Respiratory disease is a common clinical problem accounting for 25% of the workload of an average general practitioner, according to a recent estimate (Marsh and McNay, 1974). This pattern is reflected in patients presenting for anaesthesia and surgery. Diament and Palmer (1967) found spirometric evidence of significant obstructive lung disease in 35% of an unselected group of patients presenting for elective surgery. Such pre-existing respiratory disease is a potent source of complications during anaesthesia and in the period following operation. Stein and his colleagues (1962) found postoperative pulmonary complications in only 3% of patients who were normal preoperatively, but in 70% of patients with preoperative respiratory disease. Despite major advances in surgery, anaesthesia, and medicine, it appears that the overall incidence of postoperative pulmonary complications has not changed significantly in the past three decades (Wightman, 1968). It has been pointed out (Holaday, 1967) that there is an important disparity between "what is known about respiratory failure and its management and what is being done about it". At the preoperative visit, the anaesthetist should elucidate the nature of existing respiratory disorder so that a specific aetiological or pathological diagnosis can be made. Functional diagnoses such as "obstructive lung disease", while of some value in planning therapy, should not be seen as a substitute.

Many clinicians may consider prophylactic measures to be of little value in the presence of severe organic lung disease, which is essentially irreversible. That this is not the case is well illustrated by the data of Swenson, Ställberg-Stenhagen and Beck (1961). These authors identified, among patients coming for thoracic surgery, a high risk group with moderate to severe respiratory dysfunction. This group was managed with vigorous prophylactic measures to prevent respiratory complications, and it was found at the end of the study that there were fewer pulmonary complications than in a more normal group which, while showing only minor preoperative abnormalities on respiratory tests, had not been exposed to the same vigorous prophylactic measures.

The mechanisms whereby pre-existing respiratory disease may complicate anaesthesia and surgery can be divided into two categories. Firstly, respiratory disease may complicate the conduct of anaesthesia and this in itself may produce complications; secondly, respiratory disease may combine with the effects of anaesthesia and surgery to produce postoperative pulmonary complications.

The commonest manifestation of many forms of minimal preoperative respiratory disease is an increased bronchial mucosal irritability, often aggravated by the inhalation of tobacco smoke. This is shown preoperatively in the presence of a chronic cough, and it complicates anaesthesia by producing exaggerated responses to the inhalation of anaesthetic agents and to laryngeal or bronchial manipulation. This can result in coughing or breath-holding during induction, associated with laryngeal or bronchial spasm, which inhibits the uptake of inhaled anaesthetic agents. Patients suffering from bronchial asthma may be more likely to develop bronchospasm, not only in response to inhaled agents but also to a variety of intravenously administered drugs, particularly those associated with the release of histamine. More severe degrees of respiratory disease are associated with disturbed ventilation/perfusion relationships in the lung, and this will impair the uptake of inhalational anaesthetic agents (Saidman and Eger, 1967). The uptake and transfer of oxygen in these patients is also impaired, and higher than normal inspired oxygen concentrations will be required. Consequently, the clinical value of nitrous oxide is reduced. This combination of abnormalities can lead to difficult induction and maintenance of anaesthesia with spontaneous ventilation and, with a relaxant technique, can result in awareness during anaesthesia if customary assumptions are made about relationships between inspired concentrations of anaesthetic agents and total ventilation. Patients with serious mechanical abnormalities of the lungs and increased work of breathing

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may show an exaggerated response to respiratory depressant drugs, including both sedatives and narcotics given preoperatively, and to inhalational anaesthetic agents. Patients who are in overt respiratory failure with a raised arterial CO$_2$ tension will not tolerate such normal measures. Finally, the most severely disabled patients with respiratory disease may be sufficiently orthopnoeic as to be unable to tolerate the supine position for the time taken to carry out anaesthetic or surgical procedures.

Pre-existing respiratory disease may potentiate the effects of anaesthesia and surgery in producing intraoperative and postoperative complications. General anaesthesia is known to be associated with impairment of pulmonary ventilation/perfusion ratios, manifest clinically as relative hypoxaemia and increased alveolar-arterial oxygen tension difference. Hickey et al. (1973) have shown that the magnitude of these changes was greatest in patients with evidence of pre-existing airways obstruction, and in the obese.

The fundamental mechanism whereby respiratory disease predisposes to an increased incidence of postoperative pulmonary complications is that of defective clearance of pulmonary secretions. The mechanisms have been elegantly described by Palmer and Sellick (1953). Essentially, three sets of factors operate: increased volume or viscosity of secretions; defective coughing or ciliary mucosal transport; and narrowing of airways. This combination leads to retention of secretions and possible infection leading to acute infective bronchitis, pneumonia and atelectasis. Increased sputum volume is found in many disorders but is characteristic of chronic bronchitis and bronchiectasis, and increased sputum viscosity is the characteristic lesion of cystic fibrosis. Impaired coughing ability is seen in a variety of neuromuscular and skeletal disorders as well as in pulmonary disease. Ciliary transport mechanisms are defective in chronic bronchitis and in association with cigarette smoking. Narrowing of airways is seen in the whole range of obstructive lung diseases and can be caused by bronchospasm, mucus gland hyperplasia, mucosal congestion or obstructive emphysema. All of these mechanisms may be aggravated by infection.

