SPEECH THERAPY

Paradoxical vocal cord dysfunction: clinical experience and personal considerations

Disfunzione paradossa delle corde vocali: esperienza clinica e considerazioni personali

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SUMMARY

Paradoxical vocal cord dysfunction is a nosographic entity that remains to be fully elucidated as far as concerns criteria required for diagnosis and underlying aetiopathogenesis. The disorder manifests with repeated episodes of acute dyspnoea associated with a series of symptoms that may include hoarseness, globus, chest pain and "shortness of breath". A retrospective analysis of cases with acute dyspnoea referred to our Department between June 2004 and June 2005 revealed 3 patients with paradoxical vocal cord dysfunction. In 2 of these 3 cases, concomitant psychiatric morbidity was observed and the third also presented gastro-oesophageal reflux. In one patient, the episodes of dyspnoea were triggered by inspiration of irritating substances. Diagnosis of the condition requires a high level of suspicion, which is confirmed by a laryngoscopic investigation that demonstrates hyperadduction of the true vocal cords and a reduction of at least 50% in the breathing space. From a therapeutic point of view, patients with paradoxical vocal cord dysfunction require, in our opinion, a multidisciplinary approach; in fact, only a team comprising otorhinolaryngologists, phoniatricians, pulmonologists, neurologists, allergologists, psychotherapists and speech therapists is capable of defining the appropriate treatment according to the clinical and psychological characteristics of each individual patient. Our results with speech therapy, focused on respiratory and speech retraining, are reported.

KEY WORDS: Larynx • Dyspnoea • Paradoxical vocal cord dysfunction • Diagnosis • Speech therapy

RIASSUNTO

Paradoxical Vocal Cord Dysfunction (PVCD) rappresenta una entità nosografica non ancora ben definita sia per quanto riguarda i criteri diagnostici che i momenti etiopatogenetici sottostanti. Tale affezione si caratterizza per la presenza di ripetute crisi dispnoiche acute associate ad un corteo sintomatologico che può comprendere senso di vellichio, globo faringeo, dolore toracico e respiro corto. Una valutazione retrospettiva dei casi con dispnea acuta afferiti al nostro reparto nel periodo compreso tra il Giugno 2004 ed il Giugno 2005 ha permesso di identificare tre pazienti affetti da PVCD. In due dei tre casi coesisteva una comorbidità psichiatrica, in uno di questi era presente anche un reflusso gastro-esofageo. In un paziente le crisi dispnoiche erano scatenate da inalazione di irritanti. La diagnosi di tale affezione necessita di un elevato sospetto clinico e deve essere confermata con una valutazione laringoscopica che dimostri un'iperadduzione delle corde vocali vere con riduzione dello spazio respiratorio di almeno il 50%. Da un punto di vista terapeutico riteniamo che il soggetto affetto da PVCD necessiti di una gestione multidisciplinare; infatti, soltanto un team composto da otorinolaringoiatri, foniatri, pneumologi, neurologi, allergologi, psicoterapeuti e logopedisti è in grado di proporre un trattamento individualizzato, strettamente aderente alle caratteristiche cliniche e psicologiche di ogni singolo paziente. In questo lavoro riportiamo i nostri risultati ottenuti trattando i pazienti con terapia logopedica riabilitativa.

PAROLE CHIAVE: *Laringe • Dispnea • Disfunzione paradossa delle corde vocali • Diagnosi • Terapia logopedica*

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Introduction

The term paradoxical vocal cord dysfunction (PVCD) is applied to laryngeal dyskinesia when there is excessive adduction of the vocal cords during inspiration [1 2]. Diagnosis of PVCD needs adduction of the true vocal cords during inspiration, visible by means of fiberoptic laryngoscopy, which causes a decrease of at least 50% in the breathing space [3].

The first description of PVCD was made in the mid '70s and since then numerous studies have focused attention on this pathological condition, referring to it by various names: vocal cord dysfunction (VCD), irritant-VCD (I-VCD), psychogenic and variable vocal cord dysfunction, irritable larynx syndrome, transient laryngeal dyskinesia, fake asthma, Munchausen's stridor [4 5].

Despite the fact that PVCD has been known for at least 30 years, even now diagnosis is often made late or some-

times not at all. From an epidemiological point of view, the condition is rather rare and is more often found in young women between 30 and 50 years of age [6 7]. Moreover, the patients are very often quite overweight and to a certain degree suffer from anxiety disorders and depression [8-10]. There is no definite pathogenesis, even if certain factors – such as gastro-oesophageal reflux, hyper-reactive mucosa and psychiatric disorders – play some role in this condition.

