The effective operation of these accessory muscles depends essentially on the *position* of the patient. When decubitus is dorsal the muscles of the back, when lateral those of the undermost side, act but feebly; the patient therefore instinctively assumes such a posture as is best adapted for the vigorous contraction of the most powerful of the inspiratory muscles or which permits of the co-operation of the greatest number of them. Accordingly the position of the dyspnœtic patient,—excepting those cases in which he is not perfectly conscious, and does not therefore select the most comfortable attitude,—furnishes us with a useful indication of the greater or less gravity of the impediment to the respiration. Thus, the obstacle cannot be one of any very great magnitude if the patient habitually lies on his back; with every aggravation of the difficulty of breathing he assumes a higher position, and ultimately has to remain almost constantly in the sitting posture (*orthopnœa*). In the most intense forms of dyspnœa, such as occur in the asthmatic attacks connected with old-standing emphysema, in diseases of the heart associated with ascites, hydrothorax, &c., the patient is sometimes unable to sit in bed, but is obliged to pass many sleepless nights in a half-standing position or in an arm-chair.

The semi-erect posture is often adopted even when the interference with the process of respiration is not of a very serious character, especially if the affection of the lungs or air-passages is attended by a profuse fluid secretion; in this position the accumulated matters are more easily expelled on account of the greater assistance which the muscles of expiration are then able to render.

The exact seat of the obstacle to respiration has also considerable influence in determining the habitual attitude of the patient. If it involves *one side* of the chest only, as in pleurisy with effusion, it is found that breathing is more easily performed when he lies on the *affected* side, as the muscles of the sound side are thus left completely unencumbered, and the healthy lung is at liberty to dilate to its fullest extent and so to partially compensate for the disablement of the other lung. Should the patient turn to the sound side the dyspnœa is at once greatly augmented, for not merely does the hindrance to muscular contraction on the affected side continue as before, but

on the sound side also the action of the chest is hampered, and the weight of the fluid pressing on the healthy lung still further reduces its available respiratory surface. On the other hand, at the *commencement* of painful unilateral pleurisy the patient generally elects to lie on the unaffected side, as resting on the diseased side increases the pain. For the same reason when there is a painful condition of *one side* of the chest arising from other causes decubitus is usually on the sound side. These observations regarding decubitus apply only to those diseases which are characterised by the severer forms of dyspnœa; in milder cases the patient reclines indifferently on either side, according to habit.

When the function of respiration is in any way impeded, increase in frequency and in depth are the most common changes that the act of breathing undergoes. But in some rarer cases respiration is not at all accelerated, or is even somewhat *slower* than in health; inspiration, however, is then always prolonged and much more full. This type of respiration is observed in two conditions:—

1. In stenosis of the larynx or trachea, acute or chronic.
2. In lung diseases which are followed by affection of the brain.

The first condition is best illustrated by laryngeal croup—in children the most frequent cause of laryngeal stenosis; in it inspiration is prolonged, slow, and deep, and accompanied by a peculiar crowing noise (*stridor*) which is audible even at some distance. This retardation of the respiration is due to the *constriction of the rima glottidis* which must always occur on the deposit of croupous exudation above and below the glottis, but which may also take place independently of the presence of the croupous membrane, from inflammatory swelling and consequent partial paralysis (paresis) of the muscles which govern the vocal cords, especially those which relax them. It is thus only by dint of very violent effort on the part of the dilators of the glottis that the passage of air is permitted, so that *inspiration becomes laborious and deep;* superficial, though frequent, respiration would be of little avail under such circumstances. Further, in those cases in which the vocal cords are covered with exudation *expiration* also is obstructed and prolonged; and as both inspiration and expiration thus occupy a longer period respiration as

a whole is slower. Section of the recurrent laryngeal nerve in animals produces narrowing of the glottis and gives rise to the same phenomena—diminution in the frequency of respiration and deepening of each individual inspiration.

This explanation of the cause of retardation of the respiration is applicable to most cases of stenosis of the larynx, even to those in which expiration is *not* obstructed. Thus, if a patient suffering from paralysis of both posterior crico-arytenoid muscles, in which there is only *inspiratory* contraction of the glottis, should breathe rapidly he would be devoting as much time to expiration (which is free) as he does to inspiration (which is difficult); he instinctively prefers, therefore, to protract inspiration at the expense of expiration.

