The noninvasive diagnosis of impaired mechanical integrity or compliance of the trachea is most accurately made by fluoroscopic observation recorded on video tape or cineradiography, with or without benefit of artificial contrast media (contrast tracheography). In particular, localized buckling, collapse or dilatation indicative of focal tracheomalacia may thus be diagnosed in vivo and correlated with diseases of the central airways. Congenital tracheomalacia is a rare, but well described entity. Cases of acquired tracheomalacia occur with increasing frequency, but are often not clearly recognized. We contrast the dynamic behavior of the normal trachea with the abnormal dynamics characteristic of focal tracheomalacia. Such lesions may result from trauma, surgical procedures, chronic irritation, inflammation, mechanical changes, or malignancy.

By virtue of its normal flexibility or compliance, the trachea changes caliber during the respiratory cycle, i.e., inspiratory dilatation and lengthening and expiratory narrowing and shortening (Fig 1).

When this normal cyclic process is accentuated, some excessive changes in caliber may be seen, deforming either the entire trachea or a localized segment. The quantitative aspects of respiratory variations of the trachea in health and disease have been previously described, and a decrease in caliber of the thoracic segment of the trachea by 50 percent during brisk cough is usually considered at the upper limits of normal. One sees, on occasion, an even greater decrease in caliber of the trachea in individuals without known respiratory disease. In general, however, abnormal collapsibility denotes a loss of structural rigidity, i.e., softening, better expressed as abnormally increased compliance. Con-

**Figure 1. Bronchographic spot films showing variations of caliber of the normal trachea during inspiration (left) and expiration (right).**
acquired tracheomalacia

Genital flaccidity of the trachea is known as tracheomegaly or Mounier-Kuhn syndrome, but only rarely has acquired softening or tracheomalacia been described in the adult other than as a complication of prolonged tracheostomy.

Because the trachea is normally in motion during the respiratory cycle, the nature and severity of changes in caliber are best recognized during fluoroscopy and on fluorographic recordings and are usually not evident on conventional static roentgenograms. Observation of tracheal motility during respiration and coughing is, therefore, essential to the accurate analysis of its defects. Deformities in excess or, out of proportion to, variations of intrathoracic pressure or to the configuration of extrinsic masses or organs, indicate a high probability of organic disease of the tracheal wall. However, the mere presence of excessive cyclic changes of the tracheal contour does not necessarily indicate that the trachea is damaged; it may merely reflect abnormal extrinsic factors. The diagnosis thus depends on the correct interpretation of the anatomic and functional abnormalities, preferably based on recorded fluoroscopy. The present communication is specifically concerned with identification of such lesions, differentiation of deformities of the normal trachea caused by extrinsic mechanisms from true intrinsic tracheal disease, and consideration of various etiologic mechanisms.

**Response of the Normal Trachea to Extrinsic Pressure**

The trachea of patients with bronchitis and mild to moderate obstructive emphysema and no other evidence of intrinsic tracheal disease is apt to collapse considerably during severe coughing, on rare occasions even to the point of near obliteration of its lumen. This is commonly recognized in clinical practice. In such cases, its contour is uniformly affected, and is thought to reflect merely the response of a normally compliant trachea to excessive-ly high and sudden peaks of positive intrathoracic pressure. Fluorographic observation supports this interpretation.

Contiguous masses or organs (such as an ectatic aorta) may deform the trachea. A simple smooth deformity of tracheal contour in such a case does not, per se, imply intrinsic disease. This is illustrated by a large intrathoracic goiter which compresses an apparently normal trachea (Fig 2). Extrinsic deformities due to vascular anomalies, well known in the pediatric age group, may or may not be associated with organic defects of the tracheal wall.

**Pathogenesis and Pathophysiology of Acquired Tracheomalacia**

Any disease process which affects the integrity of the tracheal wall is apt to cause a localized change in the compliance of the trachea, causing increase or decrease of collapsibility of the involved segment in relation to neighboring intact areas, and disrupting the uniformity of caliber change of the trachea. Such lesions may vary in length, in cross-section, in adherence to surrounding structures, and in visible variations of thickness of the tracheal wall. The most severe lesions may cause fixed organic stenosis, while the milder lesions may merely cause transient stenosis due to focal kinking or buckling of the trachea during forced expiration or coughing. Al-
though the anatomic defect may appear trivial, or may even entirely escape detection by conventional examination methods, the functional interference with ventilation may be clinically significant by virtue of obstruction to expiratory airflow and interference with clearance of secretions. The degree of impairment of function is proportional to the length of involved segment and to the degree of stenosis. Furthermore, kinking may occur as easily at the transition between normal tracheal wall and an indurated segment, as well as in a malacic segment.