Materials and methods

A retrospective analysis has been performed of patients with acute dyspnoea referred to our Department between June 2004 and June 2005, 3 of whom were found to be affected by PVCD. All patients were assessed by means of a flexible fibrolaryngoscope and a laryngostroboscope. Diagnosis was made using the criteria proposed by Niggemann [11].

Case 1

S.D.P., a 50-year-old female with a 5-year history of vocal fatigue and persistent cough that had started about one year before coming to our attention. The patient referred that sometimes acute dyspnoea, with shortness of breath, chest pain and abnormal sounds during inspiration, occurred when she had a bout of coughing. Since she had a history of allergic rhinitis and bronchial asthma, she had been treated with topical steroids. Believing the coughing to be due to laryngeal irritation, caused by the topical steroids, the pulmonologist attempted to interrupt the treatment (administering non-topical drugs instead) but there was no obvious improvement. A previous ENT examination carried out on account of dysphonia and coughing had led to a diagnosis of dysfunctional dysphonia with hypotonic cords. The patient was referred to us for a phoniatric examination and was submitted to transnasal flexible laryngoscopy. The psychopathological profile of the patient was compatible with an anxiety disorder. During phonation, the cords were seen, in fact, to be hypotonic, but during breathing and after a few coughs the vocal cords suddenly adducted in an abnormal manner – to a greater extent in the anterior part of the glottis plane – with dyspnoea, increased coughing and panic, therefore, the examination had to be suspended. The episode of dyspnoea, with moderate stridor during inspiration, stopped spontaneously and the patient was calmed down and reassured that the event was benign.

A spectroacoustic analysis of the voice and an investigation performed with a Multi Dimensional Voice Programme (MDVP) during a non-critical stage of the condition demonstrated a profile that was compatible with dysfunctional dysphonia (Fig. 1). The patient was then invited to undergo speech therapy and initially this improved the respiratory dynamics. Respiratory rhythm exercises such as triangular breathing, squared breathing and rhythm variations (slow inspiration/rapid expiration; rapid inspiration/slow expiration) proved to be of particular importance. These exercises relaxed the muscles involved in speech and prevented the bouts of coughing. Many other exercises also have to be performed in order to obtain the best position of the resonance muscles without causing muscle tension and, above all, relaxation exercises should be carried out to eliminate nervous tension due to psychological stress. Thereafter, pneumophonic coordination exercises were performed in order to correct coupling between airflow and resonance structures, using words, sentences and phonetically balanced phrases, and specific resonance exercises to correct the equilibrium of resonance muscles and the vocal projection defects. Once the cough had considerably improved, it was then possible to apply specific laryngeal gymnastics to resolve the hypokinetic dysfunctional dysphonia. At the end of the course of speech therapy (which lasted about 20 days and was repeated each month for 3 months), the patient reported that both the cough and the episodes of dyspnoea had greatly improved. Vocal fatigue had also improved.

Case 2

C.E., a 35-year-old professional female nurse. During the winter she had had bronchial pneumonia that had left her with a cough and dyspnoea. Respiratory function tests with methacholine revealed bronchial hyperreactivity. Following accidental inspiration of sodium hypochlorite, she immediately had an acute episode of dyspnoea that was treated with iv steroids. Thereafter, the episodes of acute dyspnoea became more frequent both at work and at home. The episodes were triggered by inspiration of various substances such as detergents, soaps, paint, perfumes, to the extent that her normal everyday life suffered. A generic diagnosis of recurrent glottis spasm was made. The patient was invited to undergo allergology evaluation and a prick test, which resulted positive for graminaceous grasses. When she inhaled the alcohol used for disinfecting her arm, after the prick test, she had a severe episode of dyspnoea

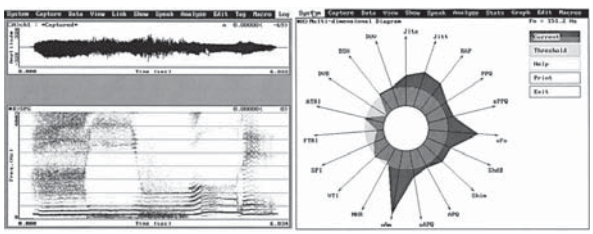

Fig. 1. Spectroacoustic analysis (left) and investigation performed with MDVP (right) of voice in Case 1 during non-critical stage. Tests compatible with dysfunctional dysphonia.

