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EDITORIAL

Upper airway obstruction: the culprits are the arytenoids

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The rapid accumulation of data on sleep apnoea syndrome recently drew the attention of investigators to the upper extrathoracic airways (UEA). These airways include the nasal and buccal airway, the pharynx (divided into naso-, oro- and hypopharynx), the larynx, and the extrathoracic trachea. These structures conduct air from the atmosphere to the alveoli. Their peculiar shape accounts for their capacity to modify the temperature and humidity of inspired air. Their involvement in the defence mechanism of the respiratory tract is well-known. UEA also serve for the initial part of digestion: biting, chewing and swallowing of food. The co-existence, at this level, of air, liquids and solids may explain the complexity of the physiological mechanisms involved.

The trachea possesses a rigid cartilagineous structure, which maintains the airway open. By contrast, the pharynx has a muscular structure. Its calibre, therefore, depends on the tone of the pharyngeal muscles. This is probably the weakest part in the chain, but not the only one. Indeed, the larynx shares with the trachea a cartilagineous supporting structure, but also has rapidly moving parts, the vocal cords, which can narrow or close the glottic orifice. To get air from the atmosphere to the alveoli a negative pressure must be created inside the alveoli and, therefore, inside the respiratory tract. A negative canalicular pressure, *i.e.* a negative transmural pressure, may collapse a compliant airway, such as the pharynx.

A fine neuromuscular mechanism maintains this airway open. Impairment of the mechanism during sleep might be responsible for the sleep apnoea syndrome. During wakefulness, narrowing or closure of the UEA can occur at different levels. The aetiology might be either functional or structural. Recently, several authors have reported acute narrowing, or closure, of the upper airways (especially the glottis) of psychological origin, occurring in both children and adults, during either rest or exercise [1–11]. Narrowing of the glottic orifice, but also of the pharynx, has also been described following topical anaesthesia of upper airways, resulting in stridor and decrease of inspiratory flow [12, 13]. These findings reflect an impairment of the reflex regulation of upper airways calibre. Upper airway obstruction during sleep has previously been reported during negative pressure ventilation with an "iron lung" [14]. Recently, it has been shown that, during these conditions, obstruction can occur either at the glottic or supraglottic level, and results from an uncoupling of upper airway muscles and diaphragm activity [15]. Indeed, the inspiratory activity of upper airway dilatory muscles precedes the activation of the diaphragm, stiffening the upper airways before the onset of airflow [16–18].

In this issue of the Journal, NAGAI and co-workers [19] present the case report of a woman with exercise dyspnoea and inspiratory stridor. She had a decrease of the inspiratory flow with rapid flow oscillations. Fibreoptic bronchoscopy showed a thickened and deformed right-sided arytenoid. During forced inspiratory the arytenoid narrowed the laryngeal orifice. In the absence of other findings, the authors attributed exercise dyspnoea to the upper airway lesions. In another paper recently published in Thorax, NAGAI and co-workers [20] presented the case report of another woman with "severe dyspnoea" "after coughing" and "marked stridor on forced inspiratory effort". There was "a clear cut decrease of the inspiratory flow, but a more normal expiratory flow". Fibreoptic bronchoscopy showed that during forced inspiratory efforts the arytenoid region moved to obstruct the laryngeal orifice. This did not occur during quiet breathing. The patient was treated with diazepam, and one week later her complaints disappeared. There was no more stridor or abnormal movements of the arytenoid region. The interest of these case reports lies in the localization of the upper airway obstruction to the arytenoid region, to my knowledge not reported until now. Decrease of the inspiratory flow pointed to the upper airways as the probable cause of obstruction. Fibreoptic bronchoscopy permitted visualization of the site of the obstruction. A more precise and elegant approach would had been to simultaneously record airflow and pressure at different levels of the upper airways. In one case, the authors suspected inflammation as the cause of the arytenoid lesion, in the second they attributed obstruction to a psychological origin.

The reader might feel somewhat frustrated at the need to search in two different European journals to find information on two related case reports. Another case report was recently published by NAGAI and co-workers [21] in Chest. Does multiplication of papers in different journals serve research better than an article discussing all of the cases together? Or does it just serve to increase the number of publications of the authors?

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