HISTORY-TAKING

The examination of the patient should commence with a thorough history. The cardinal symptoms of respiratory disease are cough, sputum production and dyspnoea. Coughing is a manifestation of increased bronchial irritability and of impaired ciliary transport of mucus, thus forcing the patient to place increased reliance on cough for clearance of bronchial secretions. Enquiries should be made as to the duration of this symptom as it is often an early one in chronic bronchitis. Cigarette smokers may be so accustomed to chronic cough that they do not consider it abnormal, and specific enquiry about "smoker's cough" may need to be made. Diamant and Palmer (1967) found 35% of their patients to have unequivocal spirometric evidence of obstructive airways disease, yet only 15% had productive cough. Cough of recent onset may imply respiratory infection or the development of new pathology such as bronchial carcinoma. Sputum production should be carefully assessed, enquiry being made about the duration and regularity of the symptom. Again, probing questioning may be required, as many patients cough bronchial secretions into the pharynx and swallow them, and thus may deny sputum production. The volume of the sputum should be noted. Excessive volumes of sputum are characteristic of chronic bronchitis, and in bronchiectasis the amounts can be extreme. Enquiry should also be made as to the quality of the sputum, both its colour and consistency. Thick yellow or green sputum is suggestive of infection.

The presence of dyspnoea implies a more serious degree of respiratory disorder than either of the previous two symptoms alone. Enquiries should be made about the duration of dyspnoea and its chronicity or otherwise. If dyspnoea is episodic in character, then triggering factors should be elucidated. The severity of dyspnoea can be graded by the response to several questions relating dyspnoea to different levels of physical activity. Seasonal influences should also be sought. Patients with chronic bronchitis are frequently worse in the winter, and those with bronchial asthma are worse during summer months. Wheezing in the chest is a variant of dyspnoea.

Additional symptoms include chest pain and haemoptysis. Patients with severe pulmonary hypertension may suffer anterior chest pain similar to that of myocardial ischaemia, and pleuritic pain may be a reflection of past pleurisy or pulmonary embolic episodes. Haemoptysis is a frequently alarming symptom, which may indicate serious pulmonary pathology but in small quantities is usually a consequence of bronchitis.

Weight loss is often seen in patients with
bronchial carcinoma and in those with chronic obstructive emphysema.

The general degree of disability may be assessed from the patient's work record, although this should be interpreted in the light of his personality.

Careful enquiry should be made into tobacco-smoking habits. There are good grounds for regarding every chronic cigarette smoker as suffering from respiratory disease even in the absence of bronchitis. Such patients have an increased incidence of respiratory complications postoperatively (Morton, 1944); they also exhibit a variety of disorders of pulmonary function, including airways obstruction and reduced lung compliance (Miller and Sproule, 1966), diminished peak flow rate, increased functional residual capacity, diminished diffusing capacity (Krumholz, Chevalier and Ross, 1965) and reduced pulmonary surfactant (Finley and Hadman, 1972). In addition, heavy cigarette smokers may have markedly elevated carboxyhaemoglobin concentrations (Birnstingl, Cole and Hawkins, 1967), which will result in a leftward shift of the haemoglobin dissociation curve which may impair oxygen transport. Patients who smoke only a pipe or cigars should be asked whether this is a lifelong habit or whether they were previously cigarette smokers, as the latter group tend to have carboxyhaemoglobin levels comparable to cigarette smokers (Castleden and Cole, 1973).

A history of previous illnesses should be taken, with specific regard to respiratory infections and respiratory complications following anaesthesia and surgery. An occupational history should also be sought in the patient with respiratory symptoms, since in addition to the well-known inhalational dust diseases of the lung, an increasing number of pulmonary lesions are being described due to inhalation of fumes and vapours such as isocyanate. A large number of allergic lung disorders, similar to farmer's lung, have been described in a wide variety of occupations.

In eliciting a history from a patient with respiratory symptoms it is valuable to attempt some quantification of abnormality. This has been done in patients with chronic bronchitis by the use of the special questionnaire developed by the Medical Research Council. This is an approach which might usefully be expanded to serve the purposes of preoperative evaluation; indeed, with careful design such a questionnaire could be self-administered and be a time-saving measure for clinical staff.

