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PULMONARY PHYSIOLOGY

Its Application to the Determination of Respiratory Impairment and Disability in Industrial Lung Disease

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An adequate appreciation of the manner in which the pneumoconioses and similar diseases produce respiratory impairment is dependent on some knowledge of respiratory function in health and disease. This chapter is written with the intention of explaining to the nonphysiologist the basic disorders of pulmonary function that may be encountered in industrial lung diseases. It is intended primarily for the industrial physician, internist, pathologist, and epidemiologist rather than the chest physician and physiologist, and as such inevitably will appear to many to be oversimplified.

The main function of the lungs is to keep the oxygen and carbon dioxide content of the arterial blood within a certain narrow range. This has to be effected despite the fact that both oxygen requirements and carbon dioxide production are constantly changing with the degree of activity of the subject. The lungs accomplish the function of gas exchange by bringing venous blood into contact with the gaseous environment over an enormous surface area—the alveolo-capillary surface. Three processes are involved in gas exchange: ventilation (often referred to as the

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bellows function), diffusion, and perfusion or blood flow. When respiratory impairment is present, either one, two, or all three processes may be affected, although almost always to varying degrees. Let us consider each process in turn.

VENTILATION

The ventilatory capacity or bellows function depends firstly on the size of the lungs (lung volumes), secondly, on the caliber of airways (airways resistance), thirdly, on the compliance or elastic properties of the lungs, and lastly, on the properties of the chest wall. Certain of the lung volumes can be measured with a spirometer; however, others require more complicated apparatus. The total volume of air that the lungs and bronchial tree contain after a maximal inspiration is known as the total lung capacity (TLC). If the subject then exhales as much air as he can, the volume of air remaining in the lungs after the expiration is known as the residual volume (RV), while that which has been expelled is known as the vital capacity (VC). It must be stressed that during the measurement of VC, the patient is permitted to take as long as he likes to complete the maneuver. If after a maximal inspiration he exhales as rapidly and forcefully as possible, then the measurement obtained is known as the forced vital capacity (FVC). In normal persons the VC and FVC are usually not significantly different; however, in certain types of obstructive airways disease, e.g., emphysema, the FVC may be considerably less than the VC as a result of collapse of the smaller airways during the forced expiration, a phenomenon known as air trapping. The volume of air remaining in the lung at the end of a normal expiration is known as the functional residual capacity (FRC), whereas the volume that can be exhaled from FRC is known as the expiratory reserve volume (ERV). Similarly, the volume of air that can be taken in from FRC is known as the inspiratory capacity (IC) and, needless to say, the sum of the ERV and IC equals the VC (Fig. 2–1). The VC, ERV, and IC can be measured

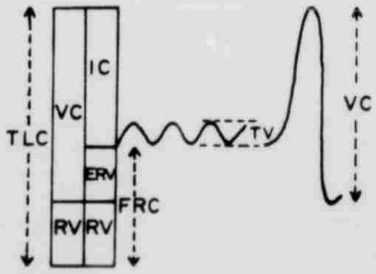


Figure 2–1 Lung volumes and a spirometric tracing of a slow vital capacity maneuver in a normal subject. (*TLC*, total lung capacity; *RV*, residual volume; *IC*, inspiratory capacity; *ERV*, expiratory reserve volume; *VC*, vital capacity; *TV*, tidal volume; *FRC*, functional residual capacity.)