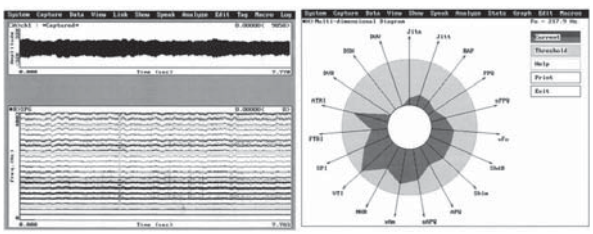

Fig. 2. Spectroacoustic analysis (left) and investigation performed with MDVP (right) of voice in Case 2 during non-critical stage. Normal tests.

with insistent cough and cyanosis. No pathological inspiration or expiration sounds were detected during the attack and auscultation of the chest was negative. The attack resolved spontaneously when the patient was invited to sit near an open window. A few days later, she was sent to the ENT department on account of recurrent episodes of acute dyspnoea. The patient did not complain of dysphonia; the spectroacoustic analysis of the voice and investigation with the MDVP were normal (Fig. 2). Throughout the examination, the patient appeared nervous but, nevertheless, underwent flexible fibre optic laryngoscopy. This endoscopic examination revealed no signs of any secondary laryngeal disorder but, during breathing, partial and sudden inspiratory adduction was observed on the vocal cords, together with a significant reduction in the glottis space associated with a severe attack of dyspnoea, cyanosis, increased coughing and panic, which required interruption of the examination. The attack of dyspnoea resolved spontaneously when the patient was reassured that the episode was benign and she was invited to breathe in slowly. The diagnosis made was irritant-vocal cord dysfunction (I-VCD); the patient was submitted to speech therapy for exercises similar to those used for reducing laryngeal muscle contraction in hyperkinetic forms of dysphonia (see Case 1). After 4 months, the patient showed considerable improvement, mainly through a psychological and speech therapy approach, focusing on breathing and speech retraining. In fact, the patient reported that while she still had attacks of dyspnoea, these were much less frequent and less severe. After 8 months, she was able to go back to her nursing career, not in the hospital wards, however (where she would have been in contact with potentially irritant substances), but in an administration office.

Case 3

A.D.R., a 47-year-old female secretary. For at least 10 years, the patient had been diagnosed as suffering from a form of bronchial asthma, for which she had always been given aerosol steroids and, more recently, also leukotriene receptor antagonists. She reported never having had any dysphonia but her anamnesis referred to fairly frequent episodes of acute dyspnoea that often required attention in the Emergency Department; on all these occasions, investigations were negative and were subsequently interpreted as being atypical panic attacks and, consequently, treated pharmacologically with very little improvement upon symptomatology. From a laryngological point of view, the laryngeal structures were always described as being normal, as far as concerns both motility and respiratory space. The patient was referred to us from the Emergency Department in a state of acute dyspnoea, with widespread cold sweating and unable to remain lying down. An examination with a flexible fibrolaryngoscope showed complete absence of any lesions in the respiratory space and the vocal cords were in perfect condition. A "closed larynx" was also observed, due to hyper-contraction of the true and false vocal cords. The patient was calmed down and asked to breathe into a plastic bag for a few minutes; thereafter, breathing again became eupnoeic and she was soon discharged without any specific treatment. She was then invited to return for further assessment that revealed a hiatus hernia but excluded the presence of dysfunctional dysphonia. Therefore, the diagnosis made was PVCD, in all probability associated with gastro-oesophageal reflux disease (GERD), and the patient was recommended treatment with proton pump inhibitors together with speech therapy to reduce laryngeal muscle contraction, as is usually advised in hyperkinetic forms of dysphonia. After approximately one year, the patient shows great improvement regarding the symptoms and the dyspnoea attacks have become much less frequent and decidedly less severe, to the extent that she has not had to return to the Emergency Department for this problem.