Experiments on animals furnish the true explanation of the slowness of respiration in the second variety of cases, those which are complicated by the occurrence of brain disease. It is well-known that the respiratory centre is situated in the medulla oblongata, close to the point of origin of the vagus nerves. It is through these nerves that the respiratory impulse is excited and the rythmical character of the movements sustained; when they are divided in the neck the frequency of respiration sinks considerably, sometimes to even one-third its normal rate per minute, while each individual inspiration becomes exceedingly deep and is accomplished only by great muscular exertion; and if the respiration of the animals so operated on be still further embarrassed, as by puncturing one side of the chest or by injecting fluid into the pleura till one lung is fully compressed, and the functionally active lung-surface in that way suddenly reduced by one-half, the breathing nevertheless remains slow and quiet as before. Similarly in men, respiration, notwithstanding the existence of a considerable obstacle to its proper performance, may be unduly retarded when the normal stimulating influence of the vagi is withdrawn, an accident which is apt to happen when their medullary roots and the adjacent parts of the brain are compressed by fluid exudation, extravasation of blood, and other changes in the basis cranii. This same symptom, prolonged and deep inspiration, is observed also in limited regional brain diseases [Heerderkrankungen] affecting even parts distant from the vagus,—in the coma which follows apoplectic seizures, and in circumscribed softening.

When the vagus is compressed by tumours in the neck, or wounded during operation or by gunshot, respiration becomes less rapid, though not to such a marked degree as when the nerve is divided in animals. I have elsewhere recorded a case of this nature, in which the respirations were 12 per minute and inspiration was abnormally deep, dependent on paralysis of the vagus from diphtheritis of the fauces.

There is a special form of dyspnœa, known as *Cheyne-Stokes' Respiration*, which is met with in some diseases of the heart and brain, and in certain other affections in the course of which cerebral complications arise. It consists in a regularly-occurring pause, lasting ¼—1 minute, during which respiration is completely suspended, and which is preceded by the following characteristic train of dyspnœal phenomena: inspiration, which is at first short and shallow, becomes gradually deeper, and finally markedly dyspnœal; when this dyspnœa has reached the acme of its severity respiration becomes again more and more superficial with each successive inspiration, and eventually comes to an absolute standstill. When the pause has continued ¼—½ minute these symptoms are repeated in exactly the same way. In the most pronounced cases the dyspnœal period, during which the respirations number about 30, occupies ½—¾ of a minute, and the pause is of nearly the same duration. The whole cycle of phenomena therefore takes up 1—1½ minutes; in less marked cases it lasts only about ¼ minute, and may easily be overlooked, especially when the pause is very short. This variety of dyspnœa rarely occurs in the early stages of any disease, usually appearing only a few weeks, days, or even hours before death; it is, therefore, with but few exceptions, a fatal indication.

Stokes' respiration is observed in a great many different affections, relatively most often in those diseases of the brain which give rise to compression in the neighbourhood of the medulla oblongata,—hæmorrhage, exudation, œdema, tumours, uræmia,—in certain cardiac diseases, such as fatty degeneration, sclerosis of the coronary arteries, stenosis of the aortic or mitral orifices, &c. As all these conditions have one common feature,—diminution of the arterial blood supply, and so of the supply of oxygen, to the brain or respiratory centre in the medulla oblongata,—Traube has assumed it as the starting point of his explanation of Stokes' phenomenon. His theory is that *the irritability of the respiratory centre is* so materially *lowered* by the inadequacy of the supply of oxygen that a much larger accumulation of carbonic acid in the blood than is usual is necessary for the excitation of the inspiratory impulse, that thus the time required for the taking up of the requisite quantity of carbonic acid from the tissues is longer, and that therefore the intervals between each inspiration are

