EDITORIAL

THE ANAESTHETIST AND RESPIRATORY DYSFUNCTION

The respiratory disorders encountered in anaesthesia and intensive care continue to threaten the lives of patients and, in some cases, the longevity of the anaesthetists who struggle with these problems. Judged by the presentations at the Anaesthetic Research Society in recent years, however, there appears to be a paradoxical waning of interest in research into pulmonary dysfunction despite the ubiquity of these clinical problems. Has this occurred because the investigative tools are costly and complex and because the problems have become esoteric and obscure? Some of the tools are certainly complicated, but the problems themselves are clearly defined and in many cases are easily investigated using diagnostic equipment found in most general hospitals. The respiratory problems seen by anaesthetists include impaired function not only of the airway but also of the chest wall and lung parenchyma as well as abnormal regulation of breathing, and this issue reviews recent research into some of these problems. To avoid having to make critical comments both on the work of my North American colleagues and on my research group at Northwick Park, I will instead outline some of these problems from the standpoint of the clinical investigator and suggest how current research can be used to improve patient care.

A common and alarming problem during anaesthesia is loss of patency of either the intrapulmonary or the upper airway, but there are few studies in which these changes are quantified. The latest development in measuring intrapulmonary airway resistance ($R_{AW}$) in anaesthetized patients (Jordan et al., 1981) is technically complex but is quite simple in basic principle. It eliminates the effect on airway calibre of unpredictable change in lung volume by the simple manoeuvre of deliberately changing lung volume and measuring $R_{AW}$ continuously as volume changes. This method can be used to answer the question: "Are the infrequent but severe changes in $R_{AW}$ seen during anaesthesia only the tip of an iceberg and do large changes in $R_{AW}$ occur which are presently unrecognized?" With this question answered, the method provides the means to unravel the causes as well as quantifying the magnitude of change in $R_{AW}$ that complicates anaesthesia.

Another fascinating problem is to determine the factors which regulate the patency of the upper airway. Credit for launching a new attack on the pathophysiology of upper airway obstruction must go to physicians interested in the rare problem of obstructive sleep apnoea. Guilleminault and Dement (1978) have edited a superb symposium on sleep apnoea in which is described the use of fibreoptic endoscopy and electromyography of upper airway muscles to elucidate mechanisms of upper airway obstruction. Another technique to examine the upper airway is to produce a plot of upper airway cross-section as a function of the distance from the lips to the trachea by a non-invasive technique using high frequency acoustic reflections measured at the mouth (Fredberg et al., 1980). It has been shown that sleep causes a reduction in tone of upper airway musculature and not only the tongue but the muscular pharyngeal tube also may collapse inwards to occlude the airway. These changes are probably analogous to the changes in the upper airway during anaesthesia. An intriguing idea is that because a loss of patency of the upper airway is a result of reduced electrical activity in the muscles of this region, patency may be restored by external electrical stimulation of these muscles! That this problem is...
Central and obstructive apnoeas in these patients were very common when i.v. opiate was used to relieve pain, but also occurred without opiate in patients given regional analgesia after operation. Apnoeic periods occurred almost entirely during sleep and were associated with oxygen desaturation. When patients with obstructive airway disease fall asleep they may develop severe hypoxia associated with obstructive apnoea and this is associated with marked electrocardiographic changes (Tirlapur and Mir, 1982). In order to explain mysterious deaths following surgery, it is tempting to extrapolate these data to postoperative patients, although none of our patients had arterial oxygen saturations of less than 72%. It does emphasize, however, the need to reconsider the type of postoperative monitoring devices that would be acceptable if a higher standard of patient safety is to be achieved.

Not only the upper airway muscles but also the intercostals show marked impairment of function during sleep and anaesthesia. This loss of rib cage function may explain not only the impaired ventilatory response to carbon dioxide during sleep and anaesthesia (Jones, 1977; Cherniack, 1981), but also the loss of compensation to loaded breathing during anaesthesia. There is an extraordinary parallel between the effects of anaesthesia and, in particular, rapid eye movement sleep, in impairing rib cage and upper airway muscle function whilst sparing diaphragm function. This may be related to the remarkable lack of spindle control of the diaphragm compared with the rich spindle innervation of other muscles. The problem of fatigue of respiratory muscles during loaded breathing on conscious subjects has recently been outlined by Rochester (1981), but it is still not clear if diaphragmatic fatigue is more important than fatigue of non-diaphragmatic respiratory muscle. It is noteworthy, however, that anaesthetized and sleeping subjects who have impaired function of intercostal and upper airway muscle have very poor compensation to partial or complete obstruction of the airway.

A quite different clinical problem is posed by damage to the blood–gas interface in the lung which may result in the adult respiratory distress syndrome (ARDS). This can be considered in terms of (a) the vast number of different damaging agents, and (b) the early recognition of change in function of the interface consequent upon damage.

Reviewed in this issue are the deleterious effects of oxygen which may be manifest either as high altitude pulmonary oedema or high oxygen lung damage. It is not known whether the latter occurs by a direct effect of molecular oxygen or is mediated from the production within cells of the highly reactive free radicals such as superoxide anion, hydroxyl radical or singlet oxygen. It has also been proposed that the effects of increased concentrations of oxygen may be mediated by the release of these free radicals from neutrophils stimulated by increased oxygen to marginate to the walls of capillaries. There is certainly tremendous current interest in the release and cell-damaging effects of these free radicals produced by neutrophils (Weissmann, Smolen and Koichak, 1980) whether mobilized by oxygen, infections or immunologically-mediated processes (Jacob et al., 1980).

The new techniques described for measuring alveolar epithelial permeability described in this issue provide a number of methods for monitoring patients who may be susceptible to diffuse interstitial lung damage and ARDS. These techniques also provide the means for confirming the suggestions of Jacob and colleagues (1980) that complement activation with an increase in C5a may be an important mediator of diffuse lung damage in man. Patients can now be identified who have a considerable increase in permeability of the alveolar–capillary interface to hydrophilic solutes. If care is taken in these patients to minimize the hydrostatic pressure in the pulmonary capillaries then pulmonary oedema may be avoided. Furthermore, a diagnosis of increased permeability made at an early stage of the disease provides an opportunity to identify and prevent the effects of factors which increase lung permeability.

It is hoped that the application of some of these ideas may enable dramatic advances to be made in patient care and that the morbidity and mortality as a result of respiratory dysfunction in patients seen by the anaesthetists will become a distant memory.

J. Gareth Jones

REFERENCES