Although the majority of cases of acquired tracheomalacia have an organic stenosis aggravated by invagination during expiration or cough, extreme cases of more diffuse tracheal disease or extensive peritracheal adhesions may cause excessive widening of the tracheal lumen with marked irregularity of its outline.16 The trachea usually distends out of proportion and unevenly during inspiration, and collapses irregularly during expiration.

The physiologic response of the trachea to changes of internal and external pressure is illustrated diagramatically in Figure 3, during normal respiration, coughing, focal tracheomalacia (right side of diagram), and severe extensive tracheomalacia (tracheomegaly) (middle portion of diagram). The latter condition, incidentally, may be indistinguishable from tracheal distortion caused by adhesions in severe pulmonary fibrosis. Of clinical significance is the fact that, in both situations (abnormally dilated or irregularly kinked or buckled trachea), there is marked interference with the functions of the trachea.

Etiology of Acquired Tracheomalacia

Tracheostomy. By far the most frequent cause of acquired tracheomalacia is the indwelling tracheostomy or endotracheal tube with inflatable cuff.17-19

![Diagram of tracheal forces and calipers](image)
Although more cases reported in the literature occurred at the tracheostomy stoma, we have seen a greater number at the site of the inflatable cuff, 1.5 or more cm below the stoma. Occasionally, one encounters an additional stenosis at the point of impingement of the tip of the tracheostomy tube. Some cases exhibit multiple such lesions. Possible pathogenic factors are pressure necrosis, impairment of blood supply, infection and cyclic friction upon the dry tracheal mucosa. The severe stricture in Figure 4 is a post-repair recurrence of a stenosing granuloma of the trachea after prolonged indwelling tracheostomy with inflatable cuff during complicated recovery from heart valve replacement. Because of these complications, inflatable cuffs are now used with great caution if at all.

Chest Trauma. Trauma to the trachea may complicate crushing chest injuries, for instance in steering wheel injuries. In this case, the fracture of the trachea was not initially recognized, but resulted in symptomatic focal tracheomalacia subsequently.

Chronic Irritation. Chronic irritation, especially in smokers, causes frequent violent cough which may weaken the tracheal wall by excessive repetitive pressure changes, superimposed on the lesions of chronic tracheobronchitis.

Inflammation. All sections of the trachea, cervical or thoracic, may reflect damage by inflammatory processes. The tomogram in Figure 5 illustrates the end result of chronic recurrent infections incurred during repeated tracheostomies. An extreme example occurred in a case of relapsing polychondritis (Fig 6) marked by progressive destruction of the tracheal cartilages, resulting in irregular dilatations of the trachea. This patient eventually succumbed to...
his disease.20

Mechanical Anatomic Factors. Pulmonary resections, especially of the upper lobes, notoriously produce stenosing tracheal deformities (Fig 7), often also compounded by infection and adhesions in the peritracheal spaces.

Malignancy. Neoplasms of the trachea, mainly cylindromas, initially reduce the tracheal lumen, and may later invade and destroy portions of the tracheal wall, including cartilage, causing tracheomalacia. The expiratory buckling of the softened tracheal wall may even simulate pedunculation during fluoroscopic observation (authors' unpublished cases).

Conclusion and Summary

Disturbances of ventilation may result from impairment of the normal compliance of the trachea. This may be manifest either as an organic stricture, or as buckling or kinking during expiration and coughing, or a combination of these mechanisms. Because the changes in tracheal caliber and contour during the respiratory cycle are rarely evident on stationary roentgenograms, lesions which affect tracheal function may only be correctly appreciated during fluoroscopy or preferably on fluorographic recordings. Accurate recognition of these lesions and assessment of the anatomic and functional defects are necessary for understanding the disease processes and for correct clinical management. The normal variations of tracheal caliber, the response of the normal trachea to extrinsic pressure, and the pathologic physiology of acquired tracheomalacia were discussed. The etiology of acquired tracheomalacia in the adult may include tracheostomy, closed chest trauma, chronic irritation, inflammation, structural and mechanical changes, and malignancy.

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