muffled or even a clear sound in the immediate vicinity of consolidated portions of the lung.

Pneumonia occurs most frequently in the lower lobes of the lung, especially on the right side, more seldom in the upper lobes or right middle lobe, or in both lower lobes at the same time. If only a part of a lobe be consolidated the form of the non-resonant region, mapped out on the surface of the chest, presents nothing characteristic; in hepatization of an entire lobe, on the contrary, we are able to trace externally its exact outline. When the whole of the lower lobe is affected resonance is diminished posteriorly as far upwards as the middle of the shoulder-blades; in condensation of the whole of the upper lobe the dulness extends downwards to the fourth or fifth rib in front, to the fourth rib laterally, and is recognisable also in the supraspinous regions; in hepatization of the right middle lobe dulness is most readily detected in the axillary line, between the fourth and fifth ribs. —As a pulmonary lobe, when completely consolidated, increases considerably in bulk the region which it renders dull to percussion is correspondingly enlarged.

In certain cases pneumonia does not pass regularly through its three stages, nor does it confine itself to the locality in which it first appears; it *wanders* from one lobe or part of the lung to another, and resolution follows at once on the onset of the inflammation, without the occurrence of hepatization; the disease may thus run a course lasting several weeks, attacking successively all the lobes of the lung, and sometimes occurring in the same parts more than once.

In the pneumonia of children, which is seldom fibrinous, but usually catarrhal and following on capillary bronchitis, the thoracic resonance is never so strikingly diminished as in that of adults, because in the former the substance of the lung is not completely emptied of air, but contains islands of permeable tissue in the midst of the consolidated parts.

As in pneumonic hepatization, which is the type of complete consolidation of the lung, so in *every other variety of pulmonary condensation, from whatever cause arising*, the percussion-sound is *muffled* or *absolutely dull*. To this category belong the different forms of *cheesy pneumonic condensation of the lung* and *chronic interstitial pneumonia* usually designated by the collective term *phthisis*. The intensity of the dulness is determined by

the degree of infiltration and blocking-up of the air-cells and by the extent of lung concerned; thus the thoracic resonance may be perfectly normal during the first stages of phthisis, and it certainly suffers but slight modification when patches of healthy lung tissue are still found between the condensed portions.—The apices of the lungs are the parts most subject to this caseous pneumonic degeneration; they may both be attacked, but seldom simultaneously, or with equal violence; at other times the disease is unilateral, occurring then most frequently on the right side. Dulness so produced is accordingly most commonly found in the supraclavicular regions and on the clavicles; if the infiltration have invaded the whole thickness of the lung, which is very frequently the case, the tone in the supraspinous regions also loses in resonance, and should it be more abundant posteriorly than anteriorly the dulness will be less marked in front than behind, or may even be absolutely wanting over the clavicles. When the condensation spreads gradually to the lower part of the superior lobe the dulness to percussion extends similarly to the infraclavicular region as far downwards as the third and fourth ribs, and also, though not to so marked a degree, to the upper part of the shoulder-blades.

Acute miliary tuberculosis never diminishes the resonance of the chest, as it does not cause consolidation of the lung substance.

Effusion of serum into the pulmonary vesicles (*œdema of the lungs*) gives rise to but very slight muffling of the percussion-sound, as the air-cells are not completely filled with fluid but still contain a certain quantity of air, the lungs being thus in a condition analogous to that presented at the end of the first stage of pneumonia.—Effusion of blood into the pulmonary vesicles (hæmorrhagic infarction) produces dulness to percussion only when the infiltration is so abundant as to totally exclude the air, and when the affected part is more than 4 cmtr. in diameter and situated near the surface of the lung.

Hæmorrhagic infarction, as the result of mitral lesion, occurs most often in the middle and lower lobes of the right lung. Infarction is also rarer in the centre than at the periphery of the lung.

2. *Dulness* due to *compression of the lung*.