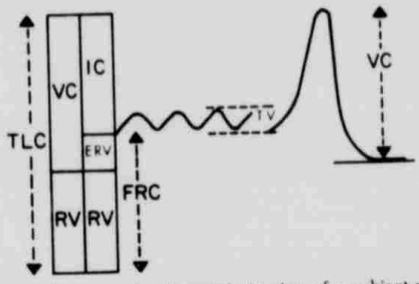


Figure 2–2 Lung volumes and spirometric tracing of a subject with obstructive airways disease. The figure is drawn to the same scale as used in Figure 2–1 and the abbreviations are the same as shown there.

with a spirometer; TLC and its derivatives, FRC and RV, require other means, viz., either closed-circuit helium equilibration, the nitrogen washout, or the body plethysmograph. The last technique is preferable if available since it measures all the gas in the thoracic cage, while with the helium equilibration and nitrogen washout methods regions of poorly ventilated lung that contain trapped gas, e.g., bullae, are not included, and thus falsely low values are obtained. Despite the more expensive apparatus necessary to measure the RV, knowledge of this lung volume is usually less helpful clinically and diagnostically than is a knowledge of the VC.

Total lung capacity can also be accurately determined from the chest film. Barnhard and his colleagues have described a method which utilizes a posteroanterior and lateral film.[1] This method has since been simplified by Reger and co-workers.[2] The lungs are treated as a series of elliptical cylindroids and allowance is made for heart size and pulmonary blood volume. This method is accurate and has application to epidemiological surveys. If the vital capacity is determined by spirometry, it becomes possible to measure the RV:TLC ratio.

In a healthy young adult, the residual volume (RV) is around 20 per cent of the total lung capacity (TLC). As the subject grows older, the RV slowly increases, so that by the age of 60 it may constitute up to 40 per cent of the TLC. The increase in RV is related to the fact that with increasing age the lung loses some of its elasticity. The decreased elastic recoil of the older person is opposed by an unchanged and relatively greater intrapleural pressure which maintains the lungs at a higher state of inflation than previously. This state of affairs used to be known as senile emphysema; however, since there is neither airways obstruction nor disruption of the alveolar-capillary surface, the term is a misnomer.

In emphysema and obstructive airways disease the RV:TLC ratio is increased, and in many instances the RV may be well over 50 per cent of the TLC (Fig. 2–2). An increased RV:TLC ratio is a frequent finding in

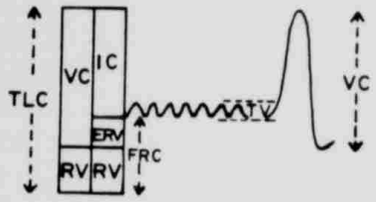


Figure 2–3 Lung volumes and spirometric tracing of a subject with diffusion fibrosis, e.g., severe asbestosis. The figure is drawn to the same scale as used in Figure 2–1 and the abbreviations are the same as shown there.

obstructive airways disease, but lesser increases in the ratio are sometimes seen in the diffusion fibroses, such as asbestosis.[3] Such increases are more apparent than real and probably are related to inadequacies in the predicted values for RV and TLC and to a relative decrease in TLC while the RV remains unchanged. In the diffusion fibroses, all the lung volumes tend to be smaller than their predicted figure. This is most evident in regard to the VC and TLC (Fig. 2–3). In contrast, and for the reasons just mentioned, changes in RV are often less spectacular. Thus in an individual subject, small increases in the RV:TLC are not necessarily diagnostic of any particular type of physiological impairment. In contrast, in epidemiological studies, knowledge of the mean ratio in groups of persons is much more useful. When it has been demonstrated that a group of subjects have a significantly increased RV/TLC ratio as compared to a comparable control group, then it can be inferred that they have a higher prevalence of obstructive airways disease.

Ventilatory capacity also depends to a large extent on the resistance to air flow in the bronchial tree. If a normal subject is asked to take in as big a breath as he can and then blow it out as rapidly and as forcefully as possible, he should be able to get out 80 per cent of his FVC in 1 second and 95 per cent in 3 seconds. The flow rates are most rapid in early forced expiration, but when the subject begins to approach residual volume there is marked slowing. This is most evident once the subject gets low his FRC, and when RV is reached, flow ceases entirely (Fig. 2–4). Subjects with obstructive airways disease, that is to say, subjects who have an increased resistance to the flow of air in and out of their lungs,