**PHYSICAL EXAMINATION**

This should begin with general examination of the patient. Particular points to be noted are evidence of recent weight loss; anatomical abnormalities surrounding the upper airway; central cyanosis which, if present, is of great significance; and finger clubbing which may indicate chronic hypoxia, chronic intrathoracic sepsis such as bronchiectasis, or bronchial carcinoma. The rate and depth of breathing should be observed, and whether the patient appears to be distressed by respiration. Extreme obesity should be noted, as it predisposes to a variety of respiratory complications.

Examination of the cardiovascular system should take particular account of the signs of right ventricular failure and pulmonary hypertension. These include a right ventricular heave, palpable pulmonary artery pulsation, accentuated second sound at the pulmonary area, fourth heart sound at the tricuspid area, hepatojugular reflux, elevated jugular venous pressure and peripheral oedema. Examination of the chest wall should seek to discover abnormalities likely to impair ventilation.

On examining the lungs, note should be taken whether abnormalities are of a widespread character, as might appear in bronchitis or bronchial asthma, or whether more localized changes are present as in bronchiectasis or bronchial carcinoma. Specific treatable lesions should be sought, such as pleural effusion and pneumothorax. The degree of airways obstruction should be estimated and, if not apparent during quiet tidal ventilation, the patient should be asked to perform the forced vital capacity manoeuvre while the lung fields are auscultated. Wheezing heard only during this manoeuvre should be interpreted with caution, however, as it may be due to closure of fairly large bronchi; this may have a different significance from the airways obstruction which is present during normal tidal ventilation (Nunn, 1969). Measurement of the time taken for a complete forced expiration can quantify the degree of airways obstruction. Values under 5 sec are normal, and over 6 sec indicate airways obstruction (Lai, Ferguson and Campbell, 1964).

Examination of the sputum should be carried out, taking note of volume, colour and consistency. Tenacious yellow or green sputum usually is indicative of active infection, although patients with active bronchial asthma may produce purulent-like sputum which on cytological examination proves to consist almost entirely of eosinophils.
A variety of general laboratory investigations are of value. The haemoglobin level should be estimated invariably, since patients with chronic respiratory disease may be polycythaemic. This leads to increased pulmonary and systemic vascular resistance, and in the presence of other disorders predisposes to the development of right heart failure. There is a prospect, as yet unproved, that haemodilution in such patients may be of clinical value (Bergentz, 1972). The electrocardiogram should be recorded in all patients with significant respiratory symptoms, particularly those with dyspnoea, and might with value be recorded in all patients over 40 and all cigarette smokers. The diagnostic value of the e.c.g. in right ventricular hypertrophy or in pulmonary hypertension is limited, but e.c.g. abnormalities of any kind, particularly if associated with impaired ventilatory capacity, have been shown to be accurate predictors of postoperative cardiopulmonary complications (Mittman, 1961). It can be argued that every patient presenting for anaesthesia and surgery should have a chest X-ray performed; not only will this assist in the immediate preparation, but the opportunity afforded by the patient's admission to hospital can be taken to have routine radiography. If abnormalities are found, particularly those suggestive of segmental inflammatory lesions, a lateral film will be of great value in localizing these precisely and thus in initiating planned regimes of postural drainage. Fluoroscopy also may be valuable in selected cases to assess movement of the diaphragm and distribution of ventilation. Loder (1955) reviewed 1,000 routine preoperative chest radiographs and found abnormalities in 11.6%; in 2.9% these were sufficiently marked to require postponement of surgery. However, Kerr (1974), in this issue, is opposed to routine chest radiography in all patients preoperatively.

PREOPERATIVE TESTING OF PULMONARY FUNCTION

A controversy has existed in the past as to whether or not tests of respiratory function need to be performed preoperatively, it having been suggested that a carefully taken history could identify all abnormal subjects (Bethune, Edmonds-Seal, and Gabriel, 1968). This is probably over-optimistic. Buist, Van Fleet and Ross (1973) studied 524 cigarette smokers who presented at an emphysema screening clinic, and found 77% with respiratory symptoms. Simple ventilatory tests exposed abnormalities in only 11%, although this figure could be raised to 44% by use of the closing volume estimation. However, 9% were asymptomatic and yet were found to have abnormalities on testing. Thus, careful testing and a carefully taken history are necessary to define the entire abnormal population.

No single test of respiratory function will provide all the desired information. The simplest test is perhaps the vital capacity manoeuvre, relating the value obtained to a predicted normal for that individual. This can be conveniently performed at the bedside and indeed Lecky, Denlinger and Ominsky (1973) have described a disposable spirometer for this purpose. The vital capacity measurement alone, however, is not of great value in predicting respiratory problems. It will identify, as abnormal, patients with both restrictive and obstructive ventilatory disorders. The former group have only a slightly enhanced risk of pulmonary complications after anaesthesia and surgery, whereas those with obstructive lung disease have a much higher risk and therefore should be specifically identified.

