Investigations carried on by means of the double stethograph show that under normal conditions inspiration is generally somewhat shorter than expiration, and that between them there is no pause,—that inspiration, having reached its termination, is at once, though almost insensibly, followed by expiration; and further, that the pause between any two respirations is exceedingly short. All parts of the thorax are not engaged in the same stage of the respiratory act at a given moment; inspiration may be completed at one point but only begun at another, or in one part expiration may be only ending when in another inspiration has already commenced. There may be absolutely no quiescent interval when respiration is much accelerated; on the other hand, it is well marked when the breathing is slow. A comparative examination of the tracings also shows that in men both sides of the chest, all the intercostal muscles, and the diaphragm, are called into action simultaneously, and that the inspiratory elevation is equal on both sides; that in young persons not unfrequently the upper part of the thorax moves more than the lower (a condition which is reversed in old age); and that in women the range of movement of the parts of the thorax decreases from above downwards.

When pathological changes take place in the respiratory apparatus the stethograph gives much the same indications as simple inspection, but usually of a more striking character; thus the slight degree of elevation shown in the inspiratory tracing of a partially or completely collapsed lung presents a marked contrast to the high curve of the sound lung. Obviously the tracings are not diagnostic of the various causes of collapse of the lung (pleuritic exudation, pneumothorax, pneumonia, phthisis, &c.), as they show merely that the respiratory movements are restricted; the greater the collapse the deeper the curve. Parts which sink in inspiration and become prominent in expiration give a reversed tracing, the inspiratory line descending the expiratory line ascending. The most characteristic tracings are those of stenosis of the larynx and trachea, and of emphysema of the lungs. In the first the expiratory curve is normal, while the inspiratory curve is much prolonged, corresponding to the long duration of inspiration produced by the blocking up of the air-passages; in emphysema it is the expiratory line which is the longer, as it is the escape of air from the chest which is most impeded. In severe and extensive emphysema the expiration is somewhat irregular, being relatively rapid in its first two-thirds; the obstacle to the expulsion of the air is then suddenly encountered, when, in spite of the powerful action of the respiratory muscles, the rest of the expiration is accomplished slowly and with difficulty. This is all very clearly shown in the tracing, the accession of the hindrance to expiration being indicated by a sharp angle. In chronic catarrh this phenomenon is quite absent, expiration being uninterrupted; it is therefore graphically distinguishable from emphysema.

Frequency of the Respiration.

This, in health, amounts to 14—18 per minute in adult men,

to a somewhat larger number in women and children, and to 40 or more in the new-born. One respiration usually corresponds to four beats of the pulse. Position has but little influence on the frequency of the respiration, though it is rather faster when sitting or standing than when lying. The most extensive control is exercised over it by the will,—it may be voluntarily deepened or made superficial, accelerated or retarded, or even arrested for 30—60 seconds. It goes on most quietly and regularly when Will and Perception are in abeyance, as in sleep.

Of the deviations from the normal frequency acceleration is more common than retardation. The rate may increase to 70, 80, or even to over 100 per minute; generally, however, it rises no higher than 40. Abnormal rapidity of respiration is called *dyspnœa*. Respiration is sometimes not merely quickened, but each inspiration may gain considerably in depth; at other times respiration is simply fuller, while its speed is scarcely increased, or may even be diminished. The latter condition also is known as dyspnœa.

Respiration is physiologically accelerated by physical exertion, such as rapid walking, running upstairs, and generally by all those circumstances which tend to increase the action of the heart. Those who are convalescent from severe acute diseases, therefore, begin to breathe more quickly as soon even as they sit up in bed, as the heart is thereby excited to more powerful and rapid contraction; the same effect is produced in some patients by mental depression or even by the knowledge of the fact that they are being observed by others. All these disturbing influences have to be carefully weighed or excluded when we wish to determine the presence or absence of abnormal frequency of respiration, a symptom which, even when but slightly developed, has always great pathological significance.

