On diseases of the chest, including diseases of the heart and great vessels : their pathology, physical diagnosis. symptoms, and treatment / by Henry William Fuller.

ON DISEASES OF THE CHEST,

INCLUDING DISEASES OF

THE HEART AND GREAT VESSELS.

BY THE SAME AUTHOR.

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ON RHEUMATISM, RHEUMATIC GOUT, AND SCIATICA:

THEIR

PATHOLOGY, SYMPTOMS, AND TREATMENT.

ON

DISEASES OF THE CHEST,

INCLUDING DISEASES OF THE

HEART AND GREAT VESSELS:

THEIR

PATHOLOGY, PHYSICAL DIAGNOSIS, SYMPTOMS,
AND TREATMENT.

BY

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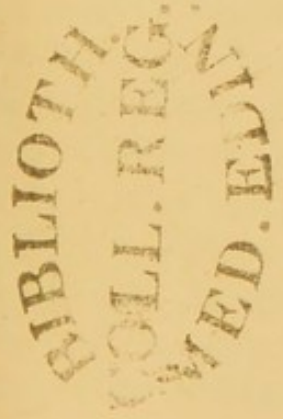

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PREFACE.

THERE are few students who do not experience misgivings on commencing the study of diseases of the chest. The subject embraces such a wide field of research, and is at once so difficult and complicated, that they are quite at a loss as to how to approach it. Unfortunately, their dislike of "dry detail," and their anxious longings after "practical" knowledge, induce them too often to attempt a method which is quite impracticable. Instead of making themselves familiar with the mechanism in which the physical signs of disease originate, and connecting each one separately with its corresponding change in the physical condition of the heart or lungs, they begin by attempting to group together certain physical signs and general symptoms as indicative of particular forms of disease. Before they are familiar with the very elements of auscultation, or have formed a clear conception of the nature and value of any of its signs, they endeavour to estimate the precise importance attaching to the presence of particular signs, and thus to diagnose existing mischief. No wonder that they so often find themselves bewildered, and are ready to discard the stethoscope, as, to them at least, a hopeless mystery. Auscultation has a language of its own, a language clear and forcible when properly understood, but otherwise obscure, mysterious, and unintelligible. This language it is almost impossible to acquire without a previous knowledge of its alphabet; and if, by extraordinary diligence and perseverance, the more zealous have

learned to interpret it correctly, the less fortunate and less persevering have notoriously failed in mastering its difficulties.

My object has been to lessen these difficulties, and to render attainable by men of ordinary capacities and ordinary opportunities a science which is indispensable to every medical practitioner. I have endeavoured to begin at the beginning, to assume nothing, and to explain every auscultatory sign by reference to the morbid condition and consequent altered mechanism in which each takes its origin. My wish has been to inculcate the necessity for regarding each physical sign, not as indicative of a certain disease, but rather as the natural consequence of a certain physical alteration in the tissues, the source and true interpretation of which must be determined by concomitant circumstances.

I have endeavoured to use the simplest language, so as to obviate the formidable difficulty presented by the confused and varied phraseology made use of by many writers on the subject, to give a definite meaning to each term which is employed, and to present a classification of the various sounds which shall be intelligible even to a novice at auscultation. How far I have succeeded it will be for others to determine; but I shall have done good service if I have even cleared the way for other labourers in the same field.

My explanation of the mechanism, and true significance of ægophony, and of sundry other abnormal sounds, is at variance with that which is generally received; but in this, and in every other instance, I have stated the grounds on which my opinion rests, and it will be for future observers and future experimentalists to estimate their real value.

Under the head of "Consumption" I have brought together a variety of facts, which serve to elucidate several points on which erroneous ideas are commonly entertained, and have discussed and expressed my views as to others about which there is considerable difference of opinion.

In that portion of the work which is devoted to the consideration of diseases of the heart and great vessels, my aim has been to simplify the subject as much as possible, to point out the conclusions which are to be drawn from recent experiments and patho-

logical research, and to present in a clear and intelligible form the diagnostic signs on which the prognosis and treatment of the various cardiac affections must be based.

I have purposely avoided encumbering my pages with the discussion of remedies and modes of treatment which experience has proved to be undeserving of confidence, and, whether in respect to diseases of the heart or lungs, have contented myself with pointing out the particular methods of treatment which have appeared to me most generally successful, and based upon the soundest physiological grounds.

Throughout the work I have availed myself of the labours of my predecessors, and have profited largely by their investigations. Whenever I have seen reason to dissent from their conclusions, I have not hesitated to avow it, and have stated the grounds of my opinion. In no other way is it possible to obtain a full discussion of the principles on which our science is founded, or to arrive at a satisfactory conclusion respecting their merits.

I need scarcely say how gratifying it would be to me to learn that the views which I have propounded have received the approval of my professional brethren; but as my sole object is to elicit the truth, I trust that those gentlemen who meet with facts which in any way elucidate the questions at issue will kindly favour me with the result of their experience.

13, Manchester Square, London, W.;
October, 1862.

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ERRATA.

At page 474, line 5, *for* "contractile" *read* "conducting."
At page 584, line 24, *for* "depressed" *read* "depraved."

Fig. 1.

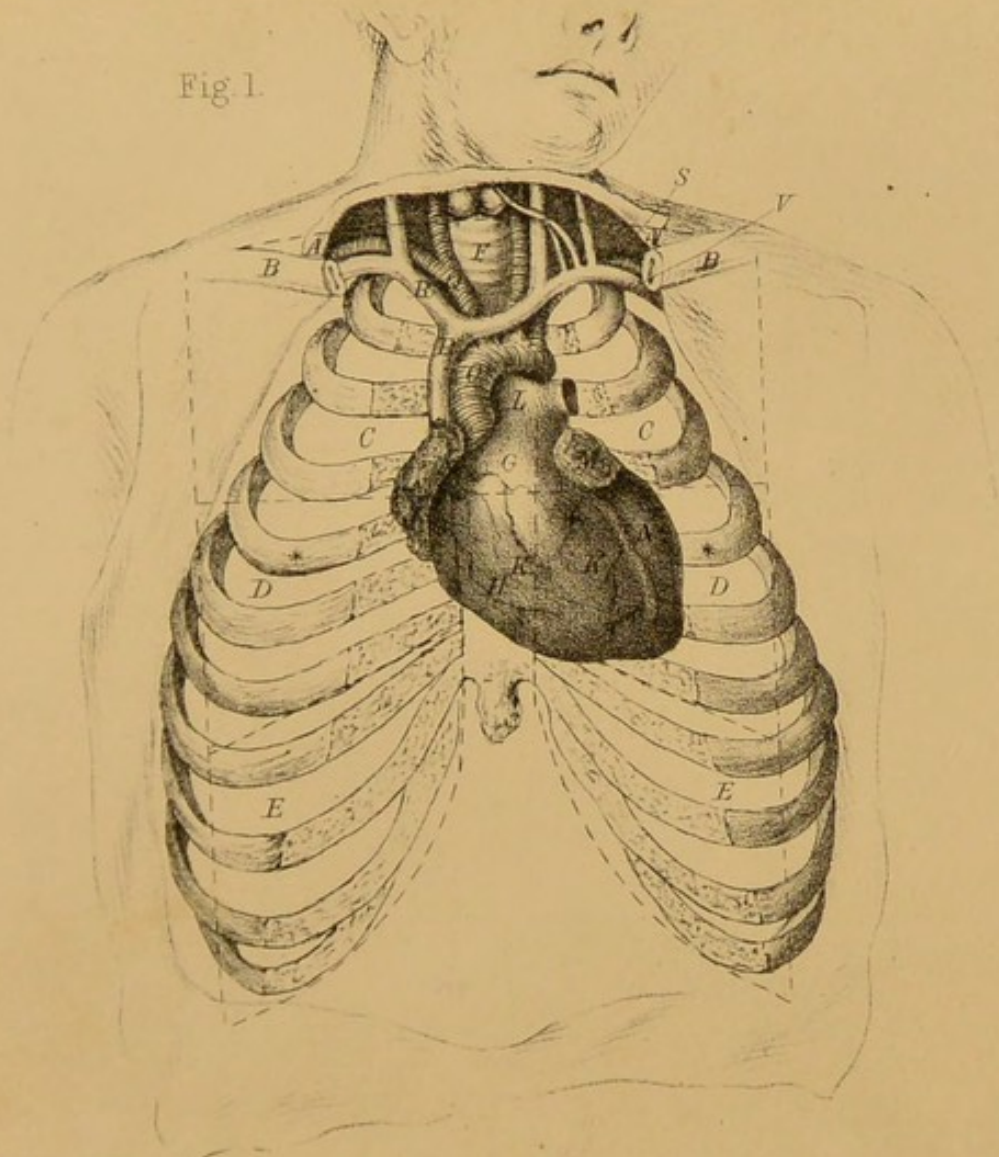
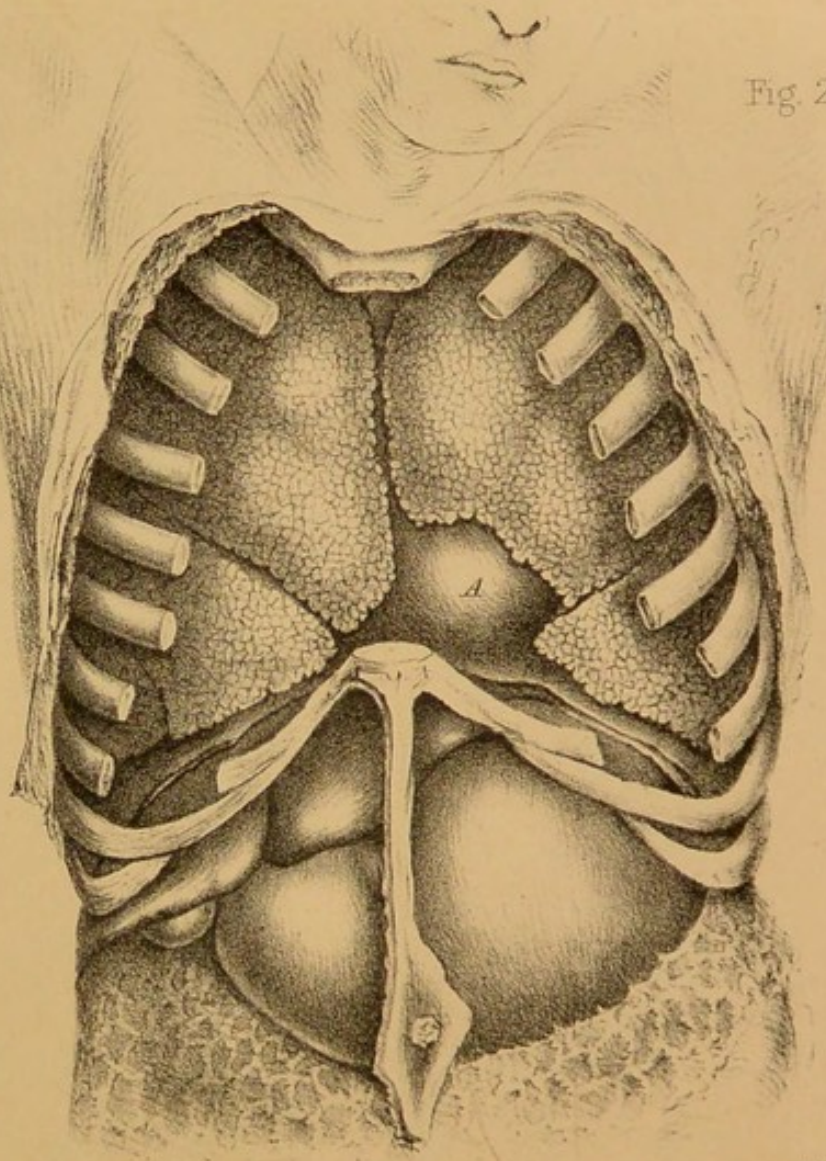


Fig. 2.



E. Burgess del. et lith.

W. West. imp.

Fig. 1. *Exhibits the relation of the heart & great vessels to the chest walls & to the different regions of the chest. A. Supra clavicular region. B. Clavicular. C. Infra clavicular. D. Mammary. E. Infra mammary. F. Supra sternal. G. Superior sternal. H. Interior sternal. + Nipples. J. Right auricle. K. Right ventricle. L. Pulmonary artery. M. Left auricle. N. Left ventricle. O. Aorta. P. Vena cava superior. Q. Arteria innominata. R. Innominata vein. S. Subclavian artery. V. Subclavian veins.* Fig. 2. *Exhibits the relative position of the Lungs & Heart & their relation to the chest walls. The lungs are supposed to be unexpanded.*

PART I.

CHAPTER I.

PRINCIPLES OF PHYSICAL DIAGNOSIS.—TOPOGRAPHY OF THE WALLS OF THE CHEST.—CONTENTS OF THE VARIOUS REGIONS.

"Felix, qui potuit rerum cognoscere causas."

When a student's attention is first directed to the physical diagnosis of diseases of the chest, he rarely forms a just estimate of its importance. He is apt either to underrate or else to overestimate its true value. In either case he starts with erroneous impressions, which, in one way or another, must ultimately cause vexation and disappointment. It may be well, therefore, to point out the true scope and object of the study we are about to enter on, in order that the beginner may be encouraged to persevere in mastering its difficulties.

The lungs, the heart, and the large vessels are placed within a bony framework, where eye cannot penetrate and the sense of touch is of little avail. Thus the senses usually employed in the investigation of disease are rendered almost useless, and as the *general* symptoms of diseases of the chest are uncertain and fallacious, the practitioner who knows not how to conduct a physical examination of the chest must treat a whole class of dangerous maladies in ignorance of the true nature of his patient's disorder. Here it is that the stethoscopist steps in. His aim is to discover the physical condition of the heart and lungs; to detect the early inroads of disease; to chronicle its onward progress, or haply note its gradual subsidence. His first object is to unveil the nature of the changes going on within the chest, with a view to the adoption of remedial measures calculated to

alleviate his patient's suffering; his next to ascertain the precise extent of those changes, in order that he may be able either to speak hopefully as to the issue or to warn the patient of his danger.

It is right, however, to approach the subject with the full and clear understanding that physical signs will not of themselves enable him to accomplish the object he has in view. As the constitutional symptoms which accompany chest diseases are often perplexing and fallacious, so the physical signs, when taken by themselves, are apt to lead to grievous error. The former are so uncertain and variable in their character that constant reference to the physical signs is required to confirm or negative their indications; whilst the latter are mere exponents of physical changes in the organs within the chest, and need all the light which can be thrown upon them by a careful consideration of the general symptoms before they can be treated as interpreters of disease or guides to its rational treatment. An exclusive reliance upon the one class of symptoms is as mischievous as a blind confidence in the other, and both prove fruitful sources of mistaken diagnosis and consequent improper treatment.

With a view to a clear understanding of the subject, I purpose beginning by an exposition of the principles on which the physical diagnosis of chest diseases is founded. It will then be seen how naturally, how necessarily the physical signs arise out of physical changes which have occurred, and how readily, after tracing morbid changes to the production of characteristic physical signs, the mind is able to invert the picture, and refer morbid sounds to their corresponding physical changes.

Physical diagnosis, then, rests on the fact that disease produces anatomical changes in the organs it affects, that such changes give rise to corresponding alterations in the physical properties of those organs, and that, knowing the physical properties of each part in a state of health, we are enabled to judge of the morbid changes which have occurred by the character of the alteration which has taken place in its physical properties. Of these we judge, 1st, by careful inspection or ocular examination of the chest; 2ndly, by palpation or manual examination; 3rdly, by mensuration; 4thly by percussion; 5thly, by auscultation; 6thly, by succussion.

The first of these methods of examination demands a previous knowledge of the form, size, and capacity of a healthy chest, the nature and position of its contents, and its action during ordinary and forced respiration, as also of the various changes it may undergo in one or all

of these particulars from influences external to the lungs, heart, and great vessels.

The three last require for their elucidation an intimate acquaintance with the laws which govern the production and transmission of sound, and, before they can be made available for the diagnosis of disease, a careful study of the sounds emitted under various circumstances by different portions of a healthy chest.

With the view of localising the physical signs, and thus rendering description as accurate as possible, it is expedient to map out the surface of the chest into regions admitting of easy recognition. The natural divisions of anterior, posterior, and lateral are not in this instance sufficiently minute, as each of the spaces thus marked out embraces an extent of surface which renders accuracy of description impossible. It therefore becomes necessary to adopt some other and smaller boundaries; and the following, which approximate closely to those proposed by many authors, will be found well defined, and sufficiently small for practical purposes:

Anteriorly on either side—1st, supra-clavicular; 2nd, clavicular; 3rd, infra-clavicular; 4th, mammary; 5th, infra-mammary.

„ in the centre—1st, supra-sternal; 2nd, superior sternal; 3rd, inferior sternal.

Laterally 1st, axillary; 2nd, infra-axillary.

Posteriorly on either side—1st, superior scapular; 2nd, inferior scapular; 3, inferior dorsal.

„ in the centre—inter-scapular.

Of these regions it will be observed that the interscapular region and the three sternal regions are single, whilst all the rest are double, existing equally on either side of the chest.

The *supra-clavicular* space has for its upper boundary a line drawn from the claviculo-scapular articulation to the upper rings of the trachea; below it has the clavicle, and inside the trachea.

The *clavicular* is that which lies beneath the inner three fifths of the clavicle, and has the bone for its anterior boundary.

The *infra-clavicular* has for its limits the clavicle above, the inferior margin of the third rib below, the sternum inside, and outside a line falling vertically from the junction of the outer and middle third of the clavicle.

The *mammary* is bounded above by the third rib; below, by the inferior margin of the sixth rib; inside, by the edge of the sternum; outside by a vertical line continuous with the outer boundary of the infra-clavicular region.

The *infra-mammary* is that portion of the anterior surface of the chest which lies below the mammary. Above it has the sixth rib; below, a curved line corresponding to the edges of the false ribs; inside, the inferior portion of the sternum; outside, a continuation of the outer boundary of the mammary region.

The *supra-sternal* is the hollowed space which lies immediately above the notch of the sternum, and is bounded on either side by the sterno-mastoid muscle.

The *superior sternal* is the space bounded anteriorly by that portion of the sternum which lies above the lower margin of the third rib.

The *inferior sternal* corresponds to that portion of the sternum which lies below the lower margin of the third rib.

The *axillary* has for its limits the axilla above; below, a line carried backwards from the lower boundary of the mammary region to the inferior angle of the scapula; in front, the outer margin of the infra-clavicular and mammary regions; and behind, the external edge of the scapula.

The *infra-axillary* has the axillary region for its upper, and the edges of the false ribs for its lower boundary; in front it is limited by the infra-mammary, and behind by the inferior dorsal region.

The *superior and inferior scapular* together occupy the space from the second to the seventh rib, and are respectively identical in their outlines with the upper and the lower fossa of the scapula.

The *inter-scapular* is the space situated between the inner margins of the scapulæ and the spines of the dorsal vertebra from the second to the sixth.

The *inferior dorsal* lies below the inferior scapula and the inter-scapular regions, which form its superior boundary; below it extends to the twelfth rib; inside to the spine; outside to a line falling vertically from the inferior angle of the scapula.

To these, then, or to somewhat similar divisions, constant reference should be made in describing the position of any alteration observed either in the walls of the chest or in the organs contained within them;

and therefore at the very outset of his investigation the student should make himself familiar with their limits.

When once this preliminary knowledge has been acquired, his next object should be to study the natural position of the thoracic organs, and ascertain precisely what portions of them correspond to these artificial external divisions. Without such a knowledge he cannot judge what signs he may expect to find there in a state of health, nor can he distinguish the accession of disease.

The following statement of the contents of the several regions will be found sufficiently minute for practical purposes:

The *supra-clavicular* region on either side contains the very apex of the lung, with portions of the sub-clavian and carotid arteries, and of the sub-clavian and jugular veins.

The *clavicular* is occupied by the parenchyma of the lung. On the right side, at its outer extremity, lies the sub-clavian artery; at the sterno-clavicular articulation, the arteria innominata; and on the left side, deeply seated, are the carotid and sub-clavian arteries.

The *infra-clavicular* has on either side the superior lobe of the lung and the main bronchus: the right bronchus lies behind, the left a little below the second costal cartilage; on the right side is the superior cava and a small portion of the right auricle of the heart; on the left is a portion of the pulmonary artery, and in some instances a portion of the sub-clavian artery. The aorta and the pulmonary artery lie immediately behind the second sterno-costal articulation—the one on the right, the other on the left side of the sternum. On the left side the lower boundary of this region very nearly corresponds to the base of the heart.

The *mammary* region differs greatly in its contents on the two sides. On the right, the lung is found in front extending down to the sixth rib, where its thin, sharp border very nearly corresponds to the lower boundary of this region. The fissure which separates the upper and middle lobes commences about the fourth cartilage; that which separates the middle and lower lobes about the fifth interspace. The right wing of the diaphragm, though not attached higher than the seventh rib, is usually pushed up by the liver as high as the fourth interspace, the lower surface

of the lung being concave to receive it. A portion of the right auricle and the superior angle of the right ventricle lie close to the sternum, between the third and the fifth ribs.

On the left side, the lung lies in front down to about the fourth sterno-costal articulation; thence its anterior border passes downwards and outwards (leaving an open space for the heart), until it reaches the fifth rib, when it curves forwards and downwards to opposite the sixth rib or interspace, and thence passes outwards almost horizontally. The open space where the heart, so to speak, comes in contact with the chest is usually about an inch and a half or two square inches in extent. The left auricle and ventricle, and a portion of the right ventricle towards the apex, are found in the left mammary region. The apex lies immediately above the sixth costal cartilage. The fissure which separates the two lobes of the left lung usually commences about the fifth interspace.

The *infra-mammary* on the right side contains the liver, with the lung protruding in front, on full inspiration; on the left, the anterior border of the spleen, and the stomach, with a portion of the left lobe of the liver, lying in front of it, towards the mesian line. The stomach usually rises to a level with the sixth rib.

The *supra-sternal* is occupied chiefly by the trachea; but on the right side is the arteria innominata. The arch of the aorta sometimes reaches to its lower border, and may be felt pulsating there, on deep pressure.

In the *superior sternal*, the lung lies in front, and immediately behind it are the ascending and transverse portions of the arch of the aorta and the pulmonary artery, from its origin to its bifurcation. The pulmonary valves are situated close to the left edge of the sternum, on a level with the lower margin of the third rib; the aortic about half an inch lower down, more deeply seated, and midway between the mesian line and the left edge of the sternum. The trachea bifurcates on a level with the second ribs.

The *inferior sternal* has lung in front on the right side throughout its whole extent, and on the left down to about the fourth sterno-costal articulation. Below this, on the left side, lies the chief part of the right ventricle of the heart, and a small portion of the left ventricle. The mitral valve is situated close to the

left edge of the sternum, on a level with the fourth sterno-costal articulation; and the tricuspid lies nearer the mesian line, and more superficially. Inferiorly is the attachment of the heart to the diaphragm, and below this again a small portion of the liver, and sometimes of the stomach.

In the *axillary*, the subjacent parts are the upper lobes of the lungs, with the main bronchi deeply seated.

The *infra-axillary* contains the lung, with its lower margin sloping down from before backwards. On the right side is the liver; on the left the stomach and spleen.

The *superior and inferior scapular* have beneath them the parenchyma of the lung.

In the *inter-scapular*, towards the centre, opposite the third dorsal vertebra, will be found the bifurcation of the trachea, the main bronchi, and the bronchial glands; on either side parenchyma of the lungs; on the left the œsophagus; and from the upper part of the fourth dorsal vertebra downwards, the descending aorta.

In the *inferior dorsal*, the lung lies superficially as low as the eleventh rib. On the right side, the liver extends downwards from the level of the eleventh rib; and on the left lie the stomach and intestines. Along the left side of the spine runs the descending aorta; and on both sides, close to the spine, is found a small portion of the kidney.

CHAPTER II.

INSPECTION OF THE CHEST—HOW TO BE CONDUCTED; WHAT TO BE OBSERVED.

A CAREFUL ocular examination of the chest will often furnish important information respecting the condition of the thoracic viscera. It shows us not only the shape of the chest and any irregularities, congenital or acquired, presented by any portion of its surface, but it makes us acquainted with the motions of the thoracic walls, the cha-

racter of the respiratory efforts, the frequency with which they are repeated, their evenness or irregularity, the ease or difficulty with which they are performed, and the part played by the various muscles of respiration. It enables us to note the rhythm* of the respiration, to estimate the exact time occupied by the acts of inspiration and expiration respectively, and to compare the movements of the two sides of the chest in regard to their symmetry, regularity and force. These are all important points in the diagnosis of disease, as will be seen when their practical bearing is explained.

But if we wish to make inspection subservient to the diagnosis of thoracic disease, it behoves us, first of all, to examine carefully the form, size, and movements of a *healthy* chest. Without this preliminary study it will be impossible to detect any physical alteration, still more to estimate its importance.

In conducting an examination of the chest whether by inspection or by any other means, it is of the utmost importance that the patient's position should be free and unrestrained, and that, whether he be lying down, sitting or standing, the plane on which he rests should be perfectly even, and his limbs in the same position on either side. Inattention to these points may lead to irregularity more or less discernible between the two sides of the thorax, when the one is carefully compared with the other, and may give rise to differences more or less marked, when corresponding portions of the chest are contrasted by the aid of mensuration, percussion, and auscultation.

The patient, then, being placed in an easy posture (sitting is the easiest and most convenient), with the surface of the chest exposed, in a good light opposite to the observer, and the arms similarly disposed on either side, the thorax should be examined in front, behind, sideways, and from above downwards. Its form, size, and movements should be noted during ordinary and forced respiration, every deviation from the healthy standard marked, and every region on the one side compared with its corresponding region on the other.

The apparent size of the thorax in relation to the body varies greatly in different individuals enjoying equally good health, and no certain rule, therefore, can be laid down respecting it. It is sufficient for our purpose at present to know that a large broad chest does not (as is

* By the term rhythm is meant the mode of progression and evolution of the sounds, which may be gradual and equable from first to last, or irregular, jerking, or interrupted.

popularly supposed) denote a healthy chest, nor a small or somewhat narrow chest an unhealthy.*

The form of a well-proportioned chest being well known to anatomists, it is needless to enter into a description of it here. Suffice it to say, that in a normal state its two sides are symmetrical in every part, and the intercostal spaces more or less distinctly visible, according as the individual is more or less fat.

Considerable alterations, however, in the form of the chest are compatible with a healthy condition of the thoracic viscera, and it therefore becomes necessary to make ourselves acquainted with the frequency of their occurrence, in order that we may be enabled to estimate correctly the indications they severally afford.

It appears from the investigations of M. Woollez† that a *perfectly* symmetrical chest is of rare occurrence, existing in scarcely more than one out of every five of the adult male population. This may be owing to causes either of physiological or pathological origin; the former unconnected with thoracic disease, being the result of congenital malformation, or of tight lacing, or else produced by the habits or pursuits of the individual; the latter the effects of existing or former pectoral disease. In either case the defects may be *general*, affecting the whole form or shape of the thorax; or *partial*, giving rise to local alterations without interfering with its general shape. But whether the defect be general or local, it is obvious that mere want of symmetry in the chest, cannot be depended on as indicating visceral disease. Its chief value is that it arrests attention and suggests inquiry as to the existence of disease at a former period, as also into the presence of characteristic signs denoting subjacent structural alteration.

When the form of the chest is altered, it may be in one or other of the following ways:—The entire thorax, or any part of it, may either bulge or fall in to an unnatural extent; there may be abnormal elevation, or lowering of the whole or any part of it, or it may be more or less distorted.

The *bulging* may be either general or local; the walls of the chest on both sides may be unusually prominent; or the prominence may be confined to one side, or it may be even more circumscribed, existing only in one or two small localities. But whatever its form, its cause,

* See 'Med.-Chir. Trans.,' vol. xxix, p. 172-4.

† 'Recherches sur l'Inspection et la Mensuration de la Poitrine.' Paris, 1838.

supposing it to arise from disease, is always the outward pressure of some force existing within the chest. Therefore, if it is general, it takes its origin in some disease which produces such pressure over the whole of either or both sides; as, for instance, general vesicular emphysema, pneumothorax, empyema, and other rarer forms of mischief; whilst if it is partial only, it is caused by an expanding force of an equally limited nature; such, for instance, as partial emphysema, circumscribed empyema, aneurismal and other intrathoracic tumours, or enlargement of the heart, liver, or spleen. Simple hydrothorax has been said to be incapable of occasioning bulging, whether general or local; but I am satisfied that the assertion is incorrect; for although it holds good as a general rule, I have met with several well-marked instances of bulging chest, which, from the effect of treatment, I cannot doubt to have resulted from long-standing and copious serous effusion into the pleural cavity, and in one case in which bulging was well-marked, the trochar afforded unmistakeable evidence of mere serous effusion.

Retraction or falling in of the chest may, in like manner, be either general or local. In both cases it results from diminution in the size of the lung, consequent either on pressure from without or on change in the lung substance. The pulmonary tissue, being deprived of elasticity, no longer offers its normal resistance and the chest walls fall in under atmospheric pressure. If the mischief, be what it may, pervades the whole of one side of the chest, the retraction or falling in of the chest walls on that side will be general; if it be limited to one or two spots, the depression will be circumscribed, occurring only at those spots. So it happens that general depression or retraction of the chest is most frequently found in cases where, at a former period, there has been extensive pleuritic effusion, and the lung is carnified and reduced to a small inelastic mass by the continued pressure of the fluid; but it is also observed to a less extent among consumptive patients, as also in cases where the lung substance is collapsed and atrophied by inaction, consequent on the occlusion of the bronchi, whether by the pressure of an aneurismal sac, a cancerous deposit, enlarged bronchial glands, or any other means. Instances of partial falling in of the chest are constantly met with in the infra-clavicular regions when tuberculous deposits exist in the apices of the lungs and occur in other parts of the thorax, according to the position of the lung substance which has undergone contraction, whether from the infiltration of

tubercle or any other matter, or from the formation of pulmonary abscess.

Elevation and lowering of the shoulders and chest walls, or certain portions of them are of common occurrence. The former is a frequent accompaniment of general emphysema, and is also seen not uncommonly on one side of the chest, when there is chronic pleuritic effusion on the other: the latter may be observed when the chest has fallen in as a result of chronic pleurisy.* Without strict investigation, however, it would be wrong to attach much importance to such phenomena, for they often exist without visceral disease, and to such an extent in some instances, that in a case recorded by Dr. Walshe, elevation of the shoulder on the one side, occurred on the same side as retraction of the parietes from chronic pleurisy.†

Distortion of the chest, when extensive, is usually the result of congenital malformation or of accidental circumstances unconnected with thoracic disease. It may take place, however, to a certain extent as a consequence of disease within the thoracic cavity. Thus chronic pleurisy when followed by compression and carnification of the lung, and consequent falling in of the chest walls, not unfrequently causes lateral curvature of the spine, the convexity being towards the sound side,* as also twisting of the ribs to such an extent in certain cases that their upper edges become external. The sternum, ribs, and clavicles may yield in any direction under the pressure of aneurismal and other tumours, and the clavicles may bend downwards and inwards in cases of extreme disorganization of the apices of the lungs.

Such are the chief modifications which may occur in the form of the chest; and if the causes on which they may depend are constantly borne in mind there will seldom be much difficulty in forming a correct estimate of any irregularity observed during the progress of visceral disease.

Valuable, however, as are the indications derived from this source, they are less so than those afforded by the movements of the chest. In a healthy person inspiration is effected partly by the elevation and expansion of the ribs, partly by the descent of the diaphragm; and the eye watching the naked chest may readily perceive the part taken by the thoracic and abdominal muscles respectively. If the descent of the

* See 'Hospital Case-book,' xxxviii; case of George Godfrey, admitted into Hope ward, Jan. 16th, 1859.

† Walshe, 'On Diseases of the Lungs and Heart,' p. 14.

diaphragm is checked, the thoracic muscles have to perform double duty, and "thoracic" respiration ensues, characterised by "heaving" of the chest. If, on the other hand, anything occurs to prevent a free expansion of the chest, the diaphragm is brought more actively into play, and diaphragmatic or abdominal breathing is the result. Thus it often happens that even a cursory survey of the respiratory movements suffices to throw important light on the seat of mischief.

But the movements of the chest, if carefully examined, may be made to furnish still more accurate information. For this purpose they may be divided into *general* and *partial*, the first being those in which the entire thorax is concerned; the last those arising from the motion of the ribs on one another, and therefore confined to particular regions.

The *general* motions during inspiration are of two kinds, viz., expansion and elevation; and during expiration the converse of these, viz., retraction and depression. During tranquil respiration, more especially in men, these costal movements are exceedingly slight, the breathing being carried on chiefly by the diaphragm; but during forced respiration, and in women who by lacing restrict the action of the diaphragm and the lower ribs, they are much more palpable and may be seen without difficulty over the entire chest. The expansion and elevation movements are intimately associated, and in health are closely proportioned to one another, but their actual extent is found to increase in a direct ratio to the mobility of the chest walls and the height of the individual, whilst the frequency of their repetition is regulated by the force, duration, and completeness of the pulmonary respiratory movements, which vary with the requirements of the system. They usually occur with greater rapidity towards the middle of the act of respiration than at its commencement or towards its close, but from first to last they take place evenly, without any jerking irregularity of rhythm. Inspiration is followed immediately by expiration; at the close of that act there is usually a pause, and then inspiration begins again. The inspiratory and expiratory efforts are of nearly equal length; and if the duration of an entire respiratory act be represented by 12, the duration of inspiration may be estimated at 6, that of expiration at 5, and that of the succeeding pause at 1, so that the period of thoracic movement is to that of rest as 11 to 1.

The *partial* costal motions arising from the movement of the ribs on one another are not very readily appreciated by the eye; indeed, during tranquil respiration they are barely perceptible, but in forced

inspiration the ribs will be seen to diverge from one another, the divergence being greatest in the lower interspaces. During expiration they converge towards each other.

Dr. Sibson maintains that the five upper ribs converge during inspiration, and moreover that their convergence is "great."* Any one, however, by placing a finger in the upper intercostal spaces of a thin but healthy person during forced respiration, may satisfy himself that such is not usually the case. During the act of expiration the finger will be compressed, and will be relieved of all pressure during inspiration. The real fact appears to be, that each of the ribs alluded to not only rises during inspiration more than the rib above it, but moves outwards and forwards to a greater extent, so that when the elevation movement takes place without the expansion movement—as it does in certain forms of disease which interfere with the due expansion of the lung—these upper ribs do "move nearer to each other," as stated by Dr. Sibson, whereas, when the expansion movement is fully performed and is carried out in due proportion to the elevation movement, the ribs, though differently placed in relation to each other, cannot be said to approximate.

But besides these costal motions, there are, as already stated, other movements connected with respiration which are deserving of close attention. As the lungs expand and the diaphragm descends, the subjacent viscera are forced down, and the abdomen consequently protrudes. In tranquil respiration this abdominal expansion movement commences before the costal, and varies in an inverse proportion to it; so that in men, in whom the costal movement is slight, the abdominal is considerable; whilst in women who lace tightly the reverse holds good. In both sexes, however, forced inspiration gives rise to thoracic movement, which is out of all proportion greater than the abdominal, and commences before it even in men.

As age advances and the costal cartilages become rigid, thoracic expansion necessarily diminishes, the breathing becomes more diaphragmatic, the movement of the sternum exceeds that of the ribs, and its lower extremity advances more than the upper. On the other hand, in infancy and childhood, the costal cartilages being extremely flexible, admit of greater movement of the ribs: the upper part of the sternum advances more than the lower, and thoracic expansion is greater, and abdominal breathing less than in the adult. Sometimes,

* 'Med.-Chir. Trans.,' vol. xxxi, p. 360.

however, if the abdomen is large and the respiration hurried and forcible, the air does not find entrance to the lungs quick enough, and in quantity sufficient to fill the space created by the descent of the diaphragm; in which case the lower end of the sternum and the adjoining cartilages will be observed to fall in at the beginning of respiration and to advance again as the chest becomes more fully inflated. In females, on the contrary, owing to the restraint of stays and tight lacing, the action of the diaphragm and lower ribs is interfered with and thoracic expansion is exaggerated.

Such then are the movements of the healthy chest and the modifications to which they are exposed from causes independent of visceral disease.

The mechanism of respiration, however, is liable to be interfered with by disease; and if the alterations so produced are carefully studied, they may be made to furnish most important information respecting the changes going on within the thorax. Sometimes the expansion and elevation of the chest walls may be simultaneously and abnormally increased or diminished: sometimes the relationship naturally existing between the elevation and expansion movements may be disturbed, the former of these actions being greatly augmented, whilst the other is lessened or almost wholly suspended: sometimes the relative proportion of the thoracic and abdominal movements may be altogether changed, the one being abnormally diminished whilst the other is increased in a corresponding degree: and sometimes, again, the respiratory rhythm may be interfered with, and may become jerking, or the expiratory movement may become of longer duration than the inspiratory. All of these changes may be easily recognised by the eye, and each possesses its proper signification.

An increase in the movements of expansion and elevation may take place simultaneously on *both* sides of the chest under three different conditions—1st. When the descent of the diaphragm is checked by mechanical causes existing within the abdominal cavity, such as pressure of the gravid uterus, dropsical effusion, flatulent distension of the stomach and bowels, and tumours of various kinds. 2ndly. When the action of the diaphragm is checked by the existence of abdominal pain, whether due to inflammation or other causes. 3rdly. When increased muscular exertion is used to overcome some obstruction situated low in the chest, as in spasmodic asthma.

Increased movement may be produced on *one* side of the chest by

anything which impairs the function of the other, such, for instance, as pleuritic effusion, pneumonia, or tubercular infiltration, pressure on the main bronchus, and other similar circumstances, and, in like manner increased action in a part of one side may be occasioned by deficient action in the remainder of that side.

Diminution of the natural movements of the chest may be observed under three conditions:—

1stly. When disease of the lungs or pleura exists of such a nature as to offer a mechanical impediment to the entrance of air, as in tubercular, pneumonic, or other infiltration of the lung substance; in pleurisy and pneumothorax, and in pressure upon, and consequent obstruction to the main bronchi. 2ndly. When, in consequence of pain in the chest, there is voluntary fixing of the chest walls, and respiration is carried on chiefly by the diaphragm, as in inflammation of the costal and upper pulmonary pleura; in pneumonia, pleurodynia, and intercostal rheumatism. 3rdly. When some paralysis of the respiratory muscles exists, as a result of cerebro-spinal mischief. A diminution of thoracic movement, however, is also observed, when an obstacle exists to the entrance of air in the upper part of the air-passages, as in croup, cynanche tonsillaris, laryngeal disease, and œdema and spasm of the glottis, as also in cases of pressure of an enlarged thyroid gland or of pharyngeal, aneurismal, or other tumours. In such cases, more especially when the amount of obstruction is great, not only do the thoracic walls not expand, but owing to the forcible descent of the diaphragm, they are actually drawn in during inspiration, whilst the abdominal walls advance in a corresponding degree. If the obstruction affects the bronchi on one side only of the chest, the phenomena referable to such obstruction will occur *on that side only*, whilst the amount of thoracic movement on the other side will be increased.

2ndly. *The relationship naturally subsisting between the elevation and expansion movements may be disturbed*, the former existing in full or overful activity, whilst the latter is almost suppressed. This is seen either partially or generally, according to the extent of mischief, in all cases where the pulmonary tissue is consolidated and rendered impermeable to air, as in tubercular, pneumonic, or other infiltration; in pleurisy, and in instances, in which the lung is compressed by intra-thoracic tumours. Volition may cause extraordinary efforts to expand the chest, and may effect its elevation; but as the lungs do not admit of inflation, no expansive movement follows. So also, when

the chest is already fully expanded, as in cases of extreme vesicular emphysema, volition may drag the chest walls upwards, but cannot effect any further expansion. Indeed, in some such instances, the chest may even fall in at the base during full inspiration.

3rdly. *The relative proportion of the thoracic and abdominal movements may be altogether changed; the one being abnormally diminished, whilst the other is increased in a corresponding degree.* Thus it is found that whatever causes a diminution of thoracic movement gives rise to a corresponding increase of diaphragmatic action, and whatever checks the action of the diaphragm produces increased thoracic movement, with heaving or thoracic respiration. It is not necessary to recapitulate the causes which lead to an increase or decrease of thoracic movement, as these have been referred to in my description of the causes of the other varieties of breathing; but it may be well to add, that any disease or injury of the spinal cord below the point where the phrenic nerve is given off will give rise to increased action of the diaphragm.

4thly. *The respiratory rhythm may be interfered with, the expiratory movement becoming of longer duration than the inspiratory.* This occurs when obstruction exists in any part of the air-passages to the free egress of the air. It receives its most constant and best marked illustration in cases of vesicular emphysema, where the elasticity and resiliency of the lung are destroyed, or when aneurismal, cancerous, or other intra-thoracic tumours are pressing upon the trachea or main bronchi; but it is also met with in many cases of tuberculous deposit, and forms a valuable accessary sign of its existence. In epilepsy, hysteria, chorea, and many other affections, the rhythm of the breathing becomes jerking and uneven, without any disease of the thoracic organs.

But other important facts, beyond mere changes in the rhythm of respiration, are discoverable by ocular inspection. The eye takes cognizance of dilatation and distension of the superficial veins of the chest and neck, and of pulsation on any part of the thoracic walls. Dilatation of the veins indicates deep-seated obstruction to the venous circulation, and suggests the necessity of examining into the condition of the heart and great vessels, and, in the event of no cause of obstructed circulation being there discovered, of searching for some pulmonary, or other intra-thoracic disease, capable of producing the obstruction. Nothing is more common than to find the superficial veins distended in connexion with extensive tubercular and cancerous deposit, and not unfrequently, when the deposit is confined to one side of the chest, the

veins of that side alone are dilated. It should be added, however, that the limitation of the distended vessels to one side is not diagnostic of pulmonary disease; for it is observed in connection with aneurism, cancer, and other forms of intra-thoracic tumours. So again in respect to pulsation on the surface of the chest. Not only does ocular inspection detect the existence of increased cardiac pulsation, and of pulsation due to aortic aneurism, but it often informs us of a wave-like pulsation at the second intercostal space, attributable indirectly to tubercular disease of the lungs; for when the lung has been excavated by vomicæ, and has subsequently contracted, the left auricle or the pulmonary artery may come into contact with the anterior surface of the chest, and give rise to pulsation in the situation above mentioned.*

CHAPTER III.

MANUAL EXAMINATION OF THE CHEST, OR PALPATION—HOW TO BE PERFORMED; INDICATIONS DERIVABLE FROM IT.

The sense of touch may often be made available in the diagnosis of thoracic disease. It acquaints us with the actual condition of the chest walls, and the nature of their movements, whether general or partial; it informs us of the character and amount of vibration communicated under certain circumstances to the parietes, and thus makes us aware of certain physical alterations in the lungs and pleura whereby that vibratory action is modified; and it indicates the presence or absence of fluctuation in the intercostal spaces, and thus gives notice of the existence or non-existence of fluid in the cavity of the pleura.

In order to arrive at satisfactory results from its employment, it is necessary to observe the various precautions already pointed out as conducive to accurate inspection. Beyond this, it need only be mentioned that the hand or the fingers should be applied to the chest gently and evenly; that the two sides of the thorax should be examined simultaneously, the one with the right hand, the other with the left; and that each corresponding portion on the two sides should be thus examined and compared.

* For further information on the subject of inspection of the chest, see Part III, Chapter I.

In many instances the unassisted eye is incompetent to determine whether the local expansion of the chest bears its due proportion to the elevation movement, as also to estimate the motion of the ribs in respect to one another. Both these important practical points may be determined by the aid of palpation. In the upper part of the chest it readily detects the absence of local expansion, and thus, by affording evidence of some physical condition which prevents the due inflation of the lung, becomes a valuable auxiliary in the diagnosis of consolidation which so often exists in the early stage of consumption. In the lower part of the chest the thumb or the index-finger, placed in one of the intercostal spaces, not only detects the absence of local expansion, but feels convergence of the ribs taking place coincidently with the continuance of the elevation movement, and thus furnishes additional presumptive proof as to the impermeability of the pulmonary tissue.

Again, touch is the only sense whereby it is possible to appreciate the character and amount of the vibration commmunicated by the voice to the chest walls—a fact of much practical importance. Thus, if, the hand be laid lightly upon the surface of the chest whilst a healthy person is speaking, a delicate, tremulous vibration will be felt, varying in a direct rate to the loudness and coarseness of the voice, and the lowness of its pitch. As a general rule, therefore, this "vocal fremitus" is more pronounced in adults than in children, and in males than in females, and is stronger during the utterance of certain sounds than it is during the utterance of others. It is more strongly felt in thin than in fat persons, whilst in children and females, more especially if stout, and having shrill, weak voices, it is often altogether absent. On the right side of the chest it is usually more marked than on the left, especially in the infra-clavicular region,* whilst in the space where the heart is uncovered by the lungs it is altogether deficient, on

* My friend Dr. Herbert Davies (on 'Diseases of the Lungs and Heart,' p. 112) asserts that, "although the natural vocal vibration is stated by some writers to be more marked on the right than the left side of the chest, the difference may be practically disregarded, and both sides may be taken to present in health the same amount of vocal fremitus." This certainly does not accord with my experience, which enables me to state that, out of 300 persons of both sexes and average health and prospects of longevity, who have come before me for examination for life assurance, and whom I have examined specially, with a view to this inquiry, above three fourths (234) have had vocal fremitus more marked on the right than on the left infra-clavicular region, the spot, above all others, where the existence or non-existence of such inequality possesses the greatest importance in relation to the presence of tubercular disease.

account of there being no proper medium for its transmission by the chest walls.

In disease the vocal fremitus may be either increased or diminished. In all cases, however, a careful comparison of the two sides of the chest, due regard being had to the marked difference naturally existing between them is necessary to determine the presence of disease. Moderate consolidation of the pulmonary tissue, whatever its cause, intensifies the vibration, by increasing the reflecting power of the bronchial tubes, preventing the diffusion of the vibrations, and forming a better medium for their transmission to the chest-walls. Thus tuberculous deposits, when of moderate extent, partial pneumonic infiltration, more especially if in the vicinity of the larger bronchi, and œdema in its early stage, increase the fremitus; whilst more extreme tuberculization of the lungs and large intra-thoracic tumours are apt to deaden, or altogether destroy the vibration, by pressing upon, or blocking up the bronchial tubes, and thus interfering with the columns of air through which the vibrations are propagated on which the vocal fremitus depends.

Again, the fremitus is annihilated when the lung is removed from the chest walls by the intervention of air or liquid in the pleural cavity —a fact which sometimes enables us to discriminate between pleuritic effusion and pneumonic or other consolidation of the lung, in which, as already stated, the vocal fremitus, as felt by the hand, is ordinarily increased, rather than diminished or destroyed.

When the removal of the lung from the parietes is due to the presence of solid matter, the fremitus may or may not be destroyed, according as the foreign matter is more or less fitted by its nature to serve as a conducting medium, and is more or less remote from the larger bronchi, where the vibrations are most intense. If the solid matter be large in amount and inelastic, it will altogether stop the vibration; if less in amount, it may only deaden it; whilst if it exist in still smaller quantity, and be contiguous to the larger bronchi, it may even raise it above the usual standard by increasing the reflecting power of the bronchi, concentrating the waves of sound, and intensifying the vibration.

The increased size of the bronchial tubes, with the adjacent consolidation often met with in chronic bronchitis, usually tends to increase the fremitus; whilst the condition of the lung which exists in vesicular emphysema produces an opposite result, by interfering with the homo-

geneity of the pulmonary tissue, and thus impairing its conducting power. In certain instances, however, where inflammation supervenes in an emphysematous lung and gives rise to pneumonic infiltration and consolidation, the vibration is increased rather than diminished.

The voice, however, is not the only source of vibration in the chest walls; every thing which throws the air contained within the lungs into a state of vibration may occasion fremitus of the parietes. The only conditions essential to its production are, that the vibratory motion be sufficiently strong, and the lungs in a state to form a good medium for its transmission. The act of coughing, therefore, may give rise to it, and so may certain ronchi; but tussive fremitus and ronchal fremitus, unlike the vocal, possess little value as indications of disease.

Palpation, however, is often able to furnish us with valuable indications of thoracic disease, besides those derivable from the vocal fremitus. It may be employed to detect the friction caused by the rubbing together of the two surfaces of the pleural membrane, or of the pericardium roughened by plastic exudation; to recognise a peculiar thrill occasioned by certain forms of valvular disease of the heart; and to guide us to a more certain knowledge of the condition of the heart itself, by informing us of the character of its impulse and of the extent of the area over which it is felt. Sometimes, when the intercostal spaces are prominent, it may assist in determining the presence of fluid in the pleural cavity; for in such cases fluctuation may be felt when the fingers are applied after the manner adopted for the detection of fluid in an abscess. More generally, however, it is necessary to percuss one portion of an intercostal space with one hand whilst a finger of the other hand is applied to another portion; and sometimes, as in cases of hydro-pneumothorax, it is even necessary to have recourse to succussion, when the splashing of the fluid will be felt. It must be remembered, however, that when the lung is adherent to the parietes of the chest and a large superficial cavity exists containing a considerable quantity of fluid, certain rhonchi will give rise to fluctuation or splashing sufficiently marked to be perceptible even by the fingers, so that the mere discovery of fluctuation in an intercostal space is not of itself sufficient to justify an opinion as to the presence of fluid in the pleural cavity. The existence of fluid in a pulmonary cavity is by no means a common cause of fluctuation; but I have met with two instances in which it was well marked when the patients were placed in a semi-erect posture, leaning somewhat forward.

CHAPTER IV.

MEASUREMENT OF THE CHEST—HOW TO BE PERFORMED; ITS INDICATIONAL VALUE.

Amongst other expedients for throwing light upon the condition of the chest is mensuration. By its aid, the comparative dimensions of the two sides, and the relative positions of their different parts, may be ascertained, and the extent of movement, the precise amount of the thoracic expansion and retraction accurately determined.

The simplest mode of measuring the circular dimensions of the chest is by means of a piece of graduated tape, or a thin whalebone measure, passed round the thorax from the mesian line, anteriorly, to the spine behind. Practically, however, considerable uncertainty attaches to this mode of mensuration, from the fact that it is almost impossible to determine accurately the precise point of the measure which corresponds to the spine, and equally difficult to carry the measure round exactly corresponding portions of the two sides. With the view of obviating these difficulties, it has been suggested by Dr. Hare that two pieces of tape, each similarly graduated, should be joined together, and padded on their inner surface, close to the line of junction. The saddle thus formed, when placed over the spine, readily adjusts itself to the spinous processes, and becomes fixed sufficiently for the purpose of mensuration. Each side of the chest is thus provided with its own graduated tape, which is more readily managed than the single tape, and greater accuracy is consequently ensured.

The circumference of the chest varies so greatly in different individuals enjoying equally good health, that little would be gained by the determination of its mean size.* Moreover, such a discovery would afford no clue to the expansibility of the thorax, and therefore practically would be of little value. The really important point for investigation is, the increase or diminution of the circular dimensions of either side, as shown *by a comparison of one side with the other*. An increase will exist under the conditions already specified as contributing to the

* The average circular dimension of the chest in an adult, at the level of the sixth cartilage, is about thirty-three inches.

production of morbid expansion of the thorax;* whilst a diminution will accompany those morbid states which give rise to retraction or falling in of the parietes.† The only point to be borne in mind, beyond the necessity for extreme caution in conducting the mensuration is, that in adults the two sides of the chest are usually unequal in size, the right, on the average, being larger than the left by about the third of an inch. This irregularity does not exist in early youth, is less marked in females than in male adults, and does not obtain in left-handed persons, clearly showing that it is due to the greater exercise to which the right side is subjected.

When it is desired to measure the expansion movements of the chest during inspiration, the double tape already described will enable us to do so with tolerable accuracy. Applied to the chest closely, but not so tightly as to interfere with respiration, it indicates and enables us to read off the amount of thoracic expansion. It shows that on a level with the sixth costal cartilage the expansion accompanying tranquil inspiration in health does not average more than a quarter of an inch in a male adult, with a chest thirty-four inches in circumference; that in forced inspiration it is increased to from one and a half to three inches, and that the difference in the circular dimensions of the chest after forced inspiration and forced expiration varies from two and a half to five and a half inches, the average being about three inches. These several amounts are contributed equally, or nearly equally, by the two sides of the chest, a slight excess only existing on the right side; so that although no inference can be drawn from variations in the total expansion of the thorax, admeasurement may assist in the detection of disease, by determining the amount of difference between the expansibility of the two sides.

Another and a very convenient mode of measuring the expansion of the chest is by means of an instrument contrived by Dr. Quain, and named by him the stethometer. It consists of a case like a watch-case, on the upper surface of which is a dial furnished with an index. This case contains a single movement, by means of which a silken cord, which passes through an aperture on one side of the case, can act on the index. When the instrument is held firm on the spine with one hand, and the cord is carried round the chest to the sternum, and fixed there by pressure of the fingers of the other hand, the degree of thoracic expansion which accompanies inspiration will be indicated by the move-

* *Ante*, p. 10. † *Ante*, p. 10.

ment of the index on the dial, consequent on the traction exerted on the cord. The expansion of one side of the chest having been thus ascertained, the cord can be carried round the opposite side, and thus any difference subsisting between the mobility of the two sides at any given point can be determined without difficulty. This instrument, like the tape, exhibits any difference which exists in the general or circular expansion of the chest on the two sides; but it fails to indicate the deficiency in the antero-posterior movement, and to discover the point at which the deficiency occurs.

Another mode of measuring the chest is by the employment of callipers. The extremity of one of the limbs of the instrument is placed on the back, whilst the other is placed on the infra-clavicular or other region the expansion of which it is desired to ascertain; and when this has been done on one side of the chest, the same operation is performed on the other, care being taken to place the extremities of the instrument on corresponding portions of the chest on the two sides. The instrument is furnished with a graduated scale, and thus the antero-posterior expansion movement of the chest may be easily and accurately ascertained. The callipers differ from the tape in not showing the circular or general expansion of the chest, but they exhibit its antero-posterior movement, and can be readily applied to the discovery of any deficiency in the antero-posterior expansion of the infra-clavicular region, or of any other given portion of the chest. In this respect they are superior to the tape and the stethometer, and in the readiness of their application they are preferable to every other form of instrument.*

Another, though practically a less applicable mode of measurement is by means of an instrument proposed by Dr. Sibson. He has named it the "chest-measurer." In principle it is a calliper with a moveable branch, to which is attached a rack, having at its extremity a small graduated dial, and an index, which serves to indicate any movement of respiration. "It can readily be applied to any part of the body, and by successive applications of it over the chest and abdomen all the movements of respiration can be ascertained with minute accuracy. It indicates the rhythm of respiration, showing whether the respiration is equal to, longer, or shorter than expiration." Such is the description given of it by Dr. Sibson—a description, however, which requires to be modified in several important particulars. The chest-

* The most convenient, and, in every respect, the best form of this instrument, is that devised by Dr. G. Nelson Edwards, and described by him in the 'Med. Times and Gazette' for December 27th, 1856.

measurer indicates only the antero-posterior movement of the chest; it does not show its circular or general expansion. Hence, if trusted to exclusively, it may lead to grievous error; for when the costal cartilages are ossified, the forward movement of the parietes of the chest is by no means in the ratio of its general expansion; and in such a case, the treacherous index would indicate an undue deficiency of thoracic expansion. Again, during laboured inspiration, when the action of the diaphragm is excessive, and the bulk of air inspired is smaller than the space left by the movement of the muscle, the lower part of the sternum and the adjoining cartilages may be actually drawn in, whilst a fair amount of general expansion takes place nevertheless. Here the indications of the chest-measurer would be completely at fault. Moreover, unless great caution is observed, the very delicacy of the instrument may occasion inaccuracy; for, as pointed out by Dr. Walshe, the torsion movement of the ribs will cause an apparent increase or decrease of the forward motion, according as the moveable rack of the instrument is fixed near their lower or upper edge. Add to these sources of uncertainty the extreme nicety required in the application of the instrument whenever accuracy of results is needed,* and the length of time necessarily consumed in making such an application, and it will be obvious that, for many purposes at least, the chest-measurer is not available by the bedside of the patient. But for localising the excess or deficiency of antero-posterior motion, and ascertaining its precise amount, the instrument, like the ordinary callipers, is extremely valuable, as it thus becomes an efficient aid in the diagnosis of consolidation, with its consequent imperfect expansion, in the early stage of phthisis.

Dr. Scott Alison has suggested another form of instrument, which he has styled the "hydrostatic pneumatoscope." It consists of a small cup containing water, connected with a graduated glass tube. The cup is covered with a piece of thin India-rubber membrane, which confines the water, and yet admits of motion being communicated to it from without, so that the slightest pressure on the India-rubber membrane causes the water to rise in the tube, and so to indicate the degree of pressure. The cup and tube are fixed on a stand, and when the instrument is to be used for the examination of the chest, "the stand is placed on a table, and the cup is made to touch the patient's chest when he has expired fully. An act of inspiration is now to be slowly and fully made. The liquid rises in the tube, and the number of degrees travelled

* In proof of this fact, see 'Med.-Chir. Trans.,' vol. xxxi, note at p. 365.

over indicate the amount of thoracic elevation and advancement. When one side of the chest has been examined, the other is to be proceeded with.* "This instrument, applied over the sub-clavicular region of a patient affected with deficiency of inspiration, in the first stage of phthisis, will indicate a deficient movement." Such is the description given by Dr. Alison; but the instrument is so liable to be broken, and, if accurate results are to be obtained, requires so much delicacy and care in its application, that for practical purposes it is not equal to the "chest-measurer" or to the simpler, yet equally efficacious callipers.

Spirometry, which has received its fullest development from Dr. Hutchinson, is another mode of estimating the expansibility of the thorax. It hinges on the fact that the lungs in health contain a certain volume of air which increases in a certain ratio with the height of the individual, and that anything which interferes with their permeability or their action will alter the volume of air they can be made to receive to the exact amount of that interference. The spirometer or instrument employed by Dr. Hutchinson for measuring what he terms the "vital capacity of the chest," or, in other words, the largest volume of air which the chest can be made to contain, consists of a cylinder closed at its upper extremity, and suspended in a reservoir of water by means of two cords fixed to opposite sides of the cylinder. Each cord passes over a pulley, and has a weight attached to its extremity, and the two weights are together sufficient to counterbalance the weight of the cylinder. When the instrument is ready for use the cylinder is nearly, but not quite full of water. A pipe, which forms a continuation of the tube through which the patient has to breathe, passes under the lower extremity of the cylinder, and rises within it above the level of the water. As the patient forces the air through this tube, each cubic inch of air which he expires displaces a corresponding amount of water, and raises the cylinder to a proportionate extent, and the exact amount of air discharged from the lungs in any given expiration is indicated by a graduated scale affixed.† By means of the "spirometer" Dr. Hutchinson has ascertained that in a person five feet in height the mean volume of air expelled from the chest by the deepest possible expiration succeeding the fullest possible

* Alison, 'On the Chest,' p. 343.

† A modification of the spirometer, constructed on the principle of an ordinary gas-meter, has been suggested by Dr. Edward Smith. A full description of it will be found in the 'Medical Circular.'

inspiration is 174 cubic inches,* and that eight additional cubic inches of air are given off at 60° Fahr., for every additional inch of stature. Exceptions to this law are occasionally met with, whilst age, weight, and other circumstances modify its influence in a determinate manner; but the law is deduced from experiments upon more than 3,000 individuals, and in most instances approximates closely to the truth.†

This fact being established, however, it becomes necessary to ascertain whether any and what amount of deficiency below this healthy standard can be relied upon as indicating the existence of disease. The advocates of spirometry assert that a deficiency of from ten to fifteen cubic inches per hundred—though, possibly, arising from physiological peculiarity—is always suspicious, whilst any further deficiency is undoubtedly morbid; hence they would infer that spirometry affords sufficiently accurate data for a positive diagnosis.

Unfortunately, experience does not warrant our assenting to this proposition. The exceptions to the law Dr. Hutchinson has laid down are so numerous as to forbid our trusting to any conclusions derived from its supposed uniformity of operation. It appears that an individual's breathing capacity,‡ which is measured by the volume of air he is able to expel at one effort from the chest, is apt to vary widely from Dr. Hutchinson's "standard of health." In one person it may exceed, in another fall below the general standard. In the former case an individual with disease in his lungs may be able to expel a volume of air fully equal to the average standard of men of his height, and may thus be marked by spirometry as healthy, whilst in the latter, with a perfectly healthy chest, he may figure in the list as unhealthy. In the course of my examinations at insurance offices, as also in private practice, I have met with several well-marked examples of both of these conditions. The fact appears to be that the *individual*, and not simply the *general* standard, is requisite as a foundation for any positive conclusions as to the existence or non-existence of disease: the man must be compared with his former self, and not with the average of other men. When once the individual healthy standard has been ascertained, spirometry is a test of infinite value; but without such a standard, the practitioner will do well to regard its indications as mere hints for his guidance, to be carefully tested by other means of diagnosis.

* 'Med.-Chir. Trans.,' vol. xxix, page 157-8.

† This is confirmed by the recent observations of Dr. Balfour. See 'Med.-Chir. Trans.,' vol. xliii, p. 263.

‡ 'Med.-Chir. Trans.,' vol. xxix, p. 143.

Unlike the instruments already described, the spirometer affords no clue to the nature or situation of the existing disease. Its indications are simply those of perfect or imperfect expansion of the lungs: it does not reveal the causes of such imperfect expansion, neither does it show whether such causes exist in the lungs themselves, in other parts of the thorax, in the abdomen, or in the nervous centres, which, if acted on by disease, may interfere with the expansion of the chest on the principle of diminished nervous energy and consequent impaired muscular action.

Hence it would appear that, for ascertaining the relative size of the two sides of the chest and the exact amount of their general expansion, mensuration by the tape or by the stethometer is most effectual; whilst the callipers and the chest-measurer form the most efficient instruments we possess for ascertaining the precise amount of antero-posterior movement of the ribs and localising the earliest inroads of disease. The spirometer is chiefly valuable as enabling us to estimate the condition of the thorax rapidly though roughly, and, by means of observations repeated at intervals, to ascertain whether any recent mischief has been set up; or whether disease, known to have existed at a former period, has been making progress, or is happily arrested. Each mode of mensuration has its peculiar advantages, and each, therefore, will be made use of by the judicious practitioner, according to the circumstances of the case.

CHAPTER V.

PERCUSSION.

We have now to make ourselves acquainted with percussion, the first of those methods of examination which enable us to ascertain on what depends any alteration discovered in the form, the size, or the movements of the chest.

Everybody knows that different substances emit different sounds when struck—that a solid, inelastic body yields a dull sound, and a hollow body, with thin, firm, elastic walls, a full-toned clear sound. Further, it is notorious that the resonance of a hollow body is modified by the nature of its contents—that an empty cask, for example, is very resonant when struck, and emits a full clear sound; that filled loosely

with wood shavings or other light substance it is still resonant, but less so than before, and yields a note less full and clear; and that filled with fluid, or with tallow or any other solid matter, its resonance is lost, it is dull on percussion, and the sound it yields is short, abrupt, and dead. The same holds good in regard to the cavity of the human chest. As the different portions of the thoracic walls and of the structures which lie beneath them vary greatly in their texture, so even in health each portion of the chest emits its characteristic sound, and offers a peculiar sensation of resistance to the finger; whilst in disease alterations are produced in these respects, according to the nature, position, and extent of the physical changes which occur in the chest walls and subjacent parts. Such are the simple yet important facts on which the employment of percussion is founded, and which, strange to say, escaped observation until Avenbrugger published his researches on the subject.* Then it became evident how valuable a method of examination had been neglected; and at the present time percussion ranks among the most important means at our disposal for exploring the different cavities of the body.

The immediate object of percussion as a means of diagnosis is the determination of the density of subjacent parts, and this is inferred partly from the degree of resistance offered to the finger and partly from the sound elicited by the percussion stroke. It is obvious, therefore, that as preliminary to the use of percussion in the diagnosis of diseases of the chest, we should make ourselves acquainted with the relative position of the thoracic viscera, their several boundaries, and their physical characters, and should endeavour to attain to perfect knowledge on three most important points, viz.:

1st. *The resistance offered and the sounds emitted on percussion by the different portions of a healthy chest*, in order that we may be able to recognise any alteration in their character occasioned by disease.

2ndly. *The conditions which govern the production of sound and regulate the degree of resistance offered by a body under percussion*, so that we may be enabled to judge of any physical change in the subjacent textures by the signs elicited on percussing the chest walls.

3rdly. *The various forms of thoracic disease and their usual seat*, with the view of being able to form an opinion as to what disorder has pro-

* Published at Vienna, A.D. 1761. Avenbrugger's work, however, appears to have attracted little attention until fifty years afterwards, when Corvisart translated it into French, and introduced the practice of percussion into the French hospitals.

duced any given change, from the position such change is found to occupy in the chest.

Let us then endeavour, in the first instance, to arrive at a clear understanding on the circumstances which govern the production of sound, and regulate the degree of resistance offered to the finger by a body under percussion.

The *sensation of resistance* is regulated almost wholly by the elasticity of the vibrating body: the more solid and inelastic the mass, the greater and more unyielding the resistance.

The *sound* is dependent upon three circumstances—1st, the force of the percussion stroke; 2ndly, the nature and bulk of the vibrating body; 3rdly, the nature of the conducting medium.

The *force of the stroke* sets the molecules of the vibrating body in motion, and thus regulates the loudness and intensity of the sound, the vibrations being stronger, and the sound louder and more intense in proportion to the force of the stroke.

The *nature and bulk of the vibrating body* lead to differences in the *quality*, *duration*, *clearness*, *fulness of tone*, and *pitch* of the resulting sound.

The *quality* of the sound varies with the form of the sonorous waves, and consequently with the size, form and composition of the vibrating body.

The *duration* of the sound is dependent on the frequency with which the vibrations are repeated, and therefore is determined, *cæteris paribus*, by the molecular elasticity of the vibrating body; the vibrations being free and unobstructed, and the sound consequently well sustained when its elasticity is great; shorter and more abrupt when its elasticity is less. The prolonged ringing sound produced by striking a gong and the short abrupt sound which results from striking the thigh, are familiar examples of the fact.

The *clearness* of the sound varies according as the vibrations take place more or less freely, and interfere with or destroy each other to a greater or less extent—conditions which are regulated by the elasticity and homogeneity of the vibrating body. If it be inelastic, so that vibrations can hardly be excited, or, if excited, do not penetrate beyond the surface and are instantly destroyed, it matters not what the nature of its texture, for the result is necessarily a short, abrupt, shallow sound—a sort of dead tap—which, for convenience sake, stethoscopists term dulness. If it be elastic but non-homogeneous the vibrations will

interfere with and destroy each other, and the sound will be, more or less, dull or muffled in consequence; whereas if it be elastic and homogeneous in structure, the vibrations will be free and non-interferent, and the resulting sound will be clear. Therefore, as all clear sounds are caused by vibrations more or less free and non-interferent, and as air furnishes the medium which admits of the most free and least interferent of all vibrations, so all clear sounds emitted by the chest convey more or less an impression of hollowness; or, in other words, of the presence of air; whilst all dull sounds, in which the vibrations are necessarily short and abrupt, convey an impression of solidity.

The *fulness* of the sound, or in other words its volume, is determined by the length of the sonorous wave and thus, *cæteris paribus*, by the size of the vibrating body, the sound being *full* when the vibrating mass is large, and *shallow** when it is small—a fact which may be exemplified by the difference existing between the sound emitted by the feeblest vibrations of a large bell, and that produced by even the strongest and most intense vibrations of a small bell. And as the force of an ordinary percussion stroke could not excite any great extent of vibration except in matter possessed of considerable molecular elasticity—a property which belongs to air alone of all the contents of the chest—it follows that in percussion of the chest walls fulness of tone implies the presence of a large amount of air beneath, and shallowness of tone its comparative absence.

Finally, the *pitch* of the sound is determined by the number of equal vibrations excited in a given time—the pitch being high when the vibrations are numerous, and low when they are few in number.

Thus, then, the sounds elicited by percussion may be clear or dull, full toned or shallow toned, high pitched or low pitched; or, resulting from a combination of these conditions, they may be "clear and full toned," "clear, but shallow toned," "dull, but full toned," "dull and shallow toned." Placed in a tabular form they stand thus:

Clear toned, and its opposite, *dull toned.*
Full toned, and its opposite, *shallow toned.*
High pitched, and its opposite, *low pitched.*

* We have no word to convey our meaning adequately, but the term "shallow" is more expressive and less objectionable than any other. We constantly speak of a full-toned instrument, and as frequently of an instrument having a poor, shallow tone. The term "empty," which is often employed, not only seems to imply the presence of a cavity or hollow space, which often does not exist, but conveys an impression which in many cases, is not produced by the sound in question.

Or combinations of these sounds; thus—

Clear and full toned . .
- Normal, as over-healthy lung, constituting what is termed "*good pulmonary resonance.*"—a fair term, and may be retained.
- Abnormal, as in pneumothorax, constituting what has been styled "*tympanitic resonance*"—a fair term, clearly expressing the character of the sound, and not suggestive of any theory as to its origin; objectionable only as encumbering our phraseology.

Clear, but shallow toned
- Never normal, except over the trachea and larger bronchi. The clearness of the sound is greater, whilst its depth or fulness is less than that even of pulmonary resonance, and far less than that of tympanitic resonance. Varieties of this sound have been termed "tubular," "tracheal," "bronchial," "cavernous," "amphoric," and "cracked-pot." The four first-named terms are extremely objectionable, as implying a theory as to the origin of the sound, and though the last-named, "amphoric," with its variety, "cracked-pot," is less objectionable, it has no pathological signification, and is therefore useless, and to be avoided. The expression "abnormally clear, but shallow toned," represents the true character of this resonance.

Dull, but full toned . .
- Never normal, except when the chest-walls are fat, flabby, or œdematous, and the clear, full sound of healthy pulmonary resonance is rendered dull and muffled in consequence.

Dull and shallow toned .
- Never normal, except in the scapular and other regions, in which a thick mass of fat or muscle is interposed between the skin and the chest-walls.

These sounds constitute "dulness" of greater or less degree—a convenient and not objectionable term, and therefore to be retained. The term "wooden," as applied to a variety of this sound, has no clinical significance, and is to be avoided as a useless encumbrance to our phraseology.

A clear and full toned sound results from the free vibrations of a large and more or less homogeneous mass possessed of considerable molecular elasticity. Its normal type, as regards percussion of the thorax, is the sound emitted by a well-developed chest, the walls of which are thin and elastic, but slightly covered with fat or muscle. From this it varies in every possible degree, until in pneumothorax, where air exists in the cavity of the pleura, it reaches its extreme

point of abnormal development, and, somewhat resembling the sound of a drum, has been termed, not inaptly, "tympanitic."

A clear but shallow toned sound arises from the vibrations of a small homogeneous mass possessed of great molecular elasticity. In the chest it results from the strong vibrations of a small quantity of air, and in a normal condition of the thoracic viscera can be elicited only over the trachea or a large bronchus. Hence this type of resonance has been termed "*tubular*," "*tracheal*," or "*bronchial*." In disease it may be met with in any part of the chest where there exist small empty superficial cavities bounded by thin tense walls capable of reflecting sound, and hence, sometimes, it has been termed "*cavernous*," and at others, when it is supposed more nearly to resemble the sound emitted on striking a jar (amphora), it has been styled "*amphoric*" resonance. These terms, however (tubular, tracheal, bronchial, cavernous, and amphoric), especially the first four when applied to diseased conditions *are* extremely objectionable, as suggesting an explanation of the sound which it is not in the power of any one to verify, and which may be clinically incorrect. In most cases this clear but shallow resonance causes an impression of emptiness. A good example of it may be obtained by filliping the inflated cheeks.

A dull but full toned sound results from the vibration of a large mass, the vibrations of which are damped or muffled. It is the tone of the muffled bell or muffled drum. In the chest it may be produced by fatness or flabbiness of the thoracic parietes, by thickening of the pleural membrane, or by the presence of a thin layer of fluid in the pleural cavity; or again, by the existence of a thin superficial layer of solidified lung or other solid matter lying over healthy air-containing lung. In either case the transmissions of vibrations from the lung beneath is impeded, and the sound, though of a full-toned character, is in consequence weakened or muffled.

A dull and shallow sound is that variety of sound which is emitted by a body not possessed of much molecular elasticity or vibratile power; it is the sound which in percussion of the chest is elicited in the scapular regions, and is usually termed "dulness." It varies in degree from slight to absolute or perfect dulness, according to the vibratile power of the part percussed. Slight dulness commences directly the vibrations are shorter, and the sound, therefore, shallower than normal pulmonary resonance. Perfect dulness and extreme shallowness are attributable to the same condition, and have the same

significance, and may be typified by the sound produced by percussing the thigh or a mass of putty. Their cause is the almost instant cessation of vibration, and the consequent shortness and abruptness of the sound.

The pitch of the sound being regulated by the number of equal vibrations excited in a given time, is dependent on a variety of circumstances. In percussion of the chest variations of pitch are not of much diagnostic value; but occasionally we may be assisted in the diagnosis of tubercles by noting a difference in the pitch of the percussion sound over corresponding portions of the two lungs. Shallow tones are generally of higher pitch than full tones, and the vibrations being shorter, the pitch of the sound is higher over solidified lung than over healthy pulmonary tissue.

Lastly, all sound is influenced by *the nature of the conducting medium.* If its elasticity be great, and its composition perfectly uniform and homogeneous, the sonorous waves are transmitted clearly and readily to the ear; if it be elastic, but non-homogeneous, the waves of sound repeatedly change their medium, and interfere with or destroy each other every time they do so, and thus they either fail to reach the ear, or else reach it much diminished in intensity and clearness. If, again, it possess but little molecular elasticity, the sonorous vibrations are soon obstructed and little or no sound is heard. In percussion of the chest, where the sound results chiefly from the vibration of air contained in the lungs, the chest walls and the external air together form the conducting medium to the observer. The atmosphere, of course, forms an excellent conductor, but the fat and the muscles which cover the chest are not possessed of much molecular elasticity, and, therefore, are very imperfect conductors. Consequently, if the thoracic parietes are thick, the sound emitted by the chest is necessarily dull and muffled when it reaches the ear, however healthy the condition of the lung beneath.

These, then, are the facts on which all percussion is founded; and we shall see their bearing exemplified in the percussion of a healthy chest, as well as in those altered conditions of the thorax and of the thoracic viscera produced by various forms of disease.

When a well-developed, healthy chest is struck lightly by the ends of the fingers, it yields a clear and somewhat full-toned sound. This resonance which is often called, "a good clear sound," does not arise from the tissue of the lung, which, when deprived of air by compression, is

inelastic, and incapable of sonorous vibration, but is caused by the vibration of the elastic chest walls, and of the air contained in the pulmonary cells and bronchial tubes beneath. It derives its precise character partly from the force of the stroke, partly from the physical condition of that portion of the chest-walls which receives the stroke, and partly from the density of the subjacent matter. The primary impulse by which the sonorous vibrations are set in motion varies, *cæteris paribus*, with the force of the stroke, and produces a corresponding loudness of sound; so that a weak stroke gives rise to feeble vibration and a weak sound, whilst a forcible stroke occasions a loud sound. But in order that clear sonorous vibrations be produced, other conditions are indispensable. Not only must the stroke be sufficiently forcible—the parts struck must possess a certain degree of elastic tension; for, if the natural elasticity of the chest walls be destroyed by ossification and stiffening of the cartilages, if flaccidity exists, or if the integuments be thickened by periosteal swelling, by great muscular development, or by œdema, fat, or other cause, the sense of resistance will be increased, the vibrations and the sound will be proportionably diminished or deadened, and more or less "dulness" on percussion will result. The subjacent matter also exercises an important influence; for the larger the amount of air present, and the greater the elasticity, and the more homogeneous or uniform the composition of the media through which the vibrations of that air are propagated, the more freely will they be transmitted to the surface, and the clearer, fuller, and more prolonged the resulting sound.*

Now, the natural spongy tissue of the lung when inflated with air presents a uniform, elastic mass, well calculated to admit of free and unresisted vibration. Consequently, when an impulse is given to the thin elastic chest walls, and is propagated to such a mass, the sense of resistance to the finger is slight, the vibrations are free, and the resulting sound is proportionably clear, full, and prolonged. When, on the other hand, the chest walls are thick or inelastic, or the subjacent structures more solid, the sense of resistance is greater, and the vibrations are not transmitted to any depth in the thorax, but are short, abrupt, and quickly returned to the ear, producing more or less dulness of sound, and conveying an impression of solidity or hardness When, again, the subjacent matters are liquid, or are solids of a soft, inelastic nature, the sense of resistance is still greater, the vibrations

* See 'Encyclopædia Metropolitana,' article "Sound," by Sir John Herschel.

are almost instantly destroyed, and the resulting sound is a sort of short, dead tap, exemplifying in the highest degree what stethoscopists term "dulness."

The sensation of resistance offered to the finger is closely proportioned to the elasticity of the part which receives the stroke; and, as the elasticity of the chest is in great measure attributable to the air therein contained, it is usually found that the degree of resistance varies inversely as the clearness of the percussion sound. The upper part of the sternum and the sternal extremity of the clavicle form exceptions to this rule, in consequence of there being a large amount of air beneath, whilst the walls at the same time are bony, and incapable of yielding under percussion; but, except in the instances referred to, the resistance is slight when the sound is clear, and considerable when the sound is dull. In proof of this, the peculiar elastic vibratory sensation imparted to the fingers on percussing the infra-clavicular regions in a healthy chest may be contrasted with the dead, inelastic feel conveyed by percussion of the thigh or of the scapular regions. Indeed, it may be laid down as a general rule that whatever deadens vibration diminishes the clearness of the sound, and increases the sense of resistance to the finger.

Thus it will be seen how anything which affects the elasticity, the thinness, and the tension of the chest-walls, or which alters the quantity of the contained air, removes the lungs from the parietes, or modifies the physical condition of the lung substance, must tend to alter the character of the sound resulting from percussion, and no less so the sense of resistance offered to the finger. And it will be readily understood that those chests, and those portions of a healthy chest, which are most elastic, least covered with fat and muscle, and which have beneath them the spongy air-filled tissue of the lungs, possess the greatest vibratile properties, emit the clearest and fullest toned sound on percussion, and, with the solitary exceptions before alluded to, offer least resistance to the finger. This being borne in mind, let us briefly inquire into the results of percussion on the various regions of a healthy chest, as it will then be seen how closely the character of the sounds and the sense of resistance to the finger accord with the condition of the thoracic parietes and with the anatomical character of the subjacent structures.

The *supra-clavicular* regions, which contain the triangular apices of the lungs, emit a tolerably clear, though shallow sound on percussion.

They vary, however, in this respect according to the degree of inflation of the lungs and the extent to which their apices rise above the clavicle. In some instances, especially where there is great muscular development, or much fatty deposit, the outer part of this region emits a dull sound, and is very inelastic and resistant.

In the *clavicular regions* the resonance is very clear towards the sternum, owing to the proximity of the trachea, but the resistance is considerable, owing to the bony nature of the chest walls; the resonance is still clear, but more distinctly pulmonary* in its character about the centre of the bone; much less clear, and, in some instances, almost dull towards the humeral extremity.

The *infra-clavicular regions* afford a good type of pulmonary resonance. The sound they yield is clear and full, the resistance slight, the elasticity manifest to the finger.

The *right mammary region* gives a clear, full sound over nearly its whole extent on ordinary gentle percussion; but, when the stroke is firm and forcible, it brings out slight dulness below the fourth interspace, consequent on the presence of the liver behind the shelving border of the lung. Between the third and fifth ribs, close to the sternum, the sound is sometimes deadened by the presence of the right auricle and the superior angle of the right ventricle of the heart.

The *left mammary region* also emits a clear, full resonance, except in those portions lying below the fourth sterno-costal articulation, where the presence of the heart deadens the sound and increases the resistance. In women a clear sound can be obtained in the *mammary* regions only by means of firm pressure on the breasts.

In the *infra-mammary*, on the right side of the chest, the resistance increases, and the sound becomes gradually duller, until at the lower margin of the chest it is perfectly dull except on deep inspiration, when the lung forces the liver down, pushes in front of it, and gives rise for a time to partial resonance on gentle percussion. Dulness, however, is still perceived on increasing the force of the stroke, and thus bringing out the shallowness which results from the presence of the liver behind. The lower part of this region on the left side is usually dull, in consequence of the presence of the spleen and the left lobe of the liver; but sometimes, when the stomach is distended with flatus, the resonance becomes exceedingly clear and full-toned, or, in other words, tympanitic below the sixth rib.

* The resonance yielded by those portions of the chest which have beneath them the spongy air-filled tissue of the lung has been termed "pulmonary" resonance.

The *supra-sternal* yields an unusually clear but shallow-toned resonance, owing to the proximity of the trachea.

In the *superior sternal*, down as low as the second rib, the resonance is unusually clear in thin persons, owing to the presence of the trachea and its bifurcations; but in certain persons a large quantity of fat accumulates at the upper part of the mediastinum, obscures the tracheal sound, and causes dulness on percussion. From the bony nature of its anterior boundary, this region is resistant under the finger.

In the *inferior sternal* the presence of the heart and great vessels, together with the left lobe of the liver, occasions dulness on percussion, which is modified in some degree during inspiration by the overlapping of a portion of the lung, and sometimes to a still greater extent by the resonance which results from flatulent distension of the stomach.

The *axillary regions* are extremely resonant at their upper part, from having beneath them the parenchyma of the lung and the main bronchi. Below the fourth interspace on the left side a clear sound is elicited on gentle percussion, and a duller sound by a more forcible stroke, which brings out the dulness occasioned by the liver; whilst on a level with and below the sixth rib complete dulness, whether with gentle or forcible percussion, results from the presence of the liver.

In the *infra-axillary* region, on the right side, there is complete dulness on percussion: on the left side pulmonary resonance may be elicited by gentle percussion, but is modified by the presence of the stomach and spleen.

In the *superior and inferior scapular* regions the sounds are dull, and the resistance great, in consequence of the thick muscular tissue which fills the superior and inferior scapular fossæ.

The *interscapular* is more resonant, and offers less resistance than the other scapular regions, though it does not yield a sound so clear or full as good pulmonary resonance, in consequence of the intervention of soft muscular tissue. With the view of eliciting a clear sound from this region, it is necessary to make the patient cross his arms in front, incline his head forwards, and bend his back, so as to put the muscles upon the stretch, and make them as tense and thin as possible.

The *inferior dorsal* regions emit a clear sound on gentle percussion, but a dull sound on forcible percussion, especially on the right side, owing to the presence of the liver. At the lower part of the left side the spleen and the stomach and intestines modify, in their respective ways, the character of the percussion sound and the sense of resistance to the finger.

Having thus traced out the principle of percussion, and seen that its effects do practically accord with what theory would have led us to expect, our attention must next be directed to the differences in the sound emitted, and in the resistance offered by the chest in different individuals, irrespectively of actual disease. In childhood and in youth the cartilages are more elastic, and the chest walls more susceptible of vibration, and the resonance, therefore, is clearer, and the resistance less than in middle age; whilst, in middle age, for the same reasons, the chest, though less clearly resonant and less yielding than in youth, is more so than in old age, when the cartilages are ossified and stiffened. In males the resonance of the chest is usually less clear than in females, partly on account of the greater development of the pectoral muscles, and partly in consequence of the greater ossification of the cartilages. In thin persons, again, the resonance of the chest, for obvious reasons, is clearer than in stout. Some chests, too, are occasionally met with in which, quite irrespective of visceral disease, or of any external peculiarity of the parietes, the resonance is much clearer, or else less clear than in the average of healthy chests. That the dulness is not due, as is often stated, to deficiency in the powers of expanding the lung, is obvious, from the fact that the persons in whom it exists are not necessarily short breathed; and I am rather inclined to attribute this peculiarity to a difference in the relative size of the respiratory and circulatory apparatus, the lungs being relatively small, and of necessity, therefore, more fully inflated when the resonance is clear; large, and therefore less fully inflated when the resonance is dull. It is almost impossible, from the nature of the case, to adduce direct proof of this position; but I am the more disposed to consider it correct, from the fact, that after forced inspiration, whilst the lungs are fully expanded, and the chest filled with air, the differences previously observed almost entirely disappear.

We will now pass on to the modes of employing percussion, and to the various considerations relative thereto. When percussion was first introduced as a means of diagnosis, it was practised directly or immediately on the chest. This "immediate" percussion—the only method of percussion employed by Avenbrugger and Laennec—is performed by striking the chest either with the palmar surface of the fingers held fully and firmly extended, or with the two or three first, or the four fingers of the right hand, held closely to each other, and so bent as that their points may be brought down perpendicularly and simultane-

ously on its surface. If the blow be sharp and quick, and the chest covered with a towel, shirt, or some thin dress, or other covering kept tightly stretched by the left hand, a sound is produced which varies with the condition of the subjacent textures, and furnishes valuable information.

But there are many objections to the general employment of "immediate percussion." In the hands of the unskilful or inexperienced operator it causes pain and suffering to the patient; it cannot be employed satisfactorily in examining the intercostal spaces; and when the integuments are anasarcous, emphysematous, loaded with fat, or flaccid, it fails in eliciting any resonance, and becomes therefore absolutely useless. These difficulties are in great measure overcome by interposing some solid substance between the chest and the percussing agent; hence, "mediate percussion," the invention of M. Piorry, has superseded the older method of "immediate percussion." M. Piorry employed as his pleximeter* or percussion-plate a piece of ivory or wood about a line in thickness, and an inch and a half in diameter, provided with two projections or handles placed at right angles to its surface, and almost at opposite points of its circumference. These enable the operator to hold the plate firmly and evenly on the chest with the left hand, whilst he percusses with the right. Various modifications of this pleximeter have been proposed, made of wood, leather, metal, and other substances; but amongst them, the only two deserving of notice are:—1st, a flat piece of ordinary India rubber, as suggested by M. Louis; and 2ndly, a pleximeter made of vulcanite. Unlike M. Piorry's wood or ivory-plate, the ordinary India rubber can be adjusted to the chest-walls with tolerable accuracy even in the thinnest persons, and its elasticity is such, that it breaks the force of the percussion stroke, and saves pain. At the same time, however, it deadens the sound—a circumstance of little consequence, when the patient can bear forcible percussion, inasmuch as our inferences must be drawn from a comparison of different parts, rather than from the actual sound produced, but of material importance when the patient's chest is tender, the percussion stroke necessarily very gentle, and the sound elicited therefore weak. In some of these cases, when the India rubber is used, it is difficult to catch and recognise slight differences of tone which are manifest when another medium is employed. This, however, does not hold good in respect to the plexi-

* So named from πλῆξις, percussion, and μέτρον, a measure.

meter made of vulcanite, which is in every respect well adapted for the purpose, and is an excellent conductor of sound.

In the method of percussion now generally had recourse to, the use of artificial pleximeters is discarded, and the index and middle fingers of the left hand are made to take their place. They offer the advantages of being constantly at hand, capable of ready application in the intercostal spaces, and of perfect adjustment to the various inequalities of the chest. They may be applied singly or together, parallel to the ribs, or at various angles with them, and not unfrequently, more especially in thin persons, it is desirable to vary the direction in which they are applied. Most persons apply their palmar surface to the chest, and this forms the most convenient and most efficient pleximeter; but some persons prefer the dorsal surface, whilst others apply the palmar surface when they wish to ascertain the resistance of the chest walls, and the dorsal when their object is simply to elicit sound. One great advantage of this digital pleximeter is, that the sound made by striking it is not so loud as that caused by striking most of the artificial pleximeters, and does not to the same extent interfere with that which is dependent on the condition of the chest.

As percussing agent, no instrument hitherto contrived proves equal to the fingers, with their tips brought to precisely the same level. When gentle percussion is practised, the index-finger may be used alone; and when a more forcible stroke is required, the index and middle fingers supported by the ball of the thumb. Sometimes the first three fingers may be used with advantage, instead of two; and sometimes again, in rough examinations of an extensive surface, when the four fingers of the left hand are employed as a pleximeter, the four fingers of the right hand may be used for percussing. In this latter case they should be held firmly extended, and made to fall horizontally, instead of perpendicularly, the palmar surface being lightly tapped against the dorsal surface of the fingers of the left hand. Thus, with the fingers of the left hand as a pleximeter, and those of the right hand as a hammer, the physician is ready armed for the operation of percussion, and can have recourse to its various modifications, according to the requirements of the case. In the determination, however, of the method to be adopted, much must depend upon the part to be examined, and upon the plan he is in the habit of employing.

Some physicians, though employing the fingers of the left hand as a pleximeter, make use of various percussing agents in lieu of the fingers

of the right hand. Thus hammers and other instruments are to be found in shops, calculated in the opinion of their inventors to supercede the hammer provided for us by nature. Practically, however, they fall short of the instrument they are intended to replace; for not only do they deprive the operator of the evidence derivable from the sense of resistance in the parts percussed, but they are apt to cause alarm to timid patients. Sometimes, however, I have derived assistance from the use of a small hammer, the end of which has a piece of India rubber affixed to it. It is the invention, I believe, of Dr. Winterlich, of Würzburgh, and possesses the advantage of producing a remarkably clear tone, and one which can be relied upon, even in the hands of an inexperienced operator. I never employ it to the exclusion of my fingers, but sometimes have recourse to it in confirmation of the evidence they afford; and it has happened on more than one occasion that in the early stage of phthisis I have been enabled thus to satisfy myself of the existence of slight dulness on percussion, and of a variation in the pitch of the percussion note on the two sides, of which I had previously entertained some doubts.

It need hardly be stated that the practice of percussion demands considerable manual dexterity, and that the correctness of its indications depends in great measure upon the mode in which it is performed. It may be well, therefore, to direct attention to certain precautions which are necessary to ensure satisfactory results.

In the first place, it is essential that the position of the patient should be rigidly attended to, and that, whether he be lying down, sitting, or standing, his body should rest on the same plane and his limbs be similarly disposed on either side, for the slightest irregularity in that respect gives rise to differences in the sounds elicited from the two sides of the chest. Sometimes the state of the patient is such as to render a recumbent posture necessary; but when this is not the case, the sitting or standing posture should be selected, for when the patient is lying down in bed not only are the sounds deadened by the effect of the bed-clothes, but the physician is often forced, for the purpose of examination, to place himself in an awkward and constrained position by no means favorable to accuracy of observation. Whilst the anterior surface of the chest is being examined, the patient should be made to hold himself upright and allow his arms to hang loosely on either side; to cross his arms in front and bend slightly forward whilst the back is

being examined, and to raise and clasp his hands above his head whilst the lateral regions are under examination.

Secondly. Attention should be directed to the condition of the thoracic parietes, inasmuch as, if "immediate" percussion be practised, the flaccidity of a mass of relaxed muscle would interfere with vibration and deaden sound, whereas that very muscular relaxation would admit of a closer approximation of the finger or pleximeter to the chest walls, and so would tend to render the sound clear if "mediate" percussion were employed. Hence, before percussion is had recourse to, the muscular tissue covering the thoracic walls should be put upon the stretch and rendered tense in the one case, but allowed to remain in a state of relaxation or flaccidity in the other.

Thirdly. The two sides of the chest should be percussed at precisely the same stage of the respiratory act; for the expansion of the lung during full inspiration not only gives rise to a decided increase in the superficial extent of surface over which good pulmonary resonance can be elicited, but, by diminishing the density of the lung substance, renders the percussion sound clearer and the sense of resistance less. Moreover, the expanded lung pushes down the liver and spleen and presses in front of the heart, giving rise to more or less resonance on percussion, where there would otherwise exist well-marked dulness. The reverse obtains after full expiration; and as the difference is very great in all these respects between the chest after a full *in*spiration and a deep *ex*piration, it behoves us in all cases of delicacy to exercise great caution in selecting the same moment or the same period of respiration for percussing the two sides. Perhaps the best method of accomplishing our object is by desiring the patient to expire deeply, or else take a deep inspiration and then hold his breath; we may then make sure for a time, at least, of having both lungs in the same state.

In disease, this caution is especially needful; for if tubercles or any disease which interferes with pulmonary expansion exist in one lung and not in the other, the clearness of the resonance and the area over which it is heard will not increase on inspiration, as they ought to do, on the diseased side, and the difference between the two sides thus rendered manifest will furnish valuable information. On the other hand, the dulness resulting from the presence of tubercles or of any disease producing pulmonary consolidation, becomes particularly apparent when the air has been expelled from the lung by a full *ex*piration; and thus differences existing between the two sides may often be

detected in delicate cases when they have previously escaped observation. So, again, when any disease exists on one side only of the chest, of a nature to prevent the expulsion of the air and the collapse of the lungs during expiration, there will not be a proper reduction in the clearness of the resonance nor of the area over which it is heard on that side after a full expiration; and this very point may sometimes enable us to determine the presence of some aneurismal or other intra-thoracic tumour pressing upon and obstructing the main bronchus.

Fourthly. Care should be taken to compare corresponding portions of the two sides of the chest; and as in percussion, just as in inspection and mensuration, our inferences as to the condition of the lungs should be deduced from the comparative rather than from the absolute sound emitted by different parts of the chest, it is essential that the same conditions in every respect should be observed in percussing the two sides. The best practical rule is to apply the finger or pleximeter with equal firmness to both sides of the chest in succession, to examine corresponding portions of the chest, and to percuss with equal force on both sides. Due allowance must of course be made for the position of the heart, liver and spleen, and for the alterations in their position which necessarily accompany the act of respiration.

Fifthly. In all cases, but more especially in doubtful cases, it is desirable to repeat the observation several times at different stages of the respiratory act and while the patient is in various postures. By taking these precautions, a careful observer will rarely fail to arrive at a correct conclusion; for any uncertainty which may have existed at one examination, in one posture, and at one period of the respiratory act, will be cleared up by an examination at another.

Sixthly. The greatest care should be taken, not only to apply the finger or the pleximeter to precisely corresponding parts on the two sides, but to apply it firmly on the spot to be examined, so that it may be closely and uniformly in contact with the chest walls. No extraneous condition exercises so great an influence in modifying the sound, and nothing, therefore, demands more constant and more jealous attention in instituting a comparison between any two corresponding portions of the chest. If, for instance, in the examination of a stout person, the finger or pleximeter be applied lightly at one time and firmly at another, a dull sound will be produced in the one case, from the muscles and integuments being alone influenced by the force of the percussion stroke, whereas in the other a clear, full sound, will be

emitted in consequence of the vibrations excited in the air contained within the lung. The rule to be ever borne in mind is, that the finger or pleximeter must be applied with equal force on both sides and always with sufficient firmness of pressure to render it, so to speak, a part of the organ about to be percussed.

Seventhly. The act of percussion should be performed by a movement of the wrist alone, the forearm and the arm being held perfectly motionless. By this means the pain or uneasiness occasioned by the weight of a blow in which the arm takes part is avoided and percussion can be practised with care and nicety, the force of the blow regulated, a precisely similar blow therefore given to different parts of the chest, and a uniformity obtained in the character of the sound elicited by successive blows at the same spot.

Eighthly. Throughout the examination, the percussing fingers should be kept at the same angle, in respect to the chest walls, and should be made to fall upon the pleximeter simultaneously. Percussion, practised with the percussing fingers, held at varying angles with the chest, is a fertile source of doubt and perplexity to the inexperienced operator, who thus elicits a new and different sound by each act of percussion, thus rendering the operation valueless, and likely to lead to an erroneous diagnosis.

Ninthly. The object of percussion being to determine the position and the density of the thoracic viscera, the force of the stroke should be proportioned to the depth at which the part to be examined is seated—gentle when it is superficial, more forcible when it is deeper seated. In this way, the density of the structures at various depths within the chest may be determined, the clearness or dulness of the sound varying greatly in many instances with the force of the stroke. Thus, the sound elicited from the lower part of the right axillary and mammary regions will be clear or dull according as the stroke is gentle or strong; or, in other words, according as the superficial lung substance is alone percussed, or the subjacent liver is made to feel the force of the impulse. It is obvious, therefore, that in every instance both gentle and forcible percussion should be practised, as by such means alone is it possible to arrive at a correct conclusion respecting the real condition of the structures at various depths within the chest.

Tenthly. Care should be taken that the percussion stroke be not strong enough to occasion pain to the patient. All fear on this score may be obviated by never giving a "heavy" blow. A sharp, quick rap,

or, in other words, a blow lightly given, is best calculated to impart the requisite impulse to the chest walls; and the speedy withdrawal of the fingers removes all impediment to free vibration. This, therefore, is the mode in which percussion should be performed, the only exception being in certain cases of disease, hereafter to be described, in which it is desired to elicit a sound known as the "cracked pot" sound, the "*bruit du pot fêlé*" of Laennec. In these cases, to which I shall presently allude, the blow should be firmer, heavier, and more sustained, as favouring those conditions on which the production of the sound depends.

Having thus explained the theory of percussion, described the method of performing it, and detailed the character of its signs in a healthy chest, it only remains to point out the alterations those signs may undergo, and how far such alterations can be made available in the diagnosis of disease.

From what has been already stated, it will be obvious that anything which interferes with the density of the matters which lie beneath the part struck will usually change or modify their molecular elasticity, and will thus occasion either an increase or a decrease in the clearness, fulness and duration of the percussion sound, and in the sense of resistance to the finger. Thus, it is found that in proportion as the subjacent textures are rendered more dense than natural, and, being so, are situated more or less superficially in the chest, so does the percussion sound pass through every gradation, from normal clearness to absolute dulness, the duration of the sound becoming at the same time proportionably shorter, and the sense of resistance greater. On the other hand, when the subjacent structures are of less than their normal density, precisely the reverse obtains: the clearness of the percussion sound is abnormally raised, its duration prolonged, and the resistance to the finger lessened. This holds good in all cases in which the quantity of air is not so extreme as to put the chest walls upon the stretch, and thus, by excessive tension, to interfere with their vibration. It is not necessary, at the present moment, to go into the particulars of every cause which may give rise to one or other of these variations; it is sufficient that their true import be recognised, and that it be fully understood that the precise nature of the change which has occurred can only be determined inferentially, by a due consideration of the position and extent of the mischief, the concomitant symptoms, and other circumstances. This much, however, it may be well to point out, that whether the cause of

the increased density be in the chest-walls, the lung-substance, the bronchi, the mediastina, or the pleural cavities ;—whether it be that the chest-walls are loaded with fat or muscle, or infiltrated with serum ; the parenchyma of the lung imperfectly inflated, as an effect of spasmodic asthma, or of any obstructive disease in the upper air-passages,* or else congested or inflamed, infiltrated with tuberculous matter, or otherwise solidified ; the bronchi enlarged or thickened, or else clogged with muco-purulent or other secretion ; the mediastina loaded with fat or other materials ; the pleural cavities filled with liquid or solid matter—the result is in all cases the same, namely, the production of more or less dulness on percussion, and decreased duration of sound, with increased resistance to the finger ; and, on the other hand that, provided the tension of the thoracic walls be not so great as to prevent free vibration, whatever diminishes density has a direct tendency to favour free vibration, to increase the duration and clearness of the percussion sound, and to lessen the resistance to the finger, whether the cause of diminished density be in the lung itself, as in emphysema, or in the pleural cavity, as in pneumothorax, or be found in the thinness, tension, and elasticity of the chest walls.

It must always be remembered, however, that the percussion sound varies in different individuals. Thus in some instances it is unusually clear, and in others unusually dull, over the entire chest, without the existence of any thoracic disease, and without its being possible to discover any extraneous cause for the peculiarity. It is only when dulness or unusual resonance on percussion exists on one side of the chest, and not on the other, or at any portion of one side as compared with the corresponding portion on the other, that any certainty can be felt as to the existence of disease.

Further, it must be borne in mind that in certain cases the *cause* of dulness or of increased clearness of resonance may not be necessarily fixed in position, nor the position of the sign therefore fixed or unchangeable. For instance, when the dulness is dependent on effusion within the pleura, or the resonance on hydro-pneumothorax, the fluid in either case will gravitate to that part of the pleural cavity which is made most dependent by the position of the patient, whilst the air, in the latter case, will as certainly rise and occupy the part which is placed uppermost. Thus, when the area over which dulness or increased clear-

* This holds good unless emphysema exists, in which case the increased density of the lung tissue is thereby masked.

ness of resonance exists is found to shift according as the patient lies on one side or on the other, reclines backwards or leans forwards; the fact of its so shifting affords valuable information respecting the nature of the mischief, and often enables us to discriminate between the dulness resulting from solidification of the lung and that which is caused by liquid effusion into the pleura. But mere unchangeableness in the position of dulness is not a *certain* criterion as to the existence of pulmonary solidification; for where adhesions have taken place between the two layers of the pleura, the movement of the fluid is restrained or prevented, and the boundaries of the dull sound are necessarily fixed and unvarying.

Another modification in the relationship subsisting between the clearness and duration of the percussion sound, and of the resistance offered to the finger, is sometimes met with in cases of phthisis, viz., increased clearness and duration of the sound coexisting with increased resistance to the finger. Practically, the existence of such an unusual complication is not of much importance, as the mischief which causes it can hardly fail to be discovered by other means. Its origin, however, should be noted and understood. The conditions necessary for its production are such as exist naturally in the upper part of the superior sternal region, viz., resistant chest walls, and an unusually large volume of air beneath. When they are found elsewhere on the chest, they are generally dependent on the formation of a superficial cavity in the lung, with a thin, tense, indurated wall adhering to the parietes of the thorax, and increasing its resistance.

M. Piorry and others have described a great variety of percussion sounds, to each of which they have endeavoured to attach a different significance; and those persons who are curious in nice distinctions may consult the works of those authors. But repeated observation has led me to the conviction that many of the sounds described exist only in the imagination of their discoverers, and that others which are met with occasionally cannot be connected with any peculiar physical change, and therefore have no pathological significance. *Practically*, then, the changes of sound to which it is necessary to direct attention are very few in number. Even these may be referred to one or other of the classes of sound described at the beginning of this chapter; and I am satisfied that in most cases it is better thus to describe them than to introduce terms which, however appropriate in certain instances, possess no prac-

tical significance, and have the great disadvantage of needlessly encumbering our phraseology, and confusing a study already sufficiently complicated and obscure.

Of these changes the first to be mentioned is a modification of the full, clear toned resonance elicited over healthy lung. It is abnormally clear and full toned, of unusual duration, and accompanied by a condition of the chest walls which renders the sense of resistance to the finger slight, and gives the impression of tension and elasticity. It is termed tympanitic resonance, and, as its name imports, resembles the sound obtained by striking a drum. It requires for its production a large space filled with air and bounded by tense elastic walls capable of reflecting sonorous vibrations. Hence it is heard of maximum intensity in cases of pneumothorax. If, however, the tension of the chest walls be extreme, not only is their power of vibration diminished, but the included air undergoes compression, the vibratory oscillation of its particles, upon which the sound depends, is interfered with, and the tympanitic quality is lessened or destroyed. So that, although a tympanitic tone is usually indicative of air in the pleural cavity, the mere fact of its not being present in full perfection does not necessarily denote the non-existence of pneumothorax.

There are two, and only two, other conditions under which this abnormally clear and full toned resonance is met with occasionally in the chest, namely, during the existence of emphysema with bulging of the thoracic parietes, as also, though more rarely, when there exists a very large tuberculous excavation in the lungs, with tense elastic walls.* In neither case, however, is the term "tympanitic" so appropriate as in most cases of pneumothorax, for although the sound is often clearer, more full toned, and of longer duration than in healthy pulmonary resonance, it is seldom so truly tympanitic as in the cases referred to, and the chest walls are more resistant.

A peculiar sound sometimes accompanies tympanitic resonance, which has been termed metallic clanging, or metallic resonance. A good imitation of it may be produced by covering the ear with the palm of one hand and lightly tapping on the back of it with the other. It is a clear ringing sound of a metallic character such as may be produced by a sharp blow on an empty cask. It may be generally obtained by sharp and somewhat forcible percussion in those cases of pneumothorax which

* This is a rare occurrence, but I have met with three well-marked examples of it.

are characterised by much bulging of the chest and consequent excessive tension of the parts, and not unfrequently may be produced over large empty thoracic cavities with tense walls. The conditions which impart this ringing character to the percussion sound are the force and sharpness of the stroke, and the tension of the walls of the air-containing cavity. The phenomenon is not of much importance in a diagnostic point of view, but is interesting as showing how completely the causes which modify the production of sound under ordinary circumstances, coincide with those which regulate its emission by the chest.

The next change is also a modification of the ordinary clear sound of pulmonary resonance. The sound is clearer but more shallow than that of healthy pulmonary resonance, and usually is of a higher pitch. The trachea and a large bronchus yield this sound in perfection, and hence it has been termed "tubular," "tracheal," or "bronchial." It is the natural sound emitted in the supra-sternal region, and must be regarded as morbid only when it exists in regions which do not usually yield this peculiar sort of resonance. Small empty superficial cavities with tense walls furnish excellent examples of it, and hence it has been termed "cavernous," but it may be also met with when any of the superficial bronchi are dilated; when a thin layer of partially condensed lung intervenes between the chest walls and a large bronchus, and in the inner part of the infraclavicular regions when the lung adherent to the upper and anterior part of the chest is pushed upwards by pleuritic effusion, and is more or less condensed by pressure. In these latter instances, inexperienced and careless auscultators are apt to mistake the nature of the disease, and to regard the unusual clearness of the percussion sound, and the increased resonance of the voice which accompanies it, as indicating the existence of a vomica. However, as the sound in question may arise from a variety of causes, it is far better to describe it as an abnormally clear, but shallow resonance, than to employ either of the terms "tubular," "tracheal," "bronchial," or "cavernous," each of which suggests a theory as to its mode of origin the correctness of which it is impossible to verify.

A modification of this clear, but shallow toned percussion sound, resulting from the size of the cavity which emits it, is known as the "amphoric" sound, and derives its title from its close resemblance to the sound obtained by percussion of an empty jar (amphora). Unlike the tympanitic sound above described, which gives an impression of fulness, this amphoric note is suggestive of shallowness or emptiness

It is produced in perfection if, when the mouth is closed, the cheeks are inflated, but not too tensely, and then filliped with the finger. Its most common source is a large superficial cavity with thin tense walls, and hence it is most commonly indicative of consumption; but a sound of a more or less amphoric character may occur under certain other circumstances, to which I shall presently have occasion to allude.

Sometimes in connection with this amphoric resonance, but occasionally without it, there is heard a peculiar sound somewhat resembling that produced by striking a cracked earthenware or metal jar, or other vessel. This is the *bruit du pôt félé* of Laennec, and is commonly called the "cracked-pot sound." It was formerly attributed to the forcible collision of air and liquid; but the fallacy of this opinion is made evident by three facts, viz.—Firstly, that the sound is producible in cavities in which no gurgling or bubbling sounds can be heard, and in which, therefore, it is probable no fluid exists; secondly, that it cannot be produced unless there is a free communication between the cavity and the external air, whereas, if it depended solely on the collision of air and liquid, the sound ought to be produced as well when the cavity is closed as when its communication with the external air is uninterrupted; and thirdly, that it is met with occasionally when no abnormal cavity exists, and when the physical and general symptoms, confirmed by the subsequent career of the patient, contra-indicate the existence of any serious disease. Therefore another explanation must be sought; and the schoolboy's trick of forming a hollow with the palms of the two hands and striking the back of one of them against the knee, furnishes a good illustration of its mode of production. If when the hands are thus struck, a portion of the contained air be forcibly expelled by the blow, a peculiar sound will be produced analogous in its character to the cracked-pot sound; whereas, if no air be suffered to escape, the sound produced will want the peculiar cracked pot character. In short, when this sound is met with in the chest, it is occasioned by the sudden expulsion of air and its forcible contact with the sides of the passages through which it is driven. It appears in fact to be a compound sound made up of the ordinary percussion sound of the part which, in these cases, is always a loud, yet short, hollow, metallic sound, and of a peculiar hissing noise resulting from the sudden and forcible expulsion of air from the lung beneath, through passages in most cases constricted or otherwise altered in character. The more elastic and yielding the chest walls, the heavier and more sustained the percussion stroke; and

the more free and uninterrupted the communication with the bronchi and upper air-passages, and through them with the external air, the larger, *cæteris paribus*, will be the quantity of air expelled by each act of percussion, the less impediment will there be to its escape, the more rapid will be its current, the more forcible its contact with the sides of the air tubes, and the louder therefore the cracked-pot sound. Hence, whenever our object is to elicit this sound, the patient should be desired to hold his mouth open so as to remove all possible obstruction to the free escape of the air, and to turn his mouth towards us, so that we may the more readily catch the sound;* whilst we on our part should not only place the finger which receives the stroke in an intercostal space, in order that the full force of the stroke may be felt by the lung beneath, but should employ somewhat slow and heavy percussion so as to maintain the direction of the impulse and prevent the resilience of the chest walls. If we take these precautions, and a large superficial cavity exists, and its walls be hard and tense, its communication with the external air through the bronchi free, and the thoracic walls beneath which it lies elastic, we shall never fail to elicit the distinctive character of the sound. But if the cavity be deep-seated in the chest, and consequently protected against the force of the percussion stroke; if its walls be dense, soft, relaxed or inelastic, so that very little impulse can be propagated to its contents, and little or no air, therefore, expelled by the act of percussion; if its communication with the bronchi be obstructed so that no portion of the contained air can be expelled, and the remainder cannot be greatly agitated; or if the mouth and nares be closed so that the escape of air is thereby prevented, then the conditions essential to the production of the cracked-pot sound are absent, and however skilful the operator, he will assuredly fail to elicit its peculiar character.

What, then, is the diagnostic value of this cracked-pot sound? In most works on auscultation it is said to denote the existence of a cavity, and by the majority of the profession it is still believed to do so. But I have often known those who thus interpret it misled as to the character of the existing disease; and I do not hesitate to affirm, as the result of long and careful observation, that it does not necessarily indicate the existence of a cavity. Dr. Hughes Bennett goes so far as to

* This precaution, though long adopted by many auscultators, was never, I believe, insisted on in print until Dr. Cotton enforced it in a paper on the cracked-pot sound, published in the 'Lancet' for 1857.

assert* that a cracked-pot resonance may be elicited in various diseases of the chest, and even when the chest is perfectly sound. This statement, however, is thoroughly opposed to common experience, and must have its foundation in some use of the term "cracked pot," which is not usually recognised in practice; for, if care be taken in applying the finger or pleximeter firmly to the chest-walls, so as to avoid the production of the jarring sound which results from the forcible expulsion of air from beneath the finger when carelessly applied, a cracked-pot sound is rarely producible. Nevertheless, it does sometimes occur under circumstances not commonly supposed to give rise to it. In no instance have I heard its distinctive character more marked than in the case of a young man, admitted into the York Ward of St. George's Hospital in the autumn of 1855, suffering from acute pneumonia of the upper lobe of the right lung. So strongly was it developed in that case, that several gentlemen who examined the patient insisted on the presence of tuberculous excavation, and yet the patient recovered perfectly in a fortnight, healthy breathing was re-established, and the cracked-pot resonance ceased. In children, too, who are suffering from bronchitis a jarring sound, resembling a cracked-pot resonance, may be sometimes elicited. My belief, therefore, is, that this sound simply indicates the sudden and forcible expulsion of a quantity of air from a space or spaces possessed of a certain degree of resilient elasticity, and through passages having a free communication with the external air, but, probably, somewhat flattened, contracted, or otherwise altered. Tuberculous or other cavities in the condition of those above alluded to are undoubtedly its most common source, and therefore it usually indicates their existence; but, as already stated, it may arise under certain conditions in pneumonia, and when, as in children, the elasticity of the chest walls is great, it may be developed to some extent during an attack of bronchitis, even when no solidification of the lung exists.

I would maintain, then, that wherever its presence is detected, its true significance must be carefully gauged before any diagnostic value is attached to it. Care must be taken to ascertain in every instance that it is not caused by the imperfect application of the finger or the pleximeter to the chest walls and the expulsion of the air from beneath it, for nothing proves a more fertile source of error to inexperienced operators than the sound which is thus produced. When this source of fallacy is guarded against, we may generally arrive at a correct con-

* 'Edinb. Monthly Journal,' January, 1856.

clusion as to the cause of the phenomenon by a careful examination as to the presence or absence of abnormal resonance or dulness on percussion, and the existence or non-existence of general and auscultatory signs of pulmonary excavation; but when there is pneumonic consolidation of the upper lobe, the diagnosis is at all times extremely difficult. On the whole, therefore, it must be concluded that this cracked-pot sound, though valuable as an accessory sign of tubercular excavation, is not a symptom to be implicitly relied upon, and in no case can be regarded as of itself sufficient to warrant a positive opinion, unless corroborated by other symptoms, general as well as physical. When strongly developed, it justifies the gravest suspicion as to the presence of some abnormal excavation in the lung, and, if persistent, may almost be regarded as pathognomonic of a vomica. It must be remembered, however, that it is liable to cease from the obstruction of the bronchus leading to the vomica, and, by the filling of the vomica with fluid secretion, and by the collapse or cicatrization of the vomica, and therefore that its temporary or even permanent cessation must not be regarded as evidence of the non-existence of a vomica.

The last change worthy of mention is a modification of the ordinary dull sound of percussion; and, from the fact that it closely resembles the peculiar sound obtained by mediate percussion of a common table, it has been invested with the title of the "wooden" sound. Essentially it is a dull sound, but it gives the impression of "hardness." It is rarely, if ever, produced by fluid in the pleural cavity, but is often developed in great intensity when adhesion has recently taken place between the two layers of the pleura by means of interposed lymph. Indeed, it has been asserted that when this sound is strongly marked, it may be regarded as almost pathognomonic of this form of mischief. Such, however, is not the case. It may exist to an equal extent in certain cases of pneumonic hepatization, as also in extensive tuberculous or other infiltration of the lung, and all that can be fairly stated of it is, that in its highly developed character it is a more common accompaniment of pleuritic adhesion than of pulmonary consolidation. In no instance is it observed more frequently than when tuberculous disease of the upper part of the lung, whether accompanied or unaccompanied by the formation of vomica, has given rise to pleuritic inflammation and to adhesion of the lung to the anterior surface of the chest. However, as it is not necessarily connected with any particular form of pul-

monary disease, and therefore has no special signification, the term is little more than a useless encumbrance to our phraseology.

There are certain apparent exceptions to the laws already enunciated as regulating the emission of sound by the chest under percussion, which are not commonly recognised or understood, and which, nevertheless, deserve special attention, as calculated to give rise to serious errors of diagnosis. They occur in instances in which the lung is supposed to present the conditions already described as contributing to the production of dulness on percussion. Thus it sometimes happens that a clear, high-pitched sound, of a somewhat metallic character, may be elicited over lung tissue more or less solidified, and that an abnormal resonance may be met with in the infra-clavicular regions when the pleural cavity is three parts full of fluid, and the lung is thereby partially compressed. Reference is made to this fact in most modern works on percussion, but no satisfactory explanation has been hitherto offered of it. The resonance alluded to has been variously described by authors as of a tracheal, tubular, metallic, empty, hollow amphoric, or tympanitic character, and, in fact, it varies greatly with the varying condition of the lung; but the point to be remembered is, that the sound is neither the dull sound of solidification, nor the peculiar resonance of healthy lung, but is clearer, and usually more shallow toned. It may differ little from healthy pulmonary resonance, so that comparative percussion of the healthy lung may be necessary to establish its abnormal character; but from this point the sound may range through every degree of intensity, until in many cases it is decidedly amphoric, and now and then very closely resembles a cracked-pot sound. The precise amount of change, however, does not serve to establish any difference in the pathological condition of the parts out of which it arises, and therefore it appears to me better to speak of the sound simply as an abnormally clear but shallow resonance, rather than to attempt to define by words the precise character of a sound which varies from day to day, and, in many cases, does not admit of accurate definition.

Well, then, the occasional existence of this abnormal resonance under the conditions above referred to, does not admit of doubt, and it is essential to determine, if possible, the causes to which it is attributable, and the mode of distinguishing it from the resonance which occurs under an apparently opposite condition of the subjacent parts.

With a view to a full understanding of the subject, I propose to bring together some of the more striking and suggestive facts connected with its various bearings.

Firstly, then, during the progress of tubercular disease of the lungs, it is observed not unfrequently that the part of the chest at which the least respiratory murmur is audible is that at which there is the clearest resonance under percussion. The existence of emphysema, which may be suggested as one method of accounting for this abnormal resonance, fails utterly in many cases in affording an explanation; for I have repeatedly noted cases in the wards of St. George's Hospital, in which this resonance existed during life, and in which no trace of emphysema was found after death. And when, in connection with this fact, it is remembered that the abnormal resonance is not always observed as a consequence of tuberculous consolidation, and that, when it does occur, it is not a persistent condition, but, with the progress of the disease, and the increase of solidification, is replaced by dulness more or less complete, the conclusion seems inevitable that its source is to be looked for not in simple pulmonary consolidation, but in some peculiar condition of the lung which is merely an accidental accompaniment of consolidation, and is apt, under certain circumstances, to arise during its progress.

So, too, in regard to inflammation of the lungs. Abnormal resonance of a clear, high-pitched, ringing, metallic character is sometimes elicited by percussion over a lung which is undergoing pneumonic consolidation, and occurs, therefore, under circumstances which, *à priori*, would lead to the expectation of partial or complete dulness on percussion.* The fact that such resonance does not *always* occur during the progress of pneumonia, and ceases as soon as complete condensation of the lung-tissue has occurred, seems to prove, as in the last case, that the cause of the abnormal resonance is not mere condensation of the pulmonary tissue, but is to be sought in some condition into which the lung is apt to pass during the process of consolidation.

The same holds good in regard to pleurisy, when there is an abundant secretion into the pleural cavity, and the lung is adherent to the anterior

* This has been often noted, though never properly explained. See a paper by Dr. Hudson, in the 'Dublin Journal,' vol. vii; a paper by Dr. Graves, in the same journal; a memoir by Dr. Roger, in the 'Archives Générales de Médecine,' vol. xxix; Dr. Stokes, 'On Diseases of the Chest,' p. 332; Dr. Walshe, loc. cit., p. 76; a report by Dr. Addison, in the 'Guy's Hospital Reports,' vol. iv, 1846; a paper by Dr. Markham, in the 'Edinb. Monthly Journal' for June, 1853; and another in the same journal for August, 1853.

and upper part of the chest. As the lung becomes partially condensed by the fluid, the upper part of the side on which the effusion exists is found to become unusually resonant on percussion, the resonance, as in the other cases, being abnormally clear, but of a shallow character.* Experiment, however, proves that lung tissue entirely deprived of air by compression yields a perfectly dull sound on percussion, and the conclusion, therefore, follows, that an explanation of the resonance must be sought in some other cause than mere pulmonary consolidation.

Viewing the above facts separately, it might appear that although a certain degree of pulmonary consolidation leads to partial dulness on percussion, and complete solidification of the lung to perfect dulness, yet, that there exists a stage between these two extremes in which the percussion sound, instead of being dull, becomes abnormally clear and resonant. But such an hypothesis is utterly repugnant to common sense, and to all that is known respecting the emission of sound by a body under percussion, and close investigation at the bedside of the patient, serves thoroughly to establish its fallacy; for it shows, as already stated, that abnormal resonance does not always occur in connection with the progress of pulmonary consolidation, and that when it does occur, it is often of very short duration, and is met with under every variety of condition in regard to the presence or absence of respiration, occurring sometimes when little or no respiratory murmur is audible, and at others when the respiration is loud, harsh, and hollow.

What, then, can be the source of this singular phenomenon? Several hypotheses have been offered in explanation, but none of them afford the slightest solution of the mystery. Some persons† who have observed this abnormal resonance chiefly in the infra-clavicular regions, have suggested that when the vesicular structure of the lung is compressed or solidified, its sound-conducting power is increased, and that it will then admit of the transmission of the sounds elicited by percussion from the trachea and larger bronchi, just as though they had been directly percussed. But the fallacy of this argument is obvious from the fact that the abnormal resonance is by no means confined to the

* Skoda, Hudson, Walshe, Davies, and many observers in this country, have noted this fact; a marked example of it, in the person of Thomas Ringrose, is at present (May 22nd, 1862) under my care, in the King's Ward of St. George's Hospital.

† Dr. C. J. B. Williams and his followers. See his 'Lectures on Diseases of the Chest.'

inner portion of the infra-clavicular regions, and to the vicinity of the larger bronchi, and is not more marked on the left side than on the right, as, from the greater proximity of the trachea, it ought to be; and further, that when complete solidification has occurred, this reputed sound-conducting power ceases, and the resonance is replaced by dulness.*

Others† who have met with this resonance in the lower part of the chest have endeavoured to account for it by the transmission through solidified lung of sound excited by the act of percussion in the stomach and intestines. But although in Dr. Addison's case, and in certain cases reported by other observers, this suggestion may be supposed to explain the phenomenon, still, its occasional occurrence in other instances over the middle portions of the lung, whilst a stratum of perfectly solidified lung, emitting a dull sound on percussion, lies immediately below the part at which the resonance is heard, suffices to prove that the phenomenon will not admit in all cases of such an explanation.

Others,‡ again, have expressed their belief in the existence of air or gaseous exhalations in the pleura over the solidified lung; and in some rare instances, when the resonance exists in the upper part of the chest, it may be difficult to *prove* that the presence of air may not possibly be its cause. But the result of careful post-mortem investigation is greatly against the agency of such a cause, and, in those instances in which the resonance is observed only at the base, or about the middle of the lungs, the phenomenon cannot be attributable to such an agency, unless, indeed, it be supposed that the air is confined in a circumscribed sac, formed by the presence of old adhesions of the pleura—an hypothesis which is at variance with reasonable expectation, and is often contradicted by the shifting in the limits of the resonance

* I have never met with a case in which this abnormal resonance was more strongly marked than in a patient admitted under my care into the York Ward of St. George's Hospital, suffering from acute pneumonia of the upper lobe of the left lung. Within thirteen days the right infra-clavicular region had passed through a stage of extraordinarily increased resonance into complete dulness on percussion, and from that again into the normal condition. At the same time the respiration, which was accompanied at first by crepitation, became markedly hollow in character, and then gradually reassumed its normal character. The expectoration was very characteristic throughout.

† Dr. Hudson, 'Dublin Journal,' vol. vii; Dr. Addison, 'Guy's Hospital Reports,' vol. iv, 1849.

‡ Drs. Stokes, Graves, and Walshe.

during life, and by the results of an inspection of the parts after death.

