CLINICAL SIGNIFICANCE OF PULMONARY FUNCTION TESTS

Upper Airway Obstruction*

John C. Acres, M.D., and Meir H. Kryger, M.D.†

Chronic upper airway obstruction is frequently unrecognized or misdiagnosed as other conditions such as asthma or chronic airflow obstruction (CAO). Clinical features such as stridor may lead to diagnosis or it may initially be suspected from the results of pulmonary function tests (PFTs) ordered for another reason. We will review the physiology and the pulmonary function tests which are necessary to understand and diagnose upper airway obstruction (UAO) and to differentiate this from lower airflow limitation. The classification of UAO based on the location of the lesion (intrathoracic or extrathoracic) and the nature of the lesion (fixed or variable) will be emphasized.

**Physiology**

The airway may be divided functionally into the following three levels: first, peripheral airways of 2 mm diameter or less; second, larger or major airways from 2 mm diameter up to the main carina; and third, the upper airways which include the trachea, larynx, pharynx, and nose or mouth. The effects of any obstruction of the upper airways will depend on several variables which include (a) the size of the airway at the site of the obstruction, (b) the location of the obstruction, (c) the nature of the lesion, and (d) phase of respiration.

Whether a lesion produces symptoms or not depends on the severity of the obstruction. For instance, it has been shown that a lesion which reduces the diameter of the upper airway to about 8 mm will produce symptoms on exercise. A 5-mm diameter airway at the site of obstruction will produce inspiratory obstruction at rest giving rise to the characteristic physical finding of stridor.

The extrathoracic upper airway is surrounded by atmospheric pressure whereas the intrathoracic upper airway is surrounded by pleural pressure. The difference between the intratracheal pressure and the external pressure is the transmural pressure. If the external pressure exceeds the intratracheal pressure, i.e., a positive transmural pressure, the airway tends to collapse. A negative transmural pressure tends to open the airway. Figure 1 diagrammatically illustrates the locations and pressures involved.

The nature of the obstruction, i.e., whether it is stiff or pliable, determines whether there will be changes in severity in relation to changes in transmural pressure. In this context, a stiff lesion causes a fixed obstruction and a pliable lesion leads to a variable obstruction.

The effect of a fixed UAO, that is, one that does not change in severity with the phase of respiration, will not be affected by the level of the obstruction. A fixed extrathoracic UAO will cause the same effects as a fixed intrathoracic obstruction.

In the case of a variable extrathoracic UAO such as that caused by vocal cord paralysis, the obstruction is increased during inspiration. The pressure inside the trachea falls on forced inspiration resulting in a greater transmural pressure at the site of the lesion worsening the obstruction. On expiration, intratracheal pressure rises which reverses transmural pressure tending to lessen the degree of obstruction and improve flow (Fig 2).

With variable intrathoracic UAO, the rise in

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*From the Department of Respiratory Medicine, St. Boniface General Hospital, Winnipeg, Manitoba.
†Assistant Professor.
Reprint requests: Dr. Kryger, Respiratory Medicine, St. Boniface Hospital, Winnipeg, Manitoba, Canada R2H 2A6

CHEST, 80: 2, AUGUST, 1981

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Figure 1. Pressures affecting caliber of upper airway.
VARIABLE EXTRATHORACIC OBSTRUCTION

pleural pressure on forced expiration may exceed the rise in intratracheal pressure resulting in a reduction in the size of the airway at the site of the lesion. The pleural pressure becomes markedly negative during forced inspiration tending to lessen the obstruction by reversing the transmural pressure resulting in improved flow (Fig 3).

LABORATORY FEATURES

In normal subjects during a forced expiration from total lung capacity (TLC), the maximum airflow is achieved during the first 25 percent of the vital capacity (VC) and is directly dependent on effort and inversely on resistance. In the remaining 75 percent of the VC, flow is limited in such a way that an increase in effort with its associated increase of pleural pressure does not result in increased flow. The precise mechanism of flow limitation over the lower 75 percent of the VC is the subject of intense current investigation and is not yet settled but involves compression of intrathoracic airways. For the purposes of this presentation, the fact that flow is limited over the lower 75 percent of the VC is the important point to remember.

With an upper airway obstruction, flow at higher lung volumes may be limited by the obstruction. At lower lung volumes, flow may be limited not by the upper airway obstruction but by the collapse of intrathoracic airways as noted above. Generally, flow measurements made at low lung volumes reflect the function of peripheral airways only, where-

Figure 2. Pressures affecting extrathoracic UAO. Ptr indicates intratracheal pressure; Patm, atmospheric pressure. Obstruction increases during forced inspiration. (Adapted from Kryger et al.9)

Figure 3. Pressures affecting intrathoracic UAO. Ptr indicates intratracheal pressure; Ppl, pleural pressure. Obstruction increases during forced expiration. (Adapted from Kryger et al.9)
as those made at high lung volumes reflect both upper and lower airway function. It thus follows that some tests that measure events that occur below 75 percent of the vital capacity such as flows at 50 percent and 25 percent of the vital capacity may not be sensitive to UAO.

**Flow Volume Loop**

Miller and Hyatt suggested that the flow volume plot of a forced expiratory and inspiratory vital capacity maneuver would be particularly helpful in categorizing UAO. They simulated major airway obstruction by having normal subjects breathe through fixed resistances and found that flow rate increased to a certain level early in both inspiration and expiration and then plateaued. The plateau was reached at lower flow rates as the resistances were progressively increased (Fig 4).