more protracted. In opposition to this hypothesis, Filehne, founding his views on some observations made on animals (which, when strongly narcotised, breathe in the above-described fashion) and on men, has shown that though the irritability of the central organ of respiration is indeed diminished, this alone is not sufficient to produce Stokes' respiration, but that it is further essential that it be less easily excited to action than the vasomotor centre. The symptoms are caused therefore in the following way: at the end of the respiratory pause there is a large disappearance of oxygen from the blood, carbonic acid has accumulated, the vasomotor centre is thereby stimulated, and the arteries (the cerebral arteries among the rest) at once contract; this produces a gradually-increasing anæmia of the respiratory centre, and inspiration becomes more and more deep; this, however, supplies the wanted oxygen to the blood, the arterial spasm is relieved, the anæmia of the respiratory centre passes off, and with it the exaggerated impulse to respiration, and breathing once more becomes superficial. When the arterial spasm has entirely subsided, so that the respiratory centre is abundantly provided with decarbonised blood, the stage of apnœa, the pause, is reached, and lasts till, by the abstraction of oxygen from the blood, the irritation of the vasomotor nervous centre is renewed and the whole series of operations again gone through. That the arteries are strongly contracted is proved by the increase of the arterial tension and of the blood-pressure, while the anæmic condition of the brain is demonstrated by the fact that in young children, at the end of each respiratory pause, immediately before the re-commencement of respiration, and also while inspiration is gaining in depth, the great fontanelle is depressed. And further, this form of dyspnœa may invariably be arrested at the very commencement of each seizure by the inhalation of nitrite of amyl, which dilates the vessels. We are thus able to produce experimentally in animals all the phenomena which go to form Stokes' type of respiration; we can diminish or even cut off the blood supply of the brain, and so set up a gradually-increasing dyspnœa, and we can then re-establish the circulation and in that way reduce the dyspnœa or even arrest respiration.

Spirometry.

(The estimation of the vital capacity of the lungs.)

By the term *vital capacity* is understood the greatest volume of air that can be inhaled by the fullest inspiration after the most forcible expiration; it must not be confounded with the term *total capacity*, which means the vital capacity with the addition of that quantity of air which still remains in the lungs even after the most complete expiration. The latter, the residual air, is displaced only when the lungs collapse on opening the thorax.

The vital capacity of the lungs may be most conveniently measured by means of Hutchinson's *Spirometer*.

This instrument consists of two cylinders, the outer of which is open above and filled with water, on which the inner one floats: the inner cylinder is open at the lower end, and properly balanced by weights placed on its upper closed end. A piece of elastic tubing, through which the patient is directed to breathe, is connected with the larger outer cylinder. In expiration the inner cylinder rises, in inspiration it sinks, and the distance through which it moves is registered on a scale graduated to show the number of cubic centimeters of air inhaled or expelled. It is seldom that the apparatus is used for determining the volume of air taken into the lungs, inspiration from the cylinder through the tube being somewhat unpleasant. To measure the amount of expired air the receiver is lowered so as to cause the index to point to a low figure on the scale, the nostrils are closed, a deep inspiration is drawn, and the air driven from the lungs through the tube and into the cylinder; the latter is buoyed upwards by the air which enters it, and remains fixed on the completion of expiration. If, for example, the index has advanced on the scale from 2,000 ccm. to 5,000 ccm., the vital capacity of the individual is said to be 3,000 ccmtr.

The vital capacity of the lungs of healthy persons varies considerably,—from 3,000 to 4,500 or 5,000 ccm. in men, and from 2,000 to 3,000 ccm. in women. Besides sex the elements whose variation seems to influence it most are height and age. It increases at the rate of 60 ccm. for every centimeter of stature above 155; thus if the vital capacity which corresponds to a height of 162 cmtr. be 3,000 ccm., that associated with a height of 167 cmtr. should be 3,300 ccm. Its relation to age is seen in an increase of about 160 ccm. from the fifteenth to the thirty-fifth year, and a decrease of about 900 ccm. from the fortieth to the sixty-fifth year; it is further very low in the extremely aged and in young children. Sedentary occupation and deficient nutrition lessen, the opposite conditions augment, the vital capacity; it also undergoes diminution in the sitting posture, and when the stomach is distended with food. The average in men of 20—40 years of age and of medium height may be assumed to be 3,600 ccm., in women 2,500 ccm.; in shorter individuals it may be 3,000 or less, while in tall persons it may mount to 4,000 or more.

The vital capacity is *diminished* in all diseases of the respiratory organs in which the expansibility of the lungs is interfered with; in the later stages of such affections it may sink to half

its normal amount, or even lower. This fall is dependent on, but is not exactly proportionate to, the extent of breathing surface encroached upon; thus, if half of the respiratory area is disabled, as by compression of one lung by pleuritic effusion, it does not follow that the vital capacity will be similarly lowered by one half, as under such circumstances the healthy lung expands more freely, and so compensates somewhat for the loss of function in the affected lung.