Others, again, as Skoda and his followers, who are conscious of the fallacy of attributing to perfectly solidified lung an increased sound-conducting power, and who are also aware of the extraordinary resonance sometimes observed over partially condensed lung have jumped to the conclusion that when the lungs are partially deprived of air, they invariably yield a tympanitic percussion sound, and that hence the source of the phenomenon.* But this doctrine is based upon two erroneous assumptions, viz., that "collapsed lungs give a distinctly tympanitic sound"† on percussion, and that under ordinary circumstances there exists sufficient distension of the lungs to interfere with and diminish the tympanitic tone of their resonance. No one who has percussed lungs which are thoroughly collapsed, will admit with Skoda that they yield a tympanitic resonance, and although it is true, as already stated, that extreme distension of any air-containing cavity, will prevent the occurrence of vibration, and thus may lead almost to dulness on percussion, as may be seen in certain cases of pneumothorax, and may be tried experimentally on tensely inflated lungs, and on a tensely inflated stomach, still it cannot be contended that such a condition exists, or can exist, under ordinary circumstances in the chest. Repeated experiments have convinced me that perfectly collapsed lungs yield a dull sound on percussion; that lungs as ordinarily met with in the dead subject, or inflated to about one quarter of the full extent, yield a clear, but not very full-toned resonance; that inflated to one half or two thirds of their full extent, their resonance is as clear, if not more so than before, and is decidedly more full toned or tympanitic; and that it is not until they are distended to their fullest extent, and the whole tissue is upon the stretch, that any diminution of clearness or fulness is perceived. Further, the consideration of certain phenomena connected with respiration establishes the same facts. After deep inspiration, the chest yields a clearer and fuller note on percussion than it does after a forcible expiration when the lungs are in great measure emptied of air; certain it is, moreover, that in most cases of partial tubercular consolidation, when the lung is in great measure deprived of air, the percussion sound is usually duller than natural; and further, that it is so during recovery from pneumonia, when the

* Skoda, 'On Auscultation,' by Dr. Markham, pp. 13 and 18.

† Skoda, loc. cit., p. 19.

lung is still only partially expanded. Indeed, over a lung which has been perfectly solidified by pneumonia, the percussion note does not regain its normal clearness and fulness until free respiration is again established. As, then in all these instances in which the lung tissue is more or less solidified, and contains less than its normal amount of air, the percussion note is duller than natural; as the same fact is observed as a result of diminishing the inflation of the lung; and as extensive tubercular deposit often occurs, and many cases of pneumonia run their course even to the production of complete hepatization without the occurrence of abnormally increased resonance, it is manifest that mere deprivation of air, whether to a small or to a large extent, will not raise the clearness of the percussion note.

To what, then, is attributable the increased resonance which sometimes, though rarely, occurs over condensed lung? From a careful consideration of the various circumstances under which this singular phenomenon is met with, I have been led to believe that it arises from the presence of air pent up in lung tissue, in the immediate vicinity of consolidated tissue—a condition which prevents the diffusion of the vibrations excited by percussion, and leads to the concentration and intensification of the resonance. Nothing can be more certain than that in many cases of pneumonia, especially when accompanied by some amount of capillary bronchitis, the gorged and distended condition of the capillary vessels, and the effusion existing in the terminal bronchi, block up the air passage from the air cells, and thus retain in them air in a state of greater or less elastic tension. The post-mortem examination of persons who have died of pneumonia show this to be a condition of very frequent occurrence. The lung cannot be compressed by any moderate degree of force; little or no air can be squeezed out of it; but no sooner is its tissue cut by the scalpel, than there issues forth a sanious frothy fluid, or, in other words, a fluid largely mixed with air. The portions of lung in which this condition exists, are those immediately above the point to which hepatization has extended, and below that to which the air has free access. They are those, in short, where crepitation or fine bubbling sounds occur, and are precisely those over which this clear toned resonance is met with. On several occasions I have traced this resonance shifting its position higher and higher in the chest, as the inflammation has spread upwards, whilst at the same time dulness on percussion has also extended upwards, and has occupied the parts immediately below it, which

previously had been the seat of this peculiarly clear resonance. In instances of tubercular deposit, the conditions essential to the retention of air in the lung tissue are of less frequent occurrence, and abnormal resonance over condensed tubercular lung is met with less frequently; but it does occur in certain instances;* and it is easy to conceive how some of the smaller bronchi may become occluded either by tenacious secretion or by the pressure of tuberculous matter; and that the obstruction thus created may, for a time at least, prevent the escape of the air from that portion of the lungs to which these bronchi lead. And, so again, in pleurisy, with an abundant secretion into the pleura. Anything, in short, which serves to occlude or obstruct a bronchus, whilst, as yet, the lung beyond the point of obstruction remains even partially distended with air, will tend to produce this abnormal resonance. It matters not whether the obstructing cause be an effusion into the terminal bronchi, as in certain cases of pneumonia, or whether it be a deposit of tubercle, cancer, or other matter either in or around the bronchial tubes, or the pressure of any tumour from without, or the pressure against the bronchi leading to the permeable portion of the lung, of lung tissue already solidified—in all cases, if air is retained, pent up beyond the cause of obstruction, and the adjacent tissue is somewhat condensed, the result is, the production of a peculiar resonance on percussion. If, as in most cases of pneumonia, the mischief commences in the air vesicles, so that the air is expelled and replaced by solid matter before the bronchi become obstructed, dulness on percussion accompanies the progress of the disease from first to last; whereas, if the mischief implicates the smaller bronchi, and the air passages become obstructed, whilst, as yet, the pulmonary cells beyond are more or less distended with air, then that condition results which is conducive to abnormally clear, though shallow resonance on percussion. Each lobule, in fact, is converted into a multilocular air-distended cavity. The same holds good in regard to tubercular or other deposit as to pneumonic exudation; but as the deposition of tubercle is usually much slower than pneumonic exudation, and less frequently leads to obstruction of the bronchi with retention of air in the pulmonary cells beyond the seat of obstruction,

* Those gentlemen who were noting my cases in St. George's Hospital, in May 1857, will remember how remarkably this was illustrated by the case of Mary Wills who was admitted into Holland Ward on the 6th of that month. See 'Hospital Case book,' xx, "Women."

it happens that the peculiar resonance in question is seldom so well marked in the former as in the latter disease. In pleurisy, if the effusion into the pleural cavity be copious, and the apex of the lung be adherent to the anterior walls of the chest, it often happens that pressure enough is exerted to obstruct some of the bronchi leading to the pervious portion of the lung, and thus to produce the condition essential to the abnormal resonance.

The accuracy of my conclusions may be tested by the following experiments, which prove that this peculiar abnormal resonance is not due to the sound from the trachea or larger bronchi transmitted through solidified lung, nor, as Skoda asserts, to mere diminution in the quantity of air and to the lung tissue being less distended in consequence, but is rather referable to the presence of pent-up air having behind it lung tissue more or less solidified, which prevents the diffusion of the sonorous vibrations.

1st. Take three healthy sheep's lungs. Let one remain in a collapsed condition;* inflate the second to about one half of its full extent, and tie the bronchus so as to prevent the escape of air; inflate the third lung thoroughly, distending it to its fullest extent, and retain it in its distended condition by a ligature on the bronchus. Then on percussing each of them by means of a pleximeter and percussion hammer, it will be found that the first yields a short, dull sound, the second a clear and full sound, the third a sound which varies according to the precise amount of pulmonary distension, but which is usually less clear and far less full than the sound yielded by the second. If the distension be extreme, the sound will be still less clear, and will approach to dulness.

2ndly. Let a lung previously inflated with air be injected through the bronchus,† and let it be injected to about one half of its full extent. When the injection has set or become solid, percuss the lung and

* I have found that lungs supposed to be collapsed vary immensely in the amount of air they contain (retained, doubtless, by viscid secretion in the bronchi), and consequently in the sound they yield on percussion. It is difficult to obtain, even from the slaughter-house, lungs which are thoroughly collapsed, but it is necessary to do so, if the experiment is to be regarded as of any value.

† These experiments are best performed with sheep's lungs, fresh from the slaughter-house. The lungs should be inflated through an injection-tube, fastened tightly into the trachea by means of ligatures, and the injection should not be commenced until the lung is inflated to at least one half its full extent. Size, mixed with a certain portion of glue and coloured with vermilion, answers well as an injecting fluid.

compare the sound produced with that elicited by percussion of the collapsed lung and of the partially inflated lungs used in the last experiment. It will then be found that if these parts only are percussed in which the superficial portions of the pulmonary tissue remain distended with air, a remarkably clear and shallow sound will be emitted, corresponding to what is termed amphoric resonance, having in some instances more or less of a metallic, ringing character. The resonance is clearer, and at the same time shallower than that yielded by any of the uninjected lungs. Percussion over those portions of the lung which lie superficially and contain no air elicits dulness more or less complete, according to the depth at which air lies beneath.

3rdly. Percuss an inflamed lung the lower part of which has undergone pneumonic hepatization. Over the perfectly hepatized portions complete dulness will be elicited by percussion; over those parts at which inflammation is commencing the percussion note will not be materially altered, whilst over those portions which, though still highly crepitant under the finger, are in a far advanced stage of inflammation, the percussion sound will vary according as the air cells or the terminal bronchi appear to be principally and primarily affected, the sound being more or less dull according as the air cells are filled to a greater or less extent with solid material, and more or less clear but shallow toned according as the smaller bronchi are obstructed, and the superficial air cells still remain distended with air, as may be tested by the spumous, frothy character of the fluid which escapes when the lung tissue is cut, and by the existence or non-existence of a granular appearance when the lung tissue is torn. These facts, though not as yet recognised or pointed out by authors, I have verified repeatedly in the dead-house of St. George's Hospital.

CHAPTER VI.

AUSCULTATION—ITS THEORY AND PRACTICE; THEORY OF THE STETHOSCOPE; ADVICE RESPECTING THE FORM OF INSTRUMENT TO BE EMPLOYED; CAUTION TO BE OBSERVED IN THE PERFORMANCE OF "IMMEDIATE" AND "MEDIATE" AUSCULTATION; MODE OF CONDUCTING AN EXAMINATION OF THE CHEST.

THE act of respiration in a healthy chest is accompanied by sounds which are occasioned by the action of the lungs and heart, and which in disease undergo modifications corresponding to the physical alterations produced in the intra-thoracic organs. These sounds, together with those produced by the voice and by the act of coughing, are audible on the external surface of the chest; and the science of auscultation consists in recognising their character and their various abnormal modifications, and in tracing each to its corresponding physical cause.

Auscultation, like percussion, may be "immediate" or "mediate." It is "immediate" when the ear is itself applied in contact with the chest walls, either bare or only thinly covered, and when, therefore, the sonorous vibrations are conveyed directly from the chest to the ear; "mediate" when an instrument is interposed between the chest and the ear and forms a conducting medium between the one and the other. Both these methods of examination have their advantages and disadvantages, and each is deserving of attentive study. The former is attended with the least fuss and parade, and is therefore useful in the case of timid persons or of children who are frightened by the application of a stethoscope;* it can be employed at all times, even when no instrument is at hand; it causes no pain or uneasiness to the patient; it is the most convenient method of exploring the posterior parts of the chest in cases not requiring much nicety of examination, and in some instances it affords the means of determining the character of sounds which, heard through the cylinder, are indistinct and indefinite. The

* An instrument so named by Laennec. The term is derived from στηθος, the chest, and, σκοπειν, to examine.

latter, on the other hand, enables us to examine the axilla and other regions where the ear cannot be placed in close apposition to the chest walls, and it provides us with the means of isolating any particular spot, and thus of tracing each sound to its exact position within the thorax. In short, every auscultator should be trained to both methods of examination, and in cases in which both can be employed it matters little which is had recourse to. It often happens that both may be employed advantageously, the one to examine the axillary and other regions which only admit of examination by the stethoscope, the other to explore the inferior, lateral regions and the whole of the posterior surface of the chest. But there are so many occasions on which it would be inconvenient, disagreeable, or even indelicate to apply the ear directly to the chest, that every one should take especial care to familiarise himself with the use of the stethoscope.

An endless variety of forms and of materials have been employed in the construction of stethoscopes, some with a view to a fancied superiority of principle, but many more out of a regard to elegance of shape, convenience, and portability. They have been made solid and hollow, rigid and flexible, of wood, bone, gutta percha, and other substances, and each form and each material has in turn found eager advocates. The truth, however, appears to be that a light-textured vibratile wood, or else gutta percha or vulcanite, is the best material for a stethoscope, and that a rigid stethoscope is the best kind of instrument for ordinary occasions. The only point to be decided, therefore, is the form of instrument to be employed.

A solid cylinder was thought by Laennec to be the form best adapted for the transmission of the chest sounds; and Dr. Hughes, in furtherance of Laennec's opinion, thinks it "quite evident that it is by the solid walls of the stethoscope that the sound is in all cases principally,and in many cases entirely conducted."* On the other hand, Dr. Williams, and most other writers attach great importance to the column of air contained in the interior of the hollow stethoscope, and recommend the employment of that form of instrument on acoustic principles quite irrespectively of its convenience and portability. The result of my own experience is in accordance with the latter opinion. Not only in the one instance does a solid piece of wood form the only medium of communication between the chest and the ear, whilst in the other a column of air serves conjointly with the wood as a medium for the

* Dr. Hughes, 'On Auscultation,' p. 80.

transmission of vibrations; but the hollowing of the stethoscope undoubtedly renders the stethoscope more vibratile, and therefore more apt to receive and propagate the sonorous vibrations.

It is commonly supposed that the column of air contained in the interior of the hollow stethoscope is set in motion by the vibration of the chest walls; and Dr. Williams and many other writers have endeavoured to point out the precise form of tube and conical excavation which is best fitted to collect and concentrate the sonorous vibrations and convey them unimpaired to the ear. The opinion, however, which forms the basis of these calculations has always appeared to me erroneous; and some ingenious experiments, devised by my friend, Dr. Davies,* afford demonstrative proof that the vibrations transmitted throughout the substance of the wood which forms the walls of the cylinder produce a greater effect in exciting the air contained within it than do the vibrations of that portion of the chest walls which is covered by the hollow end of the instrument. Thus the conclusion is forced upon us that although a hollow stethoscope forms the best conductor of the chest sounds, the precise form of the cavity of the pectoral extremity is immaterial.

The essential points in the construction of a stethoscope are, firstly, that the end applied to the chest be not so thin and sharp as to cause pain to the patient, nor so large as to render it difficult or impossible to apply it evenly and uniformly to the surface of the chest walls in a moderately thin person, and not so thin as to break easily. Secondly, that the ear-piece be large enough to cover the whole ear, and of a shape to fit and close it—a circumstance of great importance as tending to prevent the dispersion of the vibrations.

For general use I would recommend a stethoscope made of some light vibratile wood such as cedar and cherry, having a shaft from six to eight inches in length, with a clear, polished quarter-inch central canal, an ear-piece two and a half-inches in diameter, slightly and but very slightly hollowed,† and a thoracic extremity from one inch to an inch and a quarter in diameter, hollowed in a conical form, and having a broad, well-rounded rim. Such an instrument, made of a single piece of wood, or with the ear-piece made to screw, or else slip on to the

* Davies, loc. cit., pp. 203-4.

† For most persons the ear-piece should be made only slightly concave; but should the tragus and antitragus be unusually prominent, it must be made concave in a proportionate degree.

shaft, is in many respects superior to most of the stethoscopes to be met with in the shops. Practically, however, far more depends on the skill and attention of the examiner than on the form of the instrument, and therefore when a well-made stethoscope has been selected, and an ear-piece chosen which fits the ear comfortably, the student should keep to that one, and familiarise himself with its use.

Another form of stethoscope, which is sometimes had recourse to for the purpose of exploring the axilla and the lower lateral and dorsal regions, when the patient cannot be readily moved, is that known as a "flexible stethoscope." It is usually made about two feet in length, and is constructed of wire twisted into the form of a hollow cylinder, and covered with silk or worsted thread. There are few cases, however, in which its assistance is really needed, and as it conveys sound less readily than a wooden instrument, it is seldom employed in practice.

A double or bin-aural sthethoscope has been invented by Dr. Camman, of New York. It consists of two tubes—one for each ear—the thoracic ends of which fit into a hollow cylinder or cup, which is applied to the surface of the chest. It thus enables us to auscultate a particular portion of the chest with both ears at the same moment; and as all sounds are stronger when heard by both ears than when heard by one ear only, it is contended that the delicate sounds which accompany the early stages of phthisis are discoverable by this instrument more readily than by the ordinary wooden one. Unquestionably the sounds are heard louder than through the ordinary wooden instrument; but they are confused by other sounds, which result from the use of the instrument, and which it requires considerable education to distinguish from morbid chest sounds. On this account, therefore, and also on the ground that the instrument is more formidable in appearance than the ordinary wooden stethoscope, and is less portable, and otherwise less convenient, it is rarely employed by the practical physician.

Another form of bin-aural stethoscope has been invented by Dr. Scott Alison, and has been styled by him the "differential stethoscope." Like Dr. Camman's instrument, it consists of two tubes, one for each ear, but instead of being connected with one cylinder or one cup only, each tube has a separate cylinder or cup, which admits of being applied to any part of the chest. Thus, whilst Dr. Camman's instrument enables us to hear the sounds emanating from any given portion of the chest with both ears simultaneously, Dr. Alison's instrument enables us to listen to the sounds emanating from two different parts

of the chest at the same moment, and with great readiness and accuracy to compare the sounds at any spot, on the two sides of the chest by a series of consecutive observations. Both these points are very important. It is no small advantage to be able to examine two portions of the chest without shifting our position, or the position of our patient, or making any movement of the head; for these are precisely the circumstances which are most favorable for the detection of the slighest difference in the sounds on the two sides of the chest. Therefore, if this alone were attainable by the aid of Dr. Alison's instrument, it should be in the hands of every auscultator; but experience proves that it possesses other powers, which render it indispensable to every practitioner. Not only does it enable us to listen at one and the same moment to sounds emanating from two different parts of the chest, but it infallibly determines upon which side of the chest the sounds are loudest and most intense. For, strange as it may appear, it is nevertheless the fact, that when sound is conveyed to both ears simultaneously, but is louder, however slightly, on one side than on the other, the weaker sound is eclipsed or nullified, so that sound is heard on the stronger side, whereas no sound is audible on the weaker side. In the early stage of phthisis, when disease is often confined to one side, and occasions a slight difference only in the force and character of the respiratory sounds on the two sides, the value of this differential stethoscope is self-evident.

But whilst its value as an occasional aid to diagnosis is admitted, it must not be regarded as a substitute for the ordinary wooden stethoscope. It is just as formidable in appearance, and in other respects as inconvenient in practice as Dr. Camman's double stethoscope, and it requires to be handled with extreme nicety. The extremities of the tubes must be fitted closely and equally into each ear, the other extremities applied to the chest in a similar manner, and the elastic portions of the tubes kept equally straight. If these points are not strictly attended to, differences of sound will be necessarily engendered. Further, it has a disadvantage which is fatal to its use by many practitioners, viz., that the two ears of the auscultator must be of tolerably equal acuteness.

Another form of instrument, introduced by Dr. Scott Alison, and termed by him a "hydrophone,"* deserves a passing notice. It consists of a flat India-rubber bag, about three times the size, and of the

* From *υδος*, water, and *φωνη*, voice.

same thickness as a watch, partially filled with water which forms a medium between the ear and the chest walls. When the patient is very thin, and the intercostal spaces are deeply sunk, so that the surface of the chest is irregular, and the application of the ear or of the stethoscope is difficult, the hydrophone greatly facilitates auscultation. It fills up the depressions on the surface of the chest, and closes the external ear of the auscultator, and thus enables him to hear sounds issuing from the chest which would otherwise be inaudible. But it somewhat deadens the sounds as heard through an ordinary wooden stethoscope, and it does not, any more than immediate auscultation, enable the auscultator to isolate any particular part of the chest, nor to examine the axilla or other parts where the ear cannot be placed in close approximation to the chest walls, so that practically its use is restricted to those cases in which the chest is too thin and the surface too uneven to admit of being examined by the ordinary wooden stethoscope. In such cases, and especially as an aid to the flexible stethoscope, it is of some value.

In the performance of auscultation, just as of percussion, certain precautions are necessary to ensure accurate results. The chest should be uncovered, or, if exposure be inadmissible, the covering should be as thin as possible, so as not to offer any material obstruction to the transmission of sound, and of a soft yielding nature, so as not to occasion any friction or rustling which may interfere with, or overpower the sounds from within the chest. A calico chemise or nightgown, a soft towel, or an old napkin smoothly spread over the surface, answers the purpose perfectly well. The position of the patient should, if possible, be regulated in the manner pointed out in the chapter on "Percussion,"* and, for the reasons already insisted on, great care should be taken to subject each corresponding portion on the two sides of the chest to a precisely similar examination. The posture of the examiner should be easy and unconstrained; and with the view of avoiding the necessity of assuming inconvenient positions, the physician should be able to employ both ears, and should practise both ears accordingly. The examination should be conducted in a quiet room, and all friction between the stethoscope and the clothes should be strictly avoided. Care, too, must be taken not to place the stethoscope over stiff hair on the surface; for the intervention of hair between the chest and the instrument may occasion a fine crepitating sound, greatly resembling the crepitation of pneumonia, and thus may give rise to misconception.

* See p. 41.

So again, if immediate auscultation is had recourse to, the hair should be put aside from around the ear in contact with the chest; for if any portion intervenes between the ear and the chest, a noise is apt to be produced which may be readily confounded with certain morbid sounds emanating from the lungs.

But there are certain further precautions specially applicable to the use of the stethoscope. The slightest interval between any portion of the thoracic end of the instrument and the surface to which it is applied prevents the free transmission of the chest sounds to the ear, and it is therefore necessary to hold the instrument at right angles to the surface, so that every part of its extremity may be kept closely and evenly in contact with the parietes. If the patient be so thin as to render it impossible for the whole circumference of the extremity of the stethoscope to be placed in close contact with the chest, a pad of soft linen, or a piece of India rubber, or the hydrophone, may be employed to fill up the depressions between the ribs, and so to produce an even surface for its application, or, if this proceeding be not adopted, the instrument must be discarded, and the hydrophone, or else "immediate" auscultation had recourse to. Again, the character of the sounds is altered, and their transmission is interfered with if the ear be not applied to the stethoscope with a sufficient degree of pressure: whilst, on the other hand, it is obvious that forcible pressure on a thin and tender chest cannot fail to occasion pain, and in some measure to interfere with the respiratory movements. Hence the instrument should be applied firmly, yet not heavily or forcibly. In stout persons, however, it is necessary to exert more than usual pressure, in order to compress the loose adipose tissue and thus increase its conducting power; and, if any œdema of the surface exists, an amount of pressure must be employed sufficient to squeeze out all the serum contained in the cellular tissue immediately beneath the stethoscope. For, not only is serum a bad conductor of sound, but, by its escape from the cells under pressure, it may give rise to a fine bubbling, or to a creaking noise of a jerking character, the former of which, except by its continuance when the patient holds his breath, and by its disappearance after continued steady pressure with the stethoscope, is scarcely distinguishable from pneumonic exudation, and the latter from certain forms of pleuritic friction. The same holds good in a modified degree when the chest walls are emphysematous, as from a broken rib.

Beyond all this, it is in the highest degree necessary to acquire the

power of giving the undivided attention to the sounds emanating from the chest—a power only to be obtained by habit; and it is equally essential to *practice* the mechanical application of the stethoscope; to learn how to place it evenly on the chest; to hold it there firmly; to ascertain by means of the fingers whether every portion of its thoracic end is in close contact with the skin, and, if not, to adjust it properly, and then remove the contact of the fingers; to fit the ear readily to the ear-piece; and to employ sufficient, but not more than sufficient pressure. Nicety in all these matters is only to be obtained by diligent practice, and it is the more necessary because clumsiness or roughness in the use of the stethoscope at once frightens the patient and indicates an inexperienced auscultator.

In conducting a physical examination of the chest, the student will do well to observe a particular order of proceeding. On ordinary occasions the best method is to begin by carefully inspecting the chest, its form, size, and movements; next to employ the sense of touch, ascertaining in this way the amount of movement and the character of the vibrations in different parts, and then to have recourse to percussion, first on the clavicles, then on the anterior surface of the chest, proceeding from above downwards, next in the lateral regions, and lastly, on the posterior part of the chest. The stethoscope should then be made use of, and the parts examined by its means in the same order. The various methods of examination will thus severally furnish evidence as to the condition of the parts within the chest, which, taken together, will often justify our forming conclusions not warranted by the signs obtainable by any single method of examination.

It is sometimes objected that such an examination is tedious to the physician and wearisome to the patient, and therefore practically ought not to be employed. But an attentive student will soon perceive that if the examiner is skilful and well practised, and is systematic in his method of proceeding, a few minutes will usually suffice to elicit information not otherwise attainable. Even when, in obscure and difficult cases, it is necessary to devote more time and attention to the investigation of the disease, it certainly is our duty to sacrifice such time, and for once to run the risk of fatiguing our patient, rather than to undertake the treatment of the case in doubt or ignorance as to our patient's disorder. In the simplest case, mischief often occurs in situations where the general symptoms least betoken its existence; and therefore, if the whole chest be not examined before an opinion is

expressed, or a line of treatment decided on, a grievous error may be made.

The examination should be commenced whilst the patient is breathing naturally; he should then be desired to take a deep inspiration, then to speak, then to cough, and then again to breathe naturally as is his wont. By these means, as will be explained hereafter, it will often happen that much valuable information not otherwise attainable may be acquired, and the real condition of the lungs ascertained.

One difficulty occasionally presents itself, which is not to be overcome without much ingenuity and patience. Some persons, when under examination, seem incapable of breathing naturally, and equally so of taking a deep inspiration. When told to do either the one or the other, they commence a series of unnatural, awkward movements, which materially impede the entry of air into the lungs, or modify the sounds to which it gives rise; they open the mouth, raise the shoulders, and fix the chest, without the least attempt at full inspiration; or, if they do breathe efficiently, they draw in the air through the compressed lips, or else open the mouth and relax the fauces in such a manner as to occasion noises which overpower the sounds produced within the chest. Thus, not unfrequently considerable difficulty is experienced in making a patient breathe in such a manner as to enable us to judge of the condition of the lung. In some such instances our object may be attained by telling him to sigh, or to fill the chest, or to draw a deep breath, exemplifying the action by breathing deeply and noiselessly several times in succession, and desiring him to imitate our action. But not unfrequently even these expedients fail; and then the only means of effecting our object is by directing him to speak or cough for some moments consecutively, when, after the repeated short expirations which accompany those acts, a full, noiseless inspiration follows, and he does involuntarily what his previous efforts had failed to accomplish.

CHAPTER VII.

RESPIRATORY SOUNDS.

The next points for consideration are, the cause and nature of the respiratory sounds, and the modifications they undergo in disease.

It may be well to premise that two sounds are audible by auscultation accompanying each act of respiration—the one corresponding to the act of inspiration, the other to that of expiration, and named accordingly the "inspiratory" and "expiratory" sounds. These sounds are heard of varying duration, rhythm, and character in different portions of the respiratory tract. Along the pharynx, larynx and trachea they are of nearly equal duration, the latter being somewhat the most prolonged;* a distinct interval occurs between the cessation of the former and the commencement of the latter; and they are loud, dry, hoarse and hollow, as if caused by a rush of air through a tube of considerable diameter, rough and irregular on its internal surface. This is the character of what is termed "tracheal respiration."

Beneath the upper bone of the sternum, and in thin persons between the scapulæ at a point corresponding to the bifurcation of the trachea and the origin of the larger bronchi, the rhythm and character of the sounds are altogether changed. There is no longer the same interval between them,—they follow each other more closely; the inspiratory sound is considerably longer and louder than the expiratory, and both are softer, less loud and dry, less hollow and less blowing than the sounds heard over the trachea. These points characterise what has been termed "bronchial respiration," or "tubular breathing."

Passing still further along the respiratory tract to those parts of the chest which contain only the ultimate ramifications of the bronchi and

* The difference in point of duration is very slight, and, frequently is not perceptible. When it does exist, it is attributable, I believe, to the fact that, after the completion of the respiratory act a pause ensues, which admits of the uninterrupted continuance, for a few seconds, of the sonorous vibration excited during *expiration*, whereas the expiratory blast commences immediately on the cessation of inspiration, and thus destroys or prevents the continuance of the sonorous vibrations excited during that act.

the vesicular structure of the lung, the character of the sounds changes again. There no longer exists any appreciable interval between the inspiratory and expiratory sounds; the former is above twice as long as the latter, and is heard of a higher pitch—so that whilst the inspiratory sound accompanies the whole inspiratory act, and is soft, breezy, gradually developed, and continuous, the expiratory sound is short, weak, and in some persons almost, if not quite inaudible, especially on the upper part of the left side of the chest. These are the peculiarities of what has been termed "pulmonary respiration," and the sound is known as the "respiratory" or "vesicular murmur."

The source of these respiratory and expiratory sounds, and the cause of their modification at different parts of the respiratory tract, admit of satisfactory explanation. The sounds themselves are traceable to the vibrations, excited in the air by the irregularities of the surface over which it passes during each act of inspiration and expiration, as also, probably, in some slight degree to the alternate expansion and collapse of the lung tissue; the difference in their duration, rhythm, character and loudness in the various portions of the respiratory tract, to the differences in the velocity and volume of the current of air in the different parts, to the nature of the channels through which it travels, to their position as regards the chest walls, to the direction of the respiratory blast, and the consequent readiness with which the sounds are conveyed to the ear. If the several conditions attending the production of sound and its transmission to the ear in different portions of the respiratory tract are carefully considered, the cause of the observed varieties will be at once apparent.

To begin with the inspiratory sounds. What are the precise conditions observed in the first portion of the respiratory tract? The cartilaginous rings of the trachea are strongly marked, and its walls, therefore, are rough and irregular; the calibre of the tube is considerable, the volume of air passing through it is large, and as the current is drawn in with immense force, it traverses this portion of its course with great velocity. Hence the resulting sound is loud, dry, hoarse, and hollow, conveying the impression of air rushing through a tube of considerable diameter, rough and irregular on its internal surface. The trachea being superficial in position, the sound reaches the ear in all its loudness, roughness and hollowness.

Below the bifurcation of the trachea the bronchi subdivide into smaller and smaller tubes, and the cartilaginous rings which enter into

their composition, become less and less pronounced, until in the terminal ramifications of the bronchi they cease to exist, and the tubes are smooth on their internal surface. Thus differences are at once perceptible in the causes of sound in the different portions of the respiratory tract. The rush of air is the same in all, but the calibre of the tubes through which it has to pass decreases gradually, and the nature of the surface over which it travels varies greatly in the different parts of its course. In the trachea and upper air passages the air meets with little or no impediment beyond that presented by the roughness and irregularity of their walls; in the larger bronchi it has to encounter the obstacle presented by the subdivision of the bronchial tubes; whilst in the third part of its course, where it passes along the terminal bronchial ramifications into the vesicular structure of the lungs, it no longer meets with the roughness presented by the existence of the cartilaginous rings, but it has to overcome the contractile power of the air passages and is thrown into vibration by impinging upon the uneven, irregular surface of the pulmonary vesicles. This further difference is also to be remarked, that whereas the larger bronchi are well calculated from the firmness and elasticity of their walls, to reflect and propagate any sound which may be generated within them, and in some situations are close to the parietes of the chest, the smaller bronchial ramifications are devoid of cartilaginous rings to keep their sides asunder, and are mostly surrounded by the spongy tissue of the lung—a bad conductor of sound. The result is just what would have been anticipated, viz., that the inspiratory sound emitted from the larger bronchial tubes, though less loud and rough, less hollow, and less blowing than the tracheal inspiratory sound, is far louder, more rough than, and of a different character from the soft breezy, vesicular murmur which arises from the terminal portions of the respiratory tract; and further, that this vesicular murmur which is superficial in its situation and reaches the ear without difficulty is alone heard in every portion of the chest where the bronchi do not come in close proximity to the thoracic walls. Nor can the latter circumstance excite surprise after what has been stated in a previous chapter* relative to the conducting power of different media; for, independently of the fact that the superficial position of the vesicular murmur necessarily leads to its obscuring or overpowering any more deeply seated sound, it is obvious that, except when the bronchial tubes are placed in close proximity to the thoracic walls, all vibrations

* Chap. v, pp. 29 and 33.

originating in the bronchi must pass from air to membrane, and from membrane to air, many hundred times before they can reach the surface of the chest. Thus the medium of their transmission is constantly changed, and at each change their intensity is diminished.

The duration of the inspiratory sound in different portions of the respiratory tract is also regulated in accordance with fixed laws The smaller the size of the air passages through which the current of air has to pass, the weaker, *cæteris paribus*, the current which will produce sonorous vibrations therein. Thus the *in*spiratory blast will occasion sound in the vesicular structure of the lung, and in the smaller bronchial passages, before it has acquired sufficient intensity to cause sonorous vibrations in the trachea and larger bronchi, and it will serve to keep up such vibrations in the pulmonary vesicles and the terminal branches of the bronchial tubes for some time after it has ceased to occasion sonorous vibrations in the larger bronchi and the windpipe. Hence it occurs that the vesicular inspiratory sound is more prolonged than the tracheal and the larger bronchial inspiratory sounds.

So again, in regard to the rhythm of respiration and to the duration, character, and loudness of the expiratory sound in different portions of the respiratory tract. Many of the causes which give rise to sound during inspiration produce no such effect during expiration, and this holds good especially in regard to those causes of sound which exist in the pulmonary portion of the respiratory tract. Thus during expiration the current of air does not meet with resistance from the contractility of the air passages as it does in ordinary inspiration, and being no longer inwards and against the irregular surface of the air vesicles and the edges of the bronchial subdivisions, but outwards, in the same direction with those causes of vibration, it is in no way acted on by them.* Hence, as the expulsion of air from the air vesicles takes place gradually, as the current is necessarily small and weak, and as in their natural condition the walls of this portion of the air passages are smooth, so that the air meets with no obstacle in traversing them, the expiratory sound over healthy lung substance is soft and weak. Such as it is, however, it takes its origin in the collapse of the lung and the rush of air from the air vesicles, and commencing, therefore, with the commencement of expiration, it immediately succeeds the cessation of inspiration. But it is necessarily much

* The difference resulting from this arrangement may be illustrated by the effect of blowing first *against the edges* of a sheet of paper, and then *along* its surface.

shorter than the inspiratory sound; for the resistance to the exit of air from the chest is less than that to its entrance into the lungs, whilst the expiratory muscles, aided by the natural resiliency of the lungs,* and by the elasticity of the costal cartilages, form an expulsive power about one third greater than that concerned in the act of inspiration.† And to such an extent do these causes operate in influencing the duration of the act, that pulmonary expiration is found to occupy not much above one third of the time occupied by the act of inspiration.

In the other portions of the respiratory tract a material difference is observed in the rhythm of respiration and in the character and duration of the expiratory sound. The more remote any portion of the respiratory tract from the air vesicles whence the air is expelled, the larger, of course, the calibre of the tubes through which the air has to pass; the larger and stronger must be its current, in order to excite sonorous vibrations therein, and the longer, consequently, the time which must elapse before such vibrations can be produced; in other words, the longer must be the interval between the cessation of the inspiratory and the commencement of the expiratory sound. Hence, whilst in the pulmonary portion of the respiratory tract the expiratory succeeds immediately to the inspiratory sound, a distinct interval occurs between the two sounds over the bronchi, and a still longer interval over the trachea. Moreover, in the bronchi, as in the vesicular portion of the respiratory tract, the causes of sound during expiration are far less numerous than those which operate during inspiration, for the vibrating tongues formed by the sub-division of the air passages are not, as during inspiration, opposed to the current of air, but are in the same direction with it. Thus it happens that the bronchial expiratory sound, though louder and rougher than the vesicular expiratory murmur, is neither so loud nor so rough as its corresponding inspiratory sound, nor, for the reasons already stated, is it nearly so prolonged.

In the third portion of the respiratory tract the calibre of the air tubes (the trachea and larynx) is still larger, and consequently a longer time elapses after the cessation of inspiration before the expiratory blast acquires sufficient volume and strength to throw the air into

* The power of this resiliency of the lungs has been shown experimentally by Dr. Carson, in a paper published in the 'Philosophical Transactions' for 1820, and its importance on the mechanism of respiration is manifest in the symptoms observed in the emphysema, in which this elasticity of the lungs is lost.

† See a paper by Dr. Hutchinson, 'Med.-Chir. Trans.,' vol. xxix; and also Valentin, 'Lehrbuch der Physiologie des Menschen,' p. 529, quoted by Dr. Davies.

sonorous vibration. But when once this point has been reached, the force of the blast, the roughness of the internal surface of the passage, and the tension of its walls, are such as to ensure the production and maintenance of sonorous vibrations so long as the expiratory effort is continued. Indeed, the causes of sound in this portion of the respiratory tract are almost the same during expiration as during inspiration. Thus it is found that the tracheal expiratory sound, though commencing later, or, in other words, after a longer interval, than the bronchial expiratory sound, is of longer duration than that sound, and indeed is equal in that respect to the tracheal inspiratory sound.

The importance of these facts, as bearing on the diagnosis of disease, will be manifest when it is stated that the respiratory sounds undergo modifications corresponding to any physical alteration produced in the condition of the lung substance or the air passages, or in the freedom of ingress or egress of the air. At present it is sufficient to mention the circumstance, which will be illustrated and explained as each separate modification of breathing is reviewed.

And first in regard to the varieties of respiration which are compatible with a healthy condition of the lungs. What are their nature and what their causes?

The respiration already described is that of a healthy adult of middle age. In infancy, in childhood, and in old age the conditions are somewhat different, and the respiratory sounds vary accordingly. In infancy the inspiratory muscles are weak, and have great difficulty in overcoming the resistance offered to a full inspiratory effort by the elasticity of the chest walls and the resiliency of the lungs. Hence, as the diaphragm is the most powerful of the inspiratory muscles, the respiration is almost wholly abdominal; and as the resistance offered by the elasticity of the chest increases rapidly with the increase of thoracic expansion,* the breathing is necessarily short and hurried, and the lungs being imperfectly expanded, the respiratory sound is faint. In childhood, or, in other words, when the strength has increased and the muscles have become more fully developed, the chest expands freely at each inspiration; indeed, the thoracic walls, being extremely elastic, admit of great freedom of action in the lungs: the resiliency of the lung tissue is remarkable; the pulmonary air vesicles are small, so that a larger number of them exist within a given space than are to be found within the same space in

* 'Med.-Chir. Trans.,' vol. xxix.

adult life, whilst, as the walls of the chest are thin, the respiratory sounds are transmitted with great readiness to the ear. The result of these various circumstances is, that the faint respiratory sounds of infancy are replaced by breathing sounds which are louder and more intense than those heard at a more advanced age, though their character remains unchanged. This modification of breathing is termed *puerile* respiration.

In old age, on the contrary, the costal cartilages are stiffened, the elasticity of the lung tissue is diminished, and consequently the play of the lungs is lessened. The inspiratory sounds, therefore, are usually less intense than in early adult life, and the expiratory sound is more prolonged. This modification is termed *senile* respiration.

In youth the respiration is more frequent or rapid than in adult life; and in all persons, particularly in women, and in those especially who are accustomed to tight lacing, the pulmonary respiratory sounds are fuller in the upper than in the lower part of the chest, in consequence of the greater play and more thorough inflation of that portion of the lungs.* They are often particularly loud and intense in nervous persons suffering from hysteria and other similar affections, for the reason I believe that, when under the excitement of examination, such persons breathe more deeply and forcibly than under ordinary circumstances; whilst in certain individuals they are unusually weak, in consequence, I believe, of the large relative size of the lungs and the less necessity which therefore exists for the full inflation of the air vesicles.† In neither instance, however, is the character of the sound altered, nor is the peculiarity alluded to attributable to disease.

* This is strictly true, for tight lacing, or, indeed, anything which restrains the action of the lower ribs must necessarily occasion increased action of the upper part of the chest. But something more than this is needed to explain the remarkable action of the upper part of the chest in women, for the contrast between the respiratory movements in the male and female is almost as marked in naked savages as in tightly clad and civilised Europeans. Probably the cartilages of the ribs are endowed with extraordinary elasticity, and the intercostal muscles with unusual power and freedom of action in the female, with the view of obviating the great interference with respiration which would otherwise result from the enlargement of the gravid uterus, and the consequent obstacle to the descent of the diaphragm.

† Direct experiment with the spirometer has convinced me that in the majority of such cases the vital capacity is above the average, or, in other words, that the size of the lungs relatively to the body and the circulatory apparatus is unusually large; and nothing can be conceived more directly conducive to weak respiratory sounds during tranquil respiration than a breathing apparatus which, being unusually capacious, is imperfectly or partially expanded at each inspiration.

Sometimes, even when the inspiratory sound is as loud as usual, the expiratory sound is very weak, and in certain instances is unattended by any audible sound, especially in the left infra-clavicular region. Again, when corresponding portions of the chest are examined, the *in*spiratory sound is usually found to have the same character on both sides, though it is sometimes louder on the left supra- and infra-clavicular regions than it is in the corresponding regions on the right side. But the *ex*piratory sound is commonly louder and more prolonged in the supra- and infra-clavicular regions on the right side, and in the superior scapular and inter-scapular regions on the same side, than it is on the left, the difference being more perceptible at the inner than at the outer part of these regions, and more strongly marked in females than in males. Indeed, if the observation be confined to the right infra-clavicular region, it may be stated almost as a rule that the intensity and duration of the expiratory sound are greater on the right than on the left side, the difference being most marked in thin, nervous females. Again, in certain instances, the sounds of respiration on both sides of the chest are either weak and indeterminate in character, or else are replaced by an indistinct humming.

Now, it must be distinctly understood that the existence or non-existence of the varieties of breathing already described affords no criterion as to the presence of disease. They may not exist, and the patient may be perfectly free from disease; or they may be strongly marked, and he may be healthy nevertheless. But it is otherwise in regard to certain modifications of respiration, to which we have now to direct our attention. Those already mentioned derive their chief importance from the fact that, if not rightly interpreted, they might often lead to a presumption of disease: those now to be described are always indicative of an unhealthy condition of some portion of the respiratory apparatus. They consist of changes in the intensity, rhythm, and quality of the breathing sound, and it will be seen that in the two first varieties the duration of the sounds occupy an important place.

1st. In *Intensity*, the respiratory murmur may be—

(*a*) Exaggerated or increased.
(*b*) Weak or diminished.
(*c*) Weak and indeterminate.
(*d*) Absent or suppressed.

2nd. In *Rhythm* (*a*), the respiration may be jerking or interrupted (the respiration saccadée of Laennec).

(*b*) An interval of greater or less duration may intervene between the inspiratory and expiratory acts.

(*c*) The relative duration and intensity of the inspiratory and expiratory sounds may be altered.

3rdly. In *Quality*, the sounds may be—

(*a*) Coarse, harsh, or rough.

(*b*) Blowing or whiffing.

(*c*) Of a hollow character.

(*d*) Accompanied by a metallic resonance.

1st. *Intensity.*—(*a*). Exaggerated breathing, as contrasted with the natural pulmonary respiration, is characterised by the greater intensity and duration of its sounds. It is sometimes termed "*puerile*" respiration, from its resemblance to the loud respiration of children; and "*supplementary*" from the fact that it is not abnormal in character, but is simply caused by an inordinate action of certain healthy portions of the lungs, set up to supply the deficiency of respiration consequent on the inactivity of other portions which are destroyed or affected by disease.* Thus, in fact, it is due to locally increased pulmonary action; and its peculiarity is derived partly from the unusually large number of air vesicles inflated in any given space at each act of inspiration, partly from the completeness of their distension, and partly from the increased volume of air and the unwonted force and rapidity of its current in those portions of the lungs where the exaggerated breathing sound exists. Unlike the puerile respiration of children, and the naturally loud breathing which is sometimes met with in the healthy adult, exaggerated breathing is never heard diffused entirely throughout the chest.† It exists at one spot and not at another, according to the position and extent of the mischief in which it takes its origin; its intensity is greatest close to the actual seat of disease; and, after a time, it may

* The term "puerile," as applied to this variety of respiration, is singularly inappropriate, inasmuch as "puerile" breathing is a *healthy* type of respiration occurring in children, and "exaggerated" breathing is commonly met with in adults, and is essentially dependent on the presence of disease in some other portion of the lungs. The term "supplementary" is at once appropriate and significant of the true nature of this variety of breathing.

† The only exception to this rule is in "spasmodic asthma," when the nature of the complaint is well marked and obvious, and the exaggerated breathing is of only temporary duration.

become inaudible where it had previously existed in well-marked character. Owing to the close proximity of disease, which may obstruct the bronchi, and thus offer some impediment to the egress of air, the expiratory sound is often prolonged relatively as well as absolutely; but this is a modification which, though frequently coexistent with exaggerated breathing, is essentially distinct from it in its mechanism, and is attributable to causes which are not necessary to its production.

Thus, then, as anything which prevents the free play and perfect action of any portion of the pulmonary tissue may give rise to exaggerated breathing in other portions of the same or opposite lung, its presence should lead us to search out and discover the cause and seat of the obstruction of which it is the certain index. The mere fact of the existence of exaggerated breathing is positive proof of pulmonary obstruction; the cause and seat of the obstruction may vary according to circumstances. Fluid effused into the pleural cavity by compressing the lung and thus interfering with its action; aneurismal or other tumours, by pressing on and thus obstructing the bronchi; mucus or other matters blocking up the air passages; pneumonic exudation; sanguineous effusion; tuberculous or other deposits, by condensing the lung tissue; or, on the other hand, vesicular emphysema, by diminishing its elasticity, and thus impairing the action of circumscribed portions, may, one and all, produce exaggerated breathing of greater or less extent and intensity in the adjoining healthy pulmonary tissue. The only other condition in which it occurs is that of spasmodic asthma, in which the healthy tissue of the lung, suddenly released from bronchial spasm, is immediately inflated by a deep and forcible inspiration. In such a case as this the exaggerated breathing is only of temporary duration, and the nature of the malady is sufficiently obvious; but in every other instance its nature, seat, and extent, require careful investigation.

(*b*) *Weak respiration* contrasts strongly with exaggerated breathing. Its sounds, though not altered in character, are diminished in intensity and duration. Referable simply to deficient expansion of the pulmonary tissue, it may arise from any cause which directly or indirectly interferes with or controls the inflation of the lungs. Hence its causes are numerous, and require care and judgment in their discrimination. Malformations of the chest, from whatever cause arising, may give rise to this modification of breathing, and so may deficient muscular action, whether from debility, exhaustion, or paralysis, on the one hand, or as the result of pain, as in pleurisy, intercostal rheumatism, or peritonitis

on the other. Defective pulmonary elasticity, as in vesicular emphysema is another fertile source of weak respiration, and any obstruction of the air passages, whether from internal causes or from causes external to the lungs, will also give rise to it;—the chief internal causes being certain diseases of the larynx; thickening of the mucous membrane lining the bronchi, with consequent diminution in the diameter of the air tubes; an accumulation of mucus or other secretion in the trachea or bronchi; and the presence of foreign bodies or the deposit of tuberculous or other matter in the interior of the air passages;—the external causes being the pressure of enlarged bronchial glands or of aneurismal or other tumours; moderate effusions into the cavity of the pleura or pericardium; pneumo-thorax; or, in short, anything which removes the lungs from the chest walls or so far presses upon the pulmonary tissue as to interfere with the due performance of pulmonary respiration, without altogether obstructing or annihilating it. Sometimes weak respiration is of temporary duration only, or recurs in an intermittent form, indicating an equally temporary or occasional interference with the entry of air into the lungs. Thus, it occurs in whooping cough from closure of the glottis, in spasmodic asthma, pleurodynia, and other affections, and its intermittence constitutes an important element of diagnosis in cases in which aneurismal or other moveable tumours press upon the larger bronchi, and in those also in which obstruction is caused by the presence of foreign bodies in the main bronchi on either side of the chest.

(*c*) Under the head of weak respiration may also be ranged certain cases in which the breathing sounds are either weak and indeterminate in character or else are replaced by an indistinct humming. This condition of respiration is not necessarily limited to unhealthy persons, as I have met with several well-marked instances of it in persons enjoying robust health.

(*d*) *Absence or suppression of respiration.*—The total absence of respiratory sounds denotes the existence of causes which either obstruct the bronchi completely or else prevent the action of the lungs. The one essential condition is that air shall not permeate the lung tissue. Thus, the causes already mentioned as productive of weak or diminished respiratory sounds may lead, when still further developed, to their entire annihilation. Excessive tuberculous, cancerous, or other infiltration of the lung, or of any portion of it; an extreme degree of vesicular emphysema; complete occlusion of a bronchus, whether from internal

obstruction, or from external pressure as from aneurismal or other tumours; large collections of fluid or of air in the chest, compressing the entire lung and forcing it back, a carnified, impervious mass, against the vertebral column; these are some of the causes of absent respiration, the two latter being of most frequent occurrence.

2ndly. *Rhythm.*—During healthy respiration, *in*spiration and *ex*piration take place evenly; the expiratory follows closely upon the inspiratory movement, and a brief interval of repose follows the completion of each respiratory act. These general characters hold good, whatever may be the increase or decrease in the frequency of the respiration, so that the expiratory sound maintains its due relative bearing to the inspiratory, the ratio of the one to the other being about 1 to 4. But in disease various modifications are observed. Thus—(*a*) the mode of evolution of the sounds may be altered, and instead of being even and continuous from their commencement to their close, both sounds, but especially the inspiratory, may become jerking or interrupted,—evolved, as it were, by instalments. This is the *respiration saccadée* of Laennec,—the cogged-wheel respiration of some English authors. (*b*) Expiration, instead of following closely upon inspiration, may be separated from it by a distinct interval or pause. And (*c*), instead of bearing their due relative proportion to each other, the sound of expiration may be increased in intensity and duration until it becomes louder than the inspiratory murmur, and extends over the normal interval of repose, so that inspiration succeeds expiration with scarcely an appreciable interval of silence.

(*a*) The first-named variety, when existing generally over one or both lungs, is indicative of something giving rise to irregularity in the act of inspiration. Thus, timidity or nervousness whilst the patient is undergoing examination; the pain of pleurisy or intercostal rheumatism; spasmodic affections of the air passages; or, indeed, any cause which will produce occasional checks during the act of respiration, may give rise to jerking interruption in the sounds. And as the cause of such irregularity in any particular instance must be sufficiently obvious, the existence of generally diffused jerking respiration is comparatively unimportant in a diagnostic point of view. But when it exists in small portions only of the lungs, and when, more especially, it is met with in the upper part of the chest, its practical significance can hardly be overrated, inasmuch as the finely divided jerking respiration, or, as it has been termed, the

"cogged-wheel" rhythm of respiration, heard in that situation, is a frequent accompaniment of incipient tuberculization of the lungs. Its immediate cause is not quite determined. It is commonly supposed to be referable to the presence of thick, tenacious mucus, which presents some obstruction to the free ingress and egress of the air. But although this is doubtless a frequent cause of it, I am strongly inclined to the belief that it may arise independently of mucus in the air passages, as a result of obstruction, consequent on pressure caused by the presence of tubercles or some other form of consolidation. Not only is it easy to conceive how the presence of tubercles or other solid matters may press upon the smaller bronchi and produce such obstruction, but I have so often met with jerking respiration, during the early stage of tubercle, in persons who at the time were quite free from cough, expectoration, râles, and every other evidence of mucus in the air tubes, that I cannot regard it as invariably, or even generally, referable to the presence of mucus. It is always due to temporary local obstruction of the air tubes, of such a nature as will yield to the force of the inspiratory blast as soon as the expansion of the lungs has reached a certain point; but I believe that the obstruction is usually occasioned by thickening of, or pressure on, the walls of the air passages, and not by the presence of thickened mucus.

(*b*) The second variety in which a distinct interval or pause occurs between the close of inspiration and the commencement of expiration is of less importance in a diagnostic point of view. It may depend upon any cause which puts a stop to the production of sound before the close of the respiratory act; or which, on the other hand, prevents the production of sound during the commencement of expiration. Thus, the inspiratory sound, in cases of pulmonary consolidation, ceases before the accompanying expansion of the chest; whilst, on the other hand, in emphysema, when the elasticity of the pulmonary tissue is impaired, the contained air is not expelled from the air vesicles during the first portion of the expiratory act. In either case an interval occurs between the cessation of the inspiratory and the commencement of the expiratory sound; but, in the one case, this is caused by the inspiratory sound being prematurely checked, in the other by an abnormal delay in the commencement of the expiratory sound.

(*c*) The third variety is characterised by an alteration in the relative

duration and intensity of the inspiratory and expiratory sounds. The proportion which the former bears to the latter is about three to one in a state of health, and may become as one to five under the influence of disease. This excessive relative prolongation of the expiratory sound is met with only in emphysema, and is referable in part to the absolute prolongation of expiration consequent on the diminished resiliency of the lung and the swollen and thickened condition of its mucous lining membrane, and in part also to the abnormal shortening of the sound of inspiration, the sound in many cases of emphysema being heard only towards the close of the inspiratory act. But simple prolongation of the expiratory sound is of common occurrence, and its essential cause is the same in all instances, viz., a want of freedom in the egress of the air from the lungs. It may exist to a greater or less degree, and many causes may conduce to its production. One of the most common is the thickened and swollen condition of the mucous membrane of the air passages which exists in chronic bronchitis; another, the pressure of aneurismal or other tumours on the bronchi; and another, consolidation of the lungs, from whatever cause arising—whether from internal deposit or from external pressure—as in pleurisy or pneumothorax. But the most common, and therefore practically the most important, is the presence of tubercles in the lungs. Tubercular matter, when deposited in the lungs, is found either on the free surface of the bronchial mucous membrane, or in the tissue external to the air vesicles. In either case it gives rise to more or less swelling of the bronchial lining membrane and causes projections into the air passages, with a corresponding diminution in their calibre; impairs or interferes with the resiliency of the lung, and creates more or less impediment to the free egress of the air. Thus, the conditions essential to the existence of prolonged expiratory murmur are often present at an early stage of consumption, and as many of the more characteristic signs of tubercle are at that time wanting, prolonged expiration becomes a valuable diagnostic sign. It is insufficient of itself to afford evidence of the disease, but it serves to confirm in the strongest manner any evidence derived from other sources.

3rdly. *Quality.*—Anything which interferes with the normal condition of the lung, whether by solidifying or otherwise altering the density of its tissue, by producing alterations in the calibre of the air passages, or by affecting the dryness of the bronchial mucous membrane, and thus increasing the friction encountered by the inspired air, will interfere with the soft breeziness of the sound which has been de-

scribed as characteristic of healthy respiration. The earliest deviation from the natural standard *quality* of respiration is roughness, coarseness, or harshness, occasioned sometimes by rarefaction, but more commonly by solidification of the pulmonary tissue, with some degree of swelling of the bronchial mucous membrane. This variety of abnormal breathing, especially when strongly pronounced, is marked by the peculiarly blowing or whiffing character of the sounds, and by their increased duration and intensity,—characters which, though common to both sounds, especially in their highly developed types, are first perceived and are most strongly marked in the sound of inspiration. But the greater the amount of local change the greater the alteration in the sounds of respiration; and thus it happens that when the pulmonary tissue is condensed, and the sounds emanate from the large bronchi or from hollow spaces in the lungs, the character of *hollowness* is superadded to the other characters already described, and the breathing is not only rough, harsh, and blowing, but is of a distinctly hollow character.* Changes in the density of the pulmonary tissue may thus be traced by the alterations which occur in the respiratory sounds. Condensation, for instance, when only slight, and confined to the air vesicles and smaller bronchial tubes, is simply accompanied by a roughness, coarseness, or harshness of respiration, marked by a blowing or whiffing character, such as is not observed in a state of health: when the structure of the lungs is more deeply implicated, and the smaller tubes are rendered impervious, so that the vibrations conveyed to the ear are derived from bronchial tubes of a larger size, the sound, though still rough, coarse, or harsh, is more dry, more blowing, and more prolonged than in the earlier stage of the disease; whilst at a still later stage, when even greater condensation of the lung tissue has occurred, and the sounds emanate chiefly from the larger bronchi, the breathing assumes a decidedly hollow character, and somewhat resembles that

* Some theoretical considerations relative to the reinforcement of the respiratory sounds in certain instances of hollow breathing have been lately forced into more or less prominence by Skoda, of Vienna. He has attempted to ignore the effect of pulmonary consolidation in rendering more audible on the chest walls any sounds emanating from the bronchi, and has endeavoured to explain the admitted increase in the loudness of such sounds under certain circumstances by reference to the doctrine of consonance. As the matter will be discussed in connexion with increased vocal resonance, to which the same theory applies, it is needless to enter on the question at present, but I would refer the reader to pp. 102-4 of this treatise. Whatever is there stated relative to increased vocal resonance applies equally to the reinforcement of the sounds of respiration.

which is heard in health over the larger bronchial tubes. Hence this variety of breathing has been termed "bronchial breathing." When solidification is complete,—when in point of fact the lung is hepatised, as in pneumonia, and the walls of the bronchi are dry and tense, forming good reflectors of sound, a metallic resonance is superadded to the mere bronchial character, the blowing is less diffused, and a sound results which greatly resembles that produced by blowing sharply through a brass tube. This form of breathing has been called "tubular" breathing. When again, as is sometimes the case, a large bronchus, or a bronchial tube which has undergone dilatation, or a small empty vomica with tense walls, is surrounded by condensed and homogeneous lung tissue, the air passing through or into it produces a sound resembling that produced by air passing rapidly into a small cavity. This form of respiration has been styled "cavernous" respiration. When, again, the cavity is still larger, and is bounded by tense walls, as, for instance, the pleural cavity, or as in some rare cases, a large excavation in the lung, the air passing into it occasions a sound like that of air passing rapidly into an empty jar or small cask, and hence this variety of respiration has been termed "amphoric" respiration.

It will be perceived that all these varieties of hollow breathing merge gradually into each other, and differ only in the size and condition of the air passages or spaces in which the sounds take their source, and in the density, homogeneity, and conducting power of the surrounding parts. They are not peculiar to any particular form of disease nor to any one form of structural mischief, but are each indicative of a certain physical condition of the pulmonary apparatus, the nature and true significance of which must be determined by other means. Thus, for instance, a large or a dilated bronchus, surrounded by condensed pulmonary tissue, may produce a sound which cannot be distinguished from the so-called cavernous breathing, whilst, on the other hand, a small cavity, or a number of small, empty vomicæ in the lungs, may occasion respiratory sounds closely resembling the so-called bronchial and tubular breathing. So gradually do the different varieties of hollow breathing merge into each other, that it is sometimes difficult to decide whether a given sound arises from a bronchus or from an abnormal cavity in the lung tissue. All that is essential to the production of any form of hollow breathing sound is an empty space of a certain size, in free communication with the upper air passages, and surrounded by consolidated or homogeneous, yet vibratile lung. The precise nature of the hollow space and the exact cause of the consolidation or

increased homogeneity of the surrounding tissue cannot be determined solely by the auscultatory signs, but must be decided, if at all, by the history of the case, the condition of the patient, the situation of the spot at which the sound is most audible, and by the signs elicited by other means of examination. The terms, "bronchial," "tubular," and "cavernous," therefore, are objectionable, as implying a theory as to the source of the sounds, which in many instances may be erroneous, and in its practical application has often misled inexperienced auscultators. If, for convenience sake, the terms are retained, their true significance must be borne in mind, in order that undue importance may not be attached to them. None of them conveys the slightest information which is not afforded by the term "hollow," and I am strongly of opinion that we should do well to expunge such fallacious words from our phraseology and employ the term "hollow" as applicable to all abnormal breathing sounds of a hollow character.

It follows, from what has been already stated, that deviations from the healthy quality of respiration are found in the early or dry stage of bronchitis, in which the air tubes are congested and devoid of secretion; in congestion of the lungs, with distension of the vessels beneath the pulmonary vesicular membrane, causing narrowing of the air passages or irregularity of their surface; in dilatation of the bronchi; and in excavations of the lung substance, from whatever cause arising; in vesicular emphysema; and in pulmonary consolidation, whether caused by the infiltration of tubercle or other matter, by the contraction of inflammatory exudation, or by the pressure of fluid or of solid or gaseous materials in the pleural cavity;—the only exceptions being when sufficient healthy lung tissue intervenes between the ear and the part affected to mask the morbid sounds by its own natural ones, and when the amount of foreign matter in the pleura is such as to put a stop to all pulmonary sounds or prevent their conduction to the ear. In the latter case the lung is pushed upwards and backwards against the spinal column, and a hollow breathing sound will still be audible about the middle of the back, close to the spine, or, in other words, in the position of the main bronchi.

Further, it is obvious that, according as the changes capable of altering the quality of the respiratory sounds are limited to one spot or are generally diffused throughout one or both lungs, so will be the extent of the area over which the altered breathing sound is heard; and according as the changes are of a transient or enduring nature so will the alteration in the sound prove more or less persistent. These are

points which, viewed in connexion with the history of the case and the position and character of the sound itself, not unfrequently lead to a correct diagnosis as to the nature of the existing mischief.

The following tables will show at a glance the varieties of respiration in health and disease, their special characters, their mode of production and usual seat, as also the forms of disease with which the morbid varieties are usually associated.

TABLE I.

RESPIRATION IN HEALTH.

Varieties. Synonym.	*Character of the sound.*	*How produced.*	*Its usual seat.*
Pulmonary respiration — Respiratory or vesicular murmur	*Inspiration.*—A soft, diffused sound, of a breezy character, gradually developed and continuous, accompanying the whole respiratory act; followed without any appreciable interval by the *expiratory* sound, which is short, weak, and, in some persons, almost inaudible	*Inspiration.*—By vibrations excited in the inward current of air by its friction against the walls of the air passages; by the obstacles presented by the subdivision of the bronchi; and by the irregularity of the surface of the pulmonary vesicles on which the air impinges *Expiration.*—By the vibrations excited in the expired air by its friction against the walls of the air passages	The air vesicles and terminal bronchi, and therefore heard in all parts of the chest except the upper part of the sternum and the space between the scapulæ, corresponding to the roots of the larger bronchi.
Puerile respiration — Exaggerated vesicular murmur, or puerile breathing	The same as the pulmonary vesicular murmur, but exaggerated or intensified in degree	By the same causes as produce the ordinary vesicular murmur, but the sound acquires intensity in consequence of the great freedom in the action of the lungs in early childhood, the remarkable resiliency of the lung tissue, and the small size of the air vesicles which leads to there being a larger number of them in a given space than in adult life	In children, in all parts of the chest where the ordinary vesicular respiration is audible
Bronchial respiration — Bronchial breathing	Both the respiratory sounds are louder, rougher, and of a higher pitch than their corresponding vesicular murmurs; they are of a somewhat hollow, blowing character, more rapidly evolved, and follow each other less closely, so that there is an appreciable interval between the close of inspiration and the commencement of expiration. This variety of breathing merges gradually into the next variety, tracheal or tubular breathing	Its peculiar character is referable to the size and roughness of the air passages in which it originates, and to their sound-reflecting power	The upper part of the sternum and the space between the scapulæ corresponding to the roots of the bronchi.
Tracheal respiration — Tracheal or tubular breathing	A loud, dry, hoarse, and hollow sound, as if caused by the rush of air through a tube of considerable diameter, rough and irregular on its internal surface. The inspiratory and expiratory sounds are of nearly equal duration, and a distinct interval occurs between the cessation of the former and the commencement of the latter	Its peculiar character is due to the size, roughness, and sound-reflecting property of the tubes in which it originates	Over the larynx and trachea.

CHANGES WHICH OCCUR IN THE

Connected with changes.	*Variety.*	*Synonym.*	*Character of the sound.*
Inintensity	Exaggerated or increased	Supplementary or puerile* breathing	The same as the ordinary vesicular murmur of health, but exaggerated or intensified in degree: identical in character with the puerile respiration of healthy children
	Weak or diminished	Feeble respiration	The ordinary vesicular murmur, not altered in character, but simply diminished in intensity and duration
	Weak and indeterminate	Indeterminate breathing	The precise character of the sound cannot be defined; it is sometimes weak and indeterminate, and at others is replaced by an indistinct humming
	Absent or suppressed	... ...	No sound is heard
In rhythm	Jerking or interrupted	Cogged-wheel respiration, or respiration saccadée of Laennec	The character of the sounds need not be materially altered, but both sounds, especially the inspiratory, instead of being even and continuous from their commencement to their close, are jerking or interrupted—evolved, as it were, by instalments
	A distinct interval, of varying duration, may occur between the inspiratory and expiratory acts	... ...	Expiration, instead of following closely upon inspiration, may be separated from it by a distinct interval of varying duration
	The relative duration and intensity of the inspiratory and expiratory sounds may be altered	... ...	The inspiratory sound may be relatively and absolutely shorter than in health, and the expiratory sound relatively and absolutely louder and more prolonged. Sometimes, though the duration and intensity of the *in*spiratory sound are not perceptibly affected, the *ex*piratory sound may be prolonged and intensified
In quality	Coarse, harsh, or rough	... ...	Coarse, harsh and rough, in the first instance, but when strongly developed of a blowing or whiffing character. The change is perceptible in both sounds, which are increased in duration and intensity, but it is most marked in the sound of expiration. It gradually passes into the next variety
	Blowing or whiffing		
	Of a hollow character: Bronchial or tubular	Bronchial or tubular breathing	To the blowing or whiffing character of the sound last described is superadded the character of hollowness, so that the breathing is not only rough and blowing, but of a distinctly hollow character
	Of a hollow character: Cavernous	Cavernous breathing	
	Of a hollow character: Amphoric	Amphoric breathing	
	Accompanied by a metallic resonance	... ...	Whenever, under the conditions productive of hollow breathing, the walls of the air passages are tense and possess a great sound-reflecting power, a metallic character is superadded to the hollowness of the sound

* Puerile is a wrong term as applied to any form of *morbid* breathing.

SOUNDS OF RESPIRATION IN DISEASE.

How produced.	*Its usual seat.*	*Disease with which it is usually associated.*
By the inordinate action of certain healthy portions of the lungs, set up to supply the deficiency of respiration, consequent on the inactivity of other portions which are destroyed or affected by disease	Not peculiar to any portion of the lungs, and not diffused generally throughout the chest, like puerile breathing (except in spasmodic asthma, when it is of temporary duration), but limited to certain spots in the vicinity of diseased portions of the lungs	Pleurisy; aneurismal, or other tumours pressing on certain bronchi; pneumonia; tubercular deposition; vesicular emphysema; spasmodic asthma.
By any cause which interferes with and prevents the full inflation of the lungs Not clearly ascertained	Not peculiar to any portion of the lungs; often diffused throughout the chest, but sometimes limited to a part of the lung supplied by one or more particular bronchi	Deficient muscular action, from whatever cause arising; defective pulmonary elasticity, as in vesicular emphysema; and any obstruction of the air-passages, whether from internal or external causes.
By any cause which prevents the air from permeating the lung tissue	May occur in any portion of the chest, but always limited to one or more parts, and usually to the whole or some portion of one lung only	Its most common causes are extreme emphysema, and excessive effusions of air or fluid in the pleura, as the result of pleurisy or pneumothorax.
When generally diffused over the surface of the chest, it is produced by something which occasions a jerking interruption in the act of breathing, as nervousness, the pain of pleurisy, or spasmodic affection of the air passages. When confined to particular portions of the lungs, it is due to some local obstacle to the free ingress or egress of the air, usually to the pressure of tubercular deposit, or to the presence of thick mucus in the air passages	Not necessarily confined to any particular portion of the chest, but of greatest clinical significance when confined to the apices of the lungs	The early stage of phthisis, when the sound is confined to certain portions of the lungs; spasmodic affections of the air passages, when it is generally diffused over the chest.
Either by the cessation of the inspiratory sound before the close of the inspiratory act, as occurs when the air vesicles of any portion of the lungs are obstructed; or	Not peculiar to any portion of the chest	Extreme pulmonary consolidation, from whatever cause arising.
By the absence of sound during the first portion of the expiratory act, as occurs when there is any obstacle to the collapse of any portion of the lung tissue	Not peculiar to any portion of the chest	Emphysema, or extreme pulmonary consolidation.
By any cause which affects the resiliency of the lung, and produces a swollen and thickened condition of the mucous membrane of the bronchi, or otherwise causes an obstruction in the air passages	Not peculiar to any portion of the chest, but when the sound is confined to one or both apices of the lungs, it is suggestive of tubercular deposit	Emphysema; chronic bronchitis; tubercular deposit; and the pressure of aneurismal or other tumours.
By anything which alters the density of the lung tissue, produces alterations in the calibre of the air passages, or affects the dryness of the bronchial mucous membrane	Not confined to any portion of the chest; most significant at the apices of the lungs in the early stage of phthisis	The first stage of bronchitis; dilatation of the bronchi; and pulmonary consolidation, whether from the deposit of tubercle, inflammation, or any other cause.
By whatever leads to condensation of the vesicular structure of the lungs and the transmission of sound from the larger bronchi, or from any other space or cavity in the chest. The lower types of it are connected with the existence of small spaces—whether bronchi or vomicæ, in the pulmonary tissue—surrounded by consolidated tissue; the more highly developed forms, with large spaces, having good sound-reflecting walls	May occur abnormally in any portion of the chest	In pulmonary consolidation from whatever cause arising; the presence of enlarged bronchi or tuberculous cavities; and the existence of pneumothorax, with a fistulous opening between the lung and the pleural cavity.
By whatever, under the conditions productive of hollow breathing, imparts excessive tension and increased sound-reflecting power to the walls of the air-containing space	The posterior and inferior portions of the chest	Pneumonic hepatization; tuberculous cavities, with tense walls; pneumothorax, with a fistulous communication between the lung and the pleural cavity.

CHAPTER VIII.

ON THE RESONANCE OF THE VOICE IN HEALTH AND DISEASE—BRONCHOPHONY, PECTORILOQUY, AND ÆGOPHONY; AND ON THE DOCTRINE OF CONSONANCE AS APPLIED TO SOUNDS EMANATING FROM THE CHEST; AUTOPHONY; AND TUSSIVE RESONANCE.

Auscultation of the voice, as transmitted through the chest walls, is another method of obtaining information as to the condition of the lungs and their investing membrane. Varying in pitch according to the varying action of the muscles which regulate the larynx, the sounds formed by the vibrations of the vocal cords are propagated, not only upwards through the mouth, but downwards through the bronchi into the air cells of the lungs. Accordingly, if the ear be applied directly to the chest walls, or if the stethoscope be used as a medium of conduction, the voice sounds may be heard reverberating in the chest. And just as might have been expected *à priori*, it is found that their character is not necessarily proportioned to the loudness of the voice, but, *cæteris paribus*, depends on the condition of the air passages and of the surrounding pulmonary structure. Thus it becomes evident that if we make ourselves familiar with the sound of the voice as heard in the different portions of a healthy chest, and can discover the conditions which lead to an alteration in its character, auscultation of the voice will furnish a valuable index to the existence of various forms of disease.

The first points to be determined are the peculiarities of character belonging to the vocal resonance during health in different portions of the respiratory tract, and the mechanism or precise local conditions to which these peculiarities are attributable.

If the stethoscope be applied over the larnyx of a healthy person whilst speaking, the voice will be transmitted through the instrument to the ear with greater force and loudness than it reaches the other ear, inasmuch as in the one case the sound is concentrated within the narrow tube to which the stethoscope is applied, and in the other is diffused in a large space, and thereby necessarily loses in intensity.

This resonance of the voice is termed natural laryngophony, whilst that which is heard along the trachea, whether in the mesian line in front or at the lateral parts of the neck, and, to some extent, even over the spinous processes of the vertebræ behind, is called natural tracheophony. In laryngophony and tracheophony the voice seems not only to be produced but to be concentrated immediately beneath the stethoscope, and conveys the impression of passing through the instrument so as to strike directly and forcibly on the ear.

At the upper part of the sternum and on the posterior surface of the chest, between the spines of the scapula, where the larger bronchi are given off, the voice is heard less loud, less concentrated, and less distinctly articulated; it appears to be produced at a greater distance, to be more diffused, and to strike less directly against the ear, so that the words seem to be at the end of the instrument instead of passing through it into the ear. This being the sort of vocal resonance heard over the larger bronchial tube, is termed natural bronchophony.

Over smaller bronchi the articulation is still more imperfect, and the resonance is considerably weaker and less concentrated, whilst over the whole of the parietes of the chest under which lies the vesicular structure of the lung the vocal resonance is even more diffused, is no longer articulated, and, in fact, does not amount to more than an obscure buzzing or humming. Even this buzzing is sometimes absent.

But there are other modifications which require notice. As a general rule, vocal resonance is most distinct in thin persons, and is obscure or indistinct in persons whose chests are well covered with fat and muscle, and in whom consequently an imperfect conducting medium is interposed between the sound and the ear of the observer; it is strongest when the voice is grave or low toned; clearest and most distinctly articulated when it is sharp or of a high pitch; it is louder in front than behind, except just in the interscapular regions over the root of the main bronchi, and is more developed in the upper than in the lower parts of the chest; it is more intense on the right side than on the left, especially under the clavicles and in the interscapular regions, and it varies greatly in intensity in healthy persons apparently of the same physical development,—a fact analogous to the greater intensity of the respiratory murmur in some persons than in others, and, like that, attributable, I believe, to variations in the relative size of the pulmonary and circulatory apparatus, and to the consequent greater or

less degree of pulmonary expansion. To such an extent, however, does this hold good that in some perfectly healthy persons the vocal resonance over the vesicular structure of the lungs is louder than it is in others equally healthy and of the same physical development, over moderate sized bronchi; whilst in others, again, apparently under similar physical conditions, the voice sounds will be either altogether inaudible or will be heard only as an indistinct humming.

It is obvious, then, that no correct inference as to the condition of the lungs can be drawn merely from the character of the vocal resonance in any given case: in other words, there is no standard of health in this respect. The vocal resonance, which may be either weak or exaggerated, if judged of by an average standard, may be perfectly normal in the case in question, and cannot be regarded as indicative of disease, unless it be found to vary at different periods. So, too, its character in any given portion of the chest cannot of itself afford trustworthy evidence of mischief; it is only by comparison of corresponding parts on both sides of the chest, due allowance being made for the difference which naturally exists between them, that any satisfactory conclusion can be arrived at.

Further, as the resonance is found to vary with the pitch and tone of the voice, it is essential, with a view to trustworthy conclusions, that the same word, or series of words, should be repeated in a monotonous manner; and as the use of different words gives rise to marked modifications in the character of the vocal resonance and is apt to confuse the ear, it is advisable to direct the patient to repeat a single word, such as "twenty," rather than to count one, two, three, four, as is sometimes recommended. The louder the patient's utterance the greater, *cæteris paribus*, the intensity of the vocal resonance, and, therefore, as a general rule, he should be directed to speak in a full, clear voice, averting his head from the auscultator. The stethoscope should be applied to the chest walls firmly, but lightly; if too much pressure be made, the vocal resonance will be diminished, whereas, if the pressure be not sufficient to keep the instrument in firm apposition with the integuments, a tremulous, bleating character may be imparted to the resonance, and may lead the inexperienced to an incorrect diagnosis.

These points, then, being borne in mind, I will endeavour to describe the various modifications of vocal resonance which are met with in disease, and to point out their causes and precise clinical significance.

It may be stated generally that the natural vocal resonance, without material alteration in its quality, may be either increased or diminished in any and every portion of the respiratory tract, and that this increase or diminution of intensity is attributable to the physical condition of the parts whence the sound passes to the ear. But further, the *quality* of the resonance may be altered, as well as its intensity, and, instead of the natural voice sound, there may be a ringing, squeaking, quavering, or bleating resonance of a metallic or an amphoric character. These varieties are attributable to differences in the physical condition of the thoracic viscera. Placed, then, in a tabular form, the varieties of abnormal vocal resonance may be arranged as follows:

Class.	*Variety.*	*Synonym.*
Vocal resonance in disease. — Diminished	Weak or feeble.	
	Suppressed or absent.	
Increased	Simple increase.	
	Bronchophonic	(*Bronchophony*).
	Pectoriloquous	(*Pectoriloquy*).
	Amphoric.	
	Ægophonic	(*Ægophony*).

In either of the four last-named varieties, the resonance may be of a metallic, ringing character.

The varieties included under the class of "diminished resonance" require little explanation. They are characterised by nothing more than weakness or diminution in the force or intensity of the resonance, and are met with of every degree of feebleness. Sometimes, though weak, the resonance remains clear; sometimes an indistinct humming only is heard; and at others, again, the sound of the voice fails altogether to reach the surface of the chest. But, whether the resonance be altogether absent, or be only diminished in intensity, the cause of the alteration is in either case the same, viz., either weakness of the voice, or the occurrence of some physical change in the parts beneath the stethoscope calculated to interfere with the transmission of the vocal vibrations. This, of course, may vary in different cases. Sometimes bronchitis, with copious secretion, will lead to diminished resonance by obstructing the bronchi; tumours pressing upon the main bronchus will give rise to a similar result; sometimes the same effect will be produced by extensive pulmonary solidification, whether pneumonic, tuberculous, or cancerous,—consolidation so ex-

tensive, that the small stream of air which the lung still admits is unable to throw it into vibration; sometimes by the presence of cavities which have not a free communication with the bronchi; sometimes by emphysema; and at others by tumours external to the lungs, or by a thin layer of fluid in the pleura. Each of these causes, if proceeding beyond a certain extent, may not only impair, but altogether prevent the transmission of the voice sounds to the surface; but entire absence of vocal resonance is most commonly produced by extensive pleuritic effusion, pneumothorax, or the presence of very large tumours lying between the lung and the surface of the chest.

Increased resonance is a subject which requires more detailed consideration. It is always referable to one of two causes—either to consolidation of the lung tissue around the air-tubes, whereby the diffusion of the local vibrations through the ultimate ramifications of the bronchi and the air vesicles is prevented, the sound reflecting power of the tubes is increased, and the pulmonary parenchyma is rendered more homogeneous, and a better conductor of the vocal vibrations to the surface of the chest;* or else to dilatation of the bronchi, or the formation of abnormal spaces in the lungs capable of concentrating and reflecting sound with unusual intensity. In either case the condition specified may exist to a greater or less degree, and, *cæteris paribus*, the intensity and precise character of the vocal resonance will vary to a corresponding extent; so that in different cases, or at different periods of the disease, or in different parts of the chest in the same case and at the same period, every possible variety of tone may be met with, from the slightest augmentation of the natural voice sounds up to the loudest and most intensely developed form of increased vocal resonance.

* In speaking of consolidation and consequent increased homogeneity as a cause of increased vocal resonance, I would confine the observation to those cases in which the effused matter is of an elastic vibratile nature; for the presence of non-elastic material in the lung may actually diminish or altogether abrogate its voice-conducting power. This may be tried experimentally by injecting one lung with size or some other elastic material, and another with tallow or some non-elastic matter, and then listening through a stethoscope applied to one part of the lung, whilst a second person speaks through a stethoscope applied to another part of the same lung. The differences observable between the voice-conducting power of the lung, according as it is solidified by one material or another will then be strikingly apparent. I have repeated this experiment with a variety of materials; and I entertain no doubt that the varying sound-conducting power of different lungs, apparently equally condensed, is attributable to the varying elasticity of the matter to which their consolidation is due.

Five varieties, however, may be distinguished, as being sufficiently distinctive, and having each its own practical significance. The vocal, resonance may be simply increased; the voice sounds, unaltered in quality, may be heard louder than natural, but diffused over the surface of the chest, just as they are in health; or, 2ndly, their quality as well as their intensity may be altered, and they may be heard louder and clearer than natural, not distinctly articulated, and not appearing to pass through the stethoscope into the ear, but concentrated as it were beneath the stethoscope (bronchophonic resonance); or, 3rdly, they may be clearer and more distinctly articulated than natural, and may appear to be concentrated in the greatest degree, and to pass through the stethoscope directly into the ear, producing, when loud, a painful concussion on the ear of the observer (pectoriloquous resonance); or, 4thly, they may be loud, and of a metallic, ringing character, imperfectly or not at all articulated, not transmitted forcibly through the stethoscope, and conveying the impression of being produced in a large hollow space bounded by tense walls (amphoric resonance); or, again, 5thly, they may be of a tremulous, bleating, or quavering character forming a sort of Punchinello voice, synchronous with, but of a higher pitch than the voice of the patient, or else following it like a feebly whispered echo, and rarely traversing the stethoscope (ægophonic* resonance).

Now the first of these five varieties of increased vocal resonance, or that in which the voice sounds unaltered in quality, and diffused, as usual, over the surface of the chest, are heard louder and of greater intensity than natural, is met with whenever anything occurs to prevent the diffusion of the vocal vibrations through the smaller ramification of the bronchi and the pulmonary vesicles, and to increase the homogenity and vibratile power of a superficial portion of lung, the larger bronchi of which remain pervious. The obstruction to the diffusion of the vocal vibrations increases the number and strength of the vibrations in the bronchi which are still pervious, and the density of the surrounding tissue renders the bronchial tubes capable of reflecting sound more strongly than usual; whilst the increased homogeneity of the pulmonary tissue favours the transmission of the sound to the chest walls, and so to the ear of the observer. The conditions, therefore, under which simple increase of vocal resonance occurs, are slight infiltration, whether solid or semi-solid, of the lung-tissue, as from tubercle, cancer, pneu-

* From αιγος, the genitive of αιξ, a goat, and φωνη, voice.

monia, pulmonary congestion, and slight œdema of the lungs. Indeed, it would appear, that according as consolidation takes place to a greater or less extent, so the resonance of the voice increases, until it ultimately loses the character of simple increased resonance, and acquires that of the second variety.

The second variety (the bronchophonic), in which the sounds are heard louder, clearer, and more vibratory than natural, and concentrated, as it were, at the end of or beneath the stethoscope, may be produced by anything which favours the transmission to the chest walls of sound emanating from spaces of larger calibre than the small bronchial ramifications usually existing on the surface of the lung. Hence it may arise from a more advanced stage of pulmonary consolidation than that which gives rise to simple increased resonance; from partial condensation of the pulmonary tissue, as the result of external pressure; from enlargement or dilatation of the bronchial passages—a condition usually attended by thickening and induration of their walls, and by condensation of the surrounding tissue; from the presence of small empty vomicæ in the lungs; from large cavities, the walls of which are flaccid or irregular, and do not reflect sound strongly; and from dilatation of the bronchi and distension of the air-cells, in certain cases of vesicular emphysema. In all these cases, the air-containing spaces in the lungs are abnormally large, and the resonance emanating therefrom is in consequence unusually loud; or else the occurrence of condensation of the lung tissue, by causing obliteration of the air vesicles and terminal bronchi, not only checks the diffusion of the vocal vibration, but increases the sound-reflecting power of the bronchi, intensifies the resonance, and facilitates the transmission of sound from the larger bronchi to the surface of the chest. The only exception is when the extent and degree of consolidation are such that the vocal vibrations conveyed along the air passages which are still pervious, prove insufficient to throw the condensed mass into vibration.

The third variety (the pectoriloquous, the chest voice—from *pectus*, a chest, and *loquor*, to speak)—in which the sounds of the voice clearer, more ringing, and more distinctly articulated than natural, and concentrated in the highest degree, seem to pass directly through the stethoscope into the ear of the observer—is met with whenever, coincidently with great homogeneity and elasticity of the intervening lung tissue, there exist near the surface of the chest a space or spaces capable of concentrating the voice sounds, and reflecting them with great

intensity. It matters not what the nature of the space—whether a bronchus or a pneumonic, tuberculous, or other excavation; the only conditions essential to the production of pectoriloquous resonance are those above stated. Its peculiar and distinctive character is, that the merest whisper will reach the ear clearly and distinctly articulated. Its most common source is an empty tuberculous excavation in the lungs; but a large bronchus, surrounded by condensed lung, is not an unfrequent cause of it. Pneumonic abscess and cancerous excavations, being of rare occurrence, are quite exceptional causes of the phenomenon.

The fourth variety (the "amphoric," from *amphora*, a jar)—in which the sounds, though of a metallic ringing character, do not forcibly traverse the stethoscope, are very imperfectly, if at all articulated, and convey the impression of being produced in a large hollow space bounded by tense walls—derives its name from the resemblance of the sound to that produced by speaking into an empty jar. It is met with most commonly in cases of pneumothorax; but it may also arise in instances of large tuberculous excavations in the lungs, especially when the cavity communicates freely with the bronchi, and its walls are thin, smooth, and tense, and adherent to the anterior parietes of the chest.

Now, it is obvious that bronchophonic, pectoriloquous, and amphoric resonance are merely degrees or progressive developments of one and the same phenomenon, and that just as simple increased resonance is a gradual transition from natural resonance, so simple increased resonance may pass quite gradually into bronchophonic resonance, which in its turn may merge just as gradually into pectoriloquous or even into amphoric resonance. No precise line of demarcation can be drawn between the different varieties beyond those already described; and although amphoric resonance is not likely to be mistaken for the other varieties, it is often difficult to decide whether the resonance in any particular case is sufficiently concentrated to deserve the title of bronchophonic, or whether, again, in another instance, it should be termed bronchophonic or pectoriloquous. Fortunately, therefore, the decision of the question is not of material consequence; for no positive inference can be drawn as to the condition of subjacent parts simply from the existence of one or other variety. They all indicate the existence of large air-containing spaces in the lungs, an extraordinary reflecting power in the walls of the spaces in which the air is contained, and, usually, increased homogeneity of the surrounding lung tissue; but, except in extremely well-marked cases, it is impossible, from the vocal

resonance alone, to decide even approximately whether pectoriloquous resonance emanates from a large bronchus surrounded by condensed lung tissue, from a dilated bronchus, or from a vomica in the lungs.

Laennec stated that pectoriloquous resonance is a certain sign of an excavation in the lung substance, and even in the present day there are too many practitioners who so regard it, and thus are led into errors of diagnosis. Even at the apex of the lung, where no large bronchus exists, it is not of itself sufficient to warrant a conclusion as to the existence of a vomica, for loud pectoriloquous resonance may be transmitted through consolidated lung from a large neighbouring bronchus. When its character is hollow, ringing, and metallic, the presumption is strongly in favour of such a cause, though the evidence derivable merely from the sound is still insufficient to decide the question. Some of the most strongly marked instances of loud, ringing, pectoriloquous resonance I have ever met with occurred at the apex of the lung in connection with pneumonic consolidation of the upper lobe; and in some few instances I have observed this resonance in a strongly developed form over small extra-pulmonary tumours. Over large or dilated bronchi surrounded by consolidated lung it is by no means of unfrequent occurrence.

And in regard to the less developed forms of increased vocal resonance, still greater uncertainty exists. Not only may considerable increase of vocal resonance, whether simple or bronchophonic, result from comparatively slight consolidation of the lung, provided such consolidation be superficial in its position, but, on the other hand, the faintest bronchophonic resonance—not exceeding that which would be produced under certain circumstances by slight pulmonary consolidation, may result from a cavity even of considerable size, provided that its walls are flaccid and irregular, and incapable, therefore, of reflecting sound strongly; that it still contains some fluid; or, that it is seated deeply in the chest, with healthy pervious lung tissue intervening between it and the surface. Nay, more, if its communication with the bronchi is not free, vocal resonance may be inaudible over it, or, if occurring at all, may be transitory and intermittent; so that, on the one hand, the presence of pectoriloquous or bronchophonic resonance does not afford proof of a cavity in the lung, neither does its absence furnish evidence of the non-existence of a cavity.

In some instances, however, the character of the sound and the conditions under which it is heard suffice to indicate with tolerable certainty the source of the abnormal resonance. Thus, if at the apex of

the lung the vocal resonance be hollow, ringing, and metallic, and a *mere whisper* proves markedly pectoriloquous, and passes though the stethoscope into the ear, clearly and distinctly articulated, there can be little doubt that the sound emanates from an empty space occupying a very superficial position in the chest, having a free communication with the bronchi, and bounded by thin, tense, elastic walls, capable of concentrating and reflecting sound. From no other sort of space, and from no space in any other position, could a hollow, ringing, pectoriloquous resonance be conveyed to the ear under such circumstances; and as no such space exists naturally in such a position in the lungs, it is fair to conclude that the sound in question emanates from a vomica.

Some assistance in determining the cause of increased resonance may be derived in many instances from an investigation of the extent of area over which the abnormal sound can be heard. When it is of very limited extent and sharply defined, it probably results from a dilated bronchus, from a small vomica in the lungs, or from the pressure of a small extra pulmonary tumour; whereas when it is more extensive, and may be traced over a considerable portion of the chest, it is more probably due to pulmonary infiltration, whether pneumonic, tuberculous, or cancerous, and a gradual diminution in its intensity will be perceptible as the stethoscope is carried along the surface of the chest towards the healthy portion of the lung. A loud hollow metallic resonance, whether bronchophonic or pectoriloquous, if heard over an extended area, is almost necessarily derived from large bronchi surrounded by consolidated lung. The only other source of it is an enormous cavity.

There are some peculiarities attendant on the more highly developed forms of increased vocal resonance which deserve a passing notice, not on account of any practical value which attaches to them, but in consequence of a theory which has been founded upon them by Skoda and his followers.* I allude to the fact that, in certain instances, the voice heard through the stethoscope applied to the chest walls is as loud if not louder than the voice heard in the same manner over the larynx or trachea. Thus, when the cylinder is applied over a cavity in the lungs, the voice occasionally seems louder, and strikes with greater intensity on the ear than it does when auscultated over the larynx, which is its seat of production. What, then, is the cause of this reinforcement of the

* Whatever is here stated relative to the cause of increased vocal resonance, applies equally to the intensification of tussive resonance and the respiratory sounds.

voice? It is not my intention to discuss the question fully, inasmuch as it is one which requires lengthened consideration, and does not lead to any practical result; but a few remarks on bronchophonic resonance may not be out of place. First, then, it may be stated that Laennec attributed the variations perceived in the strength and clearness of the thoracic voice to variations in the sound-conducting power of the lung tissue. He considered that lung tissue in its normal condition, being non-homogeneous is a bad conductor of sound, and that, infiltrated or condensed, and thereby rendered more homogeneous, its sound-conducting power is increased; and that to such increased homogeneity and consequent increased sound-conducting power bronchophonic resonance is really attributable. But I have shown by direct experiments that the increase of homogeneity of the lung tissue, resulting from consolidation, will not lead to increased sound-conducting power in the lung, unless the consolidating matter be elastic or vibratile; and even if it were otherwise, it is obvious that mere consolidation or increase of homogeneity will not serve to explain the positive reinforcement or intensification of the voice, which is met with in certain instances of disease; neither will it suffice to explain the difference in pitch which is sometimes observed between the bronchophonic and laryngeal voice. Some further explanation must therefore be sought. Skoda, blinded by the result of imperfect experiments on the dead body, and in defiance of many facts observed in the living, asserts* "that sound is heard somewhat further through healthy than through hepatised lung;" and maintaining that "variations in the strength and clearness of the thoracic voice cannot be explained by the differences in the sound-conducting power of normal and abnormal lung parenchyma," rejects Laennec's theory *in toto*, and substitutes for it the doctrine of consonance—a doctrine at once fanciful and inconsistent with acknowledged facts. Not content with drawing attention to the possible increase of the voice sounds by consonance, under certain rare and exceptional conditions, the Viennese professor confidently proclaims that consonance is a principle of general application in the intensification of sounds audible on the chest walls, and that bronchophonic resonance and other phenomena, which Laennec attributed to the varying conducting power of healthy and diseased lung substance, are explicable only under this supposition. But those persons who are familiar with the laws of sound, must be aware that the conditions essential to the production of consonance

* Skoda, loc. cit., p. 39.

must be necessarily of rare occurrence in the air within the bronchi; and those who will observe carefully the phenomena attendant upon the sounds which Skoda has designated "consonating," will soon perceive that they commonly present characters which are quite inconsistent with the recognised acoustic phenomena of consonance.* Therefore, without denying that consonance is sometimes instrumental in intensifying, or reinforcing the vocal resonance, I am satisfied that it cannot often prove so, and that it is quite inadequate to explain the occurrence of bronchophonic resonance under ordinary circumstances.

What, then, is the cause of the phenomenon? When it occurs in connexion with consolidated lung, I believe it to be referable mainly to three causes—1st, the non-diffusion of the vocal vibrations, which results from the consolidation of the pulmonary vesicles and terminal bronchi; 2nd, the increased power possessed by the bronchi which are surrounded by condensed lung tissue of reflecting, and thereby intensifying the sound as heard by the ear; 3rd, the increase of conducting power acquired by the pulmonary parenchyma under certain conditions of consolidation and homogeneity. The effect of consolidation in preventing the diffusion and consequent weakening of the vocal vibrations, and in augmenting the reflecting power of the air passages, and thereby of inducing concentration and intensification of the voice sounds, must be obvious to all who consider the question; and although it is possible to conceive consolidation to result from infiltrated material of such an inelastic nature as to deaden all vibration, and to hinder, rather than facilitate the transmission of the voice sound to the surface of the chest; nay more, though observation and experiment † clearly prove that infiltrated matter does vary greatly in its vibratile power, and, therefore, in its powers of transmitting the voice—there is, nevertheless, abundant evidence to establish a vast increase in the conducting power of lung in certain stages of condensation and homogeneity ‡

But other circumstances are necessary to explain the positive intensification or reinforcement of the voice, which is often heard over vomicæ, dilated bronchi, &c., and also the difference of pitch observable, in some instances, between the laryngeal and thoracic voice. Here other causes

* For an excellent disquisition on the facts bearing upon Skoda's doctrine, see Walshe, loc. cit., pp. 142 to 152.

† See Note, p. 96.

‡ Dr. Scott Alison has pointed out that the increase of vocal resonance as heard through a stethoscope over solidified lungs is partly due to the fact that sonorous vibrations are transmitted more readily from solids to solids than from air to solids.

come into operation which, as regards the lungs, must be regarded as exceptional. I refer to those sources of increased or modified sound which are known as consonance, unison resonance, and echo. I quite agree with Dr. Walshe in believing that each of these agencies may come into play in the intensification of the voice sounds, under different conditions of the thoracic organs; but I am convinced that the two latter are those to which the phenomenon is most commonly attributable. For although it is conceivable that consonance should occur under certain rare and exceptional conditions, the laws which regulate its production, are such as to render its frequent occurrence in the lungs well nigh impossible; whereas, unison resonance, and echo may readily occur in many states of the pulmonary apparatus. Those who are curious in this matter, may refer to Dr. Walshe's pages, where the subject is fully discussed; and therefore, without going further into the matter, I will merely reiterate my opinion, that bronchophonic resonance is ordinarily referable to the three causes already specified, viz., non-diffusion of the vocal vibrations consequent on the obstructed condition of the air cells and the terminal bronchi; increased reflecting power of the bronchi; and augmented conducting power of the lung; but that where its intensity exceeds that of the laryngeal voice, or its pitch differs from that of the laryngeal voice, echo, or unison resonance, or both combined are usually concerned in modifying the phenomenon. Probably echo is the cause of its unusual intensification in certain examples of pulmonary consolidation, and unison resonance in cases of emphysema, whilst unison resonance and echo combine to produce the effects, in certain instances of vomicæ in the lungs. True consonance can only come into play when "the tones of the laryngeal voice chance to bear a certain mathematical relationship to the fundamental note of a resounding space in the chest."

The fifth variety of increased vocal resonance (the ægophonic), or that in which the voice as heard on the chest walls, is of a tremulous, bleating or quavering character, forming a sort of nasal Punchinello voice, is closely allied to bronchophonic resonance, being in fact nothing else than ordinary bronchophonic resonance modified, and rendered tremulous in its character. It is clear in tone, superficial in situation, and usually synchronous with the patient's voice, though sometimes following immediately after it, resembling a weak, bleating, silvery echo, vibrating as it were on the surface of the lungs; it is usually of a higher pitch than the natural voice and is not produced by every word, even though pronounced with equal force and with the same pitch; it rarely

traverses the stethoscope; is usually persistent; and though seldom continuing for longer than a few days, may endure for a period of several weeks. It is most commonly heard at the lower and posterior parts of the chest, in the vicinity of the larger bronchi, is audible over a very limited surface, and is often confined to a small space near the inferior angle of either scapula. Sometimes, however, it extends to the nipple in front, and occasionally may be traced rising gradually in the chest until it is heard at the very apex of the lung. Further, it is capable in some instances of being altered in position, or even temporarily got rid of by a change in the posture of the patient.

The cause of ægophonic resonance has proved a fertile source of theory and conjecture. Practically, this resonance is usually found as an accompaniment of effusion into the pleural cavity; is most marked when the fluid is small in quantity; rises higher in the chest with the progress of effusion, and is only heard about on a level with its surface; disappears when a large quantity of fluid has accumulated, and returns when absorption of the fluid has proceeded to a certain extent. Hence Laennec concluded that its presence is characteristic of a thin layer of fluid in the cavity of the pleura, and suggested that its peculiar character is sufficiently accounted for by flattening of the bronchi as a result of compression, and by the vibration of the thin layer of effused fluid. This opinion he fortified by reference to the peculiar character of the sound produced by the flattened mouth-pieces of the bassoon and oböe which he considered to be represented by the flattened bronchi, and further, by the experiment of listening to the voice through a bladder half filled with water placed on the interscapular region of healthy persons where natural bronchophony is very intense—a contrivance which he said imparted to the vocal resonance a tremulous ægophonic character. But even admitting the flattening of the bronchi suggested by Laennec (and its existence is more than problematical), such flattening cannot be regarded as analogous to the mouthpiece of a clarionet, which contains a thin vibrating plate; and the experiment of listening to the voice through a half-filled bladder has not succeeded in the hands of others. I have listened through a bladder half filled with water, applied not only over the interscapular regions, but over the larynx and trachea where vocal resonance is most intense, and have uniformly failed in obtaining a modification of the voice sounds in any degree resembling Laennec's ægophony. Nor have I been singular in the failure of my attempts to obtain the effect spoken of by Laennec. Dr. Davies and other experimentalists in this country and on the Continent have arrived

at the same results; and Skoda distinctly states,* that under the conditions alluded to, "the voice sounds just the same as through a piece of liver of the same thickness as the depth of water in the bladder." Further, and quite independently of experiment, extended observation and pathological research have shown that Laennec attached too much importance to the presence of fluid in the pleura as a cause of ægophonic resonance; for although they have confirmed to the fullest extent the frequent coexistence of pleuritic effusion and ægophonic resonance; nay more, though they have proved that in a pure, intensely developed and persistent form ægophonic resonance is never met with without the presence of fluid in the pleura, yet they have also shown that effusion may exist without this peculiar vocal resonance; and conversely, that ægophonic resonance may and does sometimes occur to a greater or less degree in pneumonic or tuberculous consolidation without any fluid in the pleura. Thus it has become evident that, however frequent the coexistence of the two phenomena, they cannot be regarded as necessarily in the relation of cause and effect.

I have already stated that ægophonic resonance will cease at any given spot as soon as a large quantity of fluid has accumulated, and the level of the fluid has risen above the spot at which the peculiar sound is heard; and further, that the surface over which it is heard is very limited in extent; but exceptional instances are sometimes met with which seem to negative both these statements. When adhesions exist between the lung and the costal pleura, so that the lung is retained in partial apposition with the chest walls, ægophonic resonance may continue for weeks, or even months, in spite of very copious accumulations of fluid in the pleural cavity; and under the same circumstances, if the adhesions are extensive, but partial only, it may be heard over a very large surface of the chest—sometimes, indeed, over the whole of the affected side. I have myself observed instances of both these occurrences, and instances have been put on record by various observers in this country and on the Continent.†

Further, the statement that ægophonic resonance may occur without the presence of any fluid in the pleura, requires a few words of explanation. Of the fact itself there cannot be a doubt. A peculiar resonance, not indeed so purely or so intensely ægophonic as that which accompanies pleuritic effusion, but still of a sufficiently shrill and tremulous

* Loc. cit., p. 69.

† See Laennec, 'Traité de l'Auscultation Médiate et des Maladies des Poumons et du Cœur;' Andral's 'Clin. Med.,' t. ii, obs. xxi; Walshe, loc. cit., p. 140.

character to deserve the title of ægophonic, is often heard in cases of pulmonary hepatization or tuberculous infiltration, and according to Skoda,* is met with occasionally in the interscapular region of women and children, even when the lungs are perfectly sound. I cannot say that I have ever met with such a sound in any woman or child whose lungs were healthy; nor do I think that with ordinary care a sound arising under such circumstances could be mistaken for or confounded with true ægophonic resonance; but the occurrence of such a sound over solidified lung is not very uncommon. It is most strongly marked when the ordinary voice of the patient is of a shrill, tremulous, or nasal character; and sometimes it so closely resembles the ægophonic resonance of pleurisy that it is impossible by the sound alone to determine its cause. But careful and oft repeated examinations will enable us to discriminate between the two cases. When the sound is dependent on pleuritic effusion, its seat is usually very limited, and often varies with the position of the patient; it rises higher and higher in the chest as day by day the effusion increases, and is not heard where the percussion sound is dullest, or, in other words, at the base of the lung, where the accumulation of fluid is necessarily greatest; whereas, when it arises in connection with pulmonary consolidation, it invariably occurs where the lung tissue is most condensed and the percussion sound dullest; is usually heard over a somewhat extended surface; is not affected by change of posture, and though seldom enduring above a few hours, may persist in the same spot for a considerable period in spite of continued extension of the area of dulness.

Skoda† has suggested that ægophony may "possibly be produced occasionally by a portion of mucus, &c., partially closing the mouth of the bronchial tube, imitating the thin tongue in the mouth-piece of tongued instruments," but that "probably in most cases the walls of the bronchial tubes within which the air consonates, react by impact on the air contained within them, and so give rise to the tremulous sound." The latter part of this theory, on which in fact Skoda rests the occurrence of ægophony under ordinary circumstances, is not only inexplicable, but utterly inconsistent with acknowledged facts; for if the bronchi always vibrate, as he admits they do when the air within them consonates, it is obvious that ægophony ought to be a constant accompaniment of the bronchophonic resonance which attends pneumonic hepatization, and

* Skoda, loc. cit., p. 68.

† Loc. cit., p. 72.

which, according to Skoda, results from consonance of the voice in the bronchi; whereas the very opposite is the fact, its occurrence under such circumstances being quite exceptional.

What, then, is the cause of ægophonic resonance? Setting aside those spurious cases in which an ægophonic character is said to attach to the vocal resonance in healthy women and children, and in which this peculiarity, if indeed it exists, must be attributable to the shrill, tremulous character of the natural voice—I say, setting these aside, there are two conditions which I believe to be invariable accompaniments of, and indeed essential to the production of ægophony—1st, a condition of lungs calculated to give rise to bronchophonic resonance; 2ndly, the existence of some agency able to impart to that resonance a tremulous, bleating character. This must be one of two kinds, either some tenacious secretion vibrating in the bronchial tubes and producing an effect analogous to the vibrating tongue of reed instruments, which possibly may be, though I somewhat doubt it, an occasional cause of ægophonic resonance over hepatized lungs; or, which I believe to be its source in cases of pleuritic effusion, the impulse of the vibrating and partly solidified lung against the costal pleura, an effect—viz., the repeated impulse of one solid vibrating body upon another—exactly analogous to that which takes place in the schoolboy's trick of speaking upon thin paper placed over the teeth of a comb, or in that of speaking, as Punch and Judy's showmen do, with a thin disk of metal or ivory so placed in the mouth as to lie between the lips and the teeth, and so to obstruct the egress of air from the mouth, in which case an ægophonic character is imparted to every sound by the jarring vibrations excited by the repeated impulses of the disk against the teeth. Under ordinary circumstances solidified lung lies closely in contact with the costal pleura, and practically for all purposes of vibration may be considered as connected with it; consequently, unless some cause of jarring, tremulous vibration exist within the lung itself, as in the instance of a vibrating piece of mucus in a bronchus, the vocal resonance will be purely bronchophonic, and not ægophonic. But just at the surface of a pleuritic effusion there must be a point at which the lung is barely separated from the chest walls, and in which the bronchophonic vibrations of the lung must lead to that light, jarring impulse of the visceral against the costal pleura, which analogy proves conclusively to be a frequent cause of a peculiarly tremulous, bleating sound—the sound which characterises ægophony.

Skoda laughs at the idea of three or four ounces of fluid giving rise to ægophonic resonance, and asserts that ægophony "will not be produced unless the fluid in the pleura be sufficient to deprive completely of air, by compression, a portion of lung large enough to contain a cartilaginous bronchial tube." But I am satisfied that this is not the fact, and that Laennec was correct in his statement, that a very small quantity of fluid may occasion intense ægophonic resonance. I have so often traced in the dead-house of St. George's Hospital the coexistence of scanty pleuritic effusion and ægophonic resonance, that I cannot doubt as to their mutual relation; and according to my views as to the cause of ægophony, the scantiness of the fluid is of little importance, if indeed it be not actually conducive to the production of ægophony. In fact, I consider the fluid to have no further influence over the production of ægophony than that of separating the two surfaces of the pleura to such an extent as to admit of the necessary vibratile impulse between them, and further, of interposing an imperfect conducting medium between the ear and the seat of bronchophonic resonance, which has the effect of preventing the transmission of vocal resonance, except in those parts where the stratum of fluid is very thin, and even in those parts so far diffuses the sound as to deprive it of its concentrated bronchophonic character. Consequently, if a lung be hepatized, or be otherwise in a condition to emit bronchophonic resonance, the smallest quantity of fluid may impart to that resonance an ægophonic character. It is impossible to prove that Skoda's suggestion relative to the presence of a vibrating plug of mucus in the bronchi may not *possibly* hold good, but the rarity of ægophony in patients oppressed by a quantity of tenacious mucus in their air tubes seems to render such an explanation highly improbable.

Practically, then, ægophonic resonance implies one of two conditions: either hepatized or otherwise solidified lung (or, according to its position, a large superficial bronchus or a cavity in the lungs), with tenacious mucus so placed in the air passages as to vibrate in a particular manner; or else the same condition of lung with effusion into the pleural cavity, slightly separating the two surfaces of the membrane, but admitting of their coming so far in contact at the spot where the peculiar sound is heard as to vibrate the one against the other. The latter is its common, its usual source; and when the sound is well developed and persistent, I believe it to be its invariable source. In certain instances of pneumonia, and in cases of tumours pressing into the pleural cavity,

the voice has somewhat of an ægophonic character; but if due care be taken in making the examination, and a conclusion be not arrived at until after two or three interviews, I believe with Laennec that well-developed ægophony may be depended on as proof of the existence of fluid in the pleural cavity.

Autophony, or the sound of the observer's own voice, as heard whilst his ear is applied to the chest, either directly or through the intervention of a stethoscope, affords another class of phenomena which have been called in aid of the diagnosis of thoracic disease. Few persons can have failed to remark that their own voice appears to them to sound or reverberate more strongly under certain conditions than it does under others. Thus, if the palms of the hands are placed over the ears whilst a person is speaking, the vibrations of the voice appear to the speaker to be confined within his own head, and to acquire an intensity very different from that of which he is conscious during ordinary speaking. The same effect is observed in a lesser degree when one ear only is covered. M. Hourmann went farther than this, and made a series of experiments of the same kind with substances of varying thickness, from which he concluded that the character and intensity of the resonance bear a definite proportion to the thickness and density of the substance, with which the ear is in contact. From this followed the inference that autophonic resonance would vary with the density of the contents of the thoracic cavity, and that these variations would prove valuable auxiliaries in the diagnosis of thoracic disease.* Unfortunately, observation has not borne out the practical results anticipated by M. Hourmann. There cannot be a doubt that the intensity of autophonic resonance varies greatly under different circumstances, and that consolidation of the lung is in many instances accompanied by a marked increase in its intensity; but it is equally certain that in some instances no such augmentation takes place, and that when the amount of consolidation is small, the variations of intensity are hardly perceptible. Indeed, observation has led me to believe that the intensity of autophonic resonance is attributable to the reflecting power of the substance with which the ear is in contact, rather than to its mere thickness or density; and if this be so, it must be modified as much by the condition of the chest walls as by the contents of the thoracic cavity. Be this as it may, the increase of resonance is certainly not proportioned to the mere density of the subjacent parts; and the

* 'Revue Médicale,' 1839.

numerous exceptions to the rule of there being any increase of it over consolidated lung, render autophony useless as a means of exploring the chest.*

Auscultation of the cough is another mode of examining the chest, and sometimes furnishes important information. In health, tussive resonance, as heard on the chest walls, is a short, dull, and indistinct sound, evidently produced at a distance, diffused over the surface of the chest, and accompanied by some degree of vibration of its walls. Its intensity is proportioned to the force of the cough and the thinness and elasticity of the thoracic walls, and it is heard loudest, and of a somewhat bronchophonic character, over the larger bronchial tubes. Indeed, whether in health or in disease, tussive resonance undergoes changes corresponding precisely with the alterations in the vocal and respiratory sounds under similar conditions of the thoracic organs. Thus, its intensity may be simply increased without material alteration in its character; or it may acquire a bronchophonic, pectoriloquous, amphoric, or even a quasi-ægophonic character; and, in cases where pneumothorax exists, and where large cavities are present in the lungs, it may be accompanied by metallic tinkling and amphoric echo. If fluid, mucus, or other matter be present in the air-passages, it may be attended by râles and ronchi, which, just as with respiration, will vary, *cæteris paribus*, according to the sizes of the passages or spaces in which they take their origin. In short, tussive resonance affords little or no information which may not be derived from the voice and the respiration; and in an auscultatory point of view, cough acquires its importance, not from the peculiar resonance which it produces, but from its power of removing mucus or other matters by which the air-passages are apt to become obstructed. Cough consists of a sudden and forcible expiration, followed by a quick, yet deep inspiration. The suddenness and force of the expiratory effort is often such as to dislodge obstructions in the bronchi, which cannot be got rid of in any other way; and thus a passage is cleared for the free ingress of the air during the deep inspiration, by which each paroxysm of cough is succeeded. By its means, therefore, we are often enabled to judge of the permeability or impermeability of portions of lungs over which no respiratory sounds could previously be heard; and may often discover a cavity which had

* Barth and Roger, Piorry, Skoda, and other authorities abroad, and Davies, Walshe, and others in this country, have arrived at the same conclusion.

remained uninfluenced by the voice, and the respiratory efforts in consequence of its approaches having been obstructed by mucus. By its means, again, we may sometimes discriminate between pleuritic friction, and those pseudo friction sounds which are met with occasionally in chronic bronchitis, as the result of tenacious mucus in the bronchi. For in the one case the friction sound continues uninfluenced by the act of coughing; whereas in the other it ceases as

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TABLE

RESONANCE OF THE VOICE, AS HEARD OVER THE

Variety.	*Synonym.*	*Character of sound.*
Laryngophonic	Laryngophony	The sound is loud and concentrated, in the highest degree, conveying the idea of being produced immediately beneath the stethoscope, and of passing through the instrument so as to strike directly and forcibly on the ear.
Tracheophonic	Tracheophony	
Bronchophonic	Natural Bronchophony	The sound is less loud, less concentrated, and less distinctly articulated than in tracheophony: it appears to be produced at a greater distance, to be more diffused, and to strike less distinctly against the ear, so that the words seem to be at the end of the instrument instead of passing through it into the ear.
Pulmonary	Ordinary vocal resonance	A diffused vibratory sound, amounting to little more than a humming or buzzing, and conveying the impression of distant origin. The articulation of the voice is not appreciable.

soon as the mucus by which it is caused is dislodged. In this manner cough becomes a valuable auxiliary in the exploration of the thoracic cavity.

The subjoined tables show at a glance the different varieties of vocal resonance in health and disease, their character, mode of production, and usual seat, and the diseases with which each morbid sound is usually associated:

I.

LARYNX, TRACHEA, AND CHEST WALLS, IN HEALTH.

How produced.	*Its usual seat.*
The vocal vibrations which pass directly down into the trachea, being concentrated in that narrow sound-reflecting tube, reverberate there with great intensity	Over the larynx and trachea.
The vocal vibrations which pass down the trachea and bronchi, owing partly to their being weakened by diffusion, partly to the lesser sound-reflecting power of the bronchi, as compared with the trachea, and partly to the intervention of the spongy tissue of the lung, reverberate less loudly than in the former situation	The upper part of the sternum and the posterior surface of the chest, between the spines of the scapulæ.
The vibrations of the voice which pass down the trachea and larger bronchi are obstructed, weakened, and diffused by passing through the subdivisions of the bronchi and the spongy tissue of the lung before reaching the surface of the chest	Over the whole surface of the chest, except the upper part of the sternum and the space which lies between the spines of the scapulæ, where the larger air tubes are superficial and near to the chest walls.

RESONANCE OF THE VOICE, AS HEARD

Class.	*Variety.*	*Synonym.*	*Character of sound.*
Diminished	Weak or feeble	Feeble resonance	The sound is simply less intense than it is over healthy lung tissue. Sometimes an indistinct humming only is heard
	Suppressed or absent	Absence of resonance	No sound audible
Increased	Simple increase	Increased resonance	The voice sounds, unaltered in quality, are heard louder than natural, but diffused over the surface of the chest, just as in health. This variety of increased vocal resonance gradually passes into the next variety
	Bronchophonic resonance	Bronchophony	The sound is louder and clearer than in simple increased resonance, is not distinctly articulated, and does not appear to pass through the stethoscope into the ear, but is concentrated, as it were, beneath or at the end of the stethoscope. This bronchophonic resonance passes imperceptibly into the next variety
	Pectoriloquious ditto	Pectoriloquy	The voice sounds are distinctly articulated, and transmitted directly through the stethoscope into the ear
	Amphoric ditto	Amphoric resonance	A ringing sound, of a metallic quality, not distinctly articulated, not transmitted forcibly through the stethoscope, and resembling the sound produced by speaking into an empty jar
	Ægophonic ditto	Ægophony	A tremulous, bleating, or quavering sound, forming a sort of Punchinello voice, synchronous with, but of a higher pitch, than the voice of the patient, or else following it like a feebly-whispered echo, and rarely traversing the stethoscope

HROUGH THE CHEST WALLS, IN DISEASE.

How produced.	*Its usual seat.*	*Disease with which it is commonly associated.*
By the occurrence of some physical change in the parts beneath the stethoscope, calculated to interfere with the transmission of the vocal vibrations to the surface	Not confined to any portion of the lung	Bronchitis, with copious secretion obstructing the bronchi; aneurismal or other tumours pressing upon a main bronchus; extensive inelastic pulmonary consolidation; emphysema, and slight pleuritic effusion.
By the same causes as lead to feeble resonance, if proceeding beyond a certain limit	Not confined to any portion of the lung	Each and all of the diseases above specified, when proceeding beyond a certain limit; but most commonly by extensive pleuritic effusion, pneumothorax, or the presence of large tumours lying between the lung and the surface of the chest.
By slight consolidation of the lung tissue around the air tubes, whereby the sound-reflecting power of the tubes is increased, and the pulmonary parenchyma is rendered more homogeneous, and a better conductor of the vocal vibrations to the surface of the chest	Not confined to any portion of the chest, but usually most marked and of the greatest significance towards the apices of the lungs in connection with the deposition of tubercle	In various morbid states, but, as a persistent condition, most common in the early stages of tubercular phthisis.
Sometimes by an increase of the pulmonary condensation which gives rise to simple increase of vocal resonance, and sometimes by the formation of abnormal spaces in the lungs, capable of concentrating and reflecting sound to as great a degree as moderate-sized bronchi	Not confined to any portion of the lungs, and inasmuch as it may be heard over the bronchi even in health, it is of clinical significance only when heard where bronchophony does not exist in health. Of great importance as suggestive of phthisis, when existing at the apex of the left lung	Pneumonic hepatization of the lungs; tubercular, cavernous, and other infiltrations of the lung tissue round moderate-sized bronchi; small vomicæ, as in phthisis; and dilated bronchi.
Sometimes by the condensation of lung tissue around a large bronchus, whereby the transmission of the sound to the ear is facilitated; more generally by the formation of abnormal spaces or cavities possessing smooth, sound-reflecting walls	Not confined to any portion of the lungs, but occurring most commonly at the apices and in the upper lobes	Chiefly in phthisis, but occasionally in pneumonia and other diseases in which the necessary physical conditions may exist.
By the reverberation of the voice in a large hollow space, bounded by tense sound-reflecting walls	The apices and upper portions of the lungs	Phthisis pulmonalis.
The vibration of the visceral against the costal pleura, at the point where the two surfaces are barely separated from each other, whether by fluid or by any other cause	Not confined to any portion of the lungs, but most persistent at the posterior surface of the chest, near the roots of the large bronchi, where the vocal vibrations are most intense	Chiefly in pleurisy, but also in certain instances of pneumonia, and of tumours projecting into the pleural cavity.

CHAPTER IX.

ON THE ADVENTITIOUS SOUNDS PRODUCED WITHIN THE CHEST BY THE ACT OF BREATHING.

The varieties of abnormal breathing already described are marked by modifications of the natural sounds of respiration. Those to which I have now to direct attention are characterised by the presence of sounds which find no analogue in healthy breathing, but are new and adventitious phenomena superadded to or replacing the healthy respiratory murmur. Such sounds may arise either from the air passages or cavities communicating with them, from the substance of the lung itself, or from the pleural cavities; and as they vary in character according to their seat and mode of production, it is necessary to understand the mechanism in which each takes its origin, before a proper estimate can be formed of their true significance.

First, then, in regard to those adventitious sounds which originate in the air passages or in cavities communicating with them. It has been already stated that in a healthy condition of the respiratory organs the air passages are free and open, whilst the mucous membrane which lines them is constantly moistened by a thin, watery exhalation which lubricates its surface and renders it smooth. Further, it has been shown that anything which checks the exhalation of this moisture, or produces unevenness on the surface of the bronchi, or gives rise to irregularity in or narrowing of their calibre, increases the friction encountered by the respired air, impedes its escape during expiration, and leads to a corresponding alteration in the breathing sounds. The first effects perceived by auscultation are roughness and prolongation of the sounds, especially of the sound of expiration. If the local changes are dependent upon causes of a transient nature, the roughness may gradually subside, and give place to the natural respiratory murmur. But more commonly, when mischief has gone so far as to produce roughness of breathing with prolongation of the expiratory sound, it is followed by further local changes, with a fresh train of physical phenomena. Not only does the calibre of the air passages undergo a still greater diminution, but the dryness of the bronchial mucous membrane is succeeded by morbidly

increased secretion. Thus, if the pulmonary mischief be not speedily resolved—in which case the roughness of the breathing gradually ceases—it usually happens that simple rough or harsh respiration is after a time replaced or accompanied by sounds which are dependent on the presence of secretion, and vary with its amount and character and with the altered size and condition of the bronchial tubes.

These sounds have been termed indifferently râles or rhonchi; the former being the term originally employed by Laennec, the latter derived from the Greek word ῥόγχος, being the term more generally employed in the present day.* Whatever they may be termed, however, these sounds are divisible into two species, which acquire their distinctive characters from the fact that in the one the sounds are occasioned by the vibrations into which the air is thrown by passing through channels irregularly contracted in their diameter; whilst in the other they arise from the bursting of bubbles caused by the air being forced through fluid, more or less viscid and tenacious, which has accumulated in and obstructs the air passages. The former, therefore, have been termed "dry" sounds, as contrasted with the latter, which, being connected with the presence of fluid, have been termed appropriately "moist" sounds.

But although these so-called "dry" sounds are invariably connected with narrowing of the channels through which the air passes, a very common element in their production is the presence of tough, viscid mucus or other semi-plastic material, which does not admit of the passage of air and the formation of bubbles, but which, without entirely obstructing the air tubes, adheres to their sides, contracts their diameter, and gives rise to, or admits of abnormal vibration. Indeed, in some instances the secretion and local accumulation of such mucus appears to be the chief cause of the constriction of the air passages and of the vibration

* Neither term, however, has any special signification, and each of them is inappropriate when applied to *all* the adventitious sounds originating within the air passages; for the term râle or rattle can hardly be regarded as applicable to many of the snoring, burring, cooing, and grunting sounds which are often audible in the chest, and the term rhonchus, which signifies snoring, is clearly inapplicable to the minute crepitations of pneumonia and to the fine bubbling sounds of capillary bronchitis. It appears to me better, therefore, to simplify our phraseology by substituting the word "sound" for these unmeaning or inappropriate terms. If, however, for the sake of convenience, the terms "râle" and "rhonchus" are to be retained, the former should be restricted to the sounds of bubbling, the latter to those of vibration. A distinction would thus be drawn between sounds which, though not necessarily indicative of a very different condition of the pulmonary tissue, yet take their origin in a different mechanism.

on which the production of the "dry" sounds depend; and in all cases, whatever the primary cause of the constriction—whether thickening of the bronchial mucous membrane, the pressure of tubercle or other matter, or, as often happens, irregular spasmodic contraction of the circular fibres of the air tubes—the secretion of viscid mucus at the seat of obstruction contributes greatly to the general result. This is evident from the frequent intermission of these sounds, and from their cessation for a time after the act of coughing—facts, which though quite incompatible with the theory of persistent contraction of the air passages, are strictly in keeping with the supposition that the contraction is in great measure referable to the presence of tough viscid mucus which admits of removal and change of position by the full blast of air which accompanies forcible expiration or the act of coughing. Therefore, although the impression conveyed to the ear by these so-called "dry" sounds justifies the term which has been applied to them, it must always be remembered that the use of the term does not imply a total absence of secretion, but only the absence of secretion of such a nature and in such quantity as shall lead, during the act of respiration, to the formation and bursting of air bubbles, and the consequent production of "moist" sounds.

In this limited sense the term "dry" is applicable enough; but if it be attempted, as is often done, to classify all the adventitious sounds originating within the lungs as either "dry" or "moist," then does the term "dry" become highly objectionable, as being inappropriate and calculated to mislead. Many of the sounds arising from simple abnormal vibration are connected, as already stated, with the presence of viscid secretion in the bronchi, and some even of the sounds associated with the bubbling of air through fluid in the air passages convey a distinct impression of "dryness." Such are the fine crepitations which are frequently met with in pneumonia, and the dry "clicking" which often accompanies the early stage of tubercular deposit. Moreover, a slight modification of the conditions which give rise to "dry" crepitation and "dry" clicking will occasion the setting up of "humid" crepitation and "moist" clicking; and thus sounds which pathologically acknowledge the same origin, and which differ only in the quantity and tenacity of the existing secretion, are separated and placed in contrast to each other, as if they were distinct and independent phenomena. The course, therefore, which appears to me simplest and least likely to lead to error and misapprehension is to divide all adventitious sounds originating in the air passages, according to their mechanism, into "sounds of vibration" and

"sounds of bubbling," and then the special character of each variety of vibration and each variety of bubbling may be described if necessary according to the notion it conveys to the ear.

Viewed in this light, and considered in reference to their practical significance, the adventitious sounds originating within the lungs may be arranged advantageously in the following manner:

Class.	*Variety.*	*Special Character.*	*Synonym.*
Adventitious sounds originating within the lungs. — Sounds of vibration.	Low-pitched or Grave toned.	Snoring. Cooing. Buzzing. Grunting. Creaking.	Sonorous rhonchi.*
	High-pitched or Shrill.	Whistling. Piping. Hissing.	Sibilant rhonchi.*
Sounds of bubbling.	Crepitating.	Minute, dry, rapidly evolved, crackling.	Crepitant râle,* dry crepitation, dry crackling.
	Bubbling.	Fine or small, and tolerably even-sized—bubbling.	Sub-crepitant, or muco-crepitant râle,* humid crepitation, or humid crackling.
		Coarse and irregular-sized—rattling.	Mucous or sub-mucous râle.*
		Large and hollow sounding — gurgling.	Cavernous râle.*
	Clicking.	Dry. Moist.	
	Indeterminate sounds.		

