VOCAL CORD DYSFUNCTION PRESENTING AS BRONCHIAL ASTHMA : THE ASSOCIATION WITH ABNORMAL THORACO-ABDOMINAL WALL MOTION

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ABSTRACT

A 23 year old man with glottic dysfunction presented as recurrent bronchial asthma. His symptoms were aggravated by application of a noseclip and associated with asynchronous thoraco-abdominal wall motion. The glottic dysfunction was corrected by sedation but not continuous positive airway pressure. This is the first report of vocal cord dysfunction triggered by application of a noseclip and associated with asynchronous thoraco-abdominal wall motion.

Keywords: Vocal cord dysfunction, breathing pattern, asthma.

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INTRODUCTION

Abnormal movement of the vocal cords is a rare cause of episodic noisy breathing associated with dyspnoea which may be mistaken for acute bronchial asthma[1-3]. The underlying abnormality may be glottic dysfunction related to emotional or psychiatric factors[4]. This condition should be distinguished from the intrathoracic airways obstruction associated with bronchial asthma because glottic dysfunction is a benign illness and the treatment consists of speech therapy and psychiatric counselling[3]. In this report a young man with glottic dysfunction who presented as acute recurrent bronchial asthma is described. His symptoms were precipitated by the application of a noseclip and associated with abnormal thoraco-abdominal wall motion.

CASE REPORT

A 23 year old male storekeeper was admitted to the hospital with diagnosis of "severe acute bronchial asthma" of two days duration. He gave a three year history of repeated admissions for episodes of wheezy dyspnoea associated with the sensation of tightness over the anterior neck and upper chest. There was no definite precipitating factor and he denied any nocturnal symptoms. He had no other personal or family history of allergic illnesses. During periods of remission, he played soccer for his company and was noted by his family to have a good singing voice.

On examination, he appeared moderately distressed and showed prominent use of the accessary muscles. Wheezing was heard over the entire lung fields but was maximal over the larynx and especially during inspiration. He experienced partial relief of symptoms after inhalation of aerosolized salbutamol.

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The chest radiograph, electrocardiogram and blood counts were normal. The arterial blood gas on breathing room air showed a normal alveolar-arterial gradient for oxygen tension. He did not respond to common allergens on skin prick testing.

The flow-volume curve recorded in the pulmonary function laboratory showed a small tidal volume and severe reduction of both maximal inspiratory and expiratory flow rates (Fig 1). He generated much better expiratory flows when he was asked to cough repeatedly form TLC, the transient peaks [•] and plateaus [dashed line] are superimposed on Fig 1. His airway resistance was markedly elevated at 2.6 kpa./(l./s.) [normal value 0.2-0.4 kpa./(l./s.)] and was not reduced after inhalation of salbutamol. The Jaeger Bodyscreen II was used to measure flow-volume curves and airway resistance.

The application of a noseclip in the laboratory during pulmonary function testing precipitated his symptoms of acute wheezy dyspnoea and tonic inspiratory contraction of his upper chest and accessary neck muscles with inspiratory and expiratory wheeze. The noseclip application in effect reproduced the symptom and signs of a spontaneous "asthma attack" in this patient. Thus, his ribcage (RC) and abdominal (ABD) wall motion was recorded by inductance plethysmography (Respitrace Inc.) during tidal breathing in the sitting posture *before* and *after* the application of a noseclip (Fig 2). Grossly abnormal breathing pattern was noted after application of the noseclip. There was a sharp increase in the end tidal position associated with fall in the tidal volume (VT : tidal volume equals the sum of ABD and RC signals; Fig 2). This was accompanied by asynchronous RC and ABD motion (Fig 2). The RC was held in a predominantly inspiratory position while the ABD compartment provided the major contribution to tidal volume displacement (over 80% of the sum signal). No measurements of flow-volume curve after rebreathing of helium-oxygen or single breath nitrogen slope was possible because he could not perform slow vital capacity manoeuvres with the noseclip in place. His symptoms were not relieved by the application of continuous positive airway pressure (CPAP) through a nose mask (Sleepeasy I, Respironics Inc.) of up to 10 cm H_2O[5,6].

Direct examination of the upper airways with fibreoptic bronchoscopy (Olympus BF1 T10) showed adduction of false and true vocal cords during both inspiration and expiration. There was almost complete narrowing of the glottic chink associated with stridor when he was asked to take a deep

inspiration. After the patient was sedated with an intravenous 10 mg dose of diazepam, the wheezyness ceased, and the vocal cords were observed to move normally with full abduction. No other tracheal or airway abnormality was visualised.

