Investigation of the respiratory system

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Pulmonary function tests (PFTs) are useful in determining the functional capacity of the patient and the severity of pulmonary disease, and in predicting the response to various treatments. The tests range from simple clinic or bedside measurements to those only available in specialist centres. Spirometry is the most commonly performed PFT and measures specifically the amount (volume) and/or speed (flow rate) of air that can be inhaled or exhaled. It is reported in both absolute values and as a predicted percentage of normal. Normal values vary , depending on gender, race, age and height. The most common parameters measured in spirometry are defined below and illustrated in Figure 60.5 . Basil Martin Wright , 1912–2001, member of the scientific sta ff of the Medical Research Council Research Centre, Northwick Park Hospital, Harrow , UK. Peak expiratory flow rate Peak expiratory flow rate (PEFR) is measured by a Wright peak flow meter or a peak flow gauge. This is the maximum airflow velocity achieved during an expiration delivered with maximal force from the total lung capacity . It is a reliable and reproduc - ible test but has the disadvantage of being e ff ort dependent, and it may therefore be a ff ected by abdominal or thoracic wound pain. PEFR measurements are often used in managing asthma, but there are many other causes of low PEFR such as a pr oblem with large airway patency . Forced expiratory volume in 1 second The forced expiratory volume in 1 second (FEV) is the amount 1 of air forcibly expired in 1 second. It is low in obstructive lung disease and may be normal in patients with poor gas exchange. Forced vital capacity The forced vital capacity (FVC) is the volume of air forcibly displaced following maximal inspiration to maximal expiration. The FEV and the FVC can be measured using a Vitalograph, 1

Postoperative Perioperative dyspnoea death Dynamic lung volumes, Thoracoscore transfer factor +/– split function testing Yes Offer surgery as part of multimodality management

(b) and a ratio (FEV /FVC) can be calculated (Figure 60.5 1 low ratio indicates obstruction and the test should be repeated after bronchodilators. A normal ratio (FVC and FEV reduced 1 to the same extent) indicates a restrictive pathology . There are two physiological categories of lung disease: obstructive and restrictive (Table 60.1). In obstructive condi tions such as asthma or emphysema, the flo w of air in and out of the lungs is impaired. In restrictive disease, such as lung fibrosis, the lungs have lost size or elasticity , becoming ‘sti ff ’ so that they do not fill or expand properly . Diffusion capacity The di ff usion capacity (DLCO) is a measurement of the lung’s ability to transfer gases and is often referred to as the ‘trans fer factor’. It cannot be performed at the bedside, requires the patient’s current haemoglobin level and is a test of the integrity of the lung’s alveolar–capillary surface area for gas ex change. In lung diseases that damage the alveolar walls, such). A ↓↓ ↓ - ↓↓ ↓ ↓ ↓↓ as emphysema, or that thicken the alveolar membrane, such as lung fibrosis, it may be reduced. In patients who require - surgery to remove part of their lung, for example for lung cancer, measurement of DLCO is an important determinant of ‘fitness’ for surgery and it should be measured formally as part of a lung function test.

4 4 3 3 q 2 2 Volume (litres) 1 1 1 0 2 3 4 5 6 0 Normal Obstructive Tidal volume Total lung capacity (TLC) Normal Figure 60.5 Spirometry. (a) Spirogram tracings obtained from a Vitalograph: vital capacity (FVC) 3.8 litres, FEV /FVC 82%; (ii) obstructive defect, reversible asthma, 1 FEV /FVC 40%; q after a bronchodilator, FEV 2.5 litres, FVC 3.5 litres, FEV 1 1 2.0 litres, FEV /FVC 90%. No change with bronchodilators. (b) 1 from Gray HH. Pulmonary embolism. Medicine International 1993; 4 3 2 p 1 1 2 3 4 5 6 1 0 2 3 4 5 6 Time (seconds) Restrictive VC TLC Vital capacity (VC) VC TLC Obstructive Restrictive (i) normal forced expiratory volume in 1 s (FEV) 3.1 litres, forced 1 p before a bronchodilator, FEV 1.4 litres, FVC 3.5 litres, 1 /FVC 71%; (iii) restrictive defect, /f_i brosing alveolitis, FEV 1.8 litres, FVC 1 1 Changes in lung volume in obstructive and restrictive lung disease. (Reproduced 21 : 477, by kind permission of the Medicine Group (Journals) /uni00A0 Ltd.) TABLE 60.1 Spirometry values in obstructive and restrictive lung diseases. Obstructive pattern Restrictive pattern PEFR Normal or FEV Normal or 1 FVC Normal or FEV /FVC <70

> 80 1 FEV , forced expiratory volume in 1 second; FVC, forced vital 1 capacity; PEFR, peak expiratory /f_l ow rate.