The nomenclature above employed is extremely simple, and the classification itself is not only in accordance with the mechanism of the sounds, but includes all the varieties which have the least practical significance. Other sounds or subdivisions of sounds have been dwelt upon at length by many authors as diagnostic of particular forms of bronchial effusion or special forms of pulmonary mischief, but such distinctions have no real existence, and only tend to confuse the student and complicate the study he has in hand. Nothing can be more perplexing than a needless multiplication of terms, and therefore in this classification I have confined myself to those sounds which can be traced as having a constant relation to some particular condition of the pulmonary apparatus, and consequently are of real practical importance.

* These have been termed indifferently râles and rhonchi, but, as already stated, the term rhonchus is most appropriate as applied to the sound of vibration, and the term râle to the sound of bubbling.

The precise character of the sounds of vibration varies with the force of the blast and the size, form, and condition of the channels through which the air has to pass. The stronger the current of air and the narrower the chinks or channels through which it is forced, the higher (*cæteris paribus*) the pitch, and the more whistling the character of the tone produced. And as every possible variety in the condition of the air passages may exist in different portions of the chest, so every variety in the sounds of vibration, from the deep, low-pitched note of a large tube to the shrill, high-pitched whistle of a narrow chink or channel, may be heard sometimes in a single case. The grave, low-pitched tones are usually compared to the snoring of a person asleep, and do sometimes very closely resemble it; occasionally they more nearly resemble the noise of grunting, buzzing, cooing, or creaking,* and now and then they assume a "rubbing character;" but whatever their nature, they may all be included under the general appellation of "sonorous" rhonchi; whilst those of a higher pitch, whether whistling, piping, or hissing, may be grouped under the title of "sibilant" or whistling rhonchi. Both classes of sounds are connected with the same mechanism, and the variations in their character are due to differences in the size of the air passages in which each takes its origin. As a general rule, the low-pitched "sonorous" varieties originate in the large bronchi, and the high-pitched "sibilant" varieties in the smaller tubes; but inasmuch as the calibre of the large tubes may be constricted and rendered small by disease, it is obvious that no dependence can be placed on the mere discovery of sibilant rhonchus as evidence of an affection of the smaller order of bronchi. The grave, sonorous rhonchi are undoubtedly characteristic of an affection of the larger tubes, and an extensively diffused sibilant ronchus may be relied upon as proof of an affection of the

* This is a point to which I would especially direct the attention of the student; for I have repeatedly seen this creaking and rubbing rhonchus mistaken for the friction sound of pleurisy. Unfortunately the mistake is not confined to students. Several cases have fallen under my observation which have been wrongly treated by practitioners more or less acquainted with the use of the stethoscope, under the belief that their patient was suffering from pleurisy. The mere absence of ægophony, as will be shown hereafter, is not sufficient to mark the case as one of bronchitis, but a distinction exists between the sound in the two cases which is simple and conclusive. Cough and expectoration, by getting rid of the mucus, removes the cause of the rhonchus, and thus leads to its modification or complete temporary cessation; whilst if the sound is dependent on pleuritic friction, it continues uninfluenced by the act of expectoration. Thus, if care be exercised, a satisfactory conclusion can be arrived at, often at the first, and always in the course of two or three examinations.

smaller bronchi; but when such a rhonchus is of limited extent, it may not impossibly proceed from a large bronchus which has undergone constriction.

It might be imagined, from what has been already stated relative to the source of these vibrating sounds, that they would necessarily accompany both acts of respiration. Very generally they do so to a greater or less extent, but sometimes they are heard during inspiration only, and at others are confined to the act of expiration. Varying greatly in intensity and duration, they are at one time slight, and at another so loud, as to mask or overpower the natural respiratory murmur. They may be audible not to the patient only, but to persons at some distance from him, and, when they are of a low-pitched sonorous character, they may convey a sensation of fremitus or vibration to the hand placed upon the chest walls. They rarely accompany every act of respiration, but occur irregularly and interruptedly, persisting occasionally for some length of time at a particular spot, and then, suddenly and for a time at least, ceasing—the cause of their cessation in most cases being the removal of some tough mucus by the act of coughing. In many instances, however, the high-pitched sibilant varieties are less readily got rid of by coughing and expectoration than those which are grave or of a lower pitch—a circumstance which is explicable by the comparative weakness of the blast in the smaller bronchial ramifications whence the sibilant rhonchi usually originate, and by the greater difficulty which consequently attends the removal of the tenacious mucus.

According to the form of disease by which they are occasioned, these sounds of vibration may be confined to one spot, or one particular portion of the chest, or may be diffused generally over its surface. Probably, if caused by the pressure of aneurismal or other tumours, or by the lodgment of foreign bodies within the bronchi, they will be limited in extent, and more than usually persistent; if occurring as a consequence of bronchitis, connected with the deposit of tubercle or other matters, they not only will be persistent, but will abound in those parts especially which are prone to suffer from the disease in question; whereas, if they arise in consequence of bronchitis unconnected with any local cause of irritation, they will usually extend more generally over the chest, and will subside more readily under treatment. Moreover, if the disease does not speedily subside, they will be accompanied or followed, after no lengthened period, by the

occurrence of bubbling sounds, resulting from the increased secretion which accompanies its further progress. Their most striking development is certainly met with when emphysema coexists with chronic bronchitis, in which case the expiratory sound is enormously prolonged, and the noises produced within the chest are of such variety as to defy description.

Thus, then, to sum up the practical information afforded by these sounds of vibration, it may be stated generally that their presence indicates constriction of the air passages, and the probable presence of tenacious mucus; that their persistence for a lengthened period over a limited area, leads to a presumption of some mechanical local obstruction, such as the pressure of an aneurismal or other tumour, or the presence of a foreign body in the bronchus, or, when they are confined to the apices of the lung, the irritation of tubercular deposit; whilst their existence over the entire chest, or over an extensive area, indicates the existence of wide-spread bronchitic irritation, which, if not arising from idiopathic causes, must be due to some generally diffused cause of local mischief, such as tuberculous or cancerous infiltrations. In either case their presence should lead us to expect the speedy occurrence of increased bronchial secretion, and the consequent production of bubbling sounds.

The low-pitched sonorous sounds which invariably originate in and are altogether confined to the larger bronchi, are usually of less serious import, than the high-pitched sibilant rhonchi, which, when widely diffused throughout the chest, indicate the implication of a smaller order of air tubes, and the existence of extensive affection of the respiratary apparatus, requiring immediate attention and active treatment.

The "bubbling" sounds, whatever their seat or position within the chest, are caused by the passage of the respired air through fluid more or less tenacious, which has accumulated in and obstructs the bronchial tubes. Their character, however, varies considerably, according to the consistence of the fluid, the size of the space in which it is collected, and the force with which the air is driven through it—circumstances which determine the size and number of the bubbles, the compression to which the contained air is subjected, and consequently the sound which the bubbles cause on bursting.* The less tenacious

* The influence exerted on the pitch and intensity of the sound, and on the readiness with which it is conveyed to the ear, by the nature of the space in which the bubbles

the fluid, the smaller, *cæteris paribus*, will be the size of the bubbles; the less the resistance to their passage through it, the more rapid their succession, the more frequent their bursting. Again, the greater the energy of the respiratory movements, and the greater consequently the force with which the air is driven through the fluid, the more numerous will be the bubbles in any given time, and the less distinct or separate the sound which each one produces in bursting. But the size of the tube or cavity containing the fluid exercises a further and most important modifying influence. The larger the space, the greater the freedom with which the air passes, and the larger and *more unequal* the size of the bubbles; so that the same fluid which in the smaller bronchial tubes or air passages would give rise to a fine and tolerably uniform bubbling, will in the larger tubes produce a coarse and more irregular-sized bubbling—sometimes resembling a rattling. In tubes or cavities of still larger size it will occasion a still more irregular and hollow-sounding bubbling, or, in other words, a gurgling, quite characteristic of air passing freely through liquid and producing large unequally sized bubbles; so that according to the character of the sound we are enabled to form a tolerably correct estimate of the size of the tube or cavity from which it originates, and of the consistence of the fluid by which the bubbling is caused.

Crepitation.—If the bubbles are very minute, of uniform size, burst in rapid succession, almost as in a volley, and emit a dry, crackling, or crepitating sound, rather than a liquid, bubbling sound on bursting, the air passages in which the sound originates are extremely small. Indeed, such a bubbling indicates the entrance of air into the pulmonary vesicles, and proves that no disease exists of such a nature as to render the lung impermeable and prevent the expansion of the air vesicles. This sound very closely resembles the noise one hears when a lock of dry hair is rubbed transversely and slowly between the finger and thumb close to one's own ear, or when common salt is thrown on the fire; and has been termed, not inaptly, crepitation. It is the crepitant râle or rhonchus of authors, and usually, but not invariably, accompanies the accession of pneumonia, and in some few instances is met

burst, and by the state of the surrounding pulmonary tissue, will be fully discussed hereafter. My present object is simply to point out how the character of the bubbling and the sound it produces are modified, *cæteris paribus*, by the consistence of the fluid, the size of the space in which it is collected, and the force with which the air is driven through it.

with in connection with congestion of the lungs and œdema of the pulmonary tissue.* It is almost confined to the act of inspiration, which it sometimes accompanies from beginning to end, though it occurs more commonly towards its close; it is usually persistent for some length of time at the spot where it is first evolved; is heard most distinctly after deep inspiration or after a fit of coughing, and is not checked by expectoration—circumstances which usually serve to distinguish it from the fine bubbling sound, the variety next to be described, into which it gradually merges, and with which it is confounded not unfrequently.

Some persons have been unwilling to admit crepitation to be referable to bubbling, on the ground that if it were due to the passage of air through fluid in the pulmonary cells, it should accompany expiration as well as inspiration. Hence they have suggested that it is dependent upon the sudden unfolding of the walls of the air cells, rendered dry and crackling by disease, or else upon the giving way of exudation matter which has been effused between the vesicles in the parenchyma of the lung. Neither of these theories, however, appears to me to be tenable. Observation in the dead-house proves conclusively that pneumonia does not produce dryness of the pulmonary tissue, such as could give rise to crepitation; and even were it otherwise, the dryness of the tissue should occasion crackling during the collapse of the lung almost as much as during the act of expansion. This may be seen experimentally, on inflating a dried bladder, and then compressing it so as to cause it to assume a folded condition. Nor does the second hypothesis rest on more stable ground. Indeed, in most cases, pathological research proves the absence of the cause to which the sound is attributed. Dissection of a portion of lung, in the first stage of pneumonia, shows congestion of the pulmonary capillaries, and effusion of a viscid serosanguinolent fluid into the air vesicles; but in a vast majority of instances it fails to discover the intervesicular or parenchymatous plastic

* This fact is too often lost sight of in practice, and leads those who trust too exclusively to auscultation into grievously erroneous treatment. Instances of well-marked crepitation, not to be distinguished from the crepitation of pneumonia, are met with not unfrequently during the pulmonary congestion which often accompanies continued fever and other asthenic and hæmic disorders, as also during the early stage of pulmonary œdema arising in connection with acute anasarca. It is obvious that in cases such as these the active and depressing treatment which might be appropriate in combating inflammation of the lungs would here be productive of mischievous results.

exudation, to the giving way of which this theory refers. And if it be supposed that the sound is attributable to the movement of fluid in the areolar tissue during respiration, no greater advance is made towards an explanation; for obviously, under the conditions referred to, the sound should accompany the collapse of the lung during expiration, just as much as it does its expansion during inspiration. Indeed, I cannot but think that the objection raised to the bubbling theory of crepitation rests altogether upon a false assumption. It is quite intelligible, as stated by Dr. Davies, that "the air having forced its way through fluid into the interior of the lung cells, may be expelled from those little cavities without being compelled to pass a second time through the secretion. At the end of an inspiration the cells are fully expanded, and space is afforded for the presence of air and fluid. At the commencement of the expiration, the more elastic fluid, the air, is first driven out, finding a free passage from the vesicles into the terminal bronchial ramifications."* I am satisfied, therefore, that crepitation may be regarded as the result of air bubbling through fluid in spaces of very small calibre, such as the pulmonary air cells, and that this is the only satisfactory explanation.

Fine or small, even-sized bubbling.—If the bubbles be less delicate and minute, less uniform in size, less rapidly evolved, less regular in their occurrence, and, though distinctly crepitating, are less dry in their character, and convey the impression of being connected with the bubbling of air through fluid, the bronchi or air passages in which they originate are still small, but the fluid is larger in quantity; for it is only in such tubes or spaces, and with such an amount of fluid, that fine, even-sized bubbling can be produced. This is the "sub-crepitant" or "muco-crepitant" râle of authors, and is the sound which accompanies capillary bronchitis, the first stage of œdema of the lungs, the effusion of blood or other fluids into the pulmonary tissue, and the resolution stage of pneumonia. It is, in fact, only a modification of crepitation, but, unlike the true crepitus of pneumonia, it is influenced and for a time got rid of by coughing, and accompanies expiration as well as inspiration, the fluid being in the terminal bronchi, and in such quantity that the air has to force a passage through it as well in expiration as in inspiration.

Coarse, irregular-sized bubbling; rattling.—If the bubbles be larger, of variable size, less regular in their occurrence, and distinctly liquid

* Davies, loc. cit., p. 175.

the air passages whence the sound originates are of larger size, admitting of more free and unrestrained bubbling. This is the "mucous" or "sub-mucous" râle of authors, and is the sort of bubbling which accompanies the secretion stage of bronchitis, the occurrence of œdema, hæmoptysis, or whatever leads to the presence of fluid, whether mucus, pus, blood, or serum,* in air tubes or passages of moderate size. It accompanies both respiratory movements, and is modified by the acts of coughing and expectoration, whereby the fluid in the air passages is removed, and the sounds for a time diminished or arrested. It must be remembered, however, that whatever the precise character of the bubbling, its amount does not necessarily afford an index to the quantity of fluid contained in the air passages, for these may be so filled that little or no air can pass, and little or no bubbling, therefore, can be produced. In some such instances, all respiratory sound ceases, whilst in others slight bubbling sounds still continue. In the more advanced stages of bronchitis, and during the occurrence of œdema, this happens not unfrequently, and can be detected by the presence of more or less dulness on percussion; by the absence of respiratory murmur; by the want of proper expansion of the chest walls; and, indeed, by the various signs which indicate the existence of impervious lung. But if the bubbling be small in amount, consisting of single or occasional bubbles, and the vesicular murmur remains distinctly audible, it is fair to conclude that little fluid exists in the air passages;† whilst if the bubbling be extensively diffused, and be not accompanied by a respiratory murmur, or be heard only in combination with a faint, indeterminate respiratory murmur, it is equally a legitimate conclusion that the bronchial tubes contain a large quantity of secretion.

Large, hollow-sounding bubbling; gurgling.—If, again, the bubbles be still larger and of unequal size, less numerous, and more irregular in their occurrence, often disappearing for a time, and conveying to the ear the impression of hollowness, the fluid is probably more uniform in its consistence than in the former instances, and the air passages or spaces whence the sound originates are larger. This is the gurgling or so-

* The attempts which have been made by M. Fournet and others to draw distinctions between the sounds produced under these several circumstances are calculated to give rise to endless confusion. No differences exist sufficiently well marked and of such constant occurrence as to possess the slightest diagnostic value.

† Even, under the conditions here supposed, fluid may be pent up in cavities undisturbed by the respiratory blast.

called cavernous râle of authors, and is a modification or further development of the so-called mucous or submucous râle which often merges into it. It is the sort of bubbling which occurs in the main divisions of the bronchi, in large dilatations of the bronchi, in tuberculous or other excavations in the lungs, and, in short, wherever there is an accumulation of fluid in large spaces having a free communication with the air passages. It commonly exists with both inspiration and expiration; occurs at different periods of the expiratory act, according to the relative position of the contained fluid and the communicating bronchus; is greatly modified by cough and expectoration; and when the walls of the cavity are thin, tense, and elastic, has a peculiarly ringing, metallic character.

The size of the bubbles of which gurgling is made up varies greatly in different cases, and as the character of the sound produced is regulated to some extent by the size and number of the bubbles, some authors have applied the terms "amphoric," "cavernous," and "cavernulous" to different sized gurglings, under the impression that the size and number of the bubbles form an index to the size of the cavity. Nothing, however, can be more erroneous than such a supposition. The character of the sound depends not only on the extent of the cavity, but on the nature of its walls, the freedom of its communication with the air passages, the condition of the surrounding lung tissue, the quantity and nature of the fluid contained in it, and the force with which the air is driven through that fluid. If the cavity, though large, be nearly full of fluid, and if that fluid be very viscid, and if the communication between the cavity and the air passages be not free, the amount of bubbling must be very small, whatever the size of the cavity. Again, cavities bounded by thick, inelastic walls, and surrounded by solidified, uncollapsable lung, are incapable of contracting, and equally so therefore of expelling much air during expiration. Hence it follows that they cannot receive much air during inspiration, and consequently can admit of comparatively little gurgling. On the other hand, cavities bounded by thin and elastic walls and surrounded by pervious lung tissue are greatly influenced by the respiratory movements, and expel a large portion of their contained air at each expiration. They are capable, therefore, of admitting a considerable quantity of air during inspiration, and this produces a large amount of gurgling. Moreover, if the walls are thin, tense, and elastic, the sounds reverberate loudly and

perfectly, and if the cavity is seated superficially they reach the ear without much difficulty; whereas if the walls are thick, spongy, and inelastic, the sounds are very imperfectly reflected and are not readily transmitted to the ear. Thus it often happens that a small cavity with thin, elastic walls produces more gurgling and splashing, and transmits a louder sound to the ear, than a much larger cavity bounded by thick, spongy, inelastic walls.

The statements already made show that anything which prevents the passage of air through fluid must necessarily check the production of gurgling. Hence the temporary cessation of the sound may be due to causes of several kinds—either to complete evacuation of the fluid; or to its diminution to such an extent as to bring its level below the opening of the tube or tubes communicating with the cavity; or to obstruction of the air passages which lead to the cavity. When its cessation is dependent on the complete evacuation of the fluid, the gurgling is usually replaced by hollow, breathing sound; when upon the second-mentioned cause, the gurgling may be often reproduced by change of posture and the consequent bringing of one of the openings below the level of the fluid in the cavity; and when it depends upon the third-named cause, coughing will often lead to its re-establishment by dislodging a plug of mucus or muco-purulent matter. When the obstruction is due to external pressure, cough has not much influence in restoring it, and its cessation is more permanent; and when again the space or cavity is completely full of fluid and does not admit the presence of air, gurgling cannot be produced.

Sometimes, when a cavity is unusually large, has very elastic walls, and contains a large quantity both of air and fluid, succussion of the patient, or the impulse against the cavity produced by the action of the heart, will give rise to an admixture of the air and fluid, and occasion a gurgling or splashing sound. I have had the opportunity of noting this fact on three occasions; and it is quite possible to conceive that if the communicating bronchi were obstructed, a gurgling or splashing sound might be thus produced, when no such sound was attendant upon respiration.

Another modification of bubbling is "clicking." Authors are not agreed as to the mode of origin of this sound, nor, indeed, have I ever met with a reasonable interpretation of it. Dr. Walshe says of dry clicking,* "the rhonchus, *though its mechanism is unexplained,* is of con-

* Loc. cit., p. 115.

siderable diagnostic importance;" and again, in reference to moist clicking,* "Its mechanism is almost as obscure as that of the dry crackling." But careful consideration of the conditions essential to its existence, and repeated experiments as to its mode of production, have convinced me that when it is met with in the lungs it is due to the sudden and forcible passage of air through a small bronchus, the sides of which, at one or more points, have been brought close together by external pressure, or have been agglutinated, as it were, by tenacious mucous secretion. Thus its common cause is the presence of tubercle pressing here and there upon the walls of the smaller bronchi, and not only rendering them impervious, but exciting slight local irritation, with the consequent secretion of viscid tenacious mucus. The bronchi are completely obstructed, and therefore do not admit of the production of the ordinary sounds either of bubbling or vibration; but now and then, perhaps two or three times in the course of an inspiration, their walls are separated for a moment under the pressure of the inspired air, and as the obstruction yields and the sides of the passage are forced asunder, the connecting mucus, which is drawn out into a sort of membrane, suddenly bursts, and a sharp click is produced, which conveys an impression of dryness or moisture, according to the quantity and tenacity of the fluid.

Dry clicking, like dry crepitation, originates in connection with a very small quantity of viscid secretion, and is met with almost exclusively during inspiration; whilst humid clicking, like humid crepitation or fine bubbling, is connected with a somewhat larger quantity of fluid, and though most distinct and constant during inspiration, occurs not unfrequently during expiration. When dry clicking first makes its appearance, it is audible only with forced respiration, and even then does not accompany each inspiratory act; but after a time it becomes more persistent, and is heard accompanying ordinary respiration. The dry variety passes, after a time, into the moist variety, and the latter, as the pulmonary disease progresses and softening commences, is replaced by the ordinary sound of bubbling. This arises from a diminution in the amount of pressure on or obstruction of the bronchi whence the sound originates, and from an increase in the quantity of fluid in the air passages, whereby it happens that the air no longer encounters mere films of mucus, but, by forcing its way through a more copious secretion, creates distinct bubbling.

* Loc. cit., p. 115.

Clicking, then—the "clicking" or "crackling" râle or rhonchus of authors—is commonly associated with the presence of tubercle, and the moist variety is regarded by some persons as quite characteristic of the early stage of tubercular softening. But careful observation at the bedside of the patient, borne out by the result of dissection after death, has convinced me that this opinion is erroneous. Not only have I met with dry clicking under circumstances in which the whole history and symptoms of the case, no less than the subsequent career of the patient, have satisfied me that no tubercles existed, but twice in the wards of St. George's Hospital I have met with its characters well marked, when subsequent *post-mortem* examination has enabled me to establish the non-existence of tubercle.* Therefore, although it is perfectly true that tubercle is its ordinary cause, and that when clicking is met with, as it usually is, in the infra-clavicular, supra-clavicular, or supra-scapular regions, its presence justifies the gravest suspicion of tubercle, we must not conclude without further evidence that our patient is indeed consumptive. Anything, whether tubercle or other matter, which, in one or more of the smaller bronchi, surrounded by partially consolidated lung tissue, shall occasion occlusion of such a nature as will yield now and then to the force of the respiratory blast, may prove the cause of this peculiar sound. But it is usually in the early stage of phthisis, when tubercles are sparsely scattered through the pulmonary tissue, that clicking is most frequent; for it is only when the disease is not extensive, when consolidation is of small extent, and when considerable expansion of the lung still takes place during inspiration, enabling the air to force its way through an obstructed passage, that the conditions exist which are essential to its production. The pressure of a large mass, whether of tubercle or other matter, will usually obstruct the bronchi permanently, and by presenting an insuperable obstacle to the passage of air will prevent the occurrence of clicking.

If it be asked how the clicking which results from tubercle can be distinguished from that which sometimes, though rarely, accompanies other forms of pulmonary disease, I answer that they both take their origin in the same mechanism, and that by the character of the sound alone it is impossible to discriminate between them. But I am con-

* In one of the cases alluded to the existing disease was chronic bronchitis, in the other sanguineous effusion into the lungs, as the result of valvular disease of the heart. The clicking, though well developed, was of temporary duration, and in neither instance was heard longer than five days.

vinced that oft-repeated examinations of the chest will generally assist us to a correct diagnosis. In no single instance in which there were not good grounds to suspect the existence of tubercular disease have I found clicking a persistent condition; and if when dry clicking is first observed it continues for a considerable period, and is after a time followed by humid clicking, which, in its turn, is ultimately replaced by bubbling, there cannot be a doubt as to the existence of tubercle. So that this sign, so little insisted upon by some authors,* is, in fact, of great clinical significance.

I must not quit this part of our subject without alluding to a sound which has been described by Laennec under the title of "râle crepitant sec à grosses bulles, ou craquement." It is characteristic, he says, of emphysema, is heard only during inspiration, and resembles the noise made by the sudden inflation of a dried bladder. It is a sound of rare occurrence, and of little diagnostic value; and it would not be worthy of special remark, had it not been described and first pointed out by the Father of auscultation, and adopted more recently by Skoda of Vienna. Even now, my chief object in mentioning it is to add my testimony to that of the many modern writers who refuse to assign it a place in the list of râles and rhonchi. It certainly does not possess a true bubbling character, as its name would imply, and probably is a sound of vibration connected with the extremely viscid and tenacious bronchial secretion which ordinarily accompanies vesicular emphysema. Certain it is that such a condition of secretion, exciting vibration in air passages altered by emphysema, is quite sufficient to account for its existence, and is far more likely to give rise to its production than the condition of the lung tissue, suggested by Laennec; for, with Dr. H. Davies, "I cannot conceive the pulmonary membrane which forms the lung cells to be so dry in emphysema as to be capable of crackling on expansion like a dried pig's bladder."† Further, pathology does not countenance Laennec's hypothesis; and we are bound, therefore, to reject it. Whatever the precise cause of the sound, there cannot be a doubt that at present it possesses little practical value; and my advice, therefore, to the student would be, not to encumber himself with the term given to it by Laennec, but to regard the sound, whenever he may chance to meet with it, as a subject for careful investigation.

* Dr. H. Davies does not even allude to it in his chapter on "Râles and Rhonchi," nor is any mention made of it in Skoda's work.

† Loc. cit., p. 181.

Skoda speaks of bubbling sounds which he terms "consonating." "Râles," he says,* "arising in the larynx, trachea, or either of the bronchi, may consonate within the thorax, just as the voice or the respiratory murmur consonates in the diseased states of the lung tissue already referred to, and thus become distinctly audible throughout the thorax." They are "clear and high-pitched," he says,† "formed by unequal bubbles, and accompanied by a resonance which has neither an amphoric nor a metallic character." Thus, his description answers to that of ordinary, coarse, unequal-sized bubbling, taking place in tubes surrounded by lung tissue in certain states of consolidation; and as Skoda himself admits that these so-called consonating sounds are accompanied by bronchial breathing and increased resonance of the voice, and, "generally speaking, indicate the presence either of pneumonia of of tubercular infiltration," there can be little doubt that pulmonary consolidation is a condition essential to their existence.

What, then, is the peculiarity, and what the value of these so-called "consonating râles?" In my opinion, they possess no single feature which entitles them to an independent place amongst recognised râles and rhonchi. It is universally admitted, and is strictly in accordance with the laws of acoustics, that sounds produced in any part of the air-passages may be propagated to the thoracic walls through air contained in bronchi capable of reflecting sound and through lung tissue in certain states of homogeneity and consolidation; and it is equally certain that in peculiar states of the bronchi and pulmonary tissues, all sounds generated therein will be of a high-pitched character. But, if due care be exercised in conducting the examination, there is little chance of râles propagated from other parts of the chest being mistaken for râles of local origin. The history and progress of the case, the condition of the respiration at the spot where the sounds are heard, and the presence or absence of dulness or unusual resonance on percussion will generally enable us to determine the question. Even if it were not so, and admitting, for the sake of argument, what really is not the fact, viz., that consonance is a principle of general application in the intensification of sounds propagated through the chest, still the sounds alluded to by Skoda would not possess any special significance; for Skoda himself fails to point out any certain signs by which to recognise the fact of their being generated at a distance, and reinforced and propagated by consonance. The only features which he indicates

* Loc. cit., p. 118. † Loc. cit., p. 131.

as calculated to excite suspicion as to the true nature of the case,—viz., the clearness and high pitch of the sounds, and the dulness or unusual resonance on percussion—are one and all compatible with the local production of the sounds, and their direct transmission by conduction. Therefore, my advice to the student would be to discard "consonating râles" from his vocabulary, as having at the most a problematical existence, and as being undistinguishable from other râles, and therefore possessing little diagnostic value. In discarding the term, however, he should recollect the fact, that large irregular-sized high pitched bubbling sounds heard over solidified lung do not necessarily imply the existence of a cavity immediately beneath the surface of the part auscultated, but may be sounds generated in the larynx or trachea, in a distant cavity, or in large bronchial tubes, and thence transmitted along the air in the bronchi, and through consolidated lung tissue, to the point at which they are heard. When once alive to the possibility of such occurrence, he is not likely to mistake their nature, or to err as to their true clinical significance. Sounds are rarely, if ever propagated from a distance, unless extensive alteration has occurred in the physical condition of the lung, in which case they are heard over an extended area, and, with ordinary care, may be traced back to their source.

Various sounds have been described, I believe erroneously, as emanating from the lung substance. Such, for instance, is the fine crepitant sound accompanying inspiration, which is heard sometimes at the base of the lungs, posteriorly, even in healthy persons, who have long been reclining on the back, when suddenly made to respire deeply—a sound which disappears after a few acts of respiration. This has been referred by Dr. Walshe* to the "forced unfolding of air cells which are unaffected by the calm breathing habitual to the individual." I am satisfied, however, that it does not emanate from the lung substance, but is true crepitation, due to the presence of a small quantity of secretion in the pulmonary vesicles. It is but natural to suppose that a small quantity of secretion should gradually accumulate in those portions of the lungs which are not brought into play during ordinary calm respiration, and in which, therefore, the air passages are not exposed to the drying influence of the atmosphere; and this supposition is quite consistent with the speedy cessation of the crepitus as soon as respiration commences in the part; whereas the notion that the mere unfolding of healthy lung tissue should give rise

* Loc. cit., p. 119.

to crepitation, is utterly at variance with all that is known, experimentally and practically, of the wonderful delicacy of the pulmonary apparatus, and of its smooth and noiseless action, the only perceptible sound having its origin in the friction encountered by the inspired air.

So, also, in regard to the creaking sound heard not unfrequently towards the apices of the lungs in phthisical patients during the act of inspiration; a sound which M. Fournet described as a variety of "pulmonary crumpling," and which Dr. Walshe attributes to "the unfolding of induration matter in the lung."* It is difficult to conceive the mechanism by which the presence of any solid or semi-solid matter in the lung tissue should give rise to the production of such a sound; and the frequent existence of every variety of deposit in the pulmonary tissue without its occurrence seems to negative the idea of its being so produced. Indeed, close and repeated observation has convinced me that the sound in question is a sound of vibration analogous to the creaking sometimes met with in the other parts of the chest during the existence of chronic bronchitis, and is due to a contracted condition of the bronchial tubes, and to the presence of viscid secretion in them; for, like the creaking sometimes heard in bronchitis, it is usually removable by cough and expectoration—a circumstance which serves to distinguish it from a very similar sound which sometimes results from local pleurisy.

So, again, in respect to the sound described by M. Fournet as resembling the crumpling of tissue-paper. This, I feel convinced, does not arise from the pulmonary tissue, as suggested by some authors, but is attributable to pleuritic friction. Certain it is that it is heard only at the apices of the lungs, where, in phthisical patients, pleuritic inflammation is very frequent, and is met with only during the early stage of phthisis, when it is probable that no adhesions exist, and whilst there still remains sufficient mobility of the apices of the lungs to produce the sound of friction. Further, it is persistent, and is not removable by coughing, as it would be if it were a variety of bronchial vibration. Such being the case, and as we know of no condition of the pulmonary tissue peculiar to the upper part of the lungs, where alone this sound is heard, it is fair to conclude that it does not emanate from "crumpling" of the lung tissue, as suggested by Fournet, but is referable to friction, resulting from congestion and local dryness and roughness of the pleural membrane, the peculiarity of its character being referable to its position in relation to surrounding parts.

* Loc. cit., p. 120.

Indeed, the only sound which in my opinion can be strictly referred to the lung substance is a peculiar variety of crepitation very rarely met with, and which hitherto, as far as I am aware, has never been recognised or described as distinct from ordinary crepitation. Its source, I believe to be, the presence of serosity in the areolar tissue of the lung; and its peculiarity, as contrasted with ordinary crepitation, is, that the crepitations are less numerous, and less regularly and less rapidly evolved, and that they are heard just as strikingly during the act of expiration as they are during the act of inspiration. The period of its occurrence and its duration correspond with the fine bubbling sound of capillary bronchitis—the sub-crepitant râle of many authors; and this fact, when viewed in connection with the minuteness of the crepitations, the peculiar dryness of their character, and the fact of their being unaccompanied by expectoration, serves to distinguish them from any sound known to arise from the presence of fluid in the air passages, and points to the existence of serosity in the lung tissue as the only satisfactory explanation of the phenomenon. With fluid in the areolar tissue surrounding each lobule, crepitation could not fail to be produced by every movement of the lung, whether in inspiration or expiration.

A somewhat similar sound, and referable to the same cause, viz., the movement of fluid through cellular tissue, is heard sometimes over the sternum, when the mediastinum is infiltrated with either air or serosity. The crepitations, just as in the former case, accompany expiration as well as inspiration, and may vary considerably in tone, dryness of character, and number. They may be heard during ordinary tranquil breathing, or may be audible only during forced respiration; they may be constant, or only of occasional occurrence; and may endure, for a considerable period, or may cease after a very few respirations. I have heard them strongly developed in several cases in which the mediastinal cellular tissue was infiltrated with serosity in connection with inflammation of the pericardium, and have also observed them in connection with the presence of air, as the result of accidental perforation of the chest. Their clinical importance arises from the possibility of their being mistaken, as I have known them to be, for the crepitations of pneumonia. It is strange, however, that such mistakes should be made; for the peculiarity of their character, the limitation of the area over which they are heard, and the period of

the respiratory act at which they occur, together form a safe criterion of their true origin.

The adventitious sounds which arise in the pleural cavity require a more extended notice. In a state of health the two layers of the pleural membrane being smooth, and slightly humid, glide upon each other evenly and noiselessly; but anything which roughens one or both of them, or interferes with their lubricity by drying their surface, increases the friction between them, and thus gives rise to the production of sound. Hence the sounds so produced, whatever their precise character, are commonly termed "pleuritic friction sounds." At first they are often soft and of a rustling or grazing character, and, as first suggested by Messrs. Barth and Roger, very closely resemble the sound produced when the back of one hand placed over the ear is rubbed lightly in one direction with a finger of the other hand. But as mischief advances, the sounds speedily lose this light, grazing character, and assume either a harsh, rubbing, grating, or crumpling, or else a distinctly creaking character. Very commonly they are more or less jerking or interrupted, as if the motion of the roughened surfaces over each other was momentarily suspended, and then suddenly recommenced. The precise character of the sound, however, affords no clue to the nature or extent of the pleural exudation; indeed, it appears doubtful, whether in many cases, at least, pleuritic friction sound may not be caused by mere dryness and increased vascularity of the pleural membrane. Moreover, the precise condition of the pleural membrane and the character of the exudation on its surface have far less to do with the intensity of the sound than the condition of the lung substance as to permeability, and the force of the respiratory efforts, inasmuch as free expansion of the lung is necessary to produce that friction between the two opposed surfaces of the pleura to which the sound is attributable.

The soft, grazing variety is that which occurs at the first onset of the disease; and is probably confined to the dry period of pleurisy, for its duration is very short, and as soon as dulness on percussion and other symptoms of effusion occur, it is replaced by the rubbing or creaking varieties. It is dry in character and superficial in situation, is heard only during inspiration, does not accompany each inspiratory act, is usually confined to a very limited area, and commonly passes away, or else is replaced within twenty-four hours by some other variety of pleuritic friction sound. When it arises in

connection with idiopathic pleurisy it soon disappears, and the patient is seldom seen until it is replaced by the rubbing, grating, or creaking variety. Indeed, so generally does this hold good, that in most instances when it is met with in auscultation, it indicates the circumscribed pleural inflammation which accompanies the irritation of tuberculous deposit, and often serves to establish the true character of the pains by which that inflammation is attended.

The rubbing, grating, crumpling, and creaking varieties, are the forms in which pleuritic friction sound most commonly presents itself: whichever its precise character, and whether loud, or whether faint and barely audible, it is always superficial in its seat, conveys the impression of dryness, and, though often confined to a limited area, is usually heard over a more extended surface than the grazing variety. Sometimes it is a continuous sound, the movement of the two surfaces of the pleura over each other being continuous and uninterrupted, but more commonly the motion of the pleura is more or less interrupted, and consequently the sound is made up of a series of jerks, rarely exceeding four or five in number, which in strongly marked cases occasion vibrations so distinct and forcible as to be perceptible to the patient, and to be felt even by the hand placed over the seat of their production. This is the case especially in regard to the rubbing variety. Sometimes it is confined to the act of inspiration, but generally accompanies both inspiration and expiration; in most instances it is audible during ordinary respiration, but occasionally is developed only after deep inspiration. Its duration in any one locality seldom exceeds a few days, inasmuch as the occurrence of effusion separates the two layers of membrane, and thus puts an end to the cause of the sound; but in some few cases where partial adhesions exist, keeping the two surfaces of the pleura more or less in apposition, and in the circumscribed pleurisies which often result from tubercular deposit at the apices of the lungs, I have known it continue for weeks, or even months.

Laennec asserted that pleuritic friction sound is a common accompaniment of interlobular emphysema, and one of its most characteristic physical signs; and although later and more extended observation has served to negative this statement if taken in its broad and general sense, it still remains the opinion of some, that emphysema may prove an occasional cause of it.* But I quite agree with Dr. Stokes, that it is only when the surfaces are rendered dry by an arrest of secretion,

* Thus, for instance, Dr. Walshe.—See loc. cit., p. 123-4.

or are roughened by the effusion of lymph, that their motions produce sounds perceptible to the ear. On several occasions, whilst auscultating lungs extensively emphysematous, I have detected sounds verygreatly resembling the friction sound of pleurisy; and, in some instances, the death of the patient has enabled me to determine, by *post-mortem* examination, that they did not proceed from the friction of an inflamed and lymph-covered membrane. But this is not an argument of any force in favour of the production of such sounds by the friction of subpleural emphysematous vesicles. In two only out of the instances just alluded to did any emphysematous projections exist at the spot over which the sounds had been heard during life; and it is quite certain that in *most* cases of emphysema, however strongly developed, no sounds are heard at all resembling the friction sound of pleurisy; whereas, if it were possible for irregularity or unevenness of the visceral pleura, resulting from emphysema, to occasion pleuritic friction sounds, these sounds ought to be present whenever such a lesion exists, provided only that there remains sufficient mobility of the lung to permit of a free gliding of the visceral on the costal pleura. But their existence under these circumstances is notoriously quite exceptional. Such being the case, and as sounds not to be distinguished by their mere character from the friction sound of pleurisy are sometimes heard during chronic bronchitis quite independently of emphysema, and equally so of pleuritic inflammation, I cannot doubt that the cause of the sound in the two cases is identical, viz., the vibration of viscid mucus in the air passages under certain conditions of bronchial constriction and surrounding pulmonary solidification.

Unfortunately this source of sounds which resemble friction sounds is often overlooked, or rather is not fully recognised. But I have seen too many examples of it, and have had too many opportunities of verifying the observation after death to entertain the slightest doubt on the subject. Unfortunately too, I have more than once seen the lamentable effects of erroneous treatment, founded on this mistaken diagnosis. I am anxious, therefore, to express my conviction that, with due care, the sounds alluded to ought never to be mistaken for pleuritic friction sounds. They rarely convey the impression of being very superficial in their situation, nor of being so dry as true pleural friction sounds; they are usually loudest during expiration, and are always of short duration, intermittent in their occurrence, and removable by long-continued coughing and expectoration.

Before quitting this part of our subject it may be well to advert to certain adventitious sounds originating in the parietes of the chest, which are apt to simulate and, in many instances, have been mistaken for morbid intra-thoracic sounds. Two of these, connected with the pressure of the stethoscope, I have alluded to in a previous chapter, namely, the pseudo crepitation produced when the instrument is placed over hair on the surface, and when, again, the subcutaneous cellular tissue is infiltrated with either air or fluid. But there are other sources of morbid sounds which have not as yet been mentioned. The crepitus of fractured ribs has been mistaken for pneumonic crepitation, and the creaking often heard over the dry costal cartilages of elderly persons, and occasionally also over the scapula, arising probably from some morbid condition of the cellular tissue between that bone and the chest walls, very closely resembles the creaking sound of pleural friction. The former may be recognised by its position, by its long persistence without extension of the area over which it is heard, and by its being unconnected with any of the other symptoms of pleuritic inflammation; whilst the latter may be got rid of without difficulty by a few brisk rotations of the arm.

The subjoined table exhibits at a glance the character of the adventitious sounds originating within the chest, the period of respiration at which they are usually developed, their mode of production, their ordinary seat, and the diseases of which they are commonly the index. Many, very many exceptions occur, but when the character of the sounds is strongly marked, the facts indicated by this table will generally hold good.

MORBID SOUNDS EVOLVED WITHIN THE

Class.	*Variety.*	*Synonym.*	*Character of the Sound.*
Sounds of Vibration	Low pitched or grave toned	Sonorous Rhonchi	Deep or graved toned sounds of snoring, cooing, buzzing, grunting, or creaking character, attended by fremitus, communicated more or less forcibly and extensively to the chest walls
	High pitched or Shrill	Sibilant Rhonchi	High pitched sounds of variable duration and intensity, and of irregular occurrence, of a whistling, piping, or hissing character
Sounds of Bubbling	Crepitating	Crepitant râle Crepitation, or dry crepitation	A dry crackling or crepitating sound, conveying an impression of the bursting in rapid succession, almost as in a volley, of minute bubbles of uniform size. It closely resembles the sound one hears when a lock of dry hair is rubbed transversely and slowly between the finger and thumb close to one's own ear
	Fine bubbling	Subcrepitant râle, or humid crepitation	A sound resembling the bursting of small bubbles of tolerably uniform size
	Coarse bubbling	Mucous, or sub-mucous râle	A coarse bubbling sound, conveying the impression of the bursting of somewhat large bubbles of unequal size
	Large bubbling	Cavernous râle, or gurgling	A hollow, gurgling sound, often of a metallic character, conveying the impression of very large bubbles bursting in a large space
	Dry Clicking	Dry, mucous râle, or dry clicking	A succession of three or four short crackling or clicking sounds, conveying an impression of dryness
	Moist Clicking		A succession of three or four clicking sounds less dry, and less distinctly crackling than the sounds of dry clicking

MORBID SOUNDS ASSOCIATED

Friction Sounds	Rustling or grazing Rubbing Creaking Grating Crumpling	Pleural friction sounds	A sound sometimes continuous, but often abrupt, jerking, or interrupted, conveying an impression of the friction of two surfaces. It may or may not be attended with perceptible fremitus, and may be of a rustling, grazing, rubbing, creaking grating, or crampling character.

LUNGS DURING THE ACT OF RESPIRATION.

Period of respiration at which it occurs.		*How produced.*	*Its usual seat.*	*Disease by which it is usually caused.*
Inspiration.	*Expiration.*			
——	——	Caused by the vibration excited by the passage of air through the larger bronchi, irregularly narrowed either by spasmodic contraction of their circular fibres, or by the adhesion of viscid mucus to their walls	Not peculiar to any portion of the lungs	Bronchitis, especially when in a subacute or chronic form, or associated with emphysema.
——	——	Engendered by the same cause as the sonorous rhonchi, but originating either in the smaller air passages, or in those of a larger size, which are temporarily contracted and reduced in calibre by reason of the disease	Not peculiar to any portion of the lungs.	Bronchitis.
——		Due to the passage of air through fluid in the minute air vesicles of the lungs	Base of one or both lungs	Pneumonia.
——	——	By the bubbling of air through fluid, more or less viscid, in the minute or capillary or bronchial tubes	Middle or base of one or both lungs	Capillary bronchitis or the resolution of pneumonia.
——	——	Caused by the bubbling of air through liquid in bronchial tubes of varying size	Middle portion of the lungs	Bronchitis after secretion has been thoroughly established.
——	——	Referable to the bursting of large bubbles in a space of considerable size; the gurgling and metallic quality of the sound being more pronounced the smoother and more tense and sound-reflecting the walls of the space in which the bubbling takes place	Apices of the lungs when due to—	A cavity the result of softened tubercle.
			or, in other parts when referable to—	Dilated bronchi, inflammatory abscess, or the presence of pus in the pleural cavity between which and the bronchi there exists a fistulous opening.
— — —		Caused by the forcible passage of air through a film of mucus, at a spot where the sides of a small bronchus have been brought together by external pressure, and agglutinated by tenacious secretion	Apices of the lungs	First stage of tubercular deposit.
— — —	— —	Produced in the same way as dry clicking, but associated with a more copious and less tenacious secretion.	Apices of the lungs	Commencing softening of tubercle.

WITH THE MOTIONS OF THE PLEURA.

The first variety is confined to inspiration; the others are sometimes confined to inspiration, but more commonly accompany both inspiration and expiration	By the rubbing together of the two opposed surfaces of the pleura rendered dry or roughened by inflammation.	Not peculiar to any portion of the chest	Pleurisy.

CHAPTER X.

ON SUCCUSSION, AND ON CERTAIN PECULIAR PHENOMENA CONNECTED WITH THE RESPIRATORY SOUNDS, THE BUBBLING RALES, AND THE RESONANCE OF THE VOICE AND COUGH—AMPHORIC RESONANCE; METALLIC TINKLING.

There still remains one other method by which we may sometimes obtain important information respecting the condition of the thoracic organs; I refer to succussion. So long ago as the time of Hippocrates, it was observed that if patients labouring under certain forms of thoracic disease are shaken by the shoulder whilst the ear of the observer is applied to their chests, a peculiar splashing sound may be heard on the affected side.* This sound, which closely resembles that obtained when an empty cask is shaken, and which is in fact produced by the splashing of fluid in a large, air-containing cavity, is known by the name of the "succussion sound." Its tone varies with the size of the cavity, and the condition of its walls, the density of the liquid it contains, and the relative proportions of air and liquid. The larger the relative quantity of air and the thinner the fluid, the more readily does splashing occur and the more easily, therefore, is this sound produced;† whilst the smoother and more tense the walls of the cavity in which the splashing takes place, and the thinner the fluid it contains, the more metallic the character of the sound. It may be produced by the least movement of the patient, by the act of coughing, or even by the action of the heart; and when not so producible, it may be readily excited by gently but suddenly pushing the patient backwards and forwards, or from side to side, so as to agitate the fluid. It is often perceptible to the patient himself, and may be audible by bystanders at a distance from the chest, but more commonly it is heard only when the ear of the observer is applied to the chest walls. Its duration is variable; sometimes it lasts for a few hours only, more commonly for several

* 'Hippocrates de Morbis,' lib. 11, sec. 45.

† Hippocrates observed that "those in whom much sound is heard have a smaller quantity of pus than those in whom the sound is less;" and although he does not appear to have understood the cause of this, his observation was perfectly correct.

days, but in some instances it has continued for many months, and has been known to persist even for years. In these chronic cases, which are very rare, it is perceived by the patient whenever he shifts his position suddenly, as in running, jumping, walking down stairs, or riding on horseback. It ceases immediately the pleural cavity becomes full of fluid, and when, on the other hand, all the fluid has been absorbed or evacuated, inasmuch, as already stated, it is never heard unless air and fluid coexist in the cavity.

From what has been already stated respecting the cause of this peculiar sound, it is obvious that it must have its origin in one of two conditions: either when an enormous excavation, partly filled with fluid, exists in the lung substance, or else when air and fluid coexist in the pleura. The latter is its most frequent, and as some assert its invariable cause, but as at St. George's Hospital I have met with it well marked on three occasions in which post-mortem investigation revealed an enormous tuberculous cavity but no pleuritic effusion, I am enabled to state positively that it may occur, though of necessity very rarely, in connection with excavations in the lung substance. The extent of surface over which it is heard depends in great measure upon the nature of the lesion by which it has been occasioned. If, as is commonly the case, it results from the presence of air and fluid in the pleural cavity, it will be heard over the whole of the affected side, unless indeed the lung has contracted adhesions to the thoracic parietes, in which case it will be audible at those parts only where air intervenes between the lung and the chest walls; but if it originates in a large pulmonary excavation, the surface over which it is heard will be proportionably limited in extent. I apprehend, however, that there need be little difficulty in distinguishing a succussion sound arising in a pulmonary excavation from one originating in the pleural cavity; for the limitation of the area over which it is heard is sufficient to arouse suspicion; and in pulmonary excavations the resonance rarely acquires a metallic quality at all equal in intensity to that which usually accompanies pleural succussion sound. Indeed, in the only three cases in which I have heard the sound in connection with pulmonary excavations, it had a very slightly metallic character, though it was strongly marked as a splashing sound.

Thus, then, it would appear that the forms of disease in connection with which a succussion sound may be heard are large pulmonary excavations with tense and indurated sound-reflecting walls, and pneumo-

thorax, whether arising from an ulcerated opening between the lung and the pleural cavity, or from an accidental perforation of the pleural membrane from without. Theoretically, it is possible for pneumonic abscess and other forms of pulmonary excavations to give rise to it, but I have never met with a case in point, nor can I find a record of such a case in the experience of others. Practically, therefore, phthisis pulmonalis may be regarded as almost invariably the cause of the succussion sound; for pneumothorax, when arising in the first-mentioned manner, is generally referable to tuberculous ulceration, and so also are the enormous pulmonary excavations from which the sound sometimes emanates.

Under much the same conditions as those which obtain when a succussion sound is audible, viz., during the existence of a large, air-containing cavity, certain other phenomena are sometimes observed which deserve special notice. I refer to the sounds known as amphoric echo* and metallic tinkling. Every person must be aware that on blowing, speaking, or coughing into a large empty jar with a narrow neck, the sound emitted by the observer is accompanied, or rather followed by a peculiar hollow sounding echo or reverberation, quite distinct, and sometimes even of a different pitch from the original sound. Very commonly this echo is of a humming or buzzing character, and hence was termed by Laennec, "bourdonnement amphorique," but sometimes it is high-pitched, and of an intensely metallic quality, its precise tone and character being dependent on the form, size, and reflecting power of the jar, and on the nature and strength of the vibrations by which the sound is caused. The same holds good in regard to the chest. Amphoric echo may be produced in the chest just as in the jar, but only under analogous conditions, viz., when a large air-containing cavity exists, with tense and good sound-reflecting walls. And just as in the jar, so in the chest; the more tense, and smooth, and sound-reflecting the walls, and the more distinct and forcible the original sonorous vibrations, the greater the probability of the echo being of a ringing, metallic character. The ringing quality which it ordinarily acquires under such circumstances

* This is sometimes styled "amphoric resonance," but the term is objectionable, inasmuch as "amphoric resonance" is a designation applied to a peculiar sound elicited on percussion; and if the same term is applied to the two phenomena, which have no relation to or connexion with each other, it is apt to lead to confusion. Amphoric echo is a term applicable only to the phenomenon we are discussing.

resembles that produced when an empty cask is stricken forcibly, or when a falling stone strikes the bottom of a deep well, but under certain conditions a peculiarly distinct clear tinkling sound is heard, which has been termed, not inaptly, metallic tinkling. Its distinctive characteristics, as contrasted with ordinary amphoric echo of a metallic quality, are, that it is clearer and of a higher pitch, of short duration, more purely metallic, and remarkably tinkling in its character, and recurs at distinct but irregular intervals. Laennec compared it to the sound excited by dropping grains of sand into a hollow metallic vessel; and a close imitation of it may be heard by applying the ear to a glass decanter, or a thin, hollow metallic vessel, containing a small quantity of fluid, whilst water is poured into it drop by drop.

Now, there can be no question as to the nature and cause of amphoric echo. It is merely the echo or reverberation, in a large cavity more or less adapted to reflect and concentrate sound, of certain noises excited either within the cavity itself or in its immediate vicinity by the respiration, by the râles or rhonchi, by the voice, or by coughing, by ordinary succussion, or by the impulse occasioned by the heart's action.* Its metallic quality is attributable to and varies with the form and size of the cavity, the sound-reflecting power of its walls, and the force and character of the original sonorous vibrations. The only questions which have been raised respecting it are, as to how small a cavity is capable of producing it, and as to whether a fistulous communication with the bronchi is essential to its existence. The size of the cavity has not been determined; indeed the peculiarity of the resonance appears to depend less upon the mere size of the cavity than upon the sound-reflecting power of its walls. But in regard to the question as to the necessity for the existence of a direct communication between the bronchial tubes and the cavity in which this sound occurs, much difference of opinion has been expressed. Laennec and his followers maintained that such a connection is indispensable, and even in the present day some persons profess to entertain the same opinion. But Skoda,† Barth and Rogers,† and other foreign pathologists, and most English authorities on auscultation, are opposed to this view, and I am

* I have observed this fact on four occasions; and a case in which the phenomenon was well marked, has been recorded by my late colleague, Dr. Bence Jones, in vol. viii, p. 61, of the 'Transactions of the Pathological Society.'

† Skoda, loc. cit., p. 134.

‡ 'Traité pratique d'Auscultation,' p. 213.

disposed to enrol myself amongst their number. Rarely is a case of pneumothorax met with in which amphoric echo is not producible, yet it seldom happens that the fistulous opening is not covered with effused lymph, or the access to it obstructed. And experiment leads us in the same direction; for as was pointed out by Skoda, if, whilst one "person speaks into a stethoscope placed on a stomach filled with air," another person listens through a stethoscope placed on its surface, he will hear amphoric echo sounding in the stomach, although of course no direct communication exists between the air within the cavity and that external to it. Therefore, although when this sound is heard in connection with pulmonary cavities, it is usually referable to vibrations transmitted to the cavity directly through an opening from the bronchial tubes, I do not hesitate to express the opinion that such a communication is not essential to its production; but that if the air in the cavity be separated from a moderate sized bronchus merely by a thin layer of pulmonary tissue, the sounds existing in that bronchus may pass into the air of the cavity with force sufficient to excite sonorous vibrations therein accompanied by amphoric resonance. The same holds good in respect to the cavity of the pleura; and in all cases of pneumothorax in which there is not a free communication with the bronchi, the transmission of sonorous vibrations through the walls of the cavity affords the only feasible explanation of the phenomenon.

The cause of metallic tinkling in the chest has been a subject of even greater dispute. Laennec attributed it to the reverberation produced by the falling of drops of fluid into liquid effused in the pleural cavity: Fournet, Dance, and others, supposing it to occur only when a fistulous opening exists between the pleural cavity and some bronchial tube, have referred it to the bubbling of air through liquid in the pleura, and most modern authors have connected it in one way or another with the bubbling of air through fluid, or the falling of drops of liquid into fluid in the pleura. On the other hand, Skoda* maintains that neither the presence of fluid nor the existence of a fistulous opening into the bronchi are at all essential to its existence; that it may be often heard in cases in which no opening into the bronchi can be detected after death, and therefore, that even if an opening did exist during life, it must have been far too small to transmit vibrations capable of producing metallic tinkling. He further appeals to the result of experiment, and states,† that "if a person speak into a stethoscope placed on a stomach filled with air, both metallic tinkling and amphoric echo may be heard sounding

* Loc. cit., p. 138. † Loc. cit., p. 134.

within the stomach, and this whether the stomach be partly filled with, or contain not a drop of fluid." In short, he refers this phenomenon to consonance of the voice, or of the respiratory sounds, or of abnormal sounds in the pleura or some other cavity, and asserts that it may be produced in pulmonary cavities just as in the pleura. My own conviction is, that metallic tinkling is a mere reverberation or echo of certain sounds reflected by the tense and indurated walls of a large hollow space within the chest; that it may occur without any direct communication with the bronchi; and that its peculiar character is referable chiefly to the nature and tension of the walls of the cavity and the character of the original sonorous vibrations, but in part also to circumstances tending to modify the concentration, quality, and pitch of the sound. Theoretically, therefore, it ought to be producible in pulmonary cavities just as is ordinary amphoric echo; but practically for reasons hereafter to be mentioned, its occurrence under such circumstances is extremely rare. Amongst the thousands of pulmonary excavations which I have examined, I have only met with it fully developed in a single instance; and in the case alluded to the cavity was of unusual size, considerably larger than a full-sized orange, adherent anteriorly to the parietes of the chest, bounded by smooth tense walls, surrounded on all other sides by consolidated lung tissue, and contained remarkably thin homogeneous pus.

I have already referred to Skoda's opinion, that tinkling may occur without the presence of fluid, and have detailed the experiment by which he endeavours to support that opinion. But any one may convince himself by repeating this experiment, that although an intensely metallic sounding resonance is developed, true metallic tinkling cannot be thus excited. Hence it would appear that the Viennese professor does not confine the term metallic tinkling to the sound described by Laennec as tinkling, but makes it comprise the metallic sound by which amphoric echo is usually characterised—a sound which derives its peculiarity simply from the hardness and tension of the walls within which the vibration occurs. If, then, this wide interpretation of the term is to be taken, Skoda's assertion is undoubtedly correct; but if Laennec's definition is to be followed, and a distinction is to be made between ordinary high pitched amphoric echo and true metallic tinkling—the sound as of grains of sand falling one by one into a thin metallic vessel—I am satisfied that he is in error, and that fluid is essential to the production of the phenomenon.

But experiment leads me to go further than this, and to assert that, with a view to the production of true metallic tinkling, it is necessary not only that fluid be present, but that it be thrown into active vibration. Dr. Walshe has shown that if a little water be placed in a glass decanter in which amphoric echo is occurring, no tinkling will be produced so long as the fluid remains at rest, anymore than if no fluid were present; but that when the fluid is set in motion, as it may be, by letting drops of water fall slowly on its surface, the echo which results is the purest metallic tinkling. Let the experiment be varied by substituting gruel or some other viscid fluid for the water, and it will be found that by gradually increasing the viscidity of the liquid, a point will be reached at which it will be difficult to say whether the sound emitted deserves the title of "tinkling," and that on rendering it even still more viscid, the tinkling sound will cease altogether. In short, it would appear, that the vibrations which give rise to true metallic tinkling must be very free, and cannot originate in very viscid fluid. Hence, doubtless, the rarity of the phenomenon in its purely developed form, even in cases of pneumothorax, and its almost entire absence in pulmonary cavities, even when metallic echo is well marked; the matter which is secreted being not sufficiently homogeneous or else too viscid to admit of forming the bubbles, or being otherwise thrown into the active vibration, which is necessary to the production of pure metallic tinkling.

So also it would appear that the vibrations which are productive of this peculiar sound must be excited by some cause of almost momentary duration, as if tinkling were the echo of a single note intensified and concentrated to the utmost by reflection from the walls of the cavity in which it is heard and liable to be interfered with by any vibrations set up concurrently with or shortly after it. The result of experiment seems decisive on this point. Thus, if in a decanter containing a little water a few drops of fluid be let fall slowly, drop by drop, on the surface of the water, metallic tinkling is the constant result; but pour water in a continuous stream into the decanter, and the pure tinkle is replaced by ordinary amphoric echo, oftentimes of a low pitched, buzzing character.* Thus, if care be taken in gradually increasing the rapidity with which the water is dropped, a good approximation may be arrived at in regard to the frequency with which loud vibrations may be excited without so far interfering with each other as to destroy the true tinkle.

* See Walshe, loc. cit., p. 158.

From what has been stated, it is obvious that in cavities in which no fluid exists amphoric echo alone can be heard, and that in cavities which contain both air and fluid, amphoric echo or metallic tinkling will occur, according as the original sonorous impulses are clear and separate, and do or do not produce free vibration of the fluid. The vibrations calculated to give rise to metallic tinkling may be produced by the falling of drops of fluid from the sides of the pleura into the liquid contained in the pleural cavity, or by the formation and bursting of bubbles by the entrance of air into the pleura. If the sonorous impulses thus produced are sharp and distinct, following each other slowly, so as not to interfere with and destroy each other, and if at the same time they give rise to active vibration of the fluid, metallic tinkling is the result; if, though sharp and distinct, and following each other slowly, they do not produce agitation of the fluid, amphoric echo of a metallic quality occurs, but not metallic tinkling; and if again they follow in quick succession, so as to interfere with or obstruct each other, the resulting echo is of lower pitch and only slightly metallic in quality, however sharp and forcible the original sonorous impulses, and however great the agitation of the fluid. Thus it often happens that metallic tinkling may be traced passing gradually into amphoric, buzzing echo, the conditions upon which the change depends being the gradual disappearance of the fluid, or an alteration in the character of the fluid or an increased rapidity in the occurrence of the sonorous impulses, the vibrations thereby excited becoming gradually less distinct, and of a lower pitch.

Thus, to sum up my opinion respecting the nature of amphoric echo and metallic tinkling, I would say that they are both essentially the same, viz., the echoes of sounds reverberating in a large cavity with tense, smooth, sound-reflecting walls; that they may both occur without any direct communication between the cavity and the bronchi, though the existence of such a communication is by no means unusual, and increases the probability of their occurrence; that the former—amphoric echo—whether low pitched or of an intensely high pitched and metallic quality, may be produced by the râles and rhonchi in the chest, by the voice, and by the act of coughing, and may be excited by such noises, whether they are set up in the cavity itself or in a bronchus or other contiguous cavity, and whether the cavity itself be filled with air alone, or contain both air and fluid; but that the latter—metallic tinkling—cannot occur in the chest without the presence of a homogeneous fluid which admits of free

vibration, and cannot be produced except by vibrations excited by an agency capable of imparting only a momentary impulse to that fluid.

In a practical point of view the facts stand thus:—If in a case of pneumothorax, with a fistulous communication between the pleura and the bronchi, the fistulous opening be free and above the level of the fluid, amphoric echo is usually heard alone. When with the increase of effusion the fluid rises above the level of the opening, or when again, from change of posture, the opening is brought temporarily below the level of the fluid, amphoric echo is produced if the air bubbles freely and rapidly through the liquid, but metallic tinkling if the bubbles escape slowly and intermittently, one by one. Thus, if the opening is large and free, amphoric echo is heard, whereas if it is small and partially covered or obstructed by false membrane or other secretion, metallic tinkling is the result. The acts of coughing, speaking, or moving, by producing agitation of the fluid, may give rise to tinkling, under the conditions above mentioned, even when there is no opening into the bronchi; and so possibly may the echo of râles and rhonchi in adjacent bronchial tubes, providing they have force sufficient to occasion such agitation of the fluid as is requisite for the production of metallic tinkling. Sometimes when tinkling is not producible, either by coughing, speaking, or forcible breathing, it may be distinctly heard following succussion of the patient, or as the result of a sudden change from the recumbent to an erect posture. Indeed this is perhaps the most certain method of producing it, and its cause in such cases is doubtless the fall of drops of fluid from the upper part of the cavity into the liquid below, or the bursting of air bubbles resulting from the agitation of the fluid or from the passage of air through the liquid in the chest, or through a thin film only of the liquid which has been brought by change of posture over the fistulous opening.

Thus, then, amphoric echo indicates the existence of a cavity in the chest, and its most frequent source is an air-distended pleura; but occasionally it is met with as a consequence of an enormous excavation in the lungs. Metallic tinkling very rarely occurs except in connection with an air-distended pleura containing some amount of fluid, which is commonly serous or sero-purulent rather than pure pus. Both the phenomena may, of course, arise as consequences of gangrene of the lungs, the bursting of a pneumonic abscess, or the giving way of an emphysematous air-vesicle; these occurrences, however, are so rare, that practically amphoric sounds, of whatever

nature, may be regarded as almost certain signs of far advanced tuberculous disease. They may be heard in every part of the chest, but usually are most audible about half way up posteriorly, or in the lateral regions. Amphoric echo may accompany both sounds of respiration, or may be limited to either, the limitation being generally to the inspiratory sound. Metallic tinkling may occur at any period of respiration; but when it is caused by the act of breathing it is usually, if not always, produced during inspiration, though it is sometimes prolonged into the succeeding expiration. It never occurs where amphoric echo is not producible, and generally alternates with or occurs occasionally during the continuance of that resonance. Oftentimes it is audible for a few days only; is rarely of long continuance, and seldom accompanies many successive respirations. Amphoric echo always indicates the existence of a cavity; metallic tinkling the presence of fluid in the cavity.*

* It has been objected to this supposition by Skoda, Davies, and others, that it is altogether opposed to physical laws, inasmuch as an opening which would admit air from a bronchus into the pleural cavity would also serve to let out the fluid from the pleura into the bronchus. But they appear to have overlooked an occurrence which often takes place, and which renders the explanation perfectly feasible, viz., that the opening is covered with a false membrane, which may serve as a valve to prevent the escape of fluid from the cavity, though it may not effectually bar the entrance to a few bubbles of air from the lungs.

PART II.

CHAPTER I.

In the present division of our subject I purpose describing the various diseases of the respiratory organs, and shall endeavour to point out the general symptoms by which they are each accompanied, the principal morbid changes induced by their actions, the physical signs to which they give rise, and the treatment best calculated to give relief. By so doing, I hope to enable the student to interpret the signification of different physical signs under the varying conditions of disease, and thus to utilize the knowledge he has acquired of the principles on which the diagnosis of chest disease is founded.

PLEURISY.

Pleurisy, or inflammation of the investing membrane of the lung, is one of the commonest, the most severe, and often one of the most striking of serous inflammations. Characterised from the first by chilliness and fever, acute catching pain on one side of the chest, oppression of the breathing, decubitus on the affected side, a short, dry cough, and a quick, hard pulse, it seems unlikely to be confounded with any other disorder. But its general features, though usually well marked, are by no means distinctive. They are one and all met with from time to time associated with other forms of thoracic disease, and the merest tyro in medicine must have observed how frequently one or more of them is absent, even in severe examples of the disease, and how those which are present are apt to mislead. Indeed there is no affection of the chest on which physical diagnosis throws more light than

on pleurisy, and none in which its aid is more needed by the practitioner.

It may be well to premise, as bearing upon the physical signs of the disorder, that the pleura is not only a serous membrane, but is also a shut sac, one portion of which is attached to the chest walls, whilst the other is reflected over and attached to the lungs, forming their serous envelope. Hence, on the one hand, the morbid products met with in pleurisy are, serum, lymph, blood, and pus—the usual products of serous inflammation; and, on the other, there arise phenomena resulting from the attrition of the two inflamed and roughened surfaces of the pleura, as also, it may be, from their complete or partial adhesion as a consequence of plastic exudation on their surface. Further, it may be stated that, just as with inflammation of other serous membranes, the general symptoms vary greatly under different conditions of health and constitution. In one case the catching pain in the side and other well-marked features of the complaint leave little room for doubt as to the nature of the malady. In another the pain in the side, dyspnœa and other characteristic symptoms may be absent, notwithstanding the rapid progress of mischief. These differences are doubtless attributable to causes the nature of which it is difficult to fathom; but, apart from innate constitutional peculiarities, there is reason to believe that age, sex, and the previous habits of life exercise a powerful modifying influence. Be this as it may, the fact must be constantly borne in mind whilst considering the following description of the disease.

For the sake of description, the course of acute pleurisy may be divided into three stages, characterised by well-marked anatomical differences, and corresponding to striking alterations in the physical signs and general symptoms of the disease. These are—1st. The dry stage, or stage of hyperæmia. 2nd. The stage of effusion. 3rd. The stage of absorption and resolution. Each of these varies in its general symptoms, and also, more or less, in the results which it produces, according to the age, strength, and constitutional peculiarities of the patient; but the structural changes and physical signs to which an attack of acute pleurisy *ordinarily* gives rise in a healthy person, will be seen by an inspection of the following table, in which they are collocated for the sake of comparison. The varieties of the complaint, as we meet with it in the weakly and unhealthy, and the complications which often arise during its progress, require separate consideration.

Morbid Anatomy.

First stage, or stage of hyperæmia.—The pleural membrane is much drier than natural; its normal transparency is lessened; its smoothness impaired, and its surface covered with a delicate network of finely injected blood vessels, or else studded with tufts of them in streaks or patches.

Second stage, or stage of effusion.—The sub-pleural cellular tissue is injected and œdematous; the pleural surface is extremely vascular, as in the dry stage, and the pleural sac contains lymph and serum in varying proportions. Sometimes the effusion consists of pale straw-coloured serum, with a few flakes only of albuminous

Physical Signs.

First stage, or stage of hyperæmia.—Inspection shows diminution in the respiratory movements, both of elevation and expansion, in the affected side, consequent on the pain which is felt on inspiration.

Mensuration gives similar results.

Palpation yields only negative results.

Percussion sound not materially altered.

Auscultation sometimes makes us aware of a slight, interrupted grazing friction sound on the affected side, referable to the dryness of the membrane and the congested state of the vessels; more commonly, it only tells us of a jerking unevenness in the rhythm of respiration, and of weakness or indistinctness of the respiratory murmur, consequent on the imperfect and irregular expansion of the lung. There is exaggerated respiratory murmur on the unaffected side.

Second stage, or stage of effusion.—Inspection detects increased motion on the unaffected side of the chest and great deficiency in the respiratory movements, especially of the expansion movement, on the affected side; after a time, perfect immobility of the chest walls is perceived, with general enlargement of the side, and flattening or

Morbid Anatomy.

matter floating in it; but more generally, in addition to this, a thick layer of lymph coats and roughens both surfaces of the pleura, either universally or in patches. If the congestion of the pleural membrane be excessive, a few blood globules may also escape, and the serum may be more or less distinctly blood-tinged. The quantity of fluid poured out may vary from a few drachms to many ounces, or even to several pints; and according to its amount, so is the effect produced on the lung. Whatever its quantity, compression of the lung tissue is the natural consequence. Gravitating as it does to the lowest portion of the chest, it usually gives rise, if small in amount, to partial compression of the lower lobe, which is pushed upwards, backwards, and inwards towards the root of the lung; but if it be more copious, it produces more extensive compression of the lung as it rises higher and higher in the chest, and, if no adhesions already exist, forces the entire lung backwards against the spinal column. As soon as the pleural cavity is thoroughly filled, and the lung compressed into the smallest compass, the continued effusion of fluid causes the sac to stretch, and to encroach in all directions on the surrounding

Physical Signs.

total obliteration of the intercostal spaces. Sometimes no general enlargement of the side is noted, but there is bulging of the inferior portion.

Palpation informs us of great diminution in the intensity of vocal fremitus if the amount of effusion be small, and of its absence if the effusion be copious; sometimes, though rarely, of a vibration due to friction of the roughened surfaces of the pleura; frequently, after a time, of displacement of the heart, and of the presence of fluid, as indicated by fluctuation in the intercostal spaces. The surface of the chest on the affected side is often œdematous, and is felt to be unnaturally smooth.

Mensuration proves the existence of deficiency in the movements of the chest, especially in that of expansion, and after a time of enlargement of the affected side, both in its circumference and in its antero-posterior diameter. The enlargement is most evident over the false ribs.

Percussion ascertains the existence of dulness, and an increased sense of resistance, most marked in the inferior portions of the chest, and generally terminating rather abruptly above. The line which marks the area of dulness is not

Morbid Anatomy.

structures. The parietes of the chest yield to the outward pressure, and enlargement of the entire side takes place, or bulging occurs at its inferior part; the diaphragm is thrust down, and a considerable prominence is often perceived in one or other of the hypochondria; the liver also is forced down; the mediastina are encroached upon, the heart is often displaced, and when effusion exists on the left side of the chest, is pushed over to the opposite side. After a time, if the effusion is persistent, the intercostal depressions are effaced, and, yielding to the outward pressure, the spaces may even bulge.

The lung, of course, is reduced in size, in proportion to the amount of pressure to which it is subjected. At first it is only partially emptied of air and is simply tougher and less crepitant than a healthy lung, but it yields by degrees to the pressure of the fluid, and the gradual contraction of the plastic exudation matter which coats it, and parting with all its aeriform contents lies flattened against the spine, a small, carnified mass, wrinkled perhaps on its surface, tough and leathery to the touch, and of density sufficient to sink instantly in water. In this case it is perfectly non-crepitant, but

Physical Signs,

altered by respiration, but sometimes may be made to shift by changing the patient's posture. Loud, hollow resonance, of a shallow character, is often met with in the upper part of the affected side, and sometimes, though rarely, a *quasi* cracked-pot sound may be detected there.* After a time, as effusion progresses, the entire side becomes dull on percussion, and the area of dulness extends far beyond the natural limits of the lung.

Auscultation, at an early stage, may discover pleural friction sound of a rubbing, creaking, or grating character, which ordinarily disappears, with the increase of effusion, but may possibly continue throughout the attack; total absence of the respiratory murmur, where the effusion is most abundant; weakness, or diminution of the sound, when the fluid is less copious; harshness or hollowness of breathing above the level of the effusion. In the interscapular region, at the root of the main bronchi, the respiration remains loud, hollow, and of a blowing character. The vocal resonance, in the earlier stages, is greatly increased, and in the posterior and lateral regions, especially at the root of the lung, it is often, but not invariably ægophonic. In the later stages, when the lung is much com-

* I have met with two or three marked instances of this.

Morbid Anatomy.

may be healthy in structure, and capable of re-expansion. On section it exhibits a smooth homogeneous surface, of a slaty, grey colour.

Third stage, or stage of absorption and resolution.—The seroplastic portion of the exudation is gradually absorbed, and ultimately the two surfaces of the pleura, covered with plastic material, come into apposition. Adhesion then takes place between them at one or more points, by means of the interposed lymph. Vessels are developed in this lymph, which rapidly becomes organized, and forms either a dense false membrane of fibro-cellular structure, or else mere fibro-cellular bands. In the one case the two surfaces of the pleura are firmly and universally agglutinated; in the other they are connected at a few points only.

If the effusion has been copious, and more especially if it has been of long duration, the lung is often so much compressed, and so firmly bound down by adhesions, that it is incapable of re-expansion, and becomes permanently impervious

Physical Signs.

pressed, and air ceases to enter, the respiratory sound and vocal resonance are altogether absent, and ægophony is never heard. If the heart is thrust out of its natural position, it will be heard pulsating to the right of the sternum, or possibly far beyond its normal bounds to the left. The respiration in the healthy lung is exaggerated, or compensatory.

Third stage, or stage of absorption and resolution.—*Inspection* informs us that the enlargement of the affected side is disappearing; that the intercostal spaces are regaining their normal condition, and that the mobility of the chest walls is returning.

Palpation gives us notice of returning vocal vibration and friction fremitus.

Mensuration, of a gradual return to the normal admeasurements of the chest.

Percussion gradually, though slowly yields a clearer sound, the dulness being most persistent in the inferior portions of the chest, where the compression of the lung, and the accumulation of solid plastic material is often so great, that the percussion note never regains its normal clearness.

Auscultation.—The respiratory sounds are again heard, at first weak and distant; then possibly harsh; subsequently of a normal

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to air. In this case, as the fluid is absorbed, the chest walls necessarily fall in under the effect of atmospheric pressure, and considerable distortion of the chest ensues. The affected side is diminished in every diameter, and its surface becomes irregular and uneven: in front it is often concave; the intercostal spaces decrease in width; the ribs undergo distortion, the lower ribs especially approximating and overlapping each other; the dorsal spine curves laterally, the convexity being sometimes towards the diseased side, but more generally towards the sound side, whilst a curve in the opposite direction occurs in the lumbar portion of the column; the intercostal muscles waste, in consequence of the entire loss of motion in the chest walls on the affected side; the diaphragm and subjacent viscera are often drawn upwards, and sometimes the heart is more or less displaced. These symptoms may occur when the exudation consists of lymph and serum, but they receive their most striking development in cases in which absorption fails to take place; and the fluid, whether seropurulent or purulent, is drawn off artificially, or escapes from the pleural sac spontaneously through a fistulous opening.

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character. Sometimes, as absorption progresses, and the two surfaces of the pleura come again into apposition, a friction sound reappears for a time, but ceases as soon as adhesion takes place. Occasionally pseudo râles are caused by serous infiltration of the subpleural cellular tissue. The vocal resonance, if at any time ægophonic, speedily loses this character, and becomes, at first, simply bronchophonic, and ultimately normal. The heart, if at first displaced by the effused fluid, regains its normal position, and is felt and heard pulsating under the left nipple. If, as in certain instances, the lung remains permanently impervious to air, there is entire loss of motion on the affected side; there is no return either of respiratory sounds or of vocal resonance, and the dulness on percussion is persistent. If, on the other hand, a portion of the lung—usually the upper portion—becomes partially pervious to air, percussion may elicit a hollow, but shallow resonance over it; whilst, in the same situation, the vocal resonance will be loud, and the respiration coarse and blowing.

These, then, are the morbid changes and physical signs which an uncomplicated attack of pleurisy ordinarily produces, whether in an acute or chronic form. A few remarks, however, must be added, before we pass on to the varieties of the disease, and the complications often met with during its course.

Reference has been made to the friction of the two inflamed and roughened surfaces of the pleura; and it might be inferred, from the description of the morbid appearances, that friction fremitus and friction sound must be felt and heard in most cases of pleurisy. This, however, is not so. Experience proves that friction sound and friction fremitus in pleurisy are exceptional phenomena, owing, probably, in the early stage of the complaint, to the rapidity with which effusion takes place, and to our failing to make our examination during the continuance of friction; and in the later stage—the stage of absorption—partly to the solidification the lung has undergone, whereby expansion of the chest and consequent friction of the two surfaces of the pleura is in great measure prevented, and partly also to the rapidity with which the thickly lymph-coated pleural surfaces become agglutinated. And as more motion of the lung takes place during forced, than during gentle breathing, it happens sometimes that, although friction sound is inaudible during ordinary breathing, it may be heard when the patient is directed to take a deep breath.

Again, it has been stated, that the line which marks the area of dulness may be *sometimes* made to shift its position by changing the patient's posture. Now, this is strictly and literally correct; but many authorities have asserted broadly, that, provided no adhesions exist between the two surfaces of the pleura, and that the quantity of the fluid in the pleural sac be not sufficient to compress the entire lung, the position of the dulness or resonance on percussion will vary according to the attitude in which the patient is placed; the lowest and most depending part—the part to which fluid gravitates—being always dull; and the part which, for the time being, happens to be uppermost, being always resonant on percussion. And the legitimate inference has been drawn from this statement, that when dulness is met with which is not attributable to circumscribed pleurisy, and which, nevertheless, does not shift its position with the varying attitude of the patient, it cannot be due to pleurisy, but must be referable to an interthoracic tumour, or a lung solidified by pneumonic, tuberculous, or other infiltration, which does not admit of change of position, but

necessarily occupies the same part of the chest. Nothing, however, can be more erroneous. True it is that percussion dulness referable to solidification of the lung, or to the presence of an intra-thoracic tumour, does not shift its position with the varying posture of the patient; and that when the seat of dulness is found to vary with change of posture, the dulness must be attributable to the presence of fluid. But it is not true, as the unreserved statement above referred to would lead students to believe, that when no adhesions exist to bind down the lung, or to circumscribe the effusion, the line of demarcation between dulness and normal resonance on percussion will always be found to shift its position with the varying posture of the patient. Case after case has come under my observation in St. George's Hospital, in which the contrary has been noted; indeed, in my experience, it seldom is observed to any considerable extent, except when the examination is made within a few hours after the effusion has occurred, and before the lung has suffered from compression. Even under these conditions, it is not always well marked; for when, as commonly happens, the products of inflammation consist not only of serum, but of solid albuminous and fibrinous matters in large quantity, the parietes of the chest, covered with the soft, pulpy, inelastic material, yield a dull sound on percussion, in whatever attitude the patient may be placed. All that can be fairly stated is, that the percussion note is not so dull as prior to the change in the posture of the patient; it rarely yields the good, clear resonance which is observed under similar circumstances in hydrothorax.

Another point of some importance relative to the dulness on percussion in pleurisy is the rapidity of its occurrence. From the moment that effusion commences the percussion sound in the lower part of the chest becomes comparatively dull; and as serous fluid is often poured out rapidly and early in the attack, pleuritic dulness may be clearly established within a few hours from the onset of the disease. I have known one side of the chest completely dull from pleuritic effusion within thirty-six hours after the first accession of pain in the side—a phenomenon rarely if ever witnessed in pneumonia, which produces hepatization of the lung slowly and gradually, and is, therefore, comparatively, a long time in occasioning extensive dulness on percussion.

The intensity and extent of the dulness are other points of considerable interest, as bearing on the diagnosis between pleurisy and pneumonia. The effusion of pleurisy may push aside the heart, en-

croach upon the mediastinum, and extend itself under the whole of the sternum, which, thereupon, yields a dull sound on percussion; whereas, a consolidated lung never passes beyond its natural boundary, and therefore does not give rise to dulness over more than one half of the sternum. Moreover, the dulness of pleuritic effusion is hardly to be confounded with the dulness produced by pneumonic or other solidification of the lung. It is far duller—a shorter, flatter sound, resembling the short dead tap which results from percussing the thigh; and the resistance offered to the percussing finger is beyond comparison greater than that which is met with in pulmonary consolidation from whatever cause arising.

Again, reference has been made to enlargement of the side as one of the later symptoms of pleuritic effusion. Hence, without further explanation, it might be inferred that the chest walls will only yield to long-continued outward pressure of the fluid, and that enlargement therefore, can only take place in chronic cases after effusion has existed for a considerable space of time. In most cases, undoubtedly, this inference would be correct; but instances are sometimes met with in which distinct enlargement of the side, as shown by admeasurement, occurs at a very early period of the attack. I have met with instances in which more or less local bulging has occurred in the lower part of the chest before the end of the third day of the attack; and cases are on record in which the chest has been considerably enlarged before the end of the fifth day. In my experience, however, this early enlargement has occurred only in childhood, when the chest walls are unusually yielding; and in those children only in whom the costal pleura has suffered severely, and in whom the sub-serous structures are softened and infiltrated with serum. It may possibly occur under similar circumstances in adults, but I have not chanced to meet with it.

So again, it has been stated that the intercostal spaces are apt to be effaced under the outward pressure of the fluid; and the statement, if unexplained, might lead to the conclusion that this is the common, if not the invariable result of pleuritic effusion. But it should be clearly understood that this is not the fact. Commonly, indeed, a filling up of the intercostal spaces is observed whenever effusion into the pleural sac is considerable in amount; but instances not unfrequently occur in which the side is considerably enlarged, and in which, nevertheless, there is neither bulging of the interspaces, nor very marked deficiency

in the action of the intercostal muscles during inspiration. The difference is explicable, I believe, by reference to the state of the costal pleura and the subjacent structures. When that portion of the pleural membrane is intensely inflamed, the subjacent structures become infiltrated with serum, and otherwise involved in the mischief; their contractile power is lost, and their resistance to the outward pressure of the fluid is greatly lessened. On the other hand, when the inflammation is less intense, or when the chief fury of the attack is directed on the pulmonary pleura, the intercostal muscles and adjacent structures are not paralysed, as in the former case, but continue in action, and offer great resistance to obliteration of the interspaces. The idea formerly entertained, that bulging occurs only when the pleura is distended with pus, has been clearly proved inconsistent with fact. It occurs more frequently under these circumstances than when the effusion consists of serum, but there is no necessary connection between bulging and empyema.

The opposite condition of the chest, or that produced by retraction or falling in of its walls, consequent on compression of the lung, absorption of the fluid, and contraction of the sero-plastic exudation, also requires further elucidation. The difference observed in the size of the two sides is referable not only to the falling in of the diseased side, which becomes narrower and smaller than the sound side, but to positive enlargement of the sound side, consequent on actual compensatory hypertrophy of the sound lung, on which all the work of the body devolves.

Again, it should be understood that retraction of the chest walls, and the distortion which results from it, are not in every instance persistent phenomena. The lung, as already stated, is compressed, but not necessarily diseased. Consequently, when absorption of the fluid takes place, its structure will admit of inflation; and if the plastic exudation which surrounds it and the adhesions which bind it down are not so firm as to render its expansion impossible, they may gradually yield to the inspiratory efforts, and the lung may again expand. In this case, the chest will again enlarge under the influence of the outward pressure which inspiration exerts, and the distortion of the chest will gradually pass off. Such instances are necessarily rare, and I have never met with one in which complete re-expansion of the side has taken place; but a remarkable case in point has been put on record by

Dr. Watson, which serves to establish the possibility of complete recovery.*

Thus far, then, we have discussed the phenomena usually attendant upon an uncomplicated attack of pleurisy occurring in previously healthy persons of vigorous constitution. In such persons the exudation matter is commonly very plastic, and of high vitality, readily becoming organized; so much so that anastomosing red lines have been found traversing the newly-formed false membranes, even when death has taken place after only a few days' illness. But, unfortunately, the disease is prone to arise in the old, the scrofulous, the intemperate, and the cachectic, in whom the effused lymph is almost necessarily of low vitality, and is found curdy, ill-concocted, and perfectly unorganized even after months of pleuritic suffering. Under these circumstances, the effused matters often tend to become puriform, and thus the case becomes seriously complicated. Either the chest remains permanently enlarged, or the purulent fluid which it contains seeks to escape from the cavity in which it is pent up, by perforating the pleura and discharging itself through the lungs, the thoracic walls, or the diaphragm. If, which rarely happens, the pus discharges itself through the diaphragm, peritonitis is the probable result; if it perforates the lung, symptoms of pneumonia will probably occur, and the moment of complete perforation will be marked by severe paroxyms of cough, resulting in a copious discharge of pus, or sero-purulent matter through the bronchi; but if it perforates the costal pleura, and finds an outlet through the thoracic walls, one or more soft, inelastic tumours, in which fluctuation is perceptible to the touch, will be perceived on some portion of the thoracic parietes before the skin gives way. In either of the last two cases, as soon as perforation occurs, pneumothorax will be added to the existing mischief. The physical signs of pneumothorax must be reserved for separate consideration.

Another point which requires notice in regard to empyema is, that when the pus makes its way externally, and perforates the costal pleura, the subcutaneous swellings to which its escape gives rise, may increase and decrease in size with expiration and inspiration, and if situated on the left side of the chest, may even pulsate synchronously with the heart. I have witnessed both these phenomena on two occasions, and instances have been put on record by other observers. They are of no practical

* See 'Watson's Lectures,' ed. 1, vol. ii, p. 126.

importance; but the latter acquires an interest which would not otherwise attach to it, from the fact that the student or careless practitioner might possibly mistake its real nature, and refer the pulsation to aneurism. The absence of aneurismal thrill and murmur, and of symptoms indicative of pressure on the trachea, the œsophagus, the larger veins, and the recurrent nerve, and further the presence of the physical signs and general symptoms of empyema, and not unfrequently the situation of the pulsation, will suffice to establish its true character.

Sometimes, again, when empyema exists on the left side of the chest, pulsation may be perceived in the infra-clavicular region, and over the arch of the aorta, even when no circumscribed tumour exists. I have noted this to a slight extent in several instances, but never to a degree at all likely to mislead even a student; therefore, I should not have considered it worthy of mention, had not Dr. Walshe referred to two cases in which, while the side was generally enlarged, gentle local bulging was manifest in the site of the pulsation, which was forcible enough "to jog the head at the end of the stethoscope," and in which, therefore, the question of aneurism might possibly have arisen. Such cases must be exceedingly rare; for, on inquiry amongst my friends, I cannot hear of any one who has met with an instance in point, nor can I find a record of any others than those above alluded to. Practically, too, they cannot be very important; for the presence of well-marked symptoms of empyema, backed by the feebleness of the heart's sounds over the seat of pulsation, by the absence of aneurismal thrill and murmur, by the equality in the radial pulse on the two sides, and by the absence of symptoms denoting pressure on the spine, the trachea, the œsophagus, the larger veins, and the recurrent nerve, ought at once to remove all doubt as to the real nature of the mischief.

Other modifications in the physical signs of pleurisy are occasioned by the attack being set up in persons whose lungs have previously contracted adhesions, which keep them forcibly in apposition with the costal pleura and the parietes of the chest. In these cases the physical signs of the disease are so much altered, that he who fails to bear the fact in mind, will be often misled in his diagnosis. Thus, when the lung is universally adherent to the posterior walls of the chest, it is almost impossible for any amount of effusion to stop the vocal resonance, or annihilate the respiratory sounds over the surface with which the lung is in contact, or to cause entire dulness on percussion. Even

the vocal fremitus will not cease until a large amount of fluid has been poured out. A striking case in point has recently occurred to me in St. George's Hospital. George Godfrey, aged twelve, was admitted under my care into the Hope Ward, suffering from empyema of the left side of sixteen days' duration. The whole side was considerably enlarged, and measured an inch and a half in circumference more than the sound side; it was perfectly dull on percussion anteriorly, but posteriorly did not yield such a thoroughly dull sound; and over the inner portion of the whole posterior surface of the chest diffused blowing respiration was audible, vocal fremitus could be distinguished, and vocal resonance was everywhere present, and of a hollow bronchophonic character. The pus was evacuated by an artificial opening, and enormous retraction and distortion of the chest, with curvature of the spine, ensued.

Adhesions give rise to another difficulty in the diagnosis of the disease. When the two surfaces of the pleura have become adherent at various parts by means of adventitious membrane, the effused fluid may be circumscribed, or, in other words, may not be contained in the general cavity of the pleura, but in a sac or sacs formed by the adventitious membrane. These sacs may be independent of, or may communicate with each other; but in either case, the fluid is confined within their boundaries, and does not shift its seat as fluid ordinarily does, with change of the patient's posture. Hence there may be perfect dulness on percussion over the seat of effusion, with entire absence of vocal fremitus, vocal resonance, and respiration; and there may be even local bulging; and these symptoms, though due to pleurisy, may remain unaffected by change of posture. Thus, these circumscribed collections of matter may simulate a solid tumour in the pleura or the lung, and may lead the careless and inexperienced to an erroneous diagnosis. But if due caution be observed, a mistake of this kind can seldom arise; for on the confines of the sac or sacs where the lung, compressed by the fluid, is adherent to the walls of the chest, there is usually found some dulness on percussion, and diffused hollow blowing respiration, with considerable increase of vocal resonance—conditions which are rarely met with in cases of intra-thoracic tumour, unless the adjacent lung be solidified by pneumonia, or infiltrated by tubercle or other matter, in either of which cases there would probably be other evidence as to the nature of the mischief. Moreover, in these cases of pleuritic effusion, there is entire absence of symptoms denoting

centripetal pressure—of pressure on the spine, the trachea, and larger bronchi, the œsophagus, the larger veins, and the recurrent nerve—and yet these symptoms are common attendants on intra-thoracic tumours.

Other modifications in the physical signs attendant upon pleurisy are occasioned by the occurrence of pleuro-pneumonia, or in other words by the coexistence of pleurisy and pneumonia. It might naturally be supposed that inflammation affecting the investing membrane of the lung, would be very likely to spread to the lung structure, and, conversely, that inflammation affecting the tissue of the lung would probably extend to its investing membrane. And observation in this instance is consistent with hypothesis; for it is found that the two inflammations frequently coexist, though the one is apt to predominate over the other. Pleurisy, indeed, is usually met with unassociated with pneumonia, and though inflammation of the lung may occur coincidently with pleurisy, it is rarely, very rarely, set up in sequel of that disease. But it is otherwise when pneumonia is the primary disorder; for acute inflammation of the parenchyma of the lung rarely runs its course without the supervention of pleurisy. Hence arise modifications in the physical signs of pleurisy, which vary, according to the condition of the lung at the time the pleuritic symptoms commence, and to the extent to which effusion had proceeded before the lung became implicated. Ordinarily, if the lung has undergone compression from pleuritic effusion before pneumonia commences, the supervention of that disease does not seriously modify the physical signs of pleurisy; but if, on the other hand, the lung is not solidified by pneumonic inflammation before the commencement of pleuritic effusion, it will resist to a great extent the compression of the fluid, and will continue to occupy the larger part of the pleural cavity, whilst at the same time the larger bronchi will remain pervious, and will be surrounded by a good sound-reflecting and sound-conducting structure. The result of this will be loud and widely diffused tubular breathing such as is never met with in simple pleurisy; a more than commonly intense and persistent vocal fremitus and vocal resonance; a more than ordinarily pronounced and persistent ægophony; and a very rapid extension of pleuritic dulness on percussion;—signs which will receive a development commensurate with the rapidity with which the two diseases run their course respectively.

It should be added, that acute pleurisy is often associated with, if it be not the cause of, circumscribed pericarditis and peritonitis, leading to limited plastic exudation; and that in the former case, especially,

the unwary student might mistake the rough sound of pericardial friction for the friction sound of pleurisy. The cardiac rhythm of the friction sound in the one case, and its respiratory rhythm in the other, ought, in most instances, to resolve any doubt on the subject.

Having now completed the investigation of the physical signs attendant on pleurisy, we will pass on to a consideration of the general symptoms, and then discuss the indications for treatment.

Acute pleurisy is ushered in by symptoms resembling those which mark the accession of pneumonia and pericarditis. Chilliness, sometimes amounting to actual rigors, followed by fever, with heat of skin, acute catching pain in the side, dyspnœa, a short, dry cough, a hard pulse, and a difficulty or impossibility of lying on the affected side, are the symptoms usually observed at the outset of pleurisy. But they are not all present in every instance, and some of them at least are by no means characteristic.

Rigors are the symptoms which are least constantly present, and possess perhaps least of a distinctive character. Seldom equal in severity to the rigors of pneumonia, they frequently amount to little more than mere chilliness; and in some severe attacks of pleurisy, even the sense of chilliness is altogether absent. They usually precede the "stitch in the side" by some hours or even days, but occasionally they are not observed until a later period of the attack, or after the characteristic pain has been developed. And as with the rigors, so with the fever which follows them. Sometimes, nay, generally, it is tolerably well marked, but it is rarely characterised by much intensity. Thus the skin, though hot, is often perspiring, and is seldom dry and burning, as in pneumonia.

The pain in the side, which is frequently called "a stitch in the side," is described as of a catching or stabbing character, and is undoubtedly the most striking and characteristic symptom of pleurisy. Varying in severity from the slightest "stitch" to a feeling of positive agony, it generally shows itself at the very outset of the attack, and though often fugitive at first, very soon becomes constant and localised.

Most commonly it is felt on a level with or immediately beneath one or other of the breasts, at a spot corresponding to the antero-lateral attachments of the diaphragm, but sometimes it is met with under the scapula, in the axilla, or beneath the clavicle; sometimes along the borders of the false ribs; sometimes even in the abdomen itself, especially in the right hypochondrium; whilst at others it fixes upon the

non-inflamed side, or extends over the whole of the inflamed side. This last, however, is an exceptional occurrence; for usually, no matter how extensive the area of inflammation, the pain is limited to a particular spot, and shows no disposition to shift its quarters. If it is moderate in degree, it is felt on deep inspiration, on coughing, or on sneezing, and is scarcely increased by pressure or percussion; but if it is acute, it is greatly augmented even by gentle intercostal pressure and by percussion, by lying on the affected side, by various movements of the body, and by cough; and is so greatly aggravated by inspiration, that the patient feels as if he were being stabbed each time that he attempts to inspire freely. Sometimes cases will run their course unaccompanied by pains from first to last; sometimes severe pain is persistent throughout the attack, or proves irregularly intermittent, but more commonly the pain, whatever its character, is found after a time to remit in severity, and ultimately to cease altogether.

The cause of pleuritic pain has long been a fertile topic of discussion and conjecture. Some persons have attributed it to the friction encountered by the inflamed and roughened surfaces of the pleura; and in support of their opinion, they have referred to the frequency of its occurrence at the lower portion of the chest, where thoracic motion is greatest, and where, consequently, pleuritic friction is most intense. But cases sometimes present themselves in which pleurisy runs through its various stages, accompanied by intense pleuritic friction, and in which, nevertheless, no pain is perceived from first to last. Sometimes, again, the most intense pain exists, and yet the physical signs prove beyond dispute, that the two surfaces of the pleura are separated by fluid, and that no attrition of the costal and pulmonary pleura can take place. I have met with several examples of both these phenomena. It is obvious, therefore, that some other explanation is needed; and having regard to the facts, that the pain is generally aggravated by pressure, that it is sometimes intermittent, and that it is not always felt at the seat of inflammation, it seems probable, that, in some instances at least, it is purely neuralgic, and in others a true intercostal myalgia, as suggested by Dr. Inman.

Hurried breathing, followed by dyspnœa, is, perhaps, the most constant, though not the most distinctive feature of acute pleurisy. At the outset of the disease the respiration is accelerated, in consequence of the pain which inspiration excites—pain which induces the patient to endeavour to compensate by the frequency of the breathing for its shallow

and imperfect character. But the breathing is not only short and hurried, it is jerking or irregularly interrupted; for the catching pain does not always occur at precisely the same period of each respiratory act, and consequently the thoracic movements are of unequal duration, and of a jerking or interrupted rhythm. Thus, for some time after the invasion of the disease, the breathing, though short and quick and jerking, hardly deserves the title of dyspnœa. Its increased frequency is due simply to the pain which a full inspiration excites, and not to any material encroachment on the breathing apparatus. But when effusion has occurred, the lung is encroached upon, and real dyspnœa sets in. If the pain persists, the respiratory movements will still continue jerking in rhythm, and will be more hurried even than before; whereas, if, as often happens, the pain subsides, or decreases greatly after effusion has taken place, the breathing, though still hurried, will be no longer jerking or interrupted. Sometimes the dyspnœa is most distressing to the patient, and continues so throughout the attack; sometimes it is severe at the outset of the complaint but soon diminishes in intensity; sometimes it amounts to little more than shortness of breath, which annoys, but does not oppress; and cases are not wanting in which, though the breathing is hurried, the patient makes no complaint of dyspnœa, and seems hardly conscious of its existence. Indeed, instances must have occurred to many practitioners, in which it has been difficult to persuade the patient that serious mischief existed in the chest. In some of these cases the other subjective symptoms are also absent, and then the disease is said to be latent. Of course the frequency of the respiration, when the pain has subsided is proportioned, *cæteris paribus*, to the amount and rapidity of the effusion, and to the condition of the opposite side of the chest. The larger the quantity of the fluid poured out, and the greater the rapidity of its outpouring, the more intense will be the dyspnœa. So again, if the other lung happens to be inflamed or otherwise diseased, the equilibrium between the quantity of air and the quantity of blood will be more seriously disturbed, and the dyspnœa will be greater than if such disease had not existed. Seldom, however, is the breathing very hurried, nor is the ratio naturally existing between the pulse and the breathing so such perverted as in pneumonia. In pleurisy the pulse rarely bears a smaller ratio to the breathing than 3 to 1, the respiratory being 40 when the pulse is 120 per minute, but it often happens in pneumonia that the respirations will number 60 or

64 when the pulse is only 100 or 108. Indeed, putting aside every other consideration, the amount of perversion in the ratio naturally subsisting between the pulse and the respiration will almost serve to distinguish pleurisy from pneumonia. Dr. Walshe asserts that the ratio naturally subsisting between the pulse and the breathing is more perverted in the "sitting than in the lying posture;" and, in proof of this, he cites a case, in which the ratio was as 3·39 to 1 in a recumbent posture and as 2·93 to 1 in an erect position. My own experience, however, does not accord with his in this respect; and I cannot help thinking that the result he obtained must be regarded as exceptional. Possibly it may have been due to the observation being made soon after the patient had assumed an erect posture, and before he had recovered from the disturbance of his respiration consequent on the change of position. Certain it is, that in eight cases which I examined specially with a view to this inquiry, I obtained a similar result when the examination was made soon after a change of posture; whereas in six out of the eight, a totally different result was obtained when the examination was deferred until after the patient had been kept in an upright posture for half an hour before the observations were made. In the six cases referred to, the ratio was less perverted in an erect than in a recumbent posture.

Cough is another symptom which usually accompanies acute pleurisy. Sometimes occurring as a prominent feature of the complaint, and sometimes altogether absent, even when pleuritic inflammation is intense and effusion into the pleura extensive, it is not to be depended on as a diagnostic sign. When it does occur, it is a short, half-stifled cough, dry, or accompanied only by a scanty mucous expectoration. The student, however, must not be misled by the existence of copious frothy or rusty-colored sputa. He must bear in mind that copious expectoration does not militate against the presence of pleurisy; it only shows, that if pleurisy exists, it is complicated by bronchitis or pneumonia, the signs of which will be discoverable on examination, in addition to the signs of pleurisy.

The position of the patient is another point of some importance in the diagnosis of pleurisy. Some persons have asserted that the patient lies on the affected side, some that he lies only on the sound side, whilst others have maintained that he may lie indifferently on either side, but commonly reclines on his back or in a diagonal posture between the back and the side, with his body inclined towards the affected side. The

truth appears to be that, at the outset of the complaint, the pain in the side, which is aggravated by pressure, prevents the patient lying on the affected side, and he therefore sits up in bed, or else lies on the sound side, or on his back with his body inclined to the sound side. After effusion has occurred, other influences come into operation, and lead him to alter his position. The fluid in the pleura would press upon and interfere with the action of the sound side, and so would cause dyspnœa if he continued to lie on that side, and as the pain usually ceases, or else diminishes in intensity as soon as effusion has taken place, he has no longer any reason for not lying on the affected side. He therefore assumes the posture which allows the sound side greatest freedom of action, and gives greatest relief to the dyspnœa. Thus he sits up in bed, or lies on the affected side, or reclines on his back with his head somewhat raised and his body inclined to the affected side. This, however, though generally the case, must not be regarded as an invariable rule. Instances are met with in which the patient is unable to lie down throughout the attack; others in which, from the very outset of the attack, he lies on the affected side, as the easiest position; and others again in which, though the pleura is full of fluid, he reclines indifferently on the back or on either side without augmenting his distress or increasing the difficulty of breathing. Examples in point are not very common, but they are met with often enough to prove that the decubitus in pleurisy, though sufficiently characteristic, is not to be depended on as a sign of the disease.

The general febrile symptoms attendant upon acute pleurisy do not present any characteristic features. The face is seldom flushed as in pneumonia, the skin is hot and moist, rarely dry and burning; the pulse is quick and often hard, contrasting forcibly with the soft pulse of pneumonia; the urine is high coloured, usually clear, of high specific gravity, and occasionally albuminous, as in other acute affections,* and there is seldom much delirium. The relative proportion of fibrin in the blood is augmented, as in other inflammatory disorders, but seldom to the same extent as in pneumonia.†

These, then, are the general symptoms of acute pleurisy. They may all be present in a marked degree, or one or more of them may be absent. In the former case it is difficult to mistake the nature of the complaint, even without reference to the physical signs; in the latter

* See a paper by Dr. Parkes in 'Med. Times and Gazette' for January 1st, 1859.

† See Simon's 'Chemistry.'

the aid of the physical signs is essential to a correct diagnosis. Nay more, cases are sometimes met with in which the pain and all the other subjective symptoms are wanting, and in which it is impossible without the assistance which physical diagnosis affords, to form even a conjecture as to the nature of the patient's malady. Several instances of this sort in which patients have been admitted with a chest full of fluid, and in which the existence of pleurisy had been entirely overlooked, have come before me in the wards of St. George's Hospital.

The course of pleurisy may be acute or chronic. Ordinarily, when pleuritic inflammation is set up it runs on rapidly to the production of copious exudation into the pleural cavity. But there is reason to believe that in some instances, at least, its progress may be at once arrested if vigorous and appropriate measures are adopted early. Thus, a few hours may witness the access of inflammation, the occurrence of plastic exudation on some portion of the pleura, and the entire subjugation of the disease, with adhesion of the two surfaces of the membrane. Several cases which have presented symptoms indicative of this train of events have occurred under my own observation, and some have been put on record by other practitioners. But these instances cannot be regarded as types of an ordinary attack of pleurisy. More generally, a considerable amount of fluid is effused, the lung is pressed upon and the physical signs and general symptoms already described as accompanying pleurisy are met with in a more or less fully developed form. In such cases, if appropriate treatment is adopted, the morbid action is gradually controlled, the fluid portions of the exudation are absorbed, the intercostal spaces cease to bulge, the two surfaces of the pleura which the fluid had separated again come into apposition, and become more or less generally adherent by means of the interposed lymph, the lung re-expands as the pressure of the fluid is removed, and, for all practical purposes, the chest regains its former condition. Neither spirometry nor external measurement can after a time detect any enlargement of the thorax, or any interference with the action of the lung resulting from the pleuritic seizure. And whilst these favourable changes are taking place in the physical condition of the chest, a corresponding improvement is observed in the condition of the patient. The feverish symptoms decrease and alternately disappear; the cough and dyspnœa gradually subside; the patient gets to lie indifferently on either side, he gains flesh, and his general health and strength improve.

But in other cases, although no untoward complications arise, recovery

is not so rapid or so complete. The feverish symptoms gradually subside, absorption of the fluid takes place, but the parts do not recover their former condition. The lung remains compressed by the adventitious membrane with which it has been coated during the progress of inflammation, or is bound down by firm adhesions. In either case it is incapable of expanding as the fluid is absorbed, and the necessary consequence is, that the other lung becomes hypertrophied and emphysematous, and pushes over towards the affected side of the chest, the diaphragm on that side is drawn up, the clavicle is depressed, the chest walls fall in, the thorax becomes shrunken and distorted, and the patient, deprived of the use of one lung, remains permanently short-breathed. If, as usually happens, the false membranes which form the obstacle to the expansion of the lung are thick and firm, the lung never can recover from the effect of the attack, and the distortion of the chest is irremediable. If the membranes are not so unyielding, the lung may gradually expand again, under the dilating influence of inspiration, and when it meets the collapsing chest, adhesion of the two surfaces of the pleura may take place. Sometimes, indeed, as proved by a case recorded by Dr. Watson,* complete recovery of the parts may thus gradually occur; but more commonly, the density of the adventitious membrane is sufficient to prevent the full inflation of the lung, and though the side may gradually enlarge, and the distortion disappear to a considerable extent, yet some evidence of former mischief will last for life.

In some instances, again, our utmost efforts fail to arrest effusion and produce absorption. The febrile symptoms become mitigated, but day by day the side enlarges, the intercostal spaces gradually become obliterated, or even bulge, the lung on the affected side is rendered impervious to air, and the fluid, encroaching upon the mediastinum, interferes with the action of the healthy side. In these chronic cases the patient becomes pale and cachectic in appearance; his lips are livid, his face is puffy, and the thoracic, and even the abdominal walls on the affected side become more or less œdematous. Yet, with all these evidences of obstructed circulation, there is no enlargement of the superficial veins of the chest, and none of the signs of centripetal pressure observed in cases of aneurismal or other intrathoracic tumours. The patient lies on the affected side or else on his back, with the head somewhat raised, and the body inclined towards the affected side; his

* Loc. cit., p. 106-7.

breath is short, but often not so as to cause distress; the voice is weak, and there is frequent cough, which is dry in cases of idiopathic pleurisy, but accompanied by muco-purulent or purulent expectoration if there be any coexisting affection of the lungs; the skin is dry, and usually rather hot, the pulse is frequent and often weak, and though the patient seldom experiences pain in the affected side, he is unable to sleep, suffers from hectic, and becomes gradually thinner and weaker. Indeed, his principal, if not his sole complaint, is of debility and shortness of breath.

Now, in these cases, if the patient is not relieved by having the chest walls punctured, and does not sink from exhaustion consequent on the oppression of the breathing, the effused matter after a time escapes by perforating the membrane which confines it. Sometimes the patient comes under notice before the process of perforation has commenced; and we are thus enabled to trace the establishment of a fistulous opening between the pleural sac and the external air. At others, one or more fistulous communications have been effected before the patient comes under observation. Most commonly the opening takes place through the costal pleura and the parietes of the chest; sometimes through the pulmonary pleura and the lung; more rarely, in both directions at the same time, or, in other words, through the chest walls and the lung; and, still more rarely, the matter perforates the diaphragm, and escapes into the cavity of the peritoneum. When the latter event occurs, peritonitis is set up, and runs on rapidly to a fatal issue; when the fluid forms a fistulous opening through the pulmonary pleura, there is usually some antecedent bronchitis and pneumonia, and, after a time, a sudden paroxysm of cough and dyspnœa occurs, and results in the expectoration of a large quantity—a pint or more—of purulent or sero-purulent matter, which threatens suffocation whilst it is being ejected, and leaves the patient very much exhausted. The cough and expectoration continue to a diminished extent for many days or even months; but if the case progresses favourably, they ultimately cease, the dyspnœa passes off, the patient regains his strength, and, when he recovers, has usually less distortion of his chest than when the matter has produced complete and permanent compression of the lung, and has found its way out through the parietes of the chest. Of course, as soon as perforation occurs, air is admitted into the pleural sac, and the side which continues enlarged and rounded, presents a variety of pathognomonic signs, which will be discussed under the head of Pneumothorax. Suffice it

at present to say, that if the case terminates favourably, the air is gradually absorbed, the two surfaces of the pleura come again into apposition, and the progress of the case towards recovery is marked by symptoms similar to those observed when no communication has taken place between the cavity of the pleura and the external air.

When the matter perforates the costal pleura and forces a passage through the parietes of the chest, one or more soft, inelastic tumours, in which fluctuation is perceptible to the touch, will be visible on the chest walls for some days before the skin gives way and an opening is established. Sometimes, however, patients may not come under our notice until one or more openings exist between the pleura and the external air. In either case, the student or unwary practitioner might be misled as to the real nature of the mischief; for neither the tumours nor the openings in which they result are necessarily situated in the most depending parts of the chest, but appear to form in almost any part of the thorax. Moreover, they may not communicate with the pleural sac directly, but by means of long tortuous sinuses. The discharge may be large or small in amount, according to the size of the opening, the freedom of its communication with the pleural sac, the quantity of effusion present, and the rapidity with which pus continues to be secreted; but it is often abundant, and, by the drain which it creates, gives rise to emaciation and exhaustion. Indeed, not unfrequently the patient sinks as the result of exhaustion so produced; but if he is able to bear the drain, the discharge gradually decreases, and, after a longer or shorter period—a few weeks or, possibly, some years—it ceases altogether, the opening in the parietes closes, the cough subsides, the dyspnœa passes off, the health and strength improve, and, in its ultimate progress, the case resembles those in which no communication has occurred between the pleura and the external air; the only difference being that, as the compression of the lung has been usually more complete, and the solid exudation matter larger in amount, the collapse of the chest walls and the consequent distortion is usually greater than when no fistulous communication has existed. In this instance, as in the last, pneumothorax is set up as soon as the opening is established. The consideration of the physical signs of this complication must be postponed until the subject of pneumothorax is discussed.

In what proportion of cases acute pleurisy passes on into a chronic stage has not yet been determined; and I believe that if the proportion were established, it would prove a very uncertain or fallacious guide in

respect to the prognosis of any particular case. Age, sex, habits of life, constitutional peculiarities, the plan of treatment adopted, and the period of the attack at which it was commenced, are all circumstances which exercise a remarkable influence over the course of this disease, and render deductions from any number of cases taken indiscriminately inapplicable to individual instances. Experience, however, justifies the statement, that chronic pleurisy bears a small numerical proportion to the attacks of the acute disease; that it is seen most commonly in males, and amongst the very young and the aged, in persons of a feeble or unhealthy constitution and intemperate habits, and in those cases in which appropriate treatment is not adopted, or is deferred until a late period of the attack.

The remarks just made in respect to chronic pleurisy hold good almost equally in respect to empyema.

Krause* refers to 137 cases, which he collected from various authors; and amongst these, 96 occurred in males, 23 in children, and 18 only in women. Dr. Walshe† records three cases only in females, and 19 in males; and in like manner, Heyfelder, who observed 20 cases of this disease, met with only one example of it in a female. Undoubtedly, acute pleurisy is more common in males than in females, but not to the extent indicated by empyema; and I believe that the extraordinary tendency which the disease exhibits, when occurring in men, to degenerate into empyema, is attributable principally to their intemperate habits of life, and to their neglecting to seek relief in the earlier stages of the complaint.

The causes of pleurisy undoubtedly exercise an important influence over its course, and a due appreciation of their relative bearings is essential to a correct prognosis and treatment. Exposure to cold, the irritation excited by tubercular and other deposits in the tissues, the extension of inflammation from the lung or the pericardium, and mechanical violence, such as blows on the chest or fracture of a rib, are the principal causes which are commonly supposed to excite an uncomplicated attack of pleurisy; whilst penetrating wounds of the chest, the laceration of the pulmonary pleura which is caused by the splintered ends of a fractured rib, and perforation of the pulmonary pleura resulting from inflammatory ulceration of the pleural membrane, or

* Krause 'Das Empyema,' p. 109.

† Walshe, loc. cit., p. 371.

‡ Heyfelder, 'Archives de Médecine,' 3ème série, tom. v, p. 59.

from the extension of a tuberculous excavation, are the most frequent of those which not only excite pleurisy, but lead to the admission of air into the pleural cavity, and give rise to pneumothorax.* Of the agency of the first-named cause, viz. exposure to cold, which is generally said to be the most common source of pleurisy, I feel bound to express my entire disbelief. Nothing, I think, admits of clearer proof, than that cold, however intense, and however applied, will not produce pleurisy in a healthy person; and that when exposure to cold is followed by inflammation of the pleura, the disease is due to some morbid condition of the blood, and not merely to cold. Possibly the patient might have escaped an attack if the exposure had not taken place, for his general health might have improved, and the blood might have regained its natural condition, without the supervention of pleuritic inflammation. But strictly speaking, cold is a mere accessory cause of the disease—a predisposing or exciting cause, but not the proximate or essential cause. Over-fatigue, anxiety of mind, undue excitement, or any of the thousand causes which tend to lower the vital energy and disturb the various functions of the body, might, equally with cold, have served to overcome the patient's power of resistance, and thus to excite an immediate attack. But popular feeling and casual observation combine to assign to cold the discredit of the seizure. Rigors precede or accompany the disease, and it is not surprising that the Public should mistake them for shivering, the result of exposure to cold. But the physician should be aware that something more is needed than mere exposure to cold, or to atmospheric vicissitudes, in order to induce inflammation of a membrane which is protected on all sides from the influence of cold.† The blood must be out of order, and probably contains some noxious, irritating matter, which acts directly on the pleural membrane, and excites inflammation. Experience has long since proved that a tuberculous, scrofulous, or cancerous diathesis, and the cachectic condition of the blood, which results from Bright's disease of the kidneys, intemperate habits, syphilis, pyæmia, gout, and rheumatism, are amongst the more active provocatives of pleurisy, and equally

* Suppurative disease in the abdominal viscera has been known to produce perforation of the diaphragm, and to excite pleurisy, and hydatids have given rise to the same result; but such cases are extremely rare, and only deserve notice as medical curiosities.

† For a detailed examination of the popular fallacy respecting cold, see the Introduction to the third edition of my work on "Rheumatism, Rheumatic Gout, and Sciatica," where the subject is fully discussed.

so of other serous inflammations,* and that we should at once suspect and search for one of these when called to a case of pleurisy in whch "cold" is the only apparent cause of the attack.

With regard to the other causes of the disease, it need only be observed, that those which are productive of uncomplicated pleurisy are less formidable in their consequences than those which lead to the admission of air into the pleural cavity. Pleurisy, when traceable to any of the first-named causes, excepting "cold," is usually limited in extent, easily controlled, and productive of adhesions, more or less partial, between the two layers of the pleura—an event which does not materially interfere with the function of respiration, and which, in the case of tubercular deposits, protects the patient against perforation of the pulmonary pleura, and the admission of air into the pleural cavity, with all the untoward symptoms consequent thereupon; whereas, in cases in which air finds its way into the pleural sac, and goes on accumulating there, not only is the lung very forcibly compressed, but the entire surface of the pleura is sure to be inflamed, and the products of inflammation assume a puriform character. As a consequence of these events, the immediate danger incurred is far greater, and the risk of permanent injury more probable, than in the former cases. Even under the most favourable circumstances, the lung usually fails to re-expand to its former size; and as the air and fluid are gradually got rid of, the chest walls fall in, distortion of the thorax, if not of spine, ensues, and the patient remains short-breathed for life.

Idiopathic pleurisy, occurring in a person whose lungs are not chronically diseased, seldom terminates fatally in its acute stage; so seldom that Dr. Walshe asserts that he has never lost a patient himself, and has never "known of an occurrence of the kind in the practice of others." I wish I could endorse this favorable report; but it happens that I have seen several instances of a fatal termination. Had the assertion been limited to cases in which appropriate remedies are had recourse to early in the attack, it would, I believe, have been strictly correct; but patients often neglect themselves at the outset of the disease, and do not send for a medical man until the disease has been committing its ravages for a week or ten days; and in this case death is not an unusual event. Day by day they get weaker and more exhausted,

* See an admirable paper, by Dr. Habershon, on the "Etiology of Peritonitis" in vol. xliii of the 'Med.-Chir. Trans.'

hectic sets in, the tongue becomes dry, the skin is covered with a clammy perspiration; and thus they gradually sink. During the year 1860—two such cases occurred at St. George's Hospital.

But death is not the ordinary result of acute pleurisy; indeed, recovery may be almost regarded as the rule. The course of the disease, however, differs remarkably in different cases. Sometimes the patient is seized with acute, catching pain in the side, and all the ordinary general symptoms of pleurisy, and loud but circumscribed friction sound is heard over the seat of pain. Under the effect of treatment these symptoms rapidly subside; no sign of other than circumscribed plastic exudation can be obtained, and in a few days the patient is quite well again, the two surfaces of the pleura having become adherent at the spot where the lymph was poured out. These attacks are designated attacks of "dry pleurisy;" they are often met with in cases of consumption, and give rise to the partial and often very limited adhesions which are found in the pleural cavity after death. But more frequently the disease goes on to effusion, and then, if recovery takes place, it occurs in one of two ways: either the fluid is gradually absorbed, or, absorption failing to occur, the effusion becomes chronic. In the former case recovery generally takes place, though the chest walls on the affected side often become retracted; in the latter the effusion gradually becomes purulent and a considerable proportion of the cases prove fatal, and when recovery takes place, the chest walls are almost invariably retracted, the precise degree of retraction being proportioned, as in the former case, to the extent to which the lung has been compressed by adventitious membrane, or bound down by adhesions, which prevent it from re-expanding and filling up the void created by the escape of the fluid. When, as sometimes happens, pneumothorax is added to the evils incident to pleurisy, the proportion of fatal cases is even larger than in ordinary chronic pleurisy; for when air exists in the pleura, without having been admitted through a fistulous opening caused by the escape of fluid from the pleural sac, it has usually found ingress through a wound in the pulmonary pleura, resulting from organic disease of the lung, which is a fearful though not a necessarily fatal complication of pleurisy. The precise proportion of these cases in which death speedily follows the admission of air it is difficult, if not impossible, to ascertain; that the proportion is very large is a matter of notoriety, and that temporary recovery does sometimes take place is quite susceptible of proof. I have met with five well-marked examples of it,

and one, in which the recovery was most remarkable, occurred under my care in St. George's Hospital no later than January 12, 1859.*

In practice it is often important to discriminate between pleuritic effusion and pneumonic consolidation; therefore, before passing on to the treatment of pleurisy, I will endeavour to place in juxta-position the principal points of distinction between these two disorders.

Pleuritic Effusion.

History and general symptoms.—Rigors seldom severe, and often absent. The attack commences with a sharp, catching pain in the side. The cough is dry, seldom frequent; sometimes there is little or no cough. There is seldom much burning heat of skin. The pulse is hard, and the ratio naturally subsisting between the pulse and the respiration, rarely falls below three to one. The patient lies on or inclined towards the affected side.

Inspection and Mensuration.—The side, which is almost motionless over the seat of effusion, is enlarged, rounded, and smooth; the ribs are widely separated, the intercostal spaces are more or less obliterated, and possibly the integuments are œdematous. Sometimes there is protrusion of the hypochondrium, and if the heart be displaced, as it often is, it may be seen pulsating out of its normal position.

Pneumonic Consolidation.

History and general symptoms.—Rigors, usually severe, are almost a constant precursor of the attack. The pain is not so acute as in pleurisy, nor does it catch the breath in a corresponding degree. The cough, which is severe and frequent, is accompanied by copious, and often rusty-coloured expectoration. The pulse may be full, but is frequently soft; rarely hard, as in pleurisy; and the ratio subsisting between the pulse and the respiration often falls to two to one. The patient lies indifferently on either side.

Inspection and Mensuration.—There is somewhat diminished mobility of the affected side; but its dimensions and appearance, the state of the intercostal spaces, and the position of the heart remain unaltered.

* See 'Hospital Case-Book,' xxxviii, p. 178.

Pleuritic Effusion.

Palpation.—Vocal fremitus absent, except at the root of the lung. Friction fremitus sometimes, though rarely perceptible.

Percussion.—Dulness always intense, often extending beyond the middle of the sternum; sometimes, but not invariably, altered in position by changing the posture of the patient. Under the clavicle percussion often yields a clear but shallow resonance; it shows displacement of the viscera by eliciting their characteristic percussion sounds.

Auscultation informs us that the respiratory sound and the vocal resonance are sometimes extremely weak and distant, more generally altogether absent, except at the root of the lung, where diffused hollow breathing and obscure bronchophonic or ægophonic resonance of voice may be heard. There is entire absence of fine crepitation. These signs, however, may be modified as already pointed out, by adhesion of the lung to the chest walls.

Pneumonic Consolidation.

Palpation.—Vocal fremitus increased, except when the feebleness or high pitch of the voice renders vocal fremitus imperceptible on either side.

Percussion.—Dulness rarely so intense as in pleurisy, never extending beyond the natural limits of the lung, and not altered in position by change in the patient's posture.

Auscultation reveals loud, hollow, tubular breathing, and intense bronchophonic resonance of the voice over that portion of the chest which corresponds to the consolidated lung, and fine crepitation over the adjacent portions, if inflammation is still extending.

In some instances the dulness on percussion occasioned by enlarged liver or spleen has been mistaken for the dulness of pleuritic effusion, but ordinary attention to the general symptoms and physical signs, and to the facts referred to in the first portion of this work, relative to the effect of full inspiration in increasing the area of vocal fremitus and clear resonance on percussion, will enable the careful practitioner to distinguish the dulness thus caused from that which is produced by

pleuritic effusion, and which, as already stated, is not affected by respiration. Dulness referable to simple hydrothorax, intrathoracic tumors, and tuberculous or other infiltration of the lung, will be discussed in connection with those several diseases.

With ordinary care it is impossible to confound the pain in the side and the short, interrupted breathing which result from pleurisy with the symptoms occasioned by pleurodynia, intercostal neuralgia, and other affections of the thoracic parietes. Nevertheless, such a mistake has been often made by practitioners who, in full reliance on general symptoms, have neglected to appeal to physical signs; and it may be advisable, therefore, to point out the characteristic marks of the several disorders.

The pain of pleurodynia, or rheumatism in the walls of the chest, though often undistinguishable in other respects from the pain of pleurisy, may be recognised by the fact that it is affected to a far greater degree than the pain of pleurisy by pressure on the ribs and by movement of the trunk and arm. If this be not sufficient to establish its true character, the absence of fever and of the physical signs of pleurisy, will usually serve to clear up the mystery. The only cases in which any doubt can be entertained are those in which the patient is seen soon after the accession of pain, and in which the pain co-exists with bronchitis, accompanied by febrile action. Even here the delay of a few hours will serve to set the question at rest, inasmuch as it will admit of pleuritic exudation, and the consequent production of percussion dulness, pleuritic friction sound, and ægophonic resonance of the voice.