Acceleration of the Respiration takes place as a *pathological phenomenon* in the following circumstances—

1. When there is *pain* in any part of the thorax or abdomen to which the movements of respiration are communicated. The patient breathes hurriedly but superficially, in order to avoid adding to his suffering by causing disturbance of the parts affected,—a condition of which the onset of pleurisy affords a good example. Painful affections of the ribs and of the muscles of the thorax (as in acute muscular rheumatism) are, in the same way,

associated with increased frequency of respiration, to but a slight extent, however, when the pain is located in the investing (pectoral and dorsal) muscles, most markedly when the proper respiratory muscles (the intercostals and diaphragm) are implicated.

In one case of acute rheumatism of the entire muscular system of the chest I found that the respirations numbered 40 per minute; in another case, in which the symptoms seemed to point to inflammation of the diaphragm, the respiration was over 50 per minute, while the diaphragm remained almost motionless, the slightest attempt at deep inspiration being cut short by agonising pain.

The breathing of those who are suffering from acute pain in the abdomen (as in diffuse peritonitis) is exceedingly shallow, and therefore very rapid, as the contraction and downward movement of the diaphragm and the consequent displacement of the abdominal organs are thus reduced to a minimum.

2. Increased frequency of respiration is an unfailing concomitant symptom in all *febrile conditions* of any considerable degree of intensity, from whatever cause they may arise; it does not, however, increase proportionately to the severity of the fever. In very high fever the number per minute may reach 30—40, in children 50—60, without necessarily implying the existence of any lung complication. This is attributable to various causes,—to the acceleration of the pulse and the greater velocity with which the blood circulates through the lungs, to the augmentation of the quantity of carbonic acid contained in the blood, but most of all to the abnormal elevation of temperature. Through those influences the respiratory centre in the medulla oblongata is more often and more powerfully stimulated and the reflex movements of respiration are more frequently excited.

That the rapidity of the breathing in fever depends more on the temperature of the blood than on the rate of the pulse is proved by the facts that many cases occur in which a high degree of heat and a relatively slow pulse are associated with acceleration of the respiration, and that, on the other hand, there are cases in which respiration becomes gradually slower as the temperature is reduced by the use of cold baths, the pulse-rate remaining unchanged. Dyspnœa from this cause (*heat dyspnœa*) is the result of *direct* irritation of the respiratory centre, as has been shown by numerous experiments on animals, in which the blood, as it passed through the carotids, was artificially warmed.

3. The most marked increase in the frequency of the breathing takes place in those diseases in which *the interchange of gases* that should go on *in the lungs is disturbed*, diseases whose seat may be either in the respiratory organs themselves or in other parts. In the former case the dyspnœa arises from the blocking up of some of the avenues by which the air reaches the breathing surface, either by *diminution of the calibre of the air-passages* or by *conditions which render the pulmonary vesicles impermeable*.

The cases in which the air-canals are *narrowed* may be divided into two classes, those in which the obstruction is situated in the larynx or trachea, and those in which it occurs in the larger bronchi or their finer subdivisions. To the former group belong œdema glottidis, croup, diphtheritis of the larynx, and thyroidal tumours which compress the trachea; the latter group includes especially catarrhal swelling of the bronchial mucous membrane, which, when it is extreme, affecting a large number of the finest air-tubes, produces all the effects of constriction higher up—in the larynx or trachea.

The *alveoli* may become *impervious* from *infiltration* with plastic or fluid exudation (in pneumothorax, cheesy degeneration, œdema of the lungs), or from compression (by fluid, air, tumours in the pleura, &c.), or from loss of elasticity to such an extent that they do not sufficiently expand in inspiration nor adequately contract in expiration (as in vesicular emphysema). When, from one or more of the above-named causes, the air-cells are closed the decarbonisation of the blood is interfered with, the blood becomes deficient in oxygen and contains an excess of carbonic acid, and the respiratory centres are more frequently and more strongly excited to action. The degree of the dyspnœa depends on the more or less complete occlusion of the alveoli and on the area involved in the changes which lead to this result; with certain reservations it may be said that, other conditions being the same, it increases with the magnitude of the obstacle to respiration, that is, with the diminution of the available breathing surface. As a rule it is the more marked the more suddenly the respiratory surface is reduced; thus, in collapse of one lung from pneumothorax, developed within a few hours, it reaches a high degree of intensity; but should the collapse be brought about by the pressure of a slowly-accumulating pleuritic effusion, the dyspnœa is less considerable.