This results generally from the presence of fluid in the pleural

sac. A small quantity does not obscure the sound, this taking place only when the effusion has a thickness of at least 1½ cmtr. Experiments performed on the dead body, in adult males, showed that it is only after the injection of 400 ccm. of water into the pleura that the area of dulness rises two fingers'-breadths above the posterior lower margin of the lung; when augmentation of the mass of exudation is attended also by increase of the diameter of the layer of fluid interposed between the lung and the chest-wall the percussion-sound becomes less and less clear, and when the layer measures more than 5 cmtr. in thickness the sound becomes absolutely dull. So long as the collapse of the lung from pressure is not complete we can, by percussing forcibly, demonstrate the presence of the residuum of air, the sound so elicited being distinctly more resonant.

Slight exudations gravitate towards the lower and posterior parts of the pleural cavities, so that it is to these regions that the alteration in the thoracic resonance is at first limited; as the effusion increases in volume it spreads to the sides, and then to the front, of the chest, when the non-resonant area is found to extend from the vertebral column behind to the middle line in front. The level of the fluid, as traced by percussion, is not perfectly horizontal, but is often higher posteriorly than at the side or in front. Further, as the layer of fluid is of less thickness in its upper than in its lower part the dulness is more decided towards the bases of the lungs. And finally, when the diaphragm is forced downwards, as it is, both laterally and anteriorly, when the effusion is very abundant, the non-resonance to percussion is found to be co-extensive with the displacement; thus, in exudation into the left pleura dulness is detected at the anterior and lower part of the thorax, where normally the tympanitic sound of the stomach is heard, while in exudation into the right pleura the dulness in the hepatic region is considerably increased. When the visceral and costal pleuræ have contracted adhesions in the course of a former attack of pleurisy, the fluid does not move freely in the pleural cavities, but is enclosed in such spaces as are left between the adhesions, forming the so-called encysted pleuritic effusion; in these circumstances the dulness has no characteristic shape, and a definite diagnosis is possible only by calling to our aid the other methods of exploration.

When the exudation is undergoing absorption the lung begins to expand again gradually, and the dulness diminishes—both in extent, as the level of the fluid sinks, and in intensity, as the thickness of the layer of effusion, and with it the distance between the lung and chest-wall, decrease,—the air which enters the lungs making its influence felt in clearing the percussion-sound. If, notwithstanding the complete absorption of the fluid or its discharge outwardly through perforation of the thoracic parietes or by operative puncture, the lung shows no tendency to return to its former dimensions, having become collapsed from persistent compression, the percussion-sound remains permanently dull.

Certain recent experimental investigations by Garland and by Ferber, consisting essentially of the injection of slowly-coagulating fluids into the pleural cavities of dogs, give results more or less confirmatory of the foregoing statements. A slight effusion first of all separates the peripheral part of the diaphragm from the thoracic wall, making room for itself in the complementary pleural sinus which, in ordinary superficial respiration, is not occupied by the lung; dulness begins to be appreciable only when the level of the fluid rises above the margin of the lung. The upper surface of the fluid has a constant tendency to run into the horizontal position, and alters therefore with each change in the posture of the body; its upper boundary is never perfectly horizontal, however, but presents many curves, while the mass of the fluid is continually rising and falling,—a fluctuating motion which depends to a certain extent on movements proper to the fluid itself, but principally on the movements of the respiratory organs and of the heart. It is possibly owing to these undulatory movements that when at a later stage peripheral adhesion occurs at the upper margin of the exudation it does not take place in a horizontal, but in a somewhat irregular or wavy, line. The position and outline of the mass of fluid, however, depend chiefly on the habitual posture of the body; thus it usually reaches to a considerably higher point behind than it does in front when the patient has been long confined to bed lying on his back, and it remains at this level on the formation of inflammatory adhesions, while its upper boundary is generally nearly horizontal when the patient is able to be up and to move about during the time that the exudation of lymph is going on.—Further, the distribution of the fluid, and the consequent conformation of the non-resonant area, are influenced to an important degree by the occurrence of partial adhesions between the visceral and costal pleuræ at an early stage in the disease, and also to some extent by the retractibility of the lung, this not being equal at all points even in health. In these considerations may be found the explanation of the fact that while most commonly the upper margin of the dulness is higher behind than in front in some cases it does not deviate much from the horizontal line.