The pain of intercostal neuralgia may be recognised by its paroxysmal character, and by its following the course of the affected nerve. Like pleurodynia, it simulates pleurisy, in so far that it gives rise to diminished freedom in the respiratory movements on the affected side, and to weak respiration, with jerking irregularity in its rhythm; but, like pleurodynia, it is distinguishable from pleurisy by the absence of percussion dulness, friction sound, ægophony, and other characteristic physical signs of that disease.

Having, then, determined that the patient is indeed suffering from pleurisy, what measures does it behove us to adopt for his relief?

If we see him in the stage of hyperæmia, or the first stage of the disease, the question of practising venesection may be fairly entertained, provided only that his strength be good, and that his symptoms be of a sthenic character. In many cases blood-letting is unnecessary, but in

some, in which the stitch in the side is severe and the pulse hard, it unquestionably proves extremely serviceable if it is not carried beyond what is required by the exigencies of the case. The points to be attained are, relief of pain, and moderation in the force and frequency of the pulse. The dangers to be avoided are, constitutional depression,—which so often leads to the production of suppurative instead of adhesive inflammation,—and anæmia, with its consequent protracted convalescence. The best method of making sure of the former without running undue risk of the latter is, to bleed the patient in an upright posture, and allow blood to flow in a full stream until he can take a deep breath freely, or else feels faint and exhausted. In an adult of ordinary vigour, the subject of acute pleurisy, the loss of from ten to twenty ounces of blood will usually suffice to produce one or other of these results, and a repetition of the bleeding will not be needed. If the pain continue, or the urgency of the symptoms be such that a further loss of blood is considered desirable, local depletion is preferable to venesection. Leeches should be applied to the painful side; and when they have done their duty, the leech-bites should be covered with a bread-and-water poultice, which effects the double object of fomenting the seat of pain and promoting the bleeding. Some persons recommend the employment of cupping in preference to leeches; but, on practical as well as on theoretical grounds I would strongly urge the application of leeches. The inflamed membrane lies so superficially that leeches are quite efficient in their action, and do their work without giving pain or uneasiness to the patient; whereas cupping over the painful part is often a severe infliction, and not only aggravates existing pain, but in some instances, I believe, aggravates the mischief itis intended to remove. Certain it is that on several occasions I have seen pain in the side increase after the use of the cupping glasses—an event which has never occurred after the application of leeches; and my firm conviction is, that leeches are as much preferable to cupping in the treatment of a superficial disease like pleurisy, as cupping is to leeches in the treatment of deep-seated mischief like pneumonia. If the attack of pleurisy be not very severe, leeches may be employed at the outset of the attack, to the exclusion of general venesection.

Following on, and in aid of blood letting, it is generally expedient to administer a brisk purgative; but when once the bowels have been freely relieved, nothing proves so serviceable as mercury combined with

opium. Tartar emetic, which is invaluable when the parenchyma of the lung is inflamed, has little or no control over the course of pleurisy; whereas mercury combined with opium exercises a markedly beneficial influence. It appears not only to check the effusion of inflammatory products, but to promote their reabsorption. The system, however, must be brought under its influence before these effects are fully developed, and therefore its administration should be commenced from the first, with a view to obtain its specific action as speedily as possible. The common practice is to give two or three grains of calomel every three hours, in combination with a sixth or a quarter of a grain of opium, and to continue its exhibition until the mouth is affected. But I believe that more immediate and more satisfactory results may be arrived at by applying mercury externally, and making larger use of opium internally. My usual practice is to give half-grain or grain doses of opium every three or four hours, in combination with one or two grains of calomel and half a grain of digitalis, and to have the whole side covered with a piece of linen spread with mercurial ointment. Over this is placed a poultice covered with oiled silk. In this way the action of the mercury is induced most rapidly, and apparently with far greater relief to the symptoms, and less subsequent distress to the patient than when the drug is taken wholly or principally by the mouth. Of course the patient must be carefully watched, and as soon as the slightest symptom of ptyalism is induced, the ointment must be omitted, and the internal administration of calomel suspended, or the quantity of the drug or the frequency of its repetition diminished; the object being to obtain the specific effect of mercury without aggravating the patient's sufferings by salivation.

But though the mercury be omitted, the use of opium and digitalis should be continued, and with them may be combined two grains of squills, which acts beneficially as a diuretic. If fever runs high, and the urine is loaded, an ammoniated saline draught, with the addition of a few grains of the carbonate and acetate of potash, is not only grateful to the patient, but assists in maintaining the action of the skin and kidneys, and if the tension of the system is great and the skin proves inactive, a small dose of tartar emetic may be advantageously added, either to the pills or the mixture. If pneumonia co-exists with pleurisy, the administration of tartar emetic will be urgently required. Meanwhile turpentine stupes should be applied to the chest; and when they can be borne no longer, the side

should be assiduously fomented by means of a bread-and-water poultice, or a piece of spongio-piline wetted with hot water and sprinkled with laudanum. Such appliances, I am satisfied, do something more than assuage pain and afford comfort to the patient; so much relief is often observed to follow their use that it is difficult to conceive that they can do otherwise than operate in arresting or modifying the course of the disease.

Some persons recommend the application of blisters to the seat of pain, and urge their employment not only as the most effectual method of producing counter-irritation, but as affording an absorbent surface to which mercurial ointment can be advantageously applied. Experience, however, induces me to agree with Dr. Watson and others in recommending that vesication be not employed during the active stage of the disease, or that, if employed, the blister be not placed over the seat of pain. The close proximity of a blister to any part where pain is most acute, and where intense inflammation is going on, is calculated to increase the local irritation; and in several instances which have come under my notice, the sudden disappearance of pleuritic friction coincidently with the occurrence of percussion dulness, has told, in terms not easily to be mistaken, the mischief which a blister so applied has occasioned. The same objection cannot be urged against blisters applied at a distance from the affected part; but even in this way, they afford little benefit during the more active stage of the disorder. They do not appear to check the morbid action, and the vesication which they produce may prevent their employment at a later period when their influence may be needed. Indeed, their curative action is not displayed until the pain in the side and the fever are subsiding, or, in other words, until the first activity of the disease is overpast, and the patient is suffering principally from the accumulation of fluid in the pleural sac. Then they prove our most valuable allies, and operate probably by disgorging the vessels and stimulating absorption. Certain it is that, when they are thus employed, any lingering pain yields rapidly to their influence, and that percussion and auscultation often mark the rapid disappearance of effusion.

Sometimes, however, effusion continues in spite of blisters and mercurials, or the treatment fails in producing absorption of the fluid already effused; in short, the case passes into a chronic stage. Under these circumstances, tartar emetic ointment, croton oil liniments, moxas, setons, issues, and even the actual cautery, have been had recourse to by

certain practitioners. But I cannot help regarding most of these applications as little less than barbarous engines of scientific torture. When blisters fail, the other agents are seldom of any service, and they are always productive of needless suffering. But much good may be effected by a different plan of treatment. Hitherto inflammatory fever and pain have prevailed, and the diet has been necessarily low; but the case is altered now that the symptoms of active inflammation have subsided. If the patient, at this stage, be kept too low, or be unduly depressed, it will not only be impossible to induce absorption of the fluid, but there will be great danger of its becoming sero-purulent in character. A more generous diet must therefore be given, and the general health sustained by quinine and other tonics. Meanwhile it is expedient to make full and steady trial of diuretics and absorbents. Even when a succession of blisters has failed in relieving the patient, iodurettted lotions, or iodurettted ointments,* backed by the internal administration of cinchona with tincture of iodine, iodide of potassium, and small doses of bichloride of mercury, nitre, acetate of potash, squills, digitalis, and cantharides, have sometimes effected the desired object.† The fluid has been gradually reabsorbed and recovery has ensued. Case after case has come under my observation in which this treatment, steadily pursued, has produced the most satisfactory results; and I am inclined to think that, in many instances, the unfavourable issue of pleurisy in its chronic stage is attributable to a want of tone in the system, caused by the treatment adopted. The patient is kept too low or is overmuch purged, or in some other way is unduly depressed, so that the system is unable to exercise its reparative power. In several instances, both in this country and in the Hôtel Dieu at Paris, I have

* Subjoined are formulæ I often employ:

℞ Hyd. Bichloridi, gr. iv;
Tr. Iodinii co., ʒiv—ʒvj;
Glycerini, ʒiij;
Aquæ destillatæ, ʒivss. Ft. Lotio.

℞ Hyd. Bichloridi, gr. iv—v.
Ung. Iodinii co., ʒiv—vj;
Adipis, ʒiv—ʒi. Ft. ung.

or,

℞ Hyd. Bichlor., gr. iv—v;
Potassii Iodidi, ʒij;
Aquæ destillatæ, qs. ut solventur,
Hyd. Bichlor. et Potassii Iod.
Adipis, ʒi. Ft. ung.

† A favourite diuretic is one contained in the Pharmacopœia of St. George's Hospital. It is a pill composed of digitalis, squills, and the pil. Hydrargyri.

seen patients progressing favourably until some lowering treatment has been had recourse to, and from that time the symptoms have put on an untoward aspect. Therefore, it is not without sufficient reason that I would urge the necessity of a generous diet and tonic medicines in aid of any diuretics and absorbents which may be employed in the chronic stage of pleurisy.

But if all ordinary means fail, and the pleura continues distended with fluid, so that the breathing is seriously oppressed, the general health undermined and life jeopardised, a question arises as to the propriety of relieving the patient by tapping, or, in other words, by puncturing the chest and letting out the fluid. The operation itself is simple enough, and neither difficult of performance nor formidable in its immediate results. Therefore, the sole question to be decided is as to the propriety of having recourse to its aid.

Some persons have advocated paracentesis of the chest even during the acute stage of the disorder, on the ground that if accumulation of fluid in the pleura is prevented, compression of the lung, and the various evils connected therewith, will also be obviated. But theoretical considerations are decidedly opposed to puncturing the chest, and admitting air to the inflamed pleural membrane; and practice justifies the doubts which theory suggests as to the expediency of so doing. On the one hand, it has shown that the admission of air into an inflamed serous cavity is apt to be followed by suppurative inflammation, even when the effused matters had previously consisted of mere lymph and serum; and that fatal results very commonly ensue under these circumstances;* and, on the other, it has proved that the mortality from idiopathic pleurisy is very small, and that, however extensive the mischief in the pleura, and however profuse the fluid effused, it may be generally got rid of by the influence of remedies as soon as active inflamation has been subdued. Indeed, it may be taken as thoroughly established that, during the acute stage of the idiopathic disorder, paracentesis thoracis is not curative in its action, and is almost certainly productive of mischief, and that it is only when pleurisy is associated with organic disease of the lungs, or with inflammation of the opposite lung, that we are justified in entertaining the question of its performance in the early stage of the disorder. In some such cases, where death has appeared imminent from suffocation, it has afforded temporary relief and has prolonged life; and, therefore, as a

* 'Sédillot de l'Empyeme,' p. 127.

last resource, it may be resorted to under the circumstances mentioned. But it must not be regarded as a curative agent—it is simply palliative—a mere expedient for giving temporary relief; and, even in this view, it often fails; for, although in a few instances, it has afforded the patient a respite, it has much more frequently, done little else than complicate his disorder, and lessen his chances of even imperfect recovery.

But the question assumes a very different aspect when the operation is proposed as a means of relieving a chest in which active inflammation no longer exists, but which, nevertheless, is distended with fluid which remedies have failed to get rid of. In many of these cases, the fluid in the pleura interferes with the vital functions, and jeopardises the patient's life; and we *must* interfere if life is to be prolonged. It matters not whether the disease commenced originally in an acute or in a chronic form; in other words, whether it has reached its present point rapidly or slowly; our duty is to obviate the tendency to death which the retention of the fluid in the chest occasions. The only questions which can arise are, as to the precise moment when the operation should be performed, and as to the mode of its performance, and the spot at which the opening should be made.

The first question is one on which a variety of views have been entertained, and on which, nevertheless, it is difficult to conceive how any difference of opinion can have existed. Some persons have advocated the performance of the operation even during the acute stage of the disorder; others have insisted on the propriety of postponing it until a very advanced period of the attack; some have been unwilling to recommend it under any circumstances; whilst others, fixing arbitrarily upon the fifteenth, or some other given day, of the disorder, have asserted that it may be undertaken on or after that day, but not until that day has arrived. Few arguments are required to refute each and all of these doctrines. The first is the only one which requires serious consideration, and that has been already disposed of. The objections to the others are obvious and unanswerable. A refusal to operate under any circumstances, could not be maintained in the present day in the face of the numerous recoveries which have taken place after all hope of amendment, except through the medium of the operation, has been, necessarily abandoned; and it needs no special experience of pleurisy, to understand, that the indications for the operation must be as marked in one case at the end of a fortnight, as they are in another after the

lapse of a month. And with regard to the proposition for postponing the operation until a very late period of the attack, observation has long since furnished materials for an opinion adverse to that practice. Worn out and exhausted by the long-continued irritation in his chest, and by the oppression of the breathing, resulting from the large accumulation of fluid in the pleura, the patient is ill able to bear the operation, or to withstand the drain which subsequently ensues; whilst the lung, bound down and compressed by false membranes which have gradually become thicker and firmer, is irretrievably damaged for all purposes of respiration, and is incapable of re-expanding when the fluid is withdrawn. The result, as might have been anticipated, is that the operation performed under these circumstances is commonly followed by fatal consequences, and that, even when recovery takes place, great and permanent distortion of the chest ensues.

The only practical tests as to when the operation should be had recourse to are the condition of the patient's health and respiration, and the absorption or non-absorption of the effused fluid. As long as the breathing is not seriously embarassed, and the general health does not decline, so long we are justified in making full trial of our remedies, in the hope that absorption of the fluid may be brought about. But, as soon as extreme shortness and distress of breathing, or lividity and anxiety of the countenance denote serious interference with the functions of life, delay is no longer justifiable; it becomes our duty at once to give our patient the chance which the operation affords. Even in the absence of any obvious oppression of the breathing, if day by day we note failure of the health and strength, and are not encouraged by the results of auscultation and percussion to believe that absorption is taking place, we are bound to hasten to his relief, and perform paracentesis. Sometimes, when neither deep oppression of the breathing nor rapidly failing strength proclaim the necessity for operative interference, the gradually increasing size of the chest, or the evidence of non-absorption of the fluid may warrant, or impeeraptively call for the operation; but, in this latter case, the indications as to the precise moment for its prformance are not so unequivocal as in the two former instances. Experience has proved, that by steady perseverance in a given course of treatment the fluid is sometimes absorbed after all hope of getting rid of it by natural means had been almost abandoned. Therefore, a proportional increase of caution should be observed in such cases before the operation is undertaken. Not only

should repeated blisters have been employed, and mercurial action cautiously induced, but tonics should have been administered perseveringly in combination with various diuretics and absorbents. And even, if after some weeks trial of the remedies, no evidence of absorption of the fluid can be obtained, the practitioner should still hesitate to perform the operation if the general health is not suffering, and the respiration is not materially oppressed. In this condition of affairs, he may feel doubtful as to the possibility of inducing absorption; but whatever doubts he may entertain, his right course is, not to have immediate recourse to the operation, but rather to make a tentative exploration of the chest by plunging a grooved needle into the affected side. If simple serum follows its introduction, the needle may be at once withdrawn, and the effect of remedies tried for a further period; for the operation should not be undertaken until it becomes apparent that it is impossible to get rid of the effused fluid by natural means; whereas, if the fluid appears to be pus, paracentesis should be proceeded with without delay. Every day which is suffered to pass after pus has been ascertained to exist in the pleura must necessarily expose the patient to risk by lessening his rallying powers, and increasing the impediments to the re-expansion of the lung.

I have hitherto made only slight allusion to the oft-mooted question as to whether the propriety of performing the operation can be affected by the character of the fluid to be removed. But, after what has been already insisted on, it is only necessary to repeat that the state of the patient's health and respiration, and the absorption or non-absorption of the effused fluid, are the only points which must be allowed to guide the physician's practice. The precise character of the fluid may affect his opinion as to the prognosis of the case, but it cannot militate against the propriety of the operation. If the fluid be pus, it is manifest that the operation should not be delayed, whatever the condition of the patient may be; and, if the fluid be only serum, the operation is not the less expedient and necessary if the functions of life are seriously interfered with. The utmost that can be said in such a case is, that if the general health be not declining, other remedies should be fairly and perseveringly tried before recourse is had to the operation.

The introduction of a grooved needle into the suspected side, which was originally suggested by the late Dr. Thomas Davies, is at all times desirable as preliminary to the operation of paracentesis. Not only does it serve to corroborate our diagnosis and enable us to

determine with certainty the existence of pleuritic effusion, but it also informs us of its character. If the fluid be serous, it will flow along the groove of the needle, and trickle down the patient's side; if it be puriform it will not flow so freely, and probably not a single drop will escape until the needle is withdrawn, and then a drop will make its appearance at the external orifice; or possibly none may be seen at the external orifice, but a small quantity may be visible in the groove of the needle. If no fluid escapes, and the groove of the needle is found not to contain pus, the practitioner will be made aware that no fluid is collected at the spot where he had imagined it to exist, and where, accordingly, he had introduced the needle. In this case he will have the satisfaction of feeling that the puncture has given little pain, and has done little or no harm to the patient, whilst it has been the means of preventing the introduction of the trochar or a lancet at a spot where its introduction might have been followed by dangerous consequences. But, although no evidence of effusion may have been obtained by the needle, the practitioner may feel convinced that fluid is contained in the pleural cavity, and that the existence of old adhesions of the pleura, or the presence of a thick coating of coagulable lymph at the spot where the needle was introduced has alone prevented his obtaining evidence of the fact. In this case, so harmless is the puncture of the needle that he may venture to make a tentative exploration at another part of the chest, and, if care be taken to employ a sharp needle, which shall penetrate the coating of lymph instead of driving it before it, he can hardly fail to succeed in his object. If then, on taking all necessary precautions, he fails in the second attempt to obtain proof of the presence of fluid, he will have reason to doubt the accuracy of his diagnosis, and may congratulate himself that by adopting the simple precaution of using the grooved needle, he has been saved from the dangerous error, to which his mistaken diagnosis would have led him, of tapping a chest in which no fluid existed.

Well, then, having satisfied himself, by careful examination, that the pleural cavity contains fluid, and that remedies are powerless to induce its absorption, and having had his views as to the existence of liquid corroborated by the introduction of a grooved needle, the practitioner, without further delay, should perform the operation of paracentesis. But how should he proceed in the matter? What instrument should be employed? Should any precautions be taken to prevent the entrance of air into the pleura? Where should the opening be made? Should

all the liquid be let out at once? Should the wound be kept open or healed up? These are questions which force themselves on his attention, and must be solved before the operation is attempted.

The choice of instrument must be regulated, in some measure, by the view which is entertained respecting the importance of preventing the entrance of air into the pleura. Some practitioners have laid great stress upon the non-admission of air as an element of success in puncture of the chest, and have suggested the use of canulæ, with valves and stop-cocks, as preventive of pneumothorax. Some of these instruments are very ingenious, and are calculated, as far as possible, to effect the object for which they were designed. But, in truth, they are found to be practically inoperative; for it is impossible to draw off any large quantity of liquid from the chest without admitting air into the pleura. The lung is seldom able to expand freely at once; and as the chest walls will not yield beyond a certain point, air must be allowed to find ingress into the pleural cavity, or the fluid would not flow out. I have seen two of the most perfect of these stop-cock instruments employed; and so long as precautions were taken to exclude air from the pleura, so long they failed to draw off more than a few ounces of the fluid; directly air was admitted, the liquid flowed through them freely. Therefore, however desirable it may be, theoretically, to exclude air from the pleura, it is practically impossible to do so if we wish to relieve our patient. Fortunately in this advanced stage of the complaint when *acute* inflammatory action has subsided, the admission of air is not a matter of so much importance as theoretical considerations have appeared to some persons to suggest. It does not necessarily excite suppurative inflammation of the pleura, This has been proved in many cases in which serous fluid has been drawn off by the canula without the subsequent occurrence of suppuration. Again, the existence of air in the pleura is not found to interfere with the re-expansion of the lung, in the cases now under discussion. The lung is already compressed by the fluid; and in most cases is slow in regaining its due expansion; and, as air is rapidly absorbed from the pleural cavity, its temporary admission in cases of paracentesis is, in this point of view, of little moment. I have known all traces of pneumothorax disappear within five days after the operation—a period within which it is improbable that the lung would have expanded fully even if no air had been admitted. Nevertheless, as the presence of air in the pleura

is an abnormal condition, and is certainly conducive to suppurative inflammation, every precaution should be taken to prevent its admission, when the grooved needle has shown that the effused liquid is serous in character. When, on the contrary, the grooved needle has proved the existence of pus, I believe that the admission of air is not of the slightest importance; and that, even if it were, the patient could not be relieved of the fluid without its admission. In the former instance, therefore, a small flat trochar should be used, and the opening should be closed as soon as the instrument is withdrawn; in the latter, a large trochar, or even a lancet or a bistoury, may be employed; and, unless some special circumstances contra-indicate such a course, the wound should be kept open, so as to admit of a constant discharge of matter. In the former class of cases, if the patient progresses favorably, no further operative interference will be necessary. But if rigors occur, or if much constitutional disturbance arises, the fair inference is, that suppuration has commenced; and an appeal should again be made to the decision of a grooved needle. If this shows the conclusion to be correct, a free opening should be made at once into the pleura, just as in those cases in which the needle from the first revealed the existence of pus.

The selection of the spot at which the opening is to be made is a matter of some importance, and demands the exercise of care and discrimination. Of course, if the fluid has already begun to point externally, and has occasioned the appearance of a soft, elastic tumour on the chest walls, there is no choice as to where the puncture is to be made, for nature herself has determined the question. The tumour must be opened, and a channel thus afforded for the egress of the fluid. But in most instances there is nothing of this sort to guide us, and our choice of a spot at which to introduce the trochar must be regulated by other considerations. The object to be attained is, to provide the easiest and most thorough vent for the liquid, without endangering the patient's safety. In some respects, the most dependent part of the antero-lateral portion of the chest is best fitted for the purpose; and the sixth intercostal space, a little in front of the digitations of the serratus magnus muscle is the spot often recommended. But, on making a puncture so low in the chest, some risk is incurred of perforating the diaphragm, the liver, the spleen, and other of the abdominal viscera, and thus causing the speedy death of the patient. This is no fanciful danger; it is a mischance which has occurred on more than

one occasion,* and which can hardly fail to occur if the diaphragm has been pushed upwards by enlargement of the abdominal organs. Prudence, therefore, suggests that the opening should not be made below the fifth interspace—the spot originally suggested by Laennec—and that if, on examination, the liver or the spleen appears to be enlarged, and therefore probably has forced the diaphragm high up in the chest, the puncture should be made in the fourth, instead of the fifth interspace. In a case of this sort, Laennec transfixed the diaphragm and perforated the liver by introducing the instrument in the fifth interspace; and it is well known that an enlarged liver or spleen will sometimes force itself up as high as the fifth rib. It is impossible, therefore, to take too much care in examining these points before deciding upon the spot for the operation; and as, when the fluid points spontaneously, the opening commonly occurs high up in the chest, there is reason to believe, as suggested by Dr. Stokes, that in selecting a high level at which to make the opening, we should be acting rightly for our patient's safety, and should not be compromising his chances of recovery.

Another necessary precaution is to determine, that the lung is not bound down by adhesions to the part at which it is proposed to introduce the trochar. The existence of any respiratory sound, or of vocal resonance, vocal fremitus, or resonance on percussion, is quite sufficient to prove that the lung is in close proximity with the chest walls at that particular spot, and should deter us from introducing an instrument there. So, also, in regard to the impulse of the heart, felt or seen at any spot. Further, it is important not to make the incision too near the margins of the ribs, lest the branches of the intercostal arteries be wounded. Again, considerable œdema of the integuments oftentimes exists, and some difficulty may occur in determining the precise spot at which to introduce the instrument. This may be overcome by exerting steady and continued pressure, which, by driving the fluid out of the cellular tissue beneath the fingers, discloses the ribs, and enables the operator to exercise his discretion in the matter.

When the opening has been made, and the instrument introduced into the pleural cavity, a question arises as to whether a portion only of the liquid should be drawn off, or whether the pleural sac should be emptied as far as possible. Some persons have maintained the

* See Watson, loc. cit., ed. i, vol. ii, p. 134.

propriety of the former mode of proceeding, and have adduced a variety of speculative reasons for the withdrawal of the liquid in successive portions; whilst others have advocated the latter method, and have supported their views by reference to practical results. In certain instances, success is known to have attended both modes of practice; but the weight of modern experience is greatly in favour of a full and free evacuation of the liquid, which affords the most complete and immediate relief. The only exceptions, perhaps, are cases in which the fluid has been ascertained to be serous, and those, again, in which the withdrawal of the fluid, whether serum or pus, occasions faintness. In the former, the removal of a small quantity of the liquid may relieve the tension of the vessels, enable the process of absorption to take place, and render further operative interference unnecessary; in the latter, the suspension of the operation is a matter of necessity, and not of choice, and the operation must be repeated as soon as the patient is in a condition to bear it. Some persons have proposed to make sure of emptying the chest by forcibly drawing off the fluid which accumulates below the level of the puncture. Laennec, for instance, suggested the use of a cupping-glass for the purpose; and syringes, syphons, and other instruments have been proposed by others; but common sense and practical observation are alike opposed to the practice. The lung, covered as it is with false membrane, cannot expand at once to fill up the space occupied by the fluid, and, therefore, if the liquid be withdrawn by artificial means, it must be replaced by air, or the chest walls must fall in. In either case, the patient would not be in a better position than he was before the withdrawal of the fluid; whilst the operation of removing it would have to be repeated day after day, as fresh pus is secreted, and would, necessarily, be attended with much fatigue and considerable risk to the patient. With the existing knowledge on the subject, I doubt whether any one would recommend the operation; my own feeling is strongly opposed to it.

A plan, however, has been recently proposed, which accomplishes much the same object by natural means, and seems to promise very favorable results. I allude to what has been termed the system of "drainage." With a view to carry out the plan, "a firm, long iron probe, somewhat bent," is introduced through the opening, made in the usual place, as above described, and "is then directed towards the lower and back part of the pleural cavity, the lower the better. If the end of the probe be made to press against the side of the thoracic walls, it can

be felt from the outside, through the intercostal spaces; though, perhaps, obscurely, owing to the thickness and toughness of the false membrane within. The lowest and most appropriate site in which the probe can be felt having been selected, an incision is made upon the end of the probe, which is then brought through the opening thus made. A strong piece of silk thread is passed into the eye of the probe, and drawn through the two openings, and the drainage tube—an India-rubber tube, perforated at frequent intervals, in the way recommended by Chassaignac for the healing of sinuses—being firmly tied to one end, is then drawn through by means of the silk; the ends of the tube are tied together, and the operation is complete." The benefits derivable from the adoption of this plan are said to be, that "the openings in the chest walls are always free; the matter is discharged drop by drop as it forms, so that, if the tube be suitably placed, there is never any collection of pus in the thorax; no time is given for decomposition, and the pus, therefore, is discharged in a healthy and pure state."* Now, it is obvious, from this description of the operation, that it involves a free admission of air into the pleural cavity, and therefore that it is not admissible in cases in which the effused fluid is of a serous character; but it is feasible enough, in cases of empyema, in which, as before stated, the admission of air is not productive of mischief. In some of these, as Dr. Goodfellow has shown, it may lead to the recovery of a patient in whom a single opening in the thoracic walls proves an inadequate channel for the escape of the pus. I have not as yet had an opportunity of trying it; but the objects it effects are so thoroughly in accordance with my own views as to what is desirable in cases of empyema, that I shall certainly have recourse to it when next I meet with a patient in whom operative interference is necessary.

Before quitting the subject of paracentesis, it may be well to refer somewhat more at length to the changes observed in the character of the secretion after the pleura has been punctured. Rarely does the newly secreted liquid retain a serous character, even though the pleura contained simple serum before the operation was performed. The usual tendency is to the effusion of pus, or sero-purulent fluid, and sometimes a few hours suffice to complete the change. Before the operation the effused liquid is devoid of smell, provided no air has found its way into the pleura; but after air has been admitted, the effused liquid

* See a paper by Dr. Goodfellow and Mr. De Morgan in vol. xlii 'Med.-Chir. Trans.'

is often converted into puriform matter of a low type, and emits a fetid odour. Whether the smell is attributable to the character of the pus secreted, or to decomposition resulting from its detention in the pleura, has not yet been ascertained, but I am inclined to regard the latter as the primary and potential cause of the change, and to believe that the decomposing fluid reacts on the diseased pleural membrane, and leads to its secreting pus, which is of still lower vitality, and more readily yields to the putrefactive process. But whatever the cause of the fetor, there cannot be a doubt that when it occurs immediate steps should be taken to get rid of it, and nothing answers better than injecting the pleura with warm water, containing a weak solution of permanganate of potash (ʒij of Condy's solution to Oj). At the same time the opening through the chest walls should be enlarged, or another made in a more depending position, so as to afford a freer exit to the liquid, and prevent its accumulation in the pleura. The system of drainage by Chassaignac's tube, properly carried out, would probably accomplish this object.

The prognosis, in cases where the operation is had recourse to, is a subject involved in much uncertainty. Unfortunately there are not any data from which to obtain a general estimate as to the value of paracentesis, nor are there any trustworthy grounds for an opinion as to its issue in any particular instance. The result, however, bears some relation to the quantity and quality of the effused liquid, and to the length of time the operation is deferred. The larger the quantity of the fluid, and the longer the time for which the operation is postponed, the greater, *cæteris paribus*, is the displacement of the thoracic viscera, the greater the injury they suffer from the pressure to which they are subjected, and the greater the amount of constitutional distress. As a consequence of this, the thoracic viscera return less readily to their normal condition, and the patient has less chance of rallying. Nevertheless, patients have often died when the effusion has been somewhat scanty, and others have recovered when it has been very abundant. In one of the cases treated by the system of drainage, no less than "eight quarts had escaped in twenty-four hours," and yet the patient recovered without a drawback.

The same uncertainty attaches to the character of the effused liquid as a foundation for our prognosis. In most instances, undoubtedly, mere serum in the pleura is less formidable than a sero-purulent or puriform fluid; but in many cases the liquid, which at first was serous,

has subsequently become puriform, and the case has terminated fatally. On the other hand, cases in which the secretion has been purulent, even before the chest was punctured, have gone on steadily to recovery. I have met with several examples in point, and many have been put on record by other observers. I know of no statistics showing the ratio which the cases of recovery bear in the two sets of cases respectively; but my own experience leads me to believe that the proportion is much smaller in the latter than it is in the former, in which the effused liquid is serum.

In truth, the general condition of the patient is a much more trustworthy ground for an opinion as to the issue of the operation than are the quantity and quality of the effused liquid. This, therefore, is a point to which attention should be directed, and the bearing of which should be carefully weighed, before the operation is undertaken. It is manifest that effusion is less likely to prove fatal when it results from simple pleurisy than when it is connected with a carious rib; it is less likely to have an unfavorable issue when the lungs are free from organic mischief, than when they are tuberculous, or otherwise diseased; and it will less probably run an untoward course when the patient is constitutionally sound, than when his kidneys or other organs are organically deranged, or when his health and strength are shattered by habits of intemperance. If the patient be young and vigorous, of sober habits and sound constitution, and if his digestive organs perform their functions properly, there will be far less risk of a fatal result than in persons advanced in years, or of an opposite condition of system. Indeed, judging from what I have observed in the cases which have come under my own observation, I am inclined to believe, that although the operation terminates fatally in a large proportion of those who have led an intemperate life, or are organically unsound, yet that it issues favorably in a large proportion of vigorous and temperate persons, provided only that it be not delayed too long, and that a free opening be made for the escape of the liquid.

In cases of double empyema, the prognosis, of course, is extremely unfavorable, and it is hardly right to have recourse to the operation.

Pneumothorax and Hydropneumothorax.

It has been already stated that air or gas is sometimes found in the inflamed pleural cavity, and that its presence gives rise to pathological effects and physical signs which require careful consideration. But before entering into these details it may be advisable to define what is meant by the terms pneumothorax and hydropneumothorax, and to point out the various circumstances under which these affections may arise.

The term pneumothorax is used to denote a collection of gas or atmospheric air in the cavity of the pleura; and the term hydropneumothorax, if strictly applied, signifies the coexistence of air and fluid in the same cavity. But idiopathic pneumothorax rarely exists, and even when it does, is usually accompanied, in a few hours, by the effusion of fluid in the pleura; so that, practically, the terms pneumothorax and hydropneumothorax may be regarded as almost convertible. The various causes which may lead to the presence of air in the pleural cavity will be seen on inspection of the following table, in which they are classified under four separate heads:

1st. *When no communication exists between the pleura and the external air:*

α. Spontaneous evolution of gas from the decomposition of fluid in the pleura, or from gangrene of the pleural membrane.

β. The secretion or exhalation of air by the pleura. (?)

2ndly. *When a communication takes place between the pleura and the alimentary canal:*

Resulting from inflammatory softening and perforation of the œsophagus or stomach.

3rdly. *When a communication occurs between the pleura and the atmosphere through an opening in the chest wall:*

The result of penetrating wounds of the thorax, or of parietal abscess.

4thly. *When a communication occurs between the pleura and the bronchi:*

α. The result of violence—rupture of the lung substance, and tearing of the pulmonary pleura.

β. The result of disease. Perforation of the pulmonary pleura from without inwards, as by empyema; or from within out-

wards, as by ulceration excited by hydatids, or by tubercular, cancerous, pneumonic, gangrenous, metastatic and bronchial gland abscess, or by rupture of the pulmonary pleura in emphysema, and pulmonary apoplexy.

The first and second sets of cases are of extreme rarity, and the third belongs exclusively to the province of the surgeon; so that, practically, the fourth class comprises the causes to which our attention must be specially directed. Even these vary greatly in the frequency of their operation. Thus tubercular ulceration is the efficient cause of perforation of the pulmonary pleura in a vast majority of cases. Dr. Walshe has stated 90 per cent. as the proportion of cases in which, if traumatic cases are excluded, pneumothorax is referable to tuberculous ulceration, and my own experience leads me to believe that this is a close approximation to the truth. For although of 147 cases collected by M. Saussier, 81 only are reported to have occurred in phthisical patients, it is certain that these numbers do not furnish trustworthy data for an opinion as to the causation of pneumothorax. "Tuberculous perforation is an every-day affair, which passes unnoticed" and unrecorded; whereas "perforation from gangrene, vesicular emphysema, hydatids, pulmonary apoplexy, abscess, and other rare causes, are greedily caught hold of and registered." Hence it naturally follows that when cases are collected from different sources, the result, as in M. Saussier's cases, does not show a sufficiently large proportion of tubercular disease; and in the absence of any trustworthy statistics bearing on the subject, I am constrained to refer to my own observation and to our combined experience at St. George's Hospital as evidence on the question at issue. My own experience relates to sixteen cases, in thirteen of which tuberculous ulceration was the cause of perforation, in one pneumonic abscess, and in the remaining two empyema, which emptied itself through the lung.* The *post-mortem* records of St. George's Hospital for the ten years ending December 31st, 1850, tell of twenty-three cases, in twenty-one of which tuberculous ulceration, was the cause of perforation, and in two pneumonic abscess.†

Thus, then, it is apparent that if traumatic cases are excluded, the

* These 16 cases do not include any of the instances which proved fatal in St. George's Hospital during the decennial period ending December 31, 1850.

† See St. George's Hospital Post-mortem and Case books; also 'Decennium Pathologicum,' cap. v, sec. v; by Dr. T. K. Chambers.

presence of pneumothorax may be regarded as presumptive evidence of tuberculous ulceration of the pleura, and it may be satisfactory to inquire as to the usual seat of the perforation, the age and sex of the sufferers, and the period of the phthisical disorder at which this complication is prone to arise.

Of the 13 phthisical patients to whom my notes refer 8 were males and 5 females. Of the males 1 was from fifteen to twenty years of age, 5 were from twenty to thirty-five years of age, 1 was from thirty-five to fifty years of age, and 1 was fifty-four years of age. Of the 5 females 1 was only eighteen years of age, 3 were from twenty to thirty-five years of age, and 1 was forty-two years of age.

In every instance the patient was in an advanced stage of consumption. This, however, is not a necessary condition; for Louis has recorded a case in which perforation took place within a fortnight after the phthisical symptoms first declared themselves, and experience has fully demonstrated the fact that it may occur at any period of the disease. Nevertheless, its occurrence at other than an advanced stage of the disease is quite exceptional.

In 7 of my 13 cases the perforation took place on the right side, and in 6 on the left; but in 5 of the cases I am unable to state positively at what spot the perforation occurred, inasmuch as 2 of the patients passed from my observation before a fatal termination ensued; in 2 of the others there was no *post-mortem* examination, and in 1 case the firmness of the pleural adhesions, and the consequent tearing of the lung on removal, rendered it impossible to ascertain precisely the seat of perforation. In the remaining 8 cases the pleura gave way, as below stated, viz.:

On the right side—In the middle and outer part of the upper lobe, in 1 case.
„ In the lower and posterior part of the upper lobe, in 2 cases.
„ In the outer part of the middle lobe, in 2 cases.
On the left side—In the middle and posterior part of the upper lobe, in 2 cases.
„ In the upper and posterior part of the lower lobe, in 1 case.

Of the 21 fatal cases which occurred in St. George's Hospital during the decennial period embraced in Dr. T. K. Chambers' 'Decennium

Pathologicum,' "10 were males, and 11 females. The females were all between seventeen and thirty-one. Of the males 7 were from fifteen to thirty; 2 from thirty to forty-five, and 1 forty-six years of age." In every instance, save one, the tubercular disease was far advanced. In 12 of the 21 cases the perforation had occurred on the right side, and in 9 only on the left, a fact which confirms my own observation, but is opposed to the statistics given by Dr. Walshe, relative to 74 cases collected from different authors, and to 10 observed by himself; of these 84 cases, we are told that 55 were examples of perforation on the left, and 29 only of its occurrence on the right. For obvious reasons, the results obtained from a number of cases taken indiscriminately at a large public institution are likely to be more trustworthy than those gleaned from selected cases, and I am, therefore, inclined to believe that there is probably little or no difference in the liability of the two sides of the chest to suffer from this complication of phthisis.

With respect to the precise seat of perforation, Dr. Walshe, without giving any statistics on the subject, asserts that "the pleura commonly gives way postero-laterally in the area comprised between the third and sixth ribs. I have already given the results of my own experience in the matter; but as I am disposed to give credence to the accurate statistics of a large public hospital, rather than to any generally expressed opinion, or to statistics derived from selected cases, I will again refer to our united experience at St. George's Hospital, as worked out in Dr. Chambers' report. Thus, then, in the 21 cases already alluded to as having occurred during the decennial period ending December 31st, 1850, the fistulous openings from the bronchi by vomicæ into the pleura were as follows:

On the right side—In the middle of the upper lobe, in 3 cases.
„ In the base of the upper lobe, in 1 case.
„ In the middle lobe, in 2 cases.
„ In the upper part of the lower lobe, in 1 case.
„ In the lower edge of the lower lobe, in 1 case.
„ Distributed through the upper part of the lung, in 3 cases.
„ In another case the lung contained many tubercles, but none were softened at the time of death, and any solution of continuity in the pulmonary tissue, which was condensed

by pressure of the air, seemed to have been obliterated.

On the left side—In the middle of the upper lobe, in 3 cases.
„ In the anterior part of the lower lobe, in 2 cases.
„ In the outer part of the lower lobe, in 1 case.
„ Distributed through the lung, in 1 case.
„ In 2 cases, though the lung was full of vomicæ, the exact position of the opening could not be ascertained.

These facts coincide very closely with those observed by myself, and with Dr. Walshe's statement, that perforation commonly occurs in the area comprised between the third and sixth ribs. The probable explanation of them is so well put by Dr. Chambers, that I am tempted to give it in his own words. He says*—"But how are the exposed parts," *i.e.*, the apex of the lung which is the ordinary habitat of vomicæ, and the base which is the usual seat of abscess—how are these parts "guarded? I believe by the following provision:—In breathing, the base, from the pressure of the diaphragm, and the apex, from being driven up into a corner, have less motion than the middle of the lungs, and consequently, when pleurisy occurs, they are most liable to become adherent to the thoracic walls. The adhesions thus formed not only prevent the perforations there from being detected, but they also prevent harm ensuing from them."

Thus, then, I think we are justified in stating that pneumothorax is not more prone to occur in one sex than in the other, nor on one side of the chest more than on the other; that it is most commonly met with between the ages of twenty and thirty-five, and is generally due to perforation in the middle of the chest on either side, for the reason that motion is greater there than on any part of the chest, and that the pleura is less likely to be protected against the results of perforation, by adhesion of the two surfaces of the membrane.

The general symptoms of perforation of the pleura are the sudden accession of a sharp pain in the side, accompanied by a sensation as of something giving way, and followed by intense dyspnœa. Sometimes the pain is agonising in the extreme, and all the general symptoms are well marked; at others there is little or no pain in the side, and though the breathing is very hurried, there is no sensation of dyspnœa. In

* Loc. cit., cap. v, sec. v, p. 65.

a case which I saw last year in consultation with Mr. Allen, of St. John's Wood, there was neither pain nor distress of breathing; and I have observed the same facts in several other instances.

As soon as perforation has occurred, the air, acting on the pleural membrane, excites irritation, and ordinarily gives rise to the effusion of fluid; so that the signs of hydropneumothorax may be discovered within a very few hours. Orthopnœa generally exists at first, but it often passes off after a time, and the patient lies, as he does in simple pleurisy, on his back, with his head somewhat raised, and his body slightly inclined towards the affected side. Distress and anxiety are depicted on the countenance; the complexion is pale and dusky, and the lips are more or less livid; the voice is weak; the skin moist, and often covered with a cold clammy perspiration; the pulse quick and feeble, and the respiration extremely hurried.

The physical signs of simple pneumothorax are sufficiently characteristic. There is evident convexity of the affected side, with obliteration, widening, and even bulging of the intercostal spaces, immobility or diminished movement of the chest walls, and inaction or diminished movement of the intercostal muscles, contrasting forcibly with the increased play of the opposite side of the chest and the energy of its intercostal action.

Palpation informs us that the vocal fremitus is diminished or altogether annihilated, that the intercostal spaces are more than usually elastic and resilient, and that the heart is more or less displaced.

Mensuration confirms our impression respecting the enlargement of the affected side, and the increased width of the intercostal spaces.

Percussion elicits a clear tympanitic resonance, which sometimes changes its character and becomes amphoric and of a metallic quality, over the trachea and larger bronchi. When the dilatation of the chest and the tension of the chest walls are excessive, the true tympanitic character of the sound is lost. As the mediastinum, the heart, and the diaphragm are more or less displaced, the area of clear resonance on percussion may extend considerably beyond its normal limits, and thus may transgress the middle line of the sternum; when pneumothorax occurs on the left side the pericardial dulness may be wholly replaced by tympanitic resonance.

Auscultation furnishes different results according as the amount of effused air is larger or smaller. If the quantity of air be small, the respiratory sounds are weak and distant, and the vocal resonance is weak;

if it be great, so that the lung is thoroughly compressed, the respiratory sounds and the vocal resonance are almost or altogether absent, except in the interscapular region at the root of the larger bronchi, where diffused blowing respiration and diffused but loud vocal resonance may still be audible. In some instances, even where little or no respiratory murmur is present, there may be diffused vocal resonance over the entire chest, and in others the voice, the cough, and even the sound of inspiration may be accompanied by a metallic or amphoric echo, especially when the communication with the bronchi is free. The heart's sounds are usually transmitted feebly through the distended pleura, but sometimes, as with the voice and cough, they may give rise to an echo of a metallic quality.

The signs of hydropneumothorax, as the title implies, are in part those of pleurisy and in part those of pneumothorax. The former, such as dulness on percussion, and the other signs referable to the presence of fluid, are met with in the lower or more dependent portions of the chest; the latter, including tympanitic resonance, at the upper part, to which air necessarily rises. Their precise character will be manifest by reference to what has been already stated respecting the signs of pleurisy and pneumothorax respectively; and it need only be added, that their relative position in the chest will be found to vary according to the posture of the patient.

But there are certain signs resulting from the coexistence of air and fluid in the same cavity, which are not met with in connection either with pleurisy or pneumothorax, but which frequently occur with and are very characteristic of hydropneumothorax. I allude—1st, to fluctuation, which is felt by the patient as well as by the observer when the body is abruptly jerked or shaken; 2ndly, to the ringing, splashing sound—the succussion sound of Hippocrates*—which is heard under the same circumstances; 3rdly, to the remarkable metallic tinkling which sometimes accompanies succussion of the patient, but is also apt to accompany cough or inspiration, or a sudden change in the patient's posture. The character and mechanism of these sounds will be seen by reference to the former part of this work;† and it need only be stated further, that they may be heard as well when the communication between the bronchus and the pleura has closed, as when it

* 'Hippocrates de Morbis,' lib. ii, 45.

† See chap. x, pp. 142-151.

remains open; but that metallic tinkling is not usually found to accompany inspiration unless the opening be free, so that bubbles of air can pass through the fluid during inspiration.

The quantity of air and fluid, and their proportion relatively to one another, vary greatly, and thus of course the distension of the side and the urgency of the symptoms arising therefrom are also found to vary. This difference, as far as relates to the quantity of air, is due, I believe, to a peculiarity in the form of the opening into the pleura. In some instances it is valvular, and permits the free ingress of air, but closes instantly against its egress; whilst in other cases it is fistulous, and admits of air being expelled as freely during expiration as it entered during inspiration. In the former, air necessarily accumulates in the pleura and causes enormous distension; in the latter, it exercises little influence in producing distension. Thus it happens that in certain cases, but not invariably, enlargement of the affected side and displacement of the organs reach their utmost limits in hydropneumothorax, and so does amphoric resonance of the voice, the cough, and the respiration —the amphoric character being usually most marked in proportion as the ear approaches the seat of perforation.

The general symptoms of hydropneumothorax do not differ notably from those of pneumothorax, and, therefore, need not be recapitulated.

The prognosis of pneumothorax and hydropneumothorax is always uncertain; and if an opinion is asked as to the issue of any particular case, it should be given with due caution and reserve: *cæteris paribus*, it is most favourable in traumatic cases in which the chest walls are punctured, and a communication is thus established between the air and the pleural sac, but in which, nevertheless, the lung is not wounded; and it is most unfavourable in perforative cases resulting from disease in the tissue of the lung. But the greatest uncertainty attaches to the question, irrespective of that arising from the cause of the disease. The most favourable cases will sometimes terminate fatally in the course of a few days, whilst the cases apparently the most unfavourable go on steadily to recovery. I have seen two instances of complete recovery from pneumothorax in phthisical patients, and several in which the patients recovered from the immediate effects of the attack sufficiently to return to their homes, though air and fluid still coexisted in the pleural cavity. Only last year a man under my care in the York Ward of St. George's Hospital, in whom an empyema emptied itself through the lung, recovered rapidly

without a single drawback;* and I have known several other instances of the same kind.

There are no statistics which throw any light upon the prognosis of pneumothorax, nor have I met with any feasible explanation of the differences observed in its issue in different cases. Certain it is that although the habits of life and constitutional peculiarities exercise their influence in this as in other disorders, they do not afford an adequate explanation of the vast difference observed in the course of the disease. My own experience inclines me to believe that, *cæteris paribus*, those cases are most apt to run an untoward course which are accompanied by great accumulation of air, by great displacement of the thoracic organs, and great consequent interference with the functions of life; and that, therefore, an unfavourable issue is most probable when the opening into the pleural sac is of a valvular form, or becomes so in consequence of the super-imposition of false membrane. Indeed, I question whether in some such cases it may not be desirable to relieve the tension of the side by puncturing the chest walls and allowing the air to escape. When pneumothorax does not prove rapidly fatal, the opening into the pleura may either close or remain pervious. The former condition occurs when the case terminates favourably. But closure of the opening is not necessarily a sign of a favourable issue. In some fatal cases the opening may close; in others it may remain patulous. In certain instances, again, the opening may remain patulous for months or years, and may ultimately close, and the patient may recover;* in others it may remain pervious for an equally long time without any material declension of the patient's health, though recovery does not eventually take place. Laennec mentions the case of a consumptive patient in whom all the signs of hydropneumothorax, with a pervious opening, existed at the expiration of six years; and I have noted three in one of which these signs were present at the end of eleven months; in another, at the expiration of nineteen months, and, in the third, the case referred to below,† after the lapse of twenty-seven months. Such

* Hospital Case-book, xxxviii, p. 178.

† A man named Lacey was admitted into St. George's Hospital, on February 18th, 1846, suffering from empyema of the left side, in whom this fact was strikingly exemplified. Fourteen months after the chest was punctured the side was discharging freely, and two years and three months afterwards there was still a discharge daily. Five years after the operation I saw him again. At that time the opening in the side

cases, however, are quite exceptional, and can be regarded as little else than medical curiosities.

The treatment of pneumothorax is similar to that recommended for pleurisy, due allowance being made for the nature of the malady from which the affection has originated. Opium, in full doses, with sulphuric æther, and other diffusible stimulants and antispasmodics, may be necessary to tranquillise the nervous system, overcome the collapse, relieve the dyspnœa, and subdue the agonising pain by which the accession of the disease is often marked; but as soon as the first shock of the attack is overpast, and reaction has set in, our principal reliance, in traumatic cases, must be on calomel and opium internally, and on turpentine and poppy fomentations and blisters externally. Venesection has been recommended, and, in some instances, has afforded relief; but it must be remembered that, in the majority of cases, pneumothorax arises in persons whose lungs are organically diseased, and who can ill bear loss of blood. In such cases, therefore, if bloodletting be practised, a small quantity only of blood should be drawn. For the same reason, the constitutional effects of mercury should be avoided; and if mercury is given at all, it should be administered cautiously, and in combination with medicine calculated to sustain the patient's strength. In short, the treatment which I have found most efficacious in tuberculous cases, consists of the exhibition of full doses of opium or other sedatives, followed, in the course of a few days, by quina and cinchona, the mineral acids, cod-liver oil, and a light but nutritious diet, aided from the first by turpentine and poppy fomentations, linseed-meal poultices, blisters, and the application of mercurial ointment on the side, after the manner recommended in pleurisy occurring in weakly subjects.

In non-tuberculous cases, paracentesis thoracis appears to be desirable whenever the accumulation, whether of air or fluid, is such as to produce great oppression of the breathing, and interfere with the functions of life. But in tuberculous cases, the question of paracentesis can seldom arise, inasmuch as it can only be regarded as a palliative, and, in many instances, would not be justifiable, even when viewed in that light. Nevertheless, if the mischief in the lungs is not very extensive, nor far advanced, there cannot be any reason to decline performing the operation, if the urgency of the dyspnœa, or the displacement of the thoracic

was quite closed, and the side, which had been much collapsed had expanded considerably. How long the discharge had continued it was impossible to ascertain with accuracy, but he stated that it had continued nearly three years.

viscera is such as to demand operative interference. It does not render matters materially worse, and it sometimes affords great temporary relief.

Hydrothorax.

Hydrothorax is a term applied to dropsy of the pleura, or, in other words, to a non-inflammatory accumulation of serous fluid in the pleural sacs. It commonly results from passive congestion of the pleural vessels, consequent on disease of the heart and obstruction to the pulmonary circulation, or on Bright's disease of the kidneys, or other disorders which impoverish the blood and conduce to congestion of the capillary system. But sometimes the congestion assumes an active form, and trenches closely on true inflammation. In these cases, there is more or less uneasiness in the chest, and often some accession of febrile action; but these soon pass off; and when death occurs, no plastic exudation or other inflammatory product is found in the chest. The effusion, as in the former cases, consists of nothing more than a thin, clear, yellowish green, or straw-coloured, transparent serum, in which some masses of amber-coloured, gelatiniform lymph are sometimes found floating.

Hydrothorax generally commences, and often continues for some time without any symptom to attract attention beyond gradually increasing dyspnœa and orthopnœa. There is little or no cough, no stitch in the side, no pain or tenderness on pressure as in pleurisy, no febrile heat of skin, no marked acceleration of the pulse; but the dyspnœa is more urgent, and the patient's posture is characteristic. When the effusion is very scanty, so that he is able to lie down, he invariably reclines on his back, rather than on either side, but after a short time, there is always more or less orthopnœa, and as effusion increases, the dyspnœa and orthopnœa become excessive, the lips and face extremely livid, and the extremities also cold and livid, the countenance is anxious, and the skin is bedewed with a clammy perspiration. Indeed, these symptoms are manifested to a far greater degree, and occur more rapidly than in pleurisy. This arises from the fact that pleuritic effusion is usually confined to one side of the chest; whereas hydrothorax, being commonly referable to causes which operate through the general circulation, is apt to occur simultaneously on both sides, so that the functions of respiration and circulation are more seriously disturbed in the latter than in the former affection.

The physical signs are almost identical with those which have been described as accompanying pleurisy, with the exception that in hydrothorax, as the pleura is not roughened, no friction fremitus is felt, and no friction sound heard; that percussion dulness occurs on both sides of the chest, instead of being limited to one side; that the area of dulness shifts its position with the varying posture of the patient more rapidly and more markedly than in pleurisy, and that as effusion takes place on both sides, there is little or no lateral displacement of the heart and mediastinum as in cases of abundant pleuritic effusion on one side only of the chest.

The treatment of hydrothorax must be regulated by the nature of the mischief from which it arises. It is merely a symptom of other mischief, and unless that mischief can be remedied, it is vain to expect to get rid of the fluid. If it is due to valvular or other disease of the heart, or to disease of the kidney, or to some diathetic disorder, our aim must be to alleviate the complaint by which it is occasioned, relieve the circulation, and so put a stop to further effusion. If we are unable to do this, our utmost efforts will not avail to counteract the continued outpouring of fluid; whereas, if we can mitigate the primary disease, and arrest the effusion, we have a reasonable prospect of being able to promote absorption of the fluid already effused. Frequent dry cupping, repeated blistering, and iodurettted lotions and ointments externally, and internally the administration of diuretics and purgatives, are the means to be employed in these occasions. Paracentecis-thoracis is seldom needed, and ought never to be adopted unless effusion on both sides of the chest continues in spite of remedies, and threatens immediate suffocation. Its performance, under such circumstances, might serve to afford temporary relief and prolong life, and, therefore, would be justifiable, even though it be incapable of effecting a cure.

Hæmothorax.

Hæmothorax, or hæmorrhage into the pleural cavity, never occurs as an idiopathic affection, but is occasioned sometimes by fracture of the ribs, or wounds of the chest walls, and sometimes by the outpouring of blood consequent on disease of the thoracic walls or the thoracic viscera. Thus it may result from an aneurism bursting into the pleural sac, from the giving way of the visceral pleura under the pressure of pulmonary

apoplexy, and from the rupture of vessels accompanying ulceration in various forms of disease of the lungs, the pleura, or the chest walls.* These latter causes, however, are of such rare occurrence that, in a large majority of cases, the complaint is of traumatic origin.

Practically this affection calls for very few remarks, inasmuch as it is not productive of any characteristic physical signs or general symptoms, and admits of very little relief by treatment. The physical signs resemble those observed in pleurisy, except that pleuritic friction sound is not heard in hæmothorax; again, they are similar to those which accompany hydrothorax, except that they are confined to one side of the chest, and that the dulness on percussion does not shift its position with the varying posture of the patient so constantly, and so completely as in that disease. Further, in the majority of cases, the accession and full development of the physical signs are more sudden than in either pleurisy or hydrothorax. The general symptoms consist of little more than dyspnœa and laboured breathing, the necessary results of compression of the lung and interference with its action. But when dyspnœa occurs suddenly, during the progress of visceral disease, or after injury of the chest, and, though accompanied by the physical signs of effusion on the one side of the chest, is not attended by any evidence of inflammatory action, but rather by pallor, faintness, and failure of the pulse, there is seldom much difficulty in arriving at a correct conclusion respecting the nature of the malady.

The treatment of hæmothorax may be summed up by the statement that everything must be done to obviate the tendency to death. The collapse is sometimes so great when the hæmorrhage first takes place, that diffusible stimulants are absolutely necessary to sustain life; afterwards, when reaction has occurred, pleurisy may be set up, and antiphlogistic treatment may be required accordingly. The only question which can arise, is as to the propriety of puncturing the chest at the very outset of the hæmorrhage. My own opinion is adverse to the practice, inasmuch as it is obvious that the admission of air into the pleura may be prejudicial, and that the removal of the pressure which the ex-

* Dr. Watson (loc. cit., vol. ii, p. 3, ed. i) records a remarkable case in which an enormous hæmothorax which had caused enlargement of the left side of the chest, effaced the intercostal spaces, and pushed the heart over to the right of the sternum resulted from scrofulous ulceration, which had destroyed two of the ribs and laid open one of the intercostal arteries.

travasated blood creates must favour the continuance of hæmorrhage, and so endanger the patient's safety. I am bound nevertheless to add that my objections to the operation are purely theoretical, and that M. Roux * and others have practised it with success. The question of performing the operation at a later period, when pleurisy has occurred and serous effusion has taken place, must be decided on the principles already discussed, when the subject of paracentesis in pleurisy was under consideration.

CHAPTER II.

PNEUMONIA.

We have hitherto confined our observations to pleurisy, or inflammation of the investing membrane of the lungs. We must now pass on to the consideration of pneumonia, or inflammation of the substance of the lungs, the true pulmonary tissue.

The attack is generally preceded by restlessness, followed by shivering, prostration of strength, feverish heat of skin, increased frequency of pulse, hurried respiration, and short dry cough, with a stitch or catching pain in the side, about on a level with the nipple. The restlessness and uneasiness increase, the breathing becomes more hurried and oppressed, varying in frequency from 30 to 60 in a minute; and even if the cough were dry in the first instance, it is after a time accompanied by the expectoration of stringy, adhesive, rusty-coloured mucus. The patient lies on his back, or slightly inclined to one side or the other, supported by pillows; the face is commonly flushed, the countenance anxious; the nostrils are dilated and in full action; the lips are more or less livid, the tongue is coated with a white or yellowish-white fur, the urine is high-coloured and loaded with lithates, and the pulse full and frequent, varying from 100 to 120.

Such are the usual symptoms of pneumonia. They are subject, however, to every possible variation, and possess so little of a distinctive character, that a physical examination of the chest is needed, in order that they may be interpreted correctly. With that aid, fortunately,

* Reported by Sédillot in p. 107 of his work, 'De l'Empyème.'

there is little difficulty in recognising their true character, and tracing every step in the progress of the disease.

But before discussing the physical signs of pneumonia, it may be well to look a little more in detail into certain circumstances connected with its invasion, and into the variations observed in its general symptoms.

And first, as to the circumstances connected with its invasion. Pneumonia generally occurs as a primary affection whilst the patient is apparently in good health. In some instances, however, it arises in connection with bronchitis, the inflammation spreading gradually from the larger to the smaller bronchi, and so by degrees to the air vesicles and tissue of the lungs. In other instances it appears to be occasioned by the congestion and irritation to which the lung is subjected during the progress of febrile disorders. Its accession, therefore, may be sudden, or it may be gradual and insidious.

Next, as to the variations observed in the general symptoms. Shivering is the most constant forerunner of pneumonia; but it may be altogether absent, even in severe cases. Therefore, although it is generally observed, the mere fact of its non-occurrence will not justify an opinion as to the non-existence of the disease.

Pain in the side is another symptom of variable occurrence. It is usually present to a greater or less degree, so that M. Grisolle, who examined 301 cases, with a special view to this inquiry, reports it in no less than 272 cases; and in 94 cases which occurred in the physicians' wards of St. George's Hospital, during the period of my registrarship, it was noted in 73 cases. Nevertheless, the most formidable pneumonia may exist without it, or it may be severe and catching at one period of the disease, and slight or altogether absent at another.

It has been stated—but I know not on what authority—that pain is not met with in pure, uncomplicated cases of pneumonia, and arises only when the pleura becomes involved by an extension of inflammation from the contiguous lung structure. Observation, however, has led me to doubt the accuracy of the statement; for although it is true that pain is more constantly present in pleurisy than in pneumonia, yet cases are not wanting in which wide-spread pleuritic inflammation has been unattended by pleuritic pain. On the other hand, cases of pneumonia are met with in which no evidence of pleurisy can be obtained by auscultation, and in which, nevertheless, the stitch in the side is very severe; nay more, I have traced cases to the dead-house of St. George's Hospital, in which pain in the side had existed during life, and in which, after

death, the closest inspection has only served to confirm the impression previously entertained as to the absence of pleuritic inflammation. Therefore, whilst admitting that acute, catching pain in the side is suggestive of pleuritic complication, I am bound to maintain that it does not necessarily indicate its existence. It may be, and very generally is, attributable to pleurisy; but in some instances it is referable to intercostal neuralgia, and it ought not to be regarded as of pleuritic origin, unless a careful physical examination of the chest unmistakeably attests the correctness of such an opinion. The mere presence of a metallic or quasi-ægophonic resonance of the voice is not sufficient to decide the question.* Pleuritic friction sound should be heard, or some other evidence of effusion into the pleural cavity obtained, before such a conclusion can be justified.

In corroboration of the view that pain in pneumonia is not always connected with pleuritic inflammation, is the fact that the seat of pain does not usually coincide with the seat of pneumonic inflammation. It is but fair to conclude that pleurisy, arising in connection with pneumonia, would occur in that portion of the membrane which is in apposition with the inflamed lung tissue; and, undoubtedly, when well-marked pleuropneumonia occurs, the pain is commonly referred to that portion of the chest at which evidence of pneumonia and pleurisy is to be obtained. But, in the cases now under consideration, in which proofs of pleuritic inflammation is confined to the existence of slight ægophonic resonance of the voice, the pain seldom corresponds with the seat of pneumonia. It is usually felt on a level with, or a little below the nipple; but it may occur in any other portion of the thoracic walls, and it is aggravated by causes which influence the pain of intercostal neuralgia just as much as that of pleurisy. It is increased by cough and by a full inspiration; by sudden change of posture, and by suddenly and forcibly raising the arm on the affected side; by pressure upon or percussion of the intercostal spaces over the painful part, and, in short, by whatever brings the intercostal muscles into play, or exerts any pressure upon them. Thus, it generally happens, that the patient cannot lie on the affected side, and prefers lying on his back. In certain cases, however, I have known a patient lie most comfortably on the painful side, being relieved by the uniform pressure thus exerted, though suffering, of course, some increased oppression of the breathing in consequence of his position. It is obvious, then, that in the existence of pain there

* See pp. 106-110 of this treatise.

is nothing absolutely characteristic of the disease under consideration, and little even to aid us in our diagnosis.

Dyspnœa is another symptom which varies greatly in severity. Sometimes it is extreme; sometimes so slight that the patient is hardly conscious of it, and between these two extremes every degree of embarrassment of the breathing may be observed. It always takes place, however, to a greater or less degree, and begins to manifest itself within a few hours after the commencement of the attack. The respirations may amount to 24 or 26 in a minute, almost from the very outset of the attack, and may even number 35 or 40, without any apparent difficulty of breathing; but when they attain, as they sometimes do, to 60 or 70 in a minute, it is obvious that suffocation is imminent: the face becomes livid or pale, the nostrils are dilated and in full action, the greatest anxiety is depicted on the countenance, and the patient is almost unable to speak.

Strangely enough, the dyspnœa is not proportioned to the extent of lung affected by inflammation, and does not appear to be necessarily connected with mischief in any particular portion of the lung; so that, in many cases, there may be less frequency of breathing when an entire lung is inflamed, than in others when a comparatively small portion of the lung tissue is implicated. This holds good even when the inflammatory mischief is unaccompanied by pain. The fact appears to be, that some persons possess a relatively large surplus quantity of lung tissue, which is not brought into play during tranquil breathing; and they are, therefore, less embarrassed by the inflammation and consequent inaction of a certain portion of their lung than are those who, possessing relatively small lungs, ordinarily employ the whole, or the greater part, of their respiratory apparatus. Hence, it happens that, although extreme dyspnœa is always a symptom of the gravest import, it is not, necessarily, of fatal augury; and that, viewed alone, without reference to other symptoms, it is a very uncertain guide to the extent of pulmonary inflammation.

There is yet another point relative to the respiration, which is deserving of special notice, viz., that the increase of its frequency is out of all proportion greater than that of the pulse. Thus, at the very outset of the attack, the respirations will be often 30 in the minute, whilst as yet the pulse does not exceed 80 or 84; and when the pulse rises to 100 or 110, the respirations will be found to be 45, 50, or even 60. In like manner, with the decrease of the disease, the respirations fall

in frequency more rapidly than the pulse, and soon resume their natural relative proportions. Indeed, the variations in the ratio existing between the pulse and the breathing is in no disease more marked than in pneumonia; and in none is more information to be derived from it in regard to the progress and probable issue of the disorder.

The cough of pneumonia is not in any way characteristic. Commencing almost with the first invasion of the disease, it is not always proportioned either to the extent or the intensity of the pulmonary inflammation; it is generally frequent, but seldom occurs in paroxysms; it is short and dry in the earlier part of the attack; looser and accompanied by expectoration at a somewhat later period; slight or altogether absent towards the close of fatal cases. The expectoration at first consists of nothing more than ordinary bronchial mucus, but at the expiration of a day or two, and sometimes within a few hours, it assumes a truly pneumonic character, and becomes viscid, semi-transparent, rusty-coloured, and tenacious, adhering so strongly to the vessel containing it, as to admit of the vessel being turned upside down, without its becoming detached from the sides. The rusty coloured expectoration consists of mucus intimately mixed with blood—not streaked with it as in bronchitis, but thoroughly mixed and amalgamated with it—so that it acquires a yellowish or reddish-yellow, or even a red colour, according to the quantity of the blood. If the disease be not very intense, the expectoration never attains the degree of viscidity or the depth of colour above referred to, but, though still tenacious, and adherent to the sides of the vessel, moves from one part to another, as the vessel is tilted. If the disease, however severe, progresses to a favourable termination, the sputa become more abundant, less adhesive, and less highly coloured, passing through the various shades of orange, until at length they become greenish or whitish, and resemble the expectoration of ordinary catarrh. If, on the other hand, the disease be hastening to a fatal termination, the expectoration becomes scanty, less tenacious, and of a darker or dullish brown hue, resembling the juice of prunes. Sometimes, if the type of inflammation be low, or if it be connected with tubercular deposit, the mucus may be tinged or even streaked with blood, and in another class of cases, which of late years have been more frequent than formerly, the expectoration is neither blood-tinged nor rusty coloured, but consists throughout of nearly colourless, stringy and more or less frothy mucus. In fact, the character of the expectoration varies greatly in different cases, and even

at corresponding stages of the complaint in different cases; so that it is impossible from mere inspection to draw any inference as to the stage at which the disease has arrived, or as to the precise condition of the lung. Under the microscope the rusty coloured sputa of pneumonia are seen to consist of mucus corpuscles, epithelium, exudation cells, granular matter, blood discs, oil globules, and casts more or less perfect of the ultimate subdivisions of the bronchi. Sugar has been detected in it during the height of the inflammation, and Dr. Beale has shown that an excess of the chloride of sodium is constantly present.* During the advanced stages of pneumonia, when the respiration is very frequent, the expired air is colder than natural, and contains less than the ordinary proportion of carbonic acid. This has been clearly proved by the experiments of Nysten, and is doubtless attributable to the impermeability of the lung, and to the consequent diminished activity of the chemical changes taking place on the pulmonary apparatus.

Heat of skin is one of the most constant symptoms of pneumonia. The temperature of the surface is almost always raised, and the skin is often pungently hot and burning. Sometimes the thermometer, placed under the tongue, will rise to 100 or 104, but this holds good only during the active stage of the disease; for when the breathing is much oppressed, and the disease is tending to a fatal termination, the temperature of the body sinks, the surface becomes cold, a clammy perspiration breaks out, and sudamina sometimes make their appearance.

The pulse varies greatly in different cases and at different periods of the disease. About the third or fourth day of the attack it generally beats 120 or 130 per minute, and in cases tending to a fatal issue it may attain to 160 beats per minute; but in some persons the disease may run its course with a pulse not exceeding 60 or 70. In most of such cases there is a remarkable absence of feverish excitement, and the symptoms have always appeared to me to denote passive congestion rather than truly active inflammation. Certain it is that in the more formidable types of the disease the pulse is always frequent, and in the first instance generally full and resistent; and even when the breathing has become oppressed and the pulmonary circulation embarrassed, it is still rapid, though small and feeble. In one instance only have I chanced to meet with a quiet pulse coincidently with serious and extensive pulmonary inflammation; and in the case alluded to, the pulse in-

* 'Med.-Chir. Trans.,' vol. xxxv.

creased in frequency after the first violence of the attack had abated. I am unable even to offer a conjecture as to the cause of the slowness of the pulse in these cases, but the suggestion offered by Dr. Walshe as to the pulse in such patients being still slower in health, is not in accordance with the results of general observation. The same slowness of the pulse is observed sometimes in pericarditis, peritonitis, and other inflammatory disorders; and in those cases, just as in the case of pneumonia above alluded to, I have known the frequency of the pulse increase with the subsidence of the inflammatory mischief.

The blood in pneumonia is highly charged with fibrin, so much so that the fibrinous element amounts sometimes to 13 parts per 1000, and on the average to 7·3 parts.* Consequently, when venesection is practised, the clot is highly buffed and cupped; and when the circulation through the lung is much impeded there is a great disposition to the formation of fibrinous coagula in the right cavities of the heart. In some cases of pneumonia the presence of these coagula forms a serious aggravation of the danger, and renders recovery impossible.

The brain sympathises with the general disturbance, and "cerebral symptoms" not unfrequently result. These consist of headache, which is amongst the earlier and more common accompaniments of pneumonia; of delirium, which, though less common, is by no means of unfrequent occurrence,† and of convulsions and coma, which are rarely, if ever, met with, except towards the close of fatal cases. These head symptoms when severe, are always alarming; for they denote that the nutrition of the brain is seriously interfered with; and inasmuch as this can take place only through the medium of the blood, it is obvious that the vital fluid must be either highly charged with a *materies morbi*, or else must be very imperfectly arterialized. In either case it is unfit for the proper maintenance of the vital functions, and in its effects affords a measure of the severity of the disease, of the extent and intensity of the mischief in the chest, and of the irritability of the nervous centres. My opinion, however, is opposed to the belief that the delirium occurs merely as a consequence of the impeded state of the pul-

* See Simon's 'Chemistry,' Sydenham Society's publication, vol. i, p. 260.

† A remarkable instance of violent maniacal delirium in connection with pneumonia of the upper lobe of the right lung occurred in a man (Job Warren, aged 24), admitted under my care into the Cambridge Ward of St. George's Hospital, February 7, 1862. It came on suddenly without previous headache, and subsided under the use of full doses of opium and æther.

monary circulation. I am rather inclined to regard it as invariably dependent on a poisoned blood, the morbid action of which is rendered more surely operative by its imperfect arterialization, and by some unusual excitability of the brain.

The digestive organs are usually deranged to a greater or less extent, but not in any constant or characteristic manner. Thirst is sometimes a prominent symptom, more especially among children.* The appetite is impaired or altogether lost; vomiting occurs sometimes but not generally; the tongue is usually furred, and the bowels are often but not invariably costive.

The urine is generally scanty during the height of the disease, deep coloured, loaded with lithates, and of specific gravity varying from 1015 to 1030, or even higher. Sometimes it contains a few crystals of lithic acid and of oxalate of lime, and occasionally a small quantity of albumen; the inorganic salts are deficient, the chloride of sodium especially so—a fact observed in other acute inflammatory disorders, and dependent in great measure on the nature of the food consumed. It has been found, however, that as the chlorides decrease in the urine they increase in quantity in the sputa,† and *vice versâ*, and it is therefore probable that their deficiency in the urine is partly attributable to their determination to the inflamed lung.

It will be admitted, then, that there is nothing in the general symptoms of pneumonia to guide us with certainty to a correct diagnosis. If the sputa happen to be extremely adhesive, semi-transparent, and rusty coloured, and present under the microscope the characters already described as appertaining to pneumonic expectoration, it is fair to conclude that inflammation of the pulmonary tissue is present. But pneumonic inflammation may be very severe, and yet the sputa may not be characteristic; so that it is only by reference to the physical signs that a correct conclusion can be drawn as to the nature of the disease and the stage at which it has arrived.

It has been already stated that the physical signs are dependent on the morbid changes to which inflammation gives rise in the lung and it may be well, therefore, to collocate a description of the morbid changes and physical signs which are usually observed at different stages of the disease.

By common consent the course of pneumonia has been divided into

* 'Rilliet et Barthez,' p. 99.

† See Dr. Beale's paper in 'Med.-Chir. Trans.,' vol. xxxv.

three stages, corresponding to three well-marked and distinctive conditions of the lung. The first is that of *engorgement or splenization;* the second that of *red hepatization;* the third that of *grey hepatization, or diffused suppuration of the pulmonary tissue.* The peculiarities of each condition and the physical signs by which they are each accompanied will be apparent from the following table:

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First Stage — Engorgement, or Splenization.—The pulmonary capillaries are gorged with blood, and the air cells are loaded with bloody serum, which has exuded into them, and has been rendered frothy by the admixture of air.

The inflamed lung is of a livid red or violet colour externally, and its surface is less smooth and glistening than natural; it does not collapse like a healthy lung, and is heavier and less crepitant under the finger; moreover it is less elastic than natural, so that it pits on pressure like an œdematous lung.

When cut into, a quantity of reddish, frothy fluid escapes, and the cut surface is seen to be spongy and of a brick-red colour. A gentle stream of water directed on it will wash away the greater part of the exudation, and render this condition more apparent. The lining membrane of the bronchial ramifications is of a deep red colour.

The permeability of the lung is lessened, not destroyed; so that, although the specific gravity of the

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First Stage—Inspection.—Costal movements not materially diminished except when catching pain in the side leads to their being restrained.

Palpation.—No material alteration can be perceived in the vocal fremitus.

Mensuration.—Size of affected side not increased.

Percussion.—A peculiar amphoric quality of resonance is often emitted by that portion of the chest at which pneumonia is just beginning;* but this ceases soon after the commencement of exudation and is replaced by dulness more or less marked, according to the amount of exudation and the extent of lung involved.

Auscultation.—During the early period of engorgement, and before exudation takes place, the breathing is weak in the affected parts; exaggerated in their immediate vicinity. As soon as fluid is poured out the respiratory sounds are obscured or replaced by the characteristic small crepitation of

* See pp. 54-60 of this treatise.

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lung tissue is increased, the most engorged portions will float when placed in water. Its consistence is found to be diminished, and its tissue is softer than natural, and easily torn like the spleen. Hence the term splenization.

If resolution takes place at this stage of the complaint, the sero-sanguinolent fluid which has been poured out is absorbed, the congestion gradually passes off, and the lung returns to its normal condition.

In certain instances, which during life are unaccompanied by crepitation, there is little or no frothy fluid in the air cells, but the interlobular cellular tissue is the seat of inflammation, and is distended with a reddish coloured fibrinous exudation, which presses upon and, for the time, obliterates the air vesicles.

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pneumonia—a sound as of a multitude of minute crackles, which occur in a volley towards the close of inspiration, and are not affected by coughing or expectoration. Neither bubbling râles nor rhonchi are audible, unless bronchitis be also present.

When the air cells become completely filled and the minute bronchial ramifications obstructed by exudation, the crepitation ceases; and when, as often happens, inflammation extends to the smaller bronchi and capillary bronchitis is set up, small bubbling râles are heard accompanying expiration as well as inspiration. In some few instances, especially when pneumonia arises in connection with acute rheumatism, crepitation never occurs—a fact which I have verified on several occasions, and believe to be attributable to the occurrence of exudation into the interlobular cellular tissue, and consequent immediate occlusion of the air cells. The mere non-occurrence of crepitation, therefore, is not a certain proof of the non-existence of pneumonia.

The vocal resonance is usually somewhat intensified.

If the disease is checked at this stage, and resolution takes place, crepitation gradually ceases, and is replaced by the respiratory murmur, whilst at the same time the

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Second stage. — Red hepatization.—Not only are the pulmonary capillaries gorged, but the air cells are distended with fibrinous exudation, which produces consolidation of the pulmonary tissue, so that the lung does not collapse when the chest is opened. The surface of the lung is still of a brownish red or livid red colour, but the hepatized portions no longer contain air, no longer crepitate under pressure, and sink when placed in water.

When an incision is made into the lung, the fluid which escapes is usually thicker and less in quantity than in the state of engorgement; it is of a reddish brown or claret colour, and almost free from admixture of air. The lung tissue is no longer spongy in appearance; it is evidently more compact and solid than natural, and is sometimes firm and resistant; but more commonly it is soft and friable, or easily broken down; so much so, indeed, that it may be quite rotten, and may be reduced to a state of pulp by the slighest pressure.

Its cut surface is frequently smooth, and resembles the cut surface of liver; more commonly it is granular, and occasionally it

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chest recovers its normal resonance on percussion.

Second stage — Inspection. — Costal movements diminished on the affected side; that of elevation, however, less than that of expansion. On the unaffected side they are somewhat increased.

Palpation. — Vocal fremitus varies. It is usually above the average standard, but, in some instances, it falls below it, and occasionally, when the bronchi are plugged with exudation it is altogether abrogated. Of this I have no doubt; for, on two occasions in which during life I noted entire absence of vocal fremitus, I had the opportunity of verifying by *post-mortem* examination the existence of mere pneumonic consolidation.

Mensuration. — I have never been able to satisfy myself as to the existence of any increase in the size of the affected side, but M. Grisolle asserts that he has detected slight enlargement in some rare instances.*

Percussion.—Sound usually dull, and sense of resistance to the finger considerable. In some instances, however, the diminution of clearness is not great, and in others, percussion may elicit a clear but shallow resonance. This occurs only when a superficial layer of

* 'Traité pratique de la Pneumonie,' par M. Grisolle, pp. 226-7.

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is pulpy, and almost diffluent. Sometimes it is of a uniform claret red colour, but at others mottled or variegated, partly from the presence of black pigment, and partly from the contrast afforded by the interlobular cellular tissue, and by the coats of the bronchial tubes and vessels, which are less red than the surrounding tissue.

Its torn surface is always granular, and when the granulations are carefully examined with a lens of low power, they are seen to be swollen air cells, filled with blood-tinged coagulated fibrin, which sometimes may be detached from the cell walls to which it is adherent. In some rare instances the interlobular cellular tissue appears to be the principal seat of mischief, and is seen to be infiltrated with fibrinous exudation, in which case the air vesicles are compressed and almost obliterated, instead of being distended with exudation, and the torn surface of the lung does not present the usual granular appearance.

As the second passes into

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the lung contains air, whilst solidified lung lies behind it.*

Auscultation.—The respiration is of a hollow character, sometimes diffused throughout the hepatized portion, but more commonly tubular in its centre, and harsh, diffused and blowing towards its periphery. Over those portions where tubular breathing exists there is entire absence of râles and rhonchi, and intense vocal resonance is heard, which is usually of a metallic, ringing character, and often *quasi* ægophonic. Towards the confines of the hepatized portion the vocal resonance is less intense, and loses its ringing, ægophonic character. Just beyond the limits of the hepatization, fine crepitation is often audible.

Sometimes, when the fibrinous exudation is very great and the bronchi become obstructed, neither tubular breathing, nor râles nor rhonchi, are audible over the hepatized lung; there is little or no vocal resonance, and there is absolute dulness on percussion.

* See p. 54-60 of this treatise. The cases referred to by various authors in which this resonance is attributed to the proximity of an air-distended stomach, or to the presence of a large bronchus behind a thin layer of solidified lung, are all, I believe, explicable by the condition of lung above described. If the proximity of an air-distended stomach would occasion this resonance, it ought to be present in almost every case of pneumonic consolidation of the lower lobe on the left side.

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the third stage of the disease, the hepatized lung becomes less dark-coloured, and traces of a grey or yellowish matter may be perceived here and there mixed with the reddish fluid, which escapes when the cut surface is gently scraped with a scalpel. When resolution takes place at this stage of the complaint, a serous fluid is poured out, which leads gradually to the separation and liquefaction of the solid fibrinous matter previously effused, and as the liquefied matters are expectorated the lung becomes more pervious to air.

Ultimately the exuded matter may be thoroughly got rid of by expectoration, the engorgement of the pulmonary capillaries may pass off, the colour and sponginess of the lung return, and the lung reassume its normal appearance.

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Such examples, however, are somewhat uncommon. The heart's sounds are frequently transmitted through the consolidated lung with unnatural intensity; but the facility of their transmission appears to vary with the precise species of exudation, and the consequent condition of the consolidated lung. Through some lungs which are extensively hepatized the heart's sounds are not transmitted at all; through others, also extensively hepatized, not only are the cardiac sounds transmitted, but a distinct fremitus may be conveyed to the hand.

As soon as resolution has commenced, and liquefaction of the solid exudation matter takes place, other signs begin to manifest themselves. The respiration is then accompanied by returning crepitation, or more often by moderate-sized râles, resulting from air passing to and fro through the fluid which occupies the air cells and smaller bronchial ramifications. If any of the larger bronchi contain fluid, the râles may not only become large and coarse, but, if the surrounding lung tissue remains condensed, may even give rise to the impression of gurgling.

As resolution proceeds, the respiration ceases to be tubular, and becomes at first more diffused,

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Third stage.— Grey hepatization, or diffuse suppuration of the pulmonary tissue, sometimes termed purulent infiltration. In this stage the colouring matter of the blood disappears, the solid fibrinous exudation is broken up, and is gradually replaced by liquefied exudation matter and true pus. The affected portion of the lung is of a grey or dirty grey colour, or mottled, from admixture of black pigment or red colouring matter; it is dense and impermeable to air, sinking instantly in water, and is soft and rotten, breaking down easily into a yellowish-grey pulpy mass. When an incision is made into it, its whole texture is seen to be infiltrated with pus; puriform matter, unmixed with air, oozes from every divided bronchus, aud from almost every air cell, and emits a faint, disagreeable odour; and when the liquid matter has been removed by washing, the granular texture, so characteristic

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then simply coarse and blowing, and ultimately natural; the vocal resonance becomes less intense, and loses its *quasi* ægophonic character; and the percussion note, which at first is dull, assumes by degrees its normal character. Indeed, the clearness and resonance of the percussion note returns more quickly than it does after pleurisy.

Third stage.—The physical signs in the early part of this stage of the disease resemble those already described as belonging to the preceding stage; in short, they are simply the signs of consolidation. It has been questioned whether resolution can take place when the lung has reached the stage of grey hepatization, and it is manifestly impossible, from the nature of the case, to adduce conclusive evidence of the fact. Be this as it may, it is certain, that in the event of its occurrence, it would be accompanied by signs very closely resembling those observed during resolution in the second stage of the disease, and differing from them only in the larger size of the râles.

When a cavity is in process of formation, whether as the result of simple abscess or of circumscribed gangrene, there is little or no physical indication of the fact. The percussion sound, of course, is dull if the abscess is seated super-

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of the second stage of the disease, is seen to have almost disappeared; here and there, however, it still exists partially, the granulations being of a grey colour.

Sometimes, though rarely, a circumscribed abscess results from pneumonic inflammation, so rarely, however, that when Laennec published the second edition of his work, he had met with only six cases; and, amongst the cases which proved fatal in St. George's Hospital during the ten years ending December 31st, 1850, twenty-two instances only of pulmonary abscess were met with, including cases of secondary inflammation. When it does occur,* it forms an irregular-shaped cavity, with ragged walls, containing puriform matter, and surrounded by rotten lung tissue, infiltrated with pus. When it has discharged its contents it may collapse and cicatrize, and thus, if the portion of lung involved be large, it may lead to retraction of the chest walls over the collapsed parts.

Another occasional result of pneumonic inflammation is gangrene, either circumscribed or diffused—most commonly circum-

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ficially, and respiration is almost, if not quite inaudible over the part; but there is no positive evidence of the mischief which is going on. It is otherwise, however, as soon as the abscess has formed a communication with the bronchi, and part of its contents have been evacuated. There are then, superadded to the ordinary signs of consolidation, the signs resulting from the presence of a cavity, and the admixture in it of air and fluid. The percussion sound may then be dull or of a shallow amphoric character, according to the size and position of the cavity, the condition of its walls, and the amount of air which it contains; respiration of a hollow character may or may not be audible, and may or may not be accompanied by large-sized bubbling or gurgling, (when this occurs it is usually of a metallic quality), and the vocal resonance, though varying with the freedom of communication with the bronchi, is generally much augmented, and may even attain a pectoriloquous character.

If the cavity be large and seated superficially, retraction of the

* M. Grisolle is of opinion that the formation of pneumonic abscess is connected with a debilitated constitution. He says (loc. cit., p. 332)—"Sur seize individus qui ont succombé avec des abcès pulmonaires primitifs et pour lesquels on a tenu compté de l'état constitutionel je n'en trouve que deux ayant les attributs d'une bonne constitution."

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scribed and very limited in extent. Its occurrence, however, in connection with pneumonia is even more rare than ordinary abscess. I have only met with it on three occasions, and it was not once noted amongst the 305 cases of pneumonia referred to by M. Grisolle.* Indeed it is doubtful, as Laennec suggests, whether, when it does occur, it is not attributable to some constitutional taint, some peculiarly distempered condition of the blood, and not to the violence of the pneumonic inflammation. The pulmonary arteries leading to the gangrenous parts are often filled with coagula. The gangrenous portion is of a greenish brown colour, and is sometimes soft and wet, more often diffluent, and emits an intensely fetid odour. Even during life, the abominable stench is characteristic of gangrene. It results in the gradual liquefaction and elimination of the dead matter, and the consequent formation of a cavity which may collapse and cicatrise like an ordinary pneumonic abscess.

In some instances pneumonia terminates in induration of the lung: the exudation matter, instead of undergoing the process of softening, hardens, and subsequently contracts; rendering the

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chest walls over the affected part will be perceptible after a time; but, under no other condition, does retraction of the thoracic parietes occur during the acute stage of pneumonia. The experience of Messrs. Grisolle, Woollez, and others coincide with mine in this particular. Therefore, as, except under the circumstances referred to, retraction has never been observed in any instance in which a *post-mortem* examination has proved the absence of pleuritic effusion, it is fair to conclude that, in the cases of retraction of the chest observed by Drs. Stokes and Walshe during the progress of supposed uncomplicated pneumonia, pleuritic complications must have coexisted.

Gangrene gives rise to the physical signs which accompany abscess of the lung; and, physically, is not distinguishable from ordinary softening and excavation of the lung tissue.

Induration of the lung is attended by diminution of the costal movements, by dulness on percussion, by the absence of vesicular, and the presence of hollow-sounding tubular breathing, and by increase of vocal resonance. Ordinarily there is absence of râles and rhonchi, but if bronchitis or œdema of the lung occurs, these sounds,

* Grisolle, loc. cit., p. 345.

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lung tissue tough and impervious to air, and the bulk of the lung smaller than natural. On two occasions I have met with cases in which the physical signs of pulmonary consolidation have been very persistent in sequel of pneumonia, and in which a *post-mortem* examination has shown simple consolidation and contraction of a lung to have occurred without pleuritic inflammation. Such cases, however, are extremely rare; and, in some of the instances referred to by authors as lungs contracted by pneumonia, I believe the lungs to have been collapsed, and in others to have been compressed by pleuritic effusion.

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of course, exist; and if the air tubes become clogged by secretion, there may be absence, rather than increase, of vocal resonance. After a time, as contaction of the indurated lung takes place, retraction of the chest walls occurs over the affected parts.

Drs. Stokes, Walshe, and some other authorities maintain that intense arterial injection of the lung exists in pneumonia prior to the stage of engorgement—a condition characterised, after death, by "dryness and bright vermilion colour," and distinguishable, during life, by the occurrence of respiration which is harsher, rougher, and of higher pitch than natural. My opinion, however, accords with that of Skoda, who is opposed to this supposition. Theoretically, I am unable to conceive how mere arterial injection and dryness of the pulmonary membrane can produce the signs enumerated as belonging to this condition; and practically, my observations incline me to assert with M. Grisolle, that "weakness of the respiratory murmur, attended by loss of purity and softness," usually characterises the commencement of the disease. Indeed, this is only what might have been expected from the dryness of the pulmonary membrane and the diminution of the size of the ultimate bronchial ramifications, consequent on its tumescence. Further, it is inconsistent with the result of my observations to suppose that the bright vermilion colour of the lung sometimes observed in pneumonia, is attributable specially to pneumonic injection of its tissue;

for it is not always met with in pneumonia, and sometimes it is observed in fatal cases of anæmia, and in other diseases which, during life, had not been accompanied by symptoms of pulmonary mischief. Rokitansky and others attribute it to anæmia, and it certainly does occur most frequently in cases which have been characterised during life by anæmic symptoms. Assuredly it is not the result of pneumonia, and cannot be adduced as evidence of its existence; neither is it productive of any physical signs or accompanied by any special general symptoms on which to base a line of treatment.

The existence of fine crepitation in the lung is often spoken of as pathognomonic of acute pneumonia; but it ought to be understood that this sign if viewed alone does not afford a safe criterion as to the presence of the disease in question. In some instances it is absent throughout the attack; and in many more a crepitant râle is heard in the earlier stages of the disorder, which is undistinguishable from that which accompanies capillary bronchitis. True, the crepitation which is met with in pneumonia is generally more abundant, more rapidly evolved, and is finer and of a drier character than that of capillary bronchitis; true, also, that it is commonly confined to inspiration, and is heard chiefly towards its close. But it is equally true that these characters are by no means constant, and *post-mortem* investigations enable me to testify that whilst, on the one hand, the fine bubbling râle of capillary bronchitis which accompanies expiration as well as inspiration, is met with in cases which speedily result in pneumonic consolidation of the lung tissue; so, on the other, a fine dry crepitating râle which is confined or nearly so to inspiration, and is undistinguishable by its mere character from the crepitation of pneumonia, may long persist without being accompanied by pneumonic consolidation. In short, it is only by the concurrence of different signs that it is possible to arrive at a trustworthy conclusion. Fine crepitation, occurring coincidently with intense heat of skin and rusty coloured expectoration, warrants the strongest suspicion of pneumonia, and justifies the adoption of active treatment; and if it be accompanied by marked alteration in the ratio of the pulse and respiration, and is speedily followed by dulness on percussion, and tubular breathing, the existence of acute pneumonia cannot be doubted. But crepitation, however fine, if not attended by an alteration in the ratio naturally subsisting between the pulse and the respiration, and not speedily followed by tubular breathing, cannot be relied upon as indicative of pneumonia. If it occurs without these symptoms, it is commonly indicative of capillary bronchitis with scanty

secretion; whereas, if under the same conditions it is accompanied by dulness on percussion, it is probably due to rapid œdema, or else to congestion of the lungs connected with some febrile hæmic disorder, cardiac disease, or the deposit of tubercle, all of which may produce an outpouring of non-plastic fluid into the air vesicles and terminal bronchi.

The seat of the disease will sometimes assist in determining the question; for whereas both lungs are prone to be affected in capillary bronchitis, and in passive congestion resulting from heart disease or hæmic disturbance, pneumonia is very apt to be confined to the right lung, or, at all events, to one lung. Thus, of 139 cases which I have noted with a view to this inquiry, 77 were instances in which the right lung only was affected; 51 in which the left lung only suffered; and 11 in which both lungs were more or less implicated. An analysis of 1,710 cases recorded by different authors, affords strong support to this observation; for in 908 cases the right lung only was inflamed; in 523 cases the left lung only; in 279 cases both lungs were attacked.* And when it is stated further, that of the entire number of cases of double pneumonia a large proportion commenced as cases of single pneumonia, the symptoms having existed for many days in one lung before they made their appearance in the other, it will be evident that the mere topography of the disease will often serve to decide any doubt which may exist as to the real import of a crepitation.

Some authors have asserted that, at the outset of the attack, pneumonia is usually confined to the base of one lung; and have attempted from the precise position of the crepitation, to draw an inference as to the real nature of the disease in doubtful cases. But careful and extended observation has shown that no one part of the lung is specially prone to suffer; that the proportion of cases in which the base is inflamed is only about as four to three in relation to those in which the upper part is affected, and that in a considerable number of cases the middle part of the lung is the first seat of invasion.† Further, it has been ascertained that in certain epidemics the upper lobe is more apt to be attacked than the lower ;‡ as if the precise seat of the disease is de-

* See Grisolle, loc. cit., p. 28.

† M. Grisolle gives the following statement relative to 264 cases of pneumonia which fell under his own observation, viz., that in 133 cases the lower lobes were affected, the upper in 101 cases, and the middle third of the lung in 30 cases (loc. cit., p. 34). M. Andral states that in 88 cases which he examined, the lower lobe was inflamed in 47 instances, the upper lobe in 30 instances, and the entire lung in 11 cases ('Clin. Med.,' vol. iii, p. 470).

‡ See Stokes on 'Disease of the Chest,' p. 319.

termined, not so much by the predisposition of particular portions of the lung to this special form of inflammation, as by the tendency of the materies morbi, under certain atmospheric or epidemic influences, to excite irritation in particular portions of the respiratory apparatus. However this may be, it is certain that the precise position which the mischief occupies in the lung is not a trustworthy criterion as to the nature of the mischief; and perhaps the only fact which can be definitely announced in reference to this subject is, that when the upper portion of the lung is the primary seat of pneumonia, the disease will be found, in the great majority of cases, on the right side.*

Pneumonia is more prevalent in winter than in summer,† but, relatively to the number of persons attacked, it proves little more fatal at one season than at another.‡ Indeed the danger of its attack varies with the intensity of the prevalent epidemic influence more than with the season or state of the atmosphere. At one time a large proportion of attacks prove fatal, whilst at another, even under the same treatment, and though an equally large extent of lung be implicated, death is the exception rather than the rule. *Cæteris paribus*, the danger is proportioned to the age of the patient, the extent of lung affected, and the stage of the disease at which the treatment is commenced; and in this as in other serious disorders the danger varies with the habits of life and constitutional powers of the individual. Habitual drunkards, and those who have indulged in excesses of any kind, are most exposed to attacks of the disease, least able to resist its depressing influence, and less capable of bearing the remedies which are necessary for its

* Thus M. Barth reports that in 19 cases in which he met with pneumonia of the upper lobe, the inflammation was confined to the right side in no less than 18 instances; and in like manner M. Briquet states that in 18 observations which he had made in similar cases inflammation was found affecting the right side only in 14 cases ('Archives de Médecine,' 3me série, t. vii, p. 494).

† See reports of Registrar-General; also 'Dict. de Médecine,' art. "Pneumonie," par M. Chomel. M. Grisolle (loc. cit., p. 137) gives the following analysis of 296 cases, viz.:

20	occurred in	January.	13	occurred in	July.
40	„	February.	3	„	August.
47	„	March.	5	„	September.
62	„	April.	2	„	October.
40	„	May.	22	„	November.
8	„	June.	34	„	December.

‡ Grisolle, loc. cit., p. 529-30.

subjugation. In early infancy and extreme old age the attacks generally prove fatal; between the ages of six and twelve, death very rarely occurs; between the ages of sixteen and thirty, the mortality is from 6 to 9 per cent., whilst at other periods of life the mortality ranges between one fifth and one seventh of those attacked.* Again, those patients who submit themselves to medical treatment within the first three days of the attack recover in the proportion of twelve to one; whereas amongst those who are not treated until the fourth day, the mortality rises to one in eight; and amongst those who are not seen until the seventh day or afterwards, to one in three, or even to one half.†

In my own practice the mortality has not ranged so high as the statistics just quoted would have led one to anticipate; for although patients are admitted into the hospital, of all ages, and in all stages of the complaint, I have lost only 4 out of 53 cases, and of these 4, 1 died the day after admission. Whether this favourable result is attributable to my supporting the patient and avoiding depressing treatment, it is difficult at present to decide, but, inasmuch as the statistics from which the more unfavourable results are deduced relate to cases in which

* M. Valleix reports, as the result of his observation at the "Hospice des Enfans trouvés" at Paris, that pneumonia is almost certainly fatal to new-born children; and Messrs. Rufz and Gerhard state that of 27 cases of pneumonia occurring in children between the ages of two and five years, no less than 25 proved fatal. ('Journal des Conn. Méd.-Chir.,' t. iii, p. 105.) Between the ages of six and fifteen, the same observers met with only 1 death in 80 cases; and of 116 cases occurring in persons between the ages of sixteen and thirty, 8 only had a fatal termination. Between the ages of thirty and sixty the mortality rises with each decade from one seventh to one fifth, and beyond that age the mortality is much greater; of 129 cases of pneumonia occurring in persons between the ages of sixty and ninety, no less than 77, or about three fifths, proved fatal. (See Grisolle, loc. cit., p. 518-20.) M. J. J. Leroux in his 'Cours sur les Généralités de la Médecine,' tom. vi, gives the following account of 364 cases of pneumonia:

Age.	Patients attacked.	Deaths.	Per cent.
13 to 30 ...	182 ...	17 ...	9·3
30 to 40 ...	58 ...	15 ...	25·9
40 to 50 ...	47 ...	16 ...	34·0
50 to 60 ...	55 ...	23 ...	41·8
60 to 70 ...	16 ...	9 ...	56·2
70 to 75 ...	6 ...	5 ...	83·3
	364	85	

† See Grisolle, loc. cit., p. 551.

copious venesection and other actively depressing treatment was employed, it seems not improbable that such may be the case.*

In fatal cases, death commonly occurs between the sixth and twenty-first day of the attack, the cases being quite exceptional in which the patient does not recover if he outlive the end of the third week. Further, true relapse is rare, so much so indeed, that amongst the ninety-four cases which were noted in St. George's Hospital during the period of my registrarship, there was only one well-marked example of it; and if the term be restricted to cases characterised by a recurrence of rigors, and of rusty coloured expectoration, I question whether the proportion is ever larger than 3·57 per cent., as recorded by M. Grisolle.†

Pneumonia is rarely followed by tuberculization of the lung, and even when set up in a lung already partially infiltrated with tubercle, it does not usually lead to softening of the tuberculous matter, or to any material extension of the mischief. In this respect it contrasts remarkably with bronchitis, and establishes the fact of its being physically as well as pathologically a totally distinct disorder.

The treatment of pneumonia has varied greatly at different epochs, and even now is a subject on which most discordant opinions are expressed. Venesection was formerly a very favourite remedy; and up to the time when Rasori and Laennec had recourse to the aid of tartarized antimony, it was the physician's sheet anchor in the treatment of this disease. The common practice, within the memory of living men, was to take from twenty-four to thirty-six ounces of blood at the very outset of the attack, and to repeat the bloodletting to sixteen or twenty ounces, once, twice, or even three times; and, strange as it may seem to those who have the management of cases in the present day, marked benefit appears, in some instances, to have resulted from this treatment.‡ Laennec, however, found that in his day patients could not bear the loss of so much blood; and, at the present time, there are those who maintain not only that venesection is inadmissible, but that brandy and stimulants are needed throughout the attack. Certain it is that the

* This opinion is countenanced by a paper in the 'British Med. Journ.' for August 23, 1862, by Dr. J. Hughes Bennett, in which he reports having treated 105 cases of pneumonia after much the same plan as I usually adopt, but with even greater success. He lost only 3 out of his 105 patients.

† Grisolle, loc. cit., p. 456.

‡ Grisolle, loc. cit., pp. 574-596.

"medical constitution" must have undergone some change since the days of excessive bloodletting; for the type of disease for some years past has been of a low character, and patients cannot bear great loss of blood, and do not require it for the subjugation of inflammation. But careful observation in the wards of St. George's Hospital has led me to believe that, in some cases at least, moderate venesection is of essential service in relieving the pain and modifying the severity of the attack. In cases of sthenic pneumonia characterised by intense heat and dryness of the skin, a full, resistant pulse, rusty coloured expectoration, and great oppression of the breathing, bloodletting, had recourse to at the beginning of the attack, not only affords immediate relief to the breathing, but appears to remove the extreme tension of the system, and to promote secretion. After ten or twelve, or sixteen ounces of blood have been taken, the pain in many instances ceases, expectoration takes place more easily, and alters in character; the skin becomes moister, and evidence is afforded of the action of remedies which before proved inoperative. Moreover, the effect is so well marked and immediate, that no reasonable doubt can exist as to how it is brought about. But, I am bound to say that the cases in which I have judged it expedient to employ phlebotomy are very few in number. The patient's constitution is often shattered by anxiety, excesses, or other causes; the epidemic influence is often of a low type, and, from one cause or another, cases of true sthenic pneumonia are rarely met with. In other cases, venesection from the arm is inadmissible, and bloodletting, if practised at all, must be confined to leeching and cupping.

It has been attempted by means of numerical returns to establish the necessity for bloodletting in all cases of pneumonia; and, even in the present day, not a few persons maintain that venesection always shortens the duration of the disease, mitigates its severity, and diminishes the mortality resulting from it. There are others who speak with equal confidence as to its being unnecessary in any case, and who adduce statistics in support of their view. But in this, as in most other cases where statistics are appealed to in proof of the efficacy of any particular plan of treatment, sufficient care is seldom taken to guard against the various sources of error. Thus it happens that the conclusions drawn from this source usually prove fallacious. And so it is in the present instance. Putting aside considerations connected with each patient's constitution, and with the great variety in the character of the epidemic influence in different years, and at different seasons and

in different localities, the facts already mentioned (p. 232), relative to the variations in the mortality according as the disease occurs at different ages, and is or is not early combated by treatment, nullify the result of all statistical observations from which a due regard to their influence is excluded. And when it is remembered that physicians living at the same time, and observing the disease under the same atmospheric and other conditions, have arrived at precisely opposite conclusions from the same numerical method of calculation, it needs not an elaborate argument to prove how little reliance is to be placed on the results. Common sense points out that what is serviceable in one case will prove mischievous in another, under different conditions of age, sex, constitution and the like; and experience not only endorses this view, but proves that under certain circumstances bloodletting is a palliative of extreme value. It also shows that it is unnecessary to let blood in most cases, and that excessive bloodletting, under whatever circumstances practised, impairs the strength, leads to great impoverishment of the blood, arrests the actions on which the absorption of exudation matter depends, and not only exposes the patient to risk, but even, under the most favourable conditions, induces a tardy convalescence. It is needless, therefore, to add that phlebotomy should not be resorted to, except when its employment is most clearly indicated, and that even then it should be employed very cautiously. In my own practice at St. George's Hospital and elsewhere, I have had recourse to it in pneumonia three times only within the last four years, but I am satisfied, nevertheless, that in each of those cases it was the means of affording great relief, and that cases do occur from time to time in which its employment is absolutely necessary to the wellbeing of the patient.

It is impossible to lay down a general rule as to the amount of blood which may be safely taken, or, indeed, to dogmatise in the slightest degree respecting the use of venesection in pneumonia. All that can be confidently stated is, that the indications for bloodletting should be strongly marked before it is undertaken at all; that the amount of blood to be lost must vary in each case, according to the type of the disorder and the constitution of the patient, but that at the present day it is seldom if ever necessary to draw more than from ten to sixteen ounces, and that eight or ten ounces will usually suffice; that bleeding, if resorted to at all, should be practised early, inasmuch as it may relieve the local congestion by which the early stage of the disease

is accompanied, but cannot remove, and may even cause deterioration of the matters which are effused in the subsequent stage of the disorder;* that it is seldom necessary to draw blood from the arm oftener than once, and lastly, that advanced age or the existence of pregnancy or menstruation need form no bar to its employment when the general symptoms demand its aid.

It is right to add, that whenever local pain exists, and bleeding appears necessary, cupping or leeching may be employed in addition to general venesection, inasmuch as the local abstraction of blood relieves pain more rapidly than general bloodletting, and may even cause its immediate cessation. In mild attacks, or in low type of the disease, and in weakened constitutions, it should supersede all other forms of bloodletting.

To Rasori and Laennec we are indebted for the introduction of the plan of treatment which, next to that of venesection, has obtained the greatest reputation in pneumonia—I mean the administration of large and repeated doses of tartarized antimony. Rasori, commencing with grain doses, gave as much as twenty-four or twenty-six grains in the twenty-four hours, and trusted solely to its action for effecting a cure; whilst Laennec, though following Rasori's example in giving the remedy in large and repeated doses, conceived it to be advisable to resort to moderate venesection in aid of the antimonial. He, therefore, commenced by a bleeding from the arm of from eight to sixteen ounces, Others have followed the same plan of treatment; and although nausea, vomiting, diarrhœa, and griping pain in the abdomen have sometimes resulted from the exhibition of the antimony, and in some few instances death has appeared to be occasioned by it, yet, ordinarily, it has ceased to produce vomiting or purging after the first day, and has been tolerated as perfectly as when given in smaller doses. But the amount of success resulting from the treatment has not been uniformly great. At first Laennec reported of it most favourably, but subsequently, under different atmospheric or other conditions, it failed to exert any remarkably curative action, and the proportion of deaths rose as high as that attained under other methods of treatment. Thus, of 46 patients treated by Laennec 17 died; and of 140 cases treated by Chomel 40 proved fatal. Further, Andral, who carefully watched this plan of treatment,

* It has been clearly shown by Louis and others that recovery takes place sooner when bloodletting is employed within the first four days than when it is resorted to later in the attack.

and tested it largely, expresses his doubts as to whether the disease is ever benefited by these large doses of tartarised antimony.

My own opinion is quite in accordance with the doubts expressed by Andral. In our own country, and within my time, tartar emetic has been seldom given in the doses introduced by Rasori, but I have seen quite enough of its action in the doses recommended by Dr. Walshe and others, to induce me to hesitate in recommending their administration.* More than once in hospital practice I have seen extremely dangerous depression, with profuse cold clammy sweats, produced by its exhibition in smaller doses than those just alluded to, and even when such doses have failed to occasion unpleasant symptoms, they have not appeared to exercise a more decidedly curative influence than smaller doses exhibited at longer intervals. Therefore, in my own practice I have seldom given more than a quarter of a grain at intervals of three or four hours, according to the circumstances of the case, and always combine it with saline and alkaline medicine. In such doses the remedy does not nauseate after the first time of its administration, but operates very manifestly in mitigating the severity of the symptoms. Indeed, an instance rarely occurs in which its administration is not called for at the outset of the attack, and it may be continued either alone or in combination with other remedies, until relief is obtained. Exceptional cases are sometimes met with in which the character of the pulse, on the occurrence of profuse clammy perspiration, speedily indicates serious depression of the vital powers, and in these, of course, its administration must be discontinued. Under these circumstances, salines and diffusible stimulants internally, and turpentine fomentations and blisters externally, are the appropriate remedies. Speaking generally, however, it may be fairly stated that in pneumonia no single remedy is so useful as tartar emetic, and that, administered judiciously, with due regard to the exigencies of the case, it modifies and represses the morbid action in the lungs, and rarely produces unpleasant symptoms.

In some instances, however, tartar emetic either fails in exerting a curative action, or is not well borne by the system. Cases in which

* Dr. Walshe (loc. cit., p. 426) says—"The salt should at first be given in doses of half a grain combined with dilute hydrocyanic acid, paregoric, and tincture of orange peel every hour for the first three or four hours, and the dose then increased at intervals of two hours to one grain; in the course of twelve hours the quantity may be raised to two grains, its repetition made less frequent, say every fourth hour."

hepatization proceeds with extreme rapidity, in which crepitation either does not exist at all, or is of very short duration, giving place after a few hours to intense tubular breathing, and those again, which are marked by extreme depression almost from the first, are rarely benefited by tartar emetic. The former often accompany the rheumatic diathesis, and are seen most strongly marked in connection with acute rheumatism. The latter are met with in cachectic persons, and especially under peculiar atmospheric conditions. The most efficient treatment in the one class of cases consists of calomel in repeated doses, pushed even to salivation, together with full doses of alkalies and the neutral salts. The latter is to be combated, as already stated, by salines and stimulants, with calomel and opium, if necessary. In either case the external application of turpentine fomentations is useful throughout, and blisters assist in relieving the local pain, and in promoting absorption of the plastic exudation as soon as the first fury of the attack is overpast; but if employed at the very outset of the disease, they appear to increase the feverish excitement, and are productive of evil rather than of good.

The action of mercury in pneumonia is a question on which opposite opinions have been entertained. There are persons who deny that it ever exerts a beneficial influence; and there are those, on the other hand, who laud it as the most potent remedy we possess when the disease has reached the stage of hepatization. My own opinion does not accord with either of these dogmas. Observation at the bedside has fully convinced me of the efficacy of tartarised antimony in many cases of pneumonia, long after hepatization has commenced; but it has equally satisfied me of the superiority of mercury in certain forms of the disease. Indeed, if my views are correct, the efficacy of mercury is especially conspicuous in those cases of pneumonia in which tartar emetic is of least avail; in other words, in those instances of the disease which are accompanied by a more than ordinarily plastic exudation—cases in which, as already stated, crepitation either does not exist at all, or is replaced, in a few hours, by intense tubular breathing. In these, calomel and opium, in combination with salines and small doses of tartar emetic will often produce results which are not attainable by any other means.

Of late years it has been proposed to treat pneumonia by the exhibition of small and repeated doses of alcoholic stimulants internally, whilst turpentine stupes are being applied externally; and reports

have been made from time to time in the journals, of cases so treated by Dr. Todd and others. But I suspect that these reports are calculated to convey an erroneous impression as to the result of such treatment in ordinary cases of pneumonia. They speak of the cases as being examples of ordinary sthenic pneumonia, and of the treatment—which consists essentially of half-ounce doses of brandy at intervals of an hour—as being eminently successful. My own observation of this method of treatment has been too limited to justify my expressing a positive opinion as to its efficacy; but I may state, that in four out of the only five cases in which I have seen it tried, it failed very signally in sustaining its reputation. Not only did it not afford relief, but in each instance produced aggravation of the symptoms, and was discontinued in consequence. And when it is remembered, in connection with this fact, that a large proportion of the cases in which the alcoholic treatment has been successfully employed have ranged between the ages of fifteen and thirty—a period of life during which as already shown, the mortality from pneumonia, under whatever treatment, is at its *minimum*—the inference I think may be fairly drawn, that the success which has been attributed to the action of the so-called remedy has been really due to other causes. In these, as in other disorders, stimulants are often needed in aid of other remedies; but from what I have seen, and from what I have learned from the testimony of others, I am satisfied, that now, as heretofore, their action, either for good or evil, in each particular case, depends on whether they are needed by the system, and are administered judiciously or injudiciously. It should be added, however, that the necessity for stimulants in certain cases, and at certain stages of the disease, as it has prevailed of late years in London and other large towns, is more apparent, and more frequently arises, than the experience of former years would have led us to expect.

In pneumonia, as in other acute diseases, the secretions must be regulated, and moderately antiphlogistic regimen enforced. Excessive purgation is worse than useless, as tending needlessly to exhaust the strength; but gentle laxatives should be employed when necessary, and diuretics, such as nitre and acetate of potash, may be given advantageously in aid of remedies to promote a free secretion of urine.

There are some varieties of pneumonia which demand special notice, not only as differing from ordinary cases of the disease in their symptoms and progress, but also as requiring different treatment,

and leading to different pathological results. Amongst these may be mentioned—

1st. The cases already alluded to as characterised by inflammation of the interlobular cellular tissue—cases which are especially apt to occur in rheumatic persons. These are always difficult of diagnosis, and are apt to be confounded with pleurisy. They are to be distinguished from ordinary pneumonia by the comparative absence of crepitation, and by the fact that the crepitation which exists is heard during expiration as well as during inspiration; and from pleurisy, by the rapidity with which strongly marked tubular breathing ensues, and continues, day after day, unimpaired in intensity, by the increase of the vocal fremitus, and by the increased resonance of the voice. Sometimes the inflammation is of a suppurative character, but more commonly the inflammatory exudation is fibrinous; and as the disease becomes chronic, the fibrin gradually solidifies and contracts, and produces the condition of lung described as "cirrhosis," by Dr. Corrigan. As the disease progresses the bronchi undergo dilatation, the pulmonary tissue is compressed and becomes impermeable, the lung contracts, and the side of the chest falls in. As a result of this condition, dyspnœa occurs, the costal movements are diminished, the percussion note becomes dull or else somewhat amphoric, the vocal fremitus is increased, the vocal resonance is abnormally great, the respiration is of a hollow, blowing character, and if bronchitis or œdema exist, is accompanied by sonorous rhonchi and large bubbling râles.

In the acute stage of the disease, I believe nothing is of more service than leeches and blisters externally; and, as internal remedies, calomel and opium, pushed to ptyalism, with full doses of alkalies and the neutral salts, iodide of potassium, and occasional doses of colchicum. In the more advanced stages, when the exudation matter has solidified, and has already produced compression of the lung, I do not believe that any medicine will serve to remedy the mischief which has occurred; and our chief aim should be to sustain the health, and promote free and easy expectoration, if bronchitis happens to coexist.

2ndly. Another class of cases deserving of notice is that in which inflammation, instead of spreading through an entire lobe of the lung, is confined to certain lobules, the intervening lobules remaining sound. This form of disease, which has been termed "lobular pneumonia," was formerly considered to be of frequent occurrence in infancy, and even now is so spoken of by some writers. But careful research has

shown, that in many cases the solidification of the pulmonary lobules, which was formerly attributed to pneumonia, is due, when occurring in infancy, to simple collapse of the lung. This is proved by the fact that after death, in such cases, the solidified lobules may be inflated almost to their natural size; whereas hepatized lobules do not admit of inflation. Therefore, although, as already stated, this form of pneumonia is frequently met with in infancy, it does not occur so often as was formerly supposed. In adult life it is observed occasionally as an idiopathic affection; but more commonly it arises in connection with pyæmia, and the formation of secondary abscesses in the lungs.

The physical diagnosis of this form of disease is extremely difficult. Inspection, palpation, and mensuration afford only negative information, and percussion fails to give any assistance, so long as mischief is confined to two or three lobules. Indeed, the only physical indication of the disease is to be found in the respiration, which is exaggerated in the parts adjacent to the consolidated lobules, and sometimes absent, sometimes harsh and blowing, immediately over the seat of consolidation.

3rdly. Cases in which the disease is of secondary origin, or, in other words, arises during the progress of other forms of disease. This is a most common and most dangerous form of the malady, and is apt to supervene at all ages, from earliest infancy to extreme old age. It is the principal cause of death in many cases of measles, hooping-cough, and other infantile disorders, it is apt to complicate typhus fever, rheumatic fever, variola, and other acute blood disorders, as also meningitis, and other inflammatory diseases in the adult; and it is a frequent attendant upon renal anasarca and upon phthisis pulmonalis, cancer, and other chronic disorders, connected with local organic mischief and a distempered condition of the blood.

In all these cases the physical signs are much the same as are met with in ordinary idiopathic pneumonia, except, perhaps, that in the more adynamic forms of disease the exudation is more serous, and less plastic in its nature; and that as it is more profuse, and not so readily got rid of by expectoration as in ordinary cases of pneumonia, there is a tendency to obstruction of the air passages, with entire absence of breathing, rather than to tubular breathing, over the affected portions of the lung. In a practical point of view these cases differ altogether from those of ordinary pneumonia. They partake, in many instances, more largely of congestion than of true inflammation; and the mischief, whatever it may be, is dependent on and kept up by the irritation of the *materies morbi*

out of which the primary disease arises. Hence, *inter se*, they differ greatly in character, and are more purely congestive, or more strictly inflammatory, according to the precise nature of disorder from which they take their origin. In all cases, however, "sublatâ causâ tollitur effectus," and the only rational treatment is that which endeavours to subdue the primary disease, whilst at the same time it aims at counteracting the secondary local mischief, by dry cupping, blisters, turpentine fomentations, and other topical applications. In the pneumonia of fever, brandy and quinine may be the appropriate internal remedies; alkalies in that of rheumatism; soda, colchicum, and magnesia in that of gout; compound jalap powder, bitartrate of potash, gin, and other diuretics in that of Bright's disease. I do not wish to imply by this statement that calomel and antimony are never needed in these cases; but I do mean that they are often useless, or worse than useless; and even when required for the subjugation of local inflammation should be kept subservient to the general treatment which is directed against the original disorder. I have seen too much of the mischief produced by an opposite course of treatment to doubt as to the correctness of this general rule. Indeed, it may be stated generally, that the only safe and efficient treatment is that which is calculated to mitigate or remove the blood disorder, which is the source of all the mischief, aided by such external applications as serve to mitigate local irritation or to counteract internal inflammation without unduly depressing the patient.

4thly. Cases in which the disease, though running on rapidly to consolidation, is unattended by the ordinary, general symptoms of pneumonia, and is, therefore, said to be "*latent*." Their peculiarity is simply that which their name implies and which renders the mischief very likely to be overlooked. They are met with chiefly in infants,* or else in persons advanced in years, or suffering from adynamic disorders, and the mischief partakes of the character of congestion resulting in serous effusion rather than of active inflammation. These cases constitute a large proportion of those described by Piorry under the title of "hypostatic pneumonia." Great perversion of the ratio naturally subsisting between the pulse and the respiration ought always to excite suspicion of the disease; and when once this is aroused, a physical ex-

* M. Guersent, Physician to the Hospital des Enfans, says that "three fifths of the children who die in the hospitals between birth and the conclusion of the first dentition die of pneumonia in a latent form," 'Dict. de Médecine,' vol. viii, p. 92.

amination of the chest will serve to reveal the nature and extent of the mischief; for although the general symptoms are absent, the physical signs of pulmonary consolidation are fairly marked, and differ little in their nature from those which accompany ordinary cases of pneumonia.

The treatment of this form of disease consists chiefly of measures calculated to relieve the local congestion, and in many instances, from the nature of the case, these take the form of diffusible stimulants internally and counter-irritants and derivates externally. Dry cupping is always useful, and turpentine fomentations are of great service, whereas venesection, leeches, and tartarised antimony are seldom admissible, and are generally productive of serious mischief.

5thly. Cases in which pneumonia occurs in a chronic form. This variety of disease, when unaccompanied by the deposit of tubercle or other adventitious product, in the lung is extremely rare. It is met with in two distinct forms, the one occurring most commonly as a sequel of acute pneumonia, the other as a chronic affection unattended by symptoms of the acute disease. The first is characterised by infiltration of the lung tissue, with a tough, greyish, solid matter which does not exhibit a tendency to soften, but leads gradually to contraction and shrinking of the lung and retraction of the chest walls. The lung in this case is drier and harder than in a corresponding state of ordinary hepatization, is smooth when cut, and often creaks under the scalpel. In the second variety the lung is infiltrated by a reddish, solid, but less plastic material, which after a time becomes distinctly friable and breaks up into a granular detritus. The lung in these cases, as in those last mentioned, is firmer and drier than in hepatization, and scarcely yields a trace of fluid when scraped, but presents a markedly granular surface when cut. When it is torn, the granulations are still more strikingly manifest. These cases form a connecting link between pure chronic inflammatory consolidation and tuberculous infiltration.*

The general symptoms are those of failing health and strength, with slight but habitual dyspnœa, cough, and scanty catarrhal expectoration—unaccompanied by hæmoptysis—and oppression—not pain—at the chest. If the disease supervenes on an attack of acute pneumonia, the patient does not regain his strength, but, on the contrary, becomes weaker and loses flesh; his pulse remains quick, the appetite fails, and he is troubled with feverishness, especially towards evening.

The physical signs are somewhat deceptive. The expansile move-

* See Andral 'Clin. Med.,' t. ii, p. 310.

ment of the chest is always impaired, and there is dulness on percussion; but the other signs are by no means constant. The chest walls may fall in in consequence of the shrinking of the affected lung, and this condition may be accompanied by harsh, blowing, and more or less hollow sounding respiration in some parts of the chest, whilst in others the breathing is weak, or altogether absent, or accompanied by occasional râles; the vocal resonance is increased, and the vocal fremitus intensified. Sometimes, however, if the earlier stage of the complaint has been overlooked, all these signs are wanting; the lung, as yet, has not contracted, and, consequently, there is no falling in or retraction of the chest walls; the bronchi are obstructed, so that there is entire absence of breathing sounds, whether healthy or morbid; the voice is not transmitted through the chest walls to the ear, and vocal fremitus cannot be felt. In short, the physical signs more nearly resemble those met with in pleurisy than those which are ordinarily attendant on pneumonia. A case of this sort, in which an entire lung was implicated, fell under my observation eleven years ago. The patient a young lady, aged twenty, had long been short-breathed, but otherwise had been apparently in fair health, until within three days of her death. She died of constriction of the larynx and trachea, consequent on the pressure of an enlarged thyroid. The condition of the chest was ascertained by dissection, Mr. Prescott Hewett very kindly performing the *post-mortem* examination. No trace of fluid or of pre-existing inflammation was found in the pleura, but one lung was perfectly solid. It was as large as the other lung when partially inflated, and did not collapse when the chest was opened, but filled the whole of one side of the thorax. It sank at once when placed in water. Its colour was greyish red; its consistence so firm that it creaked under the scapel, and its cut surface was smooth, and emitted scarcely a trace of fluid even when scraped with the knife. It did not contain a particle of tubercule; but the bronchial and mesenteric glands were enlarged, and contained cretaceous matter. The bronchi were filled with exudation matter. An instance in which the lower lobe of the right lung was similarly affected, and gave rise to precisely the same physical signs, was observed by M. Requin.* Such cases are apt to be mistaken for cases of old standing pleuritic effusion; and, in whatever stage they are examined, whether before or after contraction of the lung has taken place, the diagnosis is always difficult. When the upper lobe of the lung

* See Grisolle, loc. cit., pp. 350-1.

is thus affected, the case is apt to simulate consumption; for not unfrequently there is flattening of the infra-clavicular region of the affected side, with imperfect expansion, dulness on percussion, harsh, hollow breathing sound, prolonged expiration, and increased vocal and tussive resonance. The precise character and order of progression of the general symptoms, coupled with the stationary condition of the parts, as determined by several examinations at considerable intervals of time, form the only clue to the real nature of the case; but they will often suffice to excite suspicion in the mind of the careful and experienced physician, and will induce him to express a cautious opinion. Fortunately the diagnosis is not of much practical importance, inasmuch as these cases are rare, and require treatment similar to that which proves useful in tubercular phthisis. Cod-liver oil and tonics internally, daily oleaginous inunction, the occasional use of strong iodurettted and counter-irritant applications to the chest, a generous diet, proper exercise, and fresh air, are the remedies most likely to prove serviceable. In one case which I saw in consultation with Dr. Williams, and which I had the opportunity of watching for three years, this plan of treatment, aided by a twelve months' residence in Egypt, entirely removed all trace of the disease.

Gangrene of the Lung.

In my observations on pneumonia, the occurrence of gangrene of the lung was briefly alluded to, and reference was made to the morbid appearances and physical signs to which it gives rise. Mortification, however, must be regarded as an accidental complication or occasional sequence of pneumonia, rather than as a natural consequence of the disease, for it is rarely met with in sthenic cases, in which pulmonary inflammation is most intense, and sometimes occurs in instances in which pneumonia does not exist.

