Usefulness of Flow Volume Loops in Emergency Center and ICU Settings*

Kalpalatha K. Guntupalli, MD, FCCP; Venkata Bandi, MD; Corinne Sirgi, MD; Caryn Pope, RRT; Alex Rios, MD; and William Eschenbacher, MD, FCCP

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**Abbreviations:** EC=emergency center; FVL=flow volume loop; MICU=medical ICU; MVV=maximal voluntary ventilation; VC=vocal cord

In general, spirometry and flow volume loops (FVLs) are obtained electively to confirm a clinical diagnosis and quantitate the degree of functional abnormality of the lungs. We describe our experience with five cases encountered in the past year in an acute care setting (medical ICU [MICU], emergency center [EC]), where FVLs were obtained emergently which helped establish the diagnosis. In all five cases, further treatment of the patient was significantly influenced by the diagnostic FVLs. We conclude that pulmonary function tests, including FVLs, are useful but probably underutilized in the acute care setting.

**Case Reports**

**Case 1**

A 57-year-old woman with a history of metastatic breast cancer presented to the EC in respiratory distress. For 2 months prior to admission, the patient had experienced multiple episodes of shortness of breath with exercise initially, but later also at rest. On physical examination, the patient was noted to have stridor and/or inspiratory wheezing. Physical examination was also significant for cervical, supraclavicular lymphadenopathy, lymphedema of the right arm, and radiation skin changes on the neck and chest. She had been treated with modified radical mastectomy and radiation therapy 5 weeks prior to admission. Arterial blood gas values on admission to the EC were as follows: pH, 7.24; PaCO₂, 70 mm Hg; PaO₂, 78 mm Hg. She was intubated for acute respiratory failure. Chest radiograph showed right lower lobe pneumonia. A fiberoptic bronchoscopy, done while she was intubated, revealed a normal tracheobronchial tree. The patient was successfully extubated 2 days later and was able to speak properly postextubation. She was transferred to a treatment floor. She needed reintubation 3 days later for “impending respiratory failure” and was transferred back to the MICU. Because of relief of symptoms immediately following intubation, a dynamic or structural upper airway obstruction, bypassed by the endotracheal tube, was suspected and portable spirometry with FVL was done after the second extubation (Fig 1). The FVL was consistent with variable extrathoracic obstruction. Evaluation with direct laryngoscopy showed adduction of both vocal cords (Fig 2). A diagnosis of bilateral vocal cord paralysis due to radiation or metastases was made and a tracheostomy was performed with relief of symptoms and resolution of recurrent respiratory failure.

**Comments**

1. Patients with unilateral vocal cord (VC) paralysis have hoarseness of voice but are usually not short of breath. In contrast, patients with bilateral VC paralysis can phonate well because the cords can vibrate together. They are short of breath with decreased exercise tolerance during periods of increased flow requirements because of the small opening due to adduction of the VCs. This was exemplified by this vocal but breathless patient.

2. The most common cause of bilateral VC paralysis is the postthyroidectomy state. After radiation to the neck, VC paralysis has been described to occur years later. This was the most likely cause in this patient.
Figure 1. Case 1. FVL demonstrating significant inspiratory limitation consistent with variable extrathoracic obstruction. Case of bilateral VC paralysis. The dotted line is predicted normal FVL for this patient.

(3) Variable extrathoracic obstruction results usually from benign causes such as bilateral VC paralysis, subglottic stenosis, etc. Variable intrathoracic obstruction, however, is usually due to malignancy.

Case 2

A 40-year-old woman, with 100 pack-year smoking history, was admitted to the hospital for right lower lobe pneumonia, transient numbness of fingers, dysphagia, right lower lobe infiltrates, and pleural effusion. CT scan of the chest showed bilateral mediastinal and peritracheal masses with subcarinal extension and large right pleural effusion (Fig 3). A diagnostic fiberoptic bronchoscopy was performed. Significant extrinsic compression of both mainstem bronchi and trachea was seen. Because cytologic study and Wang needle aspirate were negative, a diagnostic mediastinoscopy was done that revealed non-small cell cancer. She was extubated postoperatively, but required reintubation twice for shortness of breath and impending respiratory failure. Palliative brachytherapy, chemotherapy, and external-beam radiation therapy were given. Two weeks later, she was admitted to the MICU for shortness of breath and respiratory failure requiring intubation. During this admission, repeated self-extubations promptly required reintubation for respiratory distress. Because of relief with intubation, a dynamic

Figure 2. Case 1. Lack of movement of VC's during respiration with small fixed opening. Case of bilateral VC paralysis.
airway obstruction was suspected and an FVL was performed while she was extubated. FVL was suggestive of variable intrathoracic obstruction (Fig 4). The patient refused tracheostomy and died later of worsening respiratory failure.