Oxygen saturation (S O) refers to the degree of oxygen p 2 molecules (O) carried in the blood attached to haemoglobin 2 molecules (Hb). It is a measure of how much oxygen the blood is carrying as a percentage of the maximum it could carry . The common method of monitoring the oxygenation of a patient’s haemoglobin is through a pulse oximeter. Blood gases The S O measured non-invasively with a pulse oximeter p 2 measures only oxygenation, not ventilation, and provides no information regarding a patient’s carbon dioxide or bicarbon ate levels, blood pH or base deficit. This requires arterial blood sampling or ‘blood gases’ (Table 60.2). The FEV and DLCO are often used to predict the risk 1 of postoperative dyspnoea after lung resection. The predicted postoperative values can be calculated by considering the vol ume of lung, more specifically the number of bronchopulmo nary segments, expected to be removed at surgery . For example if five segments of the left upper lobe are to be removed, the postoperative predicted FEV in a patient with a preoperative 1 FEV of 2.5 litres (85% predicted) is ((19 /uni00A0 – /uni00A0 5)/19) /uni00A0 ×/uni00A0 2.5 = 1 1.84 litres and ((19 /uni00A0 – /uni00A0 5)/19) /uni00A0×/uni00A0 85% = 62.6% predicted. This assumes that all bronchopulmonary segments are function ing (e.g. not collapsed) and contribute equally to lung func tion. Although an optimum cut-o ff of postoperative predicted FEV of 40% is widely cited, there are currently limited data 1 to provide guidance on this figure to help predict an acceptable degree of postoperative dyspnoea and quality of life. Patients should still be o ff ered surgical resection if the predicted risk of postoperative dyspnoea is moderate or high, as long as they are aware of and accept the risks of dyspnoea and associated complications. Exercise testing Other functional assessments, including the shuttle walk test, 6-minute walk test, stair climbing coupled with other tests such as oxygen saturations, as well as cardiopulmonary exercise testing (CPET), could be considered for patients at moderate or high risk of postoperative dyspnoea and may help predict surgical outcome after lung resection. In patients with moder ate to high risk of postoperative dyspnoea, using a shuttle walk test distance of >400 /uni00A0 m and CPET of >15 /uni00A0 mL/kg/min are cut-o ff values for good function. Ernest Henry Starling , 1866–1927, Professor of Physiology , University College, London, UK. The key to many aspects of practical chest surgery is an under - standing of the pleura and of the mechanics of breathing. Management of the essentially healthy pleural space is logical and simple and needs minimal technology . On the other hand, when pleural disease is advanced, for example when there is gross pleural sepsis surrounding a leaking and trapped lung, management is di ffi cult and the patient ma y require prolonged care with repeated interventions. The physiology of pleural fluid - The turnover of fluid in the human pleural space is about 1–2 /uni00A0 litres in 24 hours, with only 5–10 /uni00A0 mL of fluid present at any one time as a film, about 20 /uni00A0 /uni03BC m thick, between the visceral and parietal pleura. The mechanisms and equations given are simplifications but serve to explain the clinical conditions encountered. The fluid is produced from the capillaries of the parietal pleura as a transudate, according to the Starling capillary loop pressures. Howe ver, there is a further negative force in the pleura. The elastic content of the lung causes it to recoil and collapse if not held open by the negative pressure in the pleura. This elastic recoil exerts about 4 /uni00A0 mmHg of negative pressure and favours accumulation of fluid. The secreting forces add up to about 11 /uni00A0 mmHg in health. Pleural fluid is mainly reabsorbed (about 90%) by the visceral pleura, whose capillaries are part of the pulmonary circulation. The principal force in absorption of pleural fluid is oncotic pressure (approximately 25 /uni00A0 mmHg) - minus the di ff erence in mean capillary hydrostatic pressure of - the pulmonary capillary (8 /uni00A0 mmHg). Thus, the overall absorb - ing pressure is 25 /uni00A0 – /uni00A0 8 = 17 /uni00A0 mmHg, producing a net drying e ff ect (17 /uni00A0 – /uni00A0 11) of about 6 /uni00A0 mmHg (Figure 60.6). Gas in the pleural space There is normally no free gas in the pleural space because - the same physiological mechanism that absorbs air from a - pneumothorax prevents any gas accumulating. The partial pressures (water as saturated vapour pressure) of the gases in venous/end-capillary blood are: /uni25CF P O 40 /uni00A0 mmHg 5.3 /uni00A0 kPa 2 /uni25CF P CO 46 /uni00A0 mmHg 6.1 /uni00A0 kPa 2 /uni25CF P N 573 /uni00A0 mmHg 76.4 /uni00A0 kPa 2 /uni25CF P H O 47 /uni00A0 mmHg 6.3 /uni00A0 kPa 2 These partial pressures add up to less than atmospheric pressure (760 /uni00A0 mmHg). Free gas is therefore absorbed into the blood and lost to the atmosphere thr ough the lungs, with the gases moving in relation to their solubility (carbon dioxide quickest and nitrogen slowest) and relative concentrations in the pleural space and the blood. This does not favour nitrogen, which constitutes about 80% of atmospheric air. Breathing oxygen accelerates nitrogen removal by reducing the content - of nitrogen in the blood and increasing the gradient for its absorption. Nitrous oxide anaesthesia is dangerous in the pres - ence of a pneumothorax; nitrous oxide is very soluble and, although not normally present in the pleural space, it will be

TABLE 60.2 Arterial blood gases: ‘normal values’. pH 7.35–7.45 PaCO 4.5–6 /uni00A0 kPa (35–50 /uni00A0 mmHg) 2 PaO 11–14 /uni00A0 kPa (83–105 /uni00A0 mmHg) 2 Standard bicarbonate 22–28 mmol/L Anion gap 10–16 mmol/L Chloride 98–107 mmol/L

(b) rapidly transported into the space if the patient is given nitrous oxide to breathe.

Produced at a rate of: and reabsorbed: 0.6 mL/kg per hour or 1000 mL 80–90% into per day pulmonary capillaries 10–20% (plus protein) into lymphatics Capillary hydrostatic +32 +8 pressure Colloid –25 –5 –25 pressure 4 Elastic recoil Net drying effect 6 mmHg Figure 60.6 (a) Production and absorption of pleural /f_l uid. (b) pleural physiology. (See the text for an explanation of this simplistic physiological model.)

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