When, as in pleuro-pneumonia, pain and fever co-operate with the cause just described in accelerating the respiration, the rate shows a very rapid increase and may soon rise to 40 or more per minute; on the other hand, certain varieties of chronic consolidation of the lungs, inasmuch as their course is painless and free of fever, are accompanied by much less disturbance of the respiration than pneumonia, even when they disable a larger portion of the lung. I have seen cases of complete atelectasis of an entire lung, produced by pleural effusion of long standing, in which the respirations were not more than 24 per minute nor appreciably increased in depth.

In chronic affections in which respiration is obstructed its frequency varies greatly, particularly according as the body is well or ill nourished. Thus in phthisis pulmonalis, even when it is far advanced and the breathing surface extensively destroyed, respiration is often not at all hurried, because, on account of the emaciation, *the quantity of blood in circulation is decreased* and a much smaller volume of oxygen suffices for its decarbonisation. In those destructive processes, however, which are not attended by wasting, such as bronchiectasis, breathing is usually greatly accelerated. Respiration becomes exceedingly rapid also when a new obstacle is suddenly added to one of older standing. Emphysematous patients feel tolerably comfortable as long as their shortness of breath is due merely to inexpansibility of the air cells; but should a diffuse bronchial catarrh supervene or should the original disorder be seriously aggravated by cold weather, their dyspnœa immediately becomes very distressing.

The frequency of respiration is further increased by certain *diseases of the heart*, especially by those *valvular affections* which give rise to *overfilling of the pulmonary circulation* and thus to interruption of the free interchange of gases in the pulmonary capillaries. Among such conditions are insufficiency of the mitral valve and stenosis of the mitral orifice; in the first case a quantity of the blood in the left ventricle passes backwards into the auricle at each systole, in the second the free flow of blood from the left auricle is prevented. Under these circumstances the left auricle, and subsequently the pulmonary capillaries and veins, become engorged, the necessary consequence of which is that the blood is insufficiently arterialised in the lungs, the respiratory centre is more powerfully stimulated, and the movements of respiration more frequently repeated. A second, but less important, element in the production of dyspnœa in mitral disease is the occurrence of bronchial catarrh from the passive congestion of the circulation through the lungs. Those heart affections in which the pulmonary circulation is intact, such as diseases of the aortic valves, usually produce at first no dyspnœa; but in their later stages, when the left ventricle undergoes fatty degeneration and is no longer able to propel the whole of its contents into the aorta, and so fails to accommodate the whole of the blood which should enter it from the left auricle, the pulmonary vessels are overloaded and dyspnœa is established.

Slight dyspnœa, however, is set up in aortic disease, from the increased rapidity of the heart's contraction, and when the hypertrophied and dilated left ventricle encroaches on the neighbouring parts of the lung, causing them to shrink and so diminishing their respiratory surface. The dyspnœa attendant on large aneurisms of the aorta may be explained in the same way, though in this case it often arises from pressure on the trachea. Disease of the tricuspid valve or of the valves of the pulmonary artery very seldom occurs alone, but is usually combined with aortic or mitral disease, so that dyspnœa in these cases is of a somewhat complex origin.

Increased rapidity of the respiration may be due to diseases of the abdominal organs in which the abdominal cavity is distended, the diaphragm being pressed upwards into the chest and its movements in this way limited. Dyspnœa is thus frequently observed in cases of ovarian tumour, ascites, and tympanites, especially when the patient assumes the dorsal position. The greater the number of these exciting causes which coexist in any given case the more marked is the dyspnœa; it takes probably its most agonising form in the later stages of mitral disease, when ascites, hydrothorax, and pericardial effusion make their appearance together.

Not unfrequently we have dyspnœa proceeding from no obvious cause, or at least from no cause which corresponds to it in apparent importance. In most of these cases it is owing to a temporary diminution in the calibre of the bronchi, from spasmodic contraction of the bronchial muscles; it is therefore named *bronchial asthma*. This variety of dyspnœa occurs in paroxysms of relatively short duration, while in the intervals, when the case is not complicated with other affections, especially bronchial catarrh, respiration may be almost normal.