What has just been stated with regard to pleuritic exudation is applicable also, with certain modifications, to the transudation of fluid into the pleural cavity, *Hydrothorax;* but whilst the former, with very rare exceptions, is unilateral, the latter is invariably double, not originating simply in inflammation of the pleuræ but in disease of the heart or kidneys. Cardiac diseases, mitral disorder for example, give rise to this transudation by producing an overloaded condition of the pleural veins, and renal disease by causing hydræmia. If the quantity of fluid transuded be not excessive the intensity of the percussion-sound may be considerably modified by changing the position of the body, as the liquid always sinks to the lowest part of the thorax. Thus in patients suffering from Hydrothorax the fluid stands at the same level in front and behind so long as they maintain the upright position; but when they lie on the back the fluid gravitates to the posterior parts of the chest, and the percussion-sound becomes clearer in front. (This change in the thoracic resonance from alteration of the position of the body is also frequently observed, but to a less marked extent, in cases of ordinary pleuritic effusion.) In general the transuded fluid, which is not usually very abundant, occupies only the posterior and lower part of the pleura and is scarcely observable in front; the dulness caused by it therefore does not equal in extent or intensity that of pleuritic exudation.

Solid bodies situated between the lung and the chest-wall have the same muffling effect on the percussion note. Amongst these must be reckoned tumours of the mediastinum. The sound is similarly rendered dull when the lung is subjected to pressure by the development within itself of certain morbid growths (carcinoma, sarcoma), by tumours of the bronchial glands, by marked hypertrophy of the heart, and by large tumours of the liver or spleen which push the diaphragm upwards. In these cases and in others of a like nature the non-resonant region, even when the dulness is of great intensity and distributed over a considerable surface, has not the outline which is so characteristic of pleural exudation, but is usually somewhat irregular in conformation; obviously also the dulness is to be attributed to the solid body which gives rise to the pressure rather than to the impermeability of the portion of lung so acted upon.

Diseases of the bronchi, unaccompanied by structural change in the substance of the lung, *never diminish the thoracic resonance;* the calibre of the bronchi may be very much reduced by swelling of their mucous membrane, by very abundant catarrhal

secretion, or, as in croupous bronchitis, by firm fibrinous exudation, yet there is always sufficient room left to permit of the access of air to the alveoli; it is only when bronchial diseases invade the parenchyma of the lungs, exciting secondary infiltration and consolidation (broncho-pneumonia, bronchiectasis), that muffling of the percussion-sound is observed.

Increase in the Intensity of the Percussion-sound.

Conditions the opposite of those which obscure the percussion-sound (see p. 78 *et seq.*) increase its intensity; it is clearer therefore when the thoracic parietes are thin (in patients, for example, whose muscles and subcutaneous fat are slightly developed), as the force of the percussion stroke is more readily and with less loss of intensity conducted through them to the lungs, in which organs, accordingly, more vigorous vibration is set up. In old people therefore, and in those who are much emaciated, the percussion note is loud and clear. If the loss of tissue be confined to one side (progressive muscular atrophy) the chest, as in a case which I had an opportunity of examining, in which the right pectoralis major had almost entirely disappeared, may be so resonant at that part that the normal sound given at the corresponding point on the healthy side seems almost dull in comparison. Full inspiration intensifies the percussion note, as the volume of lung thrown into vibration is larger; in quiet respiration such differences in the intensity of the sound are not appreciable. In pulmonary emphysema the chest is sometimes abnormally resonant (the tone being also usually somewhat higher in pitch), especially in severe cases. Biermer designates this the *Bandbox sound* [Schachtelschall]. There are no other conditions to be noticed in which the thoracic resonance is simply increased in intensity. No special diagnostic value, therefore, attaches to this sign.

Pitch of the Percussion-sound.