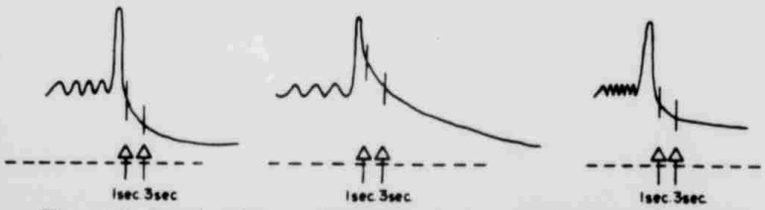


Figure 2–4 Forced expiratory volume maneuvers in a normal subject and in subjects with obstructive and restrictive pulmonary disease.

namely asthmatics, chronic bronchitics, and those with emphysema, all show a flatter curve with diminished flow rates. In many instances there is also a loss of VC. In asthma, but not in the other two diseases, the use of bronchodilators, viz. isoproterenol or epinephrine by nebulization, appreciably relieves the obstruction so that the forced expiratory volume curve becomes steeper and more closely resembles that of a normal person. As indices of obstruction both the 1 second and 3 second timed vital capacity (percentage of FVC exhaled in 1 and 3 seconds, respectively) are frequently used. Some workers, on the other hand, prefer to use the actual volume of air exhaled in the first second of a forced expiration (FEV_1), whereas others prefer the volume exhaled in the first 0.75 second ($FEV_{0.75}$). A less popular but still commonly used measurement is the maximal expiratory flow rate (MEFR), this being the rate of flow between 200 and 1200 ml. on a forced expirogram. A further measurement that also has its advocates is the MMF ($FEF_{0.25-0.75}$), or maximal expiratory flow over the middle half of a forced expiratory spirogram. All of these indices have their proponents, but there are valid reasons for preferring the FEV_1 and the MMF.

An additional method by which the ventilatory capacity can be assessed is by means of the maximal breathing capacity (MBC). This should preferably be known as the maximal voluntary ventilation (MVV) and is the maximal volume of air that is breathed over 1 minute. As the test is very tiring to subjects with obstructive airways disease, the volume is usually measured over a period of 15 or 20 seconds and the result multiplied by the necessary factor. This test is effort-dependent and has little advantage over the single breath tests. To help those physicians who were brought up on this measurement, and whose minds are too set to accommodate a change, the simple expedient of multiplying the $FEV_{0.75}$ by 40 gives an excellent approximation to the measured MVV.

All the above tests with the exception of the MVV are derived from the forced expirogram (Fig. 2–4). Most correlate reasonably well with the direct measurement of airways resistance, but it must be stressed that the forced vital capacity is not a physiological procedure. Moreover, the performance of a forced expiration leads to a marked diminution in the caliber of the larger airways. As such, the forced vital capacity is a reflection of upper airways behavior during airways compression and depends mostly on the resistance to flow in the trachea and main, segmental, and smaller bronchi. Obstruction in the smaller airways, i.e., at respiratory bronchiolar level, is less likely to be detected by the forced expirogram. In this context, it is as well to remember that around 80 per cent of the resistance to air flow is situated in the larger airways, and the resistance of the lower airways is relatively small in comparison. This will be alluded to in more detail later.

Airways resistance can be measured directly in several ways. Apart from the body plethysmograph, undoubtedly the most accurate method,[4]

the other techniques are unsuitable for general use since they are either still at the experimental stage or else involve unpleasant procedures such as the passage of an esophageal balloon. While it is possible to use the body plethysmograph in field studies, it is not as practicable as is a spirometer. Nonetheless, airways resistance determined in the body plethysmograph is in some ways a more physiological measurement since it is made during quiet panting.