There are two distinct varieties of gangrene of the lung, viz.—first, the diffused, or uncircumscribed variety; and secondly, the circumscribed. These differ greatly in their anatomical characters, but they both appear to be connected with a depraved condition of the blood analogous to that which produces the various forms of idiopathic gangrene. This, doubtless, in most cases, is their proximate cause, the local inflammation being only the accidental or exciting cause. It

must be admitted, however, that the exciting or determining causes of the disease appear to be of very different force and character in the two varieties, In the diffused form of the disorder, the slightest impediment to the pulmonary circulation suffices to excite its occurrence—nay, in some instances, uncircumscribed gangrene seems to occur without any pre-existent local obstruction; whereas the circumscribed form is rarely met with, unless intense pulmonary inflammation of a low type has existed, or secondary abscesses have formed in the lungs, or obstruction of the nutrient vessels has been brought about by the pressure of an aneurismal tumour,* or of tuberculous, cancerous, or other adventitious deposits, or still more rarely, by the presence of minute fibrinous concretions detached from the endocardium on the right side of the heart, and carried onwards with the blood to the lungs. It seems fair to conclude, therefore, that in the former variety of the disease, the blood is more deteriorated than in the latter—a view which is borne out by the class of cases in which the two varieties of gangrene occur respectively.

Mortification of the lung is extremely rare; so much so indeed, that in the *post-mortem* records in St. George's Hospital for the ten years ending 1850, it was noted in no more than nineteen instances; and I have met with only seven other cases in which its general characters and physical signs were thoroughly well marked during life. It may occur at any age, and appears from M. Boudet's inquiries to be more common in children than in adults, and more so in adults than in persons much advanced in years.† When it does occur, the gangrenous process may be

* A remarkable case of pulmonary gangrene occurring as the result of aneurismal pressure occurred under my care at St. George's Hospital, in January, 1860. For full details see 'Hospital Post-mortem Register and Case-book' for February 3rd, 1860, under the head of "Henry Barnes;" also 'Trans. Pathol. Soc.,' vol. xi, p. 62.

† The subjoined table, which throws important light on the subject of gangrene, was published, as the result of his own observations, by M. Boudet, in the 'Archives de Médecine,' September, 1843.

Age.	Number of post-mortem examinations.	Cases of gangrene of the lung.	Ratio of cases of gangrene of lung to post-mortem examinations.	Cases of various spontaneous gangrene.	Total cases of gangrene.	Ratio of cases of gangrene to post-mortem examinations.
Children .	135	5	1 to 27	9	14	1 to 9
Adults . .	156	2	1 to 78	4	6	1 to 27
Aged persons . .	220	2	1 to 110	7	9	1 to 24

confined to the lung, or may be associated with mortification in other parts of the body.

It is not an unusual accompaniment of cancrum oris; accordingly, M. Boudet's table shows that gangrene of the lung coexists with gangrene of other parts of the body more frequently in infancy than in after years.

The symptoms of gangrene vary considerably, according to the form which the disease assumes. In the diffused, or uncircumscribed form, the progress of the disease is often extremely rapid. The mischief is ushered in by utter prostration of strength; a small, weak, and frequent pulse, pallor and anxiety of countenance, and all the symptoms of rapidly failing power; coupled with these, there is frequent but feeble cough, and profuse frothy, diffluent expectoration, of a peculiar greenish colour, and intensely fetid gangrenous odour. Ere long the vital powers seem utterly oppressed or exhausted, the pulse fails, the features become collapsed, the expectoration ceases from want of power to expectorate, and the patient rapidly sinks.

The progress of circumscribed gangrene is somewhat different, and is marked by a train of symptoms which vary greatly at different periods of the disease, At first, the general symptoms are simply those of slight pneumonia, or pulmonary congestion; but they are attended by an amount of prostration which is quite disproportioned to the extent and apparent intensity of the local mischief. Sometimes, indeed, the invasion of the disease is even more insidious; there is nothing to draw attention to the condition of the chest, and depression of the vital powers is the only prominent symptom. After a time, however, expectoration commences; and the sputa, which at first are of a muco-purulent character, sometimes tinged with blood, emit a disagreeable odour; but as soon as a free communication is established between the air passages and the sloughing tissue of the lung, they not only acquire an intensely fetid gangrenous odour, but assume an appearance more or less characteristic of the disease. They lose their muco-purulent character, and become extremely liquid, or sero-purulent, and of a dirty greenish, or ash-grey colour. At the same time the breath acquires an offensive, putrid odour, the pulse becomes feeble and rapid, and there is every evidence of great and increasing prostration. Not unfrequently, the patient passes rapidly into a state of collapse, and sinks in a few hours, or it may be days, without the occurrence of any other symptom. Sometimes, though rarely,

profuse hæmorrhage occurs,* and terminates in death. Sometimes the sloughing implicates the large bronchi, and the œsophagus; sometimes it occasions perforation of the pleura, and gives rise to fatal pneumothorax, and it has been even known to cause perforation of the diaphragm, and to produce subcutaneous emphysema, by making its way through the two agglutinated layers of the pleura. At other times the disease is apt to pass into a somewhat chronic condition; the symptoms show that the patient's power of resistance is considerable; there is no longer rapid failing of the vital power, but the expectoration, which continues profuse and fetid, loses its extremely liquid character, and becomes more distinctly purulent; there is hectic fever, with night sweats and emaciation, and, after weeks, or it may be months of suffering, the patient dies utterly exhausted. In other cases the disease progresses more favourably; the sputa decrease in quantity, become decidedly purulent, and lose their fetor; the heat of skin, and hectic fever subside, the appetite returns, the patient gains flesh, and complete recovery ensues. These, however, are exceptional cases, and probably do not constitute above eight or ten per cent. of the whole. In all cases which do not speedily terminate in death, the fetor of the breath is apt to vary greatly. At one time it is insufferable, at another, nay, sometimes within a few minutes afterwards, it may be simply disagreeable; and then after the lapse of a longer or shorter period, it may again resume its poisonous quality. This alteration in the fetor and sweetness of the breath was very remarkable in a case admitted under my care into the York Ward of St. George's Hospital in October, 1858. †

The only rational explanation appears to be, that temporary obstruction of the bronchi leading to the sloughing tissue, shuts off the source of the gangrenous odour, which reappears as soon as the obstruction is removed, whether by the act of respiration, or of coughing.

The physical signs of the mischief which is going on are often obscure, and never distinctive; indeed, they are simply those of local pulmonary consolidation, followed by evidence of the breaking up of tissue, and subsequently of pulmonary excavation; they are not marked by any character calculated to throw light on the nature or cause of the disorganization. Sometimes, but not invariably, the mischief is preceded by the ordinary signs of pneumonia, and it is generally

* See St. George's Hospital 'Post-mortem Records' for 1847, p. 67. Hæmorrhage took place into a gangrenous cavity in the right lung.

† See 'Hospital Case-book,' xxxvii, p. 325.

accompanied by the signs of bronchitis as soon as the breaking up of the lung tissue has commenced.

The morbid appearances observable after death vary with the form which the disease has assumed. In the diffused form the lung tissue is extremely congested, and in a state of serous and sanguineous engorgement; its condition, indeed, resembles that of the first stage of pneumonia, except that it is more humid, and breaks down more easily. The affected portions are of a livid red, or else of a greenish, or brownish black, or ash-gray colour, more or less diffluent, and often converted into a sanious, putrid fluid, which escapes when the lung is cut, and emits an intolerably fetid, gangrenous odour. Not unfrequently some portion of the sphacelated tissue has been broken down and removed by expectoration during life, in which case an irregular excavation is found, the walls of which are composed of ragged, pulpy, mortifying tissue. In the uncircumscribed form of disease the parts in process of mortification blend gradually and insensibly with the sounder portions of the lung; whereas in the circumscribed variety the gangrenous portion of the lung is surrounded by inflamed tissue, infiltrated by exudation matter, which is more or less plastic, and maps out the limits of the affected portions. In this latter form of the disease the mortified tissue is usually found soft and diffluent, as in the uncircumscribed variety; but sometimes a sphacelated spot may be met with, harder and more compact than the tissue of sound lung, and forming a sort of core in the midst of the softer diffluent matter. Sometimes the cavities which are formed in this variety of disease, instead of being pulpy and ragged, become lined with a false membrane, which keeps up a purulent or muco-purulent secretion, and occasionally the density of this membrane appears to prevent their collapse and cicatrization. Be this as it may, these gangrenous excavations exhibit very little tendency to cicatrize, and free secretion from their internal surface has been known to go on for many years.*

In the diffused form of the disease the gangrene usually attacks a much larger portion of the lung than in the circumscribed variety; indeed an entire lobe is not unfrequently affected, and sometimes the greater part of one lung is implicated.† In the circumscribed, as also in the un-

* I have seen a smooth false membrane lining old tuberculous cavities and keeping up profuse secretion for years—in one case for no less than nine years. See also Walshe, loc. cit., p. 451.

† Laennec, loc. cit., p. 222.

circumscribed form of the disease, the mischief is usually limited to one lung, except in cases in which it ensues in secondary abscesses. In these it is scattered throughout both lungs, and is found most commonly towards their periphery, whereas in other cases the posterior portions of the lungs, and especially of the right lung, are those which are most prone to suffer.

The diagnosis of pulmonary gangrene is not in all cases an easy matter. It rests upon the peculiar fetor of the breath and expectoration, coupled with the sero-purulent character of the sputa and the physical signs of softening and excavation of the lung substance; and as a fetor not to be distinguished from gangrenous fetor may arise in cases in which no evidence of pulmonary softening is to be obtained, and in which there is reason to believe that mortification of the lung tissue does not exist, as it may also occur from sloughing of the internal surface of tuberculous and other cavities not primarily gangrenous, and as the character of the sputa will not alone serve to establish the true nature of the mischief, it is obvious that in most cases no great confidence can be felt in a diagnosis as to the existence of this disease, unless made by a very competent observer, and verified by a *post-mortem* examination. Nevertheless, when the signs already enumerated as indicative of gangrene supervene *suddenly* in a person suffering from adynamic congestion or inflammation of the lungs, and who was previously free from bronchitis and tubercular disease, there can be little doubt as to their true character, especially when gangrenous fetor of the breath has *preceded*, and is subsequently accompanied by the signs of pulmonary excavation.

Another source of difficulty, however, is the occurrence of gangrene of the lung without any perceptible fetor. I have never met with such a case; but Cruveilhier has described this variety of the disease; and Dr. Walshe, though he does not refer to instances in point, speaks of it as if it were a matter within his own cognisance. At all events, its occurrence must be extremely rare, and when it does occur it is not likely to be recognised during life.

Fortunately the difficulties incidental to the diagnosis of gangrene of the lung are not of material importance in practice; for whether the disease exists in an idiopathic form, or whether fetor be caused by sloughing of small portions of the bronchial mucous membrane, or by mortification of portions of tubercular or other cavities in the lung,

the aim of the physician in all cases must be to support the patient and obviate the tendency to death. There is nothing specific in the treatment to be employed. Whenever, and in whatever form of disease, gangrenous fetor arises, the vital powers are low and require support, and the amount and nature of that support must be determined by the precise condition of the patient, and not by any theoretical consideration as to the extent and nature of the mischief.* In the acute stage, quinine and bark in full doses, with ten or twelve minim doses of nitro-muriatic acid, opium, brandy, and diffusible stimulants are the appropriate remedies; and when the affection passes into a more chronic state, cod-liver oil, the balsam of peru in drachm doses, turpentine, and full doses of the compound tincture of benzoin, may be advantageously added. Ammonia has been recommended, but in my experience has not proved so useful as the mineral acids. Chlorate of potash has also failed in the only instance in which I have seen it employed, but its efficacy is so remarkable in cancrum oris, with which gangrene of the lung is often associated, that it deserves a further trial in full and repeated doses. It is not incompatible with the other remedies recommended, and there is no reason why it should not be administered in combination with them. Bloodletting is inadmissible, and dry-cupping and blistering, though not excluded from our list of agents by the nature of the disease, are of questionable utility. The former is most likely to prove serviceable.

The fetor of the breath may be corrected by rinsing the mouth with

* In evidence of this fact I may cite a very interesting case which occurred under my care at St. George's Hospital, in the year 1860. Stephen Deacon, aged 42, was admitted into Hope Ward on January 11th, having been seized with paralysis of the left side. The attack came on suddenly, accompanied by headache, three days before admission, whilst he was at work as a labourer. It had not been preceded by any apparent illness, and before his admission he had partially regained power over his leg, though his arm still remained motionless. His heart was healthy, the kidneys were sound, and he had no cough. On the 13th he was seized with dyspnœa, and great vital depression, and on the 14th began to cough and expectorate an enormous quantity of extremely fetid matter mixed with blood, which emitted a gangrenous odour. After the lapse of a few hours the sputa lost their puriform character and became sero-purulent and of a dirty ash-grey colour. Notwithstanding the paralysis, full doses of brandy, with bark, and ten-minim doses of nitric acid were administered; and in the course of ten days the cough had almost ceased, the expectoration was devoid of offensive odour, and the paralysis had in great measure passed off. On the 25th he left the hospital, having quite recovered, except in regard to the arm, which still remained weak.

chlorinated washes or a weak solution of permanganate of potash; and the inhalation of chlorine, will tend to modify the septic changes going on in the lung, and so to lessen the depression of the vital power. The vapour of turpentine is another agent which is reported to exercise a distinctly remedial power.* Hitherto I have had no experience of its action, but as its use need not interfere with the administration of other remedies, I should not hesitate to have recourse to its aid in severe or obstinate cases. The diet should be nutritious, and as generous as the digestive powers of the patient will permit. Wine; soup or strong beef tea, or warm milk thickened with isinglass; arrowroot, made with beef tea instead of water; eggs beaten up with brandy, blancmange, and essence of meat, are among the articles of diet which are most suitable on these occasions. Meat, with pale ale or bottled stout, may be given as soon as the patient's stomach can bear it.

Œdema of the Lungs.

Œdema of the lungs is not a condition of frequent occurrence. Arising as it does from the same class of causes which occasion œdema in other parts of the body, it should be regarded as a secondary affection, symptomatic of mischief elsewhere, rather than as a primary or idiopathic disease of the pulmonary parenchyma. It is a common accompaniment of Bright's disease of the kidneys, and of the dropsy which occurs in sequel of scarlatina; it is apt to accompany typhus fever, scurvy, and other diseases in which there exists a depraved and fluid condition of the blood; and it is sometimes the result of extreme pulmonary congestion from whatever cause arising, and thus is found associated with acute bronchitis and pneumonia, with obstructive or regurgitant disease of the mitral valve, with obstructive disease of the aortic valves, and with pressure on the pulmonary veins, whether caused by aneurism, or by cancerous or other morbid deposits in the chest. It is speedily induced by section of or pressure on the par vagum, probably as a result of defective innervation, and is usually attendant on the pulmonary congestion which precedes slow death.

By whatever cause produced, œdema of the lungs is characterised by infiltration of the air cells and interstitial areolar tissue with a thin serous fluid. When the œdema is passive and connected with chronic mischief, the fluid effused is usually colourless; whereas when the œdema

* See 'Med. Times and Gazette,' April 15, 1845, and Wiener, 'Zeitschrift,' 1853.

is acute, and arises from active congestion of the lung, it is more generally tinged with blood. It generally affects both lungs, and is diffused throughout their structure, or limited to the inferior and posterior portions, in which the laws of gravitation would naturally cause the fluid to accumulate. Oppression at the chest, with increased frequency of respiration, amounting sometimes to excessively hurried breathing, slight cough, and a thin, but sometimes rather tenacious frothy expectoration, are the general symptoms by which its accession and course are usually marked. The morbid anatomy of the parts, and the physical signs to which the disorder gives rise, will be evident from an inspection of the subjoined table:

Chronic and Passive Œdema.

Morbid Anatomy.	*Physical Signs.*
Lung of a pale greyish colour; collapses slowly and imperfectly, is inelastic, doughy, pits readily on pressure; and scarcely crepitates on being handled. On section, a large quantity of almost colourless serum, unmixed, or nearly so, with air, oozes from the surface; the lung tissue is tough and resistant, and portions of it sink instantly in water without being previously subjected to pressure. The lining membrane of the bronchi is often of a dark, livid colour, consequent on chronic vascular congestion.	*Inspection* affords no information. *Palpation* is said sometimes to disclose increased vocal fremitus. This has not been the result of my experience. *Percussion* sound duller than natural, and parietal resistance increased. Skoda speaks of a tympanitic sound on percussion, but I have never met with it. *Auscultation.*—Respiration weak or almost absent, or else harsh and coarse, and accompanied by bubbling râles, according to the precise degree of infiltration of the portion of the lung auscultated. If, as often happens, bronchitis coexists, sonorous and sibilant rhonchi will be heard.
Acute œdema. — Lung usually red; collapses slowly and imperfectly; is inelastic, but less so	*Acute œdema.*—Inspection, palpation, and percussion yield the same results in acute as in chronic

Morbid Anatomy.

than in chronic œdema, and is more crepitant on pressure; on section, a large quantity of a pale red, frothy serum escapes from the cut surface; the lung tissue is red, soft, and easily broken down, and the lining membrane of the bronchi is of a red colour, such as is seen in cases of active congestion or inflammation.

Physical Signs.

œdema; but on auscultation the vocal resonance is generally louder in the former variety, and the râle which attends it is much finer. Indeed, it ordinarily resembles the fine bubbling râle of capillary bronchitis, and is to be distinguished from it only by the lesser viscidity of the sputa, and the absence of the general signs of bronchitis. In some instances it so closely resembles the crepitation of pneumonia, that the absence of tubular breathing and other symptoms which usually attend the progress of that disease, alone enable us to discriminate between them.

The treatment of pulmonary œdema must be in strict relation with the cause from which it originates. When acute, and dependent on Bright's disease or scarlatinal dropsy, diuretics and saline purgatives are especially indicated, and the administration of squills and digitalis should not be neglected; an emetic in some instances proves extremely serviceable, and so do vapour and hot air baths. Its occurrence betokens the necessity for a tonic and stimulant treatment as soon as the chest is somewhat relieved; and nothing answers better than the tincture of the sesquichloride of iron, in combination with a diuretic and a light vegetable bitter. If it occurs during the course of typhus fever, or other disorders of an adynamic type, diffusible stimulants internally, and blisters and mustard poultices to the surface of the chest are commonly found to be the most efficient agents; whereas, if it is associated with pulmonary congestion, unconnected with a disordered condition of the blood, repeated dry cupping, aided by digitalis, expectorants, and saline purgatives, prove our most valuable and trustworthy allies.

Pulmonary Hæmorrhage, Hæmoptysis, and Pulmonary Apoplexy.

Pulmonary Hæmorrhage* is a subject of fearful interest to the physician, and of serious, nay vital, importance to the patient. Sometimes, indeed, it is the result of idiopathic congestion of the lung, and may be regarded as a primary affection of not a very serious character. But such an event is exceedingly rare. Much more commonly it is attributable to engorgement of the pulmonary vessels, induced by organic disease of the lungs or heart, and is altogether a secondary affection—a mere symptom of structural mischief in the chest. Unfortunately, too, the maladies of which it is an index, are of a serious nature, and usually tend to an untimely death. It is a frequent harbinger of consumption in its varied forms, and is often one of its early symptoms; so that too much attention cannot be bestowed in the investigation of every point relative to its occurrence.

Considerable difference of opinion exists as to the source and significance of pulmonary hæmorrhage. Some persons imagine that, when the larynx, trachea, or bronchi are congested, blood may exude from the mucous surface after the manner in which it oozes from the posterior fauces and the mucous membrane of the mouth; nay more, they affirm that exudation from the surface of the air passages is a very frequent source of pulmonary hæmorrhage, even in cases in which no local structural mischief exists. Others, without denying the possibility of hæmorrhage from the bronchial mucous membrane, assert, not only that it is of rare occurrence, but that it seldom, if ever, takes place except when the blood is altered in character, as in purpura, scurvy, and similar disorders, or where the bronchial mucous membrane is ulcerated. These writers refer to the parenchyma of the lung as the usual source of the bleeding, and they support their opinion by reference to the minute dark spots which are often found in the lungs after death in cases in which hæmoptysis has existed during life, and which are evidently caused by the outpouring of blood consequent on the giving way of the pulmonary tissue.

* Throughout this section I apply the term pulmonary hæmorrhage indifferently to all outpourings of blood which take place from any portion of the respiratory apparatus, from the epiglottis and larynx to the pulmonary parenchyma. During life it is often impossible to decide from which portion of the tract the bleeding proceeds, and even after death it is sometimes difficult to decide the question. It is therefore useless to attempt to separate the different varieties for the sake of description.

My own opinion occupies a middle place between these two extremes. Careful observation has led me to believe that, except in cases of diseased heart, or of purpura, and other hæmorrhagic disorders, bleeding seldom arises from the bronchial mucous membrane, unless that membrane is the seat of ulceration, or is acted on by aneurismal or other tumours, or by tubercular or other adventitious deposit, which causes pressure upon the bronchial vessels, and thus mechanically leads to rupture of the capillaries. But inasmuch as I have frequently seen blood ooze from the congested mucous lining of the mouth and throat, and on several occasions have traced cases to the dead-house of St. George's Hospital in which, more or less, hæmoptysis had occurred during life, and in which, nevertheless, no organic disease of the lung existed, and no evidence of any disruption of the pulmonary tissue could be discovered after death, I see no sufficient reason to doubt that, in some instances at all events, hæmorrhage may take place from the congested bronchial membrane. On the other hand, the large amount of blood which is sometimes poured out, the rapidity with which the outpouring occurs, and the evidence which, in certain instances, may be obtained after death, of the giving way of the pulmonary tissue, are facts which lead irresistibly to the conclusion that the parenchyma of the lung is a frequent source of bleeding. And when, further, it is considered that simple ulceration of the bronchial mucous membrane is of extreme rarity, that in many instances of pulmonary hæmorrhage there is entire absence even of bronchial congestion, and that the delicacy of the lung tissue is such as to render it very liable to give way under conditions productive of extreme congestion and mechanical obstruction to the pulmonary circulation, the conclusion is inevitable, that the proper tissue of the lung is a much more frequent source of hæmorrhage than the bronchial mucous membrane.

But whatever its source, pulmonary hæmorrhage is always a symptom of grave importance, as indicating interference with the circulation of vital organs. In some instances it may result from wounds or other mechanical injuries of the lung; in some, the heart may be the cause of the mischief, giving rise, by its diseased and irregular action, to undue congestion of the lung; in others, the pressure of aneurismal tumours, or of tubercule, cancer, or other adventitious matter, may produce the same effect; in others, again, pneumonic congestion, or abscess, or gangrene, may be the cause of the hæmorrhage; in others, ulceration of the larynx, trachea, or bronchi; whilst in certain instances,

extreme congestion of the bronchial mucous membrane, more especially, when connected with a spanæmic condition of the vital fluid, appears to lead to the outpouring of blood, irrespective of any organic or permanent lesion. But mischief of a serious nature must have proceeded to some extent before hæmorrhage can occur. Even in those instances in which spitting of blood is commonly supposed to be independent of local mischief, and to be of little importance, as when it occurs after violent straining efforts, or in connection with diminished atmospheric pressure during the ascent of lofty mountains, my own observation has led me to take a more serious view than that usually entertained, and to believe that in these, as in other cases, there is usually some latent mischief in the chest—some local cause of pulmonary congestion—some mechanical interference with the capillary circulation through the lungs.* In several instances which have come before me, in which persons apparently in good health have spat blood, under one of the conditions above specified, I have known the symptoms of consumption set in, and run on rapidly to a fatal termination. And although it is true that individuals are sometimes met with who have had hæmoptysis more or less profuse, and who, nevertheless, have attained to a good age, without the occurrence of phthisical symptoms, this fact does not invalidate the conclusion that their lungs are more or less organically diseased. The same fact is sometimes observed in persons who are descended from a consumptive stock, and who eventually die of phthisis. Indeed, nothing is more common than for spitting of blood to precede for years the fatal development of tubercular disease; and its occurrence only proves what most persons are prepared to admit, that tubercular disease of the lungs, if not extreme, may exist for an indefinite period in a quiescent state, without giving rise to symptoms of consumption, or, in other words, that persons

* In this observation I am borne out by Dr. Walshe. In a sensible note (loc. cit., p. 470), he remarks—"Boussingault, D'Orbigny, and Roullin make no reference to hæmoptysis as having occurred in their ascents of the Andes; De Saussure observed nothing of the sort in the ascent of Mont Blanc; and Mr. Albert Smith, whose medical education gives value to his testimony, insisting in his narrative on the difficulty of breathing experienced, is silent concerning expectoration of blood, and yet his party and guides numbered twenty-four." A large additional mass of similar evidence might now be adduced from the experience of members of the Alpine Club, as also from that of aeronauts who have rapidly attained to very high altitudes in balloons. Messrs. Glaisher and Coxwell who in this way rose to the unexampled altitude of six miles, and nearly lost their lives through the rarefaction of the atmosphere, make no mention of hæmoptysis in their detail of the symptoms which they experienced.

who have tubercles in their lungs, if placed under circumstances favourable to their health, may live on for years in the enjoyment of fair average health, and, as *post-mortem* records clearly show, may die at an advanced age of some other disorder, without having manifested any symptom of consumption, with the one exception of spitting of blood. In short, if those cases are excluded in which the sputa are slightly specked or streaked with blood—as in certain instances, of severe congestive bronchitis—or are rusty-coloured, from admixture with it as is often seen in acute pneumonia, the only exceptions I am disposed to admit to the fearful significance of hæmoptysis, are those in which it results from blows on the chest, or from mechanical injury to the lung, or in which it occurs in women, vicariously to the menstrual discharge.* In these cases, undoubtedly, it seems occasionally to take place independently of structural pulmonary disease.

It follows, from what has been already stated, that hæmoptysis, or the discharge of blood by expectoration, must always be regarded as suggestive of organic disease of the chest. It does not necessarily indicate the fact, but it is sufficient to excite the gravest suspicions, and to render imperative a close and careful examination into the condition of the thoracic viscera. It matters not whether the quantity of blood be small,—mere specks or streaks,—or whether it amounts to drachms or ounces; in either case its presence affords just cause for alarm. For although, as already stated, the researches of pathology oblige us to admit that hæmoptysis may arise from a variety of causes, they have also served to establish the fact that it is most frequently connected with tuberculization of the lung; and to such an extent does this hold good, that spitting of blood is justly regarded as one of the earliest and most important signs of pulmonary consumption.†

The quantity of blood expectorated in different cases of pulmonary hæmorrhage varies extremely. Sometimes it is so small as to escape observation; and at times, more especially in disease of the heart, is undistinguishable by the naked eye, although the microscope at once reveals blood corpuscles in the sputa; sometimes, though rarely, it is so profuse, and is poured out so rapidly, that the patient is suffocated by it, or else dies from the effect of syncope; more commonly, it is ejected slowly, a mouthful at a time, and in quantity varying from a few streaks to a drachm or several ounces.

* Most practitioners must have met with cases in point, but I would refer those who have not to a remarkable instance of vicarious menstruation from the lungs recorded by Pinel, and quoted in Dr. Watson's 'Lectures,' ed. i, vol. ii, p. 140.

† For facts corroborative of this statement see chap. iv, pp. 383-5, of this treatise.

It is commonly supposed that the quantity of blood expectorated affords a trustworthy measure of the significance of pulmonary hæmorrhage, and that a few slight streaks of blood are of little or no importance. They may arise, it is often said, from the throat, or may be the result of simple bronchitis. On the other hand, some physicians attach a fatal significance to every streak of blood in the sputa, and are disposed to doubt the correctness of those who would attribute such hæmorrhage to simple congestion of the bronchial mucous membrane. Unfortunately too, the matter, though of some practical importance, does not admit of very positive decision. There cannot be a doubt that, in the majority of cases in which spitting of blood occurs, however small the quantity of blood may be, tubercles in an active or latent state are present in the lungs. The revelations of the stethoscope, corroborated as they are by inspection in the dead-house, abundantly attest this naked fact. But this is not the whole question at issue. The point to be determined is, not simply whether spitting of blood is ever met with when there is no organic disease of the chest, but in what proportion of cases it occurs independently of structural mischief, and whether any clue to the condition of the chest can be obtained from the quantity of blood expectorated. And here we find ourselves completely at fault. Experience undoubtedly overrules the dicta of those who would deny the occurrence of hæmorrhage as a result of simple pulmonary congestion; for on several occasions I have traced cases to the dead-house of St. George's Hospital, in which there has not existed any organic disease in the chest, and in which, nevertheless, hæmoptysis, to a greater or less extent, has been observed during life. At the same time it justifies the most positive statement that such an occurrence is exceedingly rare. From the nature of the case, it is impossible to obtain trustworthy statistics to throw any light on the proportion of cases in which spitting of blood occurs irrespectively of organic mischief, or to show the relative significance of slight streaks of blood, and of drachms or ounces of blood in the sputa. Nevertheless, as on the one hand there is abundant evidence to prove that slight streaks of blood are often indicative of the presence of tubercles, and on the other cases have been met with in which a considerable quantity of blood has been ejected by coughing, as the result of simple pulmonary congestion, it is fair to conclude that no reliance can be placed upon the mere quantity of blood in the expectoration as a test of the condition of the lungs and air passages. In short, spitting of blood is

itself a fact of grave clinical significance; but in most instances the quantity of the blood ejected adds little or nothing to the information it affords. The only exception to this general rule is in the case of very profuse hæmoptysis. This, if unconnected with suspended menstruation, can only arise from excessive pulmonary congestion, or from the giving way of a large vessel. In either case, it may result from the presence of tubercle or other matter in the lung producing ulceration of one of the pulmonary vessels, or from the oozing or bursting of an aneurismal tumour; it cannot be attributable to mere idiopathic congestion.

Again, the colour of the blood in hæmoptysis is not to be depended on as a proof either of the source of the hæmorrhage, or of the condition of the parts from which it originates. The blood is usually florid, but it may be dark-coloured, or almost black; or it may be partly of an arterial, and partly of a venous tint; it is generally frothy, from admixture of air, and is manifestly ejected by the act of coughing; but if it be more profuse, it may escape in gulps from the mouth in a non-aerated condition, or may even give rise to reflex actions, which may induce the patient to declare that the blood was ejected by vomiting, and not by coughing.

Indeed, the whole question of pulmonary hæmorrhage is involved in considerable difficulty. Not only does doubt sometimes arise as to whether hæmoptysis is or is not connected with structural changes within the chest, but much uncertainty may be felt as to whether blood ejected from the mouth is derived from any portion of the respiratory apparatus. Even when the blood observed in the sputa results from pulmonary hæmorrhage, patients first become conscious of its presence, when it reaches the posterior fauces. They are apt, therefore, to assert, with the greatest confidence, that it is derived from the throat, and not from the chest. In like manner, they will sometimes refer it to the gums, the mouth, or the posterior nares. The mere appearance of the blood will not serve to elucidate the subject; but a close investigation into the state of the mouth and the pharynx, will generally show whether the hæmorrhage is referable to either of the sources indicated by the patient. And as the determination of the question is a matter of great importance, the student should make a careful examination, so as not to be misled by the earnestness of the patient's assertions.

Another source of difficulty in the diagnosis of hæmoptysis is the

occurrence of hæmatemesis, or vomiting of blood from the stomach. It has been already stated, that in severe cases of pulmonary hæmorrhage, blood is ejected rapidly by mouthfuls, and that its ejection is sometimes accompanied by efforts which the patient is unable to distinguish from vomiting. Hence, persons who have had a severe attack of hæmoptysis will often assert that they have suffered from "vomiting of blood," and even when closely questioned on the subject, will deny that they brought up the blood by coughing. But if care be exercised in conducting the inquiry, facts may generally be elicited calculated to throw light on the real nature of the malady. In cases of hæmoptysis, the family and personal history of the patient will generally indicate the probability of thoracic disease; and in corroboration of the information thus obtained, the face will usually be found flushed, and the pulse excited, full, and bounding; the seizure will have been preceded by more or less dyspnœa, slight hacking cough, a sense of weight or tightness in the chest, and a tickling at the top of the larynx; the blood will be frothy and usually of a florid colour; and although the greater part of it may have been brought up in the course of a few minutes, yet blood-streaked or blood-stained sputa will continue to be expectorated for a considerable time after "the bursting of the blood-vessel." On the other hand, hæmatemesis is usually preceded by a sense of weight and uneasiness at the epigastrium, and is sometimes followed by tenderness on pressure; the patient's face and lips are blanched, and his pulse is seldom so excited as in hæmoptysis, and is weak, rather than full and bounding; the blood ejected is not aerated, and, unless the hæmorrhage occurs rapidly from ulceration of an artery in the stomach, is of a dark, venous hue; it is brought up suddenly, and the vomiting is not followed, as in hæmoptysis, by the expectoration of blood-streaked or blood-stained sputa, and the stools are dark-coloured and pitch-like in appearance, from the quantity of blood they contain — a circumstance which never occurs in hæmoptysis, unless blood has been accidentally or wilfully swallowed. Thus, the general symptoms alone will suffice, in most instances, to stamp the case as one of hæmoptysis; but no positive conclusion ought to be drawn, and no opinion expressed, until the chest has been carefully examined. For although, on the one hand, spitting of blood may occur before any distinct evidence can be obtained of structural mischief in the lungs, and on the other, hæmatemesis may take place in persons whose lungs are structurally diseased, yet these coincidences are so rarely met with, that the finding of liquid

bubbling râles in some portion of one or both lungs would go far towards solving any doubt as to the blood being derived from the chest.

When pulmonary hæmorrhage occurs, it may or may not give rise to consolidation, or to rupture of the tissue of the lungs. Ordinarily, when the quantity of blood poured out is small, it is ejected by cough before coagulation takes place, and no local ill results ensue; but when the hæmorrhage is more profuse, and more especially in regurgitant disease of the mitral valve, when great impediment exists to the onward flow of blood through the lungs, the air cells and terminal bronchi may become distended with coagulated blood, forming firm nodulated masses, of from half an inch to four inches in diameter, blackish red, or even black in appearance, impervious to air, inelastic, and excessively firm to the touch. The masses thus formed constitute what has been termed "pulmonary apoplexy"—a singularly bad and inappropriate term, but one which was introduced by Laennec, and has obtained unusual currency in the profession. This form of local mischief is not produced by hæmorrhage from any particular source; it may occur whenever blood is present in the air passages, and finds its ways into the air vesicles. Dr. Watson gives a case in which it resulted from blood which trickled down, through the windpipe, into the lungs, during an attack of hæmorrhage resulting from ulceration of the lingual artery; * and most persons who have had frequent opportunities of witnessing *post-mortem* investigations, must have met with cases in which it has arisen in connection with hæmorrhage in cases of consumption. Nevertheless, it may be stated, that disease of the left side of the heart is the principal, though not the invariable cause of its occurrence. If the outpouring of blood into the air cells does not produce rupture of the pulmonary tissue, the apoplectic masses, though irregularly shaped, are clearly circumscribed and sharply defined, consisting of one or more lobules, firmly blocked up with fibrin; whereas if, as sometimes happens, the texture of the lungs gives way, and admits of blood infiltrating the interlobular areolar tissue, the pulmonary apoplexy is diffused, and the characteristic sharp outline of the lobular form of the mischief is wanting. The former variety of mischief is by no means uncommon, and, as already stated, usually accompanies enlargement of the left cavities of the heart, and inefficiency of the mitral valve. The latter is rare, and is seldom met with except as the result of mechanical injury to the chest, or of the

* See 'Watson's Lectures,' vol. ii, pp. 146-7.

bursting of an aneurism, or the giving way of a large vessel, in consequence of ulceration. The one is commonly found affecting both lungs, the other is almost always confined to one lung; the first generally affects several portions of the lungs; the last is usually confined to one portion of the lung. Neither the one form nor the other is necessarily accompanied by hæmoptysis, though spitting of blood is usually an attendant symptom of both, and affords the principal evidence we can obtain of their occurrence.

When the hæmorrhage is very profuse, and infiltration and rupture of the pulmonary tissue takes place, the blood may not only break up the tissue of the lung, but may burst through its serous envelope and escape into the pleural cavity. Many cases are on record of death produced in this manner.*

In the following table, the local effects produced by outpourings of blood in the lungs, and the physical signs to which they give rise, are collocated for the sake of comparison:

Morbid Changes.	*Physical Signs.*
The parenchyma of the lungs and the bronchial mucous membrane are much congested, and sometimes, though rarely, there is ulceration of the mucous surface. Tuberculous or other deposit in the lung, or aneurismal or other tumours, pressing on the lungs and producing distension and rupture of the capillaries, or, in some rare instances, ulceration of a large vessel, or insufficiency of the valves on the left side of the heart are seen to be the cause of its occurrence.	When the hæmorrhage from the lungs is slight, and pulmonary apoplexy does not occur, neither *inspection*, *palpation*, nor *percussion* affords any information as to the seat or amount of the bleeding. Auscultation, however, will usually reveal a thin bubbling râle at the spot whence the blood in the sputa is derived.
When pulmonary apoplexy exists it is usually found in the lower	When the hæmorrhage is more profuse, and accompanied by pulmonary apoplexy, an abundant bubbling râle will be heard at the seat of effusion, and will continue until coagulation takes place. Then of course all bubbling

* See Dr. Paterson's "Observations on Pulmonary Apoplexy proving Fatal by Rupture of the Periphery of the Lung by Effusion of Blood into the Pleura," in the 'Edin. Med. and Surg. Journ.,' for January, 1846.

Morbid Changes.

and posterior portion of the lungs, though it is sometimes near their anterior surface. It causes a distinct projection of the pleura if it is situated near the surface of the lung, and if, as sometimes happens, the pleura gives way, blood may be found effused into the pleural cavity. The air cells implicated in the apoplectic mass are blocked up with coagulated blood, and there may, or may not be laceration of the lung tissue. If there is no giving way of the lung structure, there are seen on the substance of the lung one or more sharply defined nodulated masses of solidified parenchyma, varying from half an inch to four inches in diameter. These masses are of a blackish red colour, inelastic, and excessively firm to the touch, do not crepitate on pressure, and sink instantly when placed in water. On section, they present a dry, and nearly homogeneous surface, sometimes slightly granular, from which a small quantity of dark, grumous blood can be obtained by scraping. In some few instances, a small clot of loosely coagulated blood exists in the centre of the apopletic clot. The adjacent lung tissue is sometimes inflamed, in which case it is loaded with a sero-sanguineous, but frothy fluid. Under these circumstances, washing with water will remove the inflammatory

Physical Signs.

will cease, and the existence or non-existence of physical signs of the mischief which has occurred will depend upon the seat and extent of that mischief.

If the hæmorrhage be small, and deeply seated in the lung, all signs of its existence will cease with the coagulation of the blood. The amount of blood effused will not seriously impair the motion of the chest, and neither inspection, palpation, percussion, nor auscultation will avail to discover its seat.

If the nodulated masses of solidified lung tissue lie superficially, percussion and auscultation may possibly yield some evidence of the mischief, but positive information can be rarely thus obtained if the nodules are few and small.

When the hæmorrhage is more profuse and the patches of pulmonary apoplexy are larger, and lie superficially, palpation, percussion, and auscultation may all furnish evidence of the seat of mischief.

Percussion will elicit a dull tone over a space corresponding to the extent of lung implicated.

Auscultation will detect a diminution or entire absence of the respiratory murmur over the affected portions, and coarse breathing, possibly accompanied

Morbid Changes.

exudation, and expose the outline of the apoplectic mass.

If laceration of the lung tissue has occurred, the defined outline of the lobular form of the disease will be absent, in consequence of the blood having passed in all directions into the interlobular cellular tissue; and the apoplectic mass will consist of coagulated blood, of various degrees of firmness, intermixed with portions of broken-down and disorganized pulmonary tissue. In some rare instances suppuration and gangrene have occurred in the apoplectic mass.

As resolution takes place the tint of the coagulum becomes lighter, and passes into a brownish and then into a yellowish red. The effused matter gradually softens, and is converted into a yellowish or rusty coloured fluid, which is removed by expectoration and absorption. The bronchi once permeable, air is readmitted, the proper lung tissue reappears, and the abnormal firmness and density of the affected portion pass off.

Physical Signs.

by bubbling in their immediate vicinity.

When the apoplectic mass is large, and is traversed by a good-sized permeable bronchus, hollow breathing and increased resonance of the voice are heard, and palpation makes us aware of increased vocal fremitus.

With the progress of resolution the hollowness of the breathing and the increase of the vocal resonance cease, râles of all kinds begin to be heard over the affected parts, and ultimately are replaced by natural vesicular breathing.

If, instead of undergoing resolution, pulmonary apoplexy is followed by pneumonic abscess or gangrene, the signs of these several affections will be met with instead of the signs of resolution.

Thus, then, it will be seen that the most careful physical examination of the chest will often fail in throwing much light upon the source of pulmonary hæmorrhage, and that were it not for the occurrence of hæmoptysis we should often remain in ignorance of its existence. But it is otherwise in regard to the diseases from which the spitting of blood originates. When once the appearance of blood in the sputa has

directed attention to the condition of the chest, the physical signs will usually enable us to determine the precise character of the mischief present; and in the great majority of cases they will teach us that, whatever the seat of the hæmorrhage, its occurrence is attributable to tubercular disease of the lung, or to disease of the left side of the heart.

The treatment of hæmoptysis must be varied according to the extent and probable cause of the hæmorrhage. If the heart is the organ at fault, digitalis, aconite, or the veratrum viride, which exercise a controlling influence over its action, should be administered internally, together with a few doses of calomel to unload the liver, and saline purges to draw off watery evacuations from the bowels, and so to relieve the circulation; at the same time venesection may be had recourse to, and cupping or dry cupping between the shoulders should be employed to mitigate the local congestion. Perfect rest must be strictly enjoined, all mental excitement avoided, and the diet restricted to milk, barley-water, iced lemonade, and other cooling beverages. In the most frequent form of hæmoptysis, viz., that which is attendant upon chronic disease of the lung when the quantity of blood is small—not exceeding a teaspoonful—and where the hæmoptysis is unattended by febrile excitement and symptoms of general pulmonary engorgement, little heed need be paid to its occurrence; the hæmorrhage is nature's mode of relieving temporary local congestion, and will prove beneficial rather than hurtful. The diet need not be altered, nor tonic medicines discontinued; and the utmost that is necessary or likely to prove useful in the way of special treatment is dry cupping between the shoulders, a dose of calomel or blue pill if the liver is inactive, a saline purge to produce a free, watery evacuation, and the administration of the mineral acids in combination with tonics and cod-liver oil.

If the hæmorrhage, though scanty, be attended with feverish heat of skin and excessive vascular action, it will not be safe to continue the use of tonic medicines or a stimulating diet. The patient must be kept low, and active measures must be taken to subdue the feverish excitement of the system. He should remain in bed, with his head and shoulders elevated; a free circulation of cool air should be kept up in the room in which he lies, his mind should not be disturbed, and silence should be enjoined. Iced water should be his drink, and his diet restricted to barley-water, cold milk, whey, and cold beef tea. A free action of the liver must be sustained, by mercurials if necessary; cooling

saline purgatives should be given, to produce watery evacuations from the bowels, and thus not only relieve the vessels, but prevent the effort of straining at stool. The excitement of the nervous and vascular systems should be tranquillised by salines, with tartar emetic, nitre, digitalis, and opium; and if local congestion be excessive, the aid of dry cupping or cupping, or even venesection, must be had recourse to. In the latter case, small and repeated bloodlettings, from six to ten ounces, according to the constitution of the individual, appear to answer better than a single full bleeding, and venesection from the arm has proved more serviceable in my hands than the loss of blood by cupping; the blood is drawn more quickly, and the effect on the pulse is more decided. But whether venesection from the arm or cupping be employed, dry cupping between the shoulders ought never to be neglected. In most instances, it will supersede the necessity for bloodletting, and it is at all times a valuable adjunct to venesection in removing pulmonary congestion. Leeches in these cases, prove comparatively useless.

If the hæmorrhage be profuse, the question cannot arise as to what measures are best suited to the patient's constitution, or the malady under which he is labouring. The sole point to be decided is, how to stay the bleeding, and obviate the tendency to death. A variety of remedies have been employed for this purpose, amongst which I may mention, as having received the greatest amount of testimony in their favour, venesection, cupping, dry cupping, the application of ice down the spine, tartar emetic, acetate of lead and opium, gallic acid, sulphuric acid, alum, matico, ergot of rye, and turpentine. My own experience leads me to testify most strongly in favour of repeated dry cupping, aided by the application of ice down the spine, and by the internal administration of full doses of digitalis.* If this fails, full and frequent doses of gallic acid, or lead, and opium may be given, if the circulation is much accelerated, and of spirits of turpentine if the bleeding is unattended by vascular excitement. The gallic acid should be given every hour, in eight- or ten-grain doses, until the hæmorrhage is subdued, or until a dark-green colour in the sputa indicates its action on the system; or, if lead is employed, it should be given in doses of two or three grains, combined with dilute acetic acid and laudanum, after the method recommended by the late Dr. Anthony T. Thompson, and the dose should be repeated every hour or every two or

* ℥j to ℥ij of the tincture, or gr. vj to gr. viij of the powder daily.

three hours, according to the urgency of the symptoms. If turpentine is had recourse to, it should be administered at brief intervals, in half-drachm doses.

If any symptoms of sinking arise, they must be met by diffusible stimulants, and must be treated on general principles, without reference to the nature of the mischief in the chest. Meanwhile, however, the administration of those remedies which are calculated to control the bleeding should be perseveringly continued.

A few words may be added by way of caution. During convalescence from an attack of hæmoptysis, and for some time afterwards, the patient should be advised to keep perfectly quiet and free from bodily or mental excitement. Nothing is more likely to determine the recurrence of hæmorrhage than public speaking, or whatever puts a strain upon the organs of respiration; or than violent bodily exertion, which accelerates the circulation and increases vascular action; or than mental excitement, which also hurries the heart's action, produces pulmonary congestion, and thus proves equally prejudicial. These are dangers which may be avoided if the patient is duly warned against them; and it behoves us therefore to place them clearly before him ere he is permitted to return to the ordinary avocations of life.

Acephalocysts in the Lungs.

Hydatids sometimes exist in the lungs, having been developed in the pulmonary tissue, or having found their way there from the liver. When developed in the lung, they may exist for some time without giving rise to notable disturbance; but as they gradually enlarge, they exert pressure on the surrounding tissues, and may occasion hæmoptysis, bronchitis, pneumonia, or even gangrene. In some instances they have been known to cause perforation of the pleura and give rise to pneumothorax; and they may also make their way through the diaphragm.

If the chest be examined whilst as yet they are in a quiescent state, and are small, and have not occasioned local irritation, the breathing may be coarse and harsh in their immediate vicinity, and there will probably be prolongation of the expiratory sound; but if they chance to be deeply seated in the lung, these symptoms will be masked by the normal sounds emitted by the healthy lung tissue which lies between them and the chest walls.

As they enlarge and excite bronchitis, pneumonia, or pleurisy, the general symptoms and physical signs of those diseases will be present; and unless some portion of an acephalocyst be expectorated or the hooklets of the echinococcus be discovered by the microscope, there will be nothing to point to the true nature of the disease.

Not unfrequently, the general symptoms are those of rapidly progressive phthisis—cough with muco-purulent expectoration, hæmoptysis, night sweats and emaciation—whilst the physical signs may at first consist of dulness on percussion, absence of breathing over the seat of the cyst, and râles and rhonchi with prolongation of the expiratory sound in its vicinity. When, after a time, the cyst bursts, there may be hollow breathing, with gurgling and pectoriloquy, just as in any other cavity which has established a free communication with the upper air passages. Here, again, the discovery of a portion of a cyst, or of the hooklets of the echinococcus in the sputa, will alone enable us to determine the nature of the disease.

When the cyst makes it way into the lungs from the liver, the pulmonary mischief will have been preceded by hepatic symptoms, and almost certainly by a sharp attack of pleurisy. At length the patient is somewhat suddenly attacked with extreme distress and difficulty of breathing; his countenance becomes anxious, his features collapsed, the skin clammy and more or less livid, and the extremities cold; incessant paroxysmal cough supervenes, vomiting occurs, and by degrees he becomes more or less deeply jaundiced. Then come symptoms of acute pneumonia and pulmonary consolidation, followed by the physical signs of excavation, the expectoration at the same time passing through every variation of tint and consistence, from that of slightly rusty coloured mucus to that of deeply bile-tinged muco-purulent matter, or of a dark brown coloured fetid fluid, containing shreds of lung tissue and entire hydatids, or portions of hydatid cysts.

In this case, as in those already referred to, the discovery of the hydatid cysts, or of the hooklets of the echinococcus, constitutes the only distinguishing mark of the disease, as the general symptoms and physical signs might be attributable to simple hepatic abscess making its way out through the lungs.

In all instances in which acephalocysts are met with in the lungs, the issue of the case is extremely doubtful. When they are developed in the pulmonary organs, statistics seem to show that although the symptoms which attend their rejection are very severe, and may continue for

many months, yet that recovery may be brought about in half the cases; whereas, when they make their way into the lungs from the liver, the constitutional disturbance is so great as to leave little hope of recovery. Nevertheless recovery has been noted in a few exceptional cases.

Treatment is of as little avail in these cases of intra-thoracic hydatids as in the case of hydatids in the liver or elsewhere. We know of no means of destroying the acephalocyst or inducing its expulsion. Therefore, the only rational means to be adopted for our patient's relief are those which are calculated to mitigate pain, assist expectoration, and sustain the failing power of the system. Opium, æther, the various expectorants and alcoholic stimulants are of the greatest service, and counter-irritants are useful in relieving the local inflammation. As soon as the first severity of the symptoms has subsided, tonics must be given freely, and the strength sustained by a nutritious diet.

CHAPTER III.

BRONCHITIS.

This disease is essentially an affection of the bronchial mucous membrane, and may arise from any cause, whether mechanical, constitutional, or epidemic, which excites irritation, congestion, and inflammation of that membrane. Thus it may follow the inhalation of the fine metallic particles which result from needle-grinding and other similar occupations; it is a prominent symptom of the so-called "hay asthma," a disorder attributable to the inhalation of the pollen of the *Anthoxanthum odoratum*, or sweet-scented spring grass,* and it is a frequent accompaniment of the local congestion induced by heart disease, aneurismal pressure, and other similar causes; it occurs as a consequence of the disordered condition of blood which accompanies tuberculosis, albuminuria, gout, and other constitutional disorders; and it is a common attendant on continued fever, the various exanthemata, and those epidemic and endemic influences which occasion influenza and common catarrh.

The disease may occur either in an acute or in a chronic form. The

* See a paper by Mr. Gordon in 'Med. Gazette,' vol. iv.

acute disease is generally ushered in by symptoms of a so-called "cold"—chilliness, followed by heat of skin, general lassitude, and aching of the limbs, uneasiness about the frontal sinuses, sneezing, running at the nose, sore throat, and hoarseness. These symptoms are soon followed by a sensation of roughness and tickling in the windpipe, with frequent dry cough, more or less tightness or uneasiness behind the sternum, and soreness diffused over the front of the chest. The cough aggravates the pain in the chest, and when severe and dry, as it usually is at first, produces a distressing sense of tearing; but in the course of a few hours, or after the lapse of a few days, it commonly becomes looser, and is then accompanied by the expectoration of a thin, saltish, frothy mucus, sometimes streaked with blood. This gradually increases in quantity, changes its character, and for some days becomes glairy, semi-transparent, and of a faintly yellowish colour. Subsequently it assumes a greyish or greenish yellow tint, and is characterised by more or less opacity and viscidity. If the attack has been severe, or if the patient be weak, the secretion after a time becomes distinctly muco-purulent, and, in some instances, may even lose its glairiness altogether, and present the character of thoroughly opaque nummulated sputa. This is the course of events when the attack is passing off, which it usually does in favourable cases between the fourth and tenth days of the disease. But it often happens that the expectoration loses its opaque and puriform character, and again becomes frothy, glairy, and tenacious. This indicates a return or an extension of the inflammation; so that the precise character of the bronchial secretion forms a valuable guide to treatment. The respiration and the pulse are both increased in frequency; the former comparatively more so than the latter, but the precise frequency of the respiration, and the existence or non-existence of actual dyspnœa, varies with the extent of the bronchial affection, and the freedom with which the secretion is expectorated. When the larger and medium sized air passages are alone affected, and expectoration is free, and secretion not excessive, the oppression of the breathing seldom amounts to actual dyspnœa; but if the air passages are widely implicated, and the mischief extends to the capillary air tubes, the symptoms are much more urgent. There is then a dreadful sense of tightness and oppression at the chest, with dyspnœa and excessive restlessness; the patient sits erect in bed, or with his body bent forwards; the countenance is anxious, the pulse quick, the face flushed, and the skin hot, and some-

times moist, whilst distension of the jugular veins, and more or less lividity of the lips, cheeks, and general surface, extending even to the fingers' ends, betokens impediment to the circulation through the right side of the heart, as a consequence of the wide-spread mischief in the lungs. If the disease terminates favourably, the symptoms of oppressed breathing gradually pass off; the cough becomes less constant; the expectoration continues free, and the pulse and the respiration decrease in frequency. But in cases tending to a fatal termination a different train of symptoms is observed: exhaustion becomes a prominent feature of the case; the patient, unable any longer to support himself erect, sinks gradually in bed, until his head is scarcely raised above the shoulders; the lividity of the countenance increases, drowsiness ensues, the pulse rises in frequency, but decreases in force; the temperature of the body falls, and the surface becomes covered with a cold, clammy perspiration. Meanwhile the cough decreases; the breathing, though more rapid, is carried on more tranquilly; the sputa almost cease, in consequence of the want of power to expectorate, and the air passages become gradually more and more loaded. After lying for some time in an unconscious or semi-conscious state, varied only by wandering or occasional delirium, and sometimes by slight convulsions, the patient ultimately dies asphyxiated.

The morbid changes, induced, in the bronchial tubes, and the physical signs resulting therefrom, will be more readily understood in their mutual relation, when viewed side by side in the following table:

Morbid Changes.	*Physical Signs.*
1*st. Dry stage.*—Congestion of the vessels of the bronchial mucous membrane, with arrest of its natural exhalation or secretion, and consequent redness, dryness, and roughness of its surface; fulness or thickening of the substance of the membrane, dependent in part on turgescence of the vessels, in part on infiltration of the submucous tissue, and giving rise to diminution more or less irregular	1*st. Dry stage.* — *Inspection* shows nothing more than hurried respiration. *Palpation* seldom affords much information, but rhonchial vibration is sometimes perceptible. *Percussion* normal. *Auscultation.*—The respiratory murmur is simply exaggerated over the non-affected portions of the lungs; but over the affected portions the sound of respiration is coarse,

Morbid Changes.

in the calibre of the affected air tubes. These changes are seldom visible to the naked eye beyond the fourth or fifth division of the bronchi; but they may extend to the smallest ramifications of the air tubes, as in so-called capillary bronchitis. They are not confined to any portion of the lungs, but where the disease is not associated with tubercles, the lower and posterior parts are their most frequent seat. There is reason to believe that throughout the complaint, but especially in its early stages, there may be, and often is, spasmodic contraction of the circular muscular fibres of the bronchi.

2nd. Moist stage.—The congestion and dryness of the bronchial lining membrane, which marks the first stage of the attack, is followed by the secretion of mucus more or less frothy, viscid, and tenacious, as already described. The air tubes are generally somewhat dilated; their lining membrane is swollen, of a dark venous colour, and often softened, and ulceration may be detected occasionally on its surface; the mucous or muco-purulent secretion is more or less closely adherent to the membrane. Microscopically, the thin, watery portion of the secretion consists of a serous fluid, loaded with

Physical Signs.

dry, and harsh, accompanied and ultimately replaced by rhonchi, or sounds of vibration which, *cæteris paribus*, are of a higher or lower pitch, according to the size of the tubes in which they take their origin. Expiration is somewhat prolonged; vocal resonance not materially altered.

2nd. Moist stage. — Inspection reveals hurried breathing, with increase in the abdominal, and decrease in the thoracic expansion. If the dyspnœa be excessive, the lower portion of the sternum may even fall in during inspiration. The expiratory movements are prolonged and manifestly inefficient.

Palpation detects the existence of rhonchial vibration. The vocal fremitus varies; sometimes it exceeds, at others falls short of the normal standard.

Percussion almost normal, except in certain instances, in which congestion or œdema of the lung, with excessive collection of secretion in the air tubes leads to

Morbid Changes.

young abortive epithelial cells; the thicker semi-transparent portion consists of a tenacious fluid, resembling white of egg, containing mucous corpuscles, and patches of small-sized epithelium; the opaque variety contains epithelium, pus, and mucous globules, some few blood-discs, exudation cells, and granular matter. In the rare form of the disease, which has been styled "plastic bronchitis," lymph is poured out, forming a false membrane, which is ejected as a cast of the bronchial tubes. In fatal cases, the trachea and bronchi are found blocked up by secretion resembling that expectorated during life, and the lungs are more or less distended with air, in consequence of its inability to escape through the obstructed tubes. Emphysema frequently complicates the morbid changes which result from this disease, and so occasionally do pulmonary collapse and pneumonia.

Physical Signs.

dulness at the posterior and inferior portions of the chest; in others, which occur more especially in children, in which obstruction of a large bronchus by a plug of thickened mucus causes pulmonary collapse, with corresponding local diminution in the clearness of the percussion note; and in others, again, in which emphysema exists, and the clearness and fulness of the percussion sound is thereby abnormally raised.

Auscultation. — Pulmonary breathing impaired or suppressed, and replaced by rhonchi which are grave or high pitched, and by râles or sounds of bubbling, which are larger or smaller according to the size of the tube in which they originate. In capillary bronchitis, a fine or minute bubbling is heard during inspiration and expiration, most commonly at the bases of both lungs, posteriorly. Vocal resonance not materially altered.

Acute idiopathic bronchitis commonly affects both lungs simultaneously and attacks the middle, lower, and posterior parts; but when bronchitis is connected with the deposit of tubercles it ordinarily selects the upper lobes. Its prognosis is extremely serious at all ages; for although, when it is confined to the larger tubes, it does not usually prove fatal except to infants and to aged, debilitated persons, yet, when it involves the smaller air passages, and is extensively diffused throughout the lungs, it is attended in all cases by excessive dyspnœa, and is fraught with great danger even to the middle-aged and vigorous adult.

The latter, if seen early in the attack and treated actively, may be rescued in most instances from the effect of its fury; but so serious is it when occurring in infants and in aged, debilitated persons, that it proves fatal to above one half of those attacked. The inflammation is always most severe and most widely diffused when the expectoration is profuse; and the danger is great in proportion to the frequency of the respiration, the lividity of the surface, and the frequency and feebleness of the pulse.

Capillary bronchitis has been mistaken for pneumonia in consequence of the sound of bubbling heard in the former disease being sometimes as fine and crepitant as in cases of pneumonia. But the absence of severe rigors at the outset of the attack, the comparative coolness and occasional moistness of the skin, the clearness or resonance of the percussion note over the affected portions of the lungs, the absence of tubular breathing, the non-existence of rusty coloured expectoration, and the occurrence of râles during expiration as well as during inspiration, together serve as distinguishing marks of the former malady.

The treatment of acute bronchitis must be varied, not only with the type and severity of the attack, but with the age, strength, and constitutional power of the patient. If the attack declares itself as a "common cold," there is seldom much necessity in the first instance for any active treatment. A full dose of opium, either alone or combined with a tumbler of white wine whey, or some other hot alcoholic stimulant, will often suffice to check the disease; or, if there be so much febrile excitement as to render such treatment inexpedient, a hot air or hot water bath or a hot leg bath, followed by an ammoniated saline draught, combined with a full dose of sweet spirits of nitre and a few drops of the liquor acet. morphiæ and of the vinum antimonii will often induce a copious action of the skin and kidneys, and thus carry off and put an end to the attack. But whether the disease commences as a "cold," or declares itself in any other form, serious symptoms oftentimes arise which require more active means for their subjugation. In the sthenic type of the disease occurring in the adult, venesection may be practised if the febrile action runs high and the violence of the other symptoms is great; but in town practice it is rarely needed, and in most cases in which bloodletting seems desirable, observation inclines me to recommend cupping between the shoulders in preference to general venesection. The local abstraction of eight or ten ounces of blood in this

manner, repeated if necessary, and followed by dry cupping, serves in most cases to relieve the congestion of the vessels and to mitigate the severity of the symptoms. This is shown by the diminished frequency of the respiration. Relief is often obtained even whilst the cupping glasses are filling; and all that can be accomplished by bloodletting in this disease may be affected in this manner more safely and with less exhaustion to the patient than by the use of general venesection. But bloodletting, however practised, must be employed with extreme caution, and must be confined to robust persons of a healthy constitution, for in the unhealthy or asthenic it soon leads to exhaustion, and thus aggravates the danger by increasing the difficulty of expectoration. Even in the strong and vigorous, care must be taken not to withdraw too much of the vital stimulant, for the principal danger to be apprehended in the advanced stages of the disease is, that the patient may not have the requisite strength to give a hearty cough and rid his lungs of the phlegm which oppresses them. In children I believe it is never necessary to open a vein, for leeches on the chest form a convenient and effective substitute for phlebotomy.

Next to bloodletting, the most powerful and efficient remedy we possess is tartarized antimony. Administered in solution, in doses of a sixth, or a quarter of a grain, every three or four hours, it appears to conduce to free secretion and generally to mitigate the symptoms of the disease; and although it is difficult, if not impossible, to obtain statistical confirmation of the fact, I believe, that employed in the manner recommended, it is quite as efficacious, and much safer as a remedy, than when given in the heroic doses (four to twelve grains daily) recommended by some authors. Even forty-minim doses of the vinum antimonii will sometimes produce so much effect on the pulse as to render it necessary to reduce the quantity of the remedy, or to omit it altogether; and on more than one occasion I have known larger doses produce alarming depression without a corresponding beneficial effect. In acute cases, however, when the pulse is forcible, and more especially when the capillary air tubes are implicated, tartarized antimony should form an ingredient of every prescription; and if carefully watched, its administration, is never attended with danger except to infants and young children. Even to them it may usually be given without fear, but it sometimes proves wonderfully depressing to infants; and from two cases which I have myself witnessed, and from others communicated to me by medical friends, I am inclined to think that,

even in very moderate doses, it may sometimes prove almost poisonous. Therefore when it is given to infants its action should be watched with more than ordinary care.

With these provisos, it may be stated that, from the very outset of the attack, the administration of tartarized antimony in a saline diaphoretic draught is advisable; and of all salines perhaps the liquor ammoniæ acetatis is the most efficacious. Digitalis is often useful as an adjunct to the mixture, and in some instances produces a magically good effect. The air passages should be fomented by the inhalation of air charged with the steam of water, the bowels should be freely but not violently acted on, the secretions stimulated if necessary by mercury, and mustard poultices or turpentine stupes applied to the chest. As soon as secretion from the air passages is fully established, a large blister should be applied to the sternum, or to either side in front, and when it has risen the blistered surface should be covered with a large bread-and-water poultice. Some persons recommend the application of a blister between the scapulæ, on the ground that it draws off more fluid in that situation than when it is applied in front; but the distress it causes by preventing the patient from lying on his back in bed much more than counterbalances any extra benefit which would thus accrue. Blisters, therefore, should be confined to the anterior surface of the chest, whilst its posterior surface is stimulated by means of mustard poultices and turpentine fomentations.

As soon as the first severity of the attack is subdued and the expectoration becomes thicker and less copious, the quantity of tartarized antimony may be decreased, and then squills, or ipecacuanha, with paregoric, or a few minims of the liquor morphiæ acetatis, will be found useful adjuncts to the mixture. At a still later stage of the complaint, when the febrile symptoms have subsided, the tartarized antimony should be omitted altogether, and if the disease shows a disposition to pass into a chronic state, quinine with squills and conium may be administered, or a draught may be given containing full doses of cinchona with five grains of carbonate of ammonia and thirty or forty minims of the compound tincture of benzoin or of the balsam of Peru. This will generally facilitate expectoration, and so relieve the dyspnœa; but if any difficulty is experienced in unloading the chest, recourse should be had to the decoction of senega, or the mistura ammoniaci, both of which are powerfully stimulating expectorants, and may be given in aid of other reme-

dies. Under no circumstances should opium be given if the secretion is copious and expectoration difficult, as it tends to paralyse the action of the air tubes, and so interferes with the ejection of the muco-purulent matter. Nitric and sulphuric æther and the æthereal tincture of lobelia are useful adjuvants in cases which are attended by bronchial spasm, and ipecacuanha proves specially useful in children. If the patient be of a gouty habit, a few drops of colchicum wine, combined with fifteen or twenty grains of the bicarbonate of soda, prove a capital expectorant; and if of a strumous or consumptive disposition, the most efficient remedy in checking the bronchial secretion will often prove to be cod-liver oil.

If, as sometimes happens, tartarized antimony fails to check or control the symptoms, calomel and opium may be tried in repeated doses. It proves at best a very inefficient substitute for antimony; but I have known it serviceable in severe and obstinate cases, probably by stimulating the secretion of the chylopoietic viscera.

If, again, from whatever cause, the disease has produced extreme oppression of the breathing, with symptoms of approaching asphyxia and failure of the vital powers, our aim must be to sustain the failing strength, induce expectoration, and so relieve the pulmonary congestion. For this purpose strong beef tea, wine, brandy, and carbonate ammonia must be given internally, whilst repeated dry cupping between the shoulders, the application of mustard poultices and turpentine fomentations, and the use of various stimulating and vesicating ointments or liniments, are resorted to externally. In truth, there is no specific treatment for bronchitis; each case is a study of itself, and must be treated on general principles, due regard being had to the age, strength, and constitutional peculiarities of the patient.

Throughout the treatment the temperature of the room should be maintained at from 63° to 68° of Fahrenheit, and its atmosphere moistened, according to the sensations of the patient, by steam from a kettle, or by the evaporation of boiling water from a dish near the bed. Under ordinary circumstances the diet should be restricted to milk, whey, broth, beef tea, gruel, and arrowroot; but if the attack be of an asthenic character, a more generous diet must be had recourse to.

Chronic Bronchitis.

Chronic bronchitis may result from any cause which excites and keeps up subacute inflammation of the bronchial mucous membrane. Sometimes it occurs in sequel of an acute attack, which has been neglected or imperfectly subdued; sometimes it is preceded by general ill-health, and by a long succession of "colds" and coughs; sometimes it comes on gradually and insidiously, without any previous "colds" or feverishness, or any of the ordinary acute symptoms, and, sometimes, it seems to be a chronic ailment, dependent on the congestion of the bronchial mucous membrane, which is often associated with cardiac derangement. Further, it is a frequent accompaniment of tubercular consumption, for which it is liable to be mistaken, and it derives a special interest from the care required to form an accurate diagnosis between the two diseases.

Like the acute disease, it varies greatly in the character and severity of its symptoms. In one class of cases the affection consists of little more than slight occasional cough, with expectoration of a greyish or greenish, or yellowish white muco-purulent matter. It commences with the approach of winter, ceases or diminishes as soon as warm weather sets in, is not attended with pain in the chest, and does not materially affect the health; indeed, the patient often feels that he would be perfectly well if he could but get rid of his cough. When it thus occurs, it constitutes the mildest form of so-called "winter cough," and depends simply on passive congestion of the air tubes with somewhat increased quantity and altered character of the bronchial secretion.

In another class of cases the cough is more severe, of more frequent occurrence, and attended with much more profuse expectoration. It is most troublesome early in the morning, and on first lying down in bed at night, and sometimes, though very severe at those times, it is comparatively quiet throughout the day. At one time expectoration is difficult, and the sputa which are comparatively scanty, consist of stringy tenacious mucus, of a greyish or yellowish white colour, occasionally streaked with blood; at another it is easy, and the sputa are more copious, muco-purulent in character, of a yellowish green colour, having a faint unpleasant odour; at another, again, the sputa are profuse, almost wholly purulent, of a nauseous, and sometimes a fetid

odour,* usually running together into one mass, but often remaining separate, and forming distinct, nummulated masses, which sink or float in water, according as they happen to be more or less aerated. There is oppression at the chest, but seldom much pain, except after a fit of coughing; the pulse is not much hurried, nor is the respiration much more frequent than natural, but the violence of the cough often leads to vomiting, and its frequency to sleepless nights, and consequent exhaustion. As a natural result of the broken rest, there ensue loss of appetite, excessive debility, night sweats, and wasting. Indeed, in some instances, attended by frequent cough and profuse expectoration, the loss of flesh is so considerable as to suggest the existence of tubercular disease, and not a few cases of the form of disease now under consideration have been regarded as consumptive by the unwary practitioner. But the absence of any family tendency to consumption, the existence of the physical signs of bronchitis in all parts of the chest, the similarity of the signs on the two sides of the chest, the prominence of rhonchi, or sounds of vibration throughout the chest, as contrasted with râles or sounds of bubbling, together with the comparative slowness of the pulse and respiration, and the absence of night sweats, and the gradual cessation of wasting which is observed after the first few weeks of the attack, combine to stamp it with distinctive characters, which, in most cases at least, are sufficiently legible to the experienced practitioner.

In another class of cases, termed dry catarrh by Laennec, there is considerable tightness and oppression at the chest, with cough almost more frequent and distressing than in the last variety, but seldom accompanied by the same debility and wasting. The expectoration is exceedingly scanty, and composed of small, semi-transparent, roundish masses of viscid, pearl-like or starch-like mucus, and there is sometimes much dyspnœa, dependent, I believe, on the temporary collapse of certain portions of the lung tissue, consequent on obstruction of the bronchi by plugs of mucus. These cases which are often met with in gouty habits, are generally associated with more or less emphysema, are dependent on passive congestion of the bronchial lining membrane, and are seldom marked by much febrile excitement.

* The fetid odour simulates that produced by gangrene of the lung. It resembles the odour of the butyrates of ethyl, and is probably connected with the presence of butyric acid, which has been detected in such cases in the sputa. See a case reported by Dr. Laycock in the 'Med. Times and Gazette' for May, 1857, p. 480.

In a fourth class of cases an opposite train of symptoms is observed. The cough, instead of being dry, irritable, and frequent, occurs in paroxysms, at considerable intervals, and is accompanied by profuse expectoration, of a thin, watery, ropy fluid, which varies in opacity, but is usually somewhat transparent, resembling gum-water. This form of the disease, which has been styled bronchorrhœa, is generally observed in elderly persons, and sometimes leads to the expectoration of half a pint of fluid in the course of an hour. A patient under my care, a short time since, in the York Ward of St. George's Hospital, presented an excellent illustration of the fact. He rarely coughed more than once or twice in twenty-four hours, and then expectorated, within twenty or thirty minutes, more than half a pint of thin, semi-transparent ropy secretion.

In whatever form the disease occurs, it generally returns every winter, or, in old persons, lasts, with few intermissions, throughout the year. Further, whatever form of the disease prevails, an acute attack may supervene at any moment, and induce the symptoms of the ordinary disease. In all cases, but especially during the existence of the second variety, which is characterised by profuse secretion, the occurrence of such an attack is extremely dangerous; for in a patient whose breathing is oppressed by the accumulation of viscid secretion in the bronchi, such an attack is necessarily of an asphyxiating character, and in elderly persons, already exhausted by a long continuance of the chronic disease, it is always a question whether the strength will be equal to the long-continued cough and profuse expectoration which is necessary to free the air passages and relieve the breathing. Indeed, there is no more frequent cause of death amongst the aged than this form of disease, which, from its proving so fatal to elderly persons, has been termed "senile bronchitis."

The morbid changes to which chronic bronchitis gives rise, and the physical signs resulting from them, are collocated, for the sake of easy comparison, in the following tables. In the first instance those changes alone are described which accompany a simple uncomplicated attack; each of its more frequent complications is then described separately, viz. pulmonary collapse, dilatation of the bronchi, and emphysema. By this means it will be seen to what physical sign each morbid change gives rise, and the student will be thus enabled more readily to detect the existence of such complication, should it arise in the course of the disease.

Morbid Changes.

The mucous lining of the bronchi is swollen and congested, and of a deep venous red colour, either generally or partially, in streaks or in patches. The more asthenic the form of the disease, the more livid or darker coloured the membrane. Its surface is uneven, and often abraded, and its substance thickened; the longitudinal and circular muscular fibres of the bronchi are much developed, and the walls of the air passages generally are hard and thickened. The bronchi are clogged with secretion, more or less viscid, varying from a semi-opaque, ropy, sero-mucous fluid to thick tenacious mucus, or to true pus. The more viscid and tenacious the secretion, the more pertinaciously does it adhere to their sides.

Physical Signs.

Inspection shows defective expansion movements of the chest and prolonged and laboured expiratory movements.

Palpation.—Vocal fremitus not materially altered; rhonchial fremitus usually perceptible.

Percussion.—Sound not materially altered, unless the accumulation of thick secretion gives rise to obstruction of any of the bronchi, with consequent impaired inflation of the lung, and temporary dulness on percussion as its result. This is to be distinguished from the dulness of pleuritic effusion by the continuance of vocal fremitus, and from that of pneumonic consolidation by the absence of tubular breathing.

Auscultation.—The respiratory sounds are comparatively deficient over the entire chest, except, perhaps, for a moment after free expectoration, when they may be heard loud, harsh, and coarse, where a moment before they had been almost inaudible. Unless the air tubes have been just emptied by expectoration, the breathing is accompanied by every variety of rhonchus, chiefly of a sonorous, but sometimes also of a sibilant character. Large coarse râles or bubbling sounds, are also present, and exaggerated or supplementary breathing is heard in the unaffected portions of the lungs. The sounds

Morbid Changes.	*Physical Signs.*
	in any portion of the lung are constantly varying, and may either cease for a time or have their character altered by cough and deep inspiration. Vocal resonance is not materially altered in most cases, but is sometimes bronchophonic, and when dulness exists on percussion may be almost wholly absent.

Bronchitis, complicated by Pulmonary Collapse.

A few years ago Dr. Gairdner, of Edinburgh, pointed out* that a particular form of condensation of the lung, formerly regarded as the result of lobular pneumonia, but now acknowledged to be referable to pulmonary collapse, is a frequent and very serious complication of bronchitis. This fact, before unknown or practically ignored, is now regarded as exercising an important influence on the issue of bronchitis, especially in old people and young children, and it may be well, therefore, to point out the causes from which it arises and the mechanism by which it is brought about. The causes of collapse, may be briefly stated thus:—First, weakness or insufficiency in the power of inspiration; secondly, the presence of some obstruction—usually thick tenacious secretion—in the bronchi; thirdly, inability to remove the obstruction by the act of coughing. These causes are obviously more likely to exist under conditions of asthenia than in persons who are strong and vigorous; and thus it is that pulmonary collapse is found to complicate bronchitis in young infants and in aged or weakly persons more frequently than when the disease occurs in the vigorous adult. It may happen slowly or suddenly, in small patches or over an extensive portion of the lung, and is a frequent source of the sudden, but often temporary, accession of dyspnœa and oppressed breathing which is observed in the course of chronic bronchitis. Sometimes, when extensive, it is the immediate cause of death, and in fatal cases of bronchitis, at whatever age occurring, it is almost invariably an

* Dr. Gairdner "On Bronchitis," 'Edin. Monthly Journal,' 1850-51.

accompaniment of the attack. Its mechanism is very simple, and is admirably illustrated by an experiment of Messrs. Mendelsohn and Traube. They introduced a shot into the bronchus of a living dog, and the lung beyond the shot became collapsed and thoroughly emptied of air. A solution of gum injected into the bronchi produced the same result. The air gradually found its way out past the obstruction and was expelled during expiration, but was prevented entering again during inspiration. So it is with a plug of tenacious mucus at the bifurcation of a bronchus. It acts the part of a ball-valve in a syringe; each expiratory blast may dislodge it so far as to admit of the escape of air around it, but not so far as to prevent its falling back into its old position, and thus closing the passage against the ingress of air during inspiration. This condition of things leads to the emptying and consequent collapse of the air vesicles beyond the seat of obstruction, with attacks of dyspnœa more or less severe, according to the size of the portion of lung affected, and more or less enduring according to the period which elapses before the obstruction is removed.

The precise nature of the morbid changes and attendant physical signs will be evident from the subjoined table; but it must be remembered that they do not replace the changes and physical signs which mark an uncomplicated attack of chronic bronchitis, but are superadded to or are associated with them, and occur in certain portions only of the lungs:

Morbid Changes.	*Physical Signs.*
Pulmonary collapse may be confined to certain lobules scattered through the lung, in which "lobular" form it has been mistaken for the effect of pneumonia, and was formerly miscalled "lobular pneumonia;" or it may be diffused over a considerable portion of one or both lungs, usually their posterior parts, and may give rise to the appearance which was formerly recognised under the title of "carnified lung."	*Inspection* rarely furnishes any information except in young children, or when an extensive and superficial portion of lung is collapsed, in which case the corresponding portions of the chest cease to expand, and when the mischief is seated in the lower portions of the lungs, may be even retracted during inspiration. *Palpation* is never of any avail in the lobular form of the disease,

Morbid Changes.

When the mischief occurs in a lobular form the air vesicles are generally emptied of air, the collapsed portions are small and sharply defined, being mapped out by the interlobular areolar tissue, and are not only hard and shrunken, but depressed below the general surface of the lung. In the diffused form the collapsed portions are larger, and the degree of collapse varies from partial emptying of the air vesicles and consequent diminution of the normal crepitus, to entire emptying of the air cells and complete collapse. The collapsed portions are of a dark venous red tint externally, and of a deep plum colour internally; they are not crepitant under the finger, convey a feeling of solidity, sink when placed in water, and, unlike lung solidified by pneumonic hepatization, their cut surface is smooth, without a trace of granulation or morbid exudation. When the mischief is of recent occurrence, a small quantity of bloody serosity can be squeezed out, but even this contains nothing more than blood corpuscles, epithelium, and other elements of normal lung tissue, mixed now and then with a few pus or mucus cells; when it is of some standing the affected parts are shrunken, and no fluid can be obtained even by the firmest

Physical Signs.

and seldom of much use in the diffused form unless the mischief is extensive. In that case it will reveal cessation of vocal and rhonchial fremitus.

Percussion never yields any evident result in the lobular form of the affection, and rarely even in the diffused form. If, however, a considerable portion of the lung is implicated, the percussion sound is at one time completely dull, at another clear and resonant, according as the lung tissue happens to be collapsed or inflated at the time of the examination. Rapid alteration in the character of the percussion sound often accompanies this form of disease.

Auscultation reveals entire absence of the respiration over the affected parts, often followed in a few hours by breathing of the same character as exists in other portions of the chest, and accompanied by the same abnormal sounds.

Morbid Changes.

pressure. Unless the mischief is of such duration that the nutrition of the parts has become impaired and their tissue wasted, the collapsed portions usually admit of being restored in great measure to their normal condition by inflation through the bronchus. When atrophy and shrinking of the lung tissue takes place, vesicular emphysema usually occurs on the confines of the collapsed portions of the lung.

Physical Signs.

In some instances, the suddenness with which the dyspnœa comes on in chronic bronchitis, and the equally sudden manner in which it passes off is almost of itself sufficient to denote the existence of pulmonary collapse; and when the presumption which has thus arisen is verified by the occurrence of rapid variations in the percussion sound, and of equally rapid alterations in the sounds of respiration—their presence at one time, their entire absence at another—the presumption amounts to a certainty. In a case which I saw some years since with Dr. Latham and Dr. Dundas, the rapidity with which changes such as these took place was most remarkable. The patient, a gentleman aged fifty-four, would be breathing quietly in the morning, and would be suddenly seized in the afternoon with an attack of dyspnœa, threatening suffocation. These attacks occurred frequently during a period of three months prior to his death, and were invariably accompanied by complete dulness on percussion, and entire absence of breathing over a considerable portion of one of the lungs, usually the lower two thirds of the right lung, where previous to the accession of dyspnœa the percussion note had been clear and the respiration distinct, though accompanied by sonorous rhonchi and coarse râles. The attacks usually lasted from one to twenty-four hours, and were followed immediately by resonance on percussion, and by a return of breathing over the affected part. Sometimes, however, the attacks were of several days' duration, and then the change in the percussion note, and the return of breathing were more gradual. The same train of phenomena has been observed by Gairdner,

West, and other observers, especially in young children and in aged persons, and are of extreme importance, as bearing upon the prognosis of the disease, the probability of an unfavourable issue being greatly increased by the occurrence of pulmonary collapse.

Bronchitis complicated by Dilatation of the Bronchi.

It has been already stated that dilatation of the air passages takes place in all cases of bronchitis, and occurs more readily in children than in adults. In most instances, however, the dilatation is only temporary. The symptoms to which it gives rise pass off with the subsidence of the bronchial inflammation, and there is no reason for doubting that the tubes reassume their normal condition. But when the bronchial inflammation has been very severe, or of long duration, and has been accompanied by much spasm and difficulty of expectoration, the bronchial muscles sometimes lose their contractility, the air tubes become more than ordinarily dilated, and the dilatation does not disappear with the subsidence of inflammation, but, on the contrary, becomes permanent. This occurs most commonly in bronchi of the third or fourth divisions, in which the cartilaginous plates are few or absent.

Dilatation of the bronchi is followed, as a natural consequence, by condensation of the surrounding lung tissue, with obliteration of some, at least, of the air cells, and a corresponding diminution in the air-containing capacity of the lung; so that, even when unaccompanied by bronchitis, this affection results in habitual shortness of breath. And when, as is much more frequently the case, it is attended by bronchial irritation and profuse muco-purulent secretion from the air passages, it leads to symptoms, general as well as physical, which simulate those produced by tubercular disease of the lungs. It may be well, therefore, without further preface, to collate the morbid changes and physical signs by which this affection is usually accompanied.

Morbid Changes.

Sometimes one or more of the bronchi undergo dilatation almost throughout their whole extent, the enlargement being tolerably uniform, and often excessive; so much

Physical Signs.

Inspection. — Expansion movement diminished, and expiratory movement much prolonged. Occasional retraction or falling in of the chest walls over the affected parts.

Morbid Changes.

so, indeed, that tubes of the fourth or fifth division may exceed the main bronchus in size, and bronchi whose diameter in a healthy state corresponds pretty closely with that of a straw, may become as large as the distended finger of a glove. This form of dilatation is accompanied by thickening of the walls of the affected bronchi, and by hypertrophy of their circular muscular fibres, whilst the lining membrane of the tubes is of a dark red colour, and secretes a thick, tenacious, muco-purulent fluid. Sometimes the dilatation is not so uniform; the bronchi are irregularly sacculated, and distended in different parts of their course, and present a series of alternate enlargements and contractions, the enlargements varying in size from that of a bean to that of a chestnut or even larger. Sometimes, again, the dilatation is almost confined to the extremities of the tubes, which form large, thin-walled, globular cavities.* In the two last-mentioned forms of dilatation, the walls

Physical Signs.

Palpation.—Vocal fremitus usually increased, and rhonchial fremitus often perceptible. The force of the vocal fremitus varies, not only with the character of the voice, but with the size of the dilatation, the degree of pulmonary condensation by which the affected air tubes are surrounded, and with their freedom from secretion and proximity to the chest walls. If the tubes are obstructed, the fremitus cannot be felt.

Percussion.—The resonance is always abnormal. If the affected tubes are deep-seated, surrounded by much condensed lung tissue, or obstructed by an accumulation of secretion, there is decided dulness on percussion; if free from secretion, distended with air, and close to the chest walls, the resonance may be abnormally clear but shallow toned. In some such instances, the cracked-pot sound may be elicited.

Auscultation.—Respiration at first is harsh and rough, accompanied, and often masked by, râles and rhonchi. When the disease

* The most remarkable instance I ever met with of this form of dilatation occurred in the person of John Jenkins, who was admitted into the Hope Ward of St. George's Hospital, in November, 1843. Several of the bronchial tubes as they approached the lower portion of the lung became enormously dilated, and terminated in cavities large enough to contain a walnut. The membrane lining these cavities was of a deep brown colour, and continuous with the lining membrane of the bronchi. In several instances these dilated cavities communicated with each other, and so enormous was the dilatation at the lower part of the right lung that the appearance was presented of a large cavity intersected by portions of condensed lung. For further particulars see 'Hospital Post-mortem book' for December 28th, 1843.

Morbid Changes.	*Physical Signs.*
of the affected tubes are atrophied and attenuated, instead of hypertrophied, and are lined by a smooth membrane, which secretes a glairy mucus, or more commonly a thick, yellowish, muco-purulent fluid. In cases of old standing, the interlobular cellular tissue is infiltrated with a black or dark coloured indurated material.	is more advanced, it is either of a hollow, blowing character, and attended by gurgling, or else, if the tubes are free from secretion, it is of a clear, hollow, ringing, metallic quality. The vocal resonance may be diminished or altogether absent if the tubes are obstructed with secretion; of a clear bronchophonic or pectoriloquous character if they are unobstructed and free from secretion. In some instances, when dilatation is excessive, distinct splashing may be heard during cough.

It will be obvious, from a comparison of these morbid changes and physical signs, that the form of disease now under consideration is productive of alterations in the physical condition of the lungs in many respects analogous to those which occur in tubercular consumption, and is consequently accompanied by physical signs, many of which are identical with those which are met with in that disease. And when to this is added the fact that the expectoration in both cases is of a muco-purulent or purulent character, that in both instances hæmoptysis may occur,* and that diarrhœa, night sweats, and considerable wasting may and often do take place, it will be readily understood how difficult in some instances must be the diagnosis between the two forms of disease. Indeed, at a first interview, it is sometimes impossible to arrive at a positive conclusion on the subject; but careful investigation continued for some weeks will seldom fail to elucidate the question. The whole course of the disease and of its physical signs is different in the two cases. In phthisis the physical signs of the disease are most marked in the upper part of the chest, and in their fully developed form are often confined to the apices of the lungs; in simple bronchial dilatation they are commonly most marked in the mammary and inferior scapular

* Hæmoptysis is not common in these cases, but I have seen it occur to a slight extent in two cases in which subsequent *post-mortem* examination proved that no tubercle existed, and that there was no valvular disease of the heart.

regions. In the former disease the signs of pulmonary excavation are usually preceded by dulness on percussion over the diseased part; whereas in the latter dulness is not produced until after the signs of excavation have been established. In the one hæmoptysis and diarrhœa are the rule, in the other the exception; in the former the progress of the general symptoms and physical signs is often very rapid, in the latter their progress is usually very slow; and although emaciation may be considerable, it seldom proceeds to the same extent as in tubercular disease. In the one the sputa are not only muco-purulent, but contain portions of the yellow, elastic areolar tissue of the lung, which can be detected by careful microscopic investigation; in the other no trace of this tissue can be discovered, however careful the search, and however profuse or purulent the expectoration. Therefore when these two forms of disease exist separately, it can rarely happen but that daily observation of the symptoms and physical signs will enable the careful practitioner to discriminate between them; but when, as sometimes happens, tubercular disease coexists with bronchial dilatation, it is impossible to form an accurate diagnosis, and it is probable that the dilated bronchial pouches would be confounded with tuberculous excavations. The physical signs and general symptoms of phthisis being present, it is difficult to conceive any combination of circumstances which could justify a positive opinion as to the existence of dilated bronchi.

Treatment of Chronic Bronchitis.

The treatment of chronic bronchitis is a subject at once of exceeding interest and unusual difficulty; for whether the disease be simple and uncomplicated, or whether it be attended by dilatation of the bronchi, pulmonary collapse, or vesicular emphysema, in either case its treatment must be regulated as much by the constitutional peculiarities of the patient, as by the precise stage of the disease, and the actual symptoms and physical signs which present themselves to our notice. It is the neglect of these circumstances which leads to the frequent want of success in the treatment of this class of affections. In every case the chronicity of the disorder is a point which must be constantly borne in mind, nor must it be forgotten that the disease is frequently connected with an enfeebled state of system; that even in the more robust and vigorous it has a tendency to exhaust the patient, and induce "bad health;" that it frequently coexists with a weak and

damaged heart; and that when it proves fatal the issue is commonly dependent on want of power to rid the air tubes of the secretion which obstructs them, rather than on the occurrence of active inflammation. Therefore from the first it behoves us to be careful not to distress our patient unnecessarily. Bloodletting is rarely if ever needed so long as the disease maintains its chronic character; and even when an accession of bronchial inflammation occurs it is still inexpedient, and in most cases dangerous, to have recourse to active depletion. The application of a few leeches to that part of the chest where fine crepitating or small bubbling râles show that active mischief is going on; or, better still, the abstraction of a few ounces of blood, not exceeding four or five, by cupping, is as much as can be safely ventured upon; and if this is followed by repeated dry cupping, it will be found to be as much as—nay, more than—is commonly needed. But even if it should fail to produce the desired effect, bloodletting must rarely be had recourse to again. It would not prove either a safe or an efficient remedy. The failure of strength is so rapid in these cases, and the accumulation of mucus consequent on its failure so dangerous in its effects, that if abstraction of blood does not at once arrest the bronchial inflammation, recourse must be had immediately to other and less lowering means for its subjugation. Counter-irritation is now the external remedy on which we must rely; and when combined with the administration of antimony, squills, ipecacuanha, digitalis, nitric æther, the sesquicarbonate of ammonia, and other expectorants, and the inhalation of an atmosphere charged with steam—to keep up, as it were, a constant fomentation of the irritated membrane—it seldom fails to lead to satisfactory results. The anterior surface of the chest should be covered with a blister whilst its posterior surface is being stimulated by means of mustard poultices, turpentine fomentations, stimulating liniments, or counter-irritant ointments. Wine and other more powerful stimulants must of course be given if the patient manifests any tendency to sinking.

But when the disease is purely chronic, and uncomplicated by acute exacerbations, and is characterised by profuse muco-purulent secretion from the bronchi, bloodletting is never needed, and if employed is of no avail. The remedies here must have special reference to three objects, viz., 1st, to subdue the irritability of the bronchial mucous membrane and allay spasm; 2nd, to promote free or easy expectoration; and 3rd, to reduce the quantity of the secretion.

Inhalations of various kinds assist in fulfilling the first-named indication; emetics, expectorants and antispasmodics facilitate the second; and the third must be attained by means of tonics and certain remedies which seem to exert a special influence in restraining discharge from the bronchial mucous membrane. The steam of water, either alone or charged with naphtha, tar, or creosote vapour, or with dilute hydrocyanic acid, æther, or iodine, or else impregnated with the active principles of conium, hyoscyamus or stramonium, may be mentioned as inhalations which sometimes prove of service in diminishing the irritability of the bronchial membrane; and they possess these special virtues beyond all remedies administered by the mouth, that they admit of almost constant use, are applied directly to the seat of mischief, and tend to keep the parts fomented and free from the impact of cold and irritating air. They should therefore be employed in every case in which frequent and distressing cough and the occasional presence, perhaps, of a few streaks of blood, denote the existence of much bronchial irritation. The inhalations of hydrocyanic acid, conium and creosote are especially useful.

Expectorants are always beneficial, as facilitating the ejection of the bronchial secretion, which, if allowed to accumulate and obstruct the air passages, proves a source of serious danger. But it must ever be borne in mind that tartarized antimony, which is of great value in the acute form of the disease, is seldom needed in the purely chronic stage of the disorder, and if administered is apt to prove hurtful rather than beneficial. There are here no active symptoms to subdue, no need to promote the flow of secretion; our object is only to render it less tenacious, and, by thus promoting its separation from the sides of the bronchial tubes, to facilitate expectoration. Therefore, although in certain cases marked by more than usual inactivity of the skin, ipecacuanha may be found useful in slightly nauseating doses, we ought not to have recourse to tartarized antimony unless the expectoration shall have become frothy, or thin, watery and ropy, indicating the accession of more active mischief. Even under such circumstances it should be used very cautiously and discontinued as soon as the more active symptoms have subsided. Generally there is little vascular excitement, and our reliance will have to be upon the more stimulating expectorants. Squills, ipecacuanha, the liquor cubebæ, naphtha, the compound tincture of benzoine, and balsam of Peru, the mistura ammoniaci of the pharmacopœia, the decoctum senegæ, the sesquicarbonate and the hydrochlorate of ammonia, combined in

some instances with small doses of paregoric or of acetate of morphia, constitute some of our more valuable remedies. In certain instances conium proves more directly sedative and anti-spasmodic than the salts of opium, and it possesses the recommendation of exerting its soothing influence without checking expectoration as opium is apt to do. In other cases attended by much spasm, the æthereal tincture of lobelia and hydrocyanic acid afford excellent results; in others, again, which occur in persons of a rheumatic diathesis, iodide of potassium exerts a magical influence. In gouty persons and in some even who have never manifested any positive symptoms of gout, colchicum, combined with small doses of carbonate of soda, gives rise to effects which are sought in vain from other remedies; and so again, several instances have come under my notice in which in successive attacks no remedy has appeared to control the cough until after mercury had been freely given with a view of stimulating the liver. In these latter cases the tongue has been coated, and the pulse frequent, the cough hard and frequent, and the expectoration very scanty, consisting of small, starch-like masses; but as soon as a free secretion of bile has been established the cough has changed its character and become loose, the expectoration free and easy, the pulse has fallen and the dyspnœa has passed off. Some of these cases have occurred in gouty persons, but neither colchicum nor any other remedy has proved of service until after the action of mercury. In some such instances a hot air bath or an ordinary warm bath is extremely serviceable.

One word of caution should, perhaps, be added, respecting the administration of opium and its salts, in cases of chronic bronchitis, accompanied by profuse muco-purulent secretion. Its action in quieting the cough, and so checking expectoration, is apt to prove extremely dangerous to old and feeble persons; for with them a few hours' cessation from cough leads to an accumulation of secretion in the air passages, which their strength hardly suffices to get rid of, and many a person has died asphyxiated in consequence of the incautious administration of the sedative. In the young and vigorous it may be often employed with advantage, as soon as all inflammatory symptoms have subsided; but extreme caution must be exercised in its administration to the old, the feeble, or exhausted. In the former it tranquillises, allays irritation, and economises strength; in the latter it induces lethargy and suffocation.

When the bronchial secretion is very profuse, and, in spite of occa-

sional expectoration, tends to accumulate in, and obstruct the bronchi, producing blueness or lividity of the face and lips, emetics prove the most useful of all remedies. Not only are they speedy and effective in their action, unloading the bronchi in a few minutes more thoroughly and with less distress to the patient than cough of some hours' duration, but, at the same time, they promote a free action of skin, and copious secretion from the liver. They thus fulfil a double purpose, and often set up a train of actions, which lead to the happiest results. So long as there is not any undue vascular excitement, or any evidence of active inflammation in the lungs, no harm can accrue from their employment, and the only question to be decided is as to the emetic which shall be selected. If the patient is young and vigorous, a grain of tartarized antimony and half a drachm of ipecacuanha may be had recourse to without fear; and if, at the same time, there be any heat or dryness of the skin, it will probably be found a more effective emetic than any other; but, in the majority of cases, I prefer an emetic of sulphate of zinc. Not only is it more speedy and less lowering in its action, but it seems in some way to restrain the amount of bronchial secretion. I have noted this effect in so many instances, that I cannot entertain any doubt on the subject, and therefore, unless there are special reasons for employing tartarized antimony and ipecacuanha, I always have recourse to sulphate of zinc.

It will sometimes happen, that notwithstanding the use of emetics and stimulating expectorants, and, in spite of the action of sedatives, the cough continues frequent, and the sputa remain profuse. The medicines do their duty in facilitating expectoration, but they fail to control the secretion from the bronchi. In these cases emaciation often occurs, and the strength fails. To remedy this condition, tonics of various kind are absolutely required, and quinine, bark, gallic acid, the mineral acids, and cod-liver oil, are among the more valuable means at our disposal; and, if the patient be pale and cachectic, iron may be given with advantage. No medicine, however, proves more effective than sulphate of zinc in gradually increasing doses. Administered in combination with quinine, conium, and cod-liver oil, I have frequently seen it subdue an attack of bronchorrhœa in the course of a few days, which had been going on for weeks unchecked, in spite of remedies; and it is the more valuable, because its administration does not in any way interfere with the administration of other tonics or expectorants. Another astringent recommended by Dr. Alison, of Edinburgh, is

tannic acid, in two or three-grain doses. A generous diet must be had recourse to, and wine is often needed.

In some instances, more especially when the skin is inactive, bronchorrhœa appears to be kept up by the extra work which devolves on the lungs in consequence of such inactivity. Common experience has proved, that a free action of the skin affords very great relief in such cases, and thus the profession in all ages have recognised the value of diaphoretics in aid of expectorants; but it has been reserved for us, in these later days, to ascertain and appreciate the assistance afforded by a full and continued action of the skin in many varieties of chronic bronchial affection. The introduction of the Turkish bath has furnished us with the means of exciting and sustaining full cutaneous action without materially weakening our patient; and, already I have met with several instances in which a bath on alternate days has speedily checked and ultimately arrested a profuse bronchial flux, which had long continued in spite of internal remedies.

Nothing, however, proves so useful in many of these cases as change of air, or change of climate. It is not the mere change from a colder to a warmer atmosphere, which exercises a beneficial influence: a uniform temperature may be maintained by artificial means, and the atmosphere may be artificially dried or moistened without any notable improvement in the cough, whereas forty-eight hours passed in another locality, even though the air may be colder and more bracing, will be often followed by an entire change in the train of symptoms. The cough will diminish, and the secretion will be reduced, probably, to one half its former quantity. Indeed, I feel so strongly on this point, having seen immediate benefit result from change of air in numerous instances, that I do not hesitate to urge the removal of the patient as soon as it becomes evident that remedies are not exercising their usual salutary influence.

The only legitimate inference in such cases is, that some atmospheric condition of the locality in which the patient is residing, for the time at least, counteracts our efforts for his relief; and, under such circumstances, it is obviously our duty to recommend the adoption of a course which experience has proved to be often successful. Persons who are extremely prone to bronchitic attacks will do well to wear a respirator during the winter months, and to seek a milder climate in the south of Europe; but, for most persons, a residence abroad is unnecessary, as I am satisfied, that in almost every instance, a climate

may be found in our native island well suited to obviate and remove the customary winter cough. The question as to the precise localities to be selected will be discussed in a future chapter.*

Chronic Bronchitis complicated by Vesicular Emphysema.

Vesicular emphysema is a common accompaniment of bronchitis, and forms one of its most serious complications. It may exist to a slight extent without the concurrence of bronchial affection; but it rarely calls for medical interference, unless the bronchi are more or less affected. The patient may be more short-breathed than natural, the breathing being slow and laboured—not hurried; he may be unable to walk quickly, to run up stairs, and otherwise to exert himself as much as usual; and he may even find it difficult to maintain a conversation without stopping frequently to take breath. But although he may be conscious of dyspnœa, and may even make complaint of being short-breathed or asthmatic, he seldom has recourse to medical advice, unless troubled with cough, the result of bronchial congestion. Then the emphysema becomes, indeed, a distressing malady, aggravating all the evils of the cough, permitting an accumulation of secretion in the air passages leading to paroxysms of suffocative dyspnœa, and sometimes producing fatal consequences. Therefore, in a practical point of view, emphysema cannot be regarded apart from chronic bronchial affections.

Vesicular emphysema appears to be an hereditary disease,† and may

* See cap. iv, pp. 413-8, of this treatise.

† Of 43 emphysematous persons whom I examined with a view to this inquiry, 26, or, in other words, 60·4 per cent., acknowledged an hereditary taint. Their ages varied from seven to sixty-five, and it will be seen from the subjoined table that among the younger sufferers the influence of an hereditary tendency was most marked. Thus:

Age of sufferers at the commencement of emphysema.	Number of sufferers at the respective ages.	Predisposed by disease in either parent.	Per-centage.
7 to 20	5	5	100·
20 to 35	8	6	75·
35 to 50	14	8	57·1
50 to 65	16	7	43·7

A very similar result has been obtained by Dr. Jackson, jun., of Boston, U.S., who reports that no less than 18 out of 28 emphysematous persons had either a father or mother, or both, afflicted with emphysema, whereas 3 only out of 50 non-emphysematous persons sprang from an emphysematous stock. Again, he states that an here-

occur at any period of life, from infancy to extreme old age. It is most common, however, in persons beyond the middle period of life, and occurs more frequently in men than in women. Consisting of distension and rupture of the air-cells, with rarifaction and loss of elasticity of the lung tissue, it has been attributed by some authorities to the effects of forcible *ex*piration,* and by others to forcible *in*spiratory efforts.† Neither of these theories, however, appears to me to be exclusively correct. The pulmonary organs are so efficiently supported on all sides, that so long as they expand and contract freely, it is difficult to conceive how the fullest inspiration, or the fullest and most forcible expiration can occasion rupture of their air cells. But it is obvious that anything which produces contraction or imperfect expansion of certain portions of their tissue, whilst the adjoining portions are distended with air, must lead to inadequate support by the surrounding tissue of those portions of the lung in which the air is contained. Under these circumstances, the air-distended vesicles may give way under the strain put upon them, as the result of atmospheric pressure. Accordingly, emphysema is often found associated with pulmonary collapse, which leads to partial and irregular inflation of the lung. In these cases, it usually exists in the immediate vicinity of the collapsed portions, and may occur as an effect of forcible inspiratory efforts. So, again, when one lung has become contracted, emphysema is usually found affecting the lung on the opposite side of the chest, the tissue of the sound lung having given way under the atmospheric pressure during inspiration.‡ On the other hand, when, from the existence of viscid tenacious secretion in the air passages, or from any other cause, air is retained in certain portions of the lungs after adjoining portions have been partially or completely

ditary taint was distinctly traced in each one of 14 persons in whom the disease occurred from early childhood, but in 2 only of other 14 persons in whom the disorder did not manifest itself until the sufferer had attained to an advanced period of life.

* See paper by Dr. Jenner 'Med.-Chir. Trans.,' vol. xl., p. 25.

† Amongst the advocates of this doctrine may be mentioned Laennec, Williams, Watson, Rokitansky, and others, and especially Dr. W. T. Gairdner, of Edinburgh, who has ably enforced his views in a series of papers published in the 'Edin. Monthly Journal' for 1850.

‡ A case illustrative of this cause of emphysema on a large scale has been recorded by me in the 'Trans. Path. Soc.,' vol. xi, p. 15. It is unique in its character, and the details will well repay perusal. Another, in which the same cause appears to me to have operated, has been recorded by Dr. Barker, in the 'Med.-Chir. Trans.,' vol. xxxiv, p. 131.

emptied, a violent fit of coughing, as in chronic bronchitis or hooping cough, may produce rupture of the air cells, in the immediate vicinity of the empty portions of the lung during the act of expiration; and even when no obstruction exists in the lung themselves, violent straining, as first shown by Dr. Jenner, may possibly lead to the same result at the apex and anterior margin of the lungs, which receive least support from the thoracic parietes. No portion of the lung is exempt from this affection. Not unfrequently the posterior and lower surface of the inferior lobes present large emphysematous patches; but the upper lobes, and the anterior edges of those lobes are its favourite seat; and it is often met with in that situation when it exists in no other part. It is not a frequent accompaniment of phthisis, though the two diseases do sometimes coexist;* it is still less frequently associated with pneumonia; but it is a common sequel of obstruction of the bronchi, from whatever cause arising. Whatever the disorder with which it is associated, it is always a consequence of pre-existing disease of the lungs or air passages, and may be limited to a few vesicles, or to one or more lobules, or it may affect one or both lungs, or parts only of either lung. Giving rise as it does to serious impediment to the pulmonary circulation, it tends to produce hypertrophy and dilatation of the right side of the heart, with venous congestion of the head and face; and there are few advanced cases of emphysema in which the pulmonary symptoms are not greatly aggravated by the congestion thereby produced. In fact, the two forms of disease act and react on each other to a degree which, when emphysema is far advanced, renders the continuance of bronchial congestion and laboured breathing almost inevitable, and keeps up, even in the mildest weather, constant wheezing and a distressing, wearing cough. But the cardiac obstruction ultimately becomes a very serious malady, quite irrespective of its action on the lungs. The right ventricle, unable to empty itself with its accustomed ease, contracts more forcibly on the contained blood, its walls yield to the gradually increasing pressure, and its cavity at length dilates to such an extent, that the auriculo-ventricular valves become inoperative. Fearful attacks of palpitation then occur, especially during the paroxysms of cough and dyspnœa, the blood is thrown back on the system, con-

* For the details of a remarkable case in confirmation of the fact see 'Trans. Path. Soc.,' vol. xi, p. 15. The patient was under my care at St. George's Hospital, and though the symptoms of emphysema did not supervene until within a week of his death, the disease was developed to an extraordinary extent.

gestion of all the internal organs takes place, and œdema of the feet and legs is the natural result. Thus dropsical effusion is a frequent termination of this sadly afflicting malady.

When slight in degree, vesicular emphysema is seldom regarded as of much importance. It produces little inconvenience beyond shortness of breath on active exertion, and even in connection with chronic bronchitis it manifests itself chiefly by the amount and character of the dyspnœa it induces. The shortness of breath is out of all proportion greater than would be expected from the nature of the concurrent symptoms; but not only so, the respiration is effected with obvious difficulty, the breathing being slow, and the expiration much prolonged, and attended with considerable wheezing. In fact, the elasticity of the lung being seriously impaired, the power of emptying the air vesicles and bronchi is necessarily diminished; the bronchial secretion, therefore, accumulates in the air passages, and presents a still further obstacle to the egress of the air, and greatly aggravates the dyspnœa. To make matters worse, there is a tendency to bronchial spasm in these cases, so that the cough and dyspnœa are generally more or less paroxysmal.

But after all, when it exists in a slight degree, emphysema is an annoyance, rather than a dangerous or distressing malady, and rarely arrests attention or calls for medical advice, except in connection with an attack of bronchitis. But it is otherwise when the disease is more fully developed. The suffering and discomfort are then so great that the patient is forced to seek advice, even when not oppressed by bronchial inflammation. He is not only short-breathed, and distressed by a constant sense of fulness and oppression at the chest, but the difficulty of breathing is aggravated by spasm, giving rise to frequent paroxysms of suffocative dyspnœa. During these attacks, which usually come on at night, he pants and often struggles for breath, loosens his clothes, throws open the door, and rushes to the open window for air; in short, he suffers severe paroxysms of asthma; is unable to lie down in bed; and often passes days and nights in a sitting posture. Even during the interval between these attacks, his sufferings are very distressing. There is so much obstruction to the pulmonary circulation, that the bronchi are always more or less congested, and cough and wheezing are present to a greater or less extent. Nor is even this the sum of his troubles. The least accession of bronchial irritation excites a vast increase of the symptoms; irregularities of diet, and ordinary dyspepsia, produce the same effect. By giving rise to flatulence and abdo-

minal distension, they occasion an upward pressure against the heart, interfere mechanically with its action, and thus aggravate the existing obstruction to the circulation; whilst, by creating an obstacle to the descent of the diaphragm, they prevent the already distended lungs from receiving the limited supply of air which would otherwise pass into them at each inspiration. The unhappy patient bears about with him abundant signs of his discomfort and suffering. Though he seldom loses flesh, his complexion is dusky and congested, the lips are dark coloured and congested, the nostrils thick, and the face and neck full, or even swollen, from turgescence of the veins, and enlargement of the accessary muscles of respiration. His expression is suggestive of anxiety and distress, the respiration is slow and laboured, effected by the joint action of all the muscles of respiration; the shoulders are elevated, the back is rounded, and the slighest exertion aggravates his cough, and occasions distressing wheezing. The pulse is weak, and usually slow, except during the existence of bronchial inflammation; the impulse of the heart is often forcible, and a severe paroxysm of cough and dyspnœa will sometimes induce an attack of palpitation. In the more aggravated cases of this disease, attended by much dyspnœa, there is utter want of strength and incapability of bodily exertion; and the patient will often remark, that on the slightest exercise his breath and his strength seem to leave him instantly and simultaneously. The disease commences gradually, is chronic in its nature, is not attended by fever, but is usually accompanied by cough, with thin, frothy mucous, or watery expectoration. The appetite in most instances remains unimpaired, and the urine clear. The bowels are generally costive.

The following are the morbid changes and physical signs induced by this disease:

Morbid Changes.

These consist of dilatation, attenuation, and rupture of the air cells; atrophy, rupture, and gradual disappearance of their septa; coalescence of their cavities, forming air cells and bladders varying in size from that of a millet seed to that of a hen's egg; compression and obliteration of the capillary blood-vessels: consequent

Physical Signs.

Inspection usually discovers alterations in the shape and movements of the chest. The chest walls are abnormally rounded and prominent on the diseased side, or on both sides if both lungs are affected; or the bulging may be confined to certain parts corresponding to the diseased portions of the lung within. The infra-

Morbid Changes.

dryness and anæmia of the emphysematous tissue, which is always pale and sometimes quite white. Oil sometimes exists in the walls of the air cells; and it is probable that fatty degeneration, and consequent loss of elasticity of the lung tissue is one of the conditions which favours the production of emphysema.* The bronchial tubes are usually dilated in old-standing cases; their mucous membrane is pale, and their circular muscular fibres are highly developed. If emphysema is general, the lungs do not collapse when the chest is opened, but instantly protrude, as if too big for their bony case. Indeed, their volume is greatly increased, and leads to the displacement of all the contiguous organs. They push in front of and displace the heart; they overlap in front of the chest at about the level of the second costal cartilages, and forcing themselves downwards, depress the diaphragm and the liver below it; and they exert so much outward pressure that their surface becomes indented, sometimes deeply, with impressions of the ribs, which form their bony prison. They are of a piebald colour, and studded with irregular groups of air bladders, which, though not very visible in the interior of the lung, form glo-

Physical Signs.

clavicular, mammary, and sternal regions are the parts in which bulging is most commonly observed, but the whole of the anterior surface of the chest is sometimes unusually prominent, and its antero-posterior diameter much increased. The clavicles and scapulæ are elevated, and the former are often ill-defined, as in fat persons, in consequence of the filling up of the supra-clavicular space by the upward pressure of the emphysematous lung. The ribs are less oblique in their position than they should be, the intercostal spaces are wide, and sometimes well marked; but in advanced cases, especially in old persons in whom the intercostal muscles become weak or paralysed, the intercostal depressions are more commonly obliterated. In some few instances in which the general symptoms of emphysema are well marked during life, the lung is atrophied and no bulging or prominence of the chest occurs either general or local. The motion of the chest walls is remarkably small; the entire thorax is dragged upwards with the shoulders as one piece, but the expansion movement is diminished or almost *nil.* This arises from the fact that, in consequence of the loss of elasticity of the lung tissue, expi-

* See a paper by Mr. Rainey, 'Med.-Chir. Trans.,' vol. xxxi.

Morbid Changes.

bular projections on its surface; they are dry, pale, or even white in parts, crepitate feebly, or not at all under the finger; do not convey a feeling of elasticity, but feel soft and yielding like a down pillow. Their specific gravity is low, so that they float high above the surface of water like an air-distended bladder. Inflation of the lung through the bronchi fills up the spaces between these emphysematous bullæ, and renders the external surface smooth and even, showing that these projections result simply from the loss of elasticity in the tissue of the diseased parts, and the consequent non-escape of the air during the natural collapse of the lung. When emphysema is partial only, the morbid appearances correspond in character though not in degree with those observed when it is more generally diffused over the chest, and the amount of displacement of the thoracic organs is in strict relation to the extent and seat of the disease.

Physical Signs.

ration fails to empty the chest, and there is little room therefore for the introduction of fresh air, and consequently little dilatation of the chest during inspiration. The respiratory efforts are laboured and powerful; the intercostal and all the accessory muscles of inspiration are seen in full activity, but the breathing, nevertheless, is chiefly abdominal, and, in aggravated cases, the lower part of the sternum and the cartilages of the lower ribs may even sink in during inspiration.

Palpation.—Vocal fremitus varies; sometimes it is normal; more commonly it either exceeds or falls short of the average standard of health. Not unfrequently the heart's action cannot be felt in the pericardial region, and sometimes it is felt much lower than natural, even in the epigastrium.

Mensuration shows great increase in the peripheral size of one or both sides, and a remarkable increase in the antero-posterior diameter of the chest.

Percussion sound is morbidly clear, and of a somewhat tympanitic quality; the area over which it is heard is more extended than natural, and owing to the very slight movement of the lungs is not materially affected either by forced inpiration or forced expiration; the precordial region often

Morbid Changes.

Physical Signs.

emits a clear resonance; the chest walls are unusually elastic and resilient under the finger.

Auscultation proves that there is little or no air in motion in the emphysematous portions of the lung, for the inspiratory sound is either short and weak, or else altogether absent, whilst the expiratory sound, if not inaudible, is weak and remarkably prolonged —the ratio of the two sounds being as 1 to 3 instead of as 3 to 1, in consequence of the diminished elasticity of the lung tissue and of the obstruction offered to the egress of the air by bronchial spasm and by mucus in the air passages. Both sounds may be jerking and interrupted, and are often marked by sibilant or sonorous rhonchi, or by bubbling bronchitic râles. The vocal resonance varies greatly. Sometimes it is diminished or altogether absent; at others, it is loud and of a bronchophonic character. The latter I believe to be its natural condition in emphysema, and its diminution or absence to be referable to obstruction of the air passages by spasm or mucous secretion. The heart's sounds are feeble or altogether inaudible.

Laennec speaks of a dry crackling as a result of sudden inflation of the emphysematous bullæ; and both he and Dr. Walshe invest the "prominent air-distended nodules" with the power of producing "a dry, grazing, friction sound." I have never been able to satisfy myself as

to the existence of either of these phenomena in emphysema; and even if their occasional existence be admitted, I should doubt their being correctly ascribed to the causes mentioned. I have never seen or known of an instance in which the lung tissue, however damaged by emphysema, has been dry enough to crackle on expansion, and I do not think it possible that the smooth, soft surface of the attenuated lung could give rise to friction. The crackling I should attribute to the bursting of bubbles caused by the occasional passage of air through viscid tenacious secretion in the air passages—the dryness of its character being referable to the viscidity and tenacity of the secretion—and the friction sound, when it exists—as it rarely does—to attrition of the two surfaces of the pleural membrane roughened by inflammation.

Pneumothorax is the only disease with which emphysema can be confounded. In both forms of disease the affected side is rounded and prominent; in both, resonance on percussion indicates the presence of air in immense quantity beneath the chest walls; and in both, feebleness or absence of respiratory sounds proves that the air is either in very slight motion, or is pent up in some cavity uninfluenced by the act of respiration. This cavity may be the pleura, or it may be the distended bladder-like pouch of an emphysematous lung. A distinction, however, may be drawn without much difficulty. Pure pneumothorax is extremely rare, if indeed, it ever exists; and when accompanied, as it is almost invariably, by liquid effusion, its characters are well marked and distinctive.* But there are other means of distinguishing pneumothorax. It comes on suddenly instead of gradually, like emphysema; it produces more urgent symptoms of dyspnœa; it gives rise to greater bulging and to resonance of a more thoroughly tympanitic character than the ordinary resonance of emphysema; and whereas emphysema, when existing to such a degree as to simulate pneumothorax, invariably affects both sides of the chest, pneumothorax is necessarily confined to one side, as it would prove instantly fatal if it were to occur on both sides.

It is stated that subpleural emphysematous air bags have been known to give way, and produce an attack of pneumothorax, but I have never chanced to meet with such a case.

Emphysema is naturally a progressive disease. Accompanied as it is by compression and obliteration of the capillary blood-vessels, and consequently by œdema and atrophy of the lung tissue, it follows that

* See pp. 204-5 of this treatise.

each attack of bronchitis, and whatever puts a strain upon the weakened organ, is almost certain to be followed by some increase of the local mischief. Repair of the injured part is impossible, for its nutrition is hopelessly interfered with; and the utmost that can be expected is, that by carefully avoiding every act of straining, violent bodily exercise, and all other acts which are calculated to make an unusual demand on the inflation of the lungs, or on their power of resistance during respiration, we may in some measure prevent the *rapid* increase of the malady. But, slowly or quickly, it assuredly will increase, whatever steps may be taken to prevent it, until at length as the functions of the lungs become more and more impaired, the act of stooping, a recumbent posture, the slightest catarrh, an attack of dyspepsia with flatulent distension of the abdomen, or anything which even temporarily creates the slightest interference with the action of the heart and lungs, will suffice to induce a violent paroxysm of dyspnœa, palpitation, and general distress.

If, then, emphysema be irremediable, what can be done to relieve the sufferings of our patient? Much good may be effected by enforcing a mode of life calculated to guard against the various circumstances which aggravate the emphysema, or induce attacks of dyspnœa. Anything which puts the patient out of health is necessarily bad for him, and everything is beneficial which conduces to tranquillity of mind and body. Regularity of habits, moderation of diet, the avoidance of cold, entire abstinence from active exertion and mental excitement, are conditions essential to his wellbeing and comfort, and, when there is much disposition to catarrh, accompanied by bronchial spasm, removal to a warm climate during the winter months, and the use of Jeffries' respirator whenever the external temperature is low and the air keen, are conditions of great importance. When these precautions are steadily observed, life may be prolonged for very many years even in aggravated cases.

But active medical interference is necessary in many cases of emphysema. This disorder is apt to be complicated by bronchitis, and in almost every instance in which a doctor's advice is sought, is attended by bronchial spasm, and bronchial congestion, with profuse secretion of mucus. The removal or mitigation of these symptoms is of the last importance to the patient, as tending not only to promote his immediate comfort, but to prevent the further extension of the disease. The means to be employed for the purpose must necessarily vary, according

as the attack is characterised by more or less extensive bronchitis and pulmonary congestion, or by spasm of the air tubes. It is unnecessary to recapitulate what has been already stated respecting the treatment of bronchial inflammation, inasmuch as what applies to cases of simple bronchitis, holds good equally in respect to bronchitis complicated by emphysema. The only remarks which are called for in this part of the subject are, that extreme caution must be exercised in having recourse to depletory measures, and, that as soon as the more active symptoms have subsided, emetics, especially the sulphate of zinc, are extremely serviceable. Further, that with the view of relieving the passive pulmonary congestion which is a distinguishing feature of these attacks, repeated dry cupping between the shoulders, and the frequent employment of hot-water leg-baths are useful expedients. If the stomach be loaded or otherwise out of order, or if there be flatulent distension of the abdomen, emetics, followed by warm carminative medicine and a gentle purge, will prove of the greatest service. If, again, there be no evidence of stomach derangement, but nevertheless the dyspnœa be considerable, the wheezing excessive, and expectoration scanty, it is fair to conclude that the symptoms are attributable chiefly to bronchial spasm, and means must be adopted accordingly. Nothing proves more useful in such cases than a full dose of opium combined with half a drachm or a drachm of Hoffman's æther and twenty drops of the æthereal tincture of lobelia. Small doses of opium frequently repeated, which are of much service in ordinary catarrh, are of little avail to an emphysematous patient, whilst a full dose given as above directed will often act like a charm, subduing the cough at once, and speedily removing the wheezing and distress of breathing. The blueness or excessive venous congestion which accompanies a paroxysm of asthma resulting from emphysema is only a temporary effect of spasm of the bronchi; whereas when it occurs as a result of bronchitis unconnected with emphysema and bronchial spasm, it is the effect of obstruction caused by excessive loading of the air passages with muco-purulent secretion. Thus it is that in the one case lividity of the surface is not a bar to the administration of opium—nay, rather calls for the employment of sedatives and antispasmodics—whilst in the other, as before stated, it contra-indicates the use of sedatives, which have the effect of checking expectoration and of so increasing the loading of the bronchi. If opium fails to afford relief, belladonna, conium, stramonium, the æthereal tincture of the Cannabis Indica, and hydrocyanic acid may be tried, and each in turn may be found of service. Even when all medicines

taken internally fail to afford relief, belladonna, camphor, and stramonium will sometimes act beneficially if introduced into cigarettes or put into a pipe and smoked. The seed-pods of the Datura tatula smoked in this manner are more powerfully antispasmodic than even the pods of the Datura stramonium, and I have known them afford relief after stramonium had completely failed. The inhalation of æther and chloroform occasionally proves serviceable, as do also the fumes of burning bibulous paper which has been steeped in nitre. The inhalation of very dilute carbonic acid has also been reported as conducive to the alleviation of bronchial spasm, but I have not had any personal experience on the subject, and cannot conceive it likely to exercise a beneficial influence.

Interlobular Emphysema.

Interlobular emphysema is the name given by Laennec to a form of disease in which air is poured out from a ruptured air vesicle into the cellular tissue which exists between the lobules of the lungs. In this form of complaint the air is not necessarily confined to the spot where it was first effused, but may pass into the cellular tissue of the mediastinum, and thence into the cellular tissue of the chest and neck. The disease is seldom connected with vesicular emphysema, and, if not produced by puncture of the lung by means of a broken rib or some similar accident, it usually arises from rupture of one or more air cells under the influence of violent straining efforts, as during childbirth, in defecation, in coughing, and in lifting heavy weights. It may be recognised during life by the suddenness of its attack. Something is felt to give way in the chest during the act of straining; shortness of breath immediately ensues, and may be followed in a short time by an emphysematous condition of the neck or chest. If the air finds its way into the subcutaneous cellular tissue, it may be felt and heard crackling beneath the finger, and such an occurrence, coupled with the history of dyspnœa occurring suddenly after an act of violent straining, cannot fail to lead to a recognition of the disease. The idiopathic disease is said to be more common in childhood than in adult life, but under all circumstances it is so uncommon that I am not able from personal observation to speak confidently as to its treatment. Theoretically, however, nothing can be of much avail, unless the air has found its way into the subcutaneous cellular tissue, in which case a

few punctures will allow the air to escape, and thus will lessen the pressure which results from the deeper-seated emphysema. Therefore, if the dyspnœa is excessive, this plan ought to be adopted, but if otherwise, it is better not to interfere, inasmuch as the mischief tends to spontaneous cure. The air which has escaped is gradually reabsorbed, and the dyspnœa ceases coincidently with its absorption.

If the air should fail to pass into the cellular tissue of the neck or chest, the diagnosis of this affection is by no means easy, for under these circumstances it is almost impossible to distinguish this form of disease from ordinary emphysema. The suddenness of the attack forms the only other clue to the nature of the mischief; and as dyspnœa may occur suddenly from a variety of causes, this cannot be depended on as a means of diagnosis.

In this, as in the former variety of emphysema, Laennec thought that a "dry crepitant rhonchus with large bubbles" is to be heard, and that the subpleural air-pouches give rise to friction sound; but I have never been able to hear those sounds, and it is almost certain, theoretically at least, that emphysema in either form cannot cause their production. In this opinion I am confirmed by many careful observers of large experience. Dr. Watson "doubts exceedingly whether" this large dry crepitation "really occurs at any time," and Dr. Herbert Davies does not hesitate to remark that Laennec's statement "is not confirmed by later observers." Dr. Walshe indeed states it as his opinion that "it is *quite possible*" that both these sounds may be produced "if the surfaces of the interlobular spaces become prominent through distension;" but inasmuch as his statement implies that he has never met with a case in support of his theoretical "possibility," his opinion cannot be allowed much weight in opposition to so much extended practical observation as has been brought to bear on the subject at issue.*

Interlobular emphysema may be easily recognised after death. Bubbles of air of various sizes pervade the surface of the lung, elevating the pulmonary pleura, and producing more or less the appearance of foam. These bubbles, instead of being stationary as are the bladder-like projections formed by the dilatation of the air cells in vesicular emphysema, can be moved to and fro through the cellular tissue by pressure of the finger. Further, the lobules of the lung may be seen mapped out and sharply defined by small bubbles of air in the surrounding cellular tissue; and sometimes, if the emphysema be extreme, the

* See p. 304 of this treatise.

interlobular cellular tissue is thoroughly inflated, and the divisions between the lobules are of considerable width. Pneumothorax is said to have been produced by the giving way of one of the subpleural air-pouches.