The theory that these dyspnœal attacks are referable to bronchial spasm is well supported by experimental evidence. Irritation of the vagus nerves in the neck is followed by *contraction of the unstriped bronchial muscles*, and therefore by diminution of the lumen of the bronchi; its effect also on the contractility of the lungs is indicated by the rising of a column of fluid in the tube of a water manometer attached to the trachea (Donders, Bert, Gerlach, &c.). It is especially the finer and smallest air-tubes which take part in this spasm, though it is not impossible that the larger bronchi may likewise be affected, as the trachea of animals has been shown to contract on electrical stimulation (Horwath). It has not yet been definitely ascertained whether the alveoli themselves also shrink on irritation of the vagus. The bronchial catarrh which almost always accompanies asthma has manifestly but an inconsiderable influence in causing the dyspnœa, as the latter occurs in paroxysms which, when they subside, are succeeded by an interval in which the breathing is easy and natural, this remission being altogether independent of any change in the catarrhal symptoms. In other cases, more rare than those just described, dyspnœa may be periodical and of great severity, while not the slightest morbid alteration may be found in the respiratory or circulatory organs. It may be assumed that these phenomena are caused by some transient irritation of the vagi, though the particular circumstances in which this irritation is excited are still unknown. In the case of a young girl, whose internal organs were perfectly healthy, I have seen the respiration become as rapid as 40 per minute, and sink again in a few days to the normal rate. For many years I have had under observation another case, that of a woman, whose organs of respiration and circulation have remained perfectly intact, but who is subject to extremely severe paroxysmal attacks of dyspnœa, accompanied by a feeling of intense anxiety, during which the frequency of the respiration increases to 70, 80, sometimes almost to 100 per minute; the violence of the seizure then gradually abates, and respiration again becomes slow and quiet. These attacks sometimes take place daily, at other times they are absent for months together. Bischoff has recorded a somewhat similar case; the breathing, during the paroxysms, was greatly increased in rapidity, sometimes to 160 per minute, but in the intervals was perfectly normal.

From *objective* dyspnœa is to be distinguished a *subjective* variety. Many persons complain of occasional shortness of breath, especially those who have a sense of weight or pain in the epigastrium from diseases of the stomach or from other causes, those who have a feeling of oppression in the cardiac region, hysterical individuals and others, in whom no trace of any physical condition which might explain this subjective dyspnœa can be detected. While it lasts no dyspnœa is objectively apparent, or the patients, while breathing superficially but at the normal rate, only now and then draw a full, deep inspiration,—a state of matters very frequently met with even in perfect health.

Genuine dyspnœa is distinguished by another peculiarity, namely the *greater depth* and therefore the *longer duration*, of each respiratory act. As a rule the depth is inversely proportionate to the frequency. Thus, in the severe acute diseases of the respiratory organs (such as pneumonia) the breathing is rapid but not appreciably increased in depth,—the respiratory muscles being evidently unequal to a task demanding so much exertion, as, in common with all the muscles of the body, they participate in the general emaciation caused by the high fever. On the other hand, in those chronic lung diseases in which the physical strength is not reduced,—vesicular emphysema for instance,—respiration is often very deep and full while the increase in its frequency is but slight. There are *two forms* in which this greater depth or longer duration of respiration may manifest itself,—it may affect principally the *inspiration* or the *expiration*. Prolonged inspiration, *inspiratory dyspnœa*, is observed when the air enters the lungs with difficulty, as in stenosis of the larynx and trachea; its most typical and simple form is seen in paralysis of the posterior crico-arytenoid muscles, the margins of the glottis in that rather rare affection being approximated to each other in inspiration and relaxed in expiration,—the reverse of the normal movements. Prolonged expiration, *expiratory dyspnœa*, occurs when the escape of air from the lungs is in any way impeded, as in diffuse bronchial catarrh associated with vesicular emphysema, though in these circumstances the inspiration also is lengthened.

The greater depth of the respiration is the result of the increased action of the ordinary respiratory muscles assisted by that of the *accessory* muscles, the latter term including those which, though they take no part in the thoracic movements when the breathing is normal and quiet, contract powerfully when it is desired to expand the chest to the utmost.