Ventilation also depends on the compliance of the lungs. This is a measure of the distensibility of the lungs and is expressed as the change in lung volume that occurs when the pressure gradient between the pleura and alveoli is changed by 1 cm. of water. It may be measured during breath holding (static compliance) or during regular breathing (dynamic compliance). If the increase in volume were directly proportional to the pressure change throughout the range of inflation and deflation, then a single value for static compliance would describe the elastic properties of the lungs. In reality this is not the case; the lungs become less compliant at higher lung volumes, and it is only in the tidal volume range that the relationship is approximately linear. Compliance also depends on lung size, and the larger the lungs, the more compliant they are. Thus an infant's lung is less compliant than is an adult's, and likewise there is a disparity in compliance of the lungs between large and small men. The reason for this can be clearly seen if one considers the hypothetical case of a man whose vital capacity is 5 liters and whose static compliance is 0.20 liter per centimeter of water. Under such circumstances an increase in his negative intrapleural pressure by 1 cm. of water increases the volume of his lungs by 200 ml. Were he then to have one lung removed, this would reduce his vital capacity to 2.5 liters, and a pressure change of 1 cm. would then increase his lung volume by only 100 ml. In short, his compliance would be halved, although the elastic properties of his lung would be unchanged. This is an oversimplification of the problem, since following pneumonectomy some compensatory overdistention of the remaining lung occurs. The latter phenomenon nonetheless is responsible for only a marginal increase in the lung volumes of the remaining lung. To get around the problem of lung size, a measurement known as specific compliance has been introduced. This relates compliance to lung volumes and is obtained by dividing the static compliance by the FRC. If the lungs become stiff and fibrotic, as frequently occurs in asbestosis, sarcoidosis, berylliosis, and certain other diseases, the compliance is markedly reduced, and values of 0.04 liter per centimeter of water and less may be found. In subjects who have a marked reduction of their compliance, the vital capacity is usually concomitantly decreased.

Dynamic compliance may be defined as the ratio of tidal volume to the difference between pressure at end-inspiration and end-expiration at points of no flow during breathing. In normal subjects, measurement of

dynamic compliance gives similar values to the static compliance; however, when the airways resistance is increased, an appreciable difference is often present. This is best explained by considering a state of affairs in which there is partial obstruction to a lobe or lung. In the unobstructed region flow is maximal and there is an appropriate increase in volume for this area; in contrast, the flow of gases into the region with the increased airways resistance takes place more slowly, thereby causing a smaller increase in volume per head of pressure.

The intrathoracic resistance of the airways may be partitioned into central and peripheral components.[5, 6] The central component includes the resistance from the trachea to roughly the eleventh generation of bronchi. In contrast, the peripheral component is made up of the resistance from the alveoli to the twelfth generation of bronchi. The central resistance in normal subjects makes up 85 to 90 per cent of the total resistance. Thus the peripheral resistance constitutes only 10 or 15 per cent of the total resistance and at higher lung volumes is negligible. It is, therefore, possible for a subject to have diffuse disease of the smaller airways and yet have a normal airways resistance and normal spirometry. Yet, although the total resistance may be normal, if the resistance in peripheral airways is increased, the lungs are less distensible.[7] The peripheral airways are usually not uniformly and diffusely affected; rather, the pathological processes producing small airways disease tend to lead to regional involvement.

Most industrial diseases affect the peripheral parts of the lung, viz., asbestosis and coal workers' pneumoconiosis. The involvement is obviously related mainly to the physics of particle deposition and to a lesser extent to the clearance mechanisms of the lungs. However, if the lesions are peripherally situated in the smaller airways, then it will be unlikely that they will be detected by conventional lung function tests, viz., spirometry and the determination of airways resistance. The detection of changes in air flow resistance in the smaller airways is a challenge of some magnitude. There are several techniques that can be used but all have their drawbacks.[8, 9]

Determination of Dynamic Compliance at Different Respiratory Rates

At a normal respiratory rate, inspired air usually has sufficient time to be distributed and to enter all areas of the lung, even though some of the smaller airways may be narrowed. At faster respiratory rates, there is less time for ingress and egress of inspired air through the narrowed airways, and as a result, there are regions of the lung that are not fully inflated with each respiration. In short, they have different time constants,

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and they fill and empty at different rates. Consequently, their dynamic compliance falls at faster respiratory rates.