Discussion

The clinical manifestation of PVCD is the presence of recurrent symptoms/signs such as dyspnoea, wheezing, stridor and shortness of breath; the attacks usually appear and disappear suddenly [12] and the majority of episodes occur during daytime. The presence of a cough has been described in over 80% of the cases [3 6 7 13 14] and is generally associated with a series of symptoms that include hoarseness, globus – a sensation of pressure in the throat – chest pain, and abnormal breathing sounds (stridor, an inspiratory sound – or wheezing, an expiratory sound) [3 15]. Inspiratory stridor, although often present, is not observed in all patients [16] and, according to Newman et al., is seen in at least one fifth of the patients [6].

It is difficult to estimate the incidence of PVCD but it is fairly certain that the condition affects overweight women more frequently and with a bimodal incidence peak. Moreover, there appears to be a high incidence of PVCD in subjects who work in hospital/health environments [16] and, while it may be considered a condition that affects adults, there has recently been a significant increase in paediatric-adolescent age patients [17-19]. Another important point is that there is a certain incidence in athletes under training, which, in this case, leads to a differential diagnosis of effort asthma [18 20-22]. From a pathogenic point of view, the mechanisms underlying PVCD are still unknown, although psychiatric factors are almost certainly involved. Newman et al., in fact, demonstrated that in 73% of the PVCD cases, there was a major psychiatric condition; about one third of the patients showed a personality disorder and about 38% of them had a history of abuse [23]. Other Authors consider PVCD a conversion disorder [24]. One of the roles still to be clarified is that played by irritant substances: it has been seen that the phlogogenic action of certain molecules can trigger a PVCD attack by means of a reflex mechanism [25]. In these cases, the term "irritant associated PVCD" or I-VCD has been suggested [26] and these subjects develop the typical clinical profile after exposure to inhalant agents [27]. It has been postulated that these patients, who are continually exposed to the irritants, develop a kind of sensitisation of the mucosa, to the extent that PVCD attacks are triggered even when they inhale substances that are not initially irritant [28]. A recent study has suggested that gastro-oesophageal

reflux (GER) may have a role [21 29]; in fact, Powell et al. observed that almost all of the patients present laryngeal manifestations compatible with gastro-oesophageal reflux disease (GERD), but, as yet, no association between PVCD and GERD has been established [2 21]. A paradoxical movement of the vocal cord, may also be caused by central neurologic aetiologies, such as cortical or upper motor neuron injury, nuclear or lower motor neuron injury, brainstem compression, Arnold-Chiari malformations I and II, myelomeningocele and, likewise the results of cerebro-vascular accidents (strokes of the posterior circulation) [15 30 31]. A neurological examination, CT scan or MRI, can indicate or exclude such conditions.

Considering the series of symptoms in PVCD, it appears evident that this disorder will be taken into consideration in differential diagnosis along with numerous other clinical conditions (bronchial asthma, exercise-induced laryngomalacia, laryngospasm, reactive airway dysfunction syndrome, GERD) [32] and very often, because this pathological condition is not well known, a diagnosis of PVCD is made incorrectly or, often very late [33]. In this sense, diagnosis is delayed, on average, for 4-10 years [6 7 10 15]. Furthermore, it is worthwhile stresssing that the presence of PVCD does not exclude other concomitant respiratory diseases [34]; in fact, Newman et al. demonstrated bronchial asthma besides vocal cord dysfunction in ≥ 50% of the patients, [23]. Indeed, subjects with PVCD are often unable to hold their breath during an attack, unlike asthmatic patients, and the respiratory symptoms (dyspnoea, in particular) sometimes resolve with simple distraction methods [9 10]. Unfortunately, since these patients do not respond to steroids, they are often considered affected by "steroid-resistant asthma" [35], thus diagnosis is delayed even further. The National Heart, Lung and Blood Institute (NHLBI) Guidelines for the Diagnosis and Management of Asthma specifically recommend considering the diagnosis of PVCD in patients who fail to respond to asthma therapy [36]. Our experience appears to confirm data available in the literature since, in all our patients, the diagnosis was reached rather late. In one patient (case 3), the delay is estimated at as much as 10 years. PVCD must be strongly suspected to be diagnosed and especially when otherwise unexplainable inspiratory stridor is present, or in subjects with wheezing, particularly above the trachea. Close attention must be paid to patients whose symptoms suddenly become worse, and also to young women with a history of mute asthma presenting sudden attacks of dyspnoea not responding to anti-asthma treatment [37]. Dyspnoea, in these subjects, is often out of proportion with the objective clinical signs. Haemogas analysis provides little information and is of very limited diagnostic help, moreover, the literature shows values that are very contradictory [4 16 24]. Chest X-rays are usually within normal limits even though, in some cases, there may be signs of hyperinflation of the lungs [1]. In our opinion, this type of diagnostic tool is not very useful in the diagnosis of PVCD since it is not easily performed during an attack.