The pitch of the percussion-sound depends on the degree of tension of the chest-wall on the one hand, and on that of the tissues of the lung on the other. The tension of both parts is increased in inspiration and diminished in expiration, so that in full inspiration the tone becomes slightly higher and in forced

expiration slightly lower; and this same rise and fall in pitch is observed both in healthy and in diseased lungs. As has been already stated, the most important of the circumstances which determine the pitch of the percussion-note is the tension of the chest-wall, the least important that of the lung substance; that the latter indeed exercises but little influence in this respect is evident from the mere fact that the inspiratory rise in pitch above referred to takes place also in certain pathological conditions in which changes in the tension of the pulmonary parenchyma are scarcely possible, such as diseases involving destruction of tissue. —The simple increase of *volume* that the lungs undergo in inspiration, apart from the forementioned greater tension, takes no part in causing the simultaneous rise in the pitch of the percussion-note; it might rather, on the contrary, be expected that the sound should become lower during inspiration, as the volume of air in vibration is greater than during expiration. This lowering of the tone, however, does not occur, as any tendency in that direction is more than compensated for by the other factors,—by the increased tension.—When the breathing is quiet the pitch remains practically the same during both phases of respiration.

In the normal thorax differences in the pitch of the percussion-sound at different parts may be recognised by a cultivated musical ear; thus, on the right it is usually found to be deeper than on the left, though occasionally the reverse condition is met with. Even on the same side of the chest the sound is not of the same pitch at all points: in the neighbourhood of the liver it is somewhat higher than at other parts,—at least in the sitting and standing positions. A slight elevation in the pitch of the tone is noticeable on the front of the thorax on raising the body from the recumbent to the sitting posture, due, evidently, to the increased tension of the thoracic parietes; this phenomenon is observed also in the dead body, a fact which excludes Rosenbach's theory, that it is caused by change in the respiration.—The variations in the pitch of the percussion-sound in the thorax are occasionally so trifling as to be appreciable only when carefully watched for; in other cases they are so marked that inexperienced examiners not unfrequently mistake them for differences in intensity, regarding the grave sound as dull in comparison with the acuter or clearer sound yielded at other points. These variations possess no diagnostic significance,

as they depend only on *physiological* differences in the tension of the chest-wall.

The percussion-note becomes *abnormally deep* in those pathological conditions which are accompanied by *diminution in the tension of the lung tissue* at any part: these are pleuritic exudation and pneumonia (Traube), of moderate intensity.

1. In *moderate pleuritic exudations*, occupying, for instance, not more than the half of the pleural cavity in front, laterally, and behind, the percussion-sound in the *infraclavicular* region * becomes deeper in pitch than at the corresponding point on the opposite side; this is caused by the gradual *retraction* of those parts of the lung still permeable to air and situated above the level of the fluid, by their slow return to their natural volume in the undistended state,—that is, by the loss of tension in those parts.

A retracted lung contains less air than one in its normal state, but the gravity of the percussion-note is not due to this, as diminution in the volume of air present in the lung usually raises the pitch of the sound, as is seen in almost every case in which the latter is muffled or dull.

2. In *pneumonia* also the pitch of the sound is lowered over those parts of the lung which remain *accessible to air*, and are situated near the hepatised portion. If, for example, the lung in its posterior aspect is completely consolidated while air is still freely admitted to the parts in front, the infraclavicular region gives an abnormally deep note on percussion. Here again, as in cases of pleuritic exudation, the same cause is in operation, deepening the sound,—decrease of tension in the non-infiltrated portions of the lung.

This lowering of the pitch of the percussion-sound is not invariably present in cases such as those described, as the retraction of the permeable parts of the lung does not always reach the precise degree which is favourable to the production of the phenomenon. Its duration must obviously be short in pneumonia, and it disappears rapidly in pleurisy also when the exudation undergoes any very great increase; I have observed it lasting only a few days, or even a still shorter time, in some cases in

* When the *anterior* part of the upper lobe of the lung is entirely emptied of air, and the *posterior* part slightly reduced in volume (as in cases of encysted exudation, glandular tumours, &c., situated in front) the percussion-sound in the supra-spinous region is abnormally low in pitch.

which the exudation was stationary. And further, at the commencement of the stage of absorption of very large effusions, when the compressed lung begins again to expand, the sound in the infraclavicular region may become abnormally deep.