When in the presence of a normal total airways resistance and normal retractive forces (a normal pressure volume curve) it is possible to demonstrate a fall in dynamic compliance at increased rates of breathing, this can be assumed to mean either that the peripheral airways have an increased resistance to flow or that the lungs are irregularly and regionally affected by emphysematous areas which are not sufficiently severe to affect the static compliance.[8,9] The measurement of dynamic compliance at different frequencies is technically difficult, is tedious to calculate, and involves the swallowing of an esophageal balloon, all of which drawbacks militate against its widespread use.

Measurement of Closing Volume

When during a vital capacity maneuver, a bolus of gas, e.g., xenon-133, is administered at residual volume, the gas is distributed mainly to the upper zones of the lungs. If during subsequent vital capacity maneuvers, the concentration of xenon-133 is measured at the mouth, a characteristic alveolar plateau develops. After the dead space is washed out, a gradually rising slope appears (Fig. 2–5). Toward the end of this gradual slope, the slope increases rapidly. It is postulated that the increased slope is a consequence of a change in emptying sequence, the lower zones having undergone airways closure with the result that the upper airways make a greater contribution to the expired gas. The junction between the slowly rising slope and the more rapidly rising slope is known

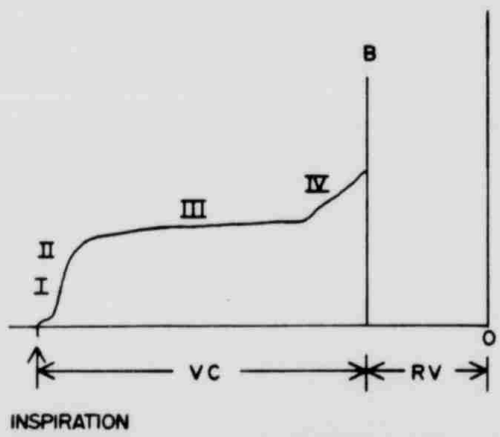


Figure 2–5 "Closing volume" measurement. Phases I and II represent the washing out of dead space. The "plateau" (phase III) ends abruptly with phase IV, and it is the junction of phase III and IV that represents the "closing volume."

as the closing volume.[10] Despite the widespread use of the title "closing volume," it has not been conclusively demonstrated that the junction of the two slopes represents airways closure.

There is some evidence that disease of the peripheral airways is associated with higher closing volumes; however, the exact significance of an elevated closing volume remains debatable. It has subsequently been shown that premature closure of airways may occur in the obese, those with thoracic deformity, heavy smokers, and asthmatics in clinical remission.[11] Nevertheless the measurement of closing volume does not involve invasive techniques and it can therefore be used in epidemiological surveys. Many subjects, however, have difficulty doing the appropriate respiratory maneuvers.

Use of Flow Volume Curves

There is some evidence that the flow volume loop might be useful in the investigation of the state of the peripheral airways.[12] The latter part of the flow volume loop is probably a reflection of the condition of the smaller airways since this part of the curve is not related to muscular effort. At these volumes expiratory flow appears to be governed by the mechanical properties of the lung surrounding the small airways and by the resistance to flow in them.

Use of the Maximal Midexpiratory Flow Rate (MMF)

While it is commonly accepted that the first part of the standard forced expiratory volume curve is effort-dependent, there is much evidence to suggest that the mid and later parts of the curve are not influenced by effort. For this reason the MMF appears to have advantages over the standard spirometric tests and perhaps is influenced more by changes in the peripheral airways than by disorders of the larger airways.[13]

Finally, ventilation is also influenced by the compliance of the thoracic cage. Skeletal disease such as kyphoscoliosis and ankylosing spondylitis may limit greatly the bellows function of the lung. Obesity and pleural thickening may have similar effects. Owing to technical difficulties, measurements of chest wall compliance have little application to the assessment of clinical chest disease.

