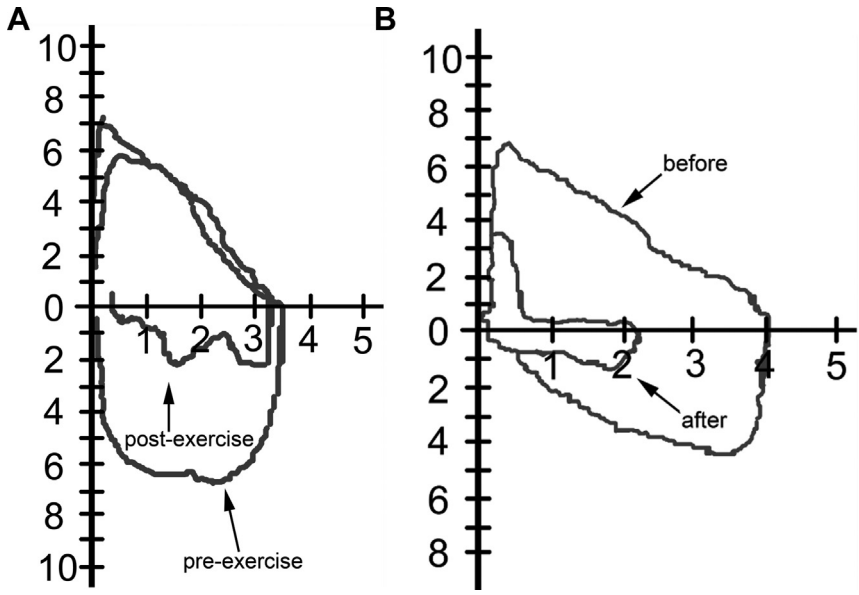

Fig. 4. Flow-volume recordings of 2 patterns of VCD: (*A*) paradoxical movement where exercise-induced adduction rather than abduction occurs on inspiration with relaxation of the cords on expiration; (*B*) spontaneous adduction on both inspiration and expiration. (*From* Weinberger M. Doshi D. Vocal Cord Dysfunction: A Functional Cause of Respiratory Distress. Breathe 2017;13:15-21; with permission.)

the precipitant for EIVCD, the precipitating factors for SVCD are variable and often unpredictable. The primary symptom of VCD is dyspnea. Diagnosis of EIVCD requires reproducing symptoms during exercise.[20] Although a treadmill is most appropriate for reproducing exercise-induced dyspnea for runners or with sports, such as basketball or soccer, an alternative device has been reported for monitoring symptoms in swimmers while they are swimming.[21]

Treatment of VCD classically has been reported to involve instruction from a speech therapist to take voluntary control of the vocal cords.[22] Patients learn to stop the inappropriate adduction of the vocal cords from continuing with this technique. Performing speech therapy techniques during vigorous and competitive exercise, however, can be difficult. Based on the vagal innervation of the vocal cords,[23–25] a rationale has been made for an inhaled anticholinergic aerosol for prevention of EIVCD by blocking the afferent pharyngeal vagal receptors. An ipratropium metered dose inhaler (Atrovent), used prior to planned exercise, has been observed to prevent EIVCD. A placebo-controlled clinical trial is needed to confirm those clinical observations.[17,26] The question has been raised as to whether a long-acting anticholinergic aerosol, such as tiotropium, would be effective as daily maintenance to prevent SVCD. Published experience regarding this approach for controlling SVCD has not yet been described, but there is a rationale for blocking the vagal effect.

The natural history of VCD was described among 28 patients identified with VCD. When contacted a median of 5 months (range of 1 week to 5 years) after the initial evaluation and diagnosis, 15 of 17 were asymptomatic; only 2 with EIVCD reported still using the ipratropium metered dose inhaler prior to exercise. All 11 SVCD patients no longer had VCD symptoms.[18] Based on those data, there appears to be a high rate of spontaneous resolution of VCD.

