the distal airways and consequently improved bronchodilation. Heliox aerosol delivery systems also suffer less particle-impaction drug loss within the delivery system, thereby increasing the bronchodilator dose available to the lungs. There may be some limitations to heliox-driven aerosol use; a hypoxic patient may require a higher FiO_2 than the usually 20% or 30% oxygen in the available heliox mixtures. By adding oxygen to the inhaled gas, which increases the FiO_2, the percentage of helium in the mixture decreases, reducing the distal deposition of the bronchodilator; this low helium-oxygen mixture may still benefit the hypoxic patient. Radionuclide studies concluded that heliox was significantly more effective than air in depositing 3.6 μm particles in the alveolar regions, and that this improvement was more pronounced in asthmatic subjects than in healthy subjects[5].

There is still great controversy regarding heliox use in acute asthma. Although Barach first described the use of helium-oxygen mixtures in asthma more than 70 years ago, the use of heliox remains sporadic and undefined. This may be due to the fact that there are no randomised double-blind placebo controlled clinical trials, since a characteristic voice change is induced by breathing helium.[1]

Heliox therapy has shown to quickly improve ventilation in intubated and non-intubated patients with acute severe asthma and respiratory acidosis.[6] In a randomised controlled trial published in 1999, heliox improved airway obstruction and dyspnoea in patients with acute severe asthma.[7] There have also been few case reports, such as this one, in which heliox seems to play an important part in the success of the treatment.[8]

Several reviews have failed to support the administration of heliox in moderate-to-severe acute asthma.[2,9] A recent Cochrane systematic review,[10] including 10 trials and 544 patients, did not support the administration of helium-oxygen mixtures to all patients with acute asthma, stating that heliox has no role in the initial treatment of these patients. However, the authors recognize that new evidence suggests certain beneficial effects in patients with more severe obstruction, although these data should be interpreted with caution.

G.I.N.A. guidelines regarding the management of acute asthma exacerbations also report that heliox use may be considered for patients who do not respond to standard therapy, reflecting the uncertainty regarding the use of heliox.

Despite the lack of concrete evidence, heliox should be considered in hospital protocols for acute asthma treatment (as in Bridgeport Hospital in Connecticut),[1] in a particular set of patients, since there is a small number who do not respond to conventional therapy but still have respiratory muscle reserve.

In conclusion, there is some evidence to support the use of heliox in parallel to conventional therapies in a variety of respiratory diseases (asthma, COPD, croup), although further work is necessary to define its precise role.

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Vocal Cord Dysfunction in a Patient with Schizophrenia

To the Editor:

Vocal cord dysfunction (VCD) is characterized by episodes of involuntary paradoxical movements caused by vocal cord adduction during inspiration, resulting in airflow obstruction. The clinical profile, which is characterized by dyspnoea and wheezing (principally in the cervical region), mimics other respiratory diseases, such as asthma.[1,2]

Patients with asthma can present VCD, which can also be seen, in isolation, in individuals without respiratory disease. Studies conducted in Brazil revealed that the prevalence of VCD in patients with severe asthma was between 15 and 20%.[3] However, VCD is commonly underdiagnosed.

The pathophysiology of VCD is unknown, but the most probably hypothesis is that its aetiology is psychogenic. Emotional factors can trigger attacks of VCD and impair its resolution, and studies have shown that patients with VCD have mild-to-severe psychological alterations, such as depression, somatisation, anxiety and personality disorders.[1,2,4]

Another hypothesis suggests that gastro-oesophageal reflux disease is one of the causes of VCD, since it provokes a reflex that closes the larynx in response to a chronic inflammatory process.[5,6] However, there is no scientific evidence confirming this hypothesis and no organic cause of VCD has yet been found.[7]

Vocal cord dysfunction is considered as one form of the ''irritable larynx syndrome'' that has been defined as a hyperkinetic dysfunction. Voice alterations have also been reported as manifestations of this syndrome.[8]

Two factors make VCD more intriguing and more difficult to diagnose. First, there is a confounding overlap between asthma attacks and VCD when they coexist. Second, VCD appears as attacks, and patients can be asymptomatic between attacks, especially those with VCD in isolation. Clinical suspicion is based on discrepancies among the clinical profile, the complementary test results, and the therapeutic response.

