

Case Report

ALL THAT WHEEZES IS NOT ASTHMA: LARYNGEAL MALIGNANCY MASQUERADING AS BRONCHIAL ASTHMA

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INTRODUCTION

The diagnosis of bronchial asthma is often made on the basis of wheezing. However, it is well known that several other conditions may cause dyspnea and wheezing. Thus, it is important to rule out other conditions before labelling a patient as bronchial asthma especially if there is poor response to medications. We present a case of well differentiated squamous cell carcinoma of larynx presenting as a case of bronchial asthma.

CASES

52 yrs/female, house wife, presented to the emergency department with complaints of insidious onset non productive cough of 6 months duration and intermittent breathlessness for 4 months.

Cough was paroxysmal in nature, more during night and increased by exposure dust and smoke. Breathlessness was associated with wheezing which was more during night. Breathlessness was intermittent in onset. It became persistent for past 1 month and since past week it was severe enough to disturb sleep of patient.

There was no associated history of any nasal discharge, fever, loss of appetite, loss of weight, chest pain, palpitations, skin rashes, joint pains, joint swelling, difficulty in swallowing. There was no significant occupational or drug exposure. There was no such illness in any family member nor any history of bronchial asthma, allergic rhinitis, pulmonary tuberculosis or ATT intake in any family member. Patient was a tobacco chewer and had significant history of biomass fuel exposure for 15 years but no history of I/V drug abuse or sexual promiscuity. Patient was taking MDI asthalin 2 puffs 3-4 times /day, Tablet Deriphyllin 400mg once daily.

Patient was also prescribed MDI seroflo 250 mg 2 puffs BD for past 1 month but was taking it on as needed basis.

Patient was admitted in another hospital for 3 days. He had no relief in the symptoms despite treatment with nebulised short acting beta 2 stimulants, I/ V corticosteroids and IV magnesium.

On examination patient was conscious and oriented. Pulse was 108 beats/ min, regular. Blood pressure was 130/ 80 mm Hg, Temperature 98.6⁰ F, Respiratory rate was 22/minute and SPO₂ 95% room air. There was no pallor, icterus, cyanosis, lymphadenopathy, clubbing, pedal edema. Patient was in respiratory distress and was not able to complete full sentences. Chest revealed bilateral diffuse biphasic ronchi which were louder on inspiration and patient was having increase in respiratory distress and voice was feeling hoarse. This lead suspicion of upper airway obstruction, though there was no clear stridor. Vocal cord pathology was suspected as patient started to have voice changes. Rest of systemic examination was normal.

Investigations showed Hemoglobin-13.6gm/dl, TLC-5200/ul, DLC= p-84, l-12, m-4 and PLC-1.9Lac., KFT= Urea-19.7, Creat-0.9, RBS-131, LFT= ALB-4.3, TBIL-1.14, DBIL-0.42, ALT-4, AST-37.3, ALP-115. X ray chest P/A view (Figure 1) and CT scan chest (Figure 2) was normal.

So patient was taken for Spirometry (Figure 3) which revealed flattening of both loops, highly characteristic of fixed upper way obstruction.

Following this a CT Scan neck was performed. CT Neck showed a large intramural soft tissue shadow occluding around 70 % of airway lumen. So, there was strong suspicion of glottic malignancy (Figure 4).

Patient was taken for urgent bronchoscopy after taken consent. Bronchoscopy revealed a large infraglottic growth which was highly vascular on NBI (narrow band imaging) and occluding almost 70 % of airway lumen (Figure 5). Histopathology revealed well differentiated squamous carcinoma.