An imitator of EIVCD is exercise-induced laryngomalacia (EIL).[27] In some cases, this may be a residual of congenital laryngomalacia.[28] Inspiratory stridor does not occur from laryngomalacia until vigorous exercise results in sufficient air movement to invaginate the arytenoids or the epiglottis, depending on the specific anatomy.[29] When exercise testing identifies upper airway obstruction, as illustrated in **Fig. 4**A, visualization is necessary to distinguish EIVCD from the much less common EIL. The treatment of EIL, if needed, is supraglottoplasty.[30,31]

In summary, VCD can be limited to occurring only with exercise, EIVCD, or can occur spontaneously, SVCD. Although these often are distinct phenotypes, some patients manifest both SVCD and EIVCD. Speech therapy is the recommended treatment of SVCD, which can provide the patient with the ability to stop VCD when it occurs. Pre-exercise treatment with an anticholinergic appears to be an effective prevention for EIVCD. A high rate of natural resolution occurs for VCD.

Hyperventilation Syndrome

Acute hyperventilation syndrome (HVS) consists of inappropriate rapid and deep breathing triggered by anxiety or stressful events. It sometimes is part of a panic attack, characterized by intense fear or discomfort, palpitations, and a racing heartbeat.[32] Panic attacks can last minutes to hours.

Patients with HVS often present with nonrespiratory symptoms. The physiologic result of hyperventilation is an increase in alveolar ventilation in excess of metabolic needs. The arterial P_{CO_2} decreases and the arterial pH increases, resulting in respiratory alkalosis.[33] The increase in pH causes protein binding of free calcium that decreases free calcium levels. That may result in symptoms of lightheadedness and numbness or tingling in the fingers, face, or feet. Muscle twitching, carpopedal spasm, and even tetany can occur. Palpitations and anxiety frequently occur. Confusion and progression to stupor or coma can occur if hyperventilation and increased pH progress. Although the subject may feel they are not getting sufficient air into the lungs, it is the symptoms resulting from the metabolic effects of hyperventilation that often are most prominent.

In a review of 44 children seen at Duke University Medical Center with diagnoses of HVS, the chief complaint was related to respiration in only 18 (43%). Although the increased depth and rate of ventilation may be apparent to the examiner, the symptoms often are not dyspnea and air hunger. Dizziness, blackout spells, fainting, shakiness, palpitations, weakness, and numbness of the hands were prominent symptoms. Spells or attacks were described frequently.[34] Ages ranged from 5 years to 16 years, with those aged 12 years to 13 years demonstrating the highest prevalence (**Fig. 5**). Similar symptoms were described in a review of experience at the Children's Hospital of Alabama where 74 children, ages 11 years to 17 years, received a diagnosis of HVS.[35] The mean age there was 13.6 years; 74% were between ages 12 years and 16 years. A history of those nonrespiratory symptoms is sufficient to suspect that the episode described involved hyperventilation.

Diagnosis of hyperventilation requires distinguishing the symptoms from other causes. Observing the deep rapid breathing can make both patient and observer suspect respiratory distress as from asthma.[36] Patients with a history of asthma may have difficulty distinguishing the dyspnea associated with asthma from the symptoms associated with hyperventilation. A study of 120 adolescents with asthma found that HVS was 10 times more common in subjects with asthma (25%) than in those without asthma (2.5%), with a higher predilection in female adolescents.[37]

Because the acute episode may not be present when a physician is evaluating the patient, diagnosing the cause of the episodes depends primarily on obtaining a history

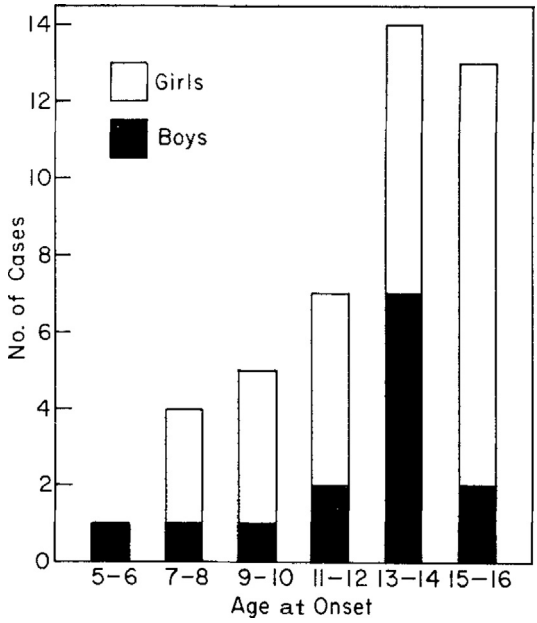

Fig. 5. Gender and age of 44 children and adolescents seen at Mayo Clinic with hyperventilation syndrome. (*From* Enzer NB, Walker PA. Hyperventilation syndrome in childhood: a review of 44 cases. J Pediatr 1967;70:523; with permission.)

of the symptoms. Confirmation of a suspicion obtained from the history can be obtained by having the patient voluntarily hyperventilate. Reproduction of the symptoms described by the patient during previous episodes confirms the suspicion determined from the history.[34]

Children with severe asthma seen during residential treatment have been observed using voluntary hyperventilation to initiate wheezing with active asthma (personal observation of MW at the National Jewish Hospital in Denver, Colorado). HVS in children often is misdiagnosed as an acute asthma exacerbation resulting in inappropriate and ineffective interventions.