The effect of a fixed lesion, either intrathoracic or extrathoracic, will be apparent during both inspiration and expiration. With a fixed UAO, the plateau and limitation of flow is seen both in the expiratory and inspiratory flow volume loops. In the case of a variable extrathoracic lesion, the alteration in the flow volume loop is seen by flow limitation and a plateau on inspiration. The reverse situation applies with a variable intrathoracic lesion (Fig 5).

**Spirometry**

A simple plot of a FVC maneuver of volume vs time, the timed spirogram, will also give some clues to the presence of upper airway obstruction. Figure 6 shows the spirogram and flow volume loop from a patient with an intrathoracic variable obstruction (a paratracheal tumor). The linear portion of the spirogram indicates constant flow which is equivalent to the plateau (ie, constant flow) of the flow volume loop. For comparison, a spirogram from a normal subject is shown on the right.

Rotman et al compared various tests in patients with upper airway obstruction and chronic obstructive pulmonary disease and normal subjects. From this study, they identified four variables which usually distinguish patients with upper airway obstruction from patients with CAO. The values are obtained from the flow volume plot and from the spirogram.

1. FIF50% ≤ 100 L/min.
2. FEF50%/FIF 50% ≥ 1
3. FEV1/PEFR ≥ 10 ml/L/min
   (The PEFR is proportionately more reduced by the UAO compared to the FEV1).
4. FEV1/FEV0.5 ≥ 1.5
   (The FEV0.5 is proportionately more reduced by obstruction of the upper airway than FEV1); this ratio can be obtained from the spirogram tracing alone.

The abbreviations used above are as follows: FIF50%, forced inspiratory flow at 50 percent of the

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**Figure 4.** Flow volume plot obtained from normal subjects breathing without external resistance and with 6 and 4 mm diameter external resistance. (Adapted from Miller and Hyatt.)

**Figure 5.** Flow volume plot in case of variable intrathoracic and variable extrathoracic obstruction. (Adapted from Kryger et al. and McCarthy.)

**Figure 6.** Spirogram and flow volume loop.
vital capacity; FEFSO%, forced expiratory flow at 50 percent of the vital capacity; PEFR, peak expiratory flow rate measured in litres per minute; FEV₁, forced expiratory volume in one second measured in milliliters; and FEV₀.₅, forced expiratory volume in ½ second measured in milliliters.

These values are readily available from most pulmonary function labs and may be included in a computer printout in those with automated reporting. The inclusion of one or more of these indices in routine reporting should lead to unsuspected cases of upper airway obstruction being discovered as well as their use in patients already suspected of having such problems.

**Mixed Upper and Lower Airway Obstruction**

The combination of CAO and UAO may arise in several circumstances, for instance, tracheal stenosis occurring after tracheal intubation for an episode of respiratory failure in a patient with chronic airflow obstruction (CAO). Using principles outlined below, the presence of UAO in patients with lower airflow limitation may be distinguished.

In the upper airways, flow is turbulent and depends on the density of the gas, whereas in peripheral airways, flow is laminar and independent of gas density. Breathing a mixture of helium and oxygen (He-O₂) increases flow in density-dependent regions even more so when turbulence is magnified by obstruction. In peripheral airways, laminar flow is not increased when breathing He-O₂. Lavelle and co-workers, using this rationale, studied patients with CAO who breathed through fixed external resistances. They showed in patients with CAO that the presence of upper airway obstruction could be established by demonstrating an increase in the flow increment at higher volumes while the subjects breathed He-O₂; at peak flow, 75 percent and 50 percent of VC, He-O₂ increased flow by 50 percent. In those with CAO not breathing through fixed resistance, there was little change in flow when breathing He-O₂. Recently Lisboa et al., using He-O₂ and more complicated methodology, showed that extrathoracic upper airway obstruction may be important in asthma.

**Distribution of Ventilation and Bronchodilator Response**

In the presence of UAO without peripheral airway obstruction, tests of distribution of ventilation such as the nitrogen washout are normal. A marked elevation in airway resistance unresponsive to bronchodilators is also often seen in patients with UAO. The combination of a normal nitrogen washout and elevated airway resistance in a patient complaining of wheezing or dyspnea should lead one immediately to suspect upper airway obstruction as the cause.

**Other Laboratory Features**

Resistance in any tube can be measured by dividing the pressure drop along the tube by the flow. In man, resistance across the upper airway is about 1 cm H₂O/L/sec, whereas most patients with symptomatic UAO are found to have a resistance between 4 and 15 cm H₂O/L/sec while breathing quietly. The resistance of the upper airway is not constant and with increasing flow (such as during exercise), there is a disproportionate rise in the resistance, probably explaining the dyspnea on exertion of these patients. During the maximum volun-
tary ventilation (MVV) maneuver, patients with UAO meet with increasing airway resistance as the respiratory rate increases. This causes a reduction in MVV that is much greater than one would suspect from the FEV₁ in the same subject.

We have reviewed the laboratory and physiologic features of upper airway obstruction. Since most lesions which cause UAO require surgical therapy, an anatomic and pathologic diagnosis is required following identification of the physiologic abnormality. This is achieved with radiologic and endoscopic techniques. In cases of severe UAO, provision for emergency treatment such as tracheostomy should be immediately available before advancing to any further investigations, such as bronchoscopy, once a physiologic diagnosis has been established.

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