DIFFUSION

After oxygen reaches the alveoli, it has to diffuse across the alveolocapillary membrane before it can be taken up by the hemoglobin of the

blood. Similarly, carbon dioxide has to diffuse in the reverse direction before it can be excreted in the expired air. Diffusion of gases depends on the difference between alveolar and pulmonary arterial partial pressure. The diffusing capacity of the lungs (D_L) for both oxygen and carbon monoxide can be measured. D_L is defined as the number of milliliters of the gas that traverse the alveolo-capillary membrane for every millimeter of mercury of pressure gradient acting across that membrane. Thus the diffusing capacity for oxygen may be expressed as follows:

$$D_{L_{O_2}} = \frac{\text{ml } O_2 \text{ uptake per minute}}{\text{Alveolar } O_2 \text{ tension} - \text{capillary } O_2 \text{ tension (mm. Hg)}}$$

Riley's method of measuring the $D_{L_{O_2}}$ is impractical for routine clinical use.[14] Carbon monoxide is a more suitable gas than oxygen for measuring D_L since it has a greater affinity for hemoglobin and exerts a negligible pressure in the capillary blood. Two methods using carbon monoxide are currently in use, the steady state and the single breath method. The former method, as described by Filley, MacIntosh, and Wright, is performed while the subject is exercising and requires arterial puncture.[15] Bates, however, has modified the technique in that he uses an end-tidal sample of expired air as being a reasonable reflection of the arterial tension of the gas.[16] The single breath test is performed at rest and does not require an arterial blood sample.[17]

Unfortunately, D_L is influenced by a variety of other factors, viz., the area of the alveolo-capillary surface and hence body size, the red cell content of the capillaries, and ventilation-perfusion abnormalities. In normal subjects the measurement of D_L is a fairly accurate reflection of the state of the alveolo-capillary membrane but this is no longer true when the lungs have an appreciable ventilation-perfusion abnormality.

Pneumonectomy will decrease the diffusing capacity for oxygen by 50 per cent. Similar reductions in D_L are seen when there has been destruction of the diffusing surface such as occurs in emphysema. The diffusing capacity may be partitioned into two components – first, that part which depends on the alveolo-capillary membrane itself (D_M), and second, that part which depends on the pulmonary capillary blood flow. Although it is possible to measure these components, the technique is complicated and not suitable for general use in a clinical pulmonary function laboratory. It is apparent, therefore, that D_L is influenced by many factors. It has been suggested that the term transfer factor should be substituted for diffusing capacity, a suggestion that has appeal to the purist. However, all but the most naive have known for years that D_L depends on several factors of which the physical state of the alveolo-capillary membrane is only one. Aside from this, when the term "transfer factor" is used in the United States it has a connotation associated with delayed hypersensitivity.

D_L is reduced in those conditions in which the lungs have lost their compliance, viz., asbestosis, sarcoidosis, berylliosis, and diffuse interstitial fibrosis, and also in conditions where the pulmonary vascular bed is destroyed. As already indicated, a marked reduction of D_L may be present in emphysema (but not in asthma), after resectional surgery, in pleural thickening, and in disease in which the number of functioning alveoli are reduced, e.g., progressive massive fibrosis and alveolar proteinosis.

PERFUSION

For oxygen exchange to occur, the gas has first to be conveyed to the alveoli. This is effected by the bellows function of the lungs. Once the gas is in the alveoli, it then has to diffuse across the alveolo-capillary membrane before it can be taken up by the blood that is perfusing the capillaries. The excretion of carbon dioxide depends on the same processes but in reverse. Unless the pulmonary capillaries are fully and adequately perfused, oxygen uptake and carbon dioxide excretion may be impaired despite normal ventilatory and diffusing capacities. The classic example of this state is pulmonary embolism.