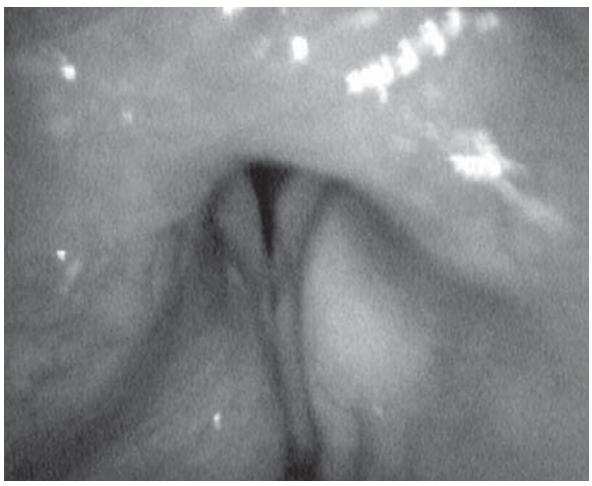

Fig. 3. Laryngostroboscopic evaluation, during an acute phase, shows adduction of two anterior thirds of vocal cords during inspiration phase, with creation of posterior glottic gap.

At present, the method of choice for diagnosing PVCD appears to be laryngoscopy [2 38]. In fact, laryngeal endoscopic investigation allows a direct view of the vocal cords and their relative function, both during inspiration and expiration. During the acute phase of an attack, the classical "diamond" aspect can be seen, that is, the adduction of the two anterior thirds of the vocal cords during inspiration, with the creation of a posterior glottic gap known as a "chink" (diamond posterior glottic shape) [6 7 13 24 39 40] (Fig. 3). In particular cases, known as biphasic PVCD, this aspect can sometimes be seen also during the expiration phase. In our opinion, and in agreement with other Authors, a diagnosis of PVCD can be considered reasonable when adduction of the true vocal cords occurs during inspiration, causing a reduction of ≥ 50% of the respiratory space [3]. Our experience confirms the usefulness of laryngoscopy with flexible fibre optics, since it allows viewing of hyperadduction of the cords during inspiration in all patients and, therefore, permits diagnosis. In some subjects, the adduction of the true vocal cords is present also during the expiration phase, albeit to a lesser degree [24]. In effect, it must be stressed that a certain degree of glottic obstruction is also present in asthma, but less than in PVCD attacks. In this case, cord adduction seems to represent a compensation mechanism avoiding, or at least reducing, collapse of the lower airways [1]. In our opinion, the biggest knot to be unravelled is its relationship with possible laryngeal lesions, which cannot exclude *ab initio* a diagnosis of PVCD. In fact, a recent report demonstrated a high percentage of laryngeal abnormalities in subjects with PVCD (acute laryngitis, chronic laryngitis, signs of GERD, laryngomalacia, vocal fold motion impairment, *sulcus vocalis*, vocal nodules, subglottic stenosis) [15]. In this regard, an objective assessment is fundamental during the acute stage because the presence of laryngeal lesions may be considered of little importance compared with the other symptoms of the patient. As already mentioned, neither haemogas analysis, nor chest X-rays appear to be useful, from a diagnostic point of view, and, the role of spirometric tests still remains to be defined: while, on the one hand, the method is capable of revealing the typical flattening (or even interruption) of the inspiration stage, it is also true that this is detected only if the patient is symp-

Table I. Diagnostic criteria for PVCD (Niggemann, 2002) [11].

Dyspnoea is predominantly inspiratory in absence of upper respiratory tract infections
No (or marginal) response to bronchodilatory treatment
Variable flattening of inspiratory limb of flow-volume curve suggesting extrathoracic airway narrowing
Respiratory-cycle dependent assessment of vocal cords during laryngoscopy under local anaesthesia
No deterioration after discontinuation of anti-asthmatic treatment