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Figure 1: X Ray Chest

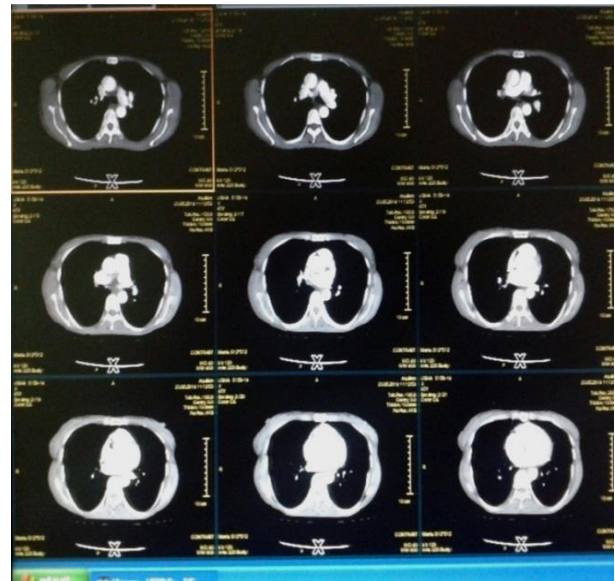


Figure 2: CT Cheat

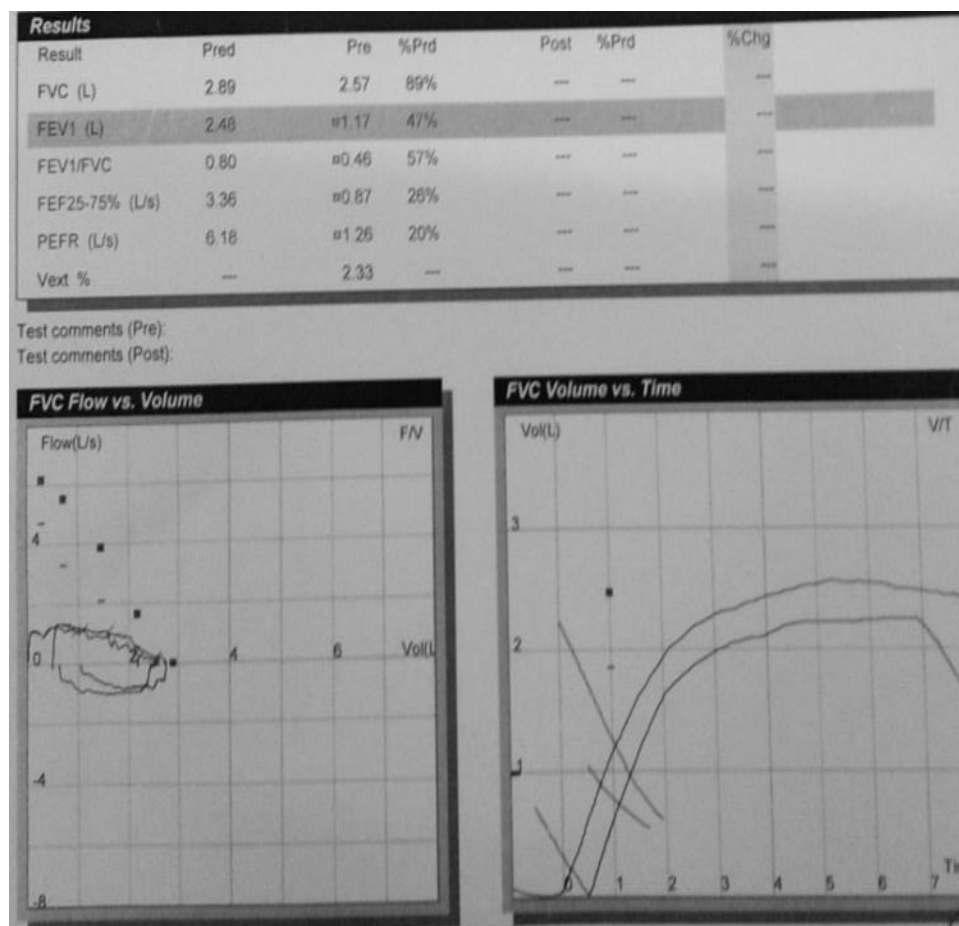


Figure 3: Spirometry

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Figure 4: CT Neck



Figure 5: Bronchoscopy

DISCUSSION

Studies have shown it the patients who have sub acute onset of upper airway obstruction (UAO) are often misdiagnosed as Bronchial or COPD. Significant upper airway obstruction may be obscured for a considerable period of time, resulting in delayed diagnosis and possible catastrophic outcome (Pratter *et al.*, 1983). First step to suspect a case of upper airway obstruction may be when there occurs incomplete or absent response to conventional asthma medications in a patient of wheezing (Newman *et al.*, 1995). Onset and rate of progression of symptoms may be important clue to the etiology. For example, foreign body aspiration often has an acute onset of symptoms, while a goiter compressing the airway or endobronchial tumor typically cause slowly progressive dyspneas. A past history of neck or thyroid surgery may direct attention to possible vocal cord while prior intubation suggests for possible vocal fold trauma or paralysis, tracheal stenosis, or tracheomalacia (Geffin *et al.*, 1971). Patients with features of dyspnea, wheeze, intractable (often barking), and recurrent pulmonary infections is suggestive of tracheobronchomalacia and hyperdynamic airway collapse (Murgu and Colt, 2006).