A characteristic, non-pathognomonic spirometric alteration occurs in VCD: the flow-volume curve shows inspiratory flow limitation, with a flattening of the curve. The definitive confirmation of the diagnosis of VCD can be obtained by means of laryngoscopy, which is currently considered the gold standard for the diagnosis. When performed during an attack, laryngoscopy shows a typical pattern: adduction of the anterior two thirds of the vocal cords, forming a cleft after inspiration (diamond-shaped image).[1,2,4]

Schizophrenia is a severe mental disease that is classically characterized by symptoms such as disordered thinking, delusions, hallucinations and flat affect, causing patients to lose touch with reality. This often causes chronic social dysfunction.[9,10]

The objective of the present study was to report a case of VCD diagnosed in a patient with schizophrenia.

A 32-year-old female patient sought medical attention, complaining of dry cough, dyspnoea and wheezing for 3 days. The family reported that, in the past 6 months, the patient had had four severe bronchospasm attacks, which prompted her to seek treatment in the emergency room. The attacks were not associated with respiratory infections, physical exercise, or inhalation of irritant agents or allergens. She was prescribed oral corticosteroids.

She had presented intermittent allergic asthma as a child, with complete remission of the disease in adolescence. At the age of 18, the patient had been diagnosed with schizophrenia, at which time she initiated psychiatric treatment. She was taking olanzapine on a daily basis. The patient's family informed that she had not suffered any form of abuse during childhood, had no toxic habits and had not gastro-oesophagic reflux symptoms. The recent respiratory attacks began after she moved from her parents' house to another city where her sister resided.

During the follow-up period, the patient continued to complain of cough, dyspnoea and wheezing. The patient communicated with family members using gestures and few words, and with the health care professional only through head movements. We prescribed nasal and inhaled corticosteroids. The patient presented wheezing upon inspiration during all periods of the day. The wheezing was exacerbated when the patient became agitated. Under these circumstances, oxygen saturation was always normal.

Despite the treatment, there was no remission of the wheezing. Upon pulmonary auscultation, wheezing was predominantly detected during inspiration and could be heard in the cervical region. Temperature curve, blood workup findings, C-reactive protein levels and oxygen saturation (determined using pulse oximetry) were all normal. Skin prick test was positive to *Dermatophagoides pteronyssinus*.

Further investigation included laryngoscopy, which revealed paroxysmal adduction of vocal cords and larynx spasms occurring during silent inspirations. The patient was not submitted to spirometry, since she was uncooperative and unable to perform the test.

After being diagnosed with VCD by videolaryngoscopy, the patient was again evaluated by the psychiatric and speech therapy teams. They were able to advance in treatment when the patient presented periods of psychological improvement. Her condition partially improved, and she remained in outpatient follow-up treatment. Asthma treatment was discontinued.

It is difficult to diagnose and manage VCD, since it sometimes simulates asthma, and since the two diseases can occur in conjunction. We underscore the fact that many patients with VCD are misdiagnosed as having severe, difficult-to-control asthma and therefore receive inappropriate treatment. Exacerbations of VCD can result in frequent visits to the emergency room and in the administration of high doses of corticosteroids, as well as in unnecessary intubations.[1,2,4] As shown in this case, patients with VCD often receive inappropriate treatment and are overmedicated. However, since most patients with VCD also suffer from asthma, the latter should not be ignored.

Unlike asthma patients, patients with VCD in isolation rarely present hypoxaemia or spirometric alterations indicative of bronchial obstruction. In the present case, oxygen saturation, peak expiratory flow and arterial blood gas analysis values were all within the normal range, even during exacerbations. The patient was unable to perform spirometry. Although the patient had a history of asthma in childhood, the clinical and laboratory data suggested that the current dyspnoea attacks were due to VCD, as showed by videolaryngoscopy. Asthma was unlikely to be active at this moment.