The simplest method of doing this is by analysis of the forced expiratory spirogram. This can be recorded by a variety of instruments, some of which are automated. From the forced expired curve, various measurements can be taken such as forced expired volume in 1 sec, the forced vital capacity, the maximum expiratory flow rate, and the maximum mid-expiratory flow rate. There seems little to choose between these various measurements, although it is held that maximum mid-expiratory flow rate is less subject to variation produced by variation in the patient's effort (Bates, Macklem and Christie, 1971). The forced expiratory manoeuvre can be repeated following inhalation of a 1% isoprenaline aerosol for 2 min. Failure to improve following the inhalation of isoprenaline, indicated by this test, need not necessarily mean that this drug is of no value in management of the patient, as the factors determining airway obstruction during forced expiration are rather different from those during normal tidal ventilation.

Peak expiratory flow rate can be measured simply at the bedside, but the results show wide variation and in epidemiological studies the measurement has been found to be a poor discriminator between normal and abnormal patients.

The maximal breathing capacity test measured over 15 sec has been suggested as an alternative, as it detects a greater number of patients at risk of postoperative complications than does the forced
expiratory manoeuvre alone. This reflects the fact that it will detect patients with both restrictive and obstructive ventilatory disorder.

A significant number of individuals who run an increased risk of pulmonary complications will not be detected by simple breathing tests. Tests of distribution of ventilation can add additional information. The classical approach has been to use the single breath nitrogen test of Fowler, and more recently the modification suggested by Anthonisen et al. (1969), which enables the measurements of closing volume to be made. Closing volume can be measured alternatively by a method based on inhalation of xenon-133.

In addition to these tests of ventilation and its distribution in selected patients with a suggestion of major lung disease, the arterial blood gases should be measured with the patient at rest and in a steady state breathing air. From the arterial PO₂ and PCO₂ one can estimate the alveolar arterial oxygen tension difference and the alveolar PO₂, thus separating the effects of hypoventilation from those of ventilation/perfusion abnormalities. It will be unusual to discover patients with CO₂ retention in the evaluation of patients for elective surgery, although Stein et al. (1962) found 5 with arterial PCO₂ greater than 45 mm Hg in a group of 63 patients assessed before elective surgery and taken at random from the hospital population. Each one of these patients developed severe postoperative complications.

Mittman (1961) has pointed out that the ideal test of respiratory function for use in preoperative evaluation would identify all patients likely to develop complications, and no patients likely to have an uncomplicated course. His own results showed that no single test approached this ideal, but that a better estimate could be made by combining the results of several tests.

In patients scheduled for pulmonary resection, the problems of assessment at preoperative examination are magnified. In addition to the localized surgical lesion to be resected, the patient may have widespread chronic lung disease, and removal of a lobe or a complete lung may reduce pulmonary function to a point below that which can sustain life. The problem is to determine what contribution to total pulmonary function is made by the lobe or lung to be resected. If it is known on the basis of radiology or bronchoscopy that there is complete occlusion of the bronchus to the lung to be resected, and if the patient’s respiratory function is adequate, then it can be predicted that the long-term outlook will be satisfactory. Measurement of the pulmonary artery pressure at rest can be done with the necessary instrumentation at the bedside; if the pressure is elevated preoperatively and pneumonectomy is proposed, then a further significant rise cannot be avoided unless there is specific evidence that the pulmonary artery to the affected lung is already occluded.

In the past, attempts at measuring individual lung function relied on differential bronchospirometry (Bjorkman et al., 1955; Ranson-Bitker, Ladurie and Silbert, 1971). This procedure is complex, requires highly specialized equipment, and may be disturbing for the patient. In recent years, alternatives have become available. Miiörner (1968) has shown good correlation between bronchospirometry and assessment based on the differential distribution of radioactive xenon-133, given either by intravenous injection or by inhalation. Lung scintiscanning with macro-aggregated human serum albumen labelled with technetium-99m, or iodine-131, has been shown to be of value in assessing operability of bronchial carcinoma and, in addition, the presence of large defects in perfusion in the contralateral lung has suggested that pneumonectomy would not be possible (Secker Walker et al., 1971). Garnett et al. (1969) developed a quantitative analysis of the lung scintiscan and showed that the results obtained with this method are comparable to those of differential bronchospirometry. The scintiscan is extremely simple to perform, requiring only an intravenous injection and that the patient is sufficiently at ease to lie supine and at rest for some 15 or 20 min. With these more modern investigations, it may be possible to determine which patients have serious abnormalities in the remaining lung, and therefore which patients would not tolerate pneumonectomy.

In summary, the clinical examination of the patient preoperatively is of cardinal importance in the detection of pre-existing respiratory disease and the prediction of postoperative and intraoperative complications. This procedure is worth performing intensively since preoperative therapy can reduce considerably the complication rate.

REFERENCES