The contribution of the vocal cords to his illness was explained to the patient. He was discharged with lorazepam and speech therapy in the outpatient department. He admitted to being a very "nervous" person but declined further psychiatric assessment. He complained of recurrent symptoms albeit of lesser severity and did not require further admissions to hospital.

DISCUSSION

The episodic wheezy dyspnoea which mimicked bronchial asthma in this patient was the result of vocal cord adduction and upper airways obstruction. This was best demonstrated by direct examination of the vocal cords during acute exacerbation of symptoms. The vocal cords play an important adaptive function in shaping the velocity of airflow during tidal breathing both in health and disease[3,7,8]. In the presence of intrathoracic airways obstruction, narrowing of the glottis during expiration may be an efficient mechanism which maintains high end-expiratory lung volumes and serve to reduce the work of inspiratory muscles[6]. Nevertheless, this adaptive mechanism may go astray and result in functional upper airway obstruction during both phases of tidal breathing and in the absence of lower airways disease as illustrated by this patient[1-4]. The mechanism for recurrent functional upper airways obstruction is unknown but psychological factors may contribute to the periodic vocal cord dysfunction[4].

The glottic dysfunction in this patient was corrected by sedation following which the vocal cords were observed to abduct normally. This observation is consistent with the notion that the abnormal motion of the vocal cords during symptomatic episodes was not due to intrinsically abnormal glottic apparatus or efferent nerves. The application of CPAP have been reported to correct the glottic dysfunction in some cases[3]. However, the application of CPAP did not have a favourable effect on either the symptoms or the breathing pattern in this patient. A normal flow volume curve could not be recorded because the application of a noseclip precipitated a wheezy episode even during remission. However, much greater expiratory flow rates were recorded during coughing when the vocal cords transiently abduct and the flow volume curve gave a better reflection of airflow from the lower airways (Fig 1). The absence of subglottic disease may be further inferred from the normal alveolar-arterial gradient for oxygen during acute illness and the normal endoscopic findings in the trachea and main airways.

Ventilatory timing and depth can be influenced by the use of respiratory apparatus. In normal subjects, the change in breathing route from nose to mouth following the application of a noseclip may result in a slower tidal frequency and higher tidal volume[9,10]. The observation in the present case that a noseclip could trigger glottic dysfunction, acute wheezyness and dyspnoea associated with rapid shallow breathing and dynamic hyperinflation (Fig 2) was clearly abnormal and had not been reported previously. Furthermore, the asynchronous motion between RC and ABD suggested that the co-ordination between different groups of chest wall muscles might also be abnormal in this syndrome[11]. The rib cage was held in an inspiratory position while the abdominal compartment made the major contribution to volume displacements during tidal breathing (Fig 2). Further definition of the patterns of respiratory muscle recruitment was not possible because respiratory muscle EMG activity and thoracic versus abdominal

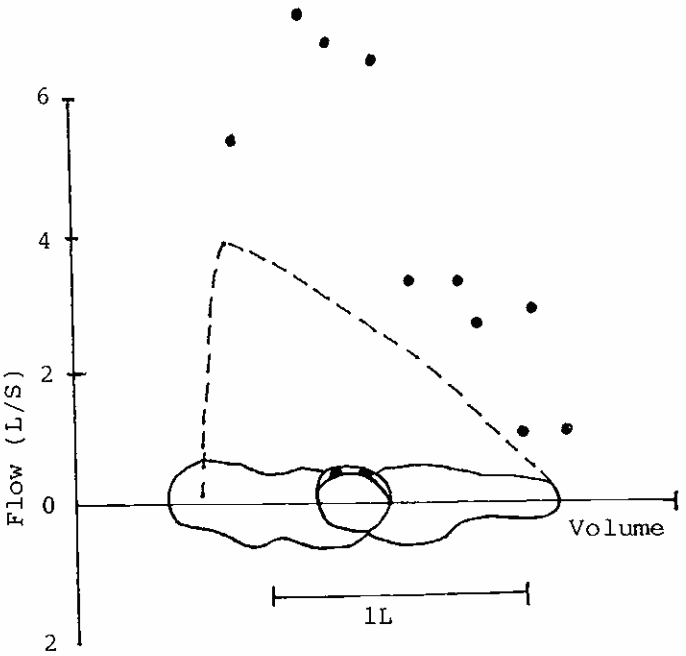

Fig 1. Flow-volume curve showing a small tidal volume and marked reduction of both maximal expiratory and inspiratory flows [solid lines]. Larger expiratory flows were measured as transient peaks [●] and plateaus [dashed line] during repeated coughs form TLC.

