CHRONIC COUGH IN CHILDREN

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CONTENTS

Cough is perhaps the most common symptom of acute and chronic respiratory diseases in children. Because of its audible nature it attracts considerable attention. An extravagant amount of time and money is spent on treating this symptom, one of the most important vital protective mechanisms of the body (Kaye et al, 1982). Therefore recurrent or chronic cough constitutes one of the most frustrating diagnostic and, therapeutic problems in practice. Frequently these patients are depressed or anxious, sometimes asthmatic patients with severe cough seek medical aid after an episode of syncope or a convulsion (Katz, 1980). Irwin et al 1981 defined a chronic persistent cough as one that (1) eluded the diagnostic and therapeutic maneuvers of at least one referring physician (2) remained persistently troublesome to the patient for at least 3 weeks.

PATHOPHYSIOLOGY OF COUGH

THE COUGH REFLEX

Each cough involves a complex reflex arc that begins with irritation of a receptor. Cough receptors are located in upper, mid, and lower peripheral airways of cats and dogs, and presumably, also in man. Those in the upper airways are located in the paranasal sinuses, ear canals, tympanic membranes, nasal airways and pharynx. In the mid airways, cough receptors are located in larynx and trachea, in the lower airways they are concentrated in the carina, bronchial bifurcations, and to a lesser extent in the peripheral airways (Widdicombe et al, 1962). Cough receptors also can be found in the pleura, diaphragm, and non pulmonary receptors sites located in the pericardium and stomach (Nodel, 1973). Investigations in animals and man indicate that there are three types of receptors in the lungs and airways :

(1) The pulmonary stretch receptors that are concerned with the depth of breathing,

(2) type j receptors that lie within the alveolar walls and are stimulated by interstitial edema, and

(3) rapidly adapting receptors that lie within the epithelium of the tracheobronchial tree and are responsible for both cough and bronchoconstriction (Bickerman, 1982).

These receptors are sensitive to mechanical stimulation from touch or foreign substances, to irritation from inflammation, to pressure from tumours or glands either within or without the bronchial tree, and to chemical irritation from noxious gases (Williams et al, 1975).

The larynx and carina and the regions of bifurcation of the major airways are areas most sensitive to mechanical stimulation (Larsel and G.E., 1024). Peripheral bronchi are remarkably insensitive to mechanical provocation, and it is a common clinical observation, that whereas even a small mechanical irritation in the major air ways calls forth a most vigorous cough, foreign bodies, or other mechanical irritants, may lodge in the peripheral bronchi may elicit no response (Jackson, 1922). Conversely, the distal airways are sensitive to chemical stimulation.

Stimulation of cough receptors creates nerve impulses that travel through the afferent pathways to the medullary cough centre, which in turn stimulates efferent nerve pathways (the vagus, phrenic, and possibly the spinal motor), (Katz, 1980).

Sympathetic nerves innervate the larynx, tracheobronchial tree, heart, and gastrointestinal tract (Dahlstrom et al., 1966). They apparently act to oppose the action of the parasympathetic nervous system. In dogs, and in man, alpha adrenergic constrictor pathways may not be important in air ways (Cabezas et al, 1971). An alternate but not widely accepted concept of cough postulates a direct reflex arc from the mucosa to the bronchial smooth muscles. According to this view, irritation of mucosal receptors initiates bronchospasm, which in turn activates cough receptors in the lungs (Nadel et al, 1962).

Mechanics of an Effective Cough :

To cough, one inspires fully then contracts the muscles of expiration against a closed glottis thus producing a marked increase in intrapleural and intra pulmonary pressures.

Ross et al, 1955), as high as 300 mmHg have been recorded in man (Sharpey, 1953). The glottis is then opened abruptly, with explosive release of air under high pressure - the bechic blast. In the trachea, the velocity of the expired air approaches the speed of sound. Because the cross sectional area of the adult trachea averages 2.5 cm^2, whereas the total cross sectional areas of all the 18th generation bronchioles totals 534 cm^2 (Weibel, 1964), the anti-stream is approximately 200 times faster in the trachea than in the periphery. One may predict therefore, that cough will be most effective in the larger, central airways. Antistream velocity is critical not only because of the force or lifting power produced, but also becuase of turbulence. The faster the movement of air, the greater the tendency to turbulent rather than laminar flow, and turbulent flow is more effective in shearing debris from the airway walls. In addition, expiratory narrowing of the major airways during cough increases the velocity of flow through these tubes. The narrowing results from pressure outside the airways exceeding pressure within the airway. Airway pressure during expiration is maximal in the alveoli and alveolar ducts (pleural pressure