Bronchitis complicated by Plastic Exudation into the Bronchi.

A complication which is sometimes met with in bronchitis, and is sufficiently remarkable to merit special notice, is the formation of plastic casts of the bronchial tubes. Just as in croup a plastic exudation takes place into the larynx and extends downwards towards the bronchi, so in this form of disease fibrinous matter is poured out into the smaller air tubes and their ramifications, and extends upwards towards the primary divisions of the bronchi. The exudation rarely reaches the larger bronchi, and never implicates the trachea and upper air passages. Indeed, the disease bears no affinity to croup, but is essentially a form of bronchial inflammation, connected probably with some peculiar diathesis which leads to the outpouring of concrete albuminous and fibrinous matter. It may occur in either sex and at any period of life; and although most frequent in delicate persons of a strumous or of a consumptive diathesis, it has been known to arise in persons of robust frame and healthy appearance, born of healthy parents, and who, neither before nor subsequently to their seizure, have exhibited any symptom of tubercular disease. Whatever its precise cause, this form of disease is exceedingly rare, and is of interest to the pathologist as well as to the practical physician. Not only are its symptoms of great severity and little under control, but its cause and true nature deserve further investigation.

The patient, possibly a healthy-looking person, is attacked with an irritative, hacking cough, which is not accompanied by much expectoration. After a period, varying from half an hour to several days, there is a considerable and rather sudden accession of dyspnœa, sometimes indeed of an alarming character, and attended by a distressing sense of constriction across the chest. The cough increases in severity, small fragments of white fibrinous matter mixed with ordinary bronchitic sputa or with blood-tinged mucus are spit up, and at length, during a violent paroxysm of cough, one or more white fibrinous casts of the bronchi are ejected, and the cough and dyspnœa in great measure pass off. The disease may then subside, or may continue for weeks or even months, marked from time to time by severe accessions of cough

and dyspnœa, invariably followed and ultimately relieved by the expectoration of fibrinous casts of the air tubes. Indeed, in one patient who came under my care at St. George's Hospital, and the particulars of whose case I communicated to the Pathological Society,* these attacks continued for several months, and recurred during a period of many years. The general health does not usually suffer much, and during the intervals between the attacks the patient's breathing may be unaffected. Sometimes even during a paroxysm of the disorder there is no pain at the chest, nor is there necessarily any heat of skin, or coating of the tongue, or loading of the urine, and the pulse, though frequent, may be soft or even weak. But the symptoms vary as much in kind and degree as they do in duration; for whilst, as already stated, there may be almost entire absence of febrile excitement and a very slight amount of mucous expectoration, there may be, on the contrary, excessive febrile distress and profuse muco-purulent bronchitic secretion, more or less tinged or mixed with blood. The secretion, however, is rarely tinged with blood, unless acute bronchitis or pneumonia be present; and perhaps the symptoms most generally met with are those which accompany that variety of chronic catarrh which is attended by scanty starch-like expectoration.

In former days the concrete masses which are expectorated in these cases were falsely termed bronchial polypi, and are described as such by Dr. Warren.† They vary from mere fragments to large pieces of from one to four inches in length, and may be either tubular or solid. Their ejection may be preceded and is often accompanied by spitting of florid blood. Sometimes the mucous expectoration is merely streaked with blood; at others the quantity of blood is considerable, and escapes in gushes; and this has led to the supposition that only the former variety of casts are the result of exudation, and that the latter are mere decolourised fibrinous coagula, resulting from ordinary hæmoptysis, and moulded in the smaller air passages.‡ But I am not inclined to coincide in this opinion. Without denying the possibility of fibrinous coagula forming in the bronchi as the result of ordinary hæmoptysis, it is undoubtedly the fact that such an occurrence is rarely observed even when hæmoptysis is very profuse; and inasmuch as when these plastic casts of the air tubes have been met with in any case, they have been so usually

* 'Pathol. Trans.,' vol. v, p. 41.

† 'Med. Trans.,' vol. i.

‡ See a paper by Mr. North in 'Med. Gazette,' vol. xxii.

not once only, as if formed by accident, but repeatedly, at intervals of days or months or even years, it is obvious that some special circumstances ought to be adduced in order to afford the slightest pretext for referring them to a cause which, to say the least, very commonly exists without giving rise to them. Moreover, long-continued observation at the bedside has led me to conclude that the white fibrinous casts now under discussion are invariably the products of true exudation. In two instances I have met with them without any hæmoptysis; and in the remarkable case which I reported to the Pathological Society, and which I have since had under my observation, the casts were sometimes rejected so soon after the accession of cough and dyspnœa that it is morally certain they would have exhibited some trace of the colouring matter of the blood if they had been attributable to bronchial hæmorrhage. Nevertheless, though some of them were solid and others tubular, and though in several instances their expulsion was preceded or else accompanied by more or less hæmoptysis, yet they were invariably white, without a trace of colouring matter. Further, I would remark that, however solid these casts appeared to be, they were always found on examination to consist of concentric laminæ, evidently deposited, or rather exuded at different periods, in successive layers; and if placed under the microscope they were seen to consist of amorphous granular matter, intermixed, *not* with blood globules, but with mucus corpuscles, compound granular cells, oil globules, and ovoid cells containing dark colouring matter such as exists in ordinary bronchial mucus.

In most of these cases of plastic exudation the prognosis is favourable. In two of the instances which I have met with complete temporary recovery took place; and in the case of the widow whose bronchial casts I brought before the Pathological Society, there has been complete temporary recovery after each of the repeated attacks which have occurred during a period of sixteen years. This patient was last in St. George's Hospital in 1858, when she again recovered thoroughly, after paroxysms of cough and dyspnœa, which for some weeks threatened her very existence. In an able report on the casts which I exhibited at the Pathological Society, Dr. Peacock has given an analysis of thirty-four cases, of which he has collected records; and of these no less than twenty are said to have recovered their health entirely; whilst in the ten who died, it appears, on investigation, that death was attributable either to phthisis, pneumonia, low fever, or violent hæmoptysis. In no instance, as far as

can be ascertained, did death arise directly from the presence of these plastic casts; on the contrary, there appears to have been little danger to life; for however urgent the cough and dyspnœa prior to and during the expulsion of the concretions, the natural efforts sufficed in every instance to expel them; and the urgency of the symptoms passed off as soon as free ingress was again afforded to the air.

The physical signs of this form of disease are peculiar. The formation of fibrinous concretions tends to obstruct the bronchi, and consequently to deprive certain portions of the lung of air. The natural result of this is weakness, or entire absence of breathing over the affected portions of the lungs. The percussion note varies in its degree of resonance or dulness, according as more or less air is pent up in the superficial portions of the lung; but if one of the larger air tubes becomes implicated, whilst as yet the portions of lung beyond it, and supplied by it, remain unaffected, collapse of those portions may take place, and complete dulness on percussion may ensue. This might lead the inexperienced practitioner to suspect the existence of pleurisy or pneumonia; but the rapidity of the disappearance of the respiratory sounds, the speedy occurrence of dulness on percussion, the absence of friction sound, and of ægophonic resonance of the voice, combine, with the non-occurrence of acute catching pain and other general symptoms, to distinguish these cases from pleurisy; whilst, in like manner, they may be distinguished from pneumonic consolidation by the rapidity of their occurrence, by the absence of tubular breathing, and by the non-existence of those general symptoms which usually mark the inroad of inflammation of the lungs. The ordinary signs of chronic bronchial inflammation are commonly present during these attacks; but acute bronchitis or pneumonia may supervene, in which case the physical signs of course undergo a corresponding change.

I know of nothing which can be relied upon to afford decided relief during the paroxysms of dyspnœa which precede or accompany the expulsion of these plastic casts of the bronchi. Calomel and opium, tartarized antimony, salines, alkalies, and blisters have been fairly tried without effect; and I have seen venesection and sedatives employed in vain. In like manner, though I have had the opportunity of watching one of these cases for many months, I have failed to satisfy myself that any class of remedies possesses the power of controlling the formation of these bronchial casts. The only medicine which appeared to prove serviceable was tartarized antimony, in moderate

doses, persevered in for a period of several weeks; and even that, on several occasions, proved utterly inoperative. But although, when the disease has once arisen, we cannot arrest its progress, I am convinced that we may do much to shield our patient from danger, by sustaining the general health, and warning him to take every precaution to avoid congestion and inflammation of the bronchial mucous membrane. Quinine, iron, and cod-liver oil should be given whenever his strength begins to flag; he should be taught to shun exposure to cold and damp, to wear one of Jeffries' respirators whenever he is likely to be subjected to variations of temperature, and on the slightest symptom of bronchitic irritation to remain indoors, in a warm atmosphere, inhaling the steam of boiling water, perseveringly, for hours. By these precautions I have reason to believe, that in one of the instances which have come under my observation, an attack was warded off on several occasions. Any bronchitic or pneumonic symptoms must be treated in the usual manner, without reference to the plastic nature of the secretion by which they are accompanied.

Epidemic Bronchitis, or Influenza.

The forms of bronchitis hitherto described are referable to endemic influences, and derive their characters, not from any peculiarity in the causes by which they are excited, but from the constitutional condition of the patient at the time of the attack, the pre-existent state of the respiratory organs, or the physical changes to which the disease accidentally gives rise. It is otherwise, however, in regard to epidemic catarrh or influenza. Its occasional prevalence in summer as well as in winter, and the facts that it occurs at long intervals in an epidemic form, pursues an erratic course, always spreading from east to west, but not necessarily affecting contiguous places at the same time;* that it disappears from the different localities within or about the same period —from four to six weeks—and that it produces fever, more or less severe, with disturbance of the nervous centres and great general prostration—all point to some specific poison as its proximate or essential cause. Indeed, the peculiarities by which its progress is marked are obviously attributable to the nature of its exciting cause, and there are few

* Those who desire to make themselves acquainted with the history of influenza, and especially with the course which the various epidemics have pursued, will do well to consult Sir Henry Holland's 'Medical Notes and Reflections,' and Dr. Robert Williams' admirable work on 'Morbid Poisons.'

circumstances within the range of our profession which, in a practical point of view, require more careful consideration.

The symptoms of influenza differ considerably in their character in different cases. In some persons catarrh is the prominent symptom; in others fever exists, almost unaccompanied by catarrh; whilst in the aged and weakly it sometimes happens that reaction does not ensue after the preliminary rigors, and that the patient dies in a few hours in a state of collapse, without the supervention of any of the ordinary symptoms of the disease.

An ordinary attack, however, greatly resembles common catarrh, and differs from it chiefly in the suddenness of its invasion, the rapid development of its symptoms, and the degree of depression by which it is accompanied. It is ushered in by chilliness, seldom amounting to actual rigors, with aching pain and soreness of the limbs, headache, and muscular and nervous prostration. In some instances angina is a prominent symptom, almost from the very outset of the attack; in others coryza is most strongly marked; whilst, in a third class of cases, disturbance of the alimentary canal, with loss of appetite, vitiated taste, nausea, or even vomiting, may prove to be the earlier and more troublesome symptoms. There is extreme uneasiness, general restlessness, and nervous prostration. Usually the skin is warm and moist, though, sometimes, towards evening, rather hot and dry; the face at one time is pale, at another flushed; the eyes stream with coryza; there is tenderness of the scalp, and aching of the jaw-bones; the alæ of the nose are red, and excoriated by acrid discharge from the nostrils, and the voice is altered, as in common "cold." At the same time the patient complains of intense frontal headache, of more or less nausea and loss of taste, of soreness and discomfort at the epigastrium, and of constriction and rawness across the chest, with frequent hard, tearing cough. If his throat be examined, it will be found to be of a dusky red colour, but not ulcerated, and seldom much swollen; the pulse is always weak, and usually, but not always, frequent; the tongue is moist, and coated with a white or a yellowish-white fur; the bowels are either costive, or else disturbed and relaxed; and the urine in most cases is clear and acid. Notwithstanding the pain and soreness at the chest, and the frequency and severity of the cough, it often happens that the physical signs of pulmonary mischief are not of a well-marked character. Sometimes, indeed, little more can be discovered than indistinctness of the respiration, with some prolongation of the expiratory sound; but,

commonly, the signs of ordinary bronchitis are present, and sonorous and sibilant rhonchi are audible in all parts of the chest. In some cases pneumonia occurs between the third and sixth days of the attack, and, in others, pleuritic inflammation is occasionally set up. Seldom, however, is the inflammation of a sthenic character. The pneumonic inflammation is not accompanied by rusty coloured sputa, and neither the pneumonia nor the pleurisy is commonly attended by pain in the side, or, at least, by any urgent increase of dyspnœa, or by the characteristic pulse of inflammation. Indeed, the mischief occurs in a latent form, and, if not discovered by a careful physical examination of the chest, will run on unheeded, until, at length, blueness of the face and lips, with clammy perspirations and coldness of the surface, indicate extensive pulmonary obstruction and failure of the powers of life.

In all cases of influenza muscular prostration and dejection of spirits are prominent features of the attack, and are infinitely greater than would have been expected from the local symptoms of the disease. The patients, if at all advanced in life, and not very vigorous, are often obliged to be supported, whilst subjected to a stethoscopic examination, and may even faint if kept long in an erect posture. Even the younger and more robust feel utterly exhausted and incapable of bodily or mental exertion.

The various epidemics of the disease have differed somewhat in the character of the symptoms produced. That of 1782 was marked by the extreme severity of the frontal headache and pain in the temples, by the amount of fever which accompanied it, and by the frequency of delirium. "The nights were passed in disturbed and unrefreshing sleep, frequently with delirium, which in general did not continue long. In some cases, however, it appears to have been the most alarming symptom of the disorder."* In the epidemic of 1837 there was seldom any delirium, but there was a great tendency to pneumonia of an extremely asthenic type; so that Sir Henry Holland described it as "scarcely maintaining true inflammation, yet simulating the character of it." So again, in some instances, the chief strain has been on the digestive organs; and excessive nausea, with frequent vomiting, tenderness at the epigastrium, and diarrhœa, have been the more prominent symptoms.

The attack usually lasts from four to ten days, and terminates in profuse sweating or diarrhœa, followed in favourable cases by gradual sub-

* 'Med. Trans.,' vol. iii, p. 69.

sidence of all the symptoms. But not unfrequently we find in its wake an abiding languor and debility, with cough and chronic bronchial irritation. So often does this occur, that it has become a matter of common remark that influenza is apt to lay the seeds of consumption. Nor are facts inconsistent with this common observation; for, although doubtless in many instances the epidemic disorder has done little more than favour the development of already existing tubercular mischief, yet it cannot be doubted that many strong and healthy persons have justly referred their ill health and consequent tubercular disease to the entire derangement of system and great vital prostration resulting from an attack of influenza.

To the aged and weakly influenza proves a very fatal disease, whilst amongst children and persons under forty years of age it rarely gives rise to fatal consequences, unless the patient is weakly and out of health at the time of the attack, or is reduced or weakened by injudicious treatment. But putting these considerations aside, and looking merely at the actual results, it cannot be doubted that influenza is one of the most fatal scourges; in proof of which, it need only be stated, that the French bills of mortality for 1837 prove that no less than 4800 persons succumbed to the complaint during the two months ending the 7th of March; and that, during the epidemic of 1557, 2000 persons are said to have died of it in a small town near Madrid, during the month of September.

In all epidemic disorders it has been proved beyond dispute that active treatment is seldom of much avail, and is often decidedly injurious. The same holds good in respect to influenza. In the milder cases it is seldom necessary to do more than confine the patient to the house, prescribe the free use of tepid diluents and entire abstinence from animal food and fermented liquors, employ a gentle purgative to unload the bowels, and a saline draught to promote the action of the skin and kidneys, and, if necessary, apply a mustard poultice or some stimulating liniment to the chest with the view of producing counter-irritation and relieving the cough. Even in the most severe examples of the disorder it is seldom judicious to have recourse to very active treatment; for whatever measures are adopted, the disease will usually run on unchecked in its course.* Therefore, as we cannot arrest the disease, our object should be to prevent the occurrence of any unto-

* In this I am confirmed by many authors. See Williams 'On Morbid Poisons,' vol. ii, p. 683.

ward symptoms rather than to waste time in vain attempts to subdue it or cut it short. So long as the symptoms are confined to headache, pains in the limbs, slight sore throat, hoarseness, and cough, the simple measures above recommended will usually accomplish all that can be done for our patient's relief. But should severe angina occur, should the bronchitis prove unusually troublesome, or should pleurisy or pneumonia supervene, more active medical interference is necessary in order to obviate serious mischief. And here it becomes necessary that sound discrimination should be exercised, and that a lesson should be learned from past observation. The supervention of pneumonia or pleurisy in these cases is sometimes accompanied by so much fever and vascular excitement as to suggest the propriety of free bloodletting. But experience has shown that these symptoms of excitement are speedily followed by symptoms of depression even in the younger and more robust, and that bleeding proves extremely dangerous, if not a fatal measure. "Experientia enim hoc comprobavit, omnes fere mortuos esse, quibus vena aperiebatur."* Even local bleeding by cupping or leeches is always a doubtful expedient. Seldom indeed, as far as my experience has gone, is bloodletting admissible in these cases. It is clearly inadmissible in the weakly and the aged; and I believe that even in the younger and more vigorous it is not needed for the relief of the symptoms. Assuredly it lowers the vital powers, and according to my observation it fails in relieving the inflammatory symptoms, whereas I have repeatedly traced the gradual disappearance of the signs of consolidation under the use of dry cupping, blistering, and turpentine fomentations, aided by internal remedies. Indeed I am satisfied that by these means we may accomplish, all that can be effected with safety to the patient. Nevertheless, some practitioners, as Dr. Robert Williams† reports, that "in general when the patient was young and the affection of the lungs limited to bronchitis, the substance being as yet unaffected, leeches to the chest, or cupping to a moderate amount, as ten or twelve ounces, were borne extremely well, and the symptoms were relieved." I am constrained, therefore, to admit that cases may occur, in which bloodletting, if judiciously employed, may prove of service, but at the same time I feel bound to caution the student and young practitioner who has not had much experience in these cases to exercise the greatest caution in the abstraction of blood. I

* This is a statement of Linnertus respecting the epidemic of 1580; *vide* 'Cyclopædia of Practical Medicine.'

† Loc. cit., p. 682.

have known rapid sinking follow bloodletting to the amount of ten ounces, and Dr. Walshe reports having seen "successive fainting fits" follow the application of a dozen leeches, in "a previously robust and healthy person."

Tartarized antimony and calomel, which are strikingly beneficial in sthenic pneumonia, are of questionable utility in influenza. The former is said to have proved successful in the hands of M. Hortloup, at the Hôtel Dieu, and Dr. T. Davies and others have spoken highly of the latter; but I cannot say that I have ever seen any marked improvement follow their administration. Indeed, in many instances a contrary effect has been observed. The patient's strength has failed rapidly under their influence, without any corresponding amelioration in the chest symptoms.

In short, the remedies on which alone I place much reliance in the treatment of this complaint are tepid diluents, freely employed, saline diaphoretic medicines, ipecacuanha and gentle purgatives, assisted if necessary by opiates, hyoscyamus, or other sedatives, and by dry cupping, blistering, and turpentine fomentations. If the patient exhibits the slightest symptoms of depression, sesquicarbonate of ammonia should be added to the mixture, and wine or brandy allowed in moderate quantity; and if there be any difficulty of expectoration, the decoctum senegæ or some other stimulating expectorant should be employed. As soon as the first fury of the attack is past, quinine should be substituted for the saline, and should be combined with squills and nitric æther, and during convalescence the salts of iron may be used advantageously.

In slight cases, the patient may be allowed white fish, light puddings, and strong beef tea throughout the attack; but in the severer forms of the disease, the diet should be restricted, in the first instance, to broth, gruel, or barley water.

In all cases, whether slight in character or severe, much care should be taken during convalescence. Tonics should be administered suited to the constitutional peculiarities of the individual; and if the slightest cough remains, some other means than mere internal remedies should be resorted to for its removal. Change of air, under these circumstances, is imperative, and if not delayed too long, will seldom fail to effect our object; but if our warning be disregarded, and no efficient means are taken to get rid of the languor, lowness of spirits, want of sound refreshing sleep, and cough, slight though it may be, which often follow this disorder, permanent ill health will ensue, and pulmonary consumption will probably terminate the patient's existence.

Bronchitis, associated with "Hay Fever," or "Hay Asthma."

There is yet another variety of catarrh which has been supposed to be connected with emanations from the *Anthoxanthum odoratum*, or sweet-scented vernal grass, and hence has been termed hay asthma, or hay fever. Its symptoms are peculiar, and resemble those produced in some persons by the powder of ipecacuanha, and sundry other substances. They consist of frontal headache, running at the eyes and nose, frequent paroxysmal sneezing, pricking sensations in the throat, excessive irritation of the air passages, with cough, usually dry, but sometimes accompanied by thin, watery mucous expectoration, soreness, oppression, and tightness of the chest, shortness of breath, and difficulty of breathing. Few persons are susceptible of the disease; but those who have once experienced an attack, are apt to suffer from it year after year, in May or June, when the grass is ripe, and haymaking is going on; nay, more, they will often experience an attack if brought into contact with the dust of dry hay at other times of the year. This, in short, constitutes a peculiarity of the disease, and together with the suddenness of its invasion, and the rapid development of its symptoms, serves to excite suspicion of its true character.

As might be expected, from the nature of the exciting cause, the duration of the hay asthma does not usually exceed a month or six weeks. Nevertheless, the complaint is so troublesome and distressing, that sufferers will have recourse to any expedient which seems to offer a prospect of relief. The most effectual remedy is avoidance of the source of irritation, as by taking a sea voyage, or by removal to the sea-side, out of reach of the smell of hay. This is the course very commonly adopted by those whose time is at their own disposal; but it is not in the power of every one to leave home; and to such persons it becomes necessary to administer medicines which shall enable the system to resist the influence of the matter which is irritating it. For this purpose, many remedies have been proposed. Mr. Gordon* reports two cases effectually cured by quinine and sulphate of iron, aided by the use of the shower-bath; Dr. Elliotson has seen relief from the inhalation of an atmosphere more or less charged with chlorine; Dr. Gream has spoken highly of the efficacy of Nux vomica; Dr. Watson has recommended arsenic; Dr. Walshe, the use of creasote inhalations; but these and all other remedies which have proved of service in certain instances, have utterly failed in others;

* 'Med. Gazette' for 1829, vol. iv.

and we are yet in want of a trustworthy remedy for this disease. In the only two well-marked instances which have ever come under my own observation, all the remedies already named had been employed in vain, and I was fortunate enough to alleviate my patient's sufferings by means of sulphate of zinc administered internally in rapidly increasing doses, whilst zinc lotion was applied to the eyes, and a lotion composed of eight grains of sulphate of zinc and an ounce of glycerine to the lining membrane of the nostrils. Relief was obtained so speedily and effectually that I am warranted in recommending a further trial of this treatment.

Spasmodic Asthma.

Spasmodic asthma is an affection dependent on spasmodic contraction of the bronchi, which gives rise to dyspnœa of a paroxysmal character. It has been termed indifferently asthma, spasmodic asthma, and bronchial asthma; but the term "spasmodic asthma" should alone be applied to the disease under consideration, inasmuch as there is a form of bronchial asthma, to which allusion will be presently made, in which there are strong reasons for doubting whether the symptoms are not attributable to paralysis rather than to spasm of the bronchial muscles. In spasmodic asthma the difficulty of breathing, which is accompanied by loud wheezing, may come on either suddenly or slowly, at any period of the day or night. Very commonly it commences during sleep, and after lasting for some hours, is terminated by expectoration. In this case it has been termed "humid" asthma; whereas, when the fit is brought to an end without expectoration, the case is said to be one of "dry" asthma. In either case the phenomena which constitute a fit of the disease are much as follow:—The patient probably wakes soon after midnight with a sense of tightness or constriction across the chest. The inability which he feels to expand the chest whilst he remains in a recumbent posture induces him to start up at once in bed; and a common posture for him to assume is that of leaning forward, with his elbows on his knees, and his head supported by his hands. In this position he will sometimes remain for hours, gasping for breath, his countenance meanwhile being anxious and distressed, the face red and congested, or else pale and rather livid; the eyes prominent and staring, the skin covered with a clammy perspiration, the extremities cold, the pulse small, feeble, and sometimes irregular. He coughs with difficulty,

so "short" is his breath, and so feeble his efforts; and if he attempts to speak, he can only articulate a few words at a time. The bowels are often somewhat relaxed, and act hurriedly, yet imperfectly, as if under the influence of spasmodic action; and the urine, which, at the commencement of a paroxysm, is copious, pale, and watery, becomes scanty and high coloured towards its close. If he finds relief in the posture above described, the patient will maintain it until the breathing becomes less laboured, and the paroxysm begins to pass off; but if he fails to do so, and experiences, as he often does, a desire for fresh air, he will open the door of his room, or throw open his window, and remain at it for hours, even in cold weather. When, at length, the fit begins to subside, he is much exhausted by the fatigue he has undergone, and generally falls asleep.

But his troubles do not cease with the termination of the paroxysm. The repose which follows is but a brief and imperfect respite from suffering. Though he may consider himself quite well during the intervals between the paroxysms, he is short-breathed, and unequal to active exertion; he is incapable of maintaining a continued conversation, and pants and wheezes if he attempts to stoop or to run up stairs. Nay, more, he is in constant dread of a fresh attack; for experience has taught him that, day after day, or for many nights in succession, the asthma will return with undiminished violence, and that when at length it ceases to recur, it will leave him weak and thoroughly exhausted.

It has been already stated that asthma is a spasmodic disease. It may be stated further, that it is an hereditary complaint; in other words, that the tendency to it may be transmitted from parent to child, through several successive generations. Like all hereditary disorders, it may occur at any period of life, and instances are not wanting of its existence in early youth. One of the most frightful examples of it I ever met with was in the person of a boy thirteen years of age. Nevertheless, the most common period for its development is between the ages of twenty-five and fifty-five; and it occurs more commonly in men than in women.

Valentin found that contraction of the rings of the trachea is induced by irritation of the par vagum; and there cannot be a doubt that the spasm of the bronchi, on which asthma depends, is excited by irritation of the filaments of that nerve connected with the lining membrane of the air tubes. Further, it cannot be doubted, that the tendency to spasm, or, in other words, to be affected by certain causes

of irritation, depends upon a constitutional peculiarity—an obscure and ill-defined condition of the nervous system—and that according as this peculiarity varies, so do the causes differ which lead to the production of asthmatic symptoms. Most asthmatic persons are affected by certain states of the atmosphere, not necessarily connected with its dryness or humidity, nor yet with its precise temperature, but probably connected with its electrical condition, or the presence or absence of ozone, or of other matters which have hitherto escaped detection. Some asthmatic persons can breathe freely in a close, damp valley; some on the summit of a hill. To one the fresh air of the country is unbearable; to another, the smoky atmosphere of a town. Nay more, so subtle and incomprehensible are the disturbing influences which give rise to the disease, that mid-London air is sometimes the most congenial atmosphere, and that mere removal from one house to another, or to a neighbouring street in the same town, has been known to put an end to a a patient's sufferings. Many persons are influenced by various circumstances which interfere with the action of the heart or disturb the pulmonary circulation; and others by the occurrence of mental excitement, or whatever tends to disturb the nervous system. Thus, a fit of anger, sudden distress, exposure to intense cold or to a storm of wind, active exertion, flatulent distension of the abdomen, and derangement of the liver and bowels, are one and all, in certain instances, productive of asthma.

Thus, then, it will be seen that whilst in every case the disease is dependent on spasm of the bronchial tubes, its exciting cause may differ widely in different instances. In one case the spasm may be of centric origin, or arising primarily from excitation of the nervous centres; in another, its source may be eccentric, that is it may originate in an impression conveyed to the par-vagum from the surface of the air passages. In either case it may be unaccompanied by organic mischief, or, in other words, may be purely spasmodic—a form of disease very rarely met with—and the patient may enjoy most perfect health during the intervals between the paroxysms, in which respect it resembles the disease already described under the title of hay asthma. But more commonly, it is associated with structural changes within the chest which impart to it a much more serious character, and render it impossible for the patient to obtain more than partial relief. Emphysema of the lungs, and organic disease of the heart and large vessels, are among its most frequent, or almost constant concomitants. And thus it happens that the sufferings of asthmatic patients, though aggravated in

paroxysms, are persistent to a greater or less extent. The asthma may be subdued for a time, but the pulmonary or heart affection will remain, and lead to shortness of breath and urgent distress on the slightest occasion. A little extra exertion, unwonted excitement, or an attack of indigestion, is almost certain to aggravate the symptoms and induce a paroxysm. Indeed, the symptoms by which an attack is usually preceded are, loss of appetite, flatulence, acid eructations, drowsiness after meals, chilliness, irritability of temper, and excessive languor; symptoms which denote derangement of the digestion, and circulation, and impairment of the nervous function.

Those persons who are affected with pure spasmodic asthma, may and do sometimes outgrow their complaint, or get rid of it by change of habits or locality; but those in whom it is associated with disease of the heart or lungs experience year by year an aggravation of their symptoms. The spasm, which characterises a fit of the disorder, tends naturally to increase the mischief in the chest, and that in turn reacts, and renders the paroxysms of asthma more frequent and more distressing. Nevertheless, the disease does not necessarily prove speedily fatal; on the contrary, with due care and attention, the short-breathed sufferer from asthma, will often drag on his miserable existence for years after his more vigorous companions have disappeared from the scene of their earthly labours.

It has been asserted by some authors that asthma and consumption never coexist, the one being antagonistic to the other. The statement, however, is erroneous; for although the two diseases are not commonly associated, the one having nothing in common with the other, occurring usually at a different period of life, and being excited by entirely different causes, it is nevertheless true that there is no incompatibility between them, and that many asthmatic patients have died consumptive. Three cases of the kind have come within my own knowledge, and few persons who have enjoyed extensive opportunities for observation can have failed to meet with similar examples. The utmost that can be truly stated is that asthma does not predispose to consumption, nor tubercular disease to spasmodic asthma.

The general symptoms of the disease are so characteristic that its physical signs become of little practical importance. They consist of excessively laboured respiration, with elevation of the chest walls, deficiency or almost entire absence of thoracic expansion, and occasional retraction or falling in of the lower ribs during inspiration. At the

same time there is almost entire absence of healthy respiratory murmur, which is replaced by sonorous or sibilant rhonchi. The only conditions under which vesicular respiration can be heard during the paroxysm are, if the patient has held his breath as long as possible, or has exhausted his chest by talking as long as he can without drawing breath, in both of which cases, as was pointed out by Laennec, the spasm is temporarily relaxed, a quiet inspiration takes place, and air can be heard entering freely into the lung.

In the treatment of asthma there are three points for consideration, namely, 1st, whether the disease is complicated by organic mischief in the chest, and if so what is likely to relieve that mischief; 2ndly, how to subdue the spasm and arrest the paroxysm; 3rdly, how to avert an attack or prevent its recurrence.

The first point is one which cannot be discussed at the present time, inasmuch as it involves the whole treatment of pulmonary and heart affections. It is obviously however of the greatest importance to determine whether there be organic mischief in the chest and to ascertain its precise nature, as the treatment of asthma, especially the preventive treatment, would be modified materially by considerations arising in connection therewith. Thus, if the disease were uncomplicated and purely spasmodic, we might safely permit our patient to take moderate exercise, provided he sedulously avoids those localities or those particular influences which experience has proved to be pernicious to him. Our efforts in such a case would have to be directed almost wholly to two points, viz., 1st, to discover a locality in which the patient can reside with comfort; and 2ndly, to invigorate his system, and render him less susceptible to the influences which excite the bronchial spasm. The first object can be attained only by repeated trials; the second, if attainable at all, is so, by a careful regulation of the digestive organs, by the frequent use of the shower bath, and by the exhibition of sulphate of zinc, quinine, arsenic, iron, and other tonics. Nothing however will countervail the influence of an atmosphere which does not suit the patient; so that the remedy we can prescribe for this variety of the complaint is repeated change of residence, until some locality be discovered in which he can breathe freely. In his search for such a spot we may often assist him by suggesting a a trial of places which differ widely in their atmospheric conditions. But additional measures are needed when the lungs are emphysematous, or the heart and large vessels more or less diseased. In such cases

active exertion must be avoided, and so must all influences, of whatever nature, which tend to accelerate the heart's action or disturb the respiration. Moreover, care must be taken to mitigate or ward off by medicinal agency any symptoms arising from the structural changes within the chest. Without due attention to these points our efforts to afford relief will be futile, and our patient will be subject to a constant recurrence of asthma.

When the paroxysm has once commenced, we can shorten or mitigate our patient's suffering only by remedies of an antispasmodic nature. Perhaps it would be more correct to say "by remedies which in the particular case in question prove antispasmodic in their operation," for relaxation of the spasm is in certain instances produced by means which do not usually exert an antispasmodic influence. Thus in cases which are connected with gout and rheumatism, colchicum and iodide of potassium prove valuable antispasmodics; strong coffee does so in others, and ipecacuanha, and many nauseants might be mentioned as sometimes efficacious. But in most cases our chief reliance must be placed upon sedatives and antispasmodics of another class. Spite of the congestion which often accompanies the earlier attacks of asthma, it is seldom advisable to have recourse to bloodletting; and so great is the prostration in advanced cases, that venesection is never admissible. Opium and the salts of morphia, belladonna, hyoscyamus, stramonium, and Indian hemp, the æthers, and the æthereal tincture of lobelia, digitalis, and hydrocyanic acid, make up the catalogue of our more valuable remedies, and great relief they oftentimes afford, though the efficacy of each of them varies in different cases. In several instances I have seen exceeding comfort derived from a mixture of opium and sulphuric æther internally, aided by the inhalation of chloroform; in others, the addition of belladonna to the mixture has appeared to exert a magical effect; and in a third class of cases, the administration of lobelia has given entire relief after the other remedies have failed. Indeed, lobelia is one of the medicines of which I entertain the highest opinion. One lady, somewhat advanced in years, who had been a martyr to asthma during the greater part of her life, assured me that she had been entirely cured of her complaint by two drachms of the æthereal tincture, taken in two doses at an interval of half an hour. She took these doses six years ago of her own accord whilst stopping in Leicestershire, at a considerable distance from any medical advice; the asthma subsided immediately, and since that time has not returned. Stramonium and other remedies had pre-

viously failed to relieve her, and so had small doses (ten to fifteen minims) of the æthereal tincture of lobelia.

Some authors have suggested doubts as to the safety of administering full doses of lobelia; and inasmuch as lobelia is a potent remedy, there are fair grounds for supposing that very large doses might give rise to disagreeable, if not dangerous effects. But I have employed it in full doses (twenty to thirty-five minims), in scores of cases of chronic bronchitis and asthma, and have never yet observed the slightest inconvenience, except occasional nausea, from its administration. I am justified, therefore, in stating, that under proper medical supervision it may be safely given in the the doses above-mentioned, and I can unhesitatingly assert, that in some cases at least it will be found extremely serviceable.

The Datura stramonium, however, is the remedy which appears to be most efficacious in checking the spasm, and relieving the asthmatic patient. Taken internally, in the form of tincture or extract, or cut into small pieces, and put into a pipe and smoked, the herb will often act like a charm, arresting the spasm, inducing expectoration, and restoring the patient to comparative comfort within a short space of time. Indeed, so speedy and so complete is the relief it affords, that asthmatic persons will often declare that they have little dread of a return of their complaint so long as they are provided with stramonium; but, like other remedies, it sometimes fails, and, if the paroxysm is strongly established before recourse is had to its assistance, the patient may find himself unable to smoke, and unable, therefore, to avail himself of its virtues.

The Datura tatula is another species of the same genus of plant which possesses valuable antispasmodic properties. Ten years ago I tried it with success in two cases, in which stramonium had failed, and other instances are on record in which it has proved superior to that drug. In this country, however, it has not been imported in any quantity, and up to the present time has been little used. But from what I have seen and what I have heard of its effects from others, I am satisfied that it well deserves a trial in obstinate cases.

In some instances a combination of stramonium, belladonna, and camphor may be employed advantageously in the form of a cigarette; in others, tobacco fumes, and the fumes arising from burning blotting-paper or from the burning of bibulous paper, saturated with nitre, will give relief; in others, again, the inhalation of æther and chloroform will serve to *check* the spasm, though it will seldom, if ever, put an end to the disease.

The Cannabis Indica has been lauded as a remedy, but I have not had any experience of its effects; digitalis proves useful in those cases especially in which the disease is complicated by heart affection; and hydrocyanic acid is a remedy which often acts beneficially in conjunction with soda and other antacid medicines, when the stomach is irritable and out of order. I have never known it arrest a paroxysm, but, on several occasions, I have seen a patient's sufferings relieved by its administration. Iodide of potassium in full doses (ten to fifteen grains) has been known to act magically in certain instances. Nitric acid in large doses, repeated at short intervals, is another remedy, which deserves a trial. I am not aware that it has ever been prescribed as a remedy for asthma; but, judging from the influence it exerts over the spasm of whooping-cough, it is fair to conclude that it may produce a similar effect in asthma. Its action is harmless, since the merest infants take it with impunity, and, even if it fails to relieve the spasm of asthma, it is likely to prove serviceable in preventing its recurrence; and this, after all, is often the most important point to which it is possible to attain in these cases.

Various other means have been adopted in obstinate cases in the vain hope of obtaining relief; amongst which, I may mention emetics, cold dash, or the shock of cold water, galvanism, hot leg baths, and the influence of an intensely hot atmosphere as in a Turkish bath. Each and all of them are said to have proved useful in certain instances, and possibly they may have done so; but it is notorious, that in many instances they have failed altogether, and in some have appeared to act prejudicially. The truth appears to be, that they are not equally adapted to every case, and that they each prove useful or prejudicial according as they are judiciously employed. As curative agents they are not entitled to our confidence, but in certain instances, they may prove useful adjuncts to other and more specific treatment.

Paralytic Asthma.

When describing spasmodic asthma, I stated that certain instances of asthma occur in which there is reason to believe that the symptoms are dependent on paralysis of the bronchial muscles, rather than on the existence of spasm. Thus, asthmatic persons are sometimes met with in whom the difficulty of breathing is obviously connected with expiration. They inspire with comparative ease, but labour in vain to empty their chest. There is comparatively little wheezing in such

cases, but not the less difficulty in expiration. In some such persons the habitual dyspnœa is doubtless connected, in part at least, with the existence of emphysema of the lungs; but, in several cases which I have watched during life, and have had the opportunity of tracing to the dead-house of St. George's Hospital, the dyspnœa has been excessive in relation to the emphysema discovered after death; and in others little emphysema could be detected, although the dyspnœa, the deficiency of the expansive movement of the thorax, and the imperfect emptying of the lungs during expiration, combined with extreme clearness and resonance of the chest on percussion, had appeared to indicate its existence. In these cases occasional exacerbations of the symptoms may arise; but the dyspnœa is more constant, and the attacks are less paroxysmal than in spasmodic asthma.

Whatever may be the cause of the symptoms in these cases, they are obviously unlike those produced by spasm of the air tubes, as typified in spasmodic asthma; and as Laennec has traced distension of the lungs with air to paralysis of the vagi nerves, and as direct experiment has confirmed the result of his investigation, it seems fair to conclude that the cases under consideration are referable to this cause. The absence of all evidence of bronchial spasm and the existence of air-distension of the lungs do not admit of a doubt, and in the absence of emphysema, paralysis of the bronchial muscles seems the only assignable cause. Possibly, the existence of this condition of the respiratory apparatus may serve to explain the beneficial effects which, in some cases of asthma, have been observed to follow the use of galvanism, cold dash, exposure to a cold atmosphere, and other similar influences.

Hæmic Asthma.

Before quitting the subject of asthma, it may be well to draw attention to certain cases in which a morbid condition of the blood is productive of much apparent distress and difficulty of breathing, irrespective of any lesion of the heart or lungs. The patient in these cases seldom complains much of shortness of breath; for although the frequency and apparent difficulty of his breathing are great, the dyspnœa does not really distress him. It resembles the laboured breathing of breathlessness from over-exertion, rather than that arising from true asthma. Ordinarily, the chest expands freely, there is little or no lividity of countenance, but nevertheless, the breathlessness continues.

The careful and experienced physician cannot possibly be misled by such cases, but they may mislead, and often have misled the student and the unwary practitioner into a belief in the existence of organic disease of the thoracic viscera. On this account they are deserving of special notice.

The characteristic features of this form of dyspnœa, are that the frequency of the respiration is not so great as its apparent distress would lead one to suppose; that in most cases it is not accompanied by lividity of countenance, or by any marks of imperfect thoracic expansion; that the ratio naturally subsisting between the pulse and the breathing is not seriously disturbed, and that an examination of the lungs and heart fails in affording any evidence of mischief calculated to explain the symptoms.

In truth, this variety of hurried breathing originates in a morbid condition of the blood, which is either impoverished or insufficiently oxygenated, or else charged with materials which it ought not to contain, and which exert a morbid influence on the thoracic viscera, and on the nervous centres whence they derive their power. Thus it is met with in certain cases of anæmia, and after excessive hæmorrhage, as also in gout, Bright's disease, and other blood disorders. The precise cause in any particular instance must be determined by the history and symptoms of the patient, but if care be taken in the investigation, a satisfactory conclusion may be easily arrived at.

The treatment must of course be varied according to the nature of the disease from which each case originates, but in every instance the patient should be well supported during the interval which must elapse before the quality of his blood can be improved, or the morbid materials with which it is charged can be eliminated and got rid of. In most cases the preparations of iron are of essential service.

Bronchitis associated with Whooping-cough.

Whooping-cough, or pertussis, is characterised by slight catarrhal fever, followed by a peculiar spasmodic cough, of which the paroxysms occur at uncertain intervals. It is met with sporadically at all seasons of the year, but sometimes prevails epidemically, thus leading to the inference that its cause must exist at all times diffused through the atmosphere, though varying at different periods in quality and intensity. Common report stamps it as infectious; and the evidence on this point is so strong

and conclusive, that although Laennec,* and some few members of our profession have hesitated to admit the fact, we may fairly state with Frank, that "nostro ævo nemo amplius de naturâ contagiosâ coqueluche dubitat." It may occur at all ages,† but is most common in childhood, for the reason that children are so susceptible to it, that few pass through early life without experiencing an attack.

The history of its introduction in the island of St. Helena,‡ proves that the disease may be propagated by means of fomites; and observations which have been made in several instances of the same sort, as also in regard to the communication of the disease from infected persons, have led to the conclusion that its period of latency is about five or six days. It may coexist with many other disorders, and is often seen concurrently with smallpox and measles,‖ and though usually occurring once only in the course of life, it has been met with twice in the same person.§ Some pathologists have ascribed it to cerebral irritation, and others to an affection of the pneumogastric nerve, but neither supposition is consistent with many acknowledged facts; and even if it be admitted that the peculiarity of its character is attributable directly or indirectly to nervous irritation, it is nevertheless true, as already stated, that the cause of that irritation—the essential or proximate cause of the disease—is an epidemic influence, a specific poison, infecting the blood, and acting through it on the nerves as well as on other parts of the body. Practically, therefore, if my opinion is correct, the disorder may be regarded as consisting essentially of bronchitic irritation, usually not very severe, excited by some epidemic influence, and accompanied by reflex spasm of the air passages. The course of the disease may be divided into three stages.—1st, the catarrhal; 2ndly, the spasmodic; 3rdly, the terminal, or stage of convalescence.

The *first stage* is marked by the usual symptoms of "common cold"—sneezing, running at the eyes and nose, hoarseness, oppression at the chest, irritation of the air passages, with dry or almost dry and often paroxysmal cough, impaired appetite, disordered bowels, and catarrhal fever. This stage lasts from three or four days to ten days or a

* 'Traité de l'Auscultation,' vol. i, p. 156.

† Dr. Watson in his 'Lectures' (ed. i, vol. ii, p. 63) alludes to a child born with whooping cough.

‡ See Williams on 'Morbid Poisons,' vol. i, p. 302.

‖ Williams, loc. cit., vol. i, p. 303.

§ Dr. Heberden relates a case in point; and there are few men of large practical experience who have not met with similar instances.

fortnight, and is not distinguished by any special features, so that it is practically impossible to determine whether, to use a nurse's phrase, the disorder may not "turn" to whooping-cough. In mild cases, the feverishness, coryza, and loss of appetite are almost absent.

The *second stage* commences as soon as the cough becomes distinctly paroxysmal, and is accompanied or followed by the characteristic whoop. The disorder now appears to consist essentially of bronchitic irritation with violent reflex spasm of the air tubes, which at uncertain intervals, by day and night, produces abrupt paroxysms of spasmodic cough. On the approach of the fit the child instinctively grasps his nurse or lays hold of a chair or table, and thus prepares, as he best may, to support the shock of the cough by which his whole frame is about to be shaken. As soon as the paroxysm has fairly commenced, the respiration becomes laboured to the greatest extent; the expiration consists of a succession of forcible, short, spasmodic coughs, which continue, without any intervening inspiration, until the air in the lung appears to be almost wholly expelled, the eyes seem about to start from their sockets, and the little patient turns red or even black in the face. Then the spasm momentarily ceases, and a prolonged and laboured inspiration takes place, accompanied by a loud cooing or whooping noise, denoting partial closure of the rima glottidis. As soon as inspiration is fairly completed, another spasmodic expiratory effort commences of the same character as the former; and a complete paroxysm, which consists of a succession of these alternate violent inspiratory and expiratory acts, may last from half a minute to a quarter of an hour. When the paroxysms are much prolonged, not only does the congestion of the head and face become excessive, but blood may burst from the distended vessels of the lungs, mouth, ears, and nostrils; convulsions may take place, and the urine and the contents of the bowels may be discharged involuntarily. Each fit is brought to an end by the occurrence either of vomiting or free expectoration, or by vomiting and expectoration taking place simultaneously, or sometimes, though rarely, through mere exhaustion, without the occurrence of discharge of any kind. The matter rejected from the stomach is of a glairy nature, semi-transparent and tenacious. That which comes from the bronchi is scanty, thin, and often streaked with blood in the earlier stages of the disease; more abundant, semi-opaque, ropy, or of a muco-purulent nature when the disease is more advanced. The former variety of bronchial secretion is expelled with difficulty, the

latter with comparative ease; and thus it happens that the earlier paroxysms of cough are commonly more severe and of longer duration than those which accompany the later stages of the complaint. As soon as the paroxysm has subsided the congestion of the head and face passes off, the pulse becomes quiet, and the respiration tranquil; so that in the course of a few minutes the little patient is again lively and apparently quite well, resumes his play or any other occupation which the cough may have interrupted, and takes his meals with a relish, as if nothing were the matter with him. This, at least, is the usual course of events in favourable cases; but in severe or unfavourable cases the patient remains feverish, pale, and exhausted, and shows a disinclination to food. In such instances there is too much cause for expecting some untoward complication, and good grounds for anxiety as to the issue.

The frequency of the paroxysms is usually found to vary according to their severity, the fits being most frequent when they are most severe. In ordinary cases, they return every hour and a half or two hours; but in some instances there may be only two or three fits in a day, whilst in others the paroxysms take place every twenty minutes or half an hour. They may occur spontaneously or without any obvious exciting cause, but more frequently they are occasioned by the act of swallowing, shouting, or laughing, by a fit of anger, or, in short, by any cause which influences the excito-motory system, and occasions reflex spasmodic action. After the third or fourth week the paroxysms usually diminish in frequency and severity, and about the eighth week the symptoms are so much mitigated that the third stage, or the stage of convalescence, may be considered to have commenced. Sometimes, however, the second stage may terminate at the end of three or four weeks, or, on the other hand, if improperly treated, it may continue for many months.

The third stage of the complaint is marked by diminution and ultimately by cessation of the symptoms. The paroxysms become milder, the intervals longer, the cough loses its convulsive character, the whoop gradually ceases, the expectoration alters its ropy appearance and becomes simply catarrhal, vomiting no longer occurs, and the patient's health improves. The average duration of this stage is from a fortnight to three weeks, but under unfavourable circumstances or inappropriate treatment it may persist for many months, the characteristic whoop remaining almost up to the last. Even after it has entirely ceased, the patient is for some time liable to a recurrence of spasmodic whooping in the event of his "catching cold" and having a cough.

In uncomplicated cases an examination of the chest does not afford much information. During the intervals of rest there will be found to be good resonance on percussion; and if the sounds of respiration are not altogether natural, they are only mixed with or replaced by rhonchi and moderate-sized râles—sounds which denote the existence of catarrh. Towards the close of a violent paroxysm the resonance of the chest becomes temporarily impaired, in consequence of the forced emptying of the air cells; at the same time the respiratory sound is entirely suspended, and is not perceptible in any part of the chest. During the brief snatches of inspiration which occur during the spasmodic fits of coughing, the sound of breathing may be sometimes heard, either natural, or mixed with wheezing; but during the prolonged whooping inspiration little vesicular breathing is audible. The air may be heard to rush into the trachea and larger bronchi, occasioning rhonchi, and if there be much fluid in the tubes, bubbling râles may also be heard; but the air does not permeate the structure of the lungs, and therefore the natural respiratory murmur is not audible. This arises from the conjoint action of two causes, viz., spasmodic narrowing of the glottis, and, spasm of the bronchial tubes; and as the action of these causes is limited to the duration of the cough, vesicular breathing is re-established, and the respiratory murmur may be heard as soon as the fit has passed off.

When the disease is mild and unattended by local inflammation, it seldom gives rise to fatal consequences; but when it is severe it is apt to be complicated by affections of the chest or head, which impart to it a very serious character, and often lead to fatal results. Acute capillary bronchitis, or pneumonia, or pleurisy may supervene, and may carry off the patient in a few days. The air cells may be ruptured by the violence of the cough, and the patient may die asphyxiated by the effusion of air into the cellular tissue of the chest; the pleura may give way, and pneumothorax may result; or convulsions, or even apoplexy may occur. Even if temporary recovery takes place, dilatation of the bronchi or emphysema may result from the long-continued straining cough, or the cerebral structures may not recover from the violent mechanical congestion to which they have been so long subjected. In this case the patient begins to squint, and may die hydrocephalic. It may be stated, however, that this chronic mischief is most apt to occur in very young children, and that immunity from it is obtained in proportion as the age is more advanced. Another complication which is apt to arise, and seriously increases the danger of the

disease, is infantile remittent fever, with a disordered condition of the bowels. Oftentimes, when this train of symptoms sets in, the motions are relaxed, and are either dark-coloured and offensive, or else pale, consisting of little else than mucus. It is difficult to determine whether these symptoms are attributable to the action of the morbific agent from which the whooping-cough originates, or whether they are not the results of a disorder engrafted on or arising coincidently with it. My own opinion, however, inclines to the former view, for the reason that the disorder of the bowels is observed only in severe cases, and on more than one occasion has appeared to me to vary with the severity of the other symptoms of whooping-cough.

The *post-mortem* appearances correspond with the symptoms observed during life. In uncomplicated cases little more is discovered than a congested condition of the mucous lining of the air passages, together perhaps with enlargement of the bronchial glands and slight effusion in the pia mater or into the ventricles of the brain, as a consequence of the long-continued congestion resulting from the cough. Even these morbid appearances are sometimes absent. But more frequently fatal cases are found to have been complicated by capillary bronchitis, pneumonia, or pleurisy, the usual pathological results of which are manifest after death. Pulmonary collapse—the so-called lobular pneumonia of former writers—is often present, as pointed out many years ago by Dr. Alderson.* In some cases there is considerable effusion of serum, and occasionally even of blood in the brain, and in cases complicated by bowel affection during life the stomach and alimentary canal have been found congested and Peyer's glands enlarged.

The character and duration of whooping-cough are apt to vary greatly, even when the disease occurs in an uncomplicated form. Some persons† have contended that it may "never put on any other form than that of a common catarrh;" and although it appears to me that the grounds on which this opinion rests are not sufficient to warrant such a positive statement, and that we should do wrong to recognise as pertussis any catarrh which is unaccompanied by the characteristic whoop, still it cannot be denied that the complaint is often exceedingly mild, and is sometimes limited to catarrh of two or three days' duration, with a few paroxysms only of spasmodic cough and stridulous inspiration. More commonly, if unchecked by proper reme-

* 'Med.-Chir. Trans.' vol. xvi, p. 91.

† Cullen and others.

dial agents, it persists for three or four months, and may run on in extreme cases for six or eight months, or even longer.

The treatment of whooping-cough is a subject which deserves very serious attention. It is commonly supposed that the disease has a definite course to run, which will be longer or shorter according to the severity of the attack, and which is not to be checked or arrested by remedial agents. The advocates of this doctrine, amongst whom may be named the major part of the profession, confine their efforts to the prevention or subjugation of any untoward symptoms, and to the mitigation of the severity of the cough. They give saline medicine and a slight sedative with ipecacuanha, or, if necessary, a little tartarized antimony; occasionally an emetic is administered; the bowels are regulated by means of a gentle purge; and stimulating embrocations are employed down the spine.

Now, there cannot be a doubt that in the majority of cases this plan of treatment, if judiciously carried out and aided by a careful avoidance of cold and by due regulation of the diet, will suffice to conduct the patient safely to the termination of the disease. But it is equally certain that the average of cases will persist, under this treatment, for a period of from eight to sixteen weeks, and that some at least will continue for a much longer period. It behoves us, therefore, to inquire whether some more efficacious plan may not be discovered.

In the year 1847 I was led to try the effect of sulphate of zinc, in rapidly increasing doses, with the view of checking the spasmodic cough. Regarding the complaint as consisting essentially of bronchitic irritation, usually not very severe, accompanied by reflex spasm of the air passages as its prominent and more important symptom, it occurred to me that sulphate of zinc might control it, if it did not put a stop to the whoop; nor were my expectations disappointed, for the remedy succeeded admirably, and I therefore adopted it in all cases of whooping-cough which subsequently came under my care. In four only of fifty-seven cases in which I had the opportunity of testing its virtues did it fail in giving marked and speedy relief. In one of these five—a male adult—it utterly failed; in two it could not be tolerated by the stomach; and in the remaining two it did not materially lessen the paroxysms in the course of three weeks, and therefore other medicines were substituted for it. In twenty-five of the remaining fifty-two cases, the severity of the paroxysms was greatly lessened by the end of a fortnight, and the whooping had ceased before the expiration of a month. In eleven it

continued five weeks, in nine of them six weeks, in five it did not cease before the end of seven weeks, and in two it continued into the eighth week. In no instance did the disease persist after the expiration of the eighth week.

These results are sufficiently striking, and contrast most favourably with the ordinary plan of treatment. Indeed, they seem to indicate the exercise of a directly remedial influence, and to suggest the idea which has more than once forced itself upon me, that the disease may be kept within very narrow bounds, and in most cases may be almost limited to the duration of an ordinary attack of bronchitis.

Thus, then, until the end of the year 1858, when I discovered how readily children tolerate large and increasing doses of belladonna,* I trusted exclusively to sulphate of zinc. Since that time I have combined the zinc with belladonna, and have obtained even more satisfactory results. The whooping has been controlled in a remarkable degree, the severity of the disease has been much diminished, and its duration lessened. Indeed, if further observation verifies my hitherto limited experience of this mode of treatment, it may be fairly stated that zinc and belladonna, judiciously employed, are capable of reducing the average duration of an uncomplicated attack of whooping-cough to three weeks or a month.†

My plan of proceeding is as follows :—During the catarrhal stage of the complaint, the patient is kept in a warm and equable atmosphere ; the diet is limited to milk and beef tea or broth ; the bowels are regulated by mild aperients ; salines and ipecacuanha are prescribed ; and mustard poultices, or even blisters, are applied, if necessary, to the chest. Leeches are rarely employed, and bleeding from the arm is never practised. As soon as the whoop declares itself, a draught is given every three or four hours, containing half a grain or a grain of sulphate of zinc, and a sixth of a grain of extract of belladonna to two drachms of syrup of orange, in from two to six of water, and an additional grain of the sulphate of zinc, and an additional sixth of a grain of belladonna, are added to each dose daily, or every alternate day, until the quantity taken daily amounts to from six grains to a drachm of zinc, and from two to six grains of the extract of belladonna, according to the age of the patient.‡ To children under a twelvemonth old I have never adminis-

* See a paper of mine in vol. xlii of the ' Med.-Chir. Trans.'

† See ' Lancet,' for July 28, 1860, p. 85.

‡ The only precautions necessary to be observed in prescribing full doses of bella-

tered more than ten grains of the zinc and two grains of the belladonna, daily, which were given in doses of a grain and a quarter of the zinc and a quarter of a grain of belladonna every three hours; whilst for children of eight or ten years of age I frequently prescribe half a drachm, or two scruples of the zinc and six grains of belladonna. If the dose be gradually and cautiously increased, the medicine will not occasion sickness; and as it neither heats nor excites the patient, it may be given as soon as the true nature of the complaint is ascertained. Its administration, however, need not preclude the exhibition of other remedies; and if there is feverish heat of skin and persistent quickness of breathing, indicating inflammation of the lung, or if the bronchial flux is great, and oppresses the breathing, it is always prudent to have recourse to auxiliary measures. In the former case it is my practice to administer the vinum antimonii in doses varying with the patient's age; in the latter, if sickness does not occur spontaneously, to order a mixture containing the vinum ipecacuanhæ, of which a dose is to be taken every evening sufficient to cause vomiting. Nothing unloads the air passages so thoroughly, promotes easy expectoration, and gives so much relief as free vomiting, and nothing, I believe, conduces more directly to the safe and favorable progress of the disease. Nevertheless, it is essential to guard against the production of sickness by the sulphate of zinc, as, if nausea is once created by its agency, the stomach will thenceforth refuse to tolerate it, and its use will have to be abandoned. Therefore it is prudent to administer it alone, in the manner recommended, and, if necessary, to give the vinum antimonii as a cough drop, with a little extract of conium or syrup of poppies.

Various remedies have been recommended by authors, and some have been vaunted as specifics. Amongst the former may be mentioned the whole class of sedatives and antispasmodics, including opium, henbane, hemlock, and lettuce, asafœtida, valerian, musk, and camphor, chloroform, and hydrocyanic acid. Amongst the latter, alum and nitric acid. The former are sometimes useful, if judiciously administered, just as they would be under similar circumstances in any other disorder; but they certainly do not materially hasten the period of convalescence, and

donna, are—1st, that the dose of the medicine be *gradually* increased; and, 2ndly, that the quantity given daily be administered in divided doses at intervals of not less than three hours. The mere occurrence of dilatation of the pupils need not be considered a bar to its exhibition. For full information respecting the action and mode of administering full doses of this drug, reference may be made to a paper of mine published in vol. xlii of the 'Med.-Chir. Transactions.'

do not deserve any special notice in connection with this disorder. Of the latter, I know little, except by repute. Alum is a very popular remedy, and appears, from the concurrent testimony of many intelligent observers, to mitigate the disease and shorten its duration. Nitric acid, in like manner, has many advocates, who assert that the disease yields rapidly to its influence. Dr. Gibb in this country, and Dr. Arnoldi, of Montreal, may be named as among those who have spoken of it in the highest terms, and who assert that, if given in full doses, (half a drachm to two drachms daily,) it subdues the disease in the course of three weeks. I am unable, from personal observation, to bear witness either for or against it; but the reports which have reached me of its efficacy are so encouraging as to induce me to consider it worthy of a trial. If the statements made respecting it are borne out by further observations, it must be regarded as a most valuable method of treatment. Garlic, cochineal, cantharides, digitalis, and many other remedies, have attracted attention at one time or another, but they have not obtained the confidence of the profession, and there is not sufficient testimony in their favour to lead to their employment in preference to the medicines already mentioned. Counter-irritation to the chest and spine has been much extolled by many writers, and there are few practitioners who do not employ stimulating embrocations during the spasmodic stage of the complaint. Some of the applications which have been recommended, such as the tartar emetic ointment and croton oil liniment, are scarcely justifiable in early childhood, and do not, I believe, give greater relief than the less formidable compound camphor and soap liniments, or turpentine fomentations. The liniment which has appeared to me most serviceable consists of equal parts of chloroform and almond oil, containing two drachms of laudanum and half a drachm of the extract of belladonna to the ounce; but I doubt very much whether any endermic treatment has a material influence over the duration of the disease. It may, and I believe it does, mitigate the paroxysms; but I question whether it renders them less frequent, or in any way prevents their recurrence.

Chloroform inhalations have been proposed with the view of shortening the paroxysms and lessening their severity; and there cannot be a doubt that chloroform so applied does have the desired effect. But may not its constant use be productive of mischief? If employed at each recurring paroxysm, it is difficult to conceive how it can fail to give rise to unpleasant symptoms. Indeed, it appears to me that this species

of inhalation cannot be regarded as a *curative* agent, and that its use must be restricted to very bad cases, in which it may be employed occasionally with the view of checking the spasm temporarily, and so of securing sleep and economising the strength of the patient.

Mr. Atcherley, of Liverpool, reports * that he has seen great benefit derived from the inhalation of nitrous fumes generated in the room which the patient occupies, by the deflagration of bibulous paper steeped in a solution of nitrate of potash. The paroxysm appears to be thereby shortened, and the spasm lessened. I have not had the opportunity of testing its virtues in whooping-cough, but it often proves serviceable in spasmodic asthma, and as the remedy is simple, and can be employed without difficulty day and night, it must, if efficacious, prove extremely valuable.

The application of a strong solution of nitrate of silver to the pharyngeal mucous membrane, and to the orifice of the glottis, is another remedy which has been thought to lessen the tendency to spasm. In some instances undoubtedly it does exercise a beneficial influence, but in many others it fails in affording the slightest relief, and therefore, whilst on the one hand I would not join with those who indiscriminately deprecate its use, I would not on the other have recourse to its employment except in appropriate cases. When, as often happens, the throat is relaxed, and the mucous lining of the pharynx congested and covered with unhealthy secretion, the stimulating effect of the application induces a more healthy action, and lessens the irritability of the parts; but when the throat is in a healthy state, local stimulation is not needed, and fails to mitigate the severity of the spasm. In short, its use should be restricted to those cases in which it would be applicable if bronchitic irritation existed irrespective of whooping-cough.

Towards the close of the disease, when all febrile symptoms have subsided, quinine and iron are often useful adjuncts to the sulphate of zinc, and change of air has been found of the greatest service; indeed, under ordinary treatment it appears in some instances to be indispensable for the entire removal of the tendency to whooping, and the restoration of health. A second change is sometimes beneficial, even without reference to the nature of the locality.

One word must be added in respect to the general management of the patient. It is too much the habit to disregard those precautions which are usually taken in cases of bronchitis, and the little patient is not only

* See 'Med. Times and Gazette,' Feb. 26, 1859.

not dieted and not confined to his room, but is even permitted to go out of doors during the catarrhal stage of the complaint. Hence I am satisfied, the frightful mortality from whooping-cough which stands recorded in the reports of the Registrar-General. In all cases during the continuance of catarrhal symptoms the diet should be restricted as in ordinary bronchitis, and in winter time it is essential not only that the patient should not leave the house throughout the whole course of the disorder, but that the temperature of his apartment should be at least 60°, and kept so as uniformly as possible, and that he should be protected from cold by wearing flannel next the skin. Even in summer time he should be strictly guarded from exposure to cold throughout the catarrhal stage of the complaint, and should not be permitted to leave the house until the severity of the complaint be overpast. So important is this precaution to the patient, that change of air should not be sought until towards the close of the disease.

Bronchitis secondary to Gout, Fever, and other Constitutional and Blood Disorders.

There are few disorders to which the human frame is liable which do not lead occasionally to the production of bronchitis. The altered and poisoned blood of typhus fever, of measles and other exanthemata, of gout and rheumatism, albuminuria and diabetes, of scurvy, secondary syphilis, and other diseases, can hardly fail, sooner or later, to produce irritation of the bronchial mucous membrane. Hence arises a morbid condition of the bronchial secretion, with cough and expectoration—in other words, bronchitis. The precise nature and extent of the pulmonary affection varies with the condition of the blood; so that, whilst in one instance excessive pulmonary congestion is a marked feature of the case, the mischief in another is limited to mere irritation and slight congestion of the mucous lining of the air passages. In either case, however, the pulmonary mischief is the consequence of a specific cause of irritation, to the removal of which our chief efforts must be directed. It is vain to bleed and to give tartarized antimony in the hope of getting rid of the congestion; the only rational and successful treatment is the employment of counter-irritation, and the exhibition of slight sedatives and expectorants, to mitigate the severity of the cough and facilitate expectoration, and of tonics to support the general health during the time which must elapse before the primary disease can be got rid of. The remedies cal-

culated to subdue the primary disease are those which exert the greatest influence on its secondary effects. Thus in bronchitis connected with a syphilitic taint, iodurated inhalations and iodide of potassium, or biniodide of mercury in conjunction with cinchona and sarsaparilla, often prove our most serviceable agents; whereas, when bronchial irritation occurs in connection with gout, our chief reliance must be on colchicum, soda, magnesia, and similar remedies. Practically, therefore, in the cases under consideration, very little treatment, save counter-irritation, is required specially for the relief of the cough; our chief aim must be to remove the primary disease and invigorate the patient, and meanwhile to keep up counter-irritation by means of mustard poultices, turpentine fomentations and blisters. Soothing, or possibly stimulating expectorants in the form of a cough-drop, are sometimes useful, but even a linctus is not always needed.

Narrowing or Obstruction of the Bronchi.

Before quitting the subject of disease of the bronchial tubes, it may be well to point out the nature of the causes which produce narrowing or complete obstruction of the bronchi, and to trace out the pathological effects and physical signs to which such obstruction gives rise.

Narrowing or obstruction of the smaller bronchi is a phenomenon of common occurrence, and may depend on causes operating within the air tubes, or on causes which exert their influence from without. Amongst the former may be mentioned thickening of the mucous membrane, such as is met with in many forms of bronchitis, the retention and accumulation of viscid tenacious mucous secretion, plastic exudation on the surface of the membrane, as often occurs in diphtheria, and the deposit of tubercle or cancer; amongst the latter may be named the pressure of tuberculous and cancerous deposits, the contraction of plastic exudation matter, whether in the lung tissue itself, or on the surface of the pulmonary pleura, and the pressure of enlarged bronchial glands, and of aneurismal and other thoracic tumours. The larger the tubes, the less liable they are to become wholly obstructed; but masses of enlarged bronchial glands and aneurismal, encephaloid, and other tumours in the thorax, do sometimes exert sufficient pressure to obstruct even a main bronchus, and frequently give rise to their perforation by ulceration.

Whatever the immediate cause of complete obstruction, the result is invariably the same, namely, collapse of that portion of the lung to

which the obstructed bronchus leads. Thus obstruction of the main bronchus will occasion collapse of the entire lung, whilst obstruction of one of the smaller tubes will be followed by collapse of correspondingly limited extent. In either case, emphysema commonly results. In the one it occurs in the opposite lung, which expands under the influence of atmospheric pressure to assist in filling up the space left unoccupied by the collapsed lung; in the other, it arises in the immediate vicinity of the collapsed portions of the lung under the operation of the same exciting cause.

The physical signs of obstructed bronchi are—first, dulness on percussion over the seat of obstruction and over the collapsed portion of the lung, unless the dulness in the latter situation be marked by emphysema, which commonly arises in its immediate vicinity; secondly, weakness or deficiency of the respiratory sounds, with greatly prolonged expiration, and occasional sonorous and sibilant rhonchi; thirdly, entire absence of respiration over the collapsed portions of the lung; fourthly, deficiency or entire absence of vocal resonance and vocal fremitus. Extreme prolongation of the expiratory sound over a limited space is perhaps the most characteristic sign of a partially obstructed bronchus.

Dyspnœa, sometimes accompanied by stridor, is the general symptom which principally attracts attention, and that varies in severity, according to the extent of the obstruction and the rapidity of its occurrence; but the rapidity with which the obstruction takes place appears to exercise a greater influence in the production of dyspnœa than the extent of the obstruction; so that the sudden obstruction of a moderate-sized bronchus will induce a paroxysm of suffocative dyspnœa, whilst obstruction of even a large tube occurring gradually, though necessarily attended by considerable frequency of respiration, will not be accompanied by any striking accession of dyspnœa, or notable distress of breathing. The former is well exemplified in many cases of bronchitis; the latter in cases of intra-thoracic tumours. The effect of partial obstruction and narrowing of the tubes in producing prolonged expiratory murmur is seen fully developed in chronic bronchitis.

The treatment of obstructed bronchi must depend on the nature of the obstructing cause, and must be in strict relation to it. Practically, however, the whole subject of obstructed bronchi acquires its interest from the light which it may throw upon the diagnosis of intra-thoracic tumours; and unfortunately in these cases remedial agents are usually of little

avail. In a diagnostic point of view the importance of the subject can hardly be overrated, as will be seen by reference to the portion of this work, where disease of the bronchial glands is discussed,* as also to the section on aneurismal tumours.

CHAPTER IV.

ON PULMONARY CONSUMPTION.

Pulmonary consumption is the commonest and most fatal malady to which the human race is subject. Occurring at every age and in every rank of life, it selects the most beautiful and most gifted as its victims, and entails months or years of weary suffering. What need then for further incentive to a careful study of its history—to a diligent inquiry, as to whether something may not be done to prevent its occurrence, retard its development, or arrest its progress?

With a view to the attainment of precision in our ideas, it will be desirable to commence by an inquiry into the nature of consumption, and into the part which is played by *pulmonary* disease in producing the fatal termination of the disorder.

Briefly, then, it may be stated, that consumption is especially a constitutional malady, closely connected with perverted nutrition and imperfect sanguification, and ultimately leading to the separation from the blood and to the deposit in various parts of the body of an unorganized material, which has been termed tubercle. This material is not deposited indifferently in all parts of the body; it affects certain parts in preference to others, and none so frequently as the lungs.† Hence, the disease is often designated by the terms "phthisis pulmonalis," "pulmonary consumption," "tubercular disease of the lungs"—terms which, though expressive of one of its commonest and most salient features, ignore its constitutional origin, and lead to the erroneous impression that it is a purely local disease of the lungs. Practically this

* See pp. 395-8 of this treatise.

† Thus, of 566 tuberculous patients examined in the dead-house of St. George's Hospital during the ten years ending December 31, 1850, no less than 517 had tubercles in the lungs. In 49 only were tubercles absent from the lungs. See St. George's Hospital 'Post-mortem and Case books,' and the analysis of their contents in Chambers' 'Decennium Path.,' chap. iii.

is of little importance, provided the nature of the malady is understood; but inasmuch as the lung affection is one only of the local results of a great constitutional disorder—a complication which, though of frequent occurrence, and a serious aggravation of the patient's danger, is not an essential part of the disease, or necessary to its fatal termination—it follows that the more appropriate titles are "consumption," "phthisis,"* or tuberculosis, which do not convey the erroneous impression of the disorder being of pulmonary origin.

In what, then, does the constitutional derangement consist? It is not easy to give a definite answer to this inquiry, but a few points may be indicated, which will serve to throw some light on the subject. Chemistry has shown that the blood in consumption contains an excess of water in relation to its solid constituents, that it exhibits a relatively increased proportion of albumen, is deficient in red corpuscles, is less alkaline than in health, and is otherwise altered in character,† whilst the general tendency of combined chemical analysis, microscopical research, and clinical observation, has been to prove not only that the nutrient fluid is vitiated or impoverished, but that its vitality is below the healthy standard. And the same inherent defect or infirmity is traceable throughout the entire organization of the body. The structural peculiarities which characterise the disorder are indicative of imperfect cell-formation and extreme delicacy of the tissues; whilst the functional derangements in their ever-changing variety afford evidence of the weakness of the different organs, and of their liabilty to become disordered on the slightest exciting cause. So that, although consumption is attended by local mischief, which produces corresponding local symptoms, there is not in the whole catalogue of human ailments a malady which more strictly deserves to be classed among constitutional disorders.

But what is the starting point of this derangement? A variety of theories have been advanced on the subject. Some persons have referred the disturbance to imperfection of the primary processes of digestion;‡ some to secondary mal-assimilation and mal-nutrition of the tissues; some to a morbid condition of the lymph;§ some to a specific

* Signifying a wasting or consuming away.

† For the detailed results of the analyses of the blood of consumptive patients by Becquerel and Rodier, Andral and Gavarret, Fricke, L'Héretier, Nicholson, Simon, Karl Popp, Elsner, and others, see Ancell on 'Tuberculosis,' pp. 8-10 and 639-642.

‡ Bennett on 'Pulmonary Tuberculosis.'

§ See Simon's lectures on 'General Pathology,' 1850.

poison in the blood;* some to want of power in the organic nervous force;† some to imperfect respiratory action; some to deficient oxygenation of the blood; and others again, to a variety of causes, which it is needless to enumerate.‡ But, in truth, these theories, however ingenious, have one fault in common. They are all too exclusive, and hence inconsistent with many acknowledged facts! And even were it not so, they would only serve to throw us one step further back in the line of causation. Admitting, for the sake of argument, that each or any one of them may be correct, we still must ask, what is the cause of the deviation from health to which the theory points? or, in other words, what is the essence of the disease?

It has been already stated that the blood is vitiated, altered in character, and of low vitality; that cell-formation takes place with diminished energy, and leads to defective structural development; that the tissues are consequently delicate, and the organs weak and easily deranged. Pathological chemistry and microscopical research enable us to go even a step beyond this, and afford ground for believing that in a portion of the recently formed blood—the "young blood," as Mr. Simon terms it—the proteiniform and oleaginous principles are not perfectly elaborated; that the oxygen, hydrogen, nitrogen, or carbon, or some of their primary compounds, are deficient or in excess; that the abnormal proteiniform compounds thus produced are less organizable than those which are formed in health, more apt to become granular, more prone to aggregation into masses; that the fatty matter is of a lower grade than healthy fat, and approximates to the nature of cholesterine; and that these deviations from the healthy type, both of the blood and of the blastema from which the tissues are formed, produce the various observed anomalies in the growth and development of the body, and give rise to products which constitute the pabulum of tubercle. Beyond this we are unable at present to proceed, for there are no trustworthy data for our guidance. Nay, more, it seems improbable that we shall ever arrive much nearer to the truth than the point here indicated. The revelations of science may ultimately throw additional light on the precise character and constitution of the blood and of the blastema at different stages of the disease, but they

* 'Thoughts on Pulmonary Consumption,' by Dr. Madden.

† Dr. Copland on 'Consumption.'

‡ For a detailed account of various theories which have been advanced respecting consumption, see Ancell, lib. cit., pp. 548-579.

can never bring to light the hidden power which regulates molecular aggregation and cell development, modifies the character and action of the red corpuscles of the blood, determines the transformation of its various constituents, and stamps the different tissues with their respective characters. This vital or formative power, which operates in accordance with fixed laws impressed upon us by our Creator, must ever be beyond man's comprehension; and all that can be surmised respecting it is, that so long as certain conditions exist, it promotes the elaboration of healthy blood, and leads to healthy assimilation, healthy nutrition, healthy excretion—in a word, to the maintenance of health; that any alteration in these conditions, by giving a fresh direction to, or otherwise interfering with, the exercise of this power modifies the results which it produces in our organization, leads to altered chemical action, and so to changes in the character of the blood; and that this derangement, which issues under certain circumstances in the formation of tubercle, constitutes the essence of consumption. Why disturbance of the system should tend in certain instances only to the production of tubercle is one of the mysteries which we shall probably ever fail to unravel. The fact is akin to many others of a similar nature, and may be illustrated by the variations in complexion, visage, form, stature, and constitution observed amongst children of different families, and even amongst children born of the same parents. And it is explicable only by supposing that there is an inherent difference in the formative power implanted in each individual, and a corresponding difference in the abnormal chemical combinations to which, when deranged, this power gives rise, and that thus it is more prone in some persons than in others to promote those peculiar abnormal combinations of the constituents of the blood which result in the formation of tubercle. Observation has led to the belief that, in some instances, repression of the skin's action may be the cause which first upsets the balance of the machine; in some impaired respiratory action, or the respiration of an unrenewed or vitiated atmosphere; in some defective action of the liver; in some imperfect digestion or primary mal-assimilation; in some secondary mal-assimilation; in some an inherited deficiency of organic nervous force; in some a derangement or exhaustion of nervous energy, whether produced by mental or physical causes; in some the introduction of a poison into the blood; and, in others, disturbance of the uterine function, or the occurrence of any morbid action which serves to depress the system and derange the general health. But, whatever the dis-

turbing cause may be, the conditions which are invariably antecedent to the formation of tubercle, are defective vital formative power, an impoverished blood, imperfect assimilation and general mal-nutrition, and these constitute the essence of consumption. This brings us to a consideration of the predisposing and exciting causes of the disease.

It will be obvious, from what has been already stated, that amongst these must be classed a great variety of agencies. The inheritance of a scrofulous or tuberculous tendency, long-continued exposure to cold and wet, improper or insufficient diet, irregularities of living, insufficient exercise, unhealthy and sedentary occupations, the constant inhalation of a vitiated atmosphere, the depressing influence of an unsuitable climate or locality, the indulgence of vicious habits, excessive sexual intercourse, over-protracted lactation and long-continued grief and mental anxiety, are amongst the causes which most powerfully predispose to the inroad of consumption; whilst irritation and congestion of the lungs, whether produced mechanically as in the case of the Sheffield fork and needle grinders, or through the agency of influenza, catarrh, and other diseases which, in the absence of a predisposition to consumption, would pass away without producing the development of that disorder, may be regarded as its more common exciting causes.*

Without going into the operation of these causes, the influence of which has been fully discussed by several authors, I feel bound to remark on some few points on which erroneous views are generally entertained.

Common observation has stamped consumption as an hereditary disorder, and amongst the public the opinion is entertained that in a vast majority of cases the disease is traceable to an hereditary taint. "We have no consumption in our family," is considered a conclusive reply to the inquiry, whether certain symptoms may not be attributable to that disease. On the other hand, a large portion of the profession has been unwilling to admit the extended operation of an hereditary tendency; and statistics have been published by various observers to prove that this influence does not operate in more than 24 or 25 per cent. of the cases met with in practice. Thus, the records of the Bromptom Hospital† are supposed to show an hereditary tendency in 246 only out of 1010 cases, or in other words to prove its influence in only 24·4 per

* For evidence respecting agencies such as these in exciting the disease, see Sir James Clark's work on 'Consumption,' and Mr. Ancell's treatise on 'Tuberculosis.'

† See report of the Brompton Hospital for Diseases of the Chest.

cent. But the truth I believe lies between these two extremes. Without denying that consumption may be acquired, or that it is acquired in many instances through the agency of causes conducive to nervous exhaustion, an impoverished blood, and feeble health, I do not hesitate to assert that we are warranted in believing that an hereditary predisposition exercises a far more extended influence than is indicated by the Brompton Hospital report. The 24·4 per cent. which is there referred to relates only to a predisposition acquired from an avowedly consumptive parent, and does not include the many cases in which the tendency has been inherited from a parent who, though he may not have exhibited at the time the report was drawn any decided symptoms of consumption, has come nevertheless from a consumptive stock, and eventually may have died consumptive, or who again, though not himself consumptive, may have lost brothers and sisters of consumption, and may have inherited and transmitted a consumptive disposition.

The great discrepancies observable in the opinions expressed by authors relative to the hereditary transmission of the disease, are explicable only by assuming a difference in the mode in which the inheritance of the tendency is calculated; * and, as the matter is one which has important bearings on many social questions, I have taken considerable trouble to arrive at a correct conclusion. The result will be seen by an inspection of the subjoined table, which shows, that if the inquiry be limited to the direct transmission of the disease from either parent, the proportion which the cases of inherited disease will bear to the whole mass of cases of consumption, will be about 25·7 per cent.; whereas, if the existence of the disease in either grandparent be considered as evidence of the transmission of the disease to the grandchildren, the proportion will rise to 43·6 per cent.; and if the predisposing influence exhibited by the death of uncles or aunts from consumption be included in the calculation, the proportion will rise to 59·5 per cent. Thus the various conflicting statements may be reconciled, and a trustworthy insight obtained as to the bearing of an intermarriage with a consumptive family.

* Thus M. Louis names one twelfth as the proportion of cases in which a consumptive disposition is inherited; M. Piorry one fourth as the proportion of his hereditary cases; the Brompton Hospital about one fourth; Dr. Cotton rather less than two fifths; M. Briquet two fifths; Dr. Copland nearly one half; M. Portal two thirds; M. Rufz somewhat more than three fifths; M. Ruysch four fifths.

TABLE exhibiting the existence or non-existence of an hereditary tendency in 385 cases of consumption. The cases were taken indiscriminately from hospital and private practice, the only cases excluded being those in which the family history could not be ascertained with tolerable certainty. In every instance in which a parent died consumptive the tendency is referred to that head; in cases in which the parents escaped, but a grandparent died consumptive, the tendency is referred to that head. In no instance is an hereditary transmission assumed in the collateral relationship of uncles and aunts, unless two or more uncles or aunts in the branch of the family had fallen victims to the disease.

	Males.	Females.			
Father consumptive .	27	21	99 or 25·7 per cent.	168 or 43·6 per cent.	229 or 59·5 per cent.
Mother do. . . .	24	16			
Father and Mother do. .	6	5			
Grandfather (paternal) do. .	11	10	69 or 17·9 per cent.		
Grandmother do. do. .	10	8			
Grandfather (maternal) do. .	8	9			
Grandmother do. do. .	8	5			
Uncles or aunts (paternal) do.	14	15	61 or 15·8 per cent.		
Uncles or aunts (maternal) do.	17	15			
No consumptive taint in either of the above relations	87	69	156 or 40·5 per cent.		
Total . . .	212	173			

It is obviously impossible to obtain any data for an opinion as to the proportion of persons who, though born of consumptive parents, pass through life without exhibiting any symptoms of the disease. The experience of life insurance offices shows that it is small, and this conclusion must be endorsed by all who have had an extended sphere of observation. Whole families are sometimes swept away, and even under favourable hygienic circumstances, a few members only ordinarily escape. M. Roche has gone so far as to assert that the children of consumptive parents almost necessarily prove victims of the disease; and if the statement were restricted to those persons whose parents were both consumptive, my own experience would have led me to concur with

M. Roche. But the proportion of cases in which a transmitted tendency to the disease is developed in the children is not so large when one parent was healthy. In that case, as far as my observation has gone, the disease is developed sooner or later in about three fifths of the children, and is especially proved to be developed in the children of that sex to which the consumptive parent belonged; so that the daughters of a consumptive mother will often fall victims to the disease whilst their brothers escape, and the sons of a consumptive father whilst their sisters escape. Whether the sex of the grandparent from whom the disease is transmitted has any influence in determining a proclivity to the disorder in the corresponding sex amongst the grandchildren, is a point which I have been unable as yet to ascertain, but the extraordinary facts lately elicited in Norway and Sweden respecting the hereditary transmission of leprosy,* renders such an influence extremely probable.

Another fallacy which commonly prevails, is respecting the age at which consumption occurs. Amongst the non-professional public, youth is considered the harvest-time of consumption, and middle age the extreme limit of the period within which the whole crop is garnered; and even in the ranks of our own profession there is too frequently a disposition to regard the disease as occurring almost exclusively in early or middle life. But nothing can be further from the truth, or more utterly at variance with modern statistics. The *post-mortem* records of St. George's Hospital show that a large per-centage of persons who die over sixty years of age are affected with pulmonary consumption,† and the valuable reports of the Registrar-General confirm in a

* See 'British and Foreign Med.-Chir. Review' for April, 1858, pp. 332-346.

† The subjoined table exhibits the number and age of the patients who died in St. George's Hospital during the ten years ending December 31, 1850, distinguishing those in whom tubercle existed in the lungs, and the per-centage which those cases bear to the total number of cases examined. See Chambers, loc. cit.

—	Number of patients examined.			Tuberculosis in lungs.				Per-centage of pulmonary tuberculosis.		
	Males.	Females.	Total.	Males.	Females.	Sex unknown.	Total.	Males.	Females.	Total.
From birth to 15 inclusive . .	94	60	154	20	17	0	37	21·2	28·3	24·0
From 15 to 30 .	377	259	636	128	82	1	211	33·9	31·5	33·1
„ 30 to 45 .	472	179	651	120	35	0	155	25·4	19·5	23·8
„ 45 to 60 .	299	139	438	66	11	0	77	22·0	7·9	17·5
Above 60 . . .	109	58	167	10	1	0	11	9·1	1·7	6·5
Age unknown .	74	37	111	14	10	2	26	18·9	27·0	23·4
Total . . .	1425	732	2157	358	156	3	517	25·1	21·3	23·9

striking manner the extended operation of a tuberculous disposition, and prove that, in relation to the number of persons living at the respective ages, the mortality from consumption does not vary materially between the ages of fifteen and seventy, but is actually somewhat less between the ages of ten and forty than it is between the ages of forty and seventy.*

A still more prevalent, and a most mischievous error relates to the predisposing agency of cold and wet, and atmospheric vicissitudes. Nothing is more common than to hear consumption attributed to the effect of wet and cold, the inclemency of the season, or the variableness of the climate in which the person is resident; and nothing, unhappily, is more consistent than the practice which is based on this supposition. Encased in warm clothes, and shut up in heated rooms from which every breath of fresh air is carefully excluded, the unfortunate victim to this prevalent superstition becomes rapidly enervated and falls an easy prey

* The subjoined table is constructed from the Registrar-General's returns for 1847, in which the deaths by phthisis in all England and Wales at the several epochs of life are given in connection with the number of persons of each specified age living in the middle of that year:

Years of age.	Mortality from phthisis.			Estimated population in 1847.			Ratio of the deaths from phthisis to the estimated population.
	Males.	Females.	Both sexes.	Males.	Females.	Both sexes.	
Under 5	2642	2559	5201	1,112,027	1,136,766	2,248,793	1 in 432·3
5 — 10	780	876	1656	1,020,042	1,022,576	2,042,618	1233·4
10 — 15	910	1432	2342	942,534	915,115	1,857,649	793·1
15 — 20	2294	3232	5526	836,845	865,094	1,701,939	307·9
20 — 25	3521	3899	7420	774,542	888,360	1,662,902	224·1
25 — 30	2983	3683	6666	657,138	722,120	1,379,258	206·9
30 — 35	2373	3094	5467	604,706	646,972	1,251,678	228·9
35 — 40	2212	2545	4757	465,847	482,871	948,718	199·4
40 — 45	1847	1903	3750	466,338	486,047	952,385	253·9
45 — 50	1534	1404	2938	345,472	349,380	694,852	236·5
50 — 55	1261	1111	2372	328,848	351,469	680,317	286·8
55 — 60	1025	874	1899	202,956	217,199	420,155	221·2
60 — 65	749	715	1464	223,801	247,912	471,713	322·2
65 — 70	515	505	1020	129,211	149,231	278,542	273·1
70 — 75	253	246	499	111,365	129,120	240,485	481·9
75 — 80	128	109	237	59,546	69,089	128,635	542·8
80 — 85	41	26	67	33,295	42,745	76,040	1134·6
85 — 90	9	10	19	10,770	14,881	25,651	1350·0
90 — 95	2	3	5	2,662	4,333	6,995	1399·0
95 and upwards	—	—	—	622	1,175	1,797	—
All ages	25,083	28,234	53,317	8,368,914	8,755,174	17,124,088	1 in 321·0

to this formidable disorder. But careful observation, corroborated as it is by statistical records, proves incontestably that the pure air of heaven which God has provided for us to breathe, and the variations of temperature, to which in His all-wise providence he has seen fit to subject us, are not so noxious or productive of ill health as man in his ignorance has oftentimes asserted. No climate is more variable than ours, and none certainly is more healthy, as proved beyond dispute by the bills of mortality. Even if considered solely in its relation to consumption, England will bear comparison with any other equally civilised country. The subjoined statistics are decisive on this point,* whilst modern research has tended to show that the vicious habits of civilised life, the confined atmosphere of the dwellings, the want of sufficient active out-door exercise, the various depressing passions, and the exhaustion of mind and body resulting from the anxious struggles and vicissitudes of life, have far more influence than wet and cold in preparing the way for the inroad of consumption. Even when the disease is already developed or far advanced, the pernicious effects resulting from over-caution in respect to exposure to atmospheric vicissitudes are often painfully apparent. Nowhere is this more strikingly exemplified than at our large hospitals, where the consumptive out-door patient who, ill clad and often ill fed, in hot weather and in cold, on wet days as on dry, has to come to the hospital twice a week for medicine, notoriously lives far longer than his brother who, more fortunate in being well fed and protected from the inclemency of the weather, is shut up in the equably heated wards of the hospital, and thus loses the bracing, invigorating stimulus of the fresh breezes of heaven.

* The following table, constructed as the result of his researches by Dr. Caspar, of Berlin (see 'British and Foreign Review,' July, 1847), exhibits the ratio of deaths from phthisis to the deaths from all causes in the following localities:

In Berlin, during	10	years, there was	1	death from phthisis to	5·7	deaths from all causes
Paris,	4	„	1	„	5·5	„
London,	2	„	1	„	6·2	„
Hamburgh,	3	„	1	„	4·6	„
Stuttgard,	10	„	1	„	4·7	„
New York,	11	„	1	„	5·	„
Philadelphia	7	„	1	„	7·7	„
Baltimore	8	„	1	„	6·7	„
Boston	7	„	1	„	5·9	„

whilst from other sources we learn (see 'Bullet. de l'Academie,' Août, 1839), that at the civil hospitals in Rome 1 in 3·4 of the deaths from all causes is due to phthisis.

„ Naples 1 2·33 „ „

Without further discussing the predisposing causes of consumption, we will pass on to the consideration of tubercle, the material which forms the characteristic anatomical element of the disorder.

Tubercle is a substance formed by a retrograde metamorphosis of an exudation from the liquor sanguinis. It is a material of imperfect organization and low vitality, unfit for the construction of new tissues, and therefore purely excrementitious. Sometimes it is deposited in one organ, sometimes in another, sometimes in all the organs of the body; but in every instance it is a foreign body, and damages the organs in which it is imbedded. In appearance it varies greatly, the varieties observed being referable in part to differences of structure, but partly also to the age and extent of the deposit. It may exist in two distinct forms. In the one it produces minute, firm, semi-transparent granules of a bluish grey colour, very different in appearance from ordinary tubercle, and known as "grey miliary tubercles." These may be sparsely scattered through the lungs or may crowd their entire tissue. In most instances they are isolated and are sometimes so minute as to be scarcely discernible by the naked eye, but more commonly they are about the size of a millet seed, and occasionally, when several of them coalesce, they may form a mass as large as a small pea. Usually firm and of a semi-cartilaginous hardness, they are occasionally softer and less resistant, and admit of being crushed between the fingers, whilst not unfrequently they are intermixed with black pigmentary matter, or else are surrounded by it.

The other and more common form of deposit is that known under the title of "crude yellow tubercle." This form of tubercle is quite opaque, and varies in colour from a dirty white to a drab or a bright buff, and in consistence from that of firm tough cheese to that of diffluent cream cheese. In many instances it exists in roundish or irregularly shaped, isolated masses; in others, a large portion of the lung is infiltrated with it; and in others, again, it forms isolated masses in one part of the lung, and in another infiltrates large portions of the lung tissue. The isolated deposits may vary in size from that of a millet seed to that of a hazel nut or a large walnut—the larger masses being formed by the aggregation of the smaller deposits, which give rise to compression and atrophy of the intervening tissue. Thus the size of the tuberculous masses is determined by the extent of lung tissue implicated in the mischief, and their shape by the form of the spaces in which the deposits take place. Not unfrequently the deposit is soft

and pultaceous in one part of the lung, firm and tough in another, extremely friable in yet another; or, if the deposit be old, it may have undergone calcareous or cretaceous transformation, and may prove of stony hardness. In another class of cases, the tuberculous matter becomes mixed up with dark-coloured carbonaceous deposit, which not only colours the tubercle, but gives a bluish-black tinge to the pulmonary tissue.

Opinions have varied as to the precise relationship subsisting between these two forms of tubercular deposit. Laennec and his followers regarded the grey miliary granulations as nascent tubercles, the germs of crude yellow tubercle, whilst others have maintained that the two forms of deposit are essentially distinct, and not merely different stages of the same deposit. Even in the present day pathologists are not agreed on the subject.* My own opinion is, that they both originate in a tubercular diathesis, and are intimately allied in their nature, but probably are each connected with somewhat different conditions of the system.† The frequent occurrence of extensive deposits of grey miliary granulations without any appearance of yellow tubercle, and of yellow tubercle without any trace of grey granulation, is of itself sufficient proof that the two forms of deposit are independent of each other; or, at least, that the grey granulation is not a stage through which yellow tubercle must necessarily pass. On the other hand, it is certain that both varieties often coexist in the same lung, and even in the same parts of an affected lung; nay more, specks of yellow tubercle may be sometimes observed imbedded in the substance of the grey granulations, and there is reason to believe that the grey granulations may be ultimately transformed into yellow tubercles. Further, the differences observed between these two varieties of tubercle disappear in great measure when recourse is had to chemical analysis and microscopical research. The microscope has shown that tubercle, whether "grey miliary" or "crude yellow tubercle," consists of granular matter, elementary molecules, and fat globules, and of so-called

* See Laennec (loc. cit.), Louis, Bayle, Rokitansky, Henle, Vogel, Lebert. A concise review of the different opinions entertained on the subject is given by Ancell, loc. cit., pp. 127-132.

† Rokitansky lends the weight of his authority to this view, when he asserts that the grey miliary tubercle differs from yellow tubercle, in being more nearly allied in composition to the normal constituents of the blood; and Dr. Cotton adds his confirmation to the fact by stating that crude tubercle is deposited under a more intense "degree of phthisis" than miliary tubercle.

tubercle corpuscles, which are imperfectly formed cells of irregular and often angular shape containing granules, but without any distinct nucleus. The relative proportion of these constituents varies greatly in different forms of tubercle, and to these variations are attributable the differences observed in the appearance of that deposit. In recent and firm tubercle, and especially in the grey miliary tubercle, the cell element prevails and fatty matter exists in the smallest possible quantity; whilst in soft tubercle of low vitality, and in old tubercle which is undergoing degeneration and softening, the fatty and granular matters are in much larger quantity, and the cells fewer in number, if not altogether absent.* Epithelium, pus globules, and portions of the yellow elastic fibrous tissue of the lung, the products of disintegration of the lung, may also be visible. When calcareous or cretaceous transformation has taken place, earthy granules are seen of irregular form and size, and crystals of cholesterine are frequently present; whilst if pigmentary matter exists, it is seen as black molecules infiltrating the tubercle, or else is collected round it into small, irregular black masses.

Chemistry, equally with the microscope, has failed to point out any essential difference between "grey miliary" and "crude yellow" tubercle. Chemists are agreed in regarding tubercle as consisting of some modified protein compound or compounds with fat and earthy salts;† they disagree only as to the precise character and relative proportion of these constituents in the specimens they have severally examined. The albuminoid matters, however, appear to exist in largest

* It has been suggested by Dr. Cotton (loc. cit., p. 17) that these differences in the relative proportion of the constituents of tubercles are due not only to the age of the tubercle and the precise stage of its degeneration, but to "the degree of phthisis" which has produced it, and that to this, in a great measure, are attributable the varieties in the course and duration of the disease. I am strongly inclined to believe that the influence here referred to does modify the character of tubercles, but it is difficult to adduce distinct proof of the fact.

† Scherer gives the following as the result of an analysis of tubercles after the salts and all foreign matters had been carefully removed, viz.:

Carbon	53·888
Hydrogen	7·112
Nitrogen	17·237
Oxygen	21·767

This corresponds with the formula $C_{43}H_{35}N_6O_{13}$. Hence, tubercle may be regarded as protein ($C_{48}H_{36}N_6O_{14}$) from which five atoms of carbon, one of hydrogen, and one of oxygen, have been removed. (See Simon's 'Chemistry,' translated by Dr. Day, p. 479.)

quantity in firm and recent tubercle; the fatty matters and the earthy salts—which consist principally of carbonate and phosphate of lime, with a small portion of some salt of soda—in old tubercle, or in tubercle which has undergone transformation subsequent to its deposition.*

Tubercle, when once deposited, may long remain very nearly in *statu quo*, or may slowly or speedily undergo transformation. The precise nature and rapidity of the change will depend in part on the original constitution of the deposit, and partly on the existing condition of the patient's system. The grey miliary tubercle does not ordinarily break up or undergo softening; it loses its transparency, and ultimately shrinks into a dense, hard, tough mass. In certain instances, however, in which it becomes the seat of yellow tubercular deposit, or undergoes transformation into yellow tubercle, it loses this character, and is liable to soften and break up; in others it becomes the seat of slight earthy deposit.

Yellow tubercle, on the contrary, is prone to soften, the process of disintegration commencing sometimes in the centre of the tuberculous mass, but more commonly at its periphery.† Softening, however, does not necessarily occur in yellow tubercle. It usually takes place sooner or later if the patient's health continues to fail; in which case the tissue entangled in the tuberculous mass or immediately surrounding the softened tubercle becomes inflamed and ulcerates, the softened scrofulous matter, mixed with portions of the disintegrated lung tissue, finds its way into the air passages, excites cough, and is then expectorated and got rid of, leaving cavities or vomicæ in the lungs. But if the patient's health improves, and the tendency to the disease is arrested, the tubercles may remain in *statu quo*, or may ultimately be absorbed, or may undergo calcareous or cretaceous transformation. After softening has taken place and the disintegrated tubercle has been removed by expectoration, the cavities which remain in the lung may cicatrize under the influence of returning health and increased vital power.

Many persons have argued against the absorption of crude tubercle, and have boldly asserted that its absorption is impossible. But there

* For full particulars respecting the chemical analysis of tubercle by Thénard, Lassaigne, Hecht, Boudet, Güterbock, Scharlau, Vogel, Lehmann, L'Heretier, Scherer, Preuss, Wood, Simon, Glover, and others, see Ancell, loc. cit., pp. 143-151. Also an analysis by Wright, 'Med. Times,' vol. ii, pp. 418-419.

† For a detailed explanation of the modes in which softening occurs, see Lebert, loc. cit.

are no valid grounds for this statement. True it is that the general health and the tone of the system rarely improve so far as to render absorption of tubercle probable, and it is equally true that from its nature and position tubercle does not easily admit of absorption; but experience at the bedside and observation in the dead-house leave little room for doubt that its absorption may take place under favorable circumstances. Its gradual disappearance from the glands of the neck and other external parts of the body admits of ocular demonstration, and the stethoscope affords presumptive proof of its occasional absorption from the lungs; whilst its almost entire absence from the textures surrounding the cicatrices of old healed vomicæ, and its occasional transformation into cretaceous or calcareous matter—which implies the disappearance of the liquid and animal matter of tubercle—complete the chain of evidence which induces me, in common with Andral, Carswell, and many recent pathologists, to regard its absorption as possible.

Tubercles may be deposited on the free surface of the mucous membrane of the air cells or bronchi, or in the pulmonary parenchyma beneath it—a fact which has an important bearing on the causation of certain physical signs.* Very generally they are deposited in isolated masses, but in the infiltrated form of tubercular deposit they block up the air cells and smaller bronchi, just after the manner of pneumonic exudation. They may be few in number, or extremely numerous, and may be distributed through every part of the lungs, though generally the seat of their deposit varies with the age of the patient and the form of the malady. In cases of acute phthisis, and more especially when the deposit is of the grey miliary variety, tubercles are apt to be disseminated through all parts of the lungs, and in children the same fact is frequently observed; but in chronic cases and in adults the deposits usually commence in the summit of the lungs, and become scarcer, or altogether absent in the lower lobes. In a diagnostic point of view, this fact is of the utmost importance; but it must ever be borne in mind that exceptions are numerous, and that every possible variation may be met with. In most instances both lungs are affected to a greater or less extent, but the tubercle is deposited earliest, and the tubercular deposit is larger in amount, and in a more advanced stage of softening at the apices than in the middle

* Thus the deposit of the tubercle in the tissue beneath the mucous surface of the air-passages gives rise, as I believe, to the more marked degrees of prolonged expiration, and leads to the remarkable phenomenon of dry clicking. (See pp. 128-131 of this treatise.)

or base of the lungs; so that when large, empty cavities exist at the apices, smaller and more recent vomicæ are found lower down, and crude unsoftened tubercle or healthy lung tissue at the base. But sometimes the middle or base of one or both lungs is alone or principally affected, and at others one lung may escape altogether, while the entire structure of the other is infiltrated with tubercle, or broken up into vomicæ. Sometimes, again, both lungs may be partially or extensively implicated, but the tubercles may be advanced, and may have undergone degeneration and softening in the one lung, whilst they are hard, and present all the characters of recent deposits in the other.

Andral, Carswell, Louis, and others, have asserted that tubercles are more commonly found in the left than in the right lung—a statement, which, if correct, would have been of diagnostic value. But careful observation in the wards and in the dead-house of St. George's Hospital has convinced me that this opinion has no valid foundation, and the statistics diligently collected by Dr. Chambers show that the statement commonly promulgated on the subject is at variance with the facts revealed by the scalpel.*

A few points connected with vomicæ require further notice. In the first place, they may vary greatly both in size and number. Sometimes, though rarely, they occur singly; at others a whole lung is riddled with them; sometimes they are no larger than a pea, whilst in certain instances they are as large as a walnut or an orange, or are even capable of containing a pint or more of fluid. The larger cavities are formed by the coalescence of smaller vomicæ, and are, therefore, irregular in shape, and usually intersected by bands of tissue which have escaped disintegration. The tissue surrounding them is generally much solidified, partly by the presence of crude tubercle, and partly by an albuminoid exudation, the result of the inflammatory process which has accompanied the deposit and subsequent softening of the tubercular mass.

* Thus among 517 consumptive patients examined in the dead-house of St. George's Hospital during the ten years ending December 31, 1850, there were 171 in whom tubercle was found in its crude, unsoftened state. Of these 32 had tubercle in the right lung and not in the left; 21 in the left and not in the right; 117 had tubercle in both lungs; whilst of 343 patients in whom the tubercular matter had softened and formed vomicæ, 56 had vomicæ in the right and not in the left lung; 59 in the left and not in the right; and in 224 vomicæ existed on both sides. In 2 cases the records are imperfect, and, without defining the side, state only that vomicæ existed on one side and not on the other; and in 2 others the description is omitted, from imperfection of the notes kept. For further details see Chambers, loc. cit., chap. iv, pp. 47, 8.

Sometimes, though rarely, the large blood-vessels which traverse those portions of the lung which are converted into vomicæ are laid open by ulceration during the process of softening, giving rise to copious and fatal hæmorrhage. But almost invariably the pressure of the tubercle has led to obliteration of the vessel and coagulation of the blood long before its coats would yield to ulceration, so that hæmorrhage from that source is extremely rare; and when hæmoptysis does occur in connection with vomicæ, the blood generally issues from the minute congested vessels on their ragged pulpy walls. Occasionally, though very rarely, a pervious vessel may be found imbedded in the bands which intersect the vomicæ,* and sometimes an obliterated vessel, which has escaped ulceration, will itself form one of the bands.

Those portions of the bronchi which are included in the tubercular mass become obstructed by the deposit of tubercle, so that during the earlier stages of a vomica there is seldom much communication between the cavity and the main bronchi; but, after a time, the obliterated bronchi yield with the surrounding lung structure to the process of ulceration, and are thus broken down and expectorated. Consequently, the patulous, ulcerated extremities of one or more pervious air tubes of considerable calibre are almost sure to be seen terminating abruptly, as if cut across, in the walls of a large vomica, and they form the openings by which air finds admission into the vomica, and the outlets by which the disintegrated tissue and the matter which is secreted from the internal surface of the cavity escape into the trachea.

As long as a tendency to disintegration continues, the internal surface of the vomicæ remains pulpy and ragged; but if the general tone of the system improves, and a tendency to repair ensues, their internal surface becomes smoother, and ultimately is lined by a sort of false membrane. Sometimes this membrane is extremely vascular, and secretes a large quantity of puriform fluid; at others it becomes dense and cartilaginous in appearance, and shows no disposition to contract, so that the cavity may remain open for years.† But more generally, when the reparative

* Louis examined 123 cases in which vomicæ existed, intersected by bands, and in five instances only did he discover a pervious blood-vessel in the bands.

† This fact was remarkably exemplified in the case of a young friend of mine, who was seen during life by the late Dr. Chambers, and by Dr. C. J. B. Williams. Those gentlemen detected a vomica at the apex of either lung, and when the patient died nine years afterwards, the cavities were found patulous, about the size of a small walnut, and lined with a dense, smooth, vascular membrane.

process is established, the fibrinous membrane contracts, the cavity closes, and the surface of the lung immediately over the seat of the vomica becomes puckered. Not unfrequently a portion of calcareousor cretaceous matter is found entangled in or immediately adjoining the cicatrix.

It would naturally occur to any one unacquainted with pathological phenomena, that ulceration occurring in the pulmonary tissue would be likely to make its way through the pleural membrane, and thus establish a direct communication with the cavity of the pleura. Such is indeed the case sometimes, though rarely—so rarely that it occurred ten times only (five times on the right and five times on the left side) amongst 168 consumptive patients, whom I carefully examined throughout the period of their residence in St. George's Hospital.* And the reason of its not occurring more frequently is at once revealed by *post-mortem* examination; for when tubercles are seated, as they often are, near the surface of the lung, they are apt to excite adhesive inflammation of the pleural membrane—not extensive inflammation attended by copious effusion, but dry, adhesive pleurisy, usually corresponding in position and limited in extent to the area of the tubercular deposit, and causing the costal and pulmonary pleura to cohere. The risk of a mixed aeriform and softened tuberculous effusion into the pleural cavity is thus avoided, and partial or local adhesion of the pleura is the only consequence. This, however, is of very constant occurrence, and is most common and most extensive at the apices of the lungs, where the tubercles are usually most numerous. Louis informs us that it was absent in one only out of 112 consumptive patients whom he examined after death, and it was present in forty-six out of fifty fatal cases of phthisis which I noted with a special view to this inquiry; two out of the remaining

* This is a considerably higher per-centage than that deduced by Dr. T. Chambers from the *post-mortem* records of St. George's Hospital. He gives 19 cases of pneumothorax (11 on the right side and 8 on the left) amongst 514 cases of tuberculosis. But, in truth, those numbers do not adequately represent the frequency of tuberculous perforation of the pleura. A certain proportion of the patients in whom this complication occurs recover sufficiently to leave the hospital; and even in a certain proportion of those who die in the hospital the effused air is absorbed, the two surfaces of the membrane become adherent, and the lesion escapes notice in the *post-mortem* record. Further, pneumothorax rarely takes place except towards the later stage of consumption, and a large proportion of the 515 cases analysed by Dr. Chambers were not, strictly speaking, fatal cases of consumption, but were rather cases in which death occurred from some other causes, though tubercles to a greater or less extent were found in the lung. If the inquiry were limited to *fatal* cases of consumption, the proportion in which pneumothorax occurs would be found infinitely larger than that which I have given above.

four cases being examples of the grey, miliary deposit. It was also present in 392 cases out of 514 cases of pulmonary tubercle (not all fatal cases of consumption) which were examined in the dead-house of St. George's Hospital during the ten years ending December, 1850.* Thus, partial or local pleurisy may be regarded as a frequent, if not a constant, complication of phthisis pulmonalis; perforation of the pleura and pneumothorax as an uncommon event. But it must not be understood that the pleurisy which accompanies phthisis is always dry and limited in extent. Commonly, indeed, it is so, and still more generally the inflammation, if acute, is limited to one side of the chest; but occasionally it exists on both,† and in a considerable proportion of cases in which it is limited to one side it is diffused over a large portion of the pleural membrane, and is accompanied by profuse effusion, which is a serious aggravation of the patient's danger, and hurries him to an untimely grave.

Other complications are also of frequent occurrence. Amongst these may be mentioned atrophy of the lung tissue, resulting from the cutting off of a due supply of blood by the pressure of tubercle on the blood-vessels; emphysema, which was observed in forty-one, or, in other words, in 7·9 per cent. of the phthisical cases examined in St. George's Hospital during a period of ten years;‡ and still more frequently bronchitis and pneumonia. The morbid condition of the blood in phthisis, and the local irritation which attends tuberculization of the lung, are apt to produce not only congestion, but active inflammation of the bronchial mucous membrane, or of the surrounding lung structure, and thus the phenomena which have been already discussed in the sections devoted to bronchitis and pneumonia are met with during the course of phthisis. Pneumonia, however, is less commonly excited by tubercular deposit than congestion and inflammation of the bronchial mucous membrane, which are prone to occur at all stages of the disease.

Another complication which is apt to arise, especially in children, is tuberculization and enlargement of the bronchial glands;§ and another, which is extremely frequent, more particularly in adults, is ulceration of

* See Chambers, loc. cit., p. 257.

† Thus it was present on both sides in no less than 29 of the tubercular cases already alluded to as have been examined in the dead-house of St. George's Hospital during the ten years ending December 31st, 1850.

‡ See Chambers, loc. cit., p. 63.

§ For a full description of the physical signs and general symptoms produced by this affection, see p. 395-8 of this treatise.

the mucous lining of the upper air passages. The epiglottis, the internal surface of the larynx, and the tracheal and bronchial mucous membrane are all liable to be implicated in the ulcerative process, and when so affected, give rise to symptoms which will be fully described hereafter.

But consumption is not merely a disease of the lungs and air passages; it is essentially a constitutional disorder, and, as stated at the commencement of this chapter, it may leave its traces in other parts besides the chest.* The brain and its membranes are prone to be the seat of tubercle; so are the liver, spleen, and kidneys; so also are the cervical and mesenteric glands, which in children are seldom exempt. The glandular structure of the bowels may be implicated in the same way, and small hard masses of yellow tubercle may be seen projecting from the

* This is seen by reference to the subjoined record of the *post-mortem* examinations of 566 tuberculous patients at St. George's Hospital during the ten years ending December, 1850, as recorded in Dr. Chambers's 'Decennium Pathologicum.' The first table shows the seat of tubercle in 517 cases of pulmonary consumption; the second exhibits their seat in the 49 cases in which tubercles did not exist in the lungs.

Tubercle in other organs conjointly with the Lungs.

Seats of tubercular deposit in cases of pulmonary consumption.	In 37 cases, from birth to 15 inclusive.	In 211 cases, from 15 to 30 inclusive.	In 155 cases, from 30 to 45 inclusive.	In 77 cases, from 45 to 60 inclusive.	In 11 cases above 60.	In 26 cases of unknown age.	In 517 cases at all ages.
1. Intestinal canal	10	71	37	14	—	9	141
2. Mesenteric glands	17	37	26	9	1	3	93
3. Kidneys	6	46	26	4	1	1	91
4. Peritoneum	6	23	8	4	—	1	42
5. Bronchial glands	10	13	6	2	—	3	34
6. Nerve centres	10	15	3	—	—	—	28
7. Spleen	6	12	4	—	—	1	23
8. Liver	5	7	1	—	—	—	13
9. Bladder and prostate gland	—	6	3	2	—	1	12
10. Organs of generation (male and female divided below)	—	5	1	2	—	1	9
11. Heart and pericardium	2	3	—	—	—	—	5
12. Male organs of generation	—	3	1	—	—	1	5
13. Female organs of generation	—	2	—	2	—	—	4
14. Axillary and cervical glands	1	—	2	1	—	—	4
15. Anterior mediastinum	—	3	—	—	—	—	3
16. Pancreas	—	—	1	—	—	—	1
17. Cranial bones	—	—	1	—	—	—	1

surface of the intestine;* or the tubercle may soften, and cause ulceration, and lead eventually to perforation of the peritoneum, and to peritonitis resulting from the escape of fæcal matter into the cavity of the abdomen; or tubercular deposit may take place in the peritoneum, and occasion chronic inflammation and ascites. Piles, and fistula in ano, are other of its common accompaniments;† fatty degeneration of the liver is another, and œdema of the lower extremities, resulting from coagulation of blood in the veins, and consequent obstruction to the circulation, is another which occasionally proves very distressing.

The physical signs of pulmonary consumption, no less than its general symptoms, correspond in great measure with the extent and condition of the tubercular deposit; and as there are three well-marked stages of tubercle, viz., those of deposition and induration, of softening, and of

Cases in which the Lungs were free from Tubercle.

Seat of tubercular deposit.	From 1 to 15.	From 15 to 30.	From 30 to 45.	From 45 to 60.	Above 60.	Age doubtful.	In 49 cases at all ages.
1. Kidneys	1	4	5	4	1	—	15
2. Bronchial glands	1	5	3	—	—	—	9
3. Peritoneum	—	3	1	2	—	1	7
4. Abdominal glands	1	2	1	1	—	1	6
5. Parts of generation	—	1	—	1	1	2	5
6. Liver	—	2	2	1	—	—	5
7. Intestinal canal	1	—	4	—	—	—	5
8. Nerve centres	2	2	1	—	—	—	5
9. Spinal bones	—	3	1	—	—	—	4
10. Parietal pleura	1	2	—	—	—	—	2
11. Cranium	—	2	—	—	—	—	3
12. Hip	2	—	—	—	—	—	2
13. Other joints	1	1	—	—	—	—	2
14. Cellular tissue and muscles	—	1	1	—	—	—	2
15. Pericardium	—	1	1	—	—	—	2
16. Shaft of femur	1	—	—	—	—	—	1
17. Spleen	—	1	—	—	—	—	1
18. Dura mater	—	1	—	—	—	—	1
19. Nervus abducens	—	—	1	—	—	—	1
20. Axillary glands	—	1	—	—	—	—	1
21. Cervical glands	—	1	—	—	—	—	1

* Louis states that he met with tuberculous ulceration of the smaller bowels in five sixths of the phthisical cases he examined after death, and ulceration of the larger bowel was almost as frequent.

† The statistics deduced from the *post-mortem* records of St. George's Hospital throw considerable doubt on the commonly received opinion as to the almost invariable connection between fistula and phthisis, and afford grounds for believing that in a large proportion of cases fistula in ano is unconnected with tuberculosis. For full details see Chambers, loc. cit., chap. vi, p. 75.

excavation, it will be desirable to discuss the physical signs of the pulmonary disease as they usually present themselves at each of these stages.

1*st stage.*—*Inspection* of the chest affords very little information unless the tubercular deposit be large in amount, or be confined to one side. In the former case an abnormal rapidity of the respiratory movements will be observable, and in both the former and the latter a decrease of expansion in the infra- and supra-clavicular regions on the affected side, and, possibly, a little flattening of the upper part of the chest walls on that side. When the deposit takes place equally on both sides, the flattening which it occasions at this early stage may escape notice.

Palpation will sometimes detect deficient expansion in the infra- or supra-clavicular regions, even when inspection has failed to do so. It also gives us notice of consolidation of the lung tissue, by making us aware of increased vocal fremitus on the affected side; the increase, however, is seldom great, and on the right side cannot be relied upon as evidence of disease. At the left apex it is a sign of considerable importance.

Mensuration will sometimes detect a slight diminution in the antero-posterior diameter of the infra-clavicular regions, and also a decrease in the local expansion movement.

Percussion is always a fallacious guide in these cases. In most instances miliary tubercles do not perceptibly affect the resonance of the chest, and even if they exist in quantity sufficient to produce a slight modification of the percussion note, they usually do so on both sides of the chest, so that the alteration of sound fails to impress the ear or to convey any definite information. Even when considerable masses of crude tubercle exist at the apex, dulness is not an invariable consequence. If they are seated superficially, the sound on percussion is dull, and the sense of resistance to the fingers is increased; but if, as often happens, some healthy or some emphysematous lung tissue intervenes between the consolidated lung and the chest walls, or if haply the superficial air cells become distended with air in consequence of any obstruction offered to its egress by the pressure of tubercle on the smaller bronchi, not only will no dulness occur, but there will be an abnormally clear though shallow resonance over the affected lung.* These alterations in the percussion note will be most perceptible in the clavicular and infra-clavicular regions; and if doubts arise as to the existence of dulness on either side, recourse should be had to percussion, practised during full inspiration, and then during deep expiration. In the former

* See chap. V, pp. 54-60, of this treatise.

case, if tubercles exist, the increase of resonance on the affected side will be slight as compared with that on the healthy side, whilst in the latter case the dulness will be far greater on the affected side than over the healthy, unaffected lung.* In all cases it is the difference of the percussion note on the two sides of the chest, rather than the actual quality of the sound, which is indicative of subjacent disease.

Auscultation may afford very valuable assistance, though in many instances it will fail altogether to do so, and in others, its indications will not suffice to establish the precise nature of the disease. Thus, when grey miliary tubercles, or a few isolated, crude tubercles, exist in a quiescent state in the lungs, they will often fail to produce any appreciable auscultatory signs. Even when they are present in somewhat larger quantity, the utmost auscultatory disturbance produced is slight harshness of the respiration, with prolongation of the expiratory murmur, and jerking irregularity or cogged wheel rhythm of the respiratory sounds—signs which are not peculiar to phthisis, but are met with in bronchitis and other forms of disease. Therefore, it is only when they occur persistently, and are confined to one side of the chest, that they can be regarded as indicating the existence of tubercle. To these however, in some instances, must be added dry clicking, which is always suggestive of tubercular deposit, and imparts a dangerous significance to the jerking respiration and prolonged expiratory murmur. These signs are usually developed earliest, and are most marked in the supra-scapular, the supra-clavicular, and the infra-clavicular regions.

When local pulmonary irritation is excited by the tubercular deposit, or by the condition of blood which has led to the deposit, the auscultatory signs of catarrh are superadded, viz., weakness or deficiency of the healthy vesicular murmur, which is replaced by coarseness of respiration, with small bubbling râles and sonorous and sibilant rhonchi. In this, however, there is nothing pathognomonic of phthisis; and it is only when these symptoms of bronchitis are persistent, and are almost confined to the apices of the lungs, being inaudible below the second intercostal space, and especially when they are confined to the apex of one lung, that their existence can be regarded as suspicious. Sometimes, however, these sounds are accompanied by the grazing or crumpling sound of pleural friction, which renders their true character apparent.

When the deposit of tubercle is more extensive, and includes within it

* For detailed precautions respecting the performance of percussion in these cases see chap. V, pp. 42-4, of this treatise.

bronchial tubes of considerable diameter, the evidence is much more conclusive. If the tubes are filled with tubercle, there is almost entire absence of vocal resonance and of respiratory murmur over the affected part, and not unfrequently exaggerated breathing in the parts immediately adjoining; whilst if the air passages remain pervious, hollow sounding respiration and increased vocal resonance are met with. Further, if the deposit be extensive, and not merely limited to the apices of the lungs, the pulmonary consolidation leads to the transmission of the sounds of the heart and large vessels to a far greater extent than in health; so that if the right lung be principally affected, the cardiac sounds may be more audible at the right than even at the left apex. But many conditions of the intervening lung tissue serve to prevent this transmission of the heart's sounds, and therefore the mere absence of this sign is not valid evidence against the existence of tubercular consolidation.

A systolic pulmonary murmur is often present in these cases at the second left sterno-costal articulation, and so is a murmur in the subclavian artery on the left side, and in the innominate artery on the right. None of these signs, however, if viewed alone, affords trustworthy evidence of tubercle. In many phthisical patients the pulmonary murmur is absent, and in many anæmic, non-phthisical persons it is present, whilst the subclavian and the innominate murmur, if not audible during tranquil respiration, may be developed in many healthy individuals by a few successive deep and forcible respiratory efforts.*

It should, perhaps, be added that in the earlier stages of consumption more reliance can be placed on the altered quality and intensity of the respiration, and on the occurrence of jerking irregularity of rhythm than on the existence of prolonged expiration or increased vocal resonance. Prolonged expiration, if unaccompanied by alteration in the quality of the respiration, may be a normal condition peculiar to the individual: and vocal resonance is subject to so many variations as to be almost

* In order to ascertain the true significance of this subclavian sound, I carefully examined 100 healthy persons who presented themselves for examination with a view to life assurance. In 3 the murmur was strongly marked in both sides of the chest, and in 9 others on the left side of the chest during tranquil respiration; in 19 it was temporarily induced on both sides, and in 46 others on the left side of the chest by the forcible effort of blowing the spirometer, whilst in the remaining 23 it was not heard either during tranquil or after forced respiration. The position of the left subclavian artery, which admits of its being compressed against the clavicle by the fully expanded lung, explains the frequency of the occurrence of the sound in the left as compared with the right side. Of course it would be more likely to be produced, and would prove a more persistent condition if the apex of the left lung were solidified by tubercle.

valueless if taken by itself as the basis for an opinion as to the condition of the lung. At the right apex increased vocal resonance can hardly be regarded even as suggestive of tubercle, unless it be excessive in degree or has succeeded to naturally weak vocal resonance.

2nd stage.—Many of the signs already described as characteristic of the first stage of the disorder now exist in an advanced degree of development, and a series of new auscultatory signs, referable to the softened tubercular deposit, are met with for the first time.

Inspection.—The eye at once notes an abnormal frequency of the breathing, a decided flattening or falling in of the chest walls on the affected side both above and below the clavicle, and great deficiency of local expansion, especially during forced inspiration.

Mensuration with the callipers shows a marked diminution in the size of the chest, both in its transverse and antero-posterior diameter, the decrease being referable in part to atrophy and interstitial contraction of the lung, and in part to retraction caused by firm pleural adhesions.

Percussion in most instances elicits evidence of a wider spread and more intense dulness.

Auscultation.—In addition to the phenomena discovered during the first stage of the disease, auscultation now announces the important fact of the presence of thin, irregular-sized bubbling râles, caused by the passage of the inspired air through the softened and liquefied tubercular deposit; it further proves that coarse and hollow-sounding respiration exists over a more extensive surface than heretofore, or possibly that the respiratory sounds have assumed a distinctly blowing character. Vocal resonance still remains extremely variable, and cannot be relied upon as a guide to the condition of the subjacent lung; and the existence or non-existence of vocal fremitus is an equally uncertain indication, inasmuch as it is dependent upon the amount of pulmonary consolidation, and not upon the occurrence of softening.

3rd stage.—Inspection reveals increasing rapidity of the respiration and extraordinary prominence of the clavicles, referable to the increasing depression or falling in of the supra-clavicular and infra-clavicular regions, consequent on the advancing tubercular disease and decreasing pulmonary expansion. Further, it sometimes makes us aware of the existence of a fluctuating impulsive movement in the upper intercostal spaces, occasionally on the right, but more frequently on the left of the sternum, caused in most instances by the action of the pulmonary artery or of the base of the heart, which has been brought into unnatural con-

tiguity with the anterior walls of the chest by the destruction of the intervening lung tissue, but referable in some cases to the systole of the left auricle, as proved by the synchronism of the impulse with the diastole of the heart.