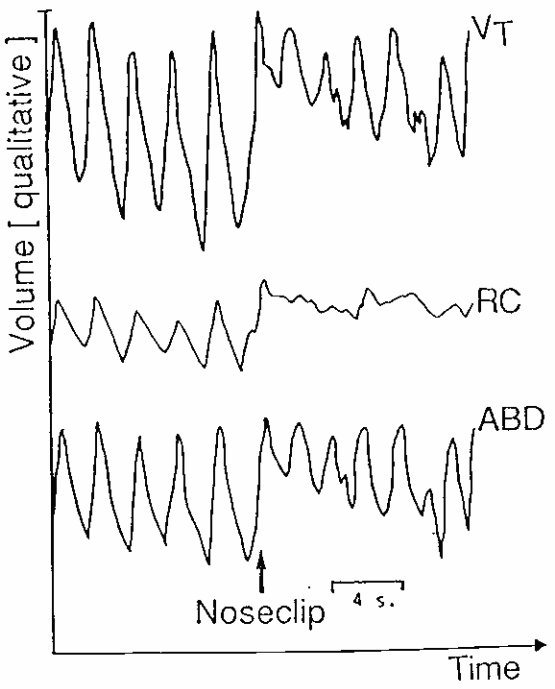

Fig 2. The sum of RC plus ABD signals which reflect the tidal volume[V_T] is in the upper panel. The individual RC and ABD displacements are in the middle and lower panels. Note the abnormal breathing pattern which followed application of a noseclip [arrow] which precipitated the signs and symptoms of an acute spontaneous attack of wheezy dyspnoea.

compartment pressures were not measured in this patient. Nevertheless, tonic contraction of the accessary inspiratory muscles of the chest wall was evidenced by inspection of the sternomastoids and palpation of the scalene muscles. This may account for his sensation of discomfort over the anterior neck and upper chest during attacks. Since no recordings of thoraco-abdominal motion was made during an actual spontaneous episode, it is not certain if the asynchronous motion observed during noseclip application reproduced exactly the breathing pattern during a naturally occurring attack of asthma in this patient. The symptoms and physical signs were however very similar.

This case report describes an unusual disorder of upper airway function which should be distinguished from bronchial asthma. Further studies may elucidate the role of chest wall muscles in this condition.

REFERENCES

1. Paterson R, Schatz M, Horton M. Munchausen's stridor: Non-organic laryngeal obstruction. Clin Allergy 1974; 4: 307-10.
2. Christopher KL, Wood RP, Eckert RC, Blager FB, Raney RA, Souhrada JF. Vocal-cord dysfunction presenting as asthma. N Engl J Med 1983; 308:1566-70.
3. McFadden ER Jr. Glottic function and dysfunction. J Allergy Clin Immunol 1987; 79:707-10.
4. Selner JC, Staudenmayer H, Koepe LW, Harvey R, Christopher K. Vocal cord dysfunction: the importance of psychological factors and provocation challenge testing. J Allergy Clin Immunol 1987; 79:726-33.
5. Lim TK. Nasal CPAP therapy in the obstructive sleep apnoea syndrome. Singapore Med J 1988; 29:157-63.
6. Lim TK. The effect of nasal CPAP on patients with chronic obstructive pulmonary disease. Singapore Med J 1990; 31:233-37.
7. Bartlett D, Remmers JE, Gautier H. Laryngeal regulation of respiratory airflow. Respir Physiol 1973; 18:194-204.
8. Collet PW, Brancatisano T, Engel LA. Upper airway dimensions and movements in bronchial asthma. Am Rev Respir Dis 1986; 133:1143-9.
9. Gilbert R, Auchincloss JH, Brodsky J, Boden W. Changes in tidal volume, frequency and ventilation induced by their measurement. J Appl Physiol 1972; 33:252-4.
10. Rodenstein DO, Mercenier C, Stanescu DC. Influence of the respiratory route on the resting breathing pattern in humans. Am Rev Respir Dis 1985; 131:163-6.
11. Mead J, Smith JC, Loring SH. Volume displacements of the chest wall and their mechanical significance. In: Roussos C, Macklem PT. eds. *The Thorax*. New York: Marcel Dekker Inc, 1985:369-92.

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