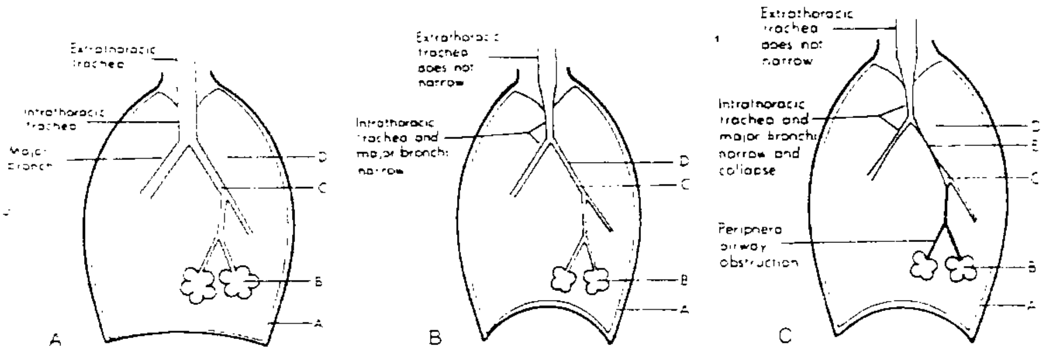

FIG. 10-5 A Airway dynamics: normal expiration. At end of quiet, passive expiration intrapleural pressure (A) is negative relative to atmospheric in range of -3 cm H_2O. Net intra-alveolar pressure (B) is positive relative to atmospheric ($+2$ cm H_2O) because the positive pressure generated by lung elasticity exceeds negative intrapleural pressure. Pressure loss across peripheral airways is small, and pressure within lumen of large airway (C) is therefore close to alveolar pressure (i.e. $+1$ to $+2$ cm H_2O), and exceeds intrapleural or intrapulmonary pressure (D). Transmural pressure gradient (D minus C) across large airway is therefore negative and there is no narrowing of lumen.

B, Airway dynamics: normal cough. During cough or forced expiration intrapleural pressure (A) is positive in range of $+100$ cm H_2O. Intra-alveolar pressure (B) is also positive ($+105$ cm H_2O). At high velocities of cough there is appreciable pressure drop across normal peripheral airways, and intraluminal pressure in large airway (C) is perhaps in range of 70 to 80 cm H_2O. This is less than intrapleural or intrapulmonary pressure (D), and transmural pressure gradient (D minus C) is positive in range of 20 to 30 cm of H_2O, compressing bronchus and narrowing lumen.

C, Airway dynamics: cough with peripheral airway obstruction. During cough in presence of peripheral airway obstruction pressure drop across distal airways is greatly exaggerated. Consequently intraluminal pressure in large airway (C) is quite low, and transmural pressure gradient (D minus C) is excessive, leading to total collapse of bronchus (E). Transmural pressure gradients of over 50 to 60 cm H_2O are probably sufficient to cause complete airway collapse. If patient coughs harder he increases his alveolar and airway pressures, but also increases pressures surrounding airways (D) an equal amount, and therefore cannot prevent bronchial closure.

(Fig. I)

plus elastic recoil) and decreases to a minimum (atomspheric) at the mouth (Fig. I). This progressive fall in intraluminal pressure from alveolus to mouth is a consequence of pressure erosion by friction between the anti stream velocity and the wall of the airway. It is a necessary requisite to air flow, since gas moves only from a region of higher pressure to one of lower pressure. The transmural pressure gradient, therefore is minimal peripherally and increases as one proceeds centrally, reaching a maximum in the main stem bronchi and trachea. Expiratory narrowing accordingly is maximal in these areas (Lorin, 1975) According to Comroe 1974, flow increases seven - fold during cough, whereas the cross sectional of intrathoracic trachea decreases to one sixth of normal, the result is forty-two fold increase in linear velocity. It has been calculated that cough is not effective beyond the sixth or seventh generation of airways (Leith, 1968).

Bronchial Squeeze :

In 1949, Dirienzo suggested that cough empties the tracheo bronchial tree by an active, peristaltic wave sweeping from bronchi to trachea. It is now apparent that cough does function by intrathoracic pressure change and that bronchial peristalsis does not occur (Huzinga 1962, Ross et al 1955, Scarpelli et al 1965). Although it is clear that bronchial peristalsis does not occur, this does not rule out the possibility of a tussive squeeze as suggested by Jackson and others (Huzinga, 1967). As the distal airways close down at low lung volume they may squeeze secretions forward, much as one squeeze tooth paste from a tube. A process such as this could slowly milk intraluminal contents to larger bronchi where it then could be handled by cough.

Mechanics of an Inadequate Cough :

All pathological conditions that lead to an in effective cough interfere with either the inspiratory or expiratory phases of cough. Patients with a variety of extrapulmonary, skeletal, and neuromuscular disorders may not cough effectively when their inspiratory and/or expiratory effects are limited due to pain, weakness or C.N.S. depression. Also cough may be in effective in pulmonary diseases characterized by reduced expiratory rates. These might include an extrinsic compression mass, endobronchial lesion, bronchial stricture, foreignbody, small tracheostomy tube, or tenacious secretions in patients with mucoviscidosis, bronchospasm or secretions in asthmatics (Irwin et al, 1977).

Complications of Cough :

These complications are :-

Musculoskeletal :

Ranges from asymptomatic elevation of serum creatinine phosphakinase to rupture of rectus abdominis muscles (Irwin et al, 1977). Rib fractures along the lateral margin of the rib cage rarely occurs in children with chronic cough (Katz, 1980).

Pulmonary :

Pneumomediastinum, pneumothorax, and subcutaneous emphysema, examples of severe complications (Katz, 1980).

Cardiovascular : These may be :

- Loss of consciousness