Palpation makes us aware of a further decrease in the expansion of the upper part of the chest; of marked vocal fremitus when the cavity is large, superficially seated, and in free communication with the upper air passages; and sometimes of rhonchial and gurgling fremitus, or even of distinct fluctuation. Further, if as sometimes happens, the disease exists principally in one lung, and that lung has shrunk considerably, and has dragged the heart along with it, palpation makes us aware of the fact that the heart is beating out of its proper place; it also informs us of any abnormal pulsation which may exist in consequence of the base of the heart or the left auricle or the pulmonary artery having been brought into contact with the anterior walls of the chest, as a result of an excavation in the intervening lung tissue.

Mensuration shows progressive diminution in the diameters of the upper part of the chest.

Percussion varies greatly in its results, according to the varying conditions of the vomicæ, and of the tissues which lie between them and the chest walls. If one or more small vomicæ exist filled in great measure by purulent secretion, or surrounded by tubercular deposit, or by consolidated lung tissue, the sound, on percussion, will be absolutely dull; whereas, if the same cavities be empty and seated superficially, and the pleural membrane be not thickened by plastic exudation, the dulness will be comparatively slight, or the sound may be even resonant, though of a shallow, amphoric character. If, again, a tolerably thick layer of healthy permeable lung tissue intervenes between the chest walls and vomicæ partially filled with fluid, or surrounded by tubercular deposit, gentle percussion may give rise to almost healthy resonance, while firm and forcible percussion will elicit the deep-seated dulness referable to the vomicæ, and the consolidated lung tissue beneath. Large empty superficial vomicæ, with thin tense walls, yield an amphoric, and possibly a cracked pot resonance; whereas, when the walls of the cavity are thick or flaccid, or, when a denser thickened pleural membrane, or a portion of consolidated lung tissue intervenes between the cavity and the chest walls, the sound elicited may be absolutely dull, or it may be of a mixed dull and amphoric character. Except in rare instances of enormous superficial vomicæ with thin tense walls, it is almost impossible to

judge of the size of a cavity by the results of percussion, inasmuch as the condition of the thoracic parietes, the distance of the vomica from the chest walls, the variations in the relative proportion of air and fluid in the cavity, and the state of the intervening lung tissue, offer insuperable obstacles to the formation of a sound judgment on the matter. When an empty cavity with resilient walls is seated superficially, and has a free communication with the upper air passages, forcible percussion will often elicit a cracked-pot sound, especially when the mouth is open, and all obstacle to the egress of air is thus removed.

Auscultation.—Provided the cavity is tolerably empty, and in free communication with the upper air passages, and that the sounds emanating from it are not masked by the intervention of a thick stratum of healthy permeable lung tissue, the respiration will be heard of a hollow, blowing, or even an amphoric and metallic character; and when the fluid contents of the cavity have accumulated sufficiently to rise above the level of the permeable bronchi which lead to it, large irregular bubbling râles or distinct gurgling, often of a metallic quality, may also be heard. In some instances well-marked metallic tinkling is produced by the cough, the respiration, and the action of the heart in large cavities which possess smooth, tense, sound-reflecting walls. Vocal resonance varies greatly, according to the precise condition of the parts, and therefore cannot be relied upon as evidence of the existence or non-existence of a cavity. If a vomica be partially filled with secretion, and the bronchi leading to it be obstructed, no vocal resonance may be heard; whereas, after the air tubes have been cleared by coughing, the resonance in one case may be nearly natural; in another simply feeble, and in yet another intensely loud, and of a bronchophonic, pectoriloquous, or amphoric and metallic character; these differences being due to the varying conditions of the walls of the cavity, and to the freedom or otherwise of its communication with the bronchi. Well-marked pectoriloquy resulting from a mere whisper is the form of vocal resonance most pathognomonic of tubercular exudation; but in many instances it is not met with, whilst, on the other hand, this and every other form of increased vocal resonance may result from dilated bronchi, surrounded by hepatized, or otherwise consolidated lung tissue.

Small cavities, partially filled with fluid, and deeply seated in the lung, seldom produce signs characteristic of a vomica, but simply occasion imperfect hollow respiration, occasional rhonchi, irregular bubbling râles, and more or less modified vocal resonance—sounds

which may originate in a large bronchus surrounded by consolidated lung. When, as often happens in cases of pulmonary excavation, the base of the heart is brought into contact with the anterior surface of the chest, causing more or less pulsation in one or more of the upper intercostal spaces, auscultation shows that the sounds of the heart are abnormally loud in that situation, and may be even louder, and apparently seated more superficially than at the fifth left interspace—the natural seat of the apex beat. Further, it often announces the existence of intense systolic murmur at the second left sterno-costal articulation, and sometimes in the first and second intercostal spaces on either side, referable to the pressure of hardened lung tissue or tubercular deposit on one or other of the larger arteries.

Succussion.—If a large cavity, possessing thin, tense, sound-reflecting walls, be seated superficially in the chest, and contain a moderate quantity of thin liquid secretion, a splashing noise may be elicited when the patient is forcibly and abruptly shaken. In a cavity of this description, placed under favourable condition, even the action of the heart may sometimes give rise to this sound.

Consumption is a malady which, beyond all others, is remarkable for the great diversity of its symptoms in different cases, and for extreme variation in the course which it runs; and as these differences are not associated with any particular stage of the tubercular deposit, but rather with peculiarities in the patient's constitution, and with the form which the malady assumes, it will be desirable to give a few sketches of the disease as it presents itself under different circumstances. In no other manner is it possible to convey an adequate idea of the complaint, or to furnish such a connected history of its symptoms as shall afford a clue to its diagnosis.

The heads under which I shall attempt to sketch the disease are, 1st, the ordinary forms of chronic phthisis; 2ndly, the acute forms of the complaint; and 3rdly, the latent or insidious forms of the disease. Its special diagnostic symptoms, and some of its more important complications, must be reserved for separate consideration.

To begin, then, with the ordinary forms of phthisis.

In many instances the earliest, and for some time the only symptoms of its approach are dyspepsia, with sick headaches, biliousness, loss of appetite, gradually increasing languor, and debility and depression of spirits. The patient feels unequal to his ordinary avocations, his nights

are restless, and in the morning he rises weary and unrefreshed. After a time emaciation commences, the flesh becomes flabby, the countenance pale, the pupil of the eye dilated, and the conjunctiva of a pearl-like whiteness; the hair falls, and in many instances the fingers become clubbed at their extremities and the finger nails incurvated and adunc. Occasionally the patient suffers from weakness and huskiness of voice, soreness of throat, and tightness across the upper part of the chest, with dull aching fugitive pains about the collar-bones, or under one or both shoulder-blades, and although he has no running at the eyes or nose, he fancies he must have "caught cold," for he feels chilly and uncomfortable, and has a short, dry, hacking cough. This occurs principally on rising in the morning and on going to bed at night. At first the cough may be so slight as not to cause him any annoyance, or even to excite the apprehension of his friends; it is regarded simply as a clearing of the throat, and appears to be occasioned by relaxation of the uvula, or by irritation in the pharynx, which in the early stage of phthisis is often rough, red, and covered with mucus. But after a time the cough increases in frequency and violence, recurs at intervals throughout the day, especially after exertion, and becomes attended by a scanty expectoration of ropy or glairy mucus, occasionally specked or tinged or streaked with blood. Little suspecting the cause of his ailment, he complains that he is short-breathed on going up stairs, and is soon exhausted by active exertion; and the physician finds that his breathing is quicker than natural, and his pulse accelerated, especially towards evening, and very deficient in force. His face flushes on the slightest excitement, and particularly after meals, whilst, in some instances, febrile paroxysms, marked by alternate chills and heats by night, and by perspirations towards morning, form a cause of serious annoyance and complaint. The bowels may act regularly, but more commonly are confined; the tongue may be clean or more or less coated, and the pulse weak or irritable, varying in frequency from 60 to 140 in the minute. The urine is at one time clear and pale, at another high-coloured, scanty, and turbid, but it simply varies with the state of the system, and does not throw any light on the condition of his chest. The menstrual discharge may be natural in quantity and quality, or it may be excessive in quantity, and may recur too frequently, but more commonly it is scanty or entirely suppressed, and replaced by profuse leucorrhœa. Not unfrequently suppression of the catamenia is the event from which the patient dates her illness.

In another class of cases the symptoms do not differ greatly from

those just described, except that they are unaccompanied by dyspepsia, and that in the earlier stages the febrile paroxysms are almost wholly absent. The appetite is fairly good, and nothing seems to disagree; the bowels act regularly, the pulse is not much accelerated, the urine is clear and natural in appearance, and the patient sleeps quietly, unless disturbed by the cough. He is weak and languid, and loses flesh; but even when pressed for a full confession of his feelings, he will assert that there is not much amiss, and that, were it not for occasional slight cough, huskiness of voice, and a sense of weakness and undue exhaustion after exercise, he should have no cause for complaint.

In yet other cases, the patient, who is usually of a florid complexion, may consider himself in good health, when, possibly after some straining effort, he is suddenly attacked with profuse hæmorrhage from the lungs, or after exposure to cold is seized with bronchitis, attended by blood-tinged or blood-streaked, frothy mucous expectoration. The only premonitory symptoms will probably have been occasional huskiness of voice, and an unusual desire to "clear his throat" on rising in the morning, more than ordinary exhaustion after taking active exercise, and a tendency to perspire on the slightest exertion. Even these symptoms will have failed to attract his attention, so that pointed and earnest enquiry has to be made before he will admit that he was at all out of health when the attack commenced. In these cases there is generally undue excitement of the circulation, with a quick and irritable pulse, feverish heat of skin, restlessness at night, and a frequent, hard cough attended by pain in the chest. Sometimes, however, though the pulse be quick, the cough is unattended by pain, and there is entire absence of fever; the difference apparently being referable to the existence or non-existence of bronchitis, or of inflammation of the pulmonary tissue.

But whatever symptoms may have inaugurated the accession of the disease, its progress will depend in part on the amount of tubercle which has been deposited, in part on the nature of the patient's constitution, and in part on the effect produced by treatment. Very commonly a large amount of tubercle is deposited in the first attack; the intervening portions of lung are congested, there is considerable bronchial irritation, and the function of respiration is seriously interfered with. If this condition of lung occurs in a person of a hitherto unimpaired constitution, the more active symptoms may possibly be subdued by judicious treatment, and considerable improvement may take

place; the cough will still remain troublesome, and the patient will be short-breathed, and a confirmed invalid; but nevertheless he may appear to be on the mend, and the symptoms of rapidly progressive disease may for a time be stayed. But if he be of a weakly constitution or strong scrofulous tendency, the disease will run on from bad to worse, notwithstanding any treatment which may be adopted. The tubercle will soften and break down into vomicæ, and whilst the disintegrated tubercular matter is being got rid of by expectoration, fresh tubercles will be deposited, and the healthy lung tissue will be still further encroached upon; the bronchitic or pneumonic symptoms may be subdued for a time, but only to reappear after a brief interval, as fresh local irritation is excited; the breathing becomes shorter and more oppressed, the cough more frequent and harassing, exciting sickness and rejection of food, and the expectoration profuse and of a purulent character, often streaked or mixed with blood. The complexion is usually pale, though the skin is hot owing to the febrile excitement which prevails; the lips are of a dusky yet pallid hue, or else of a brilliant red colour; the cheeks flush on the slightest occasion, and especially after food; the pulse is much accelerated, and distinct febrile paroxysms, which commence with alternate chills and heats, and terminate in profuse sweating and great exhaustion, occur towards noon, and again at night. Meanwhile the patient wastes, and becomes rapidly weaker; the catamenia cease, the nails become adunc, the fingers clubbed, the hair falls; pain or stitch in the side, resulting from partial or local pleurisy, is of frequent occurrence; hoarseness and partial aphonia often supervene, arising from tubercular ulceration of the larynx, and diarrhœa sets in, occasioned by the same form of mischief in the intestines; the tongue becomes extremely furred, or else clean, smooth, and red; aphthæ make their appearance in the mouth and throat, the appetite fails, and sleep is only to be wooed by full and repeated doses of sedatives; the integuments of the back become inflamed and sore, or even ulcerated, in consequence of the pressure to which they are subjected; the features become sharp and collapsed, the feet and ankles swollen and œdematous, and as death approaches slight wandering or delirium often occurs. Sometimes coma ultimately supervenes, and the patient, unconscious of his approaching end, sinks quietly and without a struggle; in other instances the brain remains unaffected, and the mind is clear and collected; the cough almost ceases, and the patient passes away in perfect tranquillity, almost

as if asleep; in others, again, the mind remains unaffected, but for many days, if not for a longer period before death the patient suffers constantly from a distressing sense of sinking and exhaustion, and is tormented with pains in the chest and bowels, oppressive shortness of breath, and excessive difficulty of expectoration, leading to frequent fits of suffocative dyspnœa, which render the death-struggle extremely painful.

The duration of the disease in the cases above described varies from a few weeks to six or eight months; and from the time when the active symptoms commence the patient is a confirmed invalid; but in many instances the malady runs a much more protracted course, and presents marked intermissions in its progress. In such cases the area of tubercular deposit is at first small, the tubercles are confined to a portion of the upper lobe, and the remainder of the lung is perfectly healthy, so that the respiratory function is not seriously interfered with. Under these circumstances, if the general health can be improved and the patient's system invigorated, the deposit of tubercles may be arrested; those already deposited may remain in a crude state for years, or even throughout a tolerably long life, the irritation they cause may pass off, the cough and other symptoms will then subside, and the patient may recover a certain degree of health. He rarely regains full bodily vigour; he remains more or less short-breathed, and liable to be affected by slight disturbing causes; but though somewhat of a valetudinarian, and prone "to catch cold," he is able to resume his ordinary avocations, and is regarded by his acquaintances as in tolerable health. Two or more such attacks as these may occur at intervals of as many months or years, before any softening of the tubercles takes place; the system rallies quickly after each attack, and the patient recovers more or less completely; though as each attack results in a fresh crop of tubercles, it leaves the lungs less equal to carry on their respiratory function, and the general health more seriously impaired. At length the deposit reaches the limit which is compatible with even feeble health; the general power of the system fails, the tubercles soften, vomicæ are formed, the patient expectorates a large quantity of puriform matter, suffers greatly from hectic fever, has frequent attacks of diarrhœa, sweats profusely at night, becomes rapidly emaciated, and sinks, as in the former case, after a few weeks or months of suffering.

Sometimes, again, the course of the disease is somewhat different. The tuberculous deposit is not extensive, and there is very little bronchitic complication so that, as in the former case, sufficient healthy

lung tissue remains to carry on respiration in a tolerably efficient manner; but the tubercles are of the soft, yellow variety, and speedily soften and break up into vomicæ. The expectoration, therefore, becomes purulent; hectic fever supervenes, and the patient loses flesh and strength. Nevertheless, in spite of the stethoscopic signs and general symptoms of far-advanced disease, there are indications which, to the practised observer, denote the possibility of the patient's rallying. The respiration is not so quick nor the pulse so much accelerated as in rapidly progressive phthisis. There is not the same burning heat of skin, or the same amount of perspiration, and the aspect of the patient is not expressive of much anxiety or of fatal constitutional disturbance. Accordingly, after a time, if the tone of the system is judiciously upheld, and no fresh deposit of tubercle takes place, the tubercular matter already deposited may be expectorated and got rid of, or may cease to excite local irritation; the cough may subside, the night sweats cease, the appetite return, the patient may regain flesh, and in great measure recover strength. In this case, as in the former, there may be intervals of months or even years during which the patient may enjoy comparatively good health, and in some instances the patient may at length shake off his tendency to the disease, so that no further deposit of tubercle may take place, and practically the disorder may be regarded as cured. But in most cases the symptoms recur at a future period, and each attack, being accompanied by fresh tubercular deposit and destruction of lung tissue, leaves the patient less able to recruit his health and strength. At length, if relapses continue to recur, the time must ultimately arrive when the amount of deposit in the lung, or the breaking down of its tissue, will be such as to render the due continuance of respiration impossible, and then the disease will run its fatal course in much the same manner as in the former instance. But it should be clearly understood that, in spite of the more formidable character of the physical signs and general symptoms, recovery may take place from the earlier attacks as perfectly as when the tubercles do not soften and break down; and that if the deposit of tubercle has not been extensive, the patient may regain apparent health, and may live many years, even after several vomicæ have been formed.

2. Under the title of *Acute Phthisis*, two distinct forms of disease, are usually comprised—the one resembling ordinary phthisis in many of its symptoms, and remarkable chiefly for the severity of the constitutional disturbance, and the extreme rapidity of its course; the other more nearly allied in its general characters to a low type of irritative fever, accompanied by congestive pneumonia. They both run their course

in from three weeks to three months from the time when the patient was supposed to have been in good health, and unless caution be exercised, they are both apt to be mistaken for some other form of malady.

The first variety, which is connected with extensive deposition of yellow tubercle, or else with tubercular infiltration of the lungs, is usually ushered in by severe rigors, followed by anxiety of countenance, heat of skin, a quick, irritable pulse, a short, hard cough, and considerable oppression of the breathing. There is pain and tightness across the chest, and a sense of heavy aching in the shoulder-blades. In the course of a few hours the cough is accompanied by frothy mucous expectoration often specked with blood, but before many days have elapsed the tubercles begin to soften, the character of the sputa is altered, and they become muco-purulent, or even purulent. At the same time the feverish heat of skin which marked the first invasion of the disease is replaced by alternate "chills and heats," and by the profuse perspirations which characterise hectic fever; the anxiety of the countenance increases; the skin is of a pallid yet dusky hue; the pulse becomes more rapid, the cough more frequent, the breathing more hurried and more oppressed, and the lips and face livid; the brain sympathises with the general distress, and wandering or low muttering delirium ensues, especially at night. So matters go on from bad to worse, until, after the lapse of a few weeks, the oppression of the breathing becomes excessive, the sensation of depression and exhaustion extreme, the skin is constantly bedewed with a clammy perspiration, sudamina make their appearance, emaciation progresses rapidly, the pulse can hardly be counted, the tongue becomes dry, the teeth are covered with sordes; and after a few days more of extreme restlessness and of constantly impending suffocation, the patient usually lapses into a state of coma or of semi-consciousness, attended with muttering delirium, and so gradually sinks. In some instances yellow tubercles are found after death extensively disseminated throughout the lungs; there are numerous small vomicæ resulting from the softening of isolated tubercular masses, and much of the intervening lung structure is extremely congested, and oftentimes consolidated by inflammatory exudation. In other cases large portions of the lung structure are thoroughly infiltrated with tubercle which in some parts is seen in a softened state, and in others has been expectorated, and has thus given rise to the formation of vomicæ.

The physical signs which are met with in these cases are by no means characteristic, and can be interpreted correctly only by constant

reference to the general symptoms. There may be deficiency in the expansion of the chest, dulness on percussion, and increased vocal fremitus, but not necessarily at the apices of the lungs; the respiration may be weak or deficient in some spots, coarse and exaggerated, or hollow sounding, and faintly metallic in others; and in others, again, the respiratory murmur may be overpowered or replaced by loud rhonchi and irregular sized, bubbling râles.

The true character of the disease which has produced these signs may generally be diagnosed if due caution be observed. The only malady with which it is likely to be confounded is pneumonia;* and between the two disorders there are many well-marked points of difference. In the tubercular disease the intensity of the constitutional disturbance is out of all proportion greater than the apparent amount of chest affection; the disease occurs equally on both sides of the chest, and is seldom accompanied by much pain or catching of the breathing; there is absence of marked and limited dulness on percussion, of true pneumonic crepitation, aud of the intensely metallic tubular breathing and sniffling bronchophonic resonance of the voice which characterises pneumonic consolidation, whilst, on the other hand, the depressed aspect of the patient, the profuse perspirations, the intense prostration, the gradually increasing but diffused dulness on percussion, and the occurrence of large irregular bubbling, and other signs of pulmonary excavation, point clearly to tubercular consolidation and excavation as the cause of all the mischief.

The second variety is connected with the deposit of innumerable grey miliary tubercles. Its symptoms resemble those which characterise the first variety, except that there is often an absence of cough and of purulent or muco-purulent expectoration, and that the feverish symptoms thereby acquire a greater prominence. If cough is present, it is usually very slight, and hardly attracts attention; and the sputa, if any exist, are very scanty, and consist of little more than frothy mucus. But the fever is considerable, the nervous depression extreme; the breathing is excessively short and oppressed, the lips and face are livid, and the whole surface of the body is of a dusky hue, and usually bathed in perspiration. In short, the general symptoms are those of low remittent fever attended by congestive pneumonia, rather than those which are usually regarded as characteristic of pulmonary consumption.

After death, the lungs are found extremely congested and every-

* In making this statement I exclude the hypothesis of cancerous and other malignant deposits in the lungs.

where studded with innumerable grey miliary tubercles, sometimes so minute as to be scarcely visible; but there are no softened tubercles, and no vomicæ.

The physical examination of the chest gives little more than negative results. There is no observable difference in the size or expansion of the chest on the two sides, nor in the intensity of the vocal fremitus, nor in the sound elicited on percussion, nor in the vocal resonance, nor in the sounds produced by cough or respiration. There is great acceleration of the breathing, and there may be slight harshness and unevenness of the respiratory murmur, and occasionally a few rhonchi or a few small râles, but there are no large bubbling râles, there is no fine crepitation, no tubular breathing, no sniffling, bronchophonic resonance of the voice, and no marked and limited dulness; indeed the dulness on percussion, if any exists, is extremely slight, and is met with equally on both sides.

Thus the physical signs, which vary little from day to day, show the fallacy of the opinion that pneumonia is present, and if duly weighed in connection with the general symptoms can hardly fail to lead to a correct diagnosis. If the oppression of the breathing be not due to pleurisy or pneumonia, it must be referable to bronchitis or phthisis. But the intense heat of the skin, the comparative absence of râles and rhonchi, the almost entire absence of sputa, and the complete absence of muco-purulent expectoration as the malady progresses, conspire to prove that the malady is not bronchitis, and, therefore, that it must be of a tubercular character. In some of these cases of acute phthisis the stomach is irritable, and vomiting occurs; in others, symptoms resembling those of meningitis arise, and in a few diarrhœa supervenes; but, if the disease runs a rapid course, these symptoms with the exception of delirium, are seldom met with in a marked degree. Even emaciation is not a constant symptom. I have seen several patients die of acute phthisis in St. George's Hospital, partly suffocated by the enormous crop of tubercles in their lungs, and partly poisoned by the noxious quality of their own blood, whilst as yet no emaciation had taken place. Indeed, their bodies after death were not only plump, but had large quantities of fat stored up in the omentum and other parts.

3. The latent, or insidious forms of phthisis present a remarkable contrast to those last described. They are characterised by persistent mental and physical depression, and by an absence of fever and of the more prominent symptoms of pulmonary disease. The patient feels low,

and weak, and depressed, but is utterly at a loss to account for his illness. He complains of general lassitude and weariness, of inability to fix his attention, and of being unequal to much exertion; but he has little or no cough, no pain in the chest, no feverish heat of skin, no symptoms, in short, to direct attention to the real seat of mischief, and being more or less dyspeptic, he usually imagines that "it is all stomach." His friends, probably, consider him nervous and hypochondriacal, and rally him on his indolence and lowness of spirits, but the physician observes signs of loss of health, which he justly attributes to deep-seated and serious mischief. He remarks that his patient's aspect is unhealthy; his pupils dilated, the conjunctivæ of a pearl-like colour, the skin unusually pale, the muscles flabby, the extremities often cold, and the pulse feeble—not necessarily much accelerated; he learns by inquiry that the appetite is impaired, the digestion weak, his sleep disturbed and unrefreshing; and together with these signs of general functional derangement and impaired constitutional power, he notes a tendency to emaciation, acceleration of the breathing, and an occasional slight bark or short dry cough after exertion. The cough is, probably, so slight as to have escaped observation, so that the patient will deny its existence; and it often remains so slight throughout the whole course of the disease, as not to form a subject of complaint. Thus the patient will go on for months, suffering, as it is thought, from nervous debility, becoming gradually thinner, but otherwise without material change in his symptoms. At length he finds himself short-breathed and unable to go up stairs without panting; he perspires on the slightest exertion; experiences chills down the spine, followed by burning heat of the palms of the hand, and not unfrequently by perspirations at night; his appetite becomes more than usually capricious, the stomach irritable, and the bowels disordered; and after a time he is attacked with griping pain in the belly, and obstinate diarrhœa sets in. For weeks or even months this may persist in spite of ordinary remedies; and even when it is controlled by appropriate treatment, it is apt to recur as soon as the medicines are discontinued. Meanwhile emaciation proceeds somewhat rapidly, profuse exhausting perspirations alternate with the diarrhœa, pains occur from time to time in the chest; the breathing becomes more oppressed, the mouth and throat become aphthous, the appetite fails, and the patient sinks.

In another class of cases the symptoms at first do not differ materially from those just described, but after a time severe abdominal pain occurs, accompanied by sickness and irregularity of the bowels; there is

gradually increasing distension of the abdomen, caused partly by the effusion of lymph and serum into the abdominal cavity, and partly by flatulent distension of the bowels, and there are great tenderness on pressure and severe febrile excitement. Those symptoms, which are due to a tuberculous condition of the peritoneum and chronic peritoneal inflammation consequent thereupon, may be brought more or less under the influence of treatment, and may thus run a longer or shorter course; but their occurrence is commonly the sign for the commencement of rapid emaciation, hectic fever, and other symptoms which bring the patient rapidly to the grave.

In yet another class of cases the symptoms resemble those above described, except in the occurrence of fistula in ano. The cough is just as slight, the nervous and physical depression as great, and emaciation is also well marked, but diarrhœa is a far less prominent feature, and appears to be replaced by the discharge from the fistula. Sometimes, indeed, a harrassing cough, or a troublesome diarrhœa may coexist with a fistula in ano; but, in the cases of latent phthisis now under consideration, neither the cough nor the diarrhœa assume any importance unless the discharge from the fistula is checked. Nor need they do so even then; for if the patient's health and strength be improved, and the tendency to further mischief arrested, the discharge may be stopped and the fistula cured without any fear of untoward consequences; but if, on the contrary, the discharge be checked whilst the constitutional disorder is still progressing, severe cough or profuse sweating will be excited or diarrhœa will set in, and the malady will assume the more ordinary features of pulmonary consumption.

In some instances of latent phthisis there is almost entire absence of cough from first to last; in others there is slight cough throughout, but it never becomes a prominent symptom; and in others again, it may be scarcely noticeable until the diarrhœa is checked, or the fistula in ano cured, when it often proves extremely troublesome, and the case assumes the ordinary characters of pulmonary consumption. In either case tubercles may be found in the lungs after death in every stage of development and degeneration, the absence of cough being in no degree attributable to the paucity of tubercular matter in the lung, or to any particular stage of its development, but rather to the absence of pulmonary irritation resulting from the local determination elsewhere set up, by the irritation of the fistula in ano, or by tubercular deposits in the glandular structure of the bowels and of the ulceration occurring there.

The general character of the symptoms of phthisis will be obvious

from the description already given of the malady, but their special features require careful consideration. Cough is one of the earliest symptoms, and in some instances is peculiar and characteristic; it is generally unpreceded and unattended by coryza, is rarely paroxysmal, and at first is dry, being due to sympathetic irritation of the larynx, and hence it is often supposed to be a nervous cough, or is referred to relaxation of the throat. At first it occurs chiefly if not exclusively in the early morning, but, as the malady proceeds, it is observed during the day, and especially after exertion, or on any change of temperature. After a time, it ceases to be dry, and is accompanied by more or less expectoration; it recurs at short intervals throughout the day, is especially troublesome at night and, in the morning, is often very severe, and not unfrequently gives rise to vomiting, especially when it occurs soon after a meal; but, in exceptional cases, it is extremely slight throughout the whole course of the disease, and is not attended by expectoration, whilst in others in which the disease arises in sequel of bronchitis, it is attended by expectoration from the date of its invasion. The expectoration is at first scanty, thin, colourless, and transparent, somewhat resembling saliva or gum-water, or it may be of a greyish colour and more or less frothy. After a time, the thin, colourless sputa lose some of their transparency, and are seen to contain specs of opaque matter, which gradually subside and form a deposit resembling the sediment in barley-water, or else remain suspended by the more ropy portions of the secretion, and float in the transparent mucous fluid in the form of striæ. If the expectoration be at first greyish coloured and frothy, it gradually becomes less aerated, more glairy and more tenacious; loses its purely grey colour and is seen to be mixed with specs or streaks of an opaque white or buff colour, and not unfrequently with specs or streaks of blood.

As the malady progresses, the character of the sputa changes again; they gradually become opaque, of a whitish or yellowish hue, and are coughed up in more distinct and more homogeneous masses; sometimes they form ragged pellets of a yellowish white colour somewhat resembling boiled rice in appearance, which sink or else partially float in a colourless semi-transparent, ropy, non-aerated mucous fluid, and at others they are accompanied by very little of this mucous fluid, but form larger masses of a buff or yellowish green colour, flocculent in appearance, yet perfectly smooth in outline, which do not easily coalesce, but remain perfectly distinct and separate from one another if expecto-

rated into a vessel containing water. These sputa have been termed nummular, from their assuming the flat, circular form of a piece of money.

At a still later stage of the disease the sputa assume an ash-grey colour, become perfectly purulent and homogeneous, and are coughed up in smooth, roundish pieces which run together and form one mass if expectorated into a dry vessel, or sink, being perfectly free from admixture with air, if expectorated into water. None of these forms of sputa, however, are perfectly characteristic of phthisis. The boiled rice sputa, the thin, semi-transparent sputa with a deposit resembling barley-water, and the opaque, circular nummulated sputa are those which are most distinctive; but they all occur occasionally in chronic bronchitis, and therefore cannot be relied upon as evidence of consumption.

In certain instances, sometimes through a period of many years, pieces of calcareous matter are expectorated, varying in size from that of a pin's head to that of a pea or a small bean; and in hardness, from that of soft gritty matter up to that of an urinary phosphatic calculus. Sometimes these harder concretions are branched, being moulded in the minute subdivisions of the bronchi. They all indicate a retrocession of the tubercle at the spot where they are formed, but not necessarily a subsidence of the disease, which oftentimes is advancing steadily in other parts of the lung at the very time when these concretions are ejected. In other instances branched *fibrinous* concretions are expectorated, formed by the coagulation of blood in the smaller bronchi.* This however is exceedingly rare.

The quantity of expectoration varies greatly in different cases, and also at different periods of the disorder. Sometimes it is profuse throughout the greater part of the attack; in other instances it is scanty at one period, and profuse—suddenly profuse, as if from the evacuation of a vomica—at another; and in others again it is comparatively insignificant in amount throughout, although large cavities are found in the lungs after death. In exceptional cases it is altogether absent, the sputa being swallowed, as they constantly are by children.

The taste of phthisical sputa is at first saline and disagreeable, but at a later period it is often sweet, owing to the formation and presence of sugar; its odour is ordinarily faint and nauseous—not fetid, unless sphacelation of some portion of the respiratory apparatus has taken place, when it becomes nauseous in the extreme, and acquires what is known as the gangrenous odour.

* See *ante*, pp. 310, 311.

The microscopical appearance of phthisical sputa varies according to the source whence they are derived. Before the tubercles have softened, or before the softened tubercular matter has found its way into the bronchi, the expectoration is derived from the mouth, and from the congested and inflamed mucous lining of the bronchi. It then consists, 1st, of salivary fluid with epithelium from the mouth; and 2ndly, of epithelium from the trachea, bronchi, and air cells, blood corpuscles, mucous corpuscles, exudation cells, and pus globules. But when the softened tubercular matter has made its way into the air passages, the products of disintegration of the lung and the tubercle are found mixed with the matters already mentioned. They consist, 1stly, of pus globules and exudation cells in large numbers, tubercle corpuscles (?), oil globules, saline crystals, amorphous earthy material, and pigmentary matter—the products more especially of disintegration of turbercle; and 2ndly, fragments of the curly yellow elastic tissue of the lung, capillary vessels, and blood corpuscles—the products of the disintegrated pulmonary tissue. These fragments of this lung tissue often afford the earliest obtainable evidence of the existence of tubercle, and in the absence of symptoms of pneumonic abscess may be regarded as almost a distinctive characteristic of phthisical sputa. In every instance, but especially in obscure cases and the earlier stages of the disease, the discovery of the curly elastic fibre of the lung is most important, as it removes all doubt as to the nature of the disorder, and imparts a significance to symptoms which might otherwise be regarded as trivial, and not requiring active medical interference.

Hæmoptysis is often one of the earliest, and is certainly one of the most frequent symptoms of phthisis. It may vary from a mere speck or streak of blood, to an ounce, a pint, or even a larger quantity, and in some instances it proves rapidly, if not immediately fatal, producing instant asphyxia, or killing indirectly after a few days by exhaustion. Seldom, indeed, does consumption run its course without hæmoptysis to a greater or less extent, but fatal hæmorrhage, as the result of tubercular ulceration, is an event of very rare occurrence. Dr. Walshe names 81 per cent. as the proportion of cases in which hæmoptysis occurs, and reports having met with two instances of rapidly fatal hæmorrhage amongst 131 analysed cases. My own experience leads me to affirm, that, if the observation be confined to adults, and if cases of acute and rapidly fatal phthisis be excluded from the calculation, hæmoptysis to a greater or less extent occurs in every instance; and it also warrants my asserting that fatal hæmorrhage is far less frequent

than is represented by Dr. Walshe's numbers. I have only met with three such cases, though I have seen many hundreds of deaths from phthisis.* In children profuse hæmoptysis is even less frequent.

Hæmoptysis is very commonly met with at the outset of the disease, and recurs at longer or shorter intervals throughout its progress. In most instances the blood is small in quantity, and only specks or streaks the sputa; but sometimes, especially in florid persons, profuse hæmorrhage occurs as the first or earliest noticed symptom. This has led to the erroneous impression that consumption is occasionally referable to hæmoptysis. The blood is usually of a florid, red colour, not unfrequently of a brick-red hue, and sometimes, though rarely, except in the later stages of the complaint, of a venous tint. As the disease advances, it is ejected more frequently, and in rather larger quantity, forming small clots, and it is more commonly met with in the sputa of males than in that of females,† and in that of adults than in that of children. When it is ejected in large quantity, the blood is frothy from admixture with air.

When very profuse in quantity, hæmoptysis tends to serious obstruction of the breathing, and possibly may thus accelerate the progress of the tubercular disease, but in smaller quantity it may act beneficially by relieving the local congestion, and it certainly does not hasten the fatal termination of the disorder. Even when very profuse it does not necessarily shorten life. It may occur once and may not recur, or it may take place frequently without producing permanent ill effects. A gentleman, whose mother was phthisical, and who died last year of consumption at the age of fifty-six, had coughed up almost yearly during the last thirty-three years, and sometimes two or three times a year, blood, varying in quantity from a few ounces to more than a pint, and yet, until the last two years, he had enjoyed good health during the intervals of the attacks. Such cases as these however are quite exceptional. In some instances the outpouring of blood into the air passages, in connection with phthisis, gives rise to the production of pulmonary apoplexy; but this occurrence also is rare—so rare, that it was met with

* I have seen several patients, supposed to be phthisical, die suddenly of hæmorrhage from the lungs; but *post-mortem* examination has revealed the existence of an unsuspected aortic aneurism, from which, and not from tuberculous ulceration, it was proved that the hæmorrhage took place.

† This is contrary to Louis' observation. He reports that hæmoptysis is more frequent in females than in males, in the proportion of three to two. Dr. Walshe's experience coincides with mine in reference to this matter.

only eight times amongst the 517 consumptive patients examined in the dead-house of St. George's Hospital during the ten years ending December 31st, 1850.*

From what has been already stated, it is obvious that the prognosis of hæmoptysis in phthisical cases must be somewhat uncertain. Ordinarily, little fear need be entertained of a fatal issue, unless the hæmorrhage be very profuse, and even then the chances are immensely in favour of temporary recovery. The most unfavourable sign is persistent frequency and irritability of the pulse, indicating great excitement of the circulation. In such cases the prognosis should be very cautious; for when once a severe attack of bleeding has occurred, profuse hæmorrhage is apt to take place at some future date. This at least is the result of my observation. In each of the three fatal cases of hæmoptysis which I have met with, profuse hæmorrhage from the lungs had previously taken place.†

Pain in the chest, especially acute pain, is rarely an attendant on the early stages of phthisis, but a dull aching uneasiness under the clavicles or the shoulder-blades is one of its most constant symptoms. As the disease advances, pain—sharp pain, though not very acute or catching—is often experienced in the chest, probably caused sometimes by simple pleurodynia, or by the morbid changes going on in the lung, but in most instances by local attacks of dry pleurisy. They differ from the pains which accompany bronchitis, in being felt in the side, or in the back, or under the shoulder-blades, rather than under the sternum, and in being aggravated by inspiration almost as much as by coughing, which alone produces much pain in bronchitis.

Dyspnœa is by no means a frequent symptom. When it does occur in any marked degree, it is not referable to unmixed phthisis, but to some coexistent mischief, such as heart disease, pleurisy, pneumonia, bronchitis, or pulmonary emphysema. In one case which fell under my care at St. George's Hospital, dyspnœa occurred to a frightful extent, and was found to be referable to rapidly developed and extensive emphysema of the lung.‡

But though dyspnœa does not occur except on active exertion, *acceleration of the breathing* is a constant and most valuable diagnostic symptom. It is one of the earliest indications of phthisis, and in the

* See Chambers, loc. cit., p. 66.

† For further particulars respecting hæmoptysis, see *ante*, chap. ii, pp. 255-262.

‡ This case, which is most interesting in a pathological point of view, is reported in the 'Trans. Path. Soc. of London.,' vol. xi, p. 15.

absence of heart disease and of any bronchitic, pneumonic, or pleuritic complication, it serves as a tolerable index to the extent of tubercular mischief. Even when the patient is at rest, and appears to be breathing tranquilly, the frequency of the respirations will prove to be above the normal standard, and on the slightest exertion may rise to a degree which is quite inconsistent with a healthy chest. Their frequency bears a marked relation to the state of the pulse, and consequently, in many instances, to the severity of the febrile symptoms; but when the lungs are extensively occupied by tubercles, the respiration is very frequent, although there be little febrile excitement.

Febrile symptoms seldom occur in the very earliest stage of the disorder, but at a somewhat later period they begin to show themselves, and in the second and third stages assume the form which is recognised as hectic. Sometimes, however, in chronic cases they are not well marked, even after vomicæ have been formed. They usually commence with chilliness, followed by burning heat of skin, and then by profuse perspiration; and this train of symptoms, which constitutes a perfect paroxysm, may occur at noon, and again towards evening. More commonly, however, they occur only at night, and the perspiration continues until early morning. The paroxysm is often imperfectly developed, the shivering being slight, though the perspiration is profuse, and not unfrequently the sweatings occur even when no distinct chilliness has been previously experienced. Indeed, at an advanced stage of the disorder perspirations break out whenever the patient falls asleep, and are extremely copious and enfeebling. No symptom is more remarkable or more distressing than these colliquative sweats in phthisis, and no source of drain on the system appears to be more exhausting. Thus, it usually happens, that when the perspirations are profuse, and persist in spite of treatment, the case runs on rapidly to a fatal termination.

Emaciation is one of the most striking and characteristic features of phthisis, and one which is always present, unless the disease has run a rapid course, and has cut off the patient ere time enough has elapsed to admit of wasting taking place. Not unfrequently it is the symptom which first attracts notice, and excites the apprehension of the patient and his friends. Referable, without doubt, to the general cachexia and to the mal-nutrition consequent thereupon, it often commences whilst the cough is as yet trifling, and before loss of appetite, diarrhœa, or perspiration have set in. When these sources of waste are superadded, it progresses with increased rapidity. The fat which

is stored up in the body disappears, the cellular tissue shrinks, the muscles waste, even the heart decreases in weight, and the whole body becomes rapidly thinner and lighter. The only structure which appears to withstand the general loss of volume is the tongue, which even up to the time of death does not materially diminish in size.

There are exceptions, however, to this law of waste. In acute and rapidly fatal phthisis the patient is sometimes asphyxiated before emaciation has taken place; and in certain chronic cases I have seen patients die whilst as yet plump, although vomicæ had long existed in the lungs. But in these latter cases the patients, although consumptive, have not succumbed to phthisis, but have been carried off by pneumonia, acute bronchitis, or some other disorder.

As a general rule, emaciation progresses least rapidly when the appetite remains good. It should be clearly understood, however, that the mere amount of food taken does not afford a trustworthy clue to the rapidity of its progress, for excessive wasting will often occur whilst food is being taken in large quantity; and in other cases it will proceed slowly, even though food be eaten sparingly. So, again, it will sometimes go on more rapidly in persons who are free from the ordinary symptoms of dyspepsia than in those who are troubled with flatulence, acid eructations, and other signs of indigestion. The fact appears to be, that it is regulated more by the mode in which the whole process of assimilation is performed, than it is by the quantity of food taken, or by the occurrence or non-occurrence of mere stomach derangement. My own impression is, that it is closely connected with the extent of tuberculization of the mesenteric glands. Be this as it may, emaciation, though not necessarily proportioned to the extent of pulmonary disease, and not even a necessary accompaniment of it, is, when it exists, a very trustworthy guide. It is always an alarming symptom of disease; and when it occurs without any apparent cause, coincidently with shortness of breath, acceleration of the pulse, and a slight, hacking cough, it may be regarded as almost conclusive evidence that tuberculization is making havoc in the lungs, and probably is implicating the mesenteric glands.

Sore throat is a symptom experienced by many consumptive patients long before pectoral mischief has declared itself, and as the tubercular disease progresses, it becomes a serious aggravation of the patient's sufferings. The affection now referred to is not the aphthous ulceration frequently met with in the later stages of consumption, but congestion of the mucous membrane lining the posterior fauces, the pharynx, and

the larynx, accompanied by a diseased condition of the follicles, and a morbidly increased secretion of viscid mucus. It is an affection analogous to that known as the "clergyman's sore throat," and is especially prone to occur in persons between the ages of twenty and forty who are weak and cachectic, and whose digestive organs are out of order. It gives rise to a constant soreness or uneasy sensation in the throat, a frequent inclination to swallow, and repeated hawking to clear the throat, which, after a time, results in the ejection of a small quantity of viscid, opaque mucus. It is often confined to the posterior fauces or pharynx, and in such cases begins and ends as a "relaxed sore throat;" but when it extends to the epiglottis and larynx, it produces more or less huskiness or hoarseness. How far this affection is connected with the deposit of tubercle in the diseased follicles of the lining membrane is a point on which different opinions are entertained; and it is probable that the truth lies between the theory which denies that tubercle is ever secreted by those follicles, and that which refers the disease invariably to the deposit of tubercle in the follicles. Certain it is, on the one hand, that the affection is met with and permanently got rid of in persons who, though weakly and dyspeptic, are not consumptive; and, on the other, that in some instances enlarged follicles may be seen, filled with a matter closely resembling tubercular matter. Further, it is certain that in persons who, in the earlier periods of their illness, have suffered from this form of sore throat, the more distinct tubercular affection of the larynx, hereafter to be described, is apt to arise at a later period.

The pulse affords an early, and therefore an important indication of consumption. In exceptional cases it may not prove above 60 or 70 in a minute throughout the whole course of the disease, but more commonly it rises to 80 or 90 at an early period, and as the malady advances may number 120 or 140. A pulse constantly above 80 in a person who is short-breathed and has a slight hacking cough, is always suggestive of tubercular disease, however stout and otherwise healthy he may appear; and a confessedly phthisical patient, whose pulse is habitually very quick and weak, has little chance of even temporary amendment.

The digestive organs sometimes remain in tolerable order, and the appetite, though capricious, is not materially impaired; but more commonly the appetite fails, and the digestive organs are disturbed to a greater or less extent, even in the earlier stage of the disease; and at a more advanced period the disturbance is very serious. At first there are pain and tenderness at the epigastrum, increased after food, together with nausea and occasional vomiting; the bowels are costive, or ex-

tremely irregular, and small aphthous ulcerations occur in the mouth. As the disease advances, tuberculization of the mesenteric glands, and of the glandular follicles of the bowels, occurs, the abdomen becomes tender on pressure, ulceration of the bowels takes place, and diarrhœa sets in, accompanied by extremely offensive evacuations, sometimes mixed with blood; nausea and vomiting after food become more frequent, and the mouth and fauces are apt to become extensively aphthous, so that deglutition is rendered painful and difficult. This, at least, is the ordinary course of the disease; but experience has proved that extensive tubercular ulceration of the bowels may be going on although constipation exists, and although there be no pain in the abdomen, no nausea or vomiting, and no tenderness on pressure. In some instances peritonitis is set up, and occasionally, though rarely, ulceration of the bowel terminates in perforation and the escape of fæcal matter into the abdominal cavity. In this case excruciating pain occurs in the abdomen, collapse ensues, and the patient usually sinks in a few hours.*

Diarrhœa is of common occurrence, and is a symptom of grave significance in doubtful cases of consumption. Oftentimes, indeed, it serves as an index to the true nature of symptoms about which some doubt had been previously entertained. The diarrhœa of phthisis is not an ordinary looseness of the bowels, nor is it due to ordinary causes; it has a special origin, and is marked by several peculiarities. It is troublesome, but not painful; it is very persistent, and is seldom amenable to ordinary remedies; it is apt to recur; and it is not usually accompanied by the furring of the tongue, and the sickness and vomiting which attend other forms of diarrhœa. Hence its character may be recognised after a few days' observation; and when, in a person suffering from cough, the bowels, which previously have been costive, become habitually relaxed, and the looseness assumes the characters above mentioned, there can be little doubt as to the true significance of the cough, or as to the nature of the coexisting constitutional malady.

The tongue is sometimes clean, at others coated; but when ulceration of the bowels is going on, it is oftentimes clean, red, and beef-steaky in appearance, and not unfrequently aphthous. Its condition, however, does not throw any light on the progress of the pectoral disease.

* A patient in whom this complication arose was admitted under my care into St. George's Hospital on the 21st of December, 1859. For full particulars of the case, which was remarkable for the entire absence of diarrhœa and other abdominal symptoms during life, see 'Trans. Path. Soc. of London,' vol. xi, pp. 103-4.

The urine in the earlier stages of the complaint is sometimes normal, but more commonly it is remarkable for the rapid variations in its character. It is at one time clear, pale, and abundant, and of low specific gravity, and in a few hours, or on the following day, scanty, high coloured, and possibly loaded with lithates. When the disease is more advanced, it generally contains an excess of lithic acid, and is often turbid. Albumen is not usually one of its constituents, though a minute quantity may be detected in it from time to time, referable to temporary congestion of the kidneys. In a certain proportion of cases, however, it is more constantly present, owing to the existence of tubercular disease of those organs.

The menstrual discharge may remain natural to the last, both in point of quantity and quality. More commonly, however, it is very profuse or else very scanty at the commencement of the disease, and ceases as soon as the phthisical symptoms are more fully developed. In some instances it ceases at the very commencement of the malady, and its sudden cessation is referred to by the patient as the cause of all her subsequent illness; indeed, the apparent connection between the suppression of the catamenia and the setting up of pectoral disease is sometimes so remarkable, that many writers on consumption have referred to cases under the title of amenorrhœal phthisis. It is almost needless to add, that the term is an improper one, the phthisical symptoms being in no wise referable to the stoppage of the monthly discharge, but rather connected with the morbid condition of the system, which led to its cessation. In many of these cases leucorrhœa is a prominent symptom.

The external and superficial glands are especially liable to become the seat of tubercular deposit in children, and form knotty swellings, which greatly disfigure the patient. In adults they are rarely enlarged. When the enlarged cervical glands suppurate, as they often do, the pectoral symptoms are usually quiescent; and it is a rare occurrence to find active mischief in the chest coexisting with tubercular suppuration in the neck. Indeed, glandular enlargement, though symptomatic of tuberculosis, and sometimes coexisting with phthisis pulmonalis, can hardly be regarded as a symptom of that disease. On the contrary, though indicative of a scrofulous disposition, it seems to be in some measure antagonistic to tubercular deposition or suppuration in the lungs.

The fingers and nails undergo a change which requires special notice; the more so as diagnostic importance has been attached to it. Sometimes, in the earlier stage of the disease, but more commonly when emaciation

has proceeded to some extent, the last joints of the fingers enlarge, their tips shrink and diminish in size, and the nails become remarkably convex and adunc. The ends of the fingers thus acquire a peculiarly rounded or clubbed appearance. There cannot be a doubt that this change of form is very remarkable, and is observed to a greater or less degree in most cases of phthisis; but, nevertheless, it cannot be regarded as distinctive. In several healthy, middle-aged, and elderly persons, I have seen this condition of the fingers strongly developed, and in some phthisical patients it does not occur even up to the last hour of their existence.

So, also, in regard to falling of the hair, which is a symptom of great frequency in consumptive cases. In many instances it is observed very early in the attack, and is a common cause of complaint long before the patient or her friends suspect the nature of the malady which is impending; but in certain instances, and more especially when the disease runs a chronic course, unaccompanied by much sweating, the hair is retained until the last.

Œdema of the extremities is not of frequent occurrence; indeed, its absence is rather remarkable, when we consider the impoverished condition of the blood, and the serious impediment offered to its circulation through the lungs. Nevertheless, the feet and ankles are apt to become œdematous towards evening, especially at the close of the disease, so that the occurrence of œdema may be regarded as of evil augury. But, except in the later stage of the complaint, and to the extent just described, I believe that œdema does not occur simply as the result of the conditions above referred to. In certain instances the legs become anasarcous, as a result of coexisting disease of the kidneys; in others, in consequence of feebleness of the heart dependent on fatty degeneration of its tissue; whilst, in certain cases, one or both of the lower extremities become excessively swollen and anasarcous, in consequence of the coagulation of blood in the veins. In this latter event, if phlebitis be present, the swelling is attended with more or less pain in the course of the veins.

The functions of the brain are seldom interfered with to any great extent. Slight wandering at night, or on waking out of sleep, is often observed towards the close of the disease; but, even in such cases, the mental powers remain unimpaired during the day. Indeed, the perceptive faculties oftentimes acquire unwonted vigour, and the imagination is unusually active. Sometimes, however, violent delirium sets

in, or the patient passes into a state of muttering delirium, and then becomes comatose. In these cases, if the irritation results from the deposit of tubercle in the brain, or in its investing membranes, a rapidly fatal issue may be anticipated. If, on the other hand, there is reason to believe that the delirium has been caused, as sometimes happens, by injudicious and depressing treatment, the prognosis would be less unfavourable, inasmuch as the mischief may then be overcome by appropriate remedies.

A strange waywardness of temper usually marks the disease throughout. The patient is at one time irritable and depressed; at another placid and singularly hopeful, maintaining up to the last a delusive hope that the malady will be subdued. So unaccountable is the apparent hopefulness in many instances, that it might almost be supposed referable to an anxiety on the part of the patient to deceive herself and her friends as to the actual state of the case. But the absence of this feature in other forms of complaint which are equally lingering and equally fatal, proves that some different explanation must be sought, and stamps it as a characteristic of consumption.

The complications of phthisis pulmonalis have been already alluded to. Pleurisy, pneumothorax, pneumonia, bronchitis, and hæmoptysis, which are some of the more important, have been discussed under their respective heads, and ulceration of the bowels has been briefly described in the present chapter. But there are yet others which require further consideration. Amongst these may be mentioned chronic tubercular inflammation of the epiglottis, larynx, and trachea, tuberculization of the bronchial glands, and irritation and inflammation of the brain or its membranes consequent on the deposit of tubercle.

It has been already stated, that hoarseness and partial loss of voice are apt to supervene in the course of pulmonary consumption. They are due to congestion and inflammation of the epiglottis and larynx, arising in consequence of local tubercular mischief, and when once established, they are apt to continue in a chronic form, and not unfrequently increase gradually in intensity. Sometimes this alteration of the voice is slight, and does not occur until the pectoral disease is far advanced; at others, it is observed very early in the attack, and becomes a prominent symptom soon after the commencement of the catarrhal symptoms, by which the invasion of the disease is marked. Many writers have alluded to cases of this kind under the title of 'Laryngeal Phthisis,' and have implied, if they have not actually

affirmed, that tubercular mischief may occur in the larynx, giving rise to symptoms of the so-called laryngeal phthisis, without the existence of any tubercular disease in the lungs. This, however, is not the fact. The tubercular deposit in the lungs may be more or less extensive, and the pectoral symptoms may be developed to a greater and less degree, but the so-called laryngeal phthisis never runs its course without the coexistence of tubercular disease of the lungs. In short, there is no such disease as laryngeal phthisis. The chronic inflammation or tubercular ulceration in the larynx often met with is merely an accessory or accidental complication of phthisis pulmonalis.

The laryngeal symptoms, however, are somewhat peculiar, and impart an entirely new complexion to the complaint. They are characterised by an alteration in the force and quality of the voice and cough, and by pain, tenderness, and a sense of dryness, pricking, and constriction over the larynx. At first there is simply occasional huskiness and "cracking" of the voice, which disappears after a forcible "hemming" or "clearing of the throat," but gradually more permanent hoarseness and partial loss of voice supervene, with a muffled and cracked sound on coughing—the cough being frequent and accompanied by a scanty mucous expectoration. As the disease proceeds, and the chordæ vocales are destroyed by ulceration, the voice degenerates into a hoarse or a squeaking whisper; the breathing in chronic cases becomes markedly stridulous; and the cough, which is constant, and attended by the expectoration of a large quantity of thin, frothy mucous or muco-purulent fluid, sometimes tinged or streaked with blood, produces a strangely mixed hoarse and stridulous sound. Yet with all this evidence of local mischief, there is seldom much febrile excitement, and at first, not unfrequently, very little pain or difficulty of breathing.

If the epiglottis remains free from mischief, there is no dysphagia, but when, as often happens in the latter stage of the disease, it becomes inflamed or ulcerated, each attempt at swallowing may give rise to a paroxysm of suffocative dyspnœa and spasmodic cough, the liquid which the patient was in the act of swallowing being rejected through the nose as well as through the mouth. This, however, is not always the case, as *post-mortem* investigations have clearly proved that the epiglottis may be ulcerated, and there may be no dysphagia nevertheless.

Sometimes, though rarely, portions of the cricoid and arytænoid cartilages become detached during the process of ulceration, or cretaceous deposits which have formed in the larynx become loose, giving rise to

excessive cough and a sense of impending suffocation. In most instances these hardened masses are ejected with expectoration during a violent fit of coughing, but occasionally they pass into the trachea and occasion great local irritation and an aggravated attack of dyspnœa. Death commonly takes place, as in uncomplicated cases of pulmonary consumption; but occasionally it results from paralysis of the glottis, induced by the extension of ulceration, or from spasm excited by the passage of foreign matters into the diseased larynx. After death, the congested mucous membrane of the larynx often exhibits a granular appearance; the edges and laryngeal surface of the epiglottis are found ulcerated, or ulcers are discovered in the ventricles of the larynx, or between the epiglottis and the chordæ vocales; the laryngeal muscles are wasted and often softened, and portions of the cartilages are sometimes ulcerated and necrosed. In many instances ulcerations exist in the upper part of the trachea, but seldom give rise to any marked symptom; in others false membrane is found effused into the larynx and upper part of the trachea,* and occasionally ulceration perforates the larynx and extends into the œsophagus, giving rise to excessive dysphagia. So common is ulceration in some part of the upper air passages, that Louis reports having met with it in the epiglottis and larynx in one out of every five consumptive patients whom he examined after death, and in the trachea in no less than one out of every three patients.

The diagnosis of phthisis pulmonalis is a matter of difficulty when the larynx is affected, inasmuch as the mischief in the upper part of the air passages obscures the sounds obtainable through the stethoscope. The general symptoms, however, are sufficiently characteristic; and when, in addition to hoarseness and partial aphonia, there are present the peculiar laryngeal cough, with thin frothy muco-purulent expectoration, semi-stridulous respiration, night sweats, emaciation, and other of the ordinary symptoms of consumption, there can be little doubt as to the true nature of the malady, however masked the chest symptoms may be. If however any doubt remains as to whether the stridor and other symptoms of laryngeal affection may not be referable to aneurismal pressure or to

* A case in which this occurred, and in which tracheotomy had to be performed, is at present (Nov. 7th, 1861) in St. George's Hospital, under the care of my colleague, Dr. Bence Jones. The patient, a man named Kent, in Fuller Ward, ejected through the opening pieces of false membrane an inch and an inch and a half in length, and about the breadth and thickness of a quill pen.

malignant or other disease of the larynx, recourse may be had to the laryngoscope—an instrument devised by M. Czermack and M. Türck for the purpose of inspecting the pharynx and larynx. By its aid the physical condition of the larynx can be readily ascertained, and all questions as to the existence of laryngeal disease set at rest.*

The prognosis of the disease is always unfavourable, from the fact already mentioned, that the laryngeal mischief is accompanied by tubercular deposit in the lungs. Generally, when hoarseness and aphonia have set in, the progress of the case is rapid, and the patient does not survive beyond a few months. Nevertheless, as with other forms of tubercular disease, its course may be almost indefinitely protracted. A phthisical patient at present under my care has suffered from hoarseness, partial loss of voice, and occasional pain and constriction in the larynx for a period of seven years, and at the present time is again rallying from an attack which for some weeks threatened to terminate fatally.

Tuberculization of the bronchial glands sometimes occurs when the lungs are free from tubercular deposit, but more commonly, it is associated with pulmonary disease, being merely a complication of that disorder. Like other scrofulous affections of the lymphatic system, it is specially prone to arise in children, and may be regarded as an exceptional occurrence in adults. Insignificant in some instances in clinical importance, it is sometimes so extensive as to give rise to symptoms far outweighing those produced by the coexistent pulmonary disease. Thus a congeries of tuberculized bronchial glands may form a large, hard, and irregularly lobulated mass in the chest,† which will compress, and may ultimately produce ulceration and perforation of the adjacent structures, giving rise to the various local and physical signs of mediastinal tumour and sometimes to symptoms which may be mistaken for croup. Twice

* The laryngoscope consists of a speculum or mirror and of a concave metallic reflector, which is perforated in its centre. The reflector is fixed before the eye by means of a common spectacle frame, or by being held between the teeth; and the physician, looking through the hole in its centre, introduces the mirror into the pharynx and holds it so as to throw light into the larynx, and at the same time to reflect an image of the parts into his own eye. In this way, after a little practice, the larynx may be readily inspected. M. Czermack, through whose kindness I was first taught how to employ this instrument, informs me that many practitioners experience at first considerable difficulty in using it, but that with a little practice the mode of employing it is soon acquired.

† In the case of Henry Truton, aged twenty-five, who was admitted under my care into the King's Ward of St. George's Hospital, on November 13th, 1861, a mass of tuberculous bronchial glands was found, which weighed more than two ounces.

I have been called in consultation to decide upon the propriety of performing tracheotomy in cases of supposed croup, when the symptoms during life, which were subsequently corroborated by *post-mortem* examination, convinced me that the mischief was due simply to a mass of enlarged bronchial glands pressing upon the trachea just above the bifurcation of the bronchi. A similar case, the particulars of which will be found in the *post-mortem* and case-book in the museum of St. George's Hospital, came under my observation in the year 1861. In each instance I was enabled to decide against the existence of croup, by observing that the stridor accompanied expiration rather than inspiration, and to diagnose the true nature of the malady by finding this peculiar respiration accompanied by dulness on percussion between the scapulæ, and by symptoms indicative of a scrofulous habit of body, and by an absence of all evidence of any other form of disease capable of producing the stridulous expiration.

Tubercle, when deposited in the bronchial glands, is prone to undergo degeneration and softening, just as it does in other parts of the body,* and sometimes it becomes the seat of cretaceous transformation. When the enlarged glands suppurate, the softened tubercular matter may make its way by ulceration into a bronchus, or into the trachea, and may then be expectorated, or it may even open into the œsophagus, and occasion more or less dysphagia. In either case the excavated gland may give rise to the physical signs of a vomica.

It is needless to detail the local symptoms of mechanical origin produced by enlarged bronchial glands, as they are identical with those occasioned by other intra-thoracic tumours.† Suffice it to say that swelling of the superficial veins of the neck or chest occurring in a child, when accompanied by lividity of the lips, puffiness of the face, œdema limited to one side, or affecting both sides of the neck and chest, constitute a train of symptoms which are extremely suggestive

* A case in which this softening occurred was admitted under my care in St. George's Hospital, on May 6th, 1861. The child, Harriet Bennett, aged four, was sent into the hospital as a case of croup with a view to operation, but as the stertor was almost confined to expiration, and there was abundant evidence of scrofulous disease, I did not consider it a case of croup, but regarded the symptoms as attributable to the pressure of a mass of bronchial glands on the trachea. After death this was found to be the case, and it was discovered further that this scrofulous mass had softened and formed an abscess which had burst into the trachea. (See 'Hospital museum Post-mortem and Case Book' for 1861.)

† They are fully discussed in the section relating to that subject.

of the disease under discussion, and that when to these symptoms are superadded dysphagia and ringing, paroxysmal cough, stridulous breathing, hectic, and emaciation, there can be no reasonable doubt as to the nature of the malady. It may be added, that whereas aneurismal and other intra-thoracic tumours are of extreme rarity or almost unknown in children, bronchial gland tumours are of somewhat common occurrence. Therefore, if pneumonia or bronchitis be not present, the presence of laboured respiration in children, attended by greatly prolonged expiration, and dulness of percussion between the scapulæ, affords sufficient grounds for suspecting the existence of enlarged bronchial glands.

The general symptoms vary greatly, according to the degree to which the enlarged glands press on the surrounding structures, and to the amount of tubercular mischief in the lungs, and the extent to which it has advanced. Ordinarily the patient's face is pale and anxious, the breathing hurried, often laboured, and sometimes stridulous; there is frequent cough, which is harsh, dry, ringing, and paroxysmal, when referable to pressure on the trachea or bronchi, or to irritation of the vagus or recurrent nerves, but hoarse, loose, and non-paroxysmal when due principally to tubercular mischief in the lungs. If the patient be a child, there will be little or no expectoration throughout, inasmuch as the softened tubercular matter will be swallowed; whereas if the patient be an adult, the sputa will be catarrhal, muco-purulent, or purulent, according to the stage at which the lung disease has arrived. Sometimes cretaceous matter from an enlarged bronchial gland may be discovered in the expectoration. If the tuberculous mass causes pressure on the trachæa or bronchi, or irritation of the recurrent nerve, not only will the breathing be stridulous, but violent paroxysms of suffocative dyspnœa may arise, accompanied by lividity of the surface, extreme anxiety of countenance, and cold, clammy perspiration. If there be pressure on and subsequently ulceration of the œsophagus, there will be gradually increasing difficulty of swallowing, and ultimately extreme dysphagia, with a sense of soreness or pain on attempting to swallow. The ordinary symptoms of pulmonary consumption will be gradually superadded to these, according as mischief in the lungs is more and more developed.

Thus, then, it will be seen that in the earlier stage of the disease there is no positive evidence of enlargement of the bronchial glands, and that inasmuch as the signs to which this malady gives rise are simply those of pulmonary irritation and local pressure—signs which may be

produced by any intra-thoracic tumour—the diagnosis of enlarged bronchial glands is extremely difficult: indeed, it can only be a matter of inference from the absence of symptoms indicating the existence of other forms of mischief which, equally with tuberculization of the bronchial glands, are capable of developing tumours and causing pressure on the structures within the chest. In adults, therefore, in whom intra-thoracic tumours of all sorts are apt to arise, the diagnosis must necessarily be very uncertain. But when, as is commonly the case, this form of disease is met with in children, the diagnosis is less difficult and less uncertain, for the reason that in them no other form of mischief is liable to occur capable of exciting many of the symptoms ordinarily attendant on this affection, and that percussion between the shoulders elicits dulness referable to the enlarged glands so much more readily than it does in adults.

The last complication of pulmonary consumption which requires special notice is inflammation of the brain or its investing membranes, due to the local irritation of tubercular deposit. Tubercles may occur in the nervous centres, independently of tubercular mischief in the lungs; and by reference to Dr. Chambers' analysis of the *post-mortem* examinations in St. George's Hospital it will be found that they did so in five cases which proved fatal during the ten years ending December, 1850. But more commonly this form of mischief is associated with tubercular disease in the lungs, and forms one of the many complications which are apt to arise during its progress. It is especially prone to occur in childhood. The first symptoms which attract attention are unusual peevishness and irritability of temper, restlessness at night, and grinding of the teeth during sleep. Shortly the patient complains of headache and occasional giddiness, the scalp is hot, and there is aversion to light and noise; febrile excitement ensues, with constipation and obstinate vomiting, and the countenance becomes distressed and anxious. After the lapse of a few hours, or possibly a few days, the headache increases in severity, the pulse becomes irregular, delirium is apt to occur, especially in adults, and twitchings of the face or actual convulsions take place, followed in many instances by strabismus, and by more or less hemiplegia or general paralysis. The pupils, which at first were contracted, eventually dilate, the delirium ceases, and extreme drowsiness or actual coma supervenes, and this may endure for periods varying from a few hours to ten days, or even longer, varied only by occasional convulsions. Sometimes the delirium is violent and boisterous, more

commonly of a quiet character, and occasionally the cerebral disturbance does not result in true delirium, but rather in a peculiar loss of mental power, which renders the patient speechless, and gives a dull, meaningless expression to his countenance. He seems to understand what is said to him; but after gazing at the speaker for a few moments, deliberately turns his head away, without attempting to speak or give an answer to any question which may be put to him.

The prognosis in these cases is extremely unfavourable. Not only is there in most instances coexistent disease in the lungs sufficient of itself to prove eventually fatal to the patient, but even temporary recovery from these attacks of delirium is rare. Cases, indeed, are met with occasionally in which the symptoms seem to betoken tubercular mischief in the brain or its membranes, and in which nevertheless the cerebral disturbance gradually subsides, and the patient regains the full possession of his faculties. But more commonly, notwithstanding occasional remissions, the extreme drowsiness or stupor, or actual coma, continue to a greater or less extent, and the patient sinks after a few days, or, at all events, within a fortnight or three weeks.

It should, perhaps, be stated, that during these attacks the chest symptoms sometimes remain in abeyance to a great extent, and, on the other hand, that constipation, which so frequently is an attendant on cerebral disorders, is seldom met with when the head symptoms occur during the advanced stage of phthisis, in which ulceration of the bowels so commonly prevails. These facts are both important, as, if not borne in mind, they might be apt to mislead.

It will be obvious, from what has been already stated, that the diagnosis of pulmonary consumption must be a matter of extreme uncertainty to a person unpractised in conducting a physical examination of the chest. Not only may the general symptoms of phthisis be obscure, but bronchitis, pneumonia, or pleurisy may be present, and mask the true nature of the disorder. On the other hand, it may not be without a certain degree of difficulty even to the most experienced auscultator; for in the earlier stages of the disease there is no one sign by which the existence of tubercle is clearly indicated, and the general symptoms may each one separately accompany bronchitis, pneumonia, or pleurisy; nay more, when referable to the presence of tubercle, they may be complicated, and masked by the existence of either of those forms of disease. And thus it happens, that if great care be not exercised in conducting the examination of the chest, and much caution observed in drawing inferences from

the signs observed, an incorrect conclusion will be arrived at. In short, the physical signs alone are oftentimes as insufficient as the general symptoms to form trustworthy data for an opinion; and the existence of consumption is only to be established by a careful inquiry into the history of the patient, by a jealous investigation into the general symptoms, and by a comparison of those symptoms with the physical signs which are revealed by an examination of the chest.

Bronchitis is the disease with which phthisis is most liable to be confounded, and it may be well, therefore, to place in a tabular form the salient diagnostic features of the two diseases.

Incipient Phthisis.	*Bronchitis.*
The cough commences gradually, without fever, and without the running at the eyes and nose which characterise an ordinary "cold."	*The cough* commences suddenly, and is usually ushered in by feverishness and running at the eyes and nose.
The cough is generally dry and hacking for some time after its commencement.	*The cough* is accompanied by expectoration almost from the first.
The expectoration is often specked or streaked with blood.	*The expectoration* is not tinged or mixed with blood.
Pain.—There is seldom much pain in the chest, and rarely any fixed pain. When pain does occur, it is usually of a dull, aching character; is met with in the sides or under the collar bones or the shoulder blades; and is not usually aggravated by coughing.	*Pain.*—There is almost invariably pain or tightness or uneasiness under the breast-bone; but rarely any pain in the sides, except immediately after, and as a result of coughing.
The morbid sounds are usually confined to the upper lobe of the lung, and are often confined to one side of the chest; they are very persistent, and even, if met with on both sides at first, are apt to subside partially or wholly on one side, whilst they continue, or even increase on the other.	*The morbid sounds* usually predominate in the lower lobes, and exist equally in both sides of the chest; they are of temporary duration, and subside gradually and equally on both sides of the chest.

For the sake of those who are commencing the investigation of diseases of the chest, it may be desirable to point out certain combinations of symptoms which are always suggestive of tubercular disease of the lungs, and should therefore lead to more than usually careful inquiry. Foremost amongst them is obstinate cough, commencing gradually, without any running of the eyes or nose, or pain in the chest—a cough which at first was dry or attended by very scanty expectoration, and has persisted in spite of ordinary remedies. If a person who gives such an account as this of his cough admits that one or more of his relatives have been delicate, asthmatic, or consumptive; that he has experienced dull, aching pain about the collar bones and upper part of the chest; that the cough and expectoration have gradually increased, and are most troublesome at night or towards morning; that on one or more occasions the sputa have been mixed or streaked or specked with blood; that he has lost strength and flesh, and has become short-breathed on going up stairs—the general evidence of early phthisis will be complete, though it will require confirmation or refutation by means of a physical examination of the chest. So, too, if emaciation has occurred without obvious cause, and more especially if it is accompanied by any of the general symptoms just mentioned, or by night sweats, or by hoarseness which is not connected with a syphilitic taint, and does not speedily subside under treatment, the gravest suspicion of phthisis may be entertained, and a careful examination of the chest is rendered necessary. So, again, if obstinate diarrhœa occurs, and in spite of ordinary remedies and careful dieting, persists with scarcely any intermission for several months, and more especially if it is not accompanied by much coating of the tongue, or by sickness or other signs of biliary derangement, but on the contrary is attended by a red, glazed, or beefsteaky tongue, and by more or less abdominal tenderness on deep pressure, the case is calculated to excite a suspicion of consumption—a suspicion which would amount almost to a certainty if the diarrhœa were attended by even the slightest cough, or by loss of flesh or shortness of breath. In like manner, chronic peritonitis occurring in an adult is almost always connected with the presence of tubercle, and renders imperative a close investigation of the state of the chest. Under any of these circumstances, a slight flattening of the chest walls or deficiency of expansion at one or both apices, a decrease in the vital capacity of the chest, the occurrence of more or less dulness on percussion in the clavicular or infra-clavicular regions, with a harshness or feebleness of the respiratory

murmur, an irregularity or jerkiness in its rhythm, the existence of prolonged expiration, or of increased vocal resonance, especially at the left apex, and the occurrence of occasional dry clickings, in addition to any other râles or rhonchi which may be present,—would one and all warrant a more serious interpretation than that which they would justify if unaccompanied by the aforementioned general symptoms. On the other hand, râles, of whatever character, if persistent at and confined to the apices of the lungs, and still more so if persistent at one apex only, and accompanied by hæmoptysis, emaciation, night sweats, and other of the general symptoms of consumption, may be regarded as almost certainly indicative of tubercular disease; but a cautious practitioner will never express a confident opinion in a doubtful case from the result of a single examination. Time is an important auxiliary in the diagnosis of obscure cases. Râles, which are due to pneumonia, bronchitis, or the presence of blood effused in an attack of hæmoptysis, and dulness, which is referable to pleuritic effusion, or to œdema of the lung, or to pneumonic consolidation, will gradually disappear, and cease to complicate and mask the signs which are referable to more permanent and organic changes in the lungs. On the other hand, signs which are due to the presence of tubercle in course of development will gradually pass from one phase to another,and thus establish the true character of the disease.

The prognosis of pulmonary consumption is necessarily unfavourable. In acute phthisis the downward course of the patient is steadily and rapidly progressive; but in certain instances of chronic phthisis the disease is of very long duration; whilst in others, and certainly the majority of cases, it runs a less protracted course, but is nevertheless marked by distinct remissions. The question therefore arises whether it is possible to prognosticate which course it will pursue, by reference to the character of the symptoms which accompany it? In some instances, undoubtedly, it is possible to do so, but in others there are not sufficient data for our guidance. It has been already stated that tubercles when once deposited may remain for years in *statu quo*, or may speedily undergo softening or disintegration, and that these peculiarities are referable in great measure to the intensity of the phthisis or constitutional disorder under the influence of which the tubercle is deposited. The same conditions regulate the entire course of the disease, and modify the effects of treatment. Thus, in any given case, if the intensity of the constitutional disturbance can be ascertained, it will not be difficult to predicate with some degree of certainty

whether the course of the pulmonary disease will be slow or rapid—whether temporary remission or suspension of the symptoms may be expected, or whether they will run on from bad to worse, unchecked by remedies. Now it happens that phthisis, or that condition of constitution which results in the deposition of tubercle, is marked by tolerably characteristic symptoms, and that when the tendency to tubercular deposition and tubercular disintegration is strongly developed, these symptoms are most prominent. Amongst them may be mentioned extreme rapidity and softness of the pulse, the early occurrence of emaciation, and of hectic with profuse perspiration, sleeplessness, entire loss of appetite, derangement of the bowels, and diarrhœa. When these symptoms are observed coincidently with great prostration, hurried breathing, and dyspnœa on the slightest exertion, the probability is, not only that there is extensive tubercular deposit in the lungs, but that the constitutional derangement is deep-seated and excessive, and that the retrograde metamorphosis of tissue will continue unchecked, and will bring the patient rapidly to his grave. On the other hand, when the pulse remains slow or is but little accelerated, when there is little quickening of the respiration, little tendency to hectic, little perspiration, and no diarrhœa, when the appetite remains good and emaciation takes place slowly—the system obviously is not overwhelmed by the disease, but possesses considerable power of resistance; the progress of the malady, therefore, will be slow; and not improbably, if appropriate treament be adopted, remissions may occur, or its course may be arrested. In cases such as these, therefore, if our patient is placed under favourable hygienic circumstances, the prognosis would be comparatively favourable, and partial recovery may be anticipated.

It has been attempted to prognosticate the issue of the complaint solely by reference to the condition of the tubercular deposit; and for this purpose it has been assumed that signs of pulmonary softening and excavation, are also signs that the disorder is tending rapidly to a fatal issue. But nothing can be more at variance with the truth. A single mass of tubercle may soften or form a vomica, and nevertheless the patient may rally, and may remain for years in tolerable health, whilst on the other hand, tubercular infiltration of the lungs may take place, and though little or no breaking down of the lung tissue may occur, the patient may sink rapidly, worn out by the hectic induced by the vitiated condition of his blood, or asphyxiated by the extent of the interference

with his respiration. In short, the amount of the tubercular deposit, viewed in connection with the general symptoms already described, forms a far more trustworthy guide to a prognosis than any evidence, however satisfactory, of the tubercle being in an advanced stage of degeneration and softening.

Of late years it has been asserted not only that remissions of the disease may occur, but that the malady may be permanently arrested. In former days such an event was regarded as simply impossible, the very fact of recovery being admitted as conclusive evidence that the diagnosis was at fault, and that the disease under which the patient was suffering could not have been phthisis. Even at the present day, there are not wanting those who maintain, and act as if they believed, that consumption is incurable. But there are no valid grounds for such an opinion. Assuming, for the sake of argument, that the deposit of tubercle has not been so extensive as to interfere materially with the functions of respiration, there is no reason why the constitutional derangement on which the formation of tubercle depends should not be checked, and the tubercle already formed got rid of. Experience fully justifies the statement that consumption does not ordinarily pursue a steadily progressive course from first to last, but like gout, consists of a series of attacks, which often occur at an interval of years, and each one of which results in a deposit of tubercle which forms an additional impediment to the due performance of the function of the lungs. Fortunately, the extent of the respiratory apparatus is greater than is needed for our ordinary requirements, and therefore the presence of a certain amount of tubercle is not incompatible with the due oxygenation of the blood and with the maintenance of tolerable health. Consequently, if the amount of tubercle already deposited is not excessive, and the general health can be improved, and the tubercular cachexia got rid of during the intervals between the attacks, the tendency to the further deposit of tubercle will be prevented, and virtually the patient will be cured. The tubercle already deposited may be absorbed, or it may remain imbedded in the lung, or it may soften and be got rid of by expectoration; but in either case, if no further deposit takes place, the functions of life will not be materially interfered with, and the patient may attain to longevity. It is only after several attacks have occurred, and successive crops of tubercle have been deposited, or after a single crop has occurred sufficiently extensive to interfere materially with the function

of respiration, and to impede the processes necessary to the oxygenation of the blood and the maintenance of health, that it is out of the power of medical treatment to restore the tone of the system and arrest the further development of mischief. Clinical observation and pathological research have left little to be desired in the way of proof, not only that recovery from phthisis is possible, but it does take place in a certain proportion of cases. They have shown that consumption is not a local disease, but consists essentially of constitutional derangement productive of impaired nutrition and deterioration of the blood; and as other constitutional disorders, which equally with phthisis result in an alteration of the quality of the blood, admit of removal, there is no *à priori* reason for doubting that under favourable circumstances the faulty condition of the system in tuberculosis may also be rectified. In numberless instances they have discovered encysted tubercles in the lungs of persons who for many years before their death had not been subject to cough, and had not exhibited any symptoms of consumption, and thus they have proved, that even after the tubercular cachexia has been fully developed, and has resulted in the deposition of tubercle, complete and permanent recovery may take place. They have shown that tubercle is an exudation from the blood; that it is of low vitality, and is not prone to become organized; and inasmuch as all other exudations from the liquor sanguinis, so long as they are unorganized, admit of being absorbed, we are constrained to conclude that the same applies to tubercle. Further, they have shown, that when tubercle is deposited in the external glands, it may remain for years in a quiescent state without producing serious inconvenience; that more commonly it gradually undergoes absorption; and that even when it softens and is eliminated by suppuration no further deposit of tubercle may take place, but the patient may live to an advanced age without the slightest recurrence of the disease. Thus they force us to believe that the same holds good in respect to the lungs, and that, whether tubercular deposit in these organs remains quiescent or undergo absorption, or be got rid of by suppuration and expectoration, the patient may recover and attain to longevity, provided only that his general health can be improved, and the condition of his blood altered, so that no fresh deposit of tubercle shall occur.

In corroboration of this view they have discovered empty vomicæ in the act of contracting and healing, and in many instances have

found them entirely healed and cicatrized, causing deep indentations on the surface of the lung. But they have carried us a point even beyond this; they have not left us to infer, simply as a matter of analogy, that absorption of tubercle may take place in the lung; for, as if to leave no doubt that where the reparative process is at work tubercular matter may be absorbed, they have proved that tubercle is seldom found in large quantity in the vicinity of pulmonary cicatrices—nay, rather, is often altogether absent, or has left as its only representatives a few pieces of cretaceous or calcareous matter, the whole of the animal constituents of the tubercle having disappeared; whilst in other instances a few small, isolated masses of tubercle—some of which are in their original crude condition, some in a state of partial transformation into cretaceous matter, some completely cretafied or calcified, and some partially or wholly encysted—alone remain to establish the nature of the disease which led to the excavation and cicatrization of the pulmonary tissue.

But there is evidence which goes even beyond this. Cases have been met with in which the general symptoms and the results of repeated examinations of the chest have mutually borne witness to the existence of phthisis, and in which, nevertheless, the symptoms have gradually subsided, the physical signs have disappeared, and the patient has regained health, and for years has remained free from chest affection. Several such cases have come under my own observation, and in three the subsequent career of the patient, and facts elicited by *post-mortem* investigation, have furnished me with ocular and demonstrative proof that complete recovery may take place under favourable hygienic conditions—recovery so complete at all events as to enable the patient to attain to an advanced age, and to live for the remainder of his life in tolerably robust health and free from all fresh accession of pulmonary symptoms.

Without entering largely into the detail of cases of arrested phthisis, many of which I have had the opportunity of noticing, I will briefly give an outline of the three cases just alluded to in which *post-mortem* examination confirmed, and thus gave additional interest to the result of careful observation during life.* The first was that of a young lady aged nineteen, whose parents were healthy and whose family were not consumptive.

* For full details of many cases of arrested phthisis see Dr. Hughes Bennett, on 'Tuberculosis;' also, one or two cases briefly referred to by Dr. Walshe.

She fell into ill health in consequence of the distress and worry incident to a disappointment respecting marriage, and speedily became short-breathed, had spitting of blood, and evinced unmistakeable symptoms of consumption. The late Dr. Chambers and Dr. C. J. B. Williams were consulted, and both pronounced her phthisical, and asserted that she had vomicæ at the apices of the lungs. After the lapse of a twelvemonth, she began to rally, the cough in great measure ceased, and the shortness of breath subsided. She lived for nine years after this, and died eventually of dropsy resulting from disease of the heart, the signs of pulmonary excavation existing at either apex up to the last. My friend and colleague, Mr. G. D. Pollock, performed the *post-mortem* examination. In the upper lobe of both lungs were two or three encysted masses of crude yellow tubercle, about the size of a hazel nut; at the right apex was a large firm cicatrix, resulting from an old vomica, and a small cavity about the size of a large hazel nut still open and lined by a dense, smooth, and highly vascular false membrane; at the left apex was a similar sized vomica also lined by a dense and vascular false membrane; the other portions of the lungs were healthy and free from tubercular deposit. From the whole history of the case, there can be little doubt, firstly, that tubercle must originally have existed in far larger quantity than was manifest on the *post-mortem* examination, and probably had been removed by absorption; secondly, that the tubercle which still remained was in a perfectly quiescent state, and was not seriously prejudicial to her health; thirdly, that no deposit of tubercle had taken place for some years before death, and that in fact consumption, or the tubercular cachexia had ceased to exist; fourthly, that for all practical purposes the patient would have recovered thoroughly, and would have got rid of her cough, had it not been for the accidental formation of the dense membrane which lined the two open vomicæ and prevented their collapse and cicatrization.

The second case was that of a gentleman who died at the age of fifty-four. Born of consumptive parents, and himself of slender frame and delicate appearance, he had profuse hæmoptysis at the age of twenty, and was pronounced phthisical by the late Drs. Chambers and Nevinson. For more than twenty years, he had rarely passed a twelvemonth without severe cough and somewhat profuse hæmoptysis, and when I first saw him, twelve years ago, his symptoms both general and physical were those of advanced phthisis. He was thin, short-breathed, and unhealthy in appearance; there was dulness on percussion and flattening under both

clavicles; auscultatory signs of vomicæ existed at either apex, and there was great deficiency of breathing in other parts of the lungs. Thinking it impossible that he could continue at work, I recommended him to take a holiday for a twelvemonth, and to travel in the East in search of health. He determined to do so, but his departure was unavoidably delayed for six months. He left London, however, and constantly shifted his quarters in England; and before he started on his trip his constitution seemed to have undergone an entire change; his complexion had lost its unhealthy hue, he had gained flesh, his cough had greatly diminished, and he stated that he was becoming less short-breathed than formerly. After an excursion of eleven months up the Nile, and through Palestine and the northern parts of Syria, he returned home apparently quite well. His cough had ceased, and he had become tolerably stout; the infra-clavicular regions had lost their flattened appearance, the dulness on percussion had in great measure disappeared, and all stethoscopic signs of pulmonary excavation had ceased. Indeed, the only indications which remained of any abnormal condition of the lungs were, imperfect expansion of the infra-clavicular regions on full inspiration, and weakness or deficiency of breathing in some parts of the upper lobes of the lungs. From the time of his return home until the day of his death, which occurred eleven years afterwards, he never had the slightest hæmoptysis. He died of ascites, resulting from disease of the liver. After death, large cicatrices were found in the apices of both lungs, and calcareous matter existed in the cicatrices; several small masses of cretaceous matter about the size of a pea were distributed through the upper lobes of both lungs, and there were also a few small, isolated, and encysted masses of crude tubercle. In this case, the family history, the spitting of blood, and other general symptoms observed during life, and repeated physical examinations of the chest, left no doubt as to the nature of the case; and the fact that he had been consumptive was further proved by inspection of the body after death; yet for nine years at least before his death, he had not exhibited any symptoms of consumption; and inspection after death confirmed the opinion which had been previously entertained, that he no longer suffered from the tubercular cachexia, and that the tubercle which had been deposited or formed in the lungs had been in great measure removed by absorption and expectoration.

The last case was that of Mary Liddon, aged fifty, who died in St. George's Hospital in the month of June, 1858, with whose earlier

history I happen to be acquainted. Her mother died young, of consumption, and she herself had frequently spat blood, and had been "asthmatic" for many years; otherwise she had enjoyed fair average health, and had been enabled to keep at work. When she first applied to me for relief in the year 1849 she presented most of the general symptoms of phthisis. She had lost flesh, though she was not much emaciated; she was cachectic in appearance, had adunc nails, was short-breathed, and was expectorating a large quantity of thin homogeneous pus. At that time there was considerable deficiency of expansion during inspiration in the right infra-clavicular region, with marked flattening and dulness on percussion in that situation; and the stethoscope informed me that in the same situation there existed gurgling and intensely increased vocal resonance; at the left apex there was some flattening of the parietes, with dulness on percussion, deficiency of the respiratory sounds, and prolonged expiration. A tonic plan of treatment was pursued, and after a time the general symptoms of phthisis began to subside, the physical signs of pulmonary excavation to disappear, and she rallied in a surprising manner. Before the expiration of eighteen months her general aspect was that of tolerably good health; the "asthma" had almost wholly ceased, and the most careful examination of the chest could only detect slight deficiency of breathing at the right apex, and feebleness of respiration, with prolongation of the expiratory sound at the left. I then lost sight of her for some years, and in the year 1856 she again applied to me. At that time she was suffering from dyspepsia, but she assured me that she "had quite got rid of her asthma." Her chest remained in precisely the same state as at the date of my last examination, except that the respiration at the left apex had become almost normal. On the 2nd of June, 1858, she was admitted into St. George's Hospital under my care, suffering from diffuse cellular inflammation of the leg and hand, of which she died in a few days.* After death a large old cicatrix was found at the right apex, and there was a small mass of old crude tubercle, of putty like consistence, immediately beneath it. In other respects the lungs were sound. There was no trace of tubercle, except in the one spot immediately adjacent to the old cicatrix, and there was no emphysema. In this case the family history of the patient, no less than the general symptoms and physical signs of disease, concurred in pointing to tubercular mischief as the cause of all her trouble, and the *post-mortem* examination confirmed

* See 'Hospital Post-mortem and Case-book' for 1858.

the opinion which had been formed, by revealing the existence of an old cicatrix at the spot where, during life, the stethoscopic signs of a vomica had been observed. After careful consideration of the history of the case and of the fact that no trace of tubercle was discovered in the left lung, and only one small mass in the right, the legitimate inference appears to be, that as her general health improved, her constitution underwent an entire change, the tendency to phthisis was overcome, and the tubercle already deposited was almost wholly absorbed. Her lungs were restored very nearly to their normal condition, and practically speaking, her consumption was cured.

A similar case, but in which our knowledge of the patient's history extended back only about three years, occurred under the care of my colleague, Dr. Pitman, in the year 1861; and another, in which the tubercular disease had made rapid progress towards recovery, was admitted in the year 1860 under the care of Dr. Page, the full particulars of which will be found at p. 35 of the 'Hospital Post-mortem and Case-book' for that year. Our museum records state that the man, who was twenty-two years of age, died of renal dropsy, and was in good bodily condition at the time of his death. His phthisical symptoms, including hæmoptysis, dated back nearly two years; after death "the upper lobe of the left lung was found *occupied in its greater part by a mass of cicatrices*," and there was also "a small, smooth, lined cavity." Several masses also of crude tubercle, not softened, existed at the apex of the right lung, indicating clearly the nature of the mischief which had resulted in the cicatrization and healing of the lung.

Thus, then, it must be admitted that recovery from pulmonary consumption is possible, nay more, that recovery does take place in a considerable proportion of cases. It may therefore be desirable to inquire, whether there are any general or physical signs indicative of the retrocession of the disease, and of the probability of permanent recovery. The peculiarities of the general symptoms, which denote a slow progression of the disease, have been already described, and it only remains to be stated, that when the disease is arrested there is not only no further evidence of declining health, but the patient regains a healthy appearance, recovers flesh and strength, and gradually loses his cough. Such an occurrence, however, would only indicate a temporary lull in the progress of the disease; it need not necessarily denote its permanent arrest. And if reference be made to a physical examination of the chest, it will be found that little assistance towards a correct pro-

gnosis is to be derived from that quarter. The physical indications of active tubercular mischief may subside, and the only remaining morbid signs may be slight flattening of the chest walls, with dulness on percussion, increased vocal resonance, and feebleness or harshness of the respiration—signs indicative simply of pulmonary contraction and consolidation. But the same phenomena are observed during a temporary suspension of the disease, and are in no degree characteristic of permanent recovery. In short, it must be admitted that there are no marks diagnostic of a permanent arrest of the disease—a fact which can only be judged of after the event by observing not only that the various physical signs which indicate pulmonary excavation and consolidation have disappeared, but that the general symptoms of active disease have subsided, and that there is progressive improvement in the health.

But, although there are no general or physical signs indicative of permanent recovery, the symptoms above referred to as denoting the probability of a slow advance of the disease, and as characterising its temporary arrest, will go far towards justifying a hope of permanent recovery, provided the malady be not hereditary. For when a tendency to the tubercular cachexia is not thoroughly ingrained in a man's constitution, but has only been developed as a result of temporarily depressing causes, it is reasonable to hope that if these causes can be got rid of, and a fresh impetus given to the function of nutrition, the further progress of the disease may be stayed. Theory points to this conclusion, and practice largely confirms it. Therefore, as the evidence of gradually improving health may be regarded as indicative of an effort of nature—and to some extent an effectual effort—to rectify the derangement which has occurred, it is fair to infer that if that improvement can be sustained, the tendency to the deposit of tubercle will be permanently overcome. In most hereditary cases the disposition to tuberculosis is too strong to admit of removal, and therefore, except under peculiar circumstances, our utmost efforts will not avail in those cases to do more than postpone the fatal event. But experience is decisive as to the possibility of recovery in non-hereditary cases; and when the patient has once begun to rally, there is no reason why the improvement should not continue, provided only that he be placed under conditions favourable to his general health. It is indispensable, however, to his permanent recovery, that he be placed under these conditions, and continue subject to them for a lengthened period—not for a few weeks or months only, but for several years. If an opposite

course is pursued, and at the expiration of a few months, when the symptoms have in some measure subsided, the patient ceases to consider himself an invalid, and subjects himself to the various depressing influences which originally upset his health and induced his illness, the phthisical tendency will probably recur with increased intensity, a fresh deposit of tubercle will take place, and as his lungs are already partly occupied by the deposit, he will "fall into a galloping consumption." It is the neglect of rational means to sustain the general health for a sufficiently long time after improvement has commenced, or in other words until the system has entirely shaken off its tendency to tbe disease, which leads to the fearful mortality from phthisis.

The average duration of the disease is a point on which it is difficult to adduce any trustworthy evidence, inasmuch as it is well nigh impossible, in most cases, to ascertain when tubercular deposition commenced. It is not improbable that tubercles may be slowly deposited in the lungs, and may long exist there in a quiescent state without giving rise to any indication of their presence. They are constantly discovered after death in the bodies of persons who, during life, were not only not suspected of being consumptive, but had not been subject to cough, and had not exhibited any symptoms of phthisis; whilst, on the other hand, persons who have had hæmoptysis, and from time to time have been subject to cough, and have manifested well-marked symptoms of phthisis, and who eventually fall victims to that disease, are often observed to pass months, or even years, during a temporary suspension of the malady, in entire freedom from cough or other notable symptom of pectoral disease. This being the case, it will be admitted that, even when the prior history of the patient can be ascertained, great uncertainty must attach to any opinion concerning the date at which tubercular deposition commenced, and that when, as in the case of inmates of hospitals, it is impossible to obtain any reliable account of the patient's ailments for some years previously, any opinion as to the duration of the malady must be a matter of the merest conjecture. All that can be safely stated is, that the disease may kill in three weeks or a month from the date of its accession, or may run a chronic course of twenty or thirty years. The average duration of the complaint is ordinarily, I believe, very much understated, from the fact that the inferences respecting its duration are drawn from the statements of hospital patients, who pay little heed to the earlier, and, as they imagine, unimportant symptoms of disease, and pertinaciously date their malady from the

occasion on which they first experienced pain in the chest, or were frightened by the occurrence of hæmoptysis, or found themselves unequal to their daily work. The statistics collected at the Hospital for Consumption, and others recorded by Louis and Bayle, accord generally with those which I have collected at St. George's Hospital;* but they differ completely from those obtained by the careful investigation of cases in private practice, where every little symptom is noted, and a much more trustworthy account can be obtained of the patient's health for years prior to the final outburst of pulmonary disease. In the former cases, by far the larger number of patients appear to die in from three to eighteen months; whereas, in the latter, the usual duration of the complaint appears to vary from about eighteen months to seven years, a discrepancy which cannot be explained by the different social position of the sufferers. Doubtless, the advantages enjoyed by the upper ranks of society in respect to medical treatment, change of air, and proper regimen, may go far towards accounting for a certain amount of the variations observed; but the difference is too great to admit of being wholly explained away in this manner, and can only be accounted for by the greater jealousy with which the upper and more

* The subjoined table exhibits the duration of pulmonary consumption, so far as the fact could be ascertained, in 772 cases:

Duration of disease.	Physicians' cases at Hospital for Consumption.	M. Louis' cases.	M. Bayle's cases.	Cases investigated by Dr. Fuller, at St. George's Hospital.	Cases in private practice, investigated by Dr. Fuller.
Less than 1 month . . .		1	1	—	—
In one month		3	1	—	—
From 1 to 3 months . .	1	11	14	1	—
3 — 6 „ . .	22	52	44	9	1
6 — 12 „ . .	66	62	64	21	2
12 — 18 „ . .	34	24	30	25	5
18 — 24 „ . .	22	17	18	27	5
2 — 3 years . . .	29	—	—	8	4
3 — 4 „ . . .	13	—	—	9	6
4 — 5 „ . . .	—	—	—	3	8
5 — 6 „ . . .	—	—	—	1	4
6 — 7 „ . . .	—	—	—	—	5
7 — 8 „ . . .	—	—	—	3	1
8 — 20 „ . . .	—	—	10	5	2
4 years and upwards	14	—	—	—	—
2 to 8 years . . .	—	23	18	—	—
Doubtful	14	—	—	6	3
Total . . .	215	193	200	118	46

educated classes are wont to watch their health, and note the earlier inroads of disease.

It is often asserted, that pregnancy retards the progress of phthisis, and many instances have come within my own knowledge in which marriage has been most improperly recommended under this idea. My experience amongst pregnant phthisical women is too limited to enable me to speak with confidence as to the effect which may be produced in this way; but theoretical considerations, and the issue of the few instances which I have met with, induce me to believe that pregnancy operates like other agencies which influence the general health. When the tubercular disease is not far advanced, and the excitement incident to marriage and pregnancy serves to rouse the system, and improve the patient's general health, it may also operate indirectly on the phthisical disease, and either partially check its progress, or lead to its suspension. But, even in this case, the debility and exhaustion consequent on child-bearing will often occasion a fresh outbreak of the disease soon after parturition, so that the patient will go just as speedily to her grave as if pregnancy had never existed. And in cases in which a patient becomes pregnant when far advanced in consumption, I am satisfied that the disease is not only not suspended, but is urged more rapidly along its downward course. Some of the general symptoms may for a time become less prominent, so that the patient or her friends may flatter themselves that the disease is stayed; but the stethoscope will reveal the fallacy of their expectations, and the case will tend speedily to a fatal issue. Such at least has been the course of events in the cases which have come under my immediate notice; and the inquiries I have made amongst those of my friends who are specially engaged in the practice of midwifery confirm the view I have been led to entertain. In a moral point of view these marriages of consumptive invalids cannot be deprecated too strongly; and socially, everybody is interested in preventing them; for the offspring of such a marriage can scarcely fail to be weakly and scrofulous, and even if they attain to manhood, will only perpetuate disease and swell the list of those, who unequal to the fatigues and duties of life, sink prematurely to the grave.

There is yet one question which demands consideration, before we enter on the subject of treatment; I mean, as to whether consumption is contagious or infectious? In Italy an impression is very prevalent that the malady is communicable from one person to another; and not only is the consumptive patient avoided, as if he were plague-stricken, but

after his death, his clothes, bedding, and everything he has made use of, is frequently burned or destroyed.* Even in our own country instances are often cited in proof of its contagious character. A wife, hitherto healthy, and apparently free from phthisical taint, nurses her consumptive husband, and falls a victim to the same form of pulmonary disease; a sister tends her gradually-failing phthisical brother, and soon afterwards shows symptoms of decline. The sequence is remarkable; and as it naturally places the two facts before us in the relation of cause and effect, it is apt to excite unnecessary apprehension; for there really is no ground for supposing that they hold that relationship to each other. The sister, probably, inherited the taint in common with her brother and other members of her family; and the wife, if free from an inherited predisposition, was not unlikely to become consumptive, worn out as she must have been by her weary, anxious watching, the sleepless nights, and long exposure to the confined atmosphere, and other depressing influences of the sick room—circumstances beyond all others powerful in fostering or exciting a tendency to phthisis. The marvel rather is, that notwithstanding the strain of long continued anxiety, and the depression inseparable from the protracted nursing of a dearly loved relative, consumption should not be developed in the survivor more frequently than observation has proved it to be. The fact, however, serves to confirm the theoretical impression that a diathesis, or peculiar state of constitution, cannot be transmitted from one person to another by the agency of contagion or infection.

But, though the non-contagious character of phthisis be admitted, it behoves the physician to warn the patient's friends of the dangers incident to long continued attendance on him, especially if the disease be in an advanced stage. It would be the height of imprudence for a healthy person, especially if young, and of a scrofulous diathesis, to sleep in the same bed, or even in the same apartment with a consumptive patient; for, although the malady would not be communicated directly from the one to the other, the close, relaxing atmosphere, and faint, nauseating odour of the sick room, and the painful scenes he would necessarily witness, would be eminently calculated to depress him, and predispose him to disease.

There are three distinct questions which require consideration in

* For a full exposition of the arguments by which the contagiousness of phthisis is advocated, see a lecture by H. Guenneau de Mussy, in 'l'Union Médicale,' No. 138, 1859.

respect to the treatment of pulmonary consumption, viz.,—first, how to prevent the occurrence of the disease in persons who seem predisposed to its invasion; secondly, how to arrest its progress whilst as yet the pulmonary mischief is limited in extent; thirdly, how to alleviate the patient's suffering and smooth his passage to the grave, when it is beyond our power to avert a fatal issue.

The first two questions involve several points on which medical men are frequently consulted, and on which much difference of opinion exists. Modern research has shown that, by invigorating the general powers of the system, and improving the functions of assimilation and nutrition, the tubercular cachexia may be removed, the formation of tubercle prevented, and its further development arrested. The question therefore arises—How is this desirable result to be brought about? Many persons imagine that warmth is the panacea which will effect our object, if the patient be placed under otherwise favourable hygienic circumstances. "The first and most effectual prophylactic," says one of our most esteemed authorities, "is residence in a warm climate." Now, here at once I find myself at issue with the opinions of many excellent and learned physicians, and no less so with popular prejudice. From our cradle to our grave we are taught to regard cold as an unmitigated evil; and as warmth is intimately mixed up in our minds with the notion of comfort, and that again with everything which is conducive to our bodily well-being, the recommendation to seek a warmer and more genial climate is quite in keeping with our inbred feelings, and is regarded as consistent with sound sense and the exercise of common prudence. But, before adopting this view, let us look more closely than is ordinarily done into certain facts of every-day life, and into others which have been elicited by recent statistical inquiry. Nothing is more certain than that many persons are conscious of enjoying better health and greater vigour in winter than in summer; the cold "seems to brace them and do them good," they breathe in it more freely and are equal to more exertion, their digestive organs are more active, and they are enabled to take many articles of diet which disagree with them and render them "bilious" in the warmer and more enervating weather of summer. There are others, and possibly they form a majority, in whom cold produces a precisely opposite effect; it "pinches and prostrates" them, lowers the whole tone of their system, interferes with their digestion, and disturbs the regular course of their other functions. The absurdity of subjecting these two classes of

persons to the same regulations in respect to temperature and climate is too obvious to be insisted on, and yet that practice is almost universal!

To take another class of facts. The result of careful observation has been to show that, although many consumptive patients are better in summer than in winter, yet that others—and amongst them many even of those whose necessities oblige them to go regularly out of doors in search of their daily bread—lose flesh less rapidly, and preserve their strength better in winter than in summer. The experience of physicians at all large hospitals, no less than amongst the upper classes in private practice, proves that to a large proportion of consumptive invalids the warm weather of spring and summer is most fatal; they complain of it as enervating their whole system, they lose whatever remains they have of appetite, feel unequal to the least exertion, and fade away apparently without the power to fight against the general depression which overwhelms them.

And yet again, the results of medical investigation into the geography of phthisis prove that cold is, if anything, preventive of the disease. Of all countries in the world there are none so exempt from the ravages of consumption as those which are included within the isothermal lines of 30° and 40° mean annual temperature. In St. Petersburg and Moscow, with a mean annual temperature of about 38° Fahr.; in Canada and the northern districts of North America; in Iceland and in the Faroe Islands, and in the northern parts of Norway, Sweden, and Lapland, the disease is comparatively rare,* whilst in France, Italy and along the northern shores of the Mediterranean, in Malta, Madeira, and other localities to which consumptive invalids are usually consigned, the ratio of mortality among the natives from phthisis equals, and in many instances exceeds, that which obtains in many parts of our own country.† And these results of careful inquiry and statistical research are corroborated by facts which from time to time are forced on our attention in the course of our professional career. There are few physicians who have not met with instances of

* Dr. Thorstenson, who practised many years in Iceland, Professor Retzius, Drs. Roberts, Schleisner, and other physicians have testified to this fact. See Schleisner, 'Brit. and For. Med.-Chir. Rev.,' April, 1850.

† Andral, Lugol, Burgess, Gourbay, and many other physicians have borne witness to this important fact, and their statements are corroborated by the valuable reports of Colonel Tulloch relative to the influence of those climates on our soldiers.

health restored and vigour regained by removal to a colder climate. Some of the most remarkable recoveries from consumption which have come within my own cognisance have been in the persons of those whose occupations or necessities have driven them to the cold regions of the north. It must be admitted, then, not only that cold is not productive of phthisis, but that in many instances it invigorates the animal economy, and thus proves antagonistic to the accession and subsequent progress of the disease. And if this be so, it follows that in certain instances, at all events, warmth must not only enervate the patient, and thus expose him to an attack of phthisis, but when the disorder is once developed must have a prejudicial effect on its progress.

I do not wish to imply by the above statement, nor to adduce the foregoing facts to prove, that warmth is always or even generally prejudicial to persons of a consumptive tendency. That would be an error precisely similar to the one against which it is my wish to protest. Nothing is more certain than that a warm atmosphere is of all things that by which many persons suffering from the tubercular cachexia are most likely to benefit. Their organization is delicate, their circulation weak, their extremities are often cold, they are pinched and prostrated by a low temperature, and they are very susceptible of damp. To such persons a warm, dry, atmosphere is invigorating in the highest degree, and without its aid medicine and the most carefully regulated diet are of little avail. My wish is rather to point out that, whereas many persons are benefited by warmth, others are equally benefited by cold, and that the opposite opinion, which is commonly and rigorously enforced in practice, is constantly leading to lamentable results. The rapid progress which consumption sometimes makes in patients who have gone abroad for their health, and which is often attributed to their having delayed their journey too long, is referable in most instances to the fact of the individuals in question being persons who are naturally benefited by a cool, bracing atmosphere, and who sink at once when sent to the warm and enervating climate of the south.

It may be asked, what facts are calculated to assist us in determining the sort of climate by which a patient is likely to be benefited? The question is somewhat difficult to answer, inasmuch as a change of climate implies not only a change of temperature, but a change in the humidity and electrical condition of the atmosphere, in the degree of its barometric pressure, in the force and direction of the wind, and probably

in many unknown but potent telluric influences productive of marked effects on the animal economy.* Nevertheless, though much uncertainty must necessarily exist, there are certain points which will serve in most instances to guide us to a correct opinion. The most important of these are—First, the sort of climate and the degree of temperature which formerly suited the patient's constitution, or, in other words, agreed best with him when he was in health; and, secondly, the state of the patient's bronchial mucous membrane at the time when his removal to another climate comes under consideration. It is obvious that if the bronchial mucous membrane is irritable the invalid cannot bear the effect of a very dry and stimulating atmosphere, however warm the locality may be. His symptoms require a soft atmosphere, and its temperature and the precise degree of necessary humidity must be determined by reference to his constitutional peculiarities. Thus, if he formerly enjoyed better health in summer than in winter, and felt in greatest vigour in very warm weather, and in an atmosphere devoid of markedly stimulating or relaxing qualities, the probability is that the climate of Syria, Persia, Rhodes, Egypt, and other parts of Northern Africa would exercise an influence on his system the good effect of which could hardly be over-estimated.

If, again, though usually better in summer than in winter, he was formerly oppressed by excessively dry heat, but enjoyed a warm and humid atmosphere, such as that of South Devon or Cornwall, the probability is that the climate of Torquay, Dawlish, Penzance, or Jersey, of Pau, Rome, the Azores, Teneriffe, Madeira, Santa Cruz, the Mauritius, or Ceylon, according to the degree of temperature required, would be found to suit his general health,and assist in subduing the irritability of the air passages.

And yet again, if he is constitutionally disposed to general languor, and has always felt as much depressed and enervated by heat, as pinched

* On what other supposition is it possible to account for the vast difference existing between the so-called "bracing air" of Brighton and the "relaxing" atmosphere of Hastings? The two places are within a few miles of each other, and both face the south; both are washed by the same waters, shone upon by the same sun, and do not differ more than one or two degrees in average thermometric range. It is useless to appeal to the more sheltered position of Hastings; for the same difference in their effect on the system is felt when the wind is sweeping over the sea from the south or south-west, and when, therefore, Hastings is not more sheltered than Brighton. The fact, I believe, is rather attributable to the difference in the geological formation of the two localities, which plays a far more important part than is commonly supposed in determining the effects of "climate" on the system.

and prostrated by cold, then, notwithstanding the irritability of his bronchial mucous membrane, a medium climate must be sought—a climate such as is to be found in Queenstown and other parts of the coast of Ireland, on the western coast of Scotland, at Buxton, Cheltenham, St. Leonards, Ventnor, and Bournmouth; or, if a somewhat higher range of temperature is necessary, in new Zealand or the Cape of Good Hope.

But a large class of consumptive patients exist in whom there is little or no irritability of the mucous membrane. In these a drier and more bracing air will generally prove of the greatest benefit; but nevertheless, as in the former cases, the selection of a locality in each particular instance must be regulated by the constitutional peculiarities of the invalid. If his circulation is languid, and he has usually felt more vigorous in summer than in winter, the invalid must repair to a warm locality; and in such a case the climate of Mentone, Hyères, Cannes, Malta, Nubia, Algeria, Upper Egypt, the northern districts of Syria, and New South Wales are likely to prove extremely beneficial. In some such cases the air of the Himalayas, the more elevated parts of the Andes, and other hill districts, has been found remarkably serviceable.

If, again, the patient has an active circulation, and has usually enjoyed better health in winter than in summer, feeling braced and invigorated by cold, he will probably derive benefit from a residence at Brighton, Margate, Aldborough, Cromer, Harrogate, or Malvern; or if a cooler and still keener air is required, from the climate of Montreal, or other places in Canada, or of certain dry localities in Russia or other northern countries. Some of the most remarkable recoveries from consumption which have come within my own cognizance, have occurred under the bracing influence of a northern clime.

Thus, then, to revert to the point at which we started in this discussion, respecting warmth and climate, it must be clearly understood that, whether in relation to the prevention or alleviation, or cure of phthisis, the influence of warmth and of change of climate will prove beneficial or otherwise, in proportion as the use made of these agencies is suited to the idiosyncrasy of the patient, and the existing condition of his bronchial mucous membrane. If the consumptive invalid be not surrounded by atmospheric and climatic influences adapted to the requirements of his organism, the skill of the physician and the utmost care and attention of the patient will generally prove unavailing to modify the morbid action going on in the animal economy, or to stay

the development of the tubercular cachexia; whereas, if those influences be favourable to his health, and care be taken to regulate his diet, and sustain the tone of his system, I am satisfied that more may be done for his relief—nay, even for his cure—than is possible in most other constitutional disorders of equal severity.

Inasmuch, then, as it is of the utmost importance to place the patient under favourable hygienic influences, it may be desirable to consider whether change of climate is always needed, and if not, what precautions should be taken to modify the character of the existing atmosphere.

There are certain cases in which change of air is indispensable. Persons of a very "chilly nature," if resident in a cold and bracing locality, must shift their quarters ere their health will improve; so too, must those who require "bracing," whose lot is cast in a warm and humid climate. These and all others who happen to be placed under circumstances unfavourable to their general health, will do wisely to migrate at the earliest opportunity. But their migration need not necessarily be to foreign climes. There are many spots in our own country, nay, even within a few miles of London, which afford to the average of consumptive patients all the change which their organism demands, and it is only the comparatively few who require a warmer climate than this country can offer to whom a residence abroad is really necessary. All persons will do well to select as their residence a spot which has ordinarily agreed well with them, and if their own home comes within this category there is no reason why they should leave it for other quarters. Temporary change of air is useful now and then, by imparting a stimulus which is not to be obtained in any other way, and to persons to whom travelling is a pleasurable excitement, and who, so long as they remain at home, are unable to shake off the cares and anxieties of business, a residence abroad may be almost indispensable. But, the majority of consumptive invalids will fare better in their own country than even in the most favoured regions abroad, especially now that the railways afford facilities for frequent change, and the Crystal Palace at Sydenham offers a never-ceasing opportunity of combining mental occupation and rational enjoyment with daily exercise in a warm and agreeable atmosphere. Mere absence from home, with its comforts and associations, is to many invalids a constant source of annoyance and regret, whilst the fatigue of a long journey and the privations incident thereto, go far towards neutralising any good effect which might otherwise be produced. Therefore with

this as with other remedies, its efficacy will depend upon the judgment with which it is employed. As a preventive of the disease, or as a corrective of that derangement of the health which marks its earlier inroads, a residence abroad, or foreign travel, with the pleasurable excitement incident thereto, may, for many persons, though not for all, be regarded as one of our most potent and most valuable remedies. But in an advanced stage of phthisis, when the patient is weak, emaciated and exhausted, the recommendation which is often given to go abroad for the winter is cruel and unwarrantable. Under these circumstances no difference of climate can compensate for the loss of home comforts, or make up for the immediate increase of suffering which the journey entails; and an early death in a lodging house abroad, which frequently ensues in the cases alluded to, is a painful commentary on the eagerness with which men vainly seek to prolong their brief existence here on earth, and on the thoughless inconsistency of physicians who recommend the journey.

When it is determined to remain at home, every care should be taken to ensure good ventilation, and to keep the temperature of the house at a point which is congenial to the feelings of the invalid. The objects to be attained are, that the patient shall not be chilled, nor on the other hand enervated by warmth, and that he shall have a free supply of pure air which he can breathe without exciting irritation of the bronchial mucous membrane. The temperature which ordinarily effects these purposes varies in different cases from 60° to 65° Fahr., and perhaps the best means of maintaining this heat and imparting the necessary moisture to the air is by the aid of hot-water pipes or of an Arnott's stove, on the top of which is placed a dish containing water. Yet, whilst counselling attention to the temperature of the house, I would guard against having it supposed that a close atmosphere is beneficial, or that confinement to the house should be recommended. Nothing can be more prejudicial to the consumptive invalid than the want of active exercise in the open air. During the summer he should spend almost the entire day out of doors, walking, riding on horseback, driving, travelling, yachting, or simply sitting in the open air; and even in winter, provided the weather be fine, he should avail himself of the warmer hours in the middle of the day to take whatever out-door exercise his strength will permit. If the cold air irritates the bronchial membrane, and excites cough, a Jeffries' respirator should be worn, with a view to heat the inspired air, and obviate

this source of trouble. But ordinarily this will not be needed; and if care be taken to guard against chills by means of appropriate clothing, it will be found that although during sedentary occupations in the house he may require the temperature to be artificially raised, the consumptive invalid will not often suffer from breathing the cool fresh air of heaven, during active exercise out of doors. It is essential, however, that his clothing be regulated to meet his necessities under the various circumstances in which he may be placed. Flannel should always be worn next to the skin; but in a warm room he should be clad less heavily than in the cold corridors or passages, and in these, again, less warmly than in the still colder air out of doors. The principle being to insulate the body and preserve its natural temperature, the amount of extra clothing should be strictly proportioned to the temperature of the atmosphere to which for the time he is exposed; in no other way can he hope to avoid chill, or to maintain his vital power. Excessive clothing in a warm room would enervate and weaken him, but deficient clothing in a cold room or out of doors would prove equally or even more prejudicial, by depressing his vital energy. It is a fact which cannot be too often impressed on the invalid, that warm clothing does not render the body more obnoxious to cold, but rather imparts power to resist it; and it is a fundamental rule which cannot safely be neglected, that a person whose circulation is feeble should be thoroughly warm before he leaves the house, and should return immediately he finds himself unequal, even with the aid of exercise, to bear up against the existing cold without losing his warmth and becoming chilled.

This brings us to the consideration of other circumstances besides warmth and change of climate which exert an influence for good or for evil on a patient suffering from the tubercular cachexia. Of these none are more important than diet and exercise.

It has been already stated that out-door exercise is an important agent in the treatment of consumption; and so efficacious has it proved in certain cases, that Dr. Rush and other consumptive invalids, who have made trial of it in their own persons, have vaunted it as a specific and certain cure. But it needs little observation to prove that it does not deserve such unbounded praise. It is preventive of phthisis, and curative in its action, precisely in the ratio in which it is proportioned to the strength of the invalid. Its primary object is to impart tone and vigour to the system; and inasmuch as the vital force of the consumptive invalid is extremely low, it behoves us to be careful lest, by

ordering an amount of exercise in excess of our patient's strength, we exhaust the little power that remains to him, and so impair the activity of the digestive functions on which the renewal of his strength depends. Active but moderate exercise excites the action of the heart and arteries, increases the appetite, and stimulates the process of assimilation; whereas, exercise carried beyond the patient's strength exhausts his vital force, impairs the appetite, diminishes the power of assimilation, and so increases the general debility. Therefore, in the treatment of phthisis, it is essential that the physician should define the amount and sort of exercise which should be taken. Walking exercise is too fatiguing for many consumptive invalids, and when this is the case, horse exercise may be employed with advantage. Driving in a carriage, especially an open carriage, may be had recourse to when walking and riding prove too fatiguing; or walking may be alternated with riding, driving, or yachting. Violent exercise, even of temporary duration, may exhaust a patient's vital power to a degree from which he may never recover, and, therefore, if he be weak, it is to be deprecated most earnestly. But moderate and frequent exercise is sure to prove beneficial, and may be carried so far as even to produce fatigue, provided the fatigue does not occasion persistent exhaustion, but is soon relieved by rest.

In addition to general bodily exercise, certain partial exercises have been found of service in the treatment of the earlier stages of consumption. Gymnastic movements of all sorts; the club exercise, fencing, the daily use of the dumb-bells; the chest expander, and the skipping rope, and even the practice of reading aloud, and singing; are each and all important aids to treatment. They increase the action of the respiratory apparatus, stimulate the circulation through the lungs, promote the development of animal heat, and gradually increase the capacity of the chest. But in the employment of these partial exercises, the same rule must be observed as to the non-production of exhaustion as was laid down in regard to general bodily exercise. And whereas these partial exercises excite the pulmonary circulation to a far greater degree than general bodily exercise, it is further necessary, whilst employing them, to watch for any indication of disturbance in the flow of blood through the lungs. Pain, or a weight or a tightness in the chest, or even excessive shortness of breath, should be a signal, either to suspend the exertion, or to modify its character; for they indicate a want of freedom in the return of blood through the pulmonary veins; and if,

in disregard of their warning, the exercise be continued, the congested vessels may relieve themselves by hæmoptysis.

Diet is a subject of equal importance to those we have hitherto discussed. Fresh air, exercise, change of climate, and judicious clothing, are all subsidiary to the one object of re-establishing healthy nutrition; and no one in the present day would venture to deny, that in the treatment of phthisis, our principal efforts should be directed to the assimilating and digestive organs, rather than to the organs of respiration. It happens that dyspepsia is one of the commonest of the early symptoms of consumption, and that, during its existence, the appetite is very capricious, and fatty matters are generally disliked, and therefore avoided. Lehmann, however, has suggested that a certain amount of fat is essential to the due performance of the digestive process, and that oily matter plays an important part in influencing the healthy metamorphosis of the albuminous constituents of the blood, and promoting cell development. The fact itself is intelligible enough; and no one who is acquainted with the valuable researches of Dr. Hughes Bennett, and has watched the effect of cod-liver oil, and other oleaginous and fatty matters on the system, can doubt its importance in a therapeutical point of view. If, then, it be admitted that the assimilation of animal and fatty matters is important, our main efforts must be directed on the one hand to the avoidance of those articles of diet which are likely to prove indigestible and to create or increase undue acidity of the stomach, whereby the assimilation of fatty matter is rendered difficult, and, on the other, to supply food containing albuminous and oleaginous principles in a form in which the weakly and disordered stomach is able to assimilate and duly prepare them for the nutrition of the body. The most carefully regulated hygienic and therapeutical treatment will fail if the diet is insufficient in quantity or faulty in quality. The prevalent error has always been a too exclusive reliance on one particular sort of diet. Some physicians have extolled the virtues of fish, and have confined their patients to that sort of food; others have maintained the efficacy of farinaceous and vegetable food alone; others of milk, eggs, or more solid animal food; whilst not a few, especially in these later days, have insisted on the necessity of enforcing a diet in which fat or oil holds a conspicuous place. But it only needs the exercise of a little common sense to establish the fallacy of each of these doctrines. As one stomach, even in a state of health, differs greatly from another in its power of dealing with certain

articles of diet, so, as might have been expected, in a state of disease these differences become even more apparent; and, without refining over much, it may be stated, that the diet suitable for consumptive patients must necessarily vary according to their individual peculiarities. "One man's meat is another man's poison," quite irrespective of the stage at which the disease has arrived, or of the symptoms by which it is accompanied. And not only so, the food which is suitable at one period of the disorder, and which the stomach proves itself equal to digest, may be quite unsuitable at another time, when that organ has acquired increased tone, or else has lost whatever little vigour it previously possessed. Thus, as it is necessary to consult our patient's feelings and constitutional peculiarities, in respect to warmth and climate, so also in respect to diet, individual idiosyncrasies must be carefully sought out and studied, if we wish to prescribe a diet which is likely to be properly digested. Remembering always the fundamental principle, that our aim must be to support our patient, and with that view to induce him to avoid indigestible matters, and to take food containing albuminous and oleaginous principles in the form best adapted to the capacity of his stomach, we must endeavour to compass our end, not by adhering to any particular kind of food, however advantageous it may appear to be theoretically, but by prescribing diet as nourishing and supporting as the stomach will bear, by carefully watching its effect, and varying it according to the result of our observation, and, at the same time, by administering such remedial agents as experience has proved likely to assist the weakened and disordered organ in the due performance of its duties.

If the disease be not accompanied by symptoms of bronchitis or of active local congestion, and if the stomach be not materially deranged, a nutritious diet should be prescribed, comprising caviare, marrow, fat bacon, and fat meat, twice or three times daily. If there be much exhaustion and depression, and the pulse be slow and the skin cool, wine and malt liquor may be added with advantage, and the patient should be ordered to take some rum and milk, or half a pint of cream with a teaspoonful of brandy on first waking in the morning, and some bread and milk, mock turtle soup, or other light, nourishing food, with a little brandy, if necessary, on retiring to rest at night. Indeed, stimulants in full doses are indispensable in many cases.

If the liver be disordered or the stomach loathes ordinary butcher's meat, venison or game may be tried, or a light fish diet may for a time

be substituted for the meat. Oysters are often digested very readily; and cod and other light white fish, including their livers, are especially to be recommended, and so is turtle soup.

If, again, the case be characterised by heat of skin, quickness of pulse, oppression and constriction across the chest, and a hard, irritable cough, the diet must be limited for a time to articles such as milk or cream, or farinaceous food. Linseed tea, the decoction of marshmallow, gum arabic, the mucilaginous material of the Fucus crispus, or Carrigheen moss, and the bitter demulcent mucilage of the Iceland moss are serviceable, not only in allaying the cough, but as bland, unirritating articles of food; and in many of these instances milk in which mutton suet has been boiled, proves extremely useful. Beverages such as whey and buttermilk, and soda, lime, or Carrara water and milk, are also to be recommended, as being at once grateful to the patient and useful adjuncts to the diet. Chocolate may be advantageously substituted for tea, and oranges, lemons, and grapes may be allowed if the stomach is not irritable and the bowels are not relaxed. It need only be added, that the amount, no less than the quality of the food, must always be proportioned to the temperament and idiosyncracy of the patient, the temperature of the air in which he resides, and the amount of exercise he is taking.

This brings me to the consideration of cod-liver oil, which may be regarded partly as an article of food, and partly as a medicinal agent. To whichever of its constituents its efficacy may be due, there cannot be a doubt that it is beyond all others the remedy on which most reliance can be placed. In the north of Europe it has long been a popular and favourite remedy, but in this country we are indebted to Dr. Hughes Bennett for our earliest knowledge of its remarkable powers in the treatment of tuberculous disorders.* Chemistry has hitherto failed to explain its action, but clinical experience leaves not the slightest doubt as to its beneficial influence. Of course its virtues are most strikingly displayed in the earlier stages of the disorder, and in cases where there is not a strong inherited predisposition to tubercular disease, but this fact amounts to nothing more than that where there is little mischief to be overcome, the oil has less difficulty in subduing it than it has when the mischief is deeper seated or more advanced.

* I cannot allow this opportunity to pass without recording my sense of the large debt of gratitude due to Dr. Hughes Bennett by the profession of this country for his persevering advocacy of cod-liver oil.

And speaking generally, it may be truly stated that the oil proves serviceable at all ages, and at all stages of the disorder. The general and local symptoms improve under its administration with a rapidity which is sometimes quite surprising; and in favourable cases the weight of the body increases, the cough and expectoration decrease, the appetite improves, and the night sweats and other unfavourable symptoms gradually disappear.