During an acute episode, children and adolescents often appear anxious or distressed with deep breathing that can use accessory muscles of respiration. Lower airway sounds usually are clear with absence of the polyphonic wheezes that characterize asthma. Some patients may have diaphoresis and tachycardia but, overall, the physical examination is normal in the absence of concomitant cardiopulmonary disease.

Treatment of an acute episode includes rebreathing into a bag combined with reassurance. A caution against rebreathing into a bag has been published by UpToDate,[38] based on a study of 14 adult volunteers who deliberately hyperventilated.[39] Nonetheless, in a report of 44 children and adolescents from Duke University, a rebreathing bag was reported as employed with universal success in all with symptoms at the time of examination.[34] The use of a rebreathing bag gradually raises the P_{CO_2}, which lowers the pH, thereby eliminating the physiologic cause of most of the physical symptoms. Any makeshift rebreathing bag, paper or plastic, works. Once the symptoms from the respiratory alkalosis are relieved, the rebreathing bag should be discontinued, and verbal reassurance then is more likely to be effective.[34]

For a child with asthma who has difficulty distinguishing hyperventilation from respiratory distress of asthma, a device, such as a peak flow meter or a hand-held portable spirometer, can be provided for the patient to use when dyspneic. If a child with asthma presents for urgent care with respiratory distress, a normal oxygen saturation by a pulse oximeter warrants consideration for a blood gas. If the P_{CO_2} is low and the pH high, hyperventilation is diagnosed, although coexistent asthma is not excluded.

The long-term outcome of children and adolescents who experience recurrent episodes of hyperventilation has been examined at the Mayo Clinic (Rochester, Minnesota).[40] Those 18 years old and younger seen at the Mayo Clinic between 1970 and 1975 diagnosed with HVS were sent a questionnaire. Thirty responses (88%) were received, providing follow-up of 2 years to 28 years. Symptoms at time of follow-up found a substantial frequency of anxiety, hyperventilation, and depression (**Table 1**).[38]

In summary, hyperventilation attacks cause a variety of symptoms other than respiratory for many children. The peak age is early adolescence, but symptoms of hyperventilation can occur throughout childhood. The most severe symptoms are those with panic attacks. Relief of acute symptoms can be obtained by use of a rebreathing bag,[34] which results in elimination of the respiratory alkaloses by increasing the P_{CO_2}, and then providing reassurance. Children and adolescents with recurring hyperventilation attacks appear to continue being troubled by anxiety and hyperventilation attacks at follow-up.

Functional Dyspnea without Hyperventilation

Functional dyspnea without hyperventilation is characterized by subjective air hunger without abnormal minute ventilation or hypoxemia. There appears to be overlap between functional dyspnea without hyperventilation and psychogenic or somatoform respiratory disorders.[41] Patients typically complain of feeling short of breath. Anxiety usually is present as the cause of the subject's dyspnea. Physical examination is

Table 1
Frequency of symptoms at the time of follow-up among the 30 children and adolescents 6–18 y of age seen at the Mayo Clinic from 1950 to 1975 with hyperventilation syndrome

Anxiety	53%
Hyperventilation	40%
Depression	35%
Cold sweats	33%
Headaches	33%
Nail biting	33%
Gastrointestinal disorders	27%
Light-headedness	27%
Dry mouth	23%
Chest pain	20%
Fear of crowds	17%
Fear of dead bodies	17%
Palpitations	13%
Paresthesias	13%

Data from Herman SP, Stickleter GB, Lucas AR. Hyperventilation syndrome in children and adolescents: long-term follow-up. Pediatrics 1981;67:183-187.

grossly normal and devoid of adventitious breath sounds. If tachypnea is present, tidal volume is likely to be low so that minute ventilatory rate is normal. Confirmation of a clinical suspicion includes demonstrating normal pulse oximetry, blood gas pH, and P_{CO_2}.