For the lungs to fulfill their function, that is to say, to maintain the arterial partial pressures of oxygen and carbon dioxide at 85 to 95 mm. and 38 to 42 mm. of mercury, respectively, requires that the ventilation perfusion ratio of the lungs as a whole be relatively normal. Maintenance of the blood gases at normal levels depends to a limited extent only on the absolute levels of ventilation and perfusion. Thus it is possible for a person to lose 50 per cent of his pulmonary ventilation and perfusion and yet have normal blood gases, a state of affairs commonly seen following pneumonectomy. Under other circumstances, a 50 per cent reduction of ventilation and perfusion can be fatal, viz., were the right lung to lose its blood supply and the left lung its ventilation. The latter situation is largely hypothetical and is only seen at postmortem; nonetheless, a little imagination shows how lesser degrees of regional mismatching of ventilation and perfusion may produce a variety of defects of gas transport. Ventilation perfusion inequalities produce two main effects:

1. An Increase in Physiological Dead Space. The anatomical dead space (V_{D_A}) is defined as that part of the tidal volume which does not participate in gas exchange. It is made up of the air in the mouth, nasopharynx, trachea, and bronchi and extends as far as the terminal bronchioles; in short, it is the conducting system of the lungs. In a normal subject it is around 150 ml. The physiological dead space (V_{D_P}) comprises the anatomical dead space plus those regions of the lung parenchyma where gas exchange is absent or less than optimal owing to either overventilation or inadequate perfusion. In a normal person the anatomi-

cal and physiological dead spaces are almost equal. When perfusio reduced or inadequate in certain regions of the lung that are adequat ventilated, then there is an increase in wasted ventilation and phy logical dead space (V_{D_p}). In normal subjects, exercise and hypervent tion produce an increase in V_{D_p}. An increase in wasted ventilation is s when the pulmonary vascular bed is compromised, when the lungs less compliant than usual, and in emphysema. Aside from an absol increase in physiological dead space, the ratio of dead space to tidal v ume (V_D/V_T ratio) is increased in these conditions as well.

2. ***Physiological Shunting or Venous Admixture.*** In the nor subject there is slight mismatching of ventilation and perfusion with result that the arterial blood is 97 per cent saturated (Pa_{O_2} 100 mm mercury). If a perfect ventilation perfusion relationship existed, the sa ration would be 97.6 per cent and the Pa_{O_2} 104 mm. When bl traverses patent capillaries that are adjacent to poorly ventilated alve it leaves the lungs with markedly reduced saturation and P_{O_2}. This t of physiological shunt, viz., blood passing through capillaries which either inadequately or not ventilated, produces systemic arterial saturation. Physiological shunts of the above type have their origin regional ventilation perfusion abnormalities such as are found in pne monia and chronic bronchitis. When a whole lobe is consolidated, d saturation can be severe and is usually evident as cyanosis.

Desaturation produced by an abnormal ventilation perfusion rat has to be distinguished from that produced by a venous to arterial shu This distinction can be made by giving the subject 100 per cent oxyge to breathe for 7 minutes. In subjects with a poor distribution and badl ventilated areas of the lung, it may require 20 minutes to wash out the n trogen from these regions. A normal subject or a patient with a physi logical shunt or a diffusion fibrosis will be found to have Pa_{O_2} of abo 600 mm., that is to say, atmospheric pressure less the partial pressures carbon dioxide and water vapor in the alveolar air. The huge alveolo-a terial gradient produced by breathing 100 per cent oxygen easily ove comes any thickening of the alveolo-capillary membrane and so ensur complete saturation. In contrast, the arterial partial pressure in a subje with an anatomical shunt will rise on pure oxygen but cannot reach 60 mm. of mercury. If the shunt is 25 per cent or less of the cardiac outpu when the patient breathes pure oxygen complete saturation is usual, th Pa_{O_2} rising to somewhere between 150 and 550 mm. of mercury. D saturation due to hypoventilation is easily distinguished from that pr duced by a diffusion fibrosis or a ventilation perfusion abnormality, sinc hypoventilation is invariably associated with carbon dioxide retention

Arterial oxygen desaturation is frequently seen in occupational lun disease and can be a consequence either of a diffusion fibrosis (beryl liosis, asbestosis) or more often of an abnormal ventilation perfusio ratio (byssinosis, progressive massive fibrosis, conglomerate silicosis).