The accessory inspiratory muscles in the neck are the *scaleni* and the *sternomastoids*. The anterior and middle scaleni raise the first rib, the posterior scalenus the second; these, however, especially in the female sex, are not entirely inactive even in

ordinary respiration. When the head is fixed the sternomastoids elevate those parts into which they are inserted,—the clavicles and sternum. Certain of the thoracic muscles also may be considered accessory muscles of inspiration.

The *pectorales* (major and minor), when the dyspnœa is very intense, raise the ribs (second to sixth) when the head and shoulders are fixed; it is by availing themselves of the action of these muscles that emphysematous patients, when suffering from an attack of asthma, seek to obtain relief in grasping firmly some object above their heads. The *serrati postici superiores* elevate the upper ribs; the *subclavius* has a similar action on the first rib when the clavicle is stationary; the *levatores costarum* (breves and longi) draw the posterior portion of each rib towards the vertebral column; the *levator anguli scapulæ*, that part of the *trapezius* which rises from the occiput and is inserted into the clavicle and acromion, and probably also the *serrati antici majores*, act as inspiratory muscles, inasmuch as they move the lower and middle ribs upwards and outwards when the shoulder is fixed. Finally, it has been proved by experiments on animals that in asphyxia the elevators of the head and spinal column aid in inspiration; in men it is only when suffocation is threatening, in croup and spasm of the glottis, that these muscles are called into action.*

While the above-mentioned muscles are to be regarded as *dilators of the thorax*, others, such as the *levatores alæ nasi* and the *levator palati mollis*, co-operate with them by enlarging the openings by which the air enters; the *sterno-hyoid*, *sterno-thyroid*, *thyro-hyoid* and *omo-hyoid* muscles also, by depressing the larynx, facilitate the admission of air into the lungs. The most important of the accessory muscles of this kind are the *crico-arytenoidei postici*; when respiration is at all embarrassed they at once contract powerfully, separate the arytenoid cartilages, and so dilate the rima glottidis.

In animals the various groups of accessory muscles of inspiration are brought into play in a certain order, according as the difficulty of breathing increases; thus Traube states that first the elevators of the upper ribs are thrown into violent contraction, then the scaleni, the elevators of the lower ribs, the sterno-hyoids and sterno-thyroids, and lastly, the serrati postici superiores. In the human subject no such order is observed, the additional movement is sometimes accomplished by one set of muscles, at other times by another. In general, however, when the dyspnœa is considerable, the cervical muscles (especially the scaleni and sternomastoids) are the first to come to the aid of the ordinary inspiratory muscles; when the dyspnœa is still more severe these are joined by the levatores alæ nasi, and in the worst cases by the muscles of the breast and shoulder-blade.

* One may easily, by simply fixing the arms, demonstrate on his own person the fact that these muscles are really instrumental in adding somewhat to the forces which dilate the chest.

Expiration, even when abnormally prolonged by the presence of diffuse bronchial catarrh or other obstacle to the emptying of the lungs, is usually effected by the elasticity of the lungs alone. When this elasticity, however, is much diminished, as in the advanced stages of emphysema, it is supplemented by the action of various muscles; the thorax is thereby as far as possible reduced in volume, and so made capable of greater expansion at next inspiration. The principal *expiratory muscles* are those of the abdomen; they compress the abdominal organs and thrust them upwards towards the diaphragm, the transverse diameter of the abdomen being shortened by the action of the *transversalis*, the long diameter by that of the *recti*. Other muscles assist in expiration by drawing the thorax downwards; the *obliqui* (external and internal) and the *triangularis* sterni depress the anterior part of the lower ribs, the *serrati postici inferiores* (antagonists of the serrati postici superiores) have a similar influence on the four lower ribs, and the *quadratus lumborum* on the lowest rib.

The existence of some difficulty of breathing may frequently be recognised at the first glance by the marked contraction of several of the accessory muscles of inspiration, such as the sternomastoid, scalenus, omohyoid, and upper part of the trapezius. Should these muscles become hypertrophied by frequent exercise of their function, as often happens in patients who have long suffered from emphysema, each contraction causes them to stand out prominently in their whole length, a phenomenon which is exceedingly striking when the neck is thin and emaciated, and when the supra-clavicular regions sink at each inspiration. The contractions of the accessory expiratory muscles also are sometimes visible.