Like every method of examination which involves the use of large or unwieldy instruments spirometry has never come to be regularly employed as an aid to diagnosis in the diseases of the thoracic organs, more especially as the data so furnished are, as a rule, less trustworthy than those easily obtainable by other means. In those cases also in which physical examination alone is sufficient to make it perfectly clear that the capacity of the lungs is diminished,—as in pleurisy with copious effusion, advanced emphysema, &c.,—it is superfluous to demonstrate that fact further with the spirometer. Spirometry is thus most useful when the decrease in the vital capacity is so trifling as to escape detection by other tests,—as when infiltration is beginning in the apices of the lungs; even then, however, it is scarcely to be expected that so slight an organic change should very markedly hamper the breathing, as complete infiltration of one apex lowers the vital capacity by little more than 100 ccm. Further, the results may vary to the extent of 100 ccm., according as the operation is performed with due care and attention to all essential details or otherwise; even persons practised in the use of the apparatus do not always succeed in raising the index to the same point. The vital capacity also differs so widely in individuals of the same height, age, and sex that, unless the usual and normal capacity of the patient under examination be known, one is warranted in making pathological deductions from it with confidence only when it falls decidedly below the physiological minimum. Finally, when the full dilatation of the lungs is from any cause prevented (as by pain), when respiration is abnormally frequent, or when the patient is very weak, spirometry can scarcely be said to be available as a means of diagnosis.

Nevertheless comparative spirometric observation, carried on for some time and often repeated,—the above-named sources of

error being excluded,—comes to be of considerable value, as indicating the progress of the disease (whether improving, stationary, or being further developed).

The vital capacity is *increased*, in health as well as in disease, by *residence in elevated regions* and by the use of certain *pneumatic apparatuses*,—by *inspiration of compressed air*, which produces greater expansion of the lungs, and by *expiration into rarefied air*, which permits of the more complete emptying of the lungs: in the latter case a portion of the residual air also is expelled, the total quantity of air displaced is greater than usual, so that the lungs are made to dilate more fully in the following inspiration.

The most widely-known pneumatic instrument is the portable apparatus introduced some years ago by Waldenburg, which is so constructed that the patient can at will inhale or expire into condensed or attenuated air. For therapeutical purposes the vital capacity may be augmented by expiration into a chamber the pressure within which is 1-60th, 1-40th, or 1-30th, less than that of the atmosphere, or by inspiration of air whose density is 1-60th—1-30th greater than that of the atmosphere. Inspiration of rarefied air does not add to the vital capacity, but it stimulates the inspiratory muscles, making them act more energetically, and in that way overcome the difficulty experienced in breathing in such circumstances. Expiration into compressed air does not affect the vital capacity, and is therefore not resorted to in the treatment of disease. Though the volume of air set in motion in each act of respiration may thus be increased either by expiration into rarefied air or by inspiration of condensed air, these two methods of accomplishing that object cannot be used indifferently. In pulmonary emphysema, for instance, in which the alveoli are abnormally distended and inelastic, taking almost no part in the movements necessary for the expulsion of air from the chest, the deficiency is chiefly in the expiration; here the *vital capacity should be increased by expiring into an atmosphere of less than the normal pressure*, as by this means the *contraction* of the vesicles themselves is favoured and a much larger volume of air (1,000—2,000 ccm. more) given out than in the most forcible expiration under ordinary conditions: inspiration of compressed air, however, would only have the effect of still further stretching the walls of the pulmonary vesicles and so of reducing the dyspnœa (for which purpose, indeed, it is sometimes practised) but not the emphysema. When, on the other hand, the lung has become partially consolidated by compression (as in cases of pleuritic effusion), in stenosis of the air-passages, and in phthisis, the lowering of the vital capacity is owing to the inability of certain parts of the lungs to expand sufficiently, so that the defect is principally in inspiration; here it is evident that the most appropriate method of treatment is to *promote the expansion of the lungs by the inhalation of condensed air*.

The benefit resulting from these proceedings is not of a merely temporary character, lasting only so long as the apparatus is in use; the changes are frequently permanent, especially if the treatment be persisted in for some time and the disease be not of a kind involving the destruction of tissue. Thus there are numerous instances on record in which pulmonary emphysema has been much diminished, in less aggravated cases actually removed, by expiration into rarefied air continued for weeks and months; not only has the dyspnœa been abolished and the vital capacity greatly augmented (sometimes nearly doubled), but the lower margin of the lungs has also been observed to rise gradually to its normal level in the thorax. Equally favourable are the results of this pneumatic treatment in chronic bronchial catarrh, and in bronchial asthma (uncomplicated, or associated with emphysema or catarrh). In the early stages of phthisis also the capacity of the lungs may be permanently increased by the inspiration of compressed air.