THE APPLICATION OF PULMONARY PHYSIOLOGY TO THE DETERMINATION OF RESPIRATORY DISABILITY

So far this chapter has been devoted to pulmonary physiology and the derangements of lung function that may be found when industrial lung disease is present. A few general comments seem pertinent at this stage. When the problem of industrial lung disease is viewed as a whole, it is evident that it matters little whether the physiological defect is obstructive or restrictive in nature, the presenting symptom is virtually always shortness of breath. Moreover, it is this symptom which is almost always related to respiratory disability and is therefore of paramount importance. Thus some elaboration is necessary concerning the relationship between dyspnea and respiratory impairment.

The commonest presenting symptom of subjects with pulmonary insufficiency is dyspnea. This complaint, like pain, is a subjective sensation and its intensity depends on both the attitude and the personality of the patient. Not uncommonly the severity of this symptom may be exaggerated in order to obtain financial gain, usually in the form of compensation. Notwithstanding all the esoteric paraphernalia of pulmonary function testing, given a modicum of clinical acumen it is usually possible to assess reasonably accurately the extent of any pulmonary insufficiency that is present by means of an adequate history and physical examination.

Perhaps at this stage some clarification of terms is necessary. Pulmonary insufficiency or respiratory impairment is best defined as an inability of the human respiratory apparatus to perform satisfactorily one or more of the three components of respiration, viz., ventilation, perfusion, and diffusion. Pulmonary disability exists when a subject is no longer capable of continuing his occupation because of respiratory symptoms. The assessment of respiratory insufficiency is the prerogative of the physician; however, the decision as to whether respiratory disability is present has often passed, rightly or wrongly, into the hands of a layman. Disability awards should be based on a complete medical evaluation and, more importantly, on the claimant's mental and physical ability to perform his job. In this context it is essential to remember that a laborer must be judged disabled if he cannot undergo the training or does not have intellectual capacity to enable him to do a desk job, even though he has sufficient pulmonary reserve to enable him to perform sedentary work. Similarly, an individual with moderate respiratory impairment can be regarded as disabled only if his job involves heavy manual labor. Were his occupation to entail mental work only, with little or no physical activity, then a moderate degree of dyspnea on exertion would not impair his working capacity. It cannot be stressed too strongly that respiratory impairment and respiratory disability are not necessarily related.

Many subjects can be shown to have marked respiratory impairment i the complete absence of disability, while others complain unceasingl of dyspnea of such severity that their activities are grossly restricted an yet may have little or no evidence of pulmonary impairment.

The sensation of dyspnea often correlates poorly with tests of pu monary function. In obstructive airways disease, it can be shown th there is a fairly good relationship between those tests that measure, e ther directly or indirectly, an increased airways resistance (the maxim voluntary ventilation, forced expiratory volume) and the presence c dyspnea. In restrictive disease this relationship is not evident. It ha been suggested that the presence of dyspnea is related to the work c breathing, and indeed there is considerable evidence in favor of thi contention. Nonetheless, there are many facts which cannot be ex plained by this hypothesis, in particular, the dyspnea experienced by subject on a ventilator, where the work of breathing is nonexistent Campbell and Howell have suggested that length tension inappro priateness provides a better explanation for the sensation of dyspnea.[9] This theory postulates that dyspnea is experienced when there is ar imbalance between the demand of the respiratory center for ventilatio and the actual ventilation produced; in short, the response is inappro priate or inadequate for the stimuli received. At the present time the lat ter hypothesis provides the best explanation for the sensation of dysp nea, but even so, much remains to be learned about this problem. Provided the above facts are borne in mind, it becomes useful to try to characterize the types of respiratory impairment that are found in the pneumoconioses.

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