Three facts, which have been elicited by careful observation, appear to afford some clue to its mode of action. The first is, that unless the patients gain weight whilst using it, the oil seldom or never proves remedial; the second, that weight and flesh may be gained during its administration although the pulmonary disease be steadily progressing; the third, that when it does act remedially, the weight gained is far beyond what would result from the oil as a mere aliment. Hence, it would appear that its operation consists in aiding digestion by supplying some principle which is essential to the assimilation of food and the establishment of healthy nutrition. This view has been uniformly maintained by Dr. Hughes Bennett, who has stated, further, that its efficacy depends on its supplying the fatty matter which is essential to the formation of healthy chyle, and which the digestive organs have become unequal to manufacture or separate from the food. My own impression is, that its influence is not so simple as is here suggested by Dr. Bennett; for, if his views were correct, it ought to act beneficially whenever the stomach is capable of retaining and digesting it, whereas cases not unfrequently occur in which, though the oil does not disagree, it fails to exercise any influence on the disease, which, nevertheless, may yield to other remedies. Further, the improvement which results from cod-liver oil is not found to follow the administration of neat's-foot and other oils which equally supply an oily matter; and as in administering cod-liver oil we are conveying into the system iodine, bromine, phosphorus, and other matters which are known to exercise a powerful influence on the animal economy, I am inclined to believe that the peculiar efficacy of cod-liver oil depends partly on the supply of oil which it affords, but partly also on its containing elements which are wanting in most cases of consumption, and which in such cases it satisfactorily supplies; and that when it fails to operate remedially, it does so in some instances, because it is not tolerated by the stomach, and in others, because it does not contain, and therefore does not supply the elements which in those particular instances are required to promote

healthy assimilation. Be this as it may, its extraordinary virtues cannot be doubted, and it ought to be administered in all cases of consumption in which it does not derange the stomach. It rarely purges or otherwise disturbs the bowels, and it does not induce congestion of the lungs, or fatty enlargement of the liver or kidneys; on the contrary, it improves nutrition generally throughout the body. Not only does it produce increase of weight, but the patients whilst taking it gain strength and colour. As a mere aliment it is extremely useful, and as a remedial agent in appropriate cases its value is inestimable.

It has been urged by Dr. Walshe,* that the oil operates less beneficially in proportion as age advances, an assertion which is quite opposed to my own experience. Had he stated that it less frequently agrees with persons advanced in years than with young persons, and therefore less commonly produces satisfactory results, I should have coincided entirely in his views; but some of the most remarkable instances of rapid improvement under its use, which have occurred in my own practice, have been in the persons of patients far beyond the middle period of life. Not only has flesh been gained in the cases referred to, but the general symptoms and physical signs have improved to a corresponding degree.

Further, it has been stated, that "intrathoracic inflammation and hæmoptysis" are contra-indications to its use. Experience at the bedside, no less than theoretical considerations, lead me to demur to this statement. In pulmonary consumption, the thoracic inflammations are attributable solely to the unhealthy condition of the blood and the local irritation of the tubercular deposit, and the hæmoptysis, in like manner is due to congestion, induced by the same agencies. If then the oil effects what its advocates profess, viz., an alteration or improvement in the condition of the blood, and a consequent improvement in the process of nutrition, it is difficult to see why its use should be abandoned at the very moment when its aid is most urgently required. Moreover, experience does not afford any warrant for suspending its use under the circumstances alluded to. My constant practice has been to continue its exhibition in combination with the remedies best calculated to relieve the inflammation or the hæmorrhage, and I have never seen the slightest inconvenience produced thereby, in cases in which it otherwise agreed.

Sometimes, however, it thoroughly disagrees with the stomach, and

* Loc. cit., p. 532.

even if it does not produce actual vomiting, it gives rise to nausea and acid eructations, together with feverish derangement of the system. In these instances it must either be omitted altogether, or administered in smaller doses, and at all events its use must be discontinued until the stomach is brought into better order, and has acquired the power to digest and assimilate it. The question of treatment in cases such as these resolves itself into that of imparting tone to the stomach, and regulating the action of the liver and other excretory organs. There is generally undue acidity of the stomach, with deficiency or irregularity in the action of the liver and bowels; and the method to be adopted in overcoming the difficulty must depend upon the degree of biliary derangement. If the stomach appears to be the organ principally at fault, the administration of the mineral acids in combination with vegetable bitters and taraxacum, will often serve to give it increased tone, and thus rectify the error; whilst if the acids are not well borne, the same result may be produced by the exhibition of an occasional alterative at night, and of the light vegetable bitters, with liq. potassæ, or the carbonates of the alkalies, or the nitrate of bismuth, during the day. But if, on the other hand, the bowels are disordered, and the motions knotty, clay coloured, or of a dark green, or almost a black colour, no good will be effected without the careful administration for some days of mercurials, with ipecacuanha, taraxacum, ox gall, podophyllin, and other remedies which in some way influence the secretion of bile and the action of the bowels. Not unfrequently, after careful attention to the digestive organs, and equal care in respect to hygienic treatment, the stomach will acquire the power of digesting the oil, and the animal economy will then be improved by the administration of the very remedy which had previously upset it. In all such cases, however, it is advisable to recommence its administration in small doses. Under ordinary circumstances, half an ounce of the oil may be given twice, three times, or even four times daily, and the dose may be cautiously increased to an ounce, or even in some instances to an ounce and a half or two ounces. But, whenever an intolerance of the oil has been once manifested, it is prudent on recommencing its administration to give it in doses not exceeding a teaspoonful twice a day; and, if that quantity be digested, the dose may be gradually increased, and repeated more frequently. It should be taken shortly before or after a meal, in order that it may be presented to the stomach at a time when digestion is in full activity, and it may be taken floating on a glass of wine, or on a dose of tonic medicine con-

taining the mineral acids or the alkalies. The kind of oil employed, whether the pale, light brown, or dark brown oil, is of little importance in a therapeutical point of view; but the paler kinds are purer and more palatable, and are sometimes retained when the coarser brown oil is rejected by the stomach.

Sometimes, however, do what we may, the stomach is unable to retain the oil, or to digest or assimilate any fatty food. Under these circumstances an attempt should be made to introduce the oil into the system through some other channel, and this may be effected either by means of the skin or the rectum. The practice of inunction is not an innovation of modern times, nor is it confined to cases of disease. Among the Romans it was employed as an hygienic luxury; and it is in constant use in the present day amongst the natives of Western Africa, who suffer little from the ravages of phthisis. Even in our own country, accidental inunction takes place to a limited extent in certain classes of the community. The young persons who are employed in wool factories where large quantities of oil are employed,* tallow chandlers, butchers, and others, are necessarily in contact with fatty matter throughout the day, and it has been observed not only that they are usually well nourished, but that they are less liable than others to suffer from consumption. But more than this, experience has established the fact that the endemic method of introducing the oil is often very serviceable in phthisis. Cases have been published by Dr. Simpson of Edinburgh, and other observers which are quite sufficient to prove its efficacy, and in my own practice several instances have occurred in which its operation has been signally beneficial. One gentleman whom I saw in consultation with the late Dr. R. Bright, and subsequently with Dr. C. J. Williams, gained a stone and a half in weight in less than three months by the persevering useof it, and the physical signs of pulmonary disease subsided greatly at the same time. In most instances however, its virtues are not displayed so strikingly as when it is administered by the mouth; and the smell of the oil when applied externally, is so extremely disagreeable, that many patients cannot be persuaded to use it. Under these circumstances, lard or the inodorous vegetable oils may be substituted for it, and undoubtedly will serve to check waste, and support the strength; but the patient's weight will seldom increase materially under their use, and I am doubtful whether, in any instance which has come under my notice, the progress of the local disease has been arrested.

* See 'Edin. Monthly Journal,' April, 1853.

Another method of introducing the oil is by the rectum, in the form of injection. Some physicians have reported favourably of its action when exhibited in this manner; but in the few cases in which I have seen it tried, it has not answered my expectations, and the strong objection which is commonly entertained to the constant use of enemata must render its administration in this way an expedient to be resorted to only in exceptional cases. When the stomach will not tolerate the oil, and the fatigue incident to its inunction, or the dislike of the patient to its employment in that manner forms a bar to its administration, then enemata containing the oil may be tried; but its exhibition in any other way than by the mouth must be regarded as a mere expedient to serve the purpose of supporting the patient temporarily, whilst the stomach—the proper receptacle of food—is being prepared for its reception, and fitted to digest it.

One word of caution may be added respecting the period of the disease at which the administration of the oil should be commenced, and the length of time for which its exhibition should be continued. It has been already stated that its effects are displayed most strikingly when it is administered early in the disease; and it need only be added, that it cannot be given too early. Pathological research has long since proved that pulmonary consumption, in an incipient stage, is a disease which admits of cure in a considerable proportion of cases—of cure so readily effected that it often takes place spontaneously, and so permanent that there is often no recurrence of the disease, even during a life protracted to an advanced age. My own experience has fully corroborated the facts thus gleaned from the field of pathology; and it has also confirmed another fact, drawn from the same field of observation, viz., that such cures are rarely effected if the pulmonary disease has made much progress. Therefore it becomes of vital importance to recognise the earliest inroads of the disease, in order that a proper regimen may be enforced, and the oil and other appropriate remedies administered whilst as yet our patient retains his strength, and the system its reparative power. It is not sufficient to persist in the administration of the oil only until some sensible improvement has taken place; the very fact of improvement should rather be regarded as a stimulus to further perseverance. The deposit of tubercle is so clearly connected with constitutional derangement, and any material alteration in the constitution is notoriously effected so very slowly, requiring not weeks or months but rather years for its completion, that if we would consult our

patient's safety, we must urge him to take the oil for many months after he considers his health re-established. If he refuses to do so, the probability will be that the improvement will be only temporary, and after a short interval of comparative tranquillity, the machine will again get out of order, mischief will recommence in an active form, and our power may not avail to arrest it; whereas, if he can be persuaded to continue taking the oil for a year or two, omitting it only three or four times in the twelvemonth, for three weeks or a month at a time, whilst he is enjoying change of air, and is otherwise under peculiarly favourable hygienic conditions, my experience leads me to believe that, in a considerable proportion of cases, the tendency to the disease will not only be arrested, but the improvement which has occurred will be maintained, even after the oil has been discontinued.

The late Dr. Theophilus Thompson* and other physicians have advocated the use of ozonised cod-liver oil, and have stated, as the result of their experience, that the force and frequency of the circulation are subdued through its agency, and inflammatory action, therefore, averted. The statement at present rests, I think, on insufficient evidence, and my own experience of its action is too limited to enable me to form any opinion on the subject. In the few cases in which I have employed the ozonised oil it has failed to exercise the controlling influence over the pulse attributed to it by Dr. Thompson; but if, on more extended observation, it should prove to exert the power claimed for it by its advocates, it would be a valuable adjunct to the treatment of cases characterised by acceleration of the pulse, and especially so when hæmoptysis is a prominent feature. The ozonisation of the oil does not appear to affect its efficacy as a remedial agent, and it therefore deserves an extended trial.

The remedies which will best assist the curative action of the oil—or, in other words, will have most effect in imparting tone to the digestive organs and improving the general health—must necessarily vary with the condition of the patient. Alkalies and their salts, phosphoric acid, or any of the mineral acids in combination with taraxacum and light bitter infusions; dulcamara in full doses, the iodide and sesquichloride of iron and other chalybeates, cinchona, quinine, strychnine, and sarsaparilla are amongst the internal remedies which I have found most useful; whilst in some instances warm baths, hot air baths, such as the Turkish bath, and cold shower baths, judiciously

* See 'Med.-Chir. Trans,' vol. xvii, p. 349.

employed, are also extremely serviceable. The same may be said of the mineral waters and mineral baths, especially those of Ems, Vichy, Cauteret, Eaux Bonnes, and Spa abroad, and of Bath, Tunbridge Wells, and Harrogate in this country; but like other agents, they prove remedial only when employed in appropriate cases. The Cimicifuga racimosa and the wild cherry bark are remedies which have obtained a high reputation in America, and an endless variety of other substances, which experience has proved to possess no special virtues, have been brought from time to time before the profession. Amongst these, the hypophosphates of lime and soda, naphtha, iodine and its compounds, hydrocyanic acid, digitalis, and other agents have been vaunted as specifics; but experience has amply proved that they are serviceable only in exceptional cases, and when administered according to the requirements of the patient. In no case do they deserve the title which their advocates have arrogated for them, even when they operate beneficially; and experience has shown that, like all other remedies, they are useless in some instances, and worse than useless in others. And the reason is not difficult to fathom. Not only does the condition of the stomach and other organs differ in different cases, and in the same case at different periods of the disease, but the general symptoms present every possible variety, according to the precise nature of the actions going on in the system.

Everybody must have observed how strangely variable the course of consumption ordinarily proves, and how frequent are the accessions of febrile disturbance and of increased cough and pain in the chest—symptoms which are commonly, though erroneously, attributed to "catching cold," and are, in fact, referable to fresh deposits of tubercle, or to the supervention of bronchitis, pneumonia, or pleurisy, excited by the unhealthy and irritating quality of the blood. It needs not much experience to understand that medicines which prove useful when the disease is pursuing its ordinary slow, uncomplicated course, must be of little avail, or positively injurious, when, by the occurrence of one of these active complications, the whole conditions of the case are altered. In short, to revert to my former statement, medicines, to be of the slightest use in this or any other disorder, must be administered according to the exigencies of the case, and in strict relation to existing symptoms.

There are other remedies which must not be passed by without special notice. I refer to bloodletting, baths, and the use of persistent deriva-

tives, such as issues, setons, blisters, and the production of pustular eruptions and discharges by means of tartar emetic, croton oil, or biniodide of mercury ointments. Small and repeated venesections, and the frequent application of a few leeches, have been recommended by authors as calculated to relieve the congestion of the lungs which so constantly accompanies phthisis, and so indirectly to cure the disease. Nothing, however, can be more opposed to sound pathology, and to the results of modern experience. A moderate abstraction of blood may possibly be requisite, if perchance active pleurisy or pneumonia should supervene; but even in that case the symptoms may usually be subdued by appropriate treatment, without loss of blood; and the idea of relieving simple congestion by repeated bloodletting, whether local or general, is simply absurd and mischievous. The congestion is due to the unhealthy and irritating condition of the circulating fluid, and though the abstraction of blood may momentarily empty the vessels and relieve the pain, the impoverishment of the vital fluid resulting therefrom must necessarily tend to the recurrence of congestion, and to an increase of the congestive tendency. Efficient constitutional treatment, aided by dry cupping and counter-irritation—not bloodletting—is the appropriate remedy for this form of congestion.

The question respecting the use of derivatives is not so easily disposed of. From the earliest periods of medical history they have been used and lauded as extremely beneficial; and the curious facts deduced from the result of clinical observation respecting the effects of apparently insignificant discharges from ulcerated surfaces, point to the necessity of caution in arriving at an opinion adverse to their employment. My own opinion is favourable to their use in aid of constitutional treatment in the early stage of the disease, provided they are so regulated as not to exhaust the patient's strength by the profuseness of the discharge induced, and not to depress him, morally and physically, by causing constant pain and annoyance, and preventing sleep. Their applicability, therefore, must depend on the mental and constitutional peculiarities of the patient. The means best adapted for the purpose of inducing discharge are the use of issues, either between the scapulæ or in the arm, or over the margin of the false ribs, or the application to the chest of one of the many ointments which induce pustular eruptions. None, in my experience, answers better than an ointment for which a formula is given below.* In the later stages of the disease derivatives of this kind

* Hydrargyri Bichloridi, gr. viii; Iodinii, ʒss; Spiritus Rectificati, ʒiss; Adipis, ℥j, ft. ung.

almost invariably depress the patient, and therefore should not be employed.

The use of baths, whether warm, tepid, or cold, vapour or hot air, is too much neglected in the treatment of consumption. This has arisen, I believe, partly from the difficulty which, until the last few years, has been experienced in obtaining baths in this country, and partly from the mischief which has frequently resulted from their improper or injudicious employment. It is not to be doubted that agents like baths must exert a powerful influence on the animal economy. Theory suggests the fact, and experience confirms it. But their agency will be for good or for evil, according as they are judiciously or injudiciously made use of. The cold shower bath will stimulate and brace one patient, but will chill and depress another; the warm bath will soothe and tranquillise one person, but enervate and render another miserable; the vapour or the hot-air bath will refresh the man whose skin is dry and inactive, and whose nervous system is oppressed or rendered irritable by the presence in the blood of materials which ought to have been thrown off by perspiration, whilst it would exhaust and reduce to an unwarrantable degree the patient whose skin is already relaxed and acting immoderately. Thus, in respect to baths as to other agents, it may be affirmed that they become remedies only when they are adapted to the requirements of the case. But it may be added, that when so employed they are most valuable adjuncts to other treatment, and ought never to be neglected. I have seen more striking benefit result from the cold shower bath and the dripping sheet, followed by active friction, than from any other remedy, except cod-liver oil. Sponging the body with cold or tepid vinegar and water is sometimes very serviceable.

There are certain symptoms in connection with phthisis which require consideration apart from the general treatment of the disease. Sometimes, for instance, dyspepsia is such a prominent feature, and so urgent in its character, as to demand special and almost exclusive treatment. Constant pain and weight at the stomach, increased after food, and oftentimes accompanied by a distressing sense of nausea, induce the patient to reject food almost entirely. These symptoms must be met by treatment specially addressed to their relief; and although it is not my purpose at present to discuss at any length the treatment of dyspepsia, it may be stated that, among the remedies which I have found most serviceable in this particular form of the complaint are bismuth, liquor potassæ, the alkaline carbonates, hydrocyanic

acid, the mineral acids, gallic acid, strychnia, and opium, preceded, if necessary, by an alterative dose of calomel or blue pill, and aided by counter-irritation at the pit of the stomach.

Cough is another symptom which requires special treatment—not when it is loose or slight in amount, but whenever it is hard, frequent, and harassing, and produces exhaustion during the day and sleeplessness at night. Amongst the remedies most serviceable in allaying the irritation are opium and its salts, especially morphia and codeine, belladonna, conium, digitalis, and hydrocyanic acid, and their action may be aided by ipecacuanha or squills, or by tolu, marsh-mallow, liquorice, Iceland moss, linseed tea, and other demulcent materials, whether in the form of a beverage, cough-drop, or lozenge.

If the cough be hard and accompanied by symptoms of febrile excitement, with pain and constriction in the chest, and a stethoscopic examination reveals commencing bronchitis or pneumonia, salines with ipecacuanha or tartar emetic will be more appropriate remedies, and their action will be assisted by occasional dry cupping between the shoulders, by stimulating embrocations to the chest, or by mustard poultices, turpentine fomentations, or blisters, according to the urgency of the case. If the febrile exacerbation be referable to pleurisy, a few leeches to the seat of pain may possibly be needed, or the administration of calomel and opium may even be required; but as the treatment of these complications has been fully discussed in the sections devoted to the subject, it is needless to recapitulate it here. If the pulse be very quick, and the cough, though extremely irritable, is not accompanied by pain or constriction across the chest, or by the physical signs of acute pneumonia or pleurisy, I know of no remedies of equal value with aconite, digitalis, and the veratrum viride, and they may be given to the exclusion of tartar emetic, or in combination with it. In some instances, especially in the early periods of the disease, the effect of these remedies in giving relief, after ordinary sedatives and expectorants have failed, is very remarkable. When there is entire absence of febrile excitement and the cough is unaccompanied by pain or constriction across the chest, and the distress which attends it is referable to difficulty in expectorating, the decoction of senega, aided by the balsam of Peru, the compound tincture of benzoin, tolu, squills, sulphuric or nitric æther, and other stimulating expectorants, appears to modify the character of the secretion, and thus, by facilitating its rejection from the bronchi, to relieve the strain of coughing. In some of

these cases the action of emetics is extremely serviceable, not only by leading to the immediate ejection of the mucus and muco-purulent matter which is loading the air tubes and oppressing the patient, but by modifying the character of the bronchial secretion, inducing a free action of the skin, unloading the whole of the internal organs, and thus promoting increased activity of digestion.

If the cough is of a spasmodic character and the breathing is accompanied by much wheezing, without any evidence of local inflammation, a cough-drop containing stramonium, lobelia, hydrocyanic acid, and æther, answers better than a mere narcotic and demulcent mixture, and in some such cases the inhalation of a few drops of æther or chloroform from off a handkerchief will subdue symptoms which have resisted the same remedies taken in a liquid form. Sometimes, when the mucous membrane of the air passages is very sensitive, and is easily irritated in a dry atmosphere, the mere fact of moistening the air with steam will prove useful in alleviating the cough, whilst in others the inhalation of dilute medicated vapours will be found more serviceable. For this purpose aqueous vapour, charged with the essential properties of conium, hyoscyamus, stramonium, or other substances, may be diffused through the atmosphere of the apartment, and breathed through one of the many forms of inhalers, whilst in other cases, in which there is little tendency to inflammation, more stimulating substances, such as vinegar, iodine, camphor, benzoin, pyroxylic spirit (naphtha), creasote, and common tar, may be beneficially inhaled in the same manner. Not unfrequently, however, the inhalation of air highly charged with moisture—whether medicated or not is of little importance—is oppressive and disagreeable to the patient, and under such circumstances relief may often be obtained by fumigating the apartments in which he resides by gently heating benzoin, myrrh, and various balsams and gum resins, and creasote, tar, camphor, iodine, and other substances, and it is stated—though I have no personal experience of the matter—by the inhalation of oxygen and compressed air.*

Sometimes the irritability of the cough is due to relaxation of the throat and elongation of the uvula, and in these cases the practice of snipping off a small portion of the uvula and of daily swabbing the throat with a strong solution of nitrate of silver, or some other

* See 'Emploi médical de l'Air comprimé,' par M. Pravaz; also 'The Compressed Air Bath: a Therapeutic Agent in various Affections of the Respiratory Organs and other Diseases,' by R. B. Grindrod, M.D.

stimulant or astringent material, will be found of the greatest benefit. A concentrated solution of tannin in glycerine has proved in my hands extremely serviceable, and so has powdered alum, applied by means of a large camel-hair brush, which has been wetted with glycerine and then covered with the alum. In certain instances, however, in which the pharynx is seen to be dry and devoid of secretion, I have found greater relief produced by the application of a mixture of two parts of the tinctura pyrethri and one part of glycerine than by any more stimulating or astringent solution. The effect of the pyrethrum in inducing increased secretion from the mucous surface is quite remarkable.

When the larynx is ulcerated the application of a solution of nitrate of silver by means of a probang, is said to be very serviceable, but experience has led me to doubt the possibility of introducing the sponge, except in rare instances in which the epiglottis and the larynx are strangely devoid of sensibility. By means of a syringe, however, the solution may be injected into the larynx without difficulty, and I have known it afford some temporary relief. The application of the solution to the pharynx has appeared to be almost equally efficacious, and as its effect is less distressing than its introduction into the larynx is apt to prove, I prefer the use of the brush or the probang to the syringe. In some instances I have known considerable benefit produced by blowing a few grains of calomel down the pharynx whilst the patient is taking a deep inspiration,* and in others relief may be obtained by local blistering or by counter-irritation induced by means of turpentine, or of a lotion composed of biniodide of mercury,† applied on a rag and covered with oiled silk, followed by the application of a bread and water or linseed poultice. The inhalations already mentioned are also in many instances attended with relief. Gargles are almost useless. In certain instances, in which suffocation is imminent, tracheotomy may be had recourse to with the effect of prolonging life.‡

* I have devised an instrument for introducing calomel or any other powder into the larynx, which answers its purpose admirably. It consists of a curved canula, the extremity of which is finely perforated like a pepper-pot, and unscrews to admit the introduction of the powder. This is passed back into the throat and held above the opening into the larynx. When it is in this position the patient is directed to inspire, and the calomel or other powder is then ejected by a current of air set in motion by means of an India-rubber ball attached to the other extremity of the canula. The instrument may be obtained from Whicker and Blaise, in St. James's Street.

† The following is a formula I often employ:—Hydrargyri Bichloridi, gr. vj; Tr. Iodinii co., ℥j; Glycerini, ℥ss; M. ft. lotio.

‡ See case in point referred to at p. 394 of this treatise.

Pain in the chest is to be met by counter-irritation. Some persons recommend that blisters should be applied, and kept open by dressing the blistered surface with the ung. sabinæ or some other stimulating application; but I much prefer the practice of letting the blister dry, and then repeating the blister if necessary. The counter-irritant effect produced is greater and occasions less distress to the patient. When the pain is not so acute as to require a blister for its relief, stimulating embrocations, or mustard cataplasms, or turpentine fomentations, may be used, or the compound tincture of iodine or the acetum cantharidis may be applied to the chest daily by means of a brush, a fresh portion of the chest being painted with the solution each day. This gives little inconvenience to the invalid, and oftentimes suffices to relieve the dull, aching pain in the infra-clavicular regions with which consumptive patients are often tormented.

Perspiration, which, when profuse, proves extremely weakening and distressing to the patient, must, of course, be combated, like all the other symptoms, by judicious constitutional treatment. But there are certain remedies and plans of treatment which appear to exercise a special control over this disagreeable symptom, and therefore should be tried when the sweating is profuse. Amongst internal remedies may be mentioned gallic acid in full doses, the mineral acids, bark, the muriated tincture of iron and opium; whilst, as external applications, the cold shower bath or the dripping sheet and sponging the body with vinegar and water often prove serviceable. In no instance ought the administration of food, together with some alcoholic stimulant on going to bed, to be neglected.

Nausea and vomiting, if attended with acidity of the stomach and not dependent on derangement of the liver, may be subdued by lime water, or by the use of effervescing draughts with hydrocyanic acid and a slight excess of soda. But when the tongue is clean and the stomach irritable without any apparent biliary derangement, I have found greater benefit from the use of strychnia or nux vomica, and sometimes from chloroform, creasote, or bismuth. Brandy and soda-water is often serviceable in these cases, and mustard cataplasms or blisters to the epigastrium are almost invariably productive of comfort, even if they do not arrest the symptoms.

Diarrhœa is another symptom the treatment of which requires the exercise of considerable judgment. If the tongue be furred and the alvine secretions pale or otherwise unhealthy, and if, moreover, there

be not any evidence of inflammatory mischief in the abdomen, a dose of calomel and opium, followed by a warm rhubarb draught or half an ounce of castor oil, will probably relieve the symptoms; whereas if the tongue be nearly clean and very red, giving evidence of long-standing intestinal irritation, the probability is that ulceration of the bowels is going on, and that calomel and rhubarb would only aggravate the mischief. In such a case chalk mixture is not of much service, and sulphate of copper and opium, acetate of lead and opium, gallic acid, ipecacuanha in full doses, the nitrate of bismuth, kino, rhatany, tormentella, and hæmotoxylum, are the more appropriate remedies, and they may be aided by starch and laudanum enemata. If the diarrhœa is attended by pain in the abdomen, poppy fomentations, poultices sprinkled with laudanum, or sinapisms, should be employed in aid of the internal remedies.

Fistula in ano is another symptom which must not be lightly dealt with. As long as the discharge is insignificant in amount and the patient's mind is not seriously disturbed by its continuance, so long it is advisable to confine our efforts to the treatment of the constitutional malady, and not to disturb the fistula. Any attempt at curing it by operation under these circumstances would probably be followed by an immediate increase of the cough and other pectoral symptoms, and, therefore, would be highly injudicious. But I do not hold with those who maintain that a fistula in ano occurring in the course of phthisis ought never to be interfered with. In some instances the discharge is profuse, and constitutes an important source of waste; and the patient is so distressed and alarmed at its continuance, that no treatment can be of avail until his nervous apprehensions are overcome. He can neither eat nor sleep for thinking of it, and his whole system is depressed in consequence. In such cases I have known the greatest benefit result from an operation, combined with the formation of an issue in the arm, the use of a proper diet, and the administration of cod-liver oil, quinine, and other appropriate remedies. Not only has the fistula healed, but the general health has improved, the patient has gained flesh, and the physical signs of pulmonary disease have materially decreased.

Chronic peritonitis, which sometimes occurs in connection with the deposit of tubercle in the peritoneum, is little under the control of medicine. Calomel, which is the remedy most serviceable in ordinary peritonitis, is of little avail, and more generally proves mischievous in

this form of disease; and perhaps opium, iodide of potassium, and cod-liver oil are the internal remedies which are not only most likely to afford relief to the local irritation, but also best calculated to subdue the constitutional irritation. Externally, applications of a remedial nature can be made, which, if they are unequal to arrest the disease, are yet efficacious in mitigating its symptoms and affording comfort to the patient. Amongst these may be mentioned bran poultices, poppy or turpentine fomentations, mustard poultices or blisters, or, better still, a lotion containing laudanum, belladonna, iodine, and glycerine, kept constantly applied to the abdomen on a piece of lint covered with flannel.

Delirium, except in connection with tubercular deposition in the brain or its membranes, is of rare occurrence in phthisis, and does not require any special treatment; but when meningitis occurs it must be treated on general principles by calomel and active purgation, aided by the application of cold to the head and leeches to the temples, the extent to which the treatment is carried being modified by the tubercular character of the disease and the constitutional power of the patient.

A few words may be added respecting the treatment of acute phthisis. Theoretically, there is no reason why the symptoms should not be combated on general principles, and practically, I believe the adoption of those principles will afford the patient the best chance of obtaining relief. This form of disease is characterised by so much asthenia that the abstraction of blood cannot be right, however great the pulmonary congestion may appear to be, and dry cuppings and counter-irritation are the expedients which offer most chances of benefit. The action of these external remedies should be aided by the internal administration of salines, digitalis, aconite, and sedatives. If the depression be not very great, some antimonial wine may be cautiously added; and if the attack be complicated by pleurisy, it may be necessary to give calomel and opium. In most instances, however, the use of fomentations and judicious counter-irritation will supersede the necessity for more active treatment. The immersion of the feet and legs in hot water for the space of an hour is useful in inducing revulsive action and promoting perspiration if the skin is hot and dry. When the more acute symptoms have subsided, and the state of the tongue indicates an improved condition of the digestive organs, cod-liver oil should be prescribed, and the other remedies given which are found to prove serviceable in the chronic forms of the malady.

CHAPTER V.

INTRA-THORACIC TUMOURS.

In the sections devoted to percussion and auscultation frequent reference was made to the signs produced by intra-thoracic tumours, and the subject was partially mooted again when tuberculous enlargement of the bronchial glands was under discussion. It may be desirable, however, to point out more distinctly what can and what cannot be done towards their diagnosis and treatment.

It should be premised that tumours in the chest are either aneurismal or produced by morbid growths, usually of a malignant or scrofulous character. Malignant tumours are sometimes attached to the parietes of the chest, but more commonly, like aneurism, they are developed in the mediastina, and ordinarily take their origin in the glandular structures. The nature of the growth has little influence on the symptoms produced, for the phenomena are not peculiar to any special form of disease, but are referable to the interference with the respiration and circulation produced by the pressure of the tumour on the surrounding parts. Thus the physical signs and general symptoms vary not only with the seat of origin of the tumour, but with its size, the direction in which it enlarges and the nature of the organs on which it consequently exerts pressure.

The physical signs are of two kinds, viz., those which indicate the presence of consolidation and those which bespeak pressure on the adjacent structures. When the tumour is attached to the parietes or occupies the anterior portion of the chest, there will be intense dulness on percussion in the region of the tumour and strongly marked parietal resistance; weakness, or absence, or some modification of the respiratory sounds; and, according as the tumour is large or small, and of an inelastic or of a vibratile nature, there will be increase or absence of vocal resonance and vocal fremitus, and increased transmission or otherwise of the sounds of the heart. But the signs of pressure will be even more important. The intercostal spaces may bulge under the influence of the pressure from within, and even the bony structures, such as the ribs, the sternum, and the clavicles, may gradually wear away, until perforation takes place, and the tumour presents itself externally. Indeed,

circumscribed pulsation is often felt, and with it a thrill transmitted from the heart or large vessels. The heart may be pushed out of its place, or the aorta may be compressed, occasioning systolic murmur; or the innominate and subclavian arteries may be pressed upon, and innominate and subclavian systolic murmurs thus produced, and the corresponding carotid and radial pulses more or less weakened; or the superior vena cava may be compressed or surrounded by the tumour, in which case the venous circulation is interfered with, so that distension of the jugular, subclavian, axillary, and other veins occurs, tortuous and swollen veins show themselves superficially on the chest and abdomen, œdema commences in the face, neck and arm, and upper part of the body, and ultimately the patient becomes drowsy from congestion of the brain and the stagnation of the blood in the venous sinuses of the dura mater; or the pulmonary artery may be subjected to pressure, which may induce a systolic pulmonary murmur, dyspnœa consequent on a deficiency in the supply of blood to the lungs, and intense congestion in the whole venous system; or the pulmonary veins may be more especially subjected to pressure, whereby dyspnœa, hæmoptysis, œdema of the lungs, and hydrothorax, will be produced; or the inferior cava may be pressed upon, and congestion of all the abdominal organs produced, which results in distension of the veins of the lower extremities, and ultimately leads to ascites and œdema of the legs and thighs; or the bronchi may be pressed upon and irritated, so that increased mucous secretion is induced, and râles, rhonchi, and prolonged expiration result; yet further, the bronchi may even be perforated by the pressure, and hæmoptysis, and violent, spasmodic, ringing cough produced; or the lung itself may be seriously encroached upon and deprived of air by the long continuance of pressure, until respiration ceases to be heard over the part, and percussion elicits intense dulness; or the tumour may give rise to excessive local irritation, and pleurisy, bronchitis, pneumonia, or even gangrene may be set up. When the tumour occupies the central and posterior portions of the chest, the dulness on percussion is seldom so marked as in the former instances, and may not be perceptible on gentle percussion, though it is brought out by a forcible stroke.

The auscultatory signs are very variable, being modified by the size and physical nature of the tumour, and by its precise relation to the surrounding organs and the walls of the chest. Sometimes the breathing is weak or almost absent, either locally or over an entire side, and

vocal resonance and vocal fremitus are also absent; but not unfrequently, when the trachea or the larger bronchi are pressed upon, the percussion note is of a tubular or amphoric character; the breathing, instead of being weak or absent, may be intensely loud and tubular, the expiratory sound prolonged, the vocal fremitus and vocal resonance increased, and the cardiac sounds transmitted far beyond their usual limit. If the phrenic and the vagi nerves or the pulmonary plexus are pressed upon by the tumour, disturbance occurs in the respiration; whilst, if the recurrent nerve is implicated, aphonia or some other modification of the voice is induced. If, as often happens, the œsophagus is pressed upon, dysphagia is the inevitable result, and if it is perforated hæmatemesis may occur; if the thoracic duct were compressed, emaciation would proceed with extreme rapidity; if the trachea is subjected to pressure, stridulous breathing and weakness of voice are induced, the cough, which is paroxysmal, is either very weak or else loud, shrill, and clanging, and the expectoration is often streaked with blood. The trachea, the bronchi, the pulmonary artery, the œsophagus, and other structures, are liable to be perforated by the tumour.

The general symptoms of intra-thoracic tumour possess little of a distinctive character. A tumour may long exist in the chest without giving rise to any notable symptoms, and the patient may not be aware of its presence until its development has led to pressure on the surrounding structures. Then occur pain, which is variable in amount, and is sometimes altogether absent, but which, when constant, as it often is in the spine, at the junction of the neck and back, is a symptom of grave significance; dyspnœa, frequently influenced by posture; wheezing and stridor, more or less distinctly marked, and peculiarly so with the expiration; cough of a paroxysmal character, peculiarly weak or else loud, ringing, and of a metallic quality; expectoration, which for a long time is simply catarrhal, or else consists of clear, gelatiniform mucus, which may subsequently become blood-tinged or more or less largely mixed with blood; palpitation; frequent attacks of giddiness and fainting; excessive restlessness during the day and want of sleep and slight wandering at night. Oftentimes the patient is able to obtain repose in one position only—the posture which happens, by force of gravity, to relieve the suffering organ from the pressure of the tumour. Thus, he sometimes sits erect in bed; or remains day and night in an arm-chair, afraid to go to bed; or, sitting

erect, he may lean towards one side or the other; or he may lie down in bed comfortably on one side, though the least attempt to move on to the other will induce violent spasmodic cough and a sense of impending suffocation; or, again, he may lean forward, with his elbows resting on his knees and his head supported by his hands. In addition to these symptoms, there may be inequality or entire absence of the pulse at one or both wrists, dysphagia, hæmatemesis, aphonia, distension of the superficial veins, and œdema of the trunk or limbs, according as the tumour presses on one organ or another, as was pointed out when the physical signs were under discussion.

There are four circumstances which will sometimes enable us to arrive approximately at a decision as to whether a tumour in the chest is aneurismal, malignant, or scrofulous. The first is the existence of aneurism or malignant, or scrofulous disease elsewhere in the body. The second is that when the tumour is aneurismal it is seldom accompanied by local inflammation, and that a murmur may be sometimes heard in a distant part of the chest, although none exists in the region of the heart; or a loud murmur may exist in both situations, but may be lost or almost inaudible at intermediate points. The third is that the symptoms induced by the pressure of an aneurismal tumour are apt to be extremely variable. A diseased mass, whether malignant, scrofulous, or fibrous, having broad and firm adhesions in the chest—arising, that is, from a broad, extended surface, as such masses do almost invariably—would not be influenced by change of posture sufficiently to effect any material diminution in the amount of pressure, whether on the spine, the œsophagus, the bronchi, or other parts; whereas an aneurismal tumour, arising, as it often does, from a narrow base, and attached only to a vessel which admits of considerable motion, may have its position altered considerably by gravity alone, so that the suffering organ may be relieved by change of posture, and the symptoms referable to pressure may be mitigated. The fourth is one to which attention was first directed by Dr. George Budd,* viz., that when the tumour is cancerous it is more apt than in the other cases to be accompanied by local symptoms of an inflammatory nature, a circumstance which he explains by stating that malignant growths often involve the root of the lung and implicate or destroy the greater part of the nerves which supply such organ. The result is that the nutrition of the lung is interfered with and inflam-

* 'Med.-Chir. Trans.,' vol. xlii.

mation is set up, just as inflammatory destruction of the eyeball ensues after division of the fifth nerve within the cranium.

It is not difficult to distinguish intra-thoracic tumours from the effect of phthisis, pneumonia, and pleurisy, though, in the absence of a careful physical examination of the chest, mistakes of this kind have been made. Thus intra-thoracic tumours may produce modifications of the percussion sound, the respiratory sounds, and the vocal resonance, analogous to those which accompany phthisis; and may also give rise to râles and rhonchi, which are not distinguishable from those which are met with in that disease. But the probability is that in the former case the abnormal phenomena would be confined to one side of the chest, whereas in phthisis they almost always exist in both; that the course which the disease has run would not tally with that which phthisis ordinarily pursues; and that, in character and intensity, the general symptoms would not correspond with those which would be expected at the particular stage of the tubercular disorder which the physical signs would appear to indicate. The aphonia, which results from pressure on the recurrent nerve, may still further complicate the case, and cause it to simulate laryngeal phthisis; but the voice, though weak, or even whispering, will not be materially altered in tone, whereas, in laryngeal phthisis, it would not only be weak, but thoroughly hoarse, harsh, and altered in character. The only case in which any doubt ought to arise is when a mass of enlarged bronchial glands excites symptoms of pressure coincidently with the symptoms of phthisis, and even then the evidence of intra-thoracic pressure, interpreted through the agency of the tubercular disease in the lungs, ought to indicate the existence and the nature of the intra-thoracic tumour.

With regard to pneumonia, its whole course is so unlike that of an intra-thoracic tumour that there is no possibility of the two diseases being confounded, unless the patient be not seen until all active symptoms have subsided. Even then the absence of the wide-spread tubular breathing and increased vocal fremitus and vocal resonance which characterise pneumonia, and, on the other hand, the presence of intense dulness on percussion and of hæmoptysis and red, gelatiniform expectoration, together with enlargement of the side, and the existence of the signs resulting from the pressure and ulceration which usually accompany intra-thoracic tumours, ought to leave little doubt as to the true nature of the disorder.

The symptoms produced by chronic pleurisy, especially when it is circumscribed, are somewhat more perplexing, but ought to be unravelled without much difficulty. The intensity of the dulness on percussion may not vary greatly in the two cases, neither may the limits of the dulness be altered materially by change of posture, and in both cases the vocal fremitus and vocal resonance may be annihilated; but in chronic pleurisy the enlargement of the side will be uniform, the intercostal spaces will often be convex, fluctuation will be perceptible in them, and there will not be enlargement of the superficial veins of the chest or other signs referable to circumscribed pressure, whereas, in the case of intra-thoracic tumour, the side, if altered in form, will be irregularly enlarged, there will be no intercostal fluctuation, signs of circumscribed pressure will almost certainly present themselves, there will usually be found some spots within the area of dulness, where the percussion sound is not completely gone or where the hollow breathing sound is still to be heard, which could not well be if liquid were the cause of dulness, and there will often be hæmoptysis and red or colourless, gelatiniform, mucous expectoration.

Of course, if intra-thoracic tumour coexists with chronic pleurisy, and the pleuritic effusion be on the same side as the tumour, the diagnosis is more difficult, the more so when the early history of the disease is wanting. But if the signs produced by pressure on the intra-thoracic organs are present, it is certain that some intra-thoracic tumour exists, and the probability will be that the pleurisy is a mere accidental complication, excited by the irritation of the tumour.

The causes and modes of death in these cases of intra-thoracic tumours are various. In most instances the patient lingers on, the sense of oppression and the distress of breathing gradually increasing, until at length the appetite fails, he becomes wretchedly thin and worn out by want of sleep, and, oppressed with anasarca, he sinks thoroughly exhausted. But sometimes he gradually succumbs in consequence of the interference with the respiration induced by pressure on the par vagum or on the phrenic nerve or on the pulmonary plexus, or by obstruction of the pulmonary artery; or he sinks rapidly from hæmorrhage resulting from perforation of the pulmonary artery or one of the larger vessels, or, in the case of an aneurismal tumour, from the giving way of the aneurismal sac; or he is cut off by pneumonia, sometimes followed by gangrene resulting from pressure of the morbid mass; or he dies suddenly from spasm of the glottis, induced by pressure on the recurrent nerve.

The treatment is necessarily of a palliative nature, and when the disease is far advanced our utmost efforts are of little avail, even to give temporary relief; but in the earlier stages much good may be effected by remedies calculated to tranquillise the circulation and regulate the secretions, as also by dry cupping, blisters, poultices, opiate and iodine lotions, and other local applications, which have the effect of subduing pain, relieving local inflammation, and producing absorption of the inflammatory matters which have been effused.

PART III.

ON THE PHYSICAL EXAMINATION OF THE HEART AND GREAT VESSELS.

CHAPTER I.

TOPOGRAPHY OF THE HEART AND GREAT VESSELS.

It has been stated that a clear knowledge of the topography of the chest is a necessary prelude to the study of pulmonary disease, but it is certain that a thorough acquaintance with the topography of the heart and great vessels is even more essential to the investigation of diseases of the circulatory apparatus. I shall endeavour, therefore, to describe the position of the circulatory organs, and their relation to the parietes of the chest.

The heart, covered by its investing membrane, occupies the lower portion of the anterior mediastinum, lying obliquely across the chest, with its base above and its apex below, the direction of its longer axis being from above downwards, forwards, and to the left. Above, the organ is kept in position by the great vessels, and below by the attachment of its pericardial covering to the diaphragm. Anteriorly, its base is on a level with the third sterno-costal articulation, and its apex with the sixth, so that virtually the entire organ extends from the third to the sixth costal cartilage, and transversely from about half an inch to the right of the sternum to within the same distance of the left nipple. Posteriorly, the base lies opposite the sixth and seventh dorsal vertebræ.

To the right of the sternum, between the third and fifth ribs, lie a portion of the right auricle and the upper portion of the right ventricle; behind the sternum lies the greater portion of the right ventricle, a small portion of the left, and the root of the great vessels; to the left

of the sternum lies the left auricle, on a level with the third costal cartilage and the upper part of the third intercostal space, and below it almost the whole of the left ventricle and a small portion of the right ventricle towards the apex. The whole of the anterior surface of the heart is overlapped by the lungs, except a small triangular portion of the right ventricle. The space where the ventricle is uncovered and comes into apposition with the chest walls is bounded on one side by a line drawn from mid-sternum, opposite the fourth costal cartilage, obliquely downwards and outwards across the lower margin of the fourth left sterno-costal articulation to the lower margin of the fifth left cartilage; on another, by a line drawn from the same part of the fifth left cartilage obliquely downwards and inwards across the sixth cartilage, and thence along the lower margin of the sixth cartilage to a line drawn vertically down the middle of the sternum; and on the other, by a line corresponding to the mesian line of the sternum, and extending from the fourth to the sixth rib. The vertical side of this triangle ordinarily measures during tranquil respiration about two inches, and the diagonal about two and a half inches, but the extent of the uncovered area varies at every stage of respiration, according to the degree to which the lungs are inflated. It is also affected by change of posture, being larger in the erect than in a recumbent posture, inasmuch as not only does the heart fall backwards and upwards under cover of the lung when a recumbent posture is assumed, but the upward pressure of the stomach and bowels also tends to push it in the same direction.

All the valves of the heart are seated behind a portion of the chest walls of about an inch in diameter. The pulmonary valves lie under the left border of the sternum opposite its junction with the third left costal cartilage; the aortic valves a little lower and nearer to the mesian line; the mitral and tricuspid valves as near as possible in the mesian line, on a level with the third intercostal space, the tricuspid being in front and the mitral behind. The second right and the second left sterno-costal articulations are the spots where the sounds of the aorta and the sounds of the pulmonary artery are respectively heard in greatest intensity, and hence the second right cartilage has been named the aortic cartilage and the second left the pulmonary cartilage.

The aorta springs from the base of the left ventricle, and lies about an inch behind the sternum, opposite the third intercostal space. Thence it ascends inclining gradually to the right, until it reaches the second costal cartilage on the right side, whence it curves across the

superior sternal region on a level with the lower border of the first costal cartilage, and, passing backwards and downwards towards the left side of the third dorsal vertebra, rests ultimately upon the left side of the bodies of the fifth and sixth vertebræ. In the first part of its course it rests upon the right pulmonary artery and the left side of the trachea, whilst the superior vena cava lies on its right side and the pulmonary artery on its left; it then passes in front of the trachea, just above its bifurcation, arches over the left bronchus, and, passing still further backwards, lies upon the left lung and the œsophagus, until ultimately it rests against the left side of the bodies of the fifth and sixth dorsal vertebræ, having the œsophagus on its right side and the lung on its left.

The pulmonary artery rises from the right ventricle, just behind the left edge of the sternum, opposite the third sterno-costal articulation, and ascends inclining slightly backwards until it bifurcates opposite the second costal cartilage.

The arteria innominata springs from the transverse portion of the arch of the aorta, whilst it lies behind the right side of the sternum, and, passing upwards and to the right in front of the trachea, it bifurcates behind the right sterno-clavicular articulation.

The left carotid artery arises from the arch of the aorta, behind the sternum, nearly in the mesian line, and, passing in front of the trachea, it tends upwards and outwards to the left sterno-clavicular articulation.

The left subclavian artery arises from the most depending part of the transverse portion of the arch of the aorta, and thence ascends almost perpendicularly to the first rib, where it turns suddenly outwards. Throughout the first portion of its course it lies close to the spine and parallel with the œsophagus.

The superior vena cava passes nearly vertically from opposite the cartilage of the first rib on the right side to the cartilage of the third rib, lying along the outer edge of the ascending portion of the arch of the aorta.

The boundaries of the space within which the heart lies are subject to variation, even when the organ is perfectly sound and the patient healthy. This results partly from the change of form which the heart undergoes in systole and diastole, partly from the change of its position occasioned by alteration in the posture of the body, and partly from the change in the size of the chest and in the position of the thoracic organs consequent on the acts of inspiration and expiration. Thus, during systole the heart twists spirally round its longitudinal axis from right to left, and its long diameter is shortened; the base is drawn

downwards and backwards, the ventricles assume a globular form, and the apex is tilted forward and passes from right to left. During diastole the converse takes place. Again, when the body is raised from a recumbent into an erect posture, the heart comes forward, and at the same time sinks downwards to the extent of an inch or even more; and it shifts in like manner from right to left, or from left to right, according as the patient lies on one side or the other. And, once again, the act of inspiration, by depressing the diaphragm, lowers the heart; whilst, conversely, expiration raises it.

CHAPTER II.

INSPECTION.

Having determined the position of the heart and great vessels, and their relation to the walls of the chest, we may proceed to discuss the means at our disposal for the examination of the circulatory apparatus within the thorax. These are—Firstly, inspection; secondly, palpation, or the application of the hand; thirdly, percussion; fourthly, auscultation.

Inspection informs us of the shape of the chest walls in the cardiac region, the condition of the integuments, and the existence or non-existence of visible pulsation, whether in the chest walls or in the larger vessels in the neck.

Without stopping to investigate the various alterations in the form of the chest which are compatible with a perfectly healthy condition of the heart and great vessels,* it may be stated generally that in a healthy person, with an ordinarily well-formed chest, the præcordial region does not differ materially from the corresponding portion of the chest on the right side, except in the fact that in the fifth intercostal space, about midway between the sternum and the left nipple, a gentle heaving or pulsation may usually be seen. This accompanies each systole of the heart, and is due to the forcible impulse of the apex of the heart against the chest walls. The area of visible impulse

* For details respecting the causes of these deviations from the normal shape of the chest see part i, chap. ii, pp. 9-11, of the present treatise.

seldom exceeds a square inch; and in stout persons, with broad, capacious chests, and in persons in whom the heart is weak and does not contract forcibly, it often happens that no impulse can be detected. In some individuals who have a short sternum the impulse is seen in the epigastrium; and in all persons change of posture, distension of the abdomen, tight lacing, and the alteration produced in the size and form of the chest, by full inspiration and full expiration, lead to corresponding changes in the position which the heart occupies, and therefore of the surface over which pulsation is visible. The heart and large vessels are all lowered during full inspiration by the descent of the diaphragm, and are all raised by its ascent during expiration.

In disease, material alterations may be observed in the form and condition of the chest walls, in the character and position of the cardiac impulse, and in the condition of the larger vessels of the neck. It frequently happens that in a state of health there is nothing to be observed in the præcordial region indicative of the presence of any organ which does not exist in a corresponding portion of the opposite side of the chest. But in disease this region may be unusually prominent or retracted, or may be the seat of pulsation of an abnormal character, or bulging and pulsation, or either of them may be observed in other parts of the chest where such phenomena ought not to exist; or the integuments may be œdematous, or covered with swollen, tortuous veins, such as are not seen in a state of health; or the larger vessels in the neck may be observed to pulsate, and to be abnormally swollen.

Abnormal prominence of the præcordial region may be due to causes acting either within or without the pericardium. Amongst the former may be mentioned hypertrophy, and dilatation of the heart; exudations of all kinds, whether serous, plastic, purulent, or hæmorrhagic, into the pericardial sac; and deposits, whether tuberculous or cancerous. Amongst the latter abscesses, aneurismal, cancerous, and other tumours in the anterior mediastinum.

The liquid effusions, more especially when copious, push forward the sternum, elevate the costal cartilages, and produce widening and bulging of the second, third, fourth, fifth, and sixth intercostal spaces; the more solid exudations, aneurismal and other tumours, and hypertrophy and dilatation of the heart, increase the prominence of the chest walls, and cause widening, but not bulging of the intercostal spaces—a fact of some importance in a diagnostic point of view. It should be added, that considerable mischief of all kinds may occur

either within or outside the pericardium, without producing prominence of the chest walls, or bulging of the intercostal spaces, and that the prominence under the conditions mentioned is most marked when the chest walls are very elastic and yielding, as in childhood or early youth.

Retraction of the chest walls in the præcordial region is stated by Dr. Walshe* to be due sometimes to absorption of pericardial effusion. But my observations do not correspond with his in this particular. It is just within the range of possibility that apparent retraction, limited to a small extent of surface, may be observed occasionally in consequence of adhesion having taken place over one portion of the heart, whilst, as yet, the remainder of the pericardial sac remains distended with fluid, but such retraction is apparent only, and referable to the abnormal prominence of the adjacent parts; and it is difficult to conceive how any real retraction can possibly take place in the præcordial region from an uncomplicated attack of pericarditis. In every instance in which I have noticed retraction, and have had the opportunity of examining the parts after death, the pericardial mischief has been accompanied by pleurisy, to which, doubtless, and not to the pericarditis, the retraction of the chest walls has been referable—the only exception being in cases which have been referred to by Skoda, in which the heart has become adherent to the anterior surface of the chest, and slight retraction of the intercostal spaces is observed consequent on its action.

The condition of the integuments is also a subject for remark in some instances, inasmuch as in cases of aneurismal, malignant, and other tumours, they are often more œdematous over the seat of mischief than in other parts of the chest. Further, the presence of swollen or distended tortuous veins may afford evidence of deep-seated pressure.

The variations which occur in the impulse of the heart, under the influence of disease, are very remarkable. Not only may the position of the impulse be shifted, but its whole character and rhythm, and the extent of surface over which it can be seen may also undergo alteration.

The apex of the heart may be visibly displaced in one of four directions, either upwards, downwards, to the right, or to the left.

The heart will be forced upwards, and its apex, therefore, must be displaced upwards, and will pulsate higher than usual in the chest, when the abdominal viscera are enlarged, the stomach or intestines

* Loc. cit., p. 181.

distended with air, or the abdomen with fluid. Ascites, and enlargement of the left lobe of the liver are especially prone to produce this result, and so is pericarditis, with copious effusion, when the patient lies very flat in bed. In such a case the pericardium undergoes distension upwards with the greatest readiness, and the base of the heart and the great vessels are thus dragged upwards and backwards as the fluid increases, and the apex of the heart is tilted upwards and outwards, so that it may even beat in the fourth intercostal space outside the nipple.*

When the heart is hypertrophied and dilated, the apex may be lowered to such an extent that sometimes it may be felt as low as the eighth rib. Again, when a patient maintains an erect or semi-erect posture during the existence of extensive effusion into the pericardial sac, the heart, by reason of its gravity, will sink to the lowest part of the fluid, and may be seen pulsating below, and usually to the left of its usual place. Aneurismal dilatations of the arch of the aorta, or of the pulmonary artery, will also depress the heart, and lower the position of its apex beat, and so also may extensive emphysema of the lungs, which serves to depress the diaphragm, to which the organ is attached.

Displacement may take place either to the right or to the left, according as disease occurs on the right or left side of the chest, or on the right or left side of the heart. Ordinarily it results from the accumulation of fluid or air in the cavity of the pleura, the displacement being to the right when the mischief is on the left side, and *vice versâ*. Extensive emphysema confined to one lung may, to some extent, produce the same effect. Sometimes, subsequent to the subsidence of acute disease in the pleural cavity, the heart is dragged over by the contraction of the effused lymph to the side on which the mischief occurred. Disease of the lung substance, leading to atrophy or diminution of bulk in the pulmonary tissue, may also occasion lateral displacement towards the side in which the atrophy occurs,† and chronic effusion into the pericardium,

* Dr. H. Davies describes displacement of the heart downwards as the invariable result of pericarditic effusion, and Dr. Walshe, on the other hand, speaks only of its displacement upwards under the same circumstances. Neither description is strictly correct. I have often seen it displaced upwards, and as often downwards, by pericarditic effusion, the result in either case being due simply to the posture of the patient, and the direction in which the fluid gravitates and distension of the pericardium occurs.

† I have seen this occur on several occasions when great atrophy of one lung, con-

which pushes the base of the heart backwards as well as upwards, and causes the whole organ to assume a more than ordinarily horizontal posture, leads to the apex pulsating far to the left of its normal position. Emphysema of the anterior part of the lung, aneurismal tumours which insinuate themselves between the heart and the anterior surface of the chest, and malignant, scrofulous, and other tumours in the anterior mediastinum, push the heart backwards, and in some instances prevent the impulse of its apex against the chest.

The extent of surface over which pulsation can be seen varies immensely. Ordinarily the impulse of the apex of the heart occasions the only pulsation which is visible on the chest walls, and the area over which that extends does not exceed a square inch. But in certain cases of hypertrophy and dilatation the heart may be seen beating over the whole præcordial region, from the third to the sixth intercostal space; whilst in certain cases of pericarditis with abundant effusion, an undulatory movement, which is synchronous with the systole of the heart, and passes from below upwards, is visible in one or all of the intercostal spaces from the second to the sixth. When the heart is excessively weak and dilated, and the patient thin, this movement may be simulated by the action of the heart itself, but when it is well marked it is very distinctive of pericardial effusion. Again, in cases of aneurismal or other tumours, extensive pulsation may be seen on the chest walls—not necessarily in the præcordial region; and in certain instances of phthisis, in which the lung has been excavated by vomicæ, the pulmonary artery may come into contact with the chest, and communicate a pulsation to the second left intercostal space. In this case, as in that of aneurismal and other tumours which give rise to pulsation, there are two distinct seats of systolic impulse, which correspond respectively with the apex of the heart and the pulsation produced by the vessel or the tumour. When from any cause the left lung is retracted and the left auricle enlarged, a diastolic impulse is sometimes visible in the second or third left intercostal space, referable to the contraction of the auricle.

The rhythm of the impulse produced by the heart is also subject to variation. Thus, instead of the single impulse ordinarily produced by the apex of the heart, there is sometimes a double impulse—the one

sequent on tubercular deposit and excavation, occurred coincidently with emphysema of the other lung. One remarkable case of this sort which occurred under my care at St. George's Hospital, is recorded in the 'Trans. Path. Soc.,' vol. xi, p. 15.

synchronous with the systole of the heart, the other occurring during the diastole. The former is visible in the fifth intercostal space, and is caused by the impulse of the apex; the other or backstroke is visible in the fourth intercostal space, and is due to the pulsation of the right ventricle during its expansion. This peculiar see-saw movement is characteristic of hypertrophy and dilatation of the heart, with or without adhesion of the pericardium. In some instances hypertrophy of the heart, especially when occurring in thin persons with a short sternum, gives rise to falling in of the epigastrium during the systole of the heart.

Distension of the large veins of the neck, especially the external jugular, is another phenomenon which inspection informs us of. It arises from pressure upon the superior cava or the vena innominata by aneurismal or other intra-thoracic tumours, or from obstruction to the circulation through the right side of the heart. When it is accompanied by pulsation it indicates insufficiency of the tricuspid valve, and of the valves seated at the junction of the internal jugular and subclavian veins. Hence it is a symptom of a largely dilated right ventricle, or of regurgitant disease of the tricuspid valve. It must be remembered, however, that even in these forms of disease pulsation of the jugulars will not ordinarily be observed until the cardiac mischief has existed for some time, and has given rise to distension of the veins sufficient to render their valves inefficient. The only exception to this is when the valves in the veins are ruptured and inoperative, in which case pulsation may be observed in an early stage of the cardiac disorder.

CHAPTER III.

PALPATION, OR THE APPLICATION OF THE HAND.

Palpation, or the application of the hand to the cardiac region, enables us to determine the position of the apex of the heart, the force with which it comes in contact with the chest, and the rhythm of its pulsation. It informs us of the movement of the ribs and of the state of vocal vibration in the præcordial region, and in certain instances it makes us aware of the existence of abnormal pulsation referable to

disease in the heart or great vessels, or of a thrill due to cardiac, pericardial, or arterial disease.

There is little to be noted by palpation respecting the impulse of the heart during health, except that it ordinarily has a certain force, is single, regular in rhythm, and synchronous with the systole of the ventricle, and occurs in the same position as the visible impulse on the chest walls; that in broad-chested, stout persons, it may be hardly perceptible, and in narrow-chested, thin persons, and in women and children, may be comparatively strong; that it is greatly increased by mental emotions, and by whatever quickens the respiration or accelerates the circulation, and that it is apparently stronger during expiration than during inspiration, when the lungs are fully inflated, and press between the heart and the chest walls.

Further, it informs us that a variety of morbid influences may induce variation in the force, character, and extent of the impulse, quite irrespectively of disease in the heart itself. It proves that when the lungs are emphysematous and press between the heart and the chest walls, the impulse is diminished or altogether annihilated; that a similar effect is observed when the heart is acting feebly, as in adynamic disorders, and when the patient is under the influence of aconite and other medicinal agents which depress the circulation—in other words, when the heart is acting feebly; as also when large, inelastic tumours occupy the anterior mediastinum, and by their bulk and want of elasticity prevent the transmission of impulse.

In like manner it shows that even when the heart is healthy there may be increased force and extent of pulsation, due to the increased facility with which the heart's impulse is transmitted to the chest walls, in consequence of pleuritic retraction of the parietes, or of consolidation of the lung tissue, or of the upward pressure of an enlarged liver or spleen, or of a greatly distended stomach or abdomen, or of the presence of small tumours in the anterior mediastinum, which are better conductors than spongy, air-distended lung. In short, it teaches us the practical lesson that increased cardiac impulse does not necessarily indicate organic disease of the heart. This is a fact which ought never to be forgotten; for if cases such as these are mistaken, as they often have been, for instances of diseased heart, an improper treatment cannot fail to be adopted. Though characterised by increased parietal pulsation, they are not accompanied by irregularity or increased force in the heart's action, and careful stethoscope exami-

nation, by revealing the absence of cardiac mischief, will generally serve to elucidate their true nature.

On the other hand, palpation proves that the heart's impulse may be greatly increased and the area of pulsation extended under a variety of morbid conditions connected with the heart and great vessels, and quite independently of external influences. The extent to which this occurs varies immensely; but to such a degree may the force of the impulse increase, that it may even shake the whole body of the patient, and impart a strong jogging motion to the head of the auscultator when placed on a stethoscope applied to the cardiac region. The area of pulsation may also undergo a corresponding extension, so that almost the whole anterior surface of the chest may heave, and pulsation may be distinctly felt on the left side of the back.

The causes of increased cardiac impulse are functional palpitation, inflammation, whether of the heart or the pericardium, and hypertrophy of the heart. Simple hypertrophy produces a great increase of impulse; hypertrophy with dilatation leads not only to increased impulse, but to an extension of the area over which it is felt. Aneurismal dilatation of the aorta or other great vessels may occasion a heaving impulse in almost any part of the thoracic walls, but is especially prone to do so on the anterior surface of the chest. In some of these instances the impulse conveys the impression of being accompanied by alternate expansion and contraction. Sometimes, again, a pulsation may exist in the second left intercostal space, which the hand perceives to be synchronous, or nearly so, with the impulse of the apex; whilst in other cases a pulsation is felt in the second or third left intercostal space, which in point of time precedes the impulse of the apex. In the former instance the phenomenon may be due to the systole of the pulmonary artery felt in that position, in consequence of retraction of the lung caused by excavation of the pulmonary tissue; in the latter it is referable to the systole of an hypertrophied left auricle.

It has been attempted to diagnose the nature of the cardiac lesion by noting the position of the increased impulse and the direction in which it is traceable. But a variety of extraneous causes may lead to the heart's pulsating a little further to the right or to the left, or to its lying more or less horizontally in the chest; and it needs little experience to prove that such data do not afford trustworthy grounds for an opinion. Indeed the hand alone will not even serve in all cases to distinguish cardiac from aneurismal pulsation.

But palpation further teaches us that disease of the heart itself may not only not augment, but may even lessen the force of the cardiac impulse to a degree which is readily appreciated by the hand. Thus, atrophy, or softening, or dilatation of the heart, may weaken its force or even render it unappreciable, and so may extensive effusion into the pericardium.

Palpation also enables us to recognise amazing differences in the character of the impulse. The shock may be abnormally sharp, abrupt, or jerking, or, on the contrary, unusually slow and heaving. The former character results from weakness and irritability; the latter, if temporary, from functional disturbance, or, if permanent, from hypertrophy. The character of aneurismal impulse is extremely variable; but it may become so forcible, and yet so abrupt, as to convey the idea of hammering, in which case it may almost be regarded as characteristic of the disorder.

Further, the hand appreciates variations in the rhythm of the impulse. Not only may it make us aware of the double impulse which is sometimes felt towards the apex of the heart, and which has been already described,* but it leads us to recognise irregularity in the force with which and the time at which the successive pulsations take place. Thus it sometimes teaches us that the successive impulses of the heart, instead of being equidistant from each other in point of time, and identical or nearly so in force, are most unequal in force, and extremely irregular in the time of their occurrence. One stroke may be forcible and the next weak, or the number either of the strong and feeble impulses, or of both, may vary indefinitely. And so also in respect to the time at which the successive impulses take place. Three or four in succession may be equidistant from each other, and these may be succeeded by an indefinite number, each succeeding the preceding one at a different interval of time, until in some instances a distinct intermission of one or more pulsations may be perceived.

The causes of the irregularity in point of time are closely connected with those which occasion irregularity of force; for the one form of irregularity is seldom observed without the other. Both the one and the other are often met with in functional palpitation, when no disease of the heart exists, and also in cases of hydrocephalus, pericarditis, and other acute disorders. In these cases, the causes of irregularity being of temporary duration, the irregularity itself is only

* *Vide ante*, pp. 457-8.

temporary. The lesions which are specially prone to occasion permanent irregularity of force are regurgitant disease of the mitral valve, and a similar affection of the aortic valves; whilst simple dilatation and fatty degeneration and flabbiness of the heart are specially productive of irregularity of rhythm. Malformations of the heart are apt to produce perversion both in the rhythm and force of the pulse; but this is not the case with simple hypertrophy of the ventricles, nor with obstructive disease of the aortic valves.

There are other phenomena, of an adventitious character, of which palpitation also takes cognizance. I allude to "friction thrill," caused by attrition of the inflamed and roughened surfaces of the pericardium, and to a peculiar "purring tremor," or thrill, caused by the forcible propulsion of the blood through orifices narrowed and roughened by disease of the valves, or through vessels roughened by atheromatous deposits, or affected with aneurismal dilatation; or, in some instances, by the irregular action of the heart or great vessels, induced by perverted innervation and a morbid condition of the blood, independently of valvular or arterial mischief. The valvular thrill being referable to an eddy produced by the forcible propulsion of blood through a roughened orifice, may diminish in intensity, or even cease altogether when the heart's action is weak and failing, and when the contraction of the orifice becomes excessive—in both of which cases the current of the blood may cease to be sufficiently full and strong to produce an eddy capable of imparting a thrill to the chest walls. In like manner, the aneurismal thrill may diminish, or even cease, if the heart's action becomes feeble, or if the aneurismal pouch becomes filled with smooth coagula of fibrin. In the rare and exceptional cases in which parietal thrill is caused by perverted cardiac action, dependent solely on perverted innervation, and a spanæmic condition of the blood, the thrill, from its nature, is necessarily of temporary duration; whereas, when due to aneurism or valvular disease, it is usually a persistent phenomenon. When occurring as the result of perverted innervation, and an altered condition of the blood, it may be felt over the entire surface of the chest, and especially over the pulmonary artery in the second left intercostal space. When due to aneurismal dilatation, it varies in position with the seat of the aneurismal tumour; and when referable to valvular disease, it varies in position according as one valve or the other is affected. The most common cause of valvular thrill is regurgitant disease of the mitral valve, occurring coincidently with hypertrophy of the left ventricle, in

which case the thrill is felt principally in the third and fourth left intercostal spaces, where the position of the lung enables the heart to come into apposition with the chest walls; but I have also felt it on two occasions in connection with extensive regurgitant disease of the aortic valves; the only other cause of it* is aortic obstruction, with hypertrophy of the ventricle, in which case the thrill is felt in the track of the aorta, and even somewhat beyond its limits; or, in other words, in the third left, and second and third right intercostal spaces, close to the sternum.

Aneurism is, perhaps, the most frequent cause of purring thrill, and the phenomenon is usually more strongly developed than in cases of mere valvular mischief. In all cases in which a thrill exists, a spanæmic condition of the blood adds greatly to its intensity.

Pericardial friction thrill is comparatively rare, and, when perceptible during the diastole as well as during the systole of the heart, is confined, as I believe, to cases in which the outer layer of the pericardium is adherent to the anterior of the chest by lymph effused in the anterior mediastinum.† The sensation which it conveys to the hand is very similar to that produced by endocardial thrill; but, although sometimes synchronous with the systole of the heart, it usually accompanies the greater part of the heart's action. It frequently shifts its position, and is felt over a large extent of surface—facts which, to a careful physician, are quite sufficient to distinguish it from valvular thrill.

Hypertrophy and dilatation of the heart may cause widening of the intercostal spaces, and effusion into the pericardium may give rise to bulging of the spaces, as already described;‡ and these facts, and also the absence of vocal vibration over the surface occupied by the enlarged and dilated heart or distended pericardium, will be readily detected by the hand.

Palpation gives us little information respecting the condition of the thoracic aorta in a state of health. If the finger be placed above the sternal notch, and pressed down behind the sternum, as far as possible

* It is conceivable that obstructive disease of the mitral valve might give rise to its occurrence, or, again, that it might arise from disease in the tricuspid and pulmonary valves. But experience does not give its warrant for the supposition, and I believe the statement above made to be strictly correct.

† This at least is certain, that this condition of the parts existed in the only seven cases in which I have ever had the opportunity of verifying the observation by inspection after death.

‡ See p. 454 of this treatise.

towards the aorta, the pulsations of the vessel may generally be felt, but in no other position does palpation even make us aware of their existence. It affords us, however, important assistance in an examination of the large vessels of the neck and other parts of the body. It tells us of an undulatory, expansile movement, which is referable to the diastole of the artery, and corresponds to each ventricular systole, and of a contraction or systole of the vessel corresponding to each diastole of the ventricles; it shows, that in the vessels near to the heart the diastole and systole are respectively coincident in point of time with the systole and diastole of the ventricles; but that, in the radial artery, and in other vessels more distant from the centre of circulation, each movement of the arteries is somewhat behind its corresponding movement in the heart, so that the radial pulse is not quite synchronous with the systole of the ventricles.* Further, it informs us of abnormal pulsation in the vessels, referable to functional derangement of the system, and also of variations in the pulse occasioned by disease in the heart and in the coats of the vessels. Thus we learn that the pulse may be quick or slow, soft or hard, small or large, weak or strong, regular or irregular in force, and other characters—intermittent, jerking, thrilling, fluttering, or bounding—each of which conditions may be indicative of disease, though not necessarily connected with any special form of mischief. Arterial thrill, as contrasted with thrill communicated to the chest walls, may originate in an altered condition of the aortic valves, or of the aorta itself, or of the blood and of the nerves supplying the vessels. Thus a peculiar thrill, resulting from aortic obstruction or aortic regurgitation, may be sometimes felt in the larger vessels all over the body; simple dilatation of the aorta, with roughening of its internal surface from atheromatous or calcified deposits on its coats, is another frequent cause of arterial thrill, and so is perverted innervation combined with an impoverished spanæmic blood.

When there is pulsation in the jugular or other large veins, palpation may discover a soft thrill in the veins somewhat resembling an arterial thrill. This is not always, nay it is not generally present in these cases, nor has it hitherto been traced to any special form of disease. So far as is known at present it simply denotes the existence of excessive regurgitation, producing more than ordinarily forcible vibration.

* This may be admirably demonstrated by the aid of Dr. S. Alison's sphygmoscope.

CHAPTER IV.

PERCUSSION.

In the section devoted to the topography of the heart and great vessels, attention has been drawn to the relations which these organs bear to the chest walls, and to the surrounding structures. We are, therefore, in a position to form an opinion as to the sound which should be elicited by percussion of the præcordial region in a state of health, and to judge of the deviations in the character of that sound which are likely to be produced by various forms of disease. In practice, however, many complications occur which would not suggest themselves to the unpractised examiner, and it may be advisable, therefore, to consider the subject in detail, rather than allow the sounds which arise to remain a matter of inference.

In the first place, it may be stated, that even in a state of health, considerable difficulty is necessarily experienced in determining accurately the size and position of the heart. The constant action of the organ, involving, as it does, perpetual change in its form and position; the incessant alterations induced by respiration in the position of the lungs; the ever-changing resonance elicited over the ribs and sternum at different stages of the respiratory act; and the proximity of the great vessels above and of the liver below—present obstacles to an accurate determination of its outlines which cannot be readily overcome. Therefore, when an attempt is made to determine by percussion the size and position of the heart and great vessels, every care must be taken to ensure a satisfactory result.

The patient should be placed in a recumbent posture, and gentle as well as forcible percussion should be practised. Gentle percussion will bring out the resonance of any lung tissue lying between the heart and the chest walls, whilst a more forcible stroke will elicit the dull sound caused by the presence of the heart behind.

Further, the intercostal spaces, as well as the ribs and sternum, should be percussed; for when the bony structures receive the impulse, it is communicated in some degree to the adjacent lungs, and a clearer resonance is elicited than would be yielded by the structures

immediately beneath the part percussed. If these precautions are carefully attended to, the physician will generally be enabled, especially in thin persons, to arrive approximately at a correct conclusion.

The following are the sounds ordinarily emitted by the chest on percussion when the patient is in a state of health.

In the præcordial region, where the heart, uncovered by lung, comes into immediate contact with the chest walls, percussion yields a short, dull sound, and conveys a sense of strong resistance to the finger. The spot where this is most distinctly marked is between the fifth and sixth ribs on the left side. When percussion is practised towards the boundaries of this uncovered space, the sound becomes somewhat clearer and more resonant, and the resistance less, in consequence of the proximity of the lungs; and in those parts where the lungs overlap the heart, and lie between it and the chest walls, the resonance and the sense of resistance to the finger vary according to the thickness of the intervening stratum of lung tissue and to the force of the percussion stroke.

The gentler the percussion employed—a gentler tap with one finger answers best—the more clearly the pulmonary resonance is brought out. The greater the thickness of the stratum of lung, the clearer and more pulmonic will be the resonance. The area over which more or less of dulness may be elicited by forcible percussion corresponds to that occupied by the heart and great vessels, and this has been already pointed out.* Suffice it to say, that it extends vertically from the upper border of the third to the lower margin of the sixth left cartilage, and transversely from about half an inch to the right of the left nipple to about half an inch beyond the right margin of the the sternum, opposite the fourth sterno-costal articulation. Of course the act of respiration, change of posture, and other circumstances which have been already referred to as influencing the position of the heart affect the area which is left uncovered by the lung, and exert a corresponding modifying effect on the result of percussion. Inspiration especially, by inflating the pulmonary tissue, forces the left lung in front of the heart, and diminishes the area of dulness, whilst expiration usually increases it.

In disease the area of præcordial dulness is liable to greater variations than it is even in health, and its increase or diminution, as the case may be, may exist in the heart itself, in its pericardium, or in disease external

* See chapter on the topography of the heart and great vessels, pp. 450-51.

to, and independent of it. Ranged in a tabular form, the causes of increased præcordial dulness may be stated as follows:

First. *External to the heart and pericardium.—a.* Morbid growths or abscess in the anterior mediastinum, or in other parts of the chest, encroaching on the mediastinum. *b.* Aneurism of the aorta or other great vessels. *c.* Consolidation of the pulmonary tissue. *d.* Pleuritic effusion in the left pleura, encroaching on this region. *e.* Retraction of the left lung, consequent on pleural adhesions, whereby a greater surface of the heart is brought into contact with the chest walls. *f.* Enlargement and upward pressure of the liver, especially of its left lobe.

Secondly. *Existing in the pericardium.—a.* Effusions, whether of serum, lymph, pus, or blood into the pericardium. *b.* Tubercular or cancerous deposits, which are usually attended by serous or serosanguineous effusion.

Thirdly. *Existing in the heart itself.—a.* Hypertrophy of the heart, *b.* Dilatation of the heart; *c.* Dilatation of the heart, attended by hypertrophy of its walls.

The causes which lead to diminution in the area of præcardial dulness are to be found in like manner in the heart itself, in the pericardium, or in disease external to it. Ranged in a tabular form, they may be as follow:

First. *External to the heart and pericardium.*—Pneumothorax and emphysema of the lung.

Secondly. *Existing in the pericardium.*—Effusion of air into the pericardium.

Thirdly. *Existing in the heart.*—Atrophy of the heart.

The causes external to the heart and pericardium which operate in producing extension of the præcardial dulness scarcely call for any remarks. Their action is self-evident, and so also is the fact that they may and possibly would occasion considerable difficulty in the diagnosis. Indeed, it often happens that the true nature of the mischief cannot be arrived at merely by the aid of percussion, and that it would escape detection if the other physical signs and the general symptoms were not appealed to. Perhaps the only lesion which requires special notice is aneurism of the aorta, or other great vessels. It must always be remembered that the extent of surface over which the presence of an aneurismal sac gives rise to dulness on percussion affords no clue to the size of the aneurism, inasmuch as from its globular form, the tumour may not come extensively in contact with the chest walls. Further, aneurism of the

aorta, even of considerable size, if arising from the posterior portion of vessels deeply seated in the chest, cannot be detected by percussion without much difficulty, especially if the patient be stout, whereas, in a thin person, a small aneurism, no larger than a walnut, if arising from the upper and anterior surface of the aorta, towards the right angle of the arch, will generally give rise to sufficient dulness on percussion to ensure its detection on careful examination.

The causes of increased præcordial dulness existing within the pericardium demand a more lengthened comment. Fluid of whatever kind in the pericardial sac, whether consisting of simple serum, the result of passive effusion or of active dropsy, or of serum mixed with lymph or pus, or blood, the products of pericardial inflammation, or of blood resulting from an injury to the heart, or from the oozing of an aneurism, must necessarily produce extension of the limits of normal dulness on percussion; and under favourable circumstances, four or five ounces will suffice to do so. In such a case the increase of dulness will be chiefly perceptible towards the base of the heart; for the pericardium admits of distension upwards more readily than in any other direction; and in a recumbent posture the heart, if unrestrained by adhesions, would also have a tendency to rise upwards.

On the other hand, when fluid has accumulated to a large extent, the elasticity of the pericardium is impaired, and the sac may undergo enormous distension—not upwards only, but backwards, laterally, and even downwards. It still retains its pyramidal form, its broad base resting on the diaphragm, and its apex seated behind the upper part of the sternum; but in cases of extreme distension, the apex may protrude even above the clavicle,* and the base pushing the diaphragm downwards, may cause a slight fulness in the epigastrium, whilst laterally its walls may extend almost from nipple to nipple, and may produce condensation of the inner and anterior margins of the lungs. In such a case as this the percussion sound is absolutely dull over the entire surface covered by the distended pericardium, and the resistance to the finger is very great, just as in pleuritic effusion.

More commonly the amount of fluid is smaller, and the pericardium, though distended, does not reach above the level of the second sterno-costal articulation; but it pushes aside the anterior borders of the lungs,

* This occurs only in cases in which the patient retains a recumbent posture. More commonly the patient sits erect or leans forward, resting the elbows on the knees, and then the dulness rarely extends above the second sterno-costal articulation.

and comes into immediate contact with the chest walls. The result is the production of dulness on percussion, traceable over an area which in ordinary cases extends from the second costal cartilage above to the cartilage of the sixth rib below, and laterally from the left nipple to about an inch, or even more, to the right of the sternum.

Percussion affords material aid in distinguishing between pericardial effusion and hypertrophy with dilatation of the heart. The dulness of pericardial effusion is developed more rapidly than that due to hypertrophy of the heart; it reaches higher in the chest, and extends lower down;* it shifts its position more completely under change of posture; it is more intense in character, and yet it is only of temporary duration, and the resistance to the finger is greater. Of course, if the exudation consist principally of plastic materials, the seat of dulness will not be so readily altered by change of posture as if the effusion consisted principally of serum, nor will the dulness rise so high in the chest, nor will it disappear so speedily; but it will be developed much more rapidly than hypertrophy of the heart, and may be readily distinguished from it by other means of diagnosis.

The effect of tuberculous or cancerous deposits in the pericardium necessarily varies with their extent and position. If they are extensive, and seated on the lateral margin of the anterior surface of the heart, they necessarily give rise to an increase in the area of dulness, even though they be not accompanied by effusion into the pericardium; whilst if seated behind the heart, they are apt to push that organ forward, and thus, by causing displacement of the anterior margins of the lungs, lead to an extension of the præcordial dulness. When accompanied, as they often are, by effusion into the pericardium, the fluid gives rise to still further extension of the area of dulness.

The causes of increased præcordial dulness existing in the heart itself are also deserving of attentive study. They are hypertrophy, hypertrophy with dilatation, and dilatation without hypertrophy. In either form of disease both sides of the heart may be hypertrophied or dilated, or the enlargement may be confined to the right or to the left side. The augmentation of bulk may vary from a slight amount, which it is difficult if not impossible to detect by percussion, up to the enormous size of a heart, mentioned by Dr. Williams, which weighed forty ounces, its

* Dulness referable to an hypertrophied heart does not, until the disease is far advanced, extend above the third left rib or below the sixth.

circumference round the base being fourteen and a half inches, its length, from the arterial orifices along the septum to the base, eight and a half inches, and the thickness of the left ventricle one to four inches; whilst near the columnæ carneæ it measured from one to five inches. In the case of even a much smaller increase of bulk than is here recorded, the lungs are pushed aside, and an unusually large surface of the heart comes into immediate contact with the chest walls, and leads to an extension of the area of præcordial dulness on percussion, and to a sense of increased resistance.

When the hypertrophy is general, and especially when it is combined with dilatation, the heart sinks downwards, in consequence of its increased weight, and its change of position is often to the full extent of the increase in its vertical diameter. From the horizontal position in which it necessarily lies, its apex beats far to the left of its usual seat of impulse, often, indeed, to the left of the nipple. It follows, therefore, that in most instances of cardiac hypertrophy an increase in the area of the percussion dulness will be perceptible in that direction. But it should be clearly understood that when the patient has been lying flat in bed for a considerable time the heart is apt to fall backwards, and even upwards, to such an extent that no downward extension of the percussion dulness may be perceptible, but, on the contrary, some extension of the dulness upwards—and this the more so when the left auricle is much dilated and hypertrophied. In several such cases I have known extension of the dulness at the base extremely well marked.

When the hypertrophy or dilatation occurs principally on the left side of the heart, the extension of dulness on percussion will be to the left, or to the right if the right cavities are the seat of enlargement.

It is only right to add, however, that when hypertrophy or dilatation exist in only a slight degree, there is nothing more difficult than to determine the fact by percussion alone. Even when the disease is further advanced, its diagnosis, by the aid of percussion, is often extremely uncertain, inasmuch as it is frequently accompanied by emphysema of the anterior margin of the lungs, and if a portion of emphysematous lung forces itself between the heart and the chest walls, the dulness which would otherwise be produced by the enlarged heart is masked, and the outline of the organ cannot be satisfactorily ascertained.

Dr. Walshe and some other authorities have stated that old adhe-

sions of the pericardium produce an increase in the area of præcordial dulness.* Nothing, however, can be more repugnant to facts, or more calculated to mislead than an unqualified statement of this kind. Observations at the bedside, confirmed by careful examinations in the dead-house, prove conclusively that adhesions of the pericardium do not even give rise necessarily to enlargement of the heart; and that, although universal, they may, by absorption, become so thin as not appreciably to thicken the walls of the heart. Common sense revolts at the idea of such adhesions producing any extension of the area of dulness; and, in truth, the statement that pericardial adhesions occasion extension of præcordial dulness will only hold good in respect to cases in which pericarditis has recently occurred, and in which, although the fluid exudation has been absorbed, a thick coating of plastic lymph remains, and has not yet undergone complete solidification. When extension of dulness upwards is observed years after an attack of pericarditis, it may be referable to thickening caused by coexistent pleurisy, or to hypertrophy and dilatation of the heart, or to the fact of the heart having become adherent to the anterior parietes of the chest by lymph effused in the anterior mediastinum, but assuredly not to lymph effused years before into the pericardium. In certain instances of old-standing and universal pericardial adhesions, which had led to atrophy of the heart, I have even noted a decrease in the normal area of præcordial dulness.

Endocarditis has no appreciable effect on the præcordial dulness, nor has valvular disease, except in virtue of the hypertrophy and dilatation to which after a time it ordinarily gives rise.

The causes which produce a diminution in the area of præcordial dulness are far less numerous and of much less importance than those which lead to its increase. Pneumothorax, and emphysema of the lungs —especially of the left lung—are the only causes, external to the heart and the pericardium, which operate in bringing about this result, and when emphysema is accompanied by bronchitis, the effect is seen most strikingly developed. Not only is the heart displaced by the dilated lung, but the lung pressing in front of it causes a diminution in the area of dulness, and a great decrease in the sense of resistance.

The only cause which can operate within the pericardium in producing a diminution of the præcordial dulness, is the presence of air or

* Walshe, loc. cit., p. 202.

gas in the pericardial sac, consequent on traumatic injury to the organ. Cases such as these are extremely rare, and practically are of little importance.

The only cause of diminished præcordial dulness existing in the heart itself is atrophy of its substance. A decrease in the bulk of the heart, by admitting a closer approximation of the anterior margins of the right and left lungs, diminishes the extent of surface over which the organ remains uncovered by pulmonary tissue, and thus lessens the area of dulness and the sense of resistance to the finger.

CHAPTER V.

AUSCULTATION.

When the ear is applied to the surface of the chest in the præcordial region, two sounds, referable to the action of the heart, are distinctly audible. These sounds follow each other in quick succession, and are separated by intervals of silence. The first or systolic sound is synchronous with the systole or contraction of the ventricles, with the stroke of the heart against the chest walls, with the pulsation of the arteries near the heart, and nearly so with the pulse of the radial artery; the second or diastolic sound is synchronous with the diastole or filling of the ventricles, with the recedence of the heart from the chest-walls, and with the systole or pulseless condition of the large arteries. The noiseless interval between the first and the second sounds is termed the first interval of silence; that which succeeds the second sound is styled the second interval of silence.

These sounds and intervals of silence are of different duration, and succeed each other in a certain order of succession, which is termed the "rhythm" of the heart. The first sound is long, the first silence extremely short; the second sound is short, and the second silence long. Both sounds and both intervals of silence are comprised within the period of time between two successive arterial pulsations, and, therefore, if we suppose the heart to be beating at the rate of 60 in a minute, they together occupy exactly a second of time. This being the case it will be readily understood that it is difficult, if not impossible, to determine

accurately the true relative duration of each sound and each interval of silence, but it may be stated roughly that if the whole period between two pulsations be divided into twenty equal parts, about seven of these will be occupied by the first sound, one by the first interval of silence, four by the second sound, and eight by the second interval of silence.* Practically, when the pulse is over 80, the first interval of silence is scarcely appreciable by the ear, so that the first sound appears to be followed immediately by the second; whereas, on the other hand, when the pulse is slow and does not exceed 50 or 60 the first interval of silence is very perceptible.

The two sounds of the heart are unlike one another in intensity, pitch, quality, and duration. The first, as heard over the left apex of heart, is apparently deep-seated, of a dull, low-pitched, booming character, at once louder and more prolonged than the second sound, which is clear, short, and flapping, and comparatively superficial. The two syllables tubb, dŭp, if clearly articulated, convey a tolerable idea of the sounds.† But there are other differences which require special consideration. The two sounds not only differ from one another, but each varies in its character at different portions of the surface of the chest. Thus at the right apex the first sound is less dull and less prolonged than at the left, and the second sound is clearer and more abrupt; whilst at the base of the heart, or rather just above the origin of the large vessels, the first sound on both sides resembles that heard at the right apex, and the second sound is louder, clearer, and more flapping than at the apex. The first sound is heard loudest at the apex, and becomes gradually weaker as it is traced upwards towards the base of the heart; the second sound is heard loudest and clearest at midsternum opposite the third cartilage, and loses in intensity as it is traced upwards or downwards.

* Laennec estimated the time occupied by the two sounds and the first interval of silence at rather more than three times the period of the second silence, whilst Volkmann, who endeavoured to measure their duration by the aid of vibrating pendulums, declares that the two sounds and the first interval of silence do not together occupy more time than the second interval of silence. Few observers would be found to bear out Laennec's estimate; but my own conviction is equally opposed to that of Volkmann, and I believe that the proportions given in the text represent very closely the relative duration of the sounds and intervals of silence when the pulse is about 76 in a minute. When the heart is pulsating very slowly Volkmann's statement may be more correct.

† Dr. C. J. B. Williams, to whom the profession are so much indebted for his contributions towards the elucidation of all auscultatory phenomena, was the first, I believe, to suggest this mode of imitating the sounds of the heart.

The extent to which both first and second sounds are propagated in the chest varies greatly; the variation being dependent in part on the intensity of the sounds themselves, but even more so on the condition of the surrounding pulmonary tissue. Emphysema, by impairing the contractile power of the lung, has the effect of preventing their transmission, and consolidation of the lung has the contrary effect. Under ordinary conditions they are more readily propagated to the left than to the right, and are often audible on the left side posteriorly when they are quite inaudible on the right.

But the sounds sometimes pass beyond the boundaries of the chest and are transmitted far along the track of the larger vessels. The conditions which conduce to this result are:—First, activity of the circulation; secondly, thickness and elasticity of the vessel; thirdly, nervous excitement. Under these circumstances both sounds are usually to be heard in the carotid and axillary arteries, and they may be audible even in the femoral. They gradually decrease in force in proportion as the vessels are further and further removed from the centre of circulation, until at length the second sound ceases to be audible, and at a still greater distance from the heart both sounds become inaudible.

It has been maintained by Dr. Walshe and other writers that the sound heard in the arteries is not conducted from the heart, but is generated in the vessels themselves by the "impulsion and friction of the blood," "the vibration of their walls," and "the current-like motion given to their column of blood by the form of the vessels." "It is not conceivable," says Dr. Walshe,* "that sound audible in the popliteal or radial artery is the mere result of conduction from the heart." A few experiments on the conduction of sound through fluid contained in elastic tubes will satisfy any one that this conclusion is incorrect, and that sonorous vibrations are transmitted along the arteries as readily as the vibrations which, in certain forms of cardiac disease, take their origin in the heart, but convey a sensation of thrill to every artery in the body. That the sounds are not generated in the vessels under ordinary circumstances, is obvious from the fact that they are rarely to be heard in the radial and popliteal arteries except under the conditions above referred to, as augmenting the sounds of the heart and favouring their transmission; whereas if they took their origin from the vessels or their contents, they ought to be invariably present. The only condition under which a sound is generated in the arteries is where their internal

* Loc. cit., p. 270.

coats are roughened by disease, and then the sound assumes the character of a murmur.

With a view to a clear comprehension of the cause of these cardiac sounds, it will be desirable to analyse a complete action of the heart, and to consider what is going on at the time of their occurrence.

A complete action of the heart comprises the following events, which take place as set forth below.

Rhythm.	*Duration.*	*Occurrences which take place at different periods of the heart's action.*
Systolic, or First Sound	$\frac{7}{20}$	The ventricles contract to expel the blood from their cavities; the mitral and tricuspid valves, which are closed partly by the action of the elastic tissue which enters into their composition,* and partly by the action of the blood in the ventricles, are rendered tense by the impulsion of the blood against their ventricular surface and by the contraction of the columnæ carneæ; the blood is driven through the aorta and pulmonary artery; the valves at the orifices of those vessels are forced back against the arterial walls by the onward current of the blood; the vessels themselves are dilated; the impulse of the heart against the chest walls takes place; the auricles become relaxed, and permit blood to flow into them from the vena cava and pulmonary veins, and the fluid contents of the ventricles are in a state of molecular collision.
First interval of silence	$\frac{1}{20}$	This is the moment which elapses after the complete systole of the ventricles before the elastic tissue which enters into the composition of the semilunar valves has effected their closure, and the aorta and pulmonary artery have contracted on the blood within them sufficiently to produce the forcible recoil of the blood on the semilunar valves which produces their sudden tension.
Diastolic or second sound	$\frac{4}{20}$	The auricles continue to receive blood from the large systemic veins; the muscular contraction of the ventricles ceases; the pressure of the blood on the ventricular surface of the auriculo-ventricular valves being removed, a certain amount of blood begins to pass through them from the auricles, and occasions passive dilatation of the ventricles; the aorta and pulmonary artery contract on the blood within them, and by forcing it against the upper surface of the semilunar valves throw them into a state of tension.

* See 'Med.-Chir. Trans.,' vol. xliv, p. 43.

Second interval of silence	$\frac{8}{20}$	The ventricles are still relaxed, and blood continues to pass into them from the auricles and into the auricles from the large vessels, until at length the auricles contract, and, by forcing their contents through the auriculo-ventricular opening, complete the dilatation of the ventricles and distend their cavities.

Throughout the whole period of the action of the heart, the two surfaces of the pericardium glide over each other to a greater or less extent.

It will be obvious, by reference to the above statement, that there are many conceivable causes of sound both during the systole and diastole of the heart, and they require careful consideration before a satisfactory conclusion can be arrived at respecting the causation of the sounds of the heart. Those which are in operation during the first sound of the heart must take precedence of the others, and may be briefly stated as follows:

1st. *The muscular contraction of the ventricles.*—The doctrine taught by many physiologists attributes to muscular contraction the principal share in the production of the first sound of the heart. Hope, Williams, Stokes, Walshe, Davies, and other physicians in this country, and many others of the leading authorities abroad, have contended for the predominance of this cause of sound, and in proof of their position have cited the fact that sound is produced by the violent or spasmodic contraction of voluntary muscles. But, although the violent contraction of voluntary muscle is to some extent soniferous, its ordinary quiet contraction is not so; and careful experiment has proved that when the heart is acting quietly, the contraction of its muscular fibres is almost if not quite unproductive of sound.* In short, it may

* Dr. Halford's admirably conceived experiments, at which I had the privilege of being present, proved this beyond dispute. When the ingress of blood into the heart was prevented, the first sound wholly ceased, although the ventricle continued to contract forcibly and rhythmically. Dr. Markham has raised an objection to any inference from this experiment, on the ground that as there was no blood for the heart to contract upon, the heart's contractions must have been abortive. But they were forcible enough to jolt the head placed on a stethoscope applied to the heart, and the eye could detect little difference between the force of their contraction when the blood was admitted into the heart, and when it was excluded. Therefore if they are to be considered abortive, the contraction of voluntary muscle which, in like manner, takes place without any substance for the muscle to contract upon, must also be regarded as abortive, and, therefore, ought not to be soniferous. Again, if the contraction of a small muscle like the ventricle of a human fœtus is productive of sound, it is difficult

be stated that during the ordinary tranquil action of the heart, muscular contraction does not contribute to the production of the first sound, but that under certain conditions of excitement, when the heart is irritable, and is acting violently, this source of sound comes into operation. In proof of which I would refer to the peculiar dull, muffled noise which is super-added to and confuses the first sound of the heart in certain instances in which the heart is acting violently—a sound which subsides as soon as the heart resumes its ordinary mode of action.

2ndly. *The sudden and forcible closure of the mitral and tricuspid valves.*—The authorities already mentioned have dwelt but little on this source of sound, but Dr. Billing and other physiologists in this country and abroad, including Rouanet, Bouillard, Bryan, Brakyn, Valentin, and Kiwish, have insisted on this sudden tension of the auriculo-ventricular valves as the sole cause of the first sound. Dr. Billing was the first to propound this view, which of late has found an able advocate and expositor in Dr. Halford; and although I consider it somewhat too exclusive if applied to all diseased conditions of the heart, Dr. Halford's recent experiments have led me to believe that in a healthy state of the circulatory apparatus, the forcible tension of the mitral and tricuspid valves is the principal source of the first sound, and, under ordinary circumstances, the sole cause of the sound as it reaches the ear applied to the chest walls. Indeed, the loudness and clearness of the first sound of the heart, when the ventricles are thin, and the impulse by no means forcible, and its weakness when the walls of the ventricles are thick and their contraction strong, are quite in keeping with the valvular origin of the sound, and utterly inconsistent with its muscular origin; so also is the fact that when the ventricles are much hypertrophied, and the auriculo-ventricular valves rigid, the first sound is scarcely audible at the apex.*

to understand why the contractions of much larger muscles, such as exist in the auricle of an adult, and still more so in the auricle of a horse or an ox, should not be attended by sound, and yet experience proves conclusively that they are not so. Further, there is no reason for believing that the ordinary contraction of the muscular tissue of the heart would be more productive of sound than the ordinary contraction of voluntary muscle, and in the latter case sound is usually absent—sound being excited only when the muscle is contracting violently.

* It is sometimes asserted that in these cases the weakness of the sound at the apex is attributable to the fact that in hypertrophy the muscular contraction is more or less imperfect. But no assertion can be more gratuitous or unfounded. So forcible are the muscular contractions in hypertrophy, that the whole chest sometimes heaves under their influence, and as the sound emitted by the contraction of the voluntary

3rdly. *The rush of blood over the surface of the ventricles, and through the aorta and pulmonary artery, and the throwing back of the semilunar valves against the sides of the vessels, the dilatation of the vessels themselves, and the molecular collision of the blood.*—The endocardium, the semilunar valves, and the internal coat of the arteries are so exquisitely smooth that the friction of the blood against them is reduced to a minimum; and as observation and experiment have proved conclusively that in a healthy condition of the arteries no appreciable sound is produced in them by the current of blood, nay, more, as it is admitted that no sound is excited by the rush of blood and the collision of its particles during the systole of the auricles, when the blood comes in contact with the irregular surface of the walls of the ventricles, it is highly improbable that any sound would arise from the current of blood or the collision of its particles during its passage from the ventricles through the exquisitely smooth ventricular opening, and equally smooth arteries, the more so as not the slightest vacuum occurs at any period of the heart's action; and, therefore, there can be no dashing of blood, and no violent collision of the blood globules amongst themselves. As well might we expect to elicit sound by succussing a patient with fluid in the pleura, as to expect sound to be produced by the motion of the blood in the cavities of the heart. Therefore I would maintain that when the circulatory apparatus is healthy, and the heart is acting tranquilly, these sources of sound do not contribute to the production of the first sound of the heart as heard through the walls of the chest. When the heart and great vessels are diseased, and more especially when under such circumstances they are acting violently and irregularly, the phenomena included in this paragraph may become distinctly soniferous and not only serve to alter the character of the first sound, but to produce a murmur.

It is maintained by some observers that the causes under discussion contribute largely to the production of the first sound as heard at the base of the heart; and Skoda and his followers speak of an arterial first sound as distinct from the cardiac first sound. The arguments, however, by which they attempt to support their position appear to me to be utterly fallacious. In the first place it is stated that the first sound may be heard in all the large arteries of the body, however distant from the centre of

muscles is never so loud as when the muscles are hypertrophied, as in the case of the pectoral muscles of the "navvy," and of the deltoid muscles of the blacksmith, so there is no reason for doubting that the same holds good in regard to the muscular structure of the heart.

circulation, and that this fact is incompatible with its transmission from the heart. But there is no valid foundation for this assertion. Indeed it is directly negatived by experiment. If a long, thin elastic India-rubber tube be filled with water, and a stethoscope be applied to one end whilst sonorous vibration is excited at the other, it will be manifest to the merest tyro in auscultation that the transmission of the first sound of the heart along the larger arteries is not only possible, but extremely probable.

Again, the fact that the first sound is sometimes heard as loud or even louder in the carotid artery than over the aortic orifice is adduced in proof of the same position; but what can be more probable than that in certain instances in which the chest walls are thick, or a portion of emphysematous lung or some other imperfect conducting medium intervenes between the aorta and the ear, the first sound in a vessel like the carotid which is not far removed from the centre of circulation, and lies quite superficially, should be heard louder than over the distant aorta.

Again, it is stated that the first and second sounds differ in character, tone, and duration, and that on that account, if the second sound is referable to the forcible closure of the valves, the first sound cannot be so. But this objection will not bear discussion. Not only are the auriculo-ventricular valves larger and thicker than the semilunar valves, but their free borders, unlike those of the semilunar valves, are attached to the heart by the tendinous cords and carneæ columnæ which are necessarily thrown into vibration by the forcible tension of the valves, and complicate the mere valvular flapping sound which would otherwise result from the closure of the valves. Further, the walls through which the sound of their tension is conducted to the ear are thicker and less vibratile than the walls to which the semilunar valves are attached. So far from being an argument against the valvular origin of the first sound, the fact alluded to is a powerful argument in its favour, for if the first sound were not more prolonged, and of a different character and tone from the second sound, assuredly it could not arise from the closure of the auriculo-ventricular valves.

Again, it is stated that in certain cases of regurgitant disease of the mitral valve, where the first sound at the left apex is replaced by a murmur, the first sound at the base of the heart is almost normal in character. This statement, if correct, would be deserving of attention, but I cannot admit that it is so. Frequently, indeed, when a mur-

mur masks the first sound at the left apex, the first sound is heard comparatively clear at the base, but so it is also at the right apex—the normal sound in both instances being referable to actions going on in the right as well as in the left chambers of the heart. In short, the clear sound of valvular flapping overpowers the murmur at the base, just as the murmur overpowers and masks the sound at the left apex, though not at the right; the slight transmission of the murmur being referable to the lowness of its pitch as compared with the sound produced by the sudden tension of the valves. The correctness of this view is proved by the fact that when the mitral valve is connected with a rigid bony ring, and is unable to flap, so that the first sound at the apex is really replaced, not merely masked, by a murmur, the murmur, though slight, is heard over the base of the heart, and, indeed, entirely replaces the normal sound. This fact, which I have not seen noticed in any work on auscultation, has enabled me on more than one occasion to diagnose the condition of the valves here referred to, and to predict that it would be found after death.

4thly. *The impulse of the heart against the walls of the chest.*—Whilst witnessing Dr. Halford's experiments, I satisfied myself that the so-called impulse of the heart bears no sort of resemblance to a blow, but is simply the result of pressure exerted by the walls of the heart as they assume a globular form during contraction; and further, that whilst the heart is acting quietly, the ear is not conscious of any sound produced by its impulse. Therefore I cannot doubt that under ordinary circumstances the so-called impulse of the heart does not contribute to the production of the first sound.

The possible causes of sound which are in operation during the period of the second sound may be briefly stated as follows:

1st. *The auricles continue to receive blood from the large systemic veins, the auriculo-ventricular valves are forced back against the walls of the ventricles, and a certain amount of blood passes from the auricles through the auriculo-ventricular openings into the ventricles.*—In the normal state the blood enters the auricles, and passes from the auricles into the ventricles in a steady current, and, as there is no roughness or other cause of friction in the lining membrane of either auricles or ventricles, and no vacuum to admit of splashing, it is highly improbable that any sound should be thereby produced capable of transmission to the surface of the chest. So also the falling back of the auriculo-ventricular valves must be unproductive of sound.

2ndly. *The dilatation of the ventricles.*—Whilst witnessing Dr. Halford's experiments I satisfied myself that this action is unproductive of sound.

Skoda has suggested that during the dilatation of the ventricles the heart recedes from the walls of the chest, and in so doing may occasion sound; and in corroboration of his theory he has cited the fact that in exceptional cases the second sound, though weak at the base, is clear at the apex of the heart, and therefore must be partly of ventricular origin. My own opinion is adverse to his theory and to the conclusion he draws from the fact cited in support of it. Direct experiment, which shows that the second sound is heard clear, loud, and unaltered in character, when the heart does not come in contact with the ribs, proves that the cause assigned by Skoda contributes little if at all to its production, and the fact cited in support of his view is more readily explicable by supposing that in the cases referred to the transmission of the sound at the base of the heart, was stopped by the intervention of fat or a portion of emphysematous lung—a condition which I have found existing in two cases precisely resembling those alluded to by Skoda. The fact, however, which is most fatal to his theory is that the recedence of the heart from the chest walls is a physical impossibility, unless it be supposed that at each diastole, the lung pushes between the heart and the chest walls—which observation clearly proves that it does not do.

3rdly. *The contraction of the aorta and pulmonary artery on the blood within them, and the impulsion of the blood against the semilunar valves.*—Theoretical considerations and experimental observations alike favour the belief that, under ordinary circumstances, the contraction of the vessels is noiseless; and inasmuch as there is nowhere any vacuum in the heart or great vessels, and consequently there can be no dashing of blood, but only a greater or less degree of pressure, it is difficult to conceive how the impulsion of the blood against the semilunar valves can be productive of sound.

4. *The sudden closure of the semilunar valves.*—This unquestionably is the principal and, as I believe, the sole cause of the second sound of the heart. Direct experiment and clinical observation alike attest this fact. As soon as the entrance of blood into the heart is stopped, and the valves therefore cease to set, the second sound ceases also. Readmit the blood and the sound instantly recommences.* Again, insert

* This I can testify from personal observation. For full details see Dr. Halford's experiments, as detailed in his pamphlet 'On the Action and Sounds of the Heart.'

the nozzle of a pair of bellows into the aorta, and cause the valves to close suddenly by air instead of blood, and a sound closely resembling the second sound is produced.* Again, hook back one or more of the semilunar valves so as to prevent them from closing the orifice of the vessel, as in the experiment detailed by Dr. Williams, and the second sound ceases, and is replaced by a murmur. Additional proof is also furnished by regurgitant disease of the aortic valves. When these valves are so damaged as not to close the orifice of the vessel, but to admit of regurgitation, the natural second sound disappears at the aortic orifice, though it continues as before at the pulmonary orifice. More decided proof can hardly be conceived.

Thus, then, to sum up the conclusions I have formed respecting the sounds of the heart in health, I would state—

1st. That both sides of the heart contribute to the production of the two sounds.

2ndly. That the synchroniety of the causes which give rise to the first and second sounds respectively on the two sides of the heart lead to a blending of the sounds generated on the two sides, so that two sounds only are heard accompanying the heart's action instead of four.

3rdly. That the first sound as heard on the chest walls is produced wholly by the sudden tension of the auriculo-ventricular valves, but is modified under certain conditions by the violent contraction of the muscular structure of the heart.

4thly. That the second sound is referable to the sudden tension of the semilunar valves.

5thly. That the differences of character observable between the first and second sounds are due to differences in the structure, position, and attachments of the auriculo-ventricular and semilunar valves respectively, the former being deep-seated and connected with tendons and muscles which, equally with themselves, are thrown into vibration by the tension to which they are subjected, whereas the latter are free from muscular and tendinous attachments, and have not the thick muscular walls of the heart interposed between them and the ear of the observer.†

* See experiments by Mr. Brakyn, reported in 'Lancet' for 1851.

† Skoda speaks of a ventricular first sound and an arterial first sound; of a ventricular second sound and an arterial second sound, and makes a distinction between the causes of the sound in the several instances. I am satisfied that the distinction has no foundation in fact, and that the first sound when normal is of ventricular, and

Thus, then, it will be obvious that circumstances which affect the action of the heart and arteries must lead to modifications in the intensity of the two sounds, and not unfrequently to alteration in their quality, pitch, and rhythm. And this holds good when the heart is healthy, as well as when it is structurally diseased.*

The first sound will be loudest in those cases in which the supply of blood is moderate and regular, the nervous influence in full force, the muscular structure of the heart thin but powerfully contractile, and the cavities of the ventricles and the auriculo-ventricular valves relatively large—conditions under which the ventricles contract forcibly, the tension and vibration of the auriculo-ventricular valves is very great, and the extent of sound-generating surface is at its maximum. Under the same conditions, the second sound will be the loudest when the current of blood is forcible, and the elasticity of the arteries great; for these are circumstances which lead to an unusually strong recoil of the blood on the semilunar valves, and to their more than usually forcible closure and vibration. Thus, in children in whom these conditions exist, the sounds are more intense than in adults, in whom the structures have lost some of their elasticity, and in whom the walls of the heart and chest are thicker, and therefore less favourable to the transmission of sound. But under no circumstances is increased intensity of the sounds observed more strikingly than when the patient is under the influence of nervous excitement. In this case the sounds are remarkably increased, the first sound more especially so, and to such a degree may this increase proceed, that the pulsation of the heart may be audible even by bye-standers. Flatulence and acrid secretions in the stomach and bowels may have a similar effect, and certain morbid

the second of arterial origin. The extraordinary readiness with which sound is propagated through fluid would hardly be credited by those who have not tested it experimentally, and in itself it affords a sufficient explanation of the propagation of the first or ventricular sound along the arteries, and of the second sound downwards into the ventricle. The rare cases which are cited as evidence that "the origin of the second sound is to be found in the ventricle," admit of another explanation, as see *ante*, pp. 478-9.

* Causes external to the heart itself may influence the transmission of the sounds to the ear, and thus may modify their intensity as heard on the chest walls; but even admitting, for the sake of argument, that the patient's posture is the same, that the structures which intervene between the heart and the chest walls are in precisely the same condition, and, in like manner, that the walls of the chest are of uniform thickness and texture, and, therefore, are possessed of the same conducting power—admitting these facts, the sounds will be found to vary according to the circumstances above stated.

poisons in the blood may exercise a similar influence, whilst all depressing and enervating diseases which weaken the heart, impair the nervous force, or render the blood less stimulating in its character, diminish the intensity of the sounds—of the first sound, by lessening the force of the ventricular contraction, and consequently, of the tension and vibration of the auriculo-ventricular valves; of the second, by diminishing the force with which the blood is driven into the arteries, and with which the artery contracts, and, therefore, by proportionably lessening the force with which the blood recoils on the semilunar valves.

But there is yet another circumstance, irrespective of disease in the heart itself, to which special reference should be made, in regard to its bearing on the intensity of the sound; I refer to the freedom with which the blood circulates through the heart. When the quantity of blood is large, or any impediment exists to the cardiac circulation, so that an excessive quantity of blood accumulates in the heart, the ventricle contracts forcibly but fruitlessly, to propel it, and a dull confused sound results from the violent, yet imperfect muscular contraction. The character of the ventricular sound is modified accordingly, and the sound is obscured and no longer clear. When, on the contrary, the quantity of blood is small, and no impediment exists to its circulation, the ventricles contract naturally, and the first sound is heard clear and distinct. This is often exemplified by the result of venesection and profuse hæmorrhage. Under the same circumstances the contractility of the artery is more than ordinarily excited, and the second sound is heard clearer than usual, and of higher pitch.

In disease of the heart these modifications of the sounds are met with in every variety. In dilatation of the heart, with moderate hypertrophy of its walls, the conditions necessary for the production of an intensely loud, though not a particularly clear first sound, are developed to the greatest degree; the heart is more extensively in contact with the anterior walls of the chest than usual; the ventricles are capacious; the valves are developed to a corresponding extent; and the muscular structure is strong enough to contract forcibly on the contained blood, and yet not thick enough to prove a serious impediment to the transmission of sound to the ear. In regard to the second sound, the same causes are in operation. The enlargement of the ventricles leads to a corresponding increase in the size of the arterial orifices, and of the valves which close them, whilst the force of their contraction leads to an unusually forcible propulsion of the blood, and to a corresponding

increase in the force of its recoil on the semilunar valves, with a consequent increase of vibration and sound. In simple dilatation, when not accompanied by such an amount of fatty degeneration of the muscular structure of the heart as to impair the contractile power of the ventricles, the conditions exist which are favourable to the production of a loud and unusually clear first sound. The heart is more than ordinarily in contact with the chest walls, the cavities of the ventricles are large, the valves consequently large; the contractile power of the ventricles is considerable, and their walls are thin, and in no way calculated to impede the transmission of sound to the ear. Therefore, as might have been expected, the sound is not only louder, but clearer and more abrupt--more distinctly valvular than in health. The second sound is also loud, in consequence of the size of the semilunar valves, and of the column of blood which reacts upon them.

In simple hypertrophy without dilatation, the walls of the heart are abnormally thick, and the cavities of the ventricles, therefore, relatively contracted; the play of the valves is unusually small, and the quantity of the blood to act upon them is also small, whilst the density of the muscular structure deadens the vibrations and prevents their transmission to the ear. Therefore at the apex, where, if it were due to muscular contraction, the first sound should be loudest, it is often inaudible; whilst it is audible over the aorta and pulmonary artery in consequence of its being transmitted along the current of the blood.* Even then, however, the sound is comparatively weak. In like manner, the smallness of the current of blood ejected from the ventricles at each systole of the heart leads to a comparatively weak recoil on the semilunar valves, and so to a diminution in the intensity of the vibration, and of the loudness and clearness of the second sound.

In atrophy of the heart, when the heart is small and the walls are thin and weak, the first sound is clear, but short and feeble, in consequence of the small size of the auriculo-ventricular valves, and of the diminution of force with which the vibration consequent on their closure takes place. The same holds good in respect to fatty degeneration and softening, fibrous or other infiltration, and whatever tends to impair the muscular power of the heart or lessen the extent of surface or the play of the valves, without at the same time increasing the thickness of the cardiac walls. Nevertheless in all these cases the first sound is loudest at the apex.

* See notes, pp. 476-7.

Serum or lymph effused into the pericardium have the effect of obscuring the intensity of the sounds, by rendering the source of their production more distant from the surface and interposing an indifferent conducting medium.

The quality and pitch of the sounds are also prone to undergo alteration. *Cæteris paribus*, the first sound becomes duller, and its pitch is lowered in proportion to the thickness of the ventricles and the rigidity or imperfect elasticity of the auriculo-ventricular valves; whilst, on the other hand, it becomes clearer, and its pitch is raised in proportion to the thinness of the ventricles and the elasticity of the auriculo-ventricular valves. The quality of the sound, however, undergoes innumerable variations, according to the precise texture of the heart, the condition of its valves, and the state of the blood and the nervous influence, and it is impossible to connect many of these slight gradations of sound with the structural and functional changes from which they originate. The most remarkable changes are those already alluded to, viz., the dull, muffled, low-pitched sound which results from extreme hypertrophy of the heart; the clear and often high-pitched sound which is produced by dilatation of the heart; and the knocking character which is imparted by nervous excitement. To these, perhaps, should be added a peculiar clanging character which is sometimes observed when the heart is irritable and the free edges of the valves are indurated.

The second sound becomes dull and has its pitch lowered by want of contractility in the arteries, and also by thickening and impaired elasticity of the semilunar valves. In certain instances in which the elasticity of the arteries is great, and thickening or rigidity affects the free edges of the valves, the sound assumes a clanging character.

The duration of the sounds also varies in strict conformity with the physical changes which occur in the condition of the heart and great vessels. Hypertrophy of the walls of the heart increases the duration of the first sound by lengthening the period which the ventricle occupies in contracting; and an undue accumulation of blood in the heart, whether caused by general plethora or by obstruction at the aortic orifice, produces a similar result, by rendering it a slow and difficult operation for the heart to expel its contents. On the contrary, simple dilatation or atrophy, or any other circumstance which weakens the heart, renders the first sound short, by impairing the contractile powers of the ventricles, and so lessening the vibrations caused

by tension of the valves. The duration of the second sound is lengthened by anything which impairs the contractility of the arteries, and also by thickening of the semilunar valves; it is shortened by extreme irritability of the arteries, and by an attenuated condition of the valves.

The duration of the first interval of silence is increased by whatever disproportionately shortens the first sound of the heart, and also by whatever impairs the elasticity of the arteries, and thus leads to delay in the recoil of the blood on the semilunar valves, or, in other words, in the commencement of the second sound; the duration of the second interval of silence, by whatever obstructs the auriculo-ventricular orifices, and thus, by causing delay in the filling of the ventricles, necessitates a longer delay than usual before the ventricles are again ready to contract. In most instances in which the pulsations of the heart are greatly reduced in frequency, the second interval of silence is disproportionately prolonged, and constitutes the principal auscultatory feature of the case.

But the rhythm of the heart's sounds may be even more seriously disturbed. Not only may one or other of the sounds or intervals of silence be inordinately prolonged or shortened, but there may be even a pause in the action of the heart, so that both sounds may cease during the period occupied by one entire action of the heart. When this occurs the sounds are said to intermit. In many instances this intermission of the sounds appears to follow some definite law; for it takes place at regular intervals,—as, for example, every fourth, fifth, or sixth beat. In other cases the intermission is irregular in its occurrence, taking place, perhaps, after two or three pulsations, then after six or seven, and then, again, perhaps, after nine or ten. Sometimes the sounds of the heart are regular, both in rhythm and intensity, during the interval which occurs between the intermission; at others they are regular in succession, but irregular in intensity, one or more weak sounds being succeeded by others comparatively loud; but more commonly they are irregular both in rhythm and intensity, each contraction of the ventricles differing from the preceding one in force and duration.

Another form of abnormal rhythm is connected with an alteration in the number as well as in the duration of the sounds of the heart. Sometimes one sound is altogether absent either at the base or the apex—so that a single sound only is heard in one of those situations. If the first sound is at fault, its absence is due to one or more of the causes already

specified, as contributing to weakness of that sound. If the second sound is inaudible at the base, the fact is explicable by the existence of one or more of the conditions which have been already pointed out as occasioning weakness of that sound, or else by its being masked by a prolonged systolic sound or murmur. In the first case, mental excitement or active exercise, by stimulating the action of the heart, will usually render the sound audible; and, in the second, the sound, if inaudible at the base, will be heard at the right apex. In some instances a triple sound, due to the reduplication of the systolic or the diastolic sound is heard, instead of the ordinary double sound; and in others four sounds, consequent on the reduplication of the first and second sounds, are heard within the period of one entire action of the heart. Indeed, reduplication, as it is termed, of the sounds of the heart, may exist in every possible variety. It may be audible at one spot, inaudible at another; it may exist at one moment, and may disappear the next; it may affect the first sound at one time, and the second shortly afterwards. In all cases its essential cause is the absence of synchronous action in the valves of either or of both sides of the heart. Thus, if one ventricle contracts before the other, the auriculo-ventricular valves on the two sides of the heart will not close simultaneously, and the first sound will be reduplicated; if the elasticity and irritability of the pulmonary artery and the aorta be unequal, the one vessel will probably contract before the other, and thus cause a want of synchronism in the closure of the semilunar valves on the two sides of the heart.

But in some instances the reduplication appears to be confined to one side of the heart; for a double first sound may be audible at the left apex and not at the right, or a double second may be audible at the aortic cartilage, and not at the pulmonary, or *vice versâ*. In these cases there is a want of synchronous action between the different segments of each set of valves—of the mitral or of the tricuspid, as the case may be, in the first instance; and of the aortic or of the pulmonary semilunar valves in the second instance.

Thickening and want of elasticity in one set of valves has been thought capable of occasioning reduplication of the sounds; but, although theoretically, the explanation seems probable, I very much doubt it being founded on fact. Certain it is, that reduplication of the sounds is at least a rare attendant on valvular mischief; whereas, it is of constant occurrence in cases which are unaccompanied by valvular dis-

ease, whilst its frequent intermission, and its appearance and disappearance under different conditions of excitement, seem to point to perverted irritability of certain portions of the heart as its essential cause.

It might be supposed that the altered rhythm of the heart would be indicated by the state of the arterial pulse, inasmuch as in a normal state, each systole of the ventricles is followed by a corresponding pulse at the wrist. And such in most instances is found to be the case. Each systole of the left ventricle, however irregular in point of time and force, is ordinarily followed by a precisely similar irregularity in the arterial pulse. But cases are met with occasionally in which this correspondence between the ventricular systole and the arterial pulse altogether fails; two complete actions of the heart, two ventricular systoles, giving rise to only one radial pulse.* This occurs as well when the rhythm of the heart is regular as well as when it is irregular, and is due to the fact that one of the two systoles of the ventricles is too feeble to cause pulsation in the radial artery.

Extreme irregularity both in the rhythm and intensity of the sounds of the heart is observed in many forms of cardiac disease, and also in persons who are in apparently good health, and in whom physical examination of the heart during life, and inspection of its structure after death fails to discover organic disease. Nay more, there are so many persons who have had an extremely irregular or intermittent pulse for years without perceiving the slightest indications of ill health, that experience compels us to doubt whether much practical significance can be attached to it. My own impression is that in many instances it is more closely connected with perverted innervation of the heart than with organic disease of its valves or muscular structure, and that in such cases life is not seriously compromised by its existence. When it does occur in a marked degree in connection with disease of the heart, the conditions which are ordinarily found to be present, are, fatty

* A case of this kind, in the person of a young woman named Cross, aged thirty-six, is at present (December 11th, 1861,) under my care in the Roseberry Ward of St. George's Hospital. The least exertion or mental excitement will rouse her heart's action sufficiently to make each systole felt at the wrist. The most remarkable instance I ever met with was one which I saw in consultation with Dr. C. J. B. Williams and Mr. Gardner, of Gloucester Terrace. In that case—probably one of fatty degeneration of the muscles, though we were not permitted to verify the fact after death—the pulse for nearly three weeks varied between 28 and 34, whilst the heart was heard contracting between 60 and 70 times in the minute. Occasionally there was only one pulse of the radial artery to three contractions of the ventricle.

degeneration and softening of the tissue of the heart, constriction of the mitral orifice, and mitral regurgitation, rupture, or acute ulceration of a valve, or of the chordæ tendineæ, or the presence of fibrinous coagula in the cavities of the heart. It may occur, however, in any form of cardiac disease, just as it may, apparently, in cases unconnected with structural alteration.

But auscultation informs us of other changes in the sounds accompanying the action of the heart, besides those connected with their intensity, pitch, duration, and rhythm; it makes us aware of adventitious sounds, which are superadded to, or entirely replace the ordinary sounds. These sounds are termed murmurs, and may arise either within the heart and great vessels, or external to them.

Those which arise within the heart are termed endocardial murmurs, the seat of their production being the cavities of the heart, or the commencement of the great vessels, whilst those which originate from mischief external to the heart itself, are termed exocardial or pericardial murmurs, the seat of their production being the pericardium, the external covering of the heart. The former invariably result from an eddy in the current of the blood capable of producing sonorous vibrations; the latter from the attrition of the two roughened surfaces of the pericardial membrane.

Endocardial murmurs are always of a more or less "blowing" character; but their quality may be simply blowing, or it may be rasping, grating, purring, whistling, or cooing, and in some instances distinctly musical. Practically, however, the precise quality of the murmur is of little importance, for the same cardiac lesion may give rise to murmurs of different qualities, according to the condition of the blood, the force of the heart's action and other modifying influences. A murmur which is loud and rasping when the heart is acting violently, may be soft and simply blowing, or may even disappear when the heart acts gently. Their pitch varies greatly, for whilst, as pointed out by Dr. Hope, the lowest pitch murmurs may be aptly represented by the word "who," pronounced in a whisper, the highest may be more nearly imitated by the letters S S pronounced in the same manner. They also vary much in their apparent distance from the surface, and likewise in their force and duration, being at one time loud and prolonged, at another weak, and of shorter duration. They vary also in the period at which they occur in relation to the sounds of the heart. Thus a murmur may be synchronous with one of the sounds of the heart, and

of equal duration with it, so as to obscure it in some instances, and in others to mask it and render it inaudible; it may even be prolonged over the succeeding sound, and may obscure that also if it does not mask it altogether; but, on the other hand, it may be of short duration, and audible only at the commencement or at the close of the sound, which may be clearly distinguished in addition to the murmur. When once developed, these endocardial murmurs are usually persistent, except when the heart's action becomes very feeble, as at the approach of death, or during syncope or extreme exhaustion, in either of which cases they often become intermittent, or may even cease altogether, in consequence of the non-occurrence of the vibration necessary to their production. *Cæteris paribus*, they are more intense the greater the force