Treatment consists of reassurance and providing evidence of normal cardiopulmonary testing and vitals during subjective air hunger. Pharmacotherapy is not indicated unless prescribed because of continuing anxiety not relieved by counseling.

Sigh Syndrome, also known as Habit Sighing

Sighing is a normal organic act described as a deep, audible prolonged breath. Although the physiologic function of sighing is not understood completely, it appears to have a role in varying tidal volume and preventing alveolar collapse.[42] Although interactions between the respiratory center and central nervous system can produce voluntary sighing, the sigh syndrome is characterized by an unusual frequency of involuntary sighing for an extended period. Sigh syndrome typically presents as an involuntary intermittent deep inspiration followed by protracted expiration. Accessory muscles of respiration may be involved; however, there is no change in oxygen saturation or ongoing respiratory rate. Sighing may be the result of emotional distress, weariness, or just an acquired habit for unapparent reasons. When asked, patients may report that they felt a need for more air.

Sigh syndrome originally was described in 1929 by White and Hahn as a rare symptom due to "nervous excitability."[43] Various terms have been used to define this entity, including sigh syndrome, sighing dyspnea, and anxiety dyspnea. For purposes of this text, the authors prefer the term, sigh syndrome or habit sighing, because it is not always apparent that afflicted patients have accompanying dyspnea or anxiety.

Episodes often occur during quiet times, with a frequency of sighing several times a minute. This pattern is absent during sleep. A study by Wong and colleagues[44] demonstrated that there was little difference in the anxiety profiles of children with sigh syndrome compared with those without sigh syndrome. There are no specific physiologic tests that distinguish sigh syndrome. Unless there is concomitant organic illness, serum electrolytes and blood gas are normal. Chest radiograph findings are negative and nonspecific in sigh syndrome. Pulmonary function findings in sigh syndrome are normal.[45]

Sigh syndrome appears to be a benign and self-limited disorder that disturbs primarily the parents observing the child but not the child with the repeated sighing. Neither school nor usual activities are affected by the sighing, which appears more common during periods of quiet activity, such as watching TV. A study of 40 patients with sigh syndrome found that none reported clinical symptoms at an 18-month follow-up period.[46] Treatment consists largely of providing reassurance to the parents. It is important to validate patient symptoms and discuss that sigh syndrome is a real, albeit benign, diagnosis. With correct diagnosis and reassurance, the prognosis appears to be good for eventual spontaneous cessation.

Functional Component of Asthma

Asthma is the most common chronic disease of childhood, affecting millions of children annually in the United States alone.[47] Children with asthma can demonstrate a functional component that can complicate or confound treatment. A study of 206 children with persistent asthma found that 5% reported dysfunctional breathing despite normal pulmonary function.[48] A functional component of asthma was more common in girls compared with boys, and these patients also experienced poorer asthma control.

A functional component of asthma in a child should be considered when complaints of dyspnea or hyperventilation are present in the absence of physiologic abnormalities of asthma, demonstrable by measuring pulmonary function. Treatment should be aimed at educating the patient and family regarding asthma symptoms and management. Objective self-assessments with a peak flow meter or portable spirometer can provide the patient and caregivers with a means to distinguish anxiety-induced dyspnea from actual asthma.

SUMMARY OF FUNCTIONAL RESPIRATORY DISORDERS

FRDs can be as troublesome as physical disorders. A chronic cough may occur from multiple causes. The repetitive nature and absence once asleep distinguish the habit cough syndrome from organic disorders. The functional causes of dyspnea may occur in the presence or absence of physiologic causes. Dysfunctional breathing can confound and complicate diagnosis and treatment of asthma. Those caring for children with respiratory disease need to be as familiar with these functional disorders as they are with organic disease.

CLINICAL CARE POINTS

- Morbidity from functional disorders can match or exceed organic disorders.
- Functional disorders have clinical characteristics that enable identification - they are not diagnoses of exclusion.
- Recognize when symptoms encountered are different from organic diseases with which you are familiar.
- When unusual symptoms are encountered, do not just treat what you know; search the literature for what you don't know.
- Recognizing that a disorder is functional requires skillful application of the art of medicine.
- Make the correct diagnosis before beginning treatment.

DISCLOSURE

The authors have nothing to disclose.

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