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Cigarette smoking more than 10 pack years increases the possibility that wheezing is due to COPD, but also increases the risk bronchogenic carcinoma or laryngeal malignancy. Typically, significant anatomic obstruction precedes overt symptoms. By the time exertional dyspnea occurs, the airway diameter is likely to be reduced to about 8 mm and dyspnea at rest develops when the airway diameter reaches 5 mm, coinciding with the onset of stridor (Ernst *et al.*, 2004). Stridor is a loud, musical sound of constant pitch that usually connotes obstruction of the larynx or upper trachea.

These symptoms are especially prominent during exercise and also may be aggravated by a change in body position (neck flexion) (Forgacs, 1978). The patient may complain more breathless in recumbent position and may have a severely disrupted sleep pattern. Even some patients may present as sleep apnea syndrome with day time sleepiness. Maneuvers that increase air flow, such as voluntary hyperventilation, accentuate stridor (Forgacs, 1978). If neck flexion changes the intensity of stridor, it suggesting a thoracic outlet obstruction. When the obstructing lesion is below the thoracic inlet, both inspiratory and expiratory stridor may be heard which may be mistaken for loud wheeze. Muffling of the voice without hoarseness represents a supraglottic process and Hoarseness is the sign of a laryngeal abnormality (Loudon and Murphy, 1984).

Upper airway is the conducting passages extending from the nose or mouth to the main carina. UAO may be functional or anatomic and may develop acutely or sub-acutely. Being tubular in nature narrowing of the upper respiratory tract has an exponential effect on airflow because linear airflow is a function of the fourth power of the radius (Khosh and Lebovics, 2001). The narrowest portion of the larynx is at the glottis in adults and the subglottis in infants. Laryngeal obstruction has a particular importance because the larynx is the narrowest portion of the upper airway and in acute cases needs emergency management (Dickison, 1987).

Etiology of upper airway obstruction depends upon the age and onset of obstruction. Infectious causes and foreign bodies are more common in children and onset tends to be more acute while post traumatic and malignancies are more common in adults and tend to present with subacute onset and hence may be missed in early stages. Various etiologies can be grouped as:

1. Infections:

- Suppurative parotitis
- Retropharyngeal abscess
- Tonsillitis
- Ludwig's angina
- Epiglottitis
- Laryngitis
- Laryngotracheobronchitis (croup)
- Diphtheria

2. Traumatic Causes:

- Laryngeal trauma/ Hemorrhage
- Airway burn
- Facial trauma (mandibular or maxillary fractures)

3. Foreign Bodies

4. Iatrogenic Causes:

- Tracheal stenosis post-tracheostomy
- Tracheal stenosis post-intubation
- Mucous ball from transtracheal catheter

5. Vocal Cord - paralysis / Vocal cord dysfunction

6. Tumors: Laryngeal tumors (benign or malignant)/ esophageal lesions
Laryngeal papillomatosis

7. Tracheal Stenosis: (caused by intrinsic or extrinsic tumors)

8. Angioedema: Anaphylactic reactions C1 inhibitor deficiency, Angiotensin-converting enzyme inhibitors

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9. Vascular Rings: Right-sided aortic arch

Investigations in UAO:

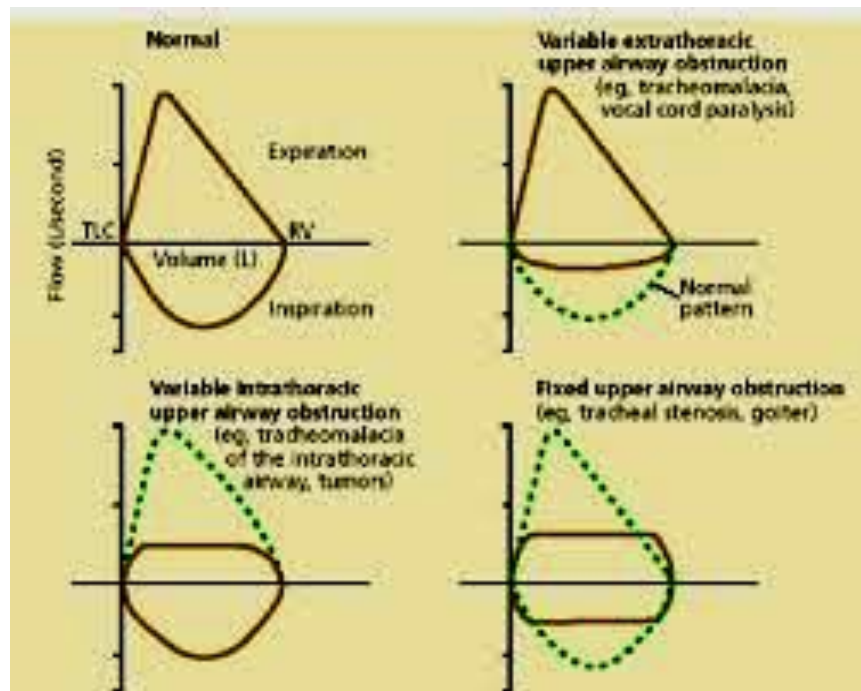
The most important diagnostic tool if UAO is suspected is a quick history and physical examination. Many times, management of a patient with UAO must start simultaneously with the diagnostic process. It is useful to separate patients with potential UAO into those with severe symptoms and impending respiratory failure and those with a more indolent course and less severe symptoms.

Plain Chest and Neck Radiographs: Plain neck and chest films may show features as tracheal deviation, extrinsic compression, or radiopaque foreign bodies. In one study, only 13 of 53 tracheal tumors were evident to the radiologist on the standard PA roentgenogram (Aboussouan and Stoller, 1994).

Computed Tomography: Computed tomography (CT) is important in investigating UAO in the stable patient or in the unstable patient with an already secured airway. It can also provide information on the degree and extension of airway compromise in UAO. The sensitivity of CT scanning for detecting upper airway disease is 97 percent versus 66 percent compared to routine chest roentgenogram (Aboussouan and Stoller, 1994). The use of paired inspiratory dynamic and expiratory multislice HRCT has proved helpful for the diagnosis of tracheomalacia. Tracheomalacia is generally defined as a reduction in cross-sectional area of greater than 50 percent on expiratory images (Lee and Boiselle, 2010).

MRI: This is best used to investigate vascular structures surrounding central airways, such as vascular rings or aneurysms that may compress the trachea.

Spirometry: Its sensitivity is low in early stages as abnormalities in flow-volume loop develop only when airway lumen is less than 8 mm in diameter. This corresponds to an obstruction of more than 80 percent of the tracheal lumen. Also the forced expiratory volume in 1s (FEV1) remains above 90 percent of control until a 6-mm orifice is created (Owens and Murphy, 1983). But analysis of the flow-volume loops may be helpful suggesting the location and functional severity of the obstruction. Typical patterns of the flow-volume loop may be seen, depending on whether the obstruction to flow is “fixed” or “variable,” and whether the site of the obstruction is above or below the thoracic outlet or suprasternal notch (Miller and Hyatt, 1969).



Bronchoscopy: Rigid or flexible bronchoscopy is the most effective tool in establishing diagnosis and frequently provides the best way to secure the airway as well to deliver treatment including laser therapy,

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photoresection, electrocautery, electrosurgery, balloon bronchoplasty, and tracheal stenting once the airway has been secured and the patient stabilized (Ayers and Beamis, 2001).

Management of UAO:

Establishing a secure and patent airway is the most important goal in the resuscitation of a patient with acute UAO. A quick evaluation considering age group, history, physical examination, and clinical circumstances helps determine the site and cause of obstruction, the severity of the obstruction, and the need to establish an airway urgently. Several medical and surgical approaches are available in the management of UAO. The selection of the intervention will depend on the cause of UAO and the urgency to obtain a secure airway.

Heimlich Maneuver: In the outpatient setting the most common cause of UAO is obstruction of the larynx with a foreign body. Heimlich maneuver is recommended for relief of the airway obstruction in adults and children one to eight years of age. Repeat abdominal thrusts may be needed to clear the airway.