Pneumatometry.

To Waldenburg also belongs the credit of having been the first to apply the estimation of the force exerted in respiration,—an operation which, till his time, was regarded as of merely physiological interest (Valentin, Donders, Hutchinson, &c.),—to the diagnosis of diseases of the lungs. The manometer in most common use consists of a glass tube open at both ends, bent double, and supported on a wooden stand; each vertical limb is about 270 mm. high and is half-filled by a column of mercury, the top of which marks the point assumed as zero. One limb is turned horizontally at its upper end and is inserted into a caoutchouc tube; this is connected with a mask which fits closely on the face, absolutely excluding the air from the mouth and nose. On drawing breath through the caoutchouc tube the column of mercury rises in that limb of the manometer nearest to the patient and sinks in the other, movements which are, of course, reversed in expiration. The extent of the fluctuation is read-off on millimeter scales, of which there are two, one on each side of the instrument, placed on the glass tubes, and reaching above and below the zero points. Obviously the numbers so obtained must be doubled, as the quicksilver descends in the one limb exactly the same distance that it ascends in the other.

It has been abundantly proved by the researches of the above-mentioned observers that in healthy persons the *power* put forth in *expiration exceeds that of inspiration*, usually by 20—30 mm. on the scale just described. The following table gives more nearly the limits within which the pressure of forcible inspiration and expiration may vary in health:—

	Inspiration. Mm.	Expiration. Mm.
In moderately strong men ...	... 70—100	80—130
In very powerfully-built men	... 120—160	150—220
In women	... 50— 80	60—110

These points on the scale are reached only when the movements of respiration are executed rapidly and with the utmost exertion of the respiratory muscles; the mercury is then maintained at its maximum height for scarcely a second, and falls again immediately. When inspiration and expiration are performed slowly the manometer indicates a much lower pressure, and the mercury remains longer at its highest point, oscillating slightly upwards and downwards. For practical purposes the former method recommends itself to the investigator, as we thereby ascertain the positive maximum of inspiratory and expiratory power the patient is capable of exerting.

As these pneumatometric signs have such a wide range even within the bounds of perfect health, it is important to know at what point they overstep these limits and acquire a pathological significance. Generally speaking it may be inferred that an *absolute* diminution of pressure exists in those cases in which it is decidedly below the physiological minimum; further, in largely-built individuals the physiological *mean* should be taken as the standard, any downward deviation from which is to be regarded as indicating disease; above all, however, the comparison of the inspiratory with the expiratory manometric pressure furnishes the best guide to a decision in this matter.

The diseases of the respiratory organs may be divided into two principal pneumatometric types: in the first the conditions of health are reversed, the positive expiratory pressure being less than the negative inspiratory force, while the latter may be somewhat increased, normal, or subnormal; in the second the negative inspiratory pressure is lower than the physiological minimum, while the positive expiratory force may be normal or subnormal, in the latter case being still slightly greater than the inspiratory force. To the first class, that in which *the expiratory pressure is diminished*, belong *pulmonary emphysema* (the lowering of pressure being here due to diminution in the elasticity of the lung-tissue), *bronchitis* and *nervous asthma*. In the second class, in which it is particularly the *inspiratory power* that is lessened, are found *phthisis pulmonum* (the manometric difference being observable here even when the disease is of but slight extent), *stenosis of the larynx and trachea*, and generally those diseases of the respiratory organs which offer increased resistance to the expansion of the lungs (pneumonia, pleurisy); in the more advanced stages of such cases the expiratory pressure may also be decreased. Among diseases of the heart mitral lesions most usually produce diminution of expiratory power, the result of the consecutive hyperæmia of the lungs (Waldenburg). Tumours of the abdominal organs (pregnancy, effusions, &c.), inasmuch as they impede expiration rather than inspiration, tend chiefly to lessen the expiratory force (Eichhorst).

It is worthy of notice that in comparing the pneumatometric with the spirometric signs, in health and disease, we find no evidence of any fixed relation between respiratory pressure and vital capacity; the former may be high while the latter is low, and *vice versa*.