The symptoms of VCD tend to occur mainly during the day, unlike those of asthma attacks, which occur primarily in the evening and early morning. Stress, physical activity, infections of the upper respiratory tract, inhaled irritants and contact with health professionals are just some of the factors reported to trigger the attacks, but they are not specific and can also trigger asthma exacerbations. In the case reported here, the patient had diurnal attacks, which were associated with periods of agitation.

A diagnosis of VCD should be considered whenever clinical data, laboratory test results and therapeutic efficacy are discrepant.[1–4] A specific questionnaire for the clinical suspicion of VCD was recently developed in order to identify patients who are more likely to present the disease.[3]

There is a consensus in the literature that wheezing in the cervical region is characteristic of patients with VCD and is extremely important for the disease's diagnosis, as occurred in the present case. As reported, although our patient was unable to perform pulmonary function tests, the diagnosis of VCD was confirmed through laryngoscopy.

The treatment of VCD consists of psychotherapy and speech therapy. Respiratory exercises typically used for treating hyperfunctional voice disorders are employed in order to reduce laryngeal muscle tension. Since beta-2 agonists and inhaled corticosteroids tend to produce poor results, they should be used only in patients with concomitant asthma. A multidisciplinary team is needed in order to increase the chances of treatment success.[1,2,4]

Some authors have suggested that this condition is a type of conversion disturbance, which is not fictitious because the patient can neither consciously control the dysfunction nor voluntarily reproduce the paradoxal movements.[1,2,4] Despite the association with psychiatric disorders, there have been no previous reports of VCD in patients with schizophrenia. This disease is essentially characterized by fragmentation of thought processes and, perhaps, neuropsychological mechanisms could trigger DPV. There is no known cure for it, but treatment can provide some degree of symptom relief, allowing patients to live a satisfactory, productive life.[9,10]

We highlight the fact that VCD is under-diagnosed, and that many patients are overmedicated for asthma. Therefore, the hypothesis of this diagnosis should always be considered.

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Stevens-Johnson syndrome: a case report

To the Editor:

Stevens-Johnson syndrome (SJS) is a life-threatening vesiculobullous disease characterized by an acute eruption that involves the skin and mucous membranes. Various etiologic factors have been implicated as a cause of SJS, including infection, vaccination, drugs, systemic diseases, physical agents, and food. Drugs are the most commonly blamed.

The incidence of SJS is estimated to be between 1.1 and 7.1 cases per million person-years.[1] SJS is currently considered to be a part of bullous disease syndromes [SJS, SJS-toxic epidermal necrolysis (TEN) overlap syndrome, and TEN] in which keratinocyte cell death results in subepidermal separation. In SJS, skin detachment is limited to less than 10% of the body surface area (BSA). TEN requires skin detachment of more than 30% of the BSA. An overlap group of SJS/TEN has been defined with erosions between 10% and 3 0% of the BSA.[1–3] The pathogenesis of SJS has yet to be clarified.

We report a 10-year-old male patient who was admitted to our department with a widespread bullous, erythematous pruritic eruption, bilateral conjunctivitis, oedema of lips and eyelids, haemorrhagic crusts of the lips, superficial erosions of the hard palate, an ulceration on the penile meatus (Fig. 1), and high fever (38.2 °C). The symptoms first started 2 days before, approximately 7 h after intramuscular administration of the 2nd dose of cefazolin (2 × 500 mg, i.m) which had been prescribed with the diagnosis of acute pharyngitis. He did not take any other drug apart from the antibiotics. He had no history of adverse drug reactions and had not been treated with cefazolin before. Other personal and family history was unremarkable. The clinical diagnosis of drug induced SJS was made with epidermal detachment of 7–8% of total body surface area.[1–3]

Laboratory results on admission revealed WBC count, 2330/mm^3; lymphocytes, 25%; platelets, 160000/mm^3; erythrocyte sedimentation rate (ESR), 36/h; C-reactive protein, 5.08 mg/dl (0-1); aspartate aminotransferase (AST), 54 U/l (15-41); alanine aminotransferase (ALT), 16U/l (14-54). Blood urea nitrogen (BUN), 41 mg/dl; creatinin and