The percussion-sound in the above-mentioned circumstances, in pleuritic exudation and in pneumonia, may be simply lowered in pitch, or it may also be attended by a tone of somewhat tympanitic character, or may even eventually become perfectly tympanitic; in another class of cases of the same diseases the note is tympanitic from the very outset, without having been previously lowered in pitch (see p. 97 *et seq.*).

Elevation of the pitch from *pathological* causes never occurs alone, unaccompanied by decrease in the intensity: the sound generally rises in pitch as it loses in clearness. An abnormally high-pitched percussion-sound is thus of no special diagnostic importance, as it never exists alone as a pathological phenomenon.

The Tympanitic Percussion-sound.

This sound owes its name to its resemblance to the sound of a drum, though the timbre in the two cases is not exactly identical. In its physical characters it closely approaches the musical tone,* inasmuch as it is a sound the pitch of which can be easily and accurately determined. The percussion-sounds of the larynx (with the mouth open or closed), of the distended cheeks, and of the stomach and bowel, illustrate well the differences in the pitch of the tympanitic note.

Tympanicity is not an unusual symptom in many of the diseases of the organs of respiration, but is never found on percussing the normal chest. In tracing the manner in which it is produced we must go back to its physical cause, which is most simply demonstrated by percussing the larynx or intestine,

* Gerhard has tried to prove, by means of König's sensitive gas-flame, that the tympanitic tone is composed of a succession of similar and regular sonorous waves, the non-tympanitic sound, on the other hand, of dissimilar waves. If a tympanitic sound or a tone of low pitch be received in the funnel of the apparatus and directed on the flame, a series of equal indentations, with non-tympanitic sounds a series of unequal indentations, appears in the curved bands of light on the rotating reflecting prism. H. Jacobson, on the contrary, whose experiments I have had the pleasure of witnessing, denies that the tympanitic sound is distinguished from the non-tympanitic by any marked difference in the flame-tracing.

or by striking on the upper part of a drinking-glass, and so throwing into vibration the column of air contained in it.

If we percuss with the hammer on a pleximeter held over the mouth of an empty or only partially filled vessel (a glass or jug), a tone is heard which is louder the nearer the margin, and the more forcibly, the stroke is delivered. This sound is *exquisitely tympanitic*, and retains the same pitch whether we percuss forcibly or gently, with a narrow or a broad pleximeter. On performing this experiment with different glasses the tone will be found to vary in pitch with the breadth and depth of each vessel, *being high in direct proportion to the shallowness of the glass and the width of its mouth.* The tympanitic character is given to the tone by the vibration of the column of air within the glass and the regular reflection of the sonorous waves from its smooth inner surface.

The conditions presented by the larynx, stomach, and bowel, are exactly similar to those detailed above; we have here to deal with a body of air set in vibration by the percussion-stroke, and enclosed in a cavity whose walls reflect the waves of sound with perfect uniformity. That the tympanicity of the intestinal percussion-note is due simply to the vibration of the air contained in the bowel may be proved negatively. If the whole bowel, or a portion of it, be removed from the abdomen and fully inflated, a ligature being placed on each end, the sound it gives to percussion is no longer tympanitic; the tense intestine becomes capable of entering into simultaneous vibration with the air it surrounds when the shock of the blow is communicated to it; but the vibrations of a membrane (that is, of a *solid* body), being different in kind and extent from those of the intestinal gases, the different sonorous waves interfere with each other, become broken and irregular, and constitute merely a *sound*,—not a *tone* in the physical sense. But as soon as a portion of the air is allowed to escape, the relaxed state of the bowel deprives it of the power of vibrating, and the sound again takes on the tympanitic character.

The alteration in the intestinal percussion-sound thus brought about by *artificial* means finds its parallel in the change which accompanies pathological *distension* of the *bowel* within the abdomen *by gas*; in cases of meteorism of the intestines tympanicity disappears, the percussion-sound over

the abdomen becomes louder and deeper in pitch, but not tympanitic.