tomatic. It is sometimes possible to see limitations in the expiratory stage as well, and, on rare occasions, an increase in pulmonary volume (as in asthma) [1]. Some Authors maintain that the methacholine test can trigger an attack, at least in some patients [35]. In effect, and as already pointed out, it must be stressed that the presence of PVCD does not exclude the presence of other respiratory diseases (bronchial asthma, first of all) [23 34]. In this respect, it is believed that a differential diagnosis between PVCD and asthma cannot be based exclusively upon a bronchial provocation test, even though some Authors have observed that, in the majority of PVCD patients, the pulmonary volume is altered after challenging with methacholine [2 23]. Admittedly, this phenomenon is linked with PVCD and asthma both being present, in a fairly significant percentage of patients, or it reflects a laryngeal response to irritant agents [2]. This means that the presence of inspiratory modifications in the spirometric curve (flattening/interruption) after the methacholine test, while not supplying a diagnosis, should, at least, warn the physician concerning the possible presence of PVCD. On the grounds of what has been described so far, it is easy to understand why PVCD is often wrongly diagnosed since differential diagnosis with an attack of asthma (and effort asthma) is, in itself, difficult [22]. To facilitate this distinction, several diagnostic criteria for PVCD have been proposed (Table I) [11]. While our experience is somewhat limited, it appears to confirm that these criteria are useful. We believe that if laryngeal function is normal when severe inspiratory dyspnoea is present, and the symptoms rapidly resolve without the administration of steroids, then PVCD should be strongly suspected. At present, there is no generally accepted treatment for the condition. Over the years, speech therapy, pharmacological treatment and psychotherapy have been used without obtaining indisputable results [41]. When managing an acute attack of dyspnoea, it is very important to reassure the patient and explain that his/her condition is "benign". It is also advisable to distract the patient during respiration [11 37]; in fact, these calming procedures were very efficacious in all our patients. In one case, we had to perform re-breathing manoeuvres to control the acute anxiety brought on by the dyspnoea attack. In particular cases, different types of anaesthetics (Propofol, for example) have been used to interrupt dyspnoea attacks [1]. We personally believe that this therapeutic approach is somewhat aggressive and probably out of proportion with the real needs of the patient. In effect, the management of an attack is relatively simple due to the fact that the attack is actually self-limiting. Management of the conditions that trigger or contribute to an attack is definitely more complex. When psychosomatic or clearly psychiatric disorders are present, treatment should be directed at the basic disorder. Besides administering psychopharmacological drugs (when necessary), treatment should also include psychotherapy, respiratory rehabilitation and speech therapy, plus anti-allergy and anti-reflux drugs in the case of allergies or gastro-oesophageal reflux [3 43-47]. Botulinic treatment has also been demonstrated to be efficacious for, at least temporarily, reducing the frequency of the attacks [10 42]. Our experience certainly confirms an association with psychiatric disorders present in two of our three patients, both of whom were, nevertheless, submitted to psychiatric assessment. The exact pathogenic significance of this association has yet to be explained. The methods and efficacy of respiratory and speech retraining therapy have been described elsewhere [3 10 13 44]. Reassurance and specific exercises focusing the attention of the patient on rhythmic passive inspiration and active expiration, upper body relaxation, easy voice onset and resonant exercise are used [3 13 43 44]. Thus, many authors recommend vocal exercises similar to those used to reduce the contraction of laryngeal muscles in hyperkinetic forms of dysphonia [1 10]. In fact, the speech therapy advised is centred on relaxation methods that emphasize techniques for reducing cordal hypertone and strategies for decreasing laryngopharyngeal and cervix muscle tension. The aim of the training is also to achieve automatic application of the relaxation techniques at the first signs of an attack of dyspnoea in order to block its progress and speed up the process. Significant clinical improvement has been observed in 88% of the patients when the main approach has been through psychological and speech therapy focused on respiratory and speech retraining [13].

In our opinion, and in agreement with other Authors, a PVCD patient should, nevertheless, undergo an initial multidisciplinary assessment. In fact, only a team of otorhinolaryngologists, phoniatricians, pulmonologists, neurologists, allergologists, psychotherapists and speech therapists can suggest the most suitable treatment for the individual patient according to his/her clinical and psychological manifestations [3].

Conclusions

PVCD is a nosographic entity that has yet to be defined as far as concerns the criteria used in diagnosis and its underlying aetiopathogenesis. Correct clinical-instrumental assessment together with a strong suspicion of the condition is fundamental in order to reach a diagnosis. Treatment should be focused on the pathogenic aspects detected during the various examinations and, therefore, should be tailor-made to suit the specific requirements of each patient.

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