Racemic Epinephrine: Racemic epinephrine administered by means of a nebulizer has been proven to be effective in treating croup (laryngotracheobronchitis) in the pediatric population decreasing morbidity, mortality, and hospital stay but is not effective in the treatment of epiglottitis and may be deleterious (Quan, 1992). Racemic epinephrine also is used to treat postextubation laryngeal edema, which has been reported to occur from 2.3% to 6.9% (Quan, 1992). These patients should remain in the intensive care unit under careful observation until it is confirmed that the UAO has resolved or greatly improved.

Corticosteroids: Corticosteroids have been used to treat UAO because of their potential beneficial effect in reducing airway edema. Randomized trials have confirmed the usefulness of corticosteroids in the treatment of croup with decreases in the need for intubation and hospital stay. However, the treatment of epiglottitis with steroids is controversial and often contraindicated (Kairys *et al.*, 1989).

Heliox: Heliox, a helium-oxygen gas mixture, is effective in reducing the work of breathing by decreasing airway resistance to turbulent flow in the density-dependent pressure drop across the airway obstruction. Heliox has been used in several conditions including post-extubation laryngeal edema, tracheal stenosis or extrinsic compression, status asthmaticus, and angioedema. The use of heliox in patients with severe UAO should only be used to provide temporary support pending definitive diagnosis and management (Boorstein *et al.*, 1989).

Endotracheal Intubation: In most cases of UAO, the patency of the upper airway can be reestablished with endotracheal intubation after rapid assessment of the patient's airway anatomy. Evaluation is done with assessment of mouth opening (>40 mm), dentition, cervical spine mobility (flexion-extension), thyromental distance (normal is >3 finger breadths) and the function of the temporomandibular joint. These features are key to subsequent success and avoidance of complications (American Society of Anesthesiologists Task Force on Management of the Difficult Airway, 2003).

Surgical Interventions: Overall, emergency laryngotracheal intubation is effective in approximately 97% of cases. Thus, a surgical airway is needed in only 3% of such emergencies. Surgical airway is considered emergently mainly in cases of laryngotracheal trauma, foreign body lodged in the pharyngolaryngeal area or severe anatomic deformity caused by trauma.

Cricothyroidotomy: It is the procedure of choice in the emergency setting; it is easier to perform, simpler, and more likely to be successful than tracheotomy. It is recommended that cricothyroidotomy to be converted to tracheotomy if longer than 72 hours of use is anticipated. This is because intraluminal diameter of the trachea is narrowest at the level of the cricoid and there is concern that prolonged use of a cricothyroidotomy may cause subglottic injury and lead to subglottic narrowing.

Tracheostomy: It is probably the last option available to establish an airway in acute UAO. The only indication for emergency tracheostomy is laryngeal trauma as it is a relative contraindication to cricothyroidotomy and laryngotracheal intubation. This procedure is time consuming and requires expertise and attention to detail (Grillo, 2003).

Laser Therapy: Carbon dioxide or neodymium yttrium aluminum garnet (Nd:YAG) laser therapy can be used to treat intraluminal tracheobronchial lesions like tracheal webs, to treat benign obstructive lesions (papillomas), or as palliative therapy for malignant tracheobronchial lesions.

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Tracheal Stents can be helpful to maintain a patent airway in patients with tracheal obstruction caused by benign or malignant conditions.

They are put either via rigid or flexible bronchoscopy. Airway stents can provide prolonged palliation from an unresectable recalcitrant benign stenosis or rapidly recurrent endoluminal tumor (Wood *et al.*, 2003).

A specific complication is of UAO is Postobstructive pulmonary edema. It is the development of pulmonary edema following UAO without evidence of any other underlying cardiopulmonary condition. There are two types of post obstructive pulmonary edema.

Type I: it follows a sudden, severe airway obstruction such as post-extubation, laryngospasm, epiglottitis, croup, strangulation, choking, and hanging. So, Type I is associated with any cause of acute UAO. The exact pathogenesis is unclear but the current theory is that young patients are able to generate extremely high negative intrathoracic pressure, which increases venous return, decreases cardiac output, and causes fluid transudation into the alveolar space (Van Kooy and Gargiulo, 2000).

Type II: Pulmonary edema develops after surgical relief of long-term UAO. Reported causes include tonsillectomy and removal of upper airway tumors. Post obstructive pulmonary edema usually occurs within one hour of a precipitating event but it has reported to occur up to six hours later. The cause of type II post obstructive pulmonary edema is less clear, but it appears that the obstructing lesion produces a modest level of positive end expiratory pressure (PEEP) and increases end-expiratory lung volume. The sudden removal of this PEEP may then lead to interstitial fluid transudation and pulmonary edema (Wilms and Shure, 1988).

The treatment of post obstructive pulmonary edema is supportive with supplemental oxygen, intubation, and application of low levels of PEEP (5 cm H₂O). The role of diuretics in this setting is unclear. Most patients respond promptly to appropriate treatment and have full recovery (Van Kooy and Gargiulo, 2000).

Conclusion

Upper airway pathology may with varied number of symptoms. So, always assess upper airway in patients who present with wheezy chest and are not responding to your treatment as response is expected. Radiology gives clue in most cases, so, scrutinize these carefully before going for other investigations.

REFERENCES

- Aboussouan L and Stoller JK (1994).** Diagnosis and management of upper airway obstruction. *Clinics in Chest Medicine* **15**(1) 35-53.
- American Society of Anesthesiologists Task Force on Management of the Difficult Airway (2003).** Practice guidelines form management of the difficult airway: an updated report by the American Society of Anesthesiologists Task Force on Management of the Difficult Airway. *Anesthesiology* **98** 1269–1277.
- Ayers ML and Beamis JF Jr (2001).** Rigid bronchoscopy in the twenty first century. *Clinics in Chest Medicine* **22** 355–364.
- Boorstein JM, Boorstein SM, Humphries GN et al., (1989).** Using helium–oxygen mixtures in the emergency management of acute upper airway obstruction. *Annals of Emergency Medicine* **18** 688-90.
- Dickison AE (1987).** The normal and abnormal pediatric airway. Recognition and management of obstruction. *Clinics in Chest Medicine* **8**(4) 583-96.
- Ernst A, Feller-Kopman D, Becker HD et al., (2004).** Central airway obstruction. *American Journal of Respiratory and Critical Care Medicine* **169** 1278–1297.
- Forgacs P (1978).** The functional basis of pulmonary sounds. *Chest* **73** 399.
- Geffin B, Grillo HC, Cooper JD and Pontoppidan H (1971).** Stenosis following tracheostomy for respiratory care. *Journal of the American Medical Association* **216** 1984-88.
- Grillo HC (2003).** Development of tracheal surgery: A historical re- view. Part 1: Techniques of tracheal surgery. *The Annals of Thoracic Surgery* **75** 610–619.
- Kairys SW, Olmstead EM and O'Connor GT (1989).** Steroid treatment of laryngotracheitis: a meta-analysis of the evidence from randomized trials. *Pediatrics*, **83** 683-93.

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Khosh MM and Lebovics RS (2001). Upper airway obstruction. In: Parrillo JE, Dellinger RP, edition, *Critical Care Medicine* (USA, St. Louis: Mosby) 808-25.

Lee KS and Boisselle PM (2010). Update on multi detector computed tomography imaging of the airways. *Journal of Thoracic Imaging* **25** 112.

Loudon R and Murphy RL Jr (1984). Lung sounds. *American Review of Respiratory Disease* **130** 663.

Miller RD and Hyatt RE (1969). Obstructing lesions of the larynx and trachea: clinical and physiologic characteristics. *Mayo Clinic Proceedings* **44** 145.

Murgu SD and Colt HG (2006). Tracheobronchomalacia and excessive dynamic airway collapse. *Respirology* **11** 388.

Newman KB, Mason UG 3rd and Schmaling KB (1995). Clinical features of vocal cord dysfunction. *American Journal of Respiratory and Critical Care Medicine* **152** 1382.

Owens GR and Murphy DM (1983). Spirometric diagnosis of upper airway obstruction. *Archives of Internal Medicine* **143** 1331–1334.

Pratter MR, Hingston DM and Irwin RS (1983). Diagnosis of bronchial asthma by clinical evaluation. An unreliable method. *Chest* **84** 42.

Quan L (1992). Diagnosis and treatment of croup. *American Family Physician* **46**(3) 747-55.

Van Kooy MA and Gargiulo RF (2000). Post obstructive pulmonary edema. *American Family Physician* **62** 401-4

Wilms D and Shure D (1988). Pulmonary edema due to upper airway obstruction in adults. *Chest* **94** 1090-2.

Wood DE, Liu YH, Vallieres E et al., (2003). Airway stenting for malignant and benign tracheobronchial stenosis. *The Annals of Thoracic Surgery* **76** 167-74.