The physical cause of the normal tympanicity of the intestines at once explains also the non-tympanicity of the normal thoracic sound. The lung in the thorax being always during life slightly over-distended, not only the pulmonary air but the pulmonary tissue also vibrates in response to the percussion-stroke; the sonorous undulations originating in the tissues of the lung mingle with and disturb those of the air, just as the vibrations of the tense intestinal wall obscure those of the intestinal gases, so that no *tone* is given out, but only a *sound* of a non-musical character. This explanation is supported by the fact that the lung, when *relaxed* and reduced to its normal size by removal from the body, yields a note of *tympanitic* quality, only the *air* within it, and *not* the *lung-tissue*, being thrown into vibration; and if the lung be now inflated the tympanitic tone disappears and is replaced by the ordinary percussion-sound of the lung while it is still within the thoracic cavity.

This simple phenomenon—that the lung when raised from the thorax and shrunk to its normal volume gives a tympanitic sound, the distended lung a non-tympanitic sound, to percussion—has been the subject of numerous controversies. Does the tympanicity of the sound depend on the vibration of the pulmonary air? or of the pulmonary substance? or of both combined? These are questions to which very different answers have been given by Wintrich, Mazonn, Körner, Hoppe, Geigel, Schweigger, &c.

Wintrich holds that the tympanitic sound given by the shrunken lung in the dead body originates in the pulmonary parenchyma, and not in the air contained in the alveoli or bronchi; the pulmonary vesicles are too small, the smallest body of air which can give rise to a tympanitic tone having, according to this author, a height of at least six lines, and the pulmonary sound also is somewhat higher in pitch, even than that obtainable from a column of air six lines high; neither can the air in the bronchi be considered as the cause of the tympanitic sound, as narrowing or dilatation of the principal bronchus, or even closure of the same by ligature, makes no change on the pitch of the note,—an effect, however, which is invariably produced by similarly altering the conditions of a column of air contained in an open cavity (see p. 91). Rosenbach, on the other hand, states that such a change *does* follow closure of the principal bronchus; that in these circumstances the sound becomes to a corresponding extent deeper.

The tympanicity of the percussion-sound of the collapsed lung in the dead body can be satisfactorily accounted for, not by regarding the pulmonary cells as columns of air surrounded by membranous walls,

and each one capable independently of entering into vibration,—their small size forbidding such an assumption,—but rather by considering them as *continuous*, forming *one large resonant cavity* subdivided by very thin membranous septa which are everywhere of homogeneous structure. *These septa, like the relaxed walls of the intestine or stomach, can indeed reflect sound, but are themselves unable to enter into vibration, as they lack the principal condition necessary to the performance of this function,—a sufficient degree of tension;* it is thus physically impossible to cause them to vibrate. So soon, however, as, by the inflation of the lung, this condition is realised, the tympanitic quality of the percussion-sound is lost, because now not merely the pulmonary air, but also the tense pulmonary tissue, responds to the percussion-stroke, and the substance of the lung, being a *solid body*, naturally gives vibrations quite different in nature from those of the *air* in the alveoli. The result is that these dissimilar sonorous waves to a certain extent neutralize each other and render the production of a tympanitic or musical tone impossible.*

The percussion-sound of the thorax is *tympanitic* in the following pathological conditions:

1. In cases of *excavation of the lung substance;*
2. In cases of *accumulation of air (gas) in the pleural cavity;*
3. When the *tension of the lung tissue is diminished.*

1. Tympanitic Percussion-sound in Pulmonary Cavities.

Pulmonary excavations, being invariably in direct communication with the bronchi, always contain air, and thus form resonant or reverberating chambers similar to the glass used in the experiment already described. To the production of the true tympanitic sound, however, certain conditions are necessary:

a. This air-space must be surrounded by *firm* walls, capable of reflecting waves of sound. The condensed tissue in which vomicæ are usually enclosed constitutes a solid wall of this character; in the absence of this induration, which is comparatively rare, no tympanitic sound is heard.

b. The cavity must be of a certain size,—as large as a pigeon's egg at least,—and be situated quite close to the surface of the lung; over deep-seated cavities, even when they are of much greater dimensions than above indicated, the percussion-

* Although *tympanicity* is more nearly related to the physical idea of a *tone* than to that of a *sound*, the latter term (as in the expression *tympanitic sound*) being the one best known and longest in use, is retained in the following pages.