Comments

This patient had variable intrathoracic obstruction due to compression of the tracheobronchial tree with tumor. Unlike variable extrathoracic obstruction, which is usually due to benign causes, variable intrathoracic obstruction is generally due to malignancy.

Case 3

A 36-year-old nonsmoking, diabetic woman, presented to the emergency department with shortness of breath, hoarseness, cough, and wheezing of 2 weeks’ duration. Four months prior to hospital admission, she had prolonged hospitalization at another facility for complications due to diabetic ketoacidosis. She required mechanical ventilation and tracheostomy for an unknown period at that time. History was also significant for asthma during childhood. Because of the history of asthma, the patient was diagnosed as having an asthma exacerbation during the current hospital admission and given several albuterol treatments with minimal relief. On admission to the MICU, the patient was in acute respiratory distress with respiratory rate of 32/min and pulse rate of 120/min. Stridor was heard, but no wheezing was heard on auscultation of lungs. Chest radiograph was clear. Because of clear lung examination with continued respiratory distress, a portable spirometry and FVL was performed while breathing room air (Fig 5) and helium oxygen mixture (80:20) (Fig 6). Both FVLs were consistent with fixed airway obstruction. A tomogram and MRI were done subsequently.

The tomogram revealed a 1.5-cm segment of irregular nodular narrowing of the trachea 3.5 cm distal to the glottis (Fig 7). MRI of the trachea confirmed an abnormal soft-tissue intensity involving the lateral aspects of the subglottic area at the T1-T2 level with tracheal narrowing (Fig 8).

Comments

This patient was being treated for asthma exacerbation. As a general rule, shortness of breath in an asthmatic is due to exacerbation of asthma. But one needs to be aware of other possibilities, as highlighted by this case. Large airway obstruction causes turbulence in airflows leading to increased work of breathing and reduced flows for the same amount of work. By substituting a less dense but more viscous gas like heliox, the flows could be favorably improved leading to reduced work of breathing.
CASE 4

A 48-year-old Hispanic man presented to the emergency department of Ben Taub General Hospital complaining of "worsening of asthma symptoms." The patient immigrated from Mexico 10 years earlier. He was diagnosed as having asthma 12 years ago. The patient received treatment with steroids and bronchodilator aerosols. Medical history was significant for rhinorrhea, shortness of breath, and hoarseness. Dyspnea and hoarseness worsened over the last 2 years. In the emergency department, the patient was given β2-agonist aerosol treatments. As part of an ongoing clinical research protocol for acute asthma exacerbation, FVL was done, which showed a fixed airway obstruction (Fig 9). Closer physical examination revealed granulomatous lesions in both nares and hypopharynx. A laryngoscopic evaluation revealed a subglottic mass. A presumptive diagnosis of rhinoscleroma was made that was later confirmed by culture of *Klebsiella rhinoscleromatis*. Histopathologic study of the subglottic mass was consistent with rhinoscleroma. The patient underwent an elective tracheostomy for airway control and was started on a regimen of ciprofloxacin. An endoscopic laser resection of the scleroma was done 10 days later. Steroids and β2-aerosol treatment were discontinued and the patient remained asymptomatic.

Comments

Rhinoscleroma is a chronic granulomatous infection of the upper airways caused by *K rhinoscleromatis*. It is a chronic inflammatory granulomatous reaction involving the nose, larynx, and occasionally the trachea and bronchi.

CASE 5

A 45-year-old woman was admitted to the hospital for shortness of breath, chest pain, and numbness of fingers. She was hospitalized to rule out myocardial infarction. She was discharged from the hospital 10 days earlier after being treated for asthma exacerbation. The patient was diagnosed as having asthma 2 years earlier, with multiple exacerbations requiring frequent EC visits and 6 hospitalizations in the preceding 6 months. She also admits to "worsening of voice" and "choking of throat" during exacerbations. The patient was a smoker and labeled as a steroid-dependent asthmatic. On current admission, she was tachycardic with a pulse rate of 156/min, respiratory rate of 22/min, with stridor and/or inspiratory wheezing on auscultation. Arterial blood gas studies revealed a pH of 7.53, Pco2 of 25, and Po2 of 213 on 100% non-rebreather mask. Physical examination was significant for cushingoid facies and proximal muscle weakness. Myocardial infarction was ruled out. Pulmonary service evaluation was requested for poorly controlled asthma. Because of multiple visits and questionable stridor, a pulmonary function test and direct laryngoscopy were performed. FVL indicated variable extrathoracic obstruction (Fig 10) and direct laryngoscopy confirmed the cause to be paradoxic bilateral vocal cord adduction during inspiration. The steroid therapy was tapered, and speech therapy and psychiatry consults were obtained for counseling and treatment. A panic disorder with mild agoraphobia was diagnosed by the psychiatry consultant. FVLs several years before and after current hospital admission were normal, con-

![Flow vs Volume Graph](image)

**Figure 5.** Case 3. FVL in patient with tracheal stenosis while breathing room air.

![Flow vs Volume Graph](image)

**Figure 6.** Case 3. FVL in patient with tracheal stenosis while breathing helium-oxygen mixture. Flow rates are improved compared with values while breathing room air (see Fig 5).
sistent with functional variable extrathoracic obstruction during exacerbations due to paroxysmal VC dysfunction (Figs 11 and 12).

Comments

This patient was diagnosed as having steroid-dependent asthma, but the acute major component was VC dysfunction. The patient was suffering from side effects of long-term steroid usage with cushingoid facies and muscle weakness.

DISCUSSION

Because acute respiratory failure is the most common reason for admission to MICUs, the differential diagnosis for acute presentation of dyspnea becomes critical. Acuteness of the medical problem usually requires immediate intervention in many instances. Therefore, exact cause of shortness of breath or respiratory failure may be masked in these patients. This is particularly applicable to patients with dynamic obstruction of the airways, in which endotracheal intubation may alleviate the problem and not be apparent again until after extubation.

Upper airway obstruction is defined as obstruction at/or above the carina. Normal coronal tracheal airway diameter is 10 to 25 mm. FVLs may not show abnormality until the diameter is reduced to 8 mm or less. Patients with upper airway obstruction may not become symptomatic until either the lumen is compromised significantly or the flow rate increases across a fixed compromised lumen as with exercise. Generally, shortness of breath with exercise occurs when airway lumen is reduced to 8 mm or less, and shortness of breath at rest with stridor, with reduction to 5 mm or less. The dynamics of intrathoracic and extrathoracic airways differ resulting in different flow characteristics during inspiration and expiration. Variable intrathoracic obstruction has fairly well-preserved flows during inspiration. Expiratory flow is significantly cut off when the airway is compressed during expiration by positive pleural pressure. In variable intrathoracic obstruction, marked reduction in maximum voluntary ventilation (MVV), peak expiratory flow rate, forced expiratory flow rate between 25% and 75% of FVC, FEV₁, and possibly FVC is seen. Usually, a characteristic flattening of expiratory loop distinguishes from those with emphysema, in whom curvilinear expiratory loop is seen.

In contrast, variable extrathoracic obstruction causes inspiratory flow compromise because of marked negative intraluminal pressure below the lesion while the expiratory flow is fairly well preserved. Variable extrathoracic obstruction does share a marked reduction in MVV with variable intrathoracic obstruction, but reduction in peak inspiratory
flow rate and forced inspiratory volume in 1 s (FIV₁) with marked slowing of inspiratory flow is also seen. The expiratory flow loop tends to have normal contour. With inspiration, a slow, but fairly constant flow is seen.³⁴⁸ Ratio of expired to inspiratory flow at mid-vital capacity is normally 0.9 but is high in variable extrathoracic obstruction and low in variable intrathoracic obstruction.

Fixed obstructions, whether intrathoracic or extrathoracic behave similarly. A moderate reduction in PEFR and MVV is seen. Mid-vital capacity is normal with a plateau of expiratory flow loop and the inspiratory flow. All these can be obtained by simple spirometry.

Endotracheal intubation masked and delayed the diagnosis in cases 1 and 2; both had different etiologies for airway obstruction. Both of these highlight certain observations: (1) variable extrathoracic obstruction is usually due to benign causes such as tracheal or subglottic stenosis, bilateral VC paralysis, or granulation tissue, as highlighted by case 1; (2) variable intrathoracic obstruction, however, is usually due to malignancy, as seen in case 2. We believe that the extrinsic compression by the malignant nodes and infiltration of airways was relieved with each intubation and positive pressure ventilation by splinting and keeping the airway open. Both of these cases also emphasize the usual clinical observation that immediate resolution of symptoms upon securing artificial airway and worsening with extubation should alert one to the possibility of large airway obstruction bypassed by endotracheal tubes. Character of voice can be helpful in defining the nature of the problem. Stridor with normal voice, as in case 1, usually indicates bilateral VC paralysis.¹⁹ Thyroidectomy is the most common cause of bilateral VC paralysis.¹⁹ It may be unrecognized for years. Malignancy of larynx, neck, or mediastinum causing pressure or invasion of the vagus and its recurrent branches usually involves one side first and then both sides.⁹ This or postradiation changes were postulated as the cause in our patient. Often bilateral VC paralysis is unrecognized clinically because voice in adults or cry in infant is quite normal. In bilateral VC paralysis, both cords remain weakly adducted and vibrate passively but equally during expiration. Bilateral VC paralysis is the most common cause of variable extrathoracic obstruction.⁹ Patients with unilateral VC paralysis are not short of breath but have hoarseness of voice due to differences in tension between normal and paralyzed VCs.

Cases 3, 4, and 5 emphasize that not all patients with history of acute asthma or diagnosis of asthma have reactive airways when presenting with shortness of breath. These cases highlight the importance of considering other diagnoses in patients labeled as asthmatics when the clinical course is atypical. High index of suspicion is required to pursue alternative
All these patients carried a diagnosis of asthma and were treated as asthmatics for years. Accurate diagnosis led to radically different treatment strategies in these three patients. Case 3 needed surgical intervention to address the subglottic stenosis. Prevalence of tracheal stenosis after tracheostomy is 10 to 19%. Functionally significant stenosis requiring surgery is seen in about 8% of cases.2,11 This patient probably had stomal stenosis as indicated by the location of the stenotic segment. Stomal stenosis usually occurs with a soft-tissue stenotic segment seen on the lateral wall, as in this case, at or below the cords. Biphasic stridor is usually seen with subglottic or tracheal stenosis. Presence of a stridor signifies severity of obstruction and reduction of airway lumen to 5 mm or less. However, it is not helpful in localizing the site of lesion.2

Case 4 needed tracheostomy, antibiotics, and laser resection of the chronic infection. Rhinoscleroma is a chronic inflammatory granulomatous reaction involving the nose, larynx, and occasionally the trachea and bronchi. The infectious agent, *K. rhinoscleromatis* is a Gram-negative, nonmotile, glucose- and mannitol-fermenting bacillus. It persists for months to years in humans, the only known susceptible host.12,13 Clinical manifestations include nasal obstruction (94%), nasal deformity (32%), hoarseness (12%), and epistaxis (11%).14 Our case represents an unusual presentation since he had an erroneous diagnosis of asthma for a long time.

The treatment consists of a combination of surgery and antimicrobials. Third-generation cephalosporins, quinolones, and macrolides are effective. Surgery is indicated for obstructive lesions of the airways using CO₂ laser.15

The fifth patient was labeled as a steroid-dependent asthmatic for 2 years. The unusual features that triggered suspicion for VC dysfunction include multiple hospital admissions, history of choking sensation in the throat, change in voice with exhaustion, no improvement with steroid therapy, and acuity of onset. The patient had VC dysfunction that improved with time and voice training. Paradoxic VC motion is a syndrome that may be seen by itself or in patients with asthma. In this entity, the VCs, instead of abducting during inspiration, adduct and compromise the inspiratory loop.16-18 In a recent report of 52 patients referred to a national asthma center for difficult-to-treat asthma, 5 (9.6%) had only VC dysfunction while 17 (37.6%) had asthma and VC dysfunction.18 This report alerts one to consider this

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**Figure 11.** Case 5. FVL of patient with VC dysfunction 1½ years before acute episode. Note the normal expiratory limb with minimal inspiratory flattening.

**Figure 12.** Case 5. FVL of patient with VC dysfunction 2 months after resolution of acute episode. Note the improved inspiratory flow loop.
entity in patients with the label of asthma but who have no wheezing on auscultation, monophonic wheezing, or prominent wheezing in the neck. Short-term, these patients respond well to breathing helium-oxygen mixtures. On a long-term basis psychiatric counseling and speech therapy are needed to prevent or abort an attack.16-18

VC dysfunction can be inspiratory or expiratory. FVLs are normal between attacks. Clues to diagnosis include the following: (1) poor reproducibility on spirometry and FVL; (2) resolution of stridor when mouth breathing is switched to nose breathing; and (3) resolution of attack while breathing helium-oxygen mixture, a low-density, high-viscosity gas. Asthma and VC dysfunction are not mutually exclusive diagnoses. Attacks are usually precipitated by irritants. On laryngoscopy, classic appearance is ad-
duction of the anterior two-thirds of VC with a posterior diamond chink visible during inspiration. Occasionally the same can also be seen during expiration. Diagnosis is usually made by laryngoscopy during an attack.2,16-18

In summary, bedside FVLs can be useful in the workup of patients with shortness of breath in the acute care setting.

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