Psychogenic stridor: diagnosis and management

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Abstract
Psychogenic stridor is a rare cause of apparent acute upper airway obstruction, usually occurring in young female adults. The alarming presence of inspiratory stridor leads to suspicion of organic disease, with urgent management directed towards treatment of suspected pathology. Careful history taking may suggest the correct diagnosis and early examination of the larynx, preferably by fibreoptic nasendoscopic examination, may identify the underlying abnormality of paradoxical adduction of the vocal cords on inspiration, accounting for the stridor. This characteristic feature allows a positive diagnosis to be made and may avoid potentially harmful and inappropriate interventions. Three cases are presented, illustrating an evolution of management. (J Accid Emerg Med 1997;14:330-332)

Keywords: psychogenic stridor; fibreoptic nasendoscopy

Although functional upper airway obstruction and stridor is rare, its presentation often suggests life threatening upper airway disease, and management is often directed along urgent lines to protect the airway or even establish alternative means of breathing through a tracheostomy. This is partly because the diagnosis, even recently, has been considered one of exclusion.¹ It is important to appreciate, however, that there are characteristic signs of psychogenic stridor, and recognition of these by direct laryngeal examination may spare the patient inappropriate treatment.

Case reports
CASE 1
A 12 year old girl was admitted through the accident and emergency (A&E) department with a short history of stridor which had woken her from sleep. On examination she had predominant inspiratory stridor and appeared distressed. There was a mild tachycardia but she was normotensive and apyrexial. Interestingly her voice was virtually normal. A brief period of pulse oximeter monitoring showed no desaturation. In view of the stridor it was decided to perform an examination under anaesthetic to exclude or confirm suspected epiglottitis and proceed on the basis of the findings.

Induction and intubation proved uneventful and complete endoscopic examination of the upper airway showed no abnormality. Extubation was likewise uneventful. The patient was observed overnight. Interview the following

2 Safar P. Ventilatory efficacy of mouth-to-mouth artificial respiration. JAMA 1958;167:335-41.
morning suggested stresses at home but no specific management was instigated for this and the patient was discharged home without follow up.

CASE 2
A 12 year old girl was referred urgently through A&E with intermittent inspiratory stridor. This had been present for a period of approximately two weeks and had been unsuccessfully treated with inhaled salbutamol for a putative diagnosis of asthma. When seen, the patient was undistressed with no stridor. Careful history taking revealed her to be under considerable stress as a result of attempting to gain a secondary school place out of her local area. Full otolaryngological examination was normal as was peak expiratory flow and examination and radiology of her chest. Her voice was normal. A diagnosis of psychogenic stridor was made. Follow up arrangements were made with the speech therapy department but resolution of her schooling arrangements seemed to cure the problem.

CASE 3
A 13 year old girl was referred to the ENT department from the paediatric department, having been admitted the previous day with a diagnosis of worsening asthma of short duration, poorly responsive to her prescribed medication of inhaled salbutamol and beclometasone. When she was admitted it became apparent that her noisy breathing was the result of intermittent inspiratory stridor. Initial ENT assessment revealed no pathology. Further interviewing suggested that stridor only developed when the patient’s mother arrived on the ward. Following this initial assessment it was then possible to re-examine the patient during an attack of stridor induced by maternal arrival. On flexible nasendoscopy the characteristic findings of paradoxical vocal cord movement were observed, thus confirming the diagnosis. Again the voice remained normal during the attack of stridor.

This patient was managed through the speech therapy department. Although there was initial family resistance to the idea that “stress” could be the underlying factor this was eventually accepted. The situation was, however, complicated by continued prescribing of anti-asthma medication by the patient’s general practitioner, thus undermining the diagnosis.

Discussion
Psychogenic stridor is not a new condition. Osler described it in 1902 and it has subsequently been reported as Munchausen’s stridor, factitious asthma, vocal cord dysfunction presenting as asthma, hysterical stridor, and functional airway obstruction. The alarming noise made by the patient is usually frightening to them, their relatives, and often their medical attendants.

All our patients had solely inspiratory stridor yet two had been treated for a diagnosis of asthma before this was realised. This accords with the experience of O’Connell et al., who found that 12 of 20 patients were initially diagnosed as asthma, and three of the 20 were diagnosed as having exercise induced bronchospasm. This emphasises the potential for clinical error: the sound signal frequency of asthmatic wheeze and stridor is similar and distinction is based only on their timing in the respiratory cycle. Although not present in this series, expiratory vocal cord dysfunction—part of the spectrum of vocal cord dysfunction—is reported and makes clinical distinction from asthma considerably harder. Blood gas analysis may be useful in the acute phase of psychogenic stridor, and the presence of a normal oxygen and a normal or even low carbon dioxide partial pressure favours this diagnosis (a low PaCO2 occurs as a result of hyperventilation.) However, some cases of psychogenic stridor have presented with collapse and cyanosis, and entities such as exacerbation of acute asthma may present with these results. This investigation is therefore of limited use in establishing the diagnosis.

A feature common to all these patients was the presence of a normal voice during an attack. Such a finding is a valuable pointer to the diagnosis. Speech occurs in the expiratory phase of respiration and this tends to be normal in psychogenic stridor, although in Lacy’s review of the 48 patients appearing in the literature, 10 had a history, current or past, of dysphonia or aphonia. The crucial phase of the respiratory cycle here was the inspiratory phase. During normal inspiration the vocal cords become more widely abducted as total lung capacity is reached. In psychogenic stridor the characteristic feature is paradoxical adduction of the vocal cords on inspiration, which results in turbulent airflow and the noise of stridor. This is combined with the ability to abduct normally, which is seen in expiration. If normal abduction is not seen, then the alternative diagnosis of bilateral recurrent laryngeal nerve paralysis must be entertained. Examination of the larynx during the acute phase by either indirect laryngoscopy or, ideally, fiberoptic nasendoscopy is not difficult to perform and enables one to visualise the source of the noise and confidently make a diagnosis. This has been shown to be possible in 75% of patients undergoing mirror examination in this condition. Flexible nasendoscopy is likely to improve upon this rate.

Our first two cases remain diagnoses of exclusion, as all we know is that they had a normally appearing larynx when we examined them. It is very unlikely that the diagnosis can be positively made under anaesthetic, as the psychogenic drive for paradoxical movement has been abolished. We would also suggest that the use of heavy sedation before performing laryngeal examination may cause loss of the paradoxical movement and is best avoided if psychogenic stridor is suspected. With our third case we were able to visualise the larynx and make a positive diagnosis at the time. Doing this enables further intervention to be avoided and energies to be channelled along more fruitful lines.
Once the diagnosis is confirmed the simplest treatment is reassurance. Sedation with diazepam may also be of some value. Breathing a helium/oxygen mixture has been suggested and used, but this may probably function through a placebo effect. Although the patients we describe showed no desaturation, there are examples of published reports of airflow limitation sufficient to have led to intervention in the form of a tracheostomy. Certainly if such distress is present then laryngeal examination may be difficult, but we and others would suggest that endotracheal intubation is the preferred initial management to secure the airway. The condition occurs predominantly but not exclusively in young females under psychosocial stress. Previous investigators have considered this to be a conversion disorder and the laryngeal spasm may be an unconscious somatic expression of emotional conflict. Long term treatment is usually in the form of psychotherapy or speech therapy, although relapse is common in times of stress.

While it is imperative that the presence of organic pathology in actual upper airway obstruction is sought and treated if present, early detection of paradoxical vocal cord movement will allow an early diagnosis. In addition it will allow a positive diagnosis of the condition to be made, as opposed to a "diagnosis of exclusion" and will avoid unnecessary and potentially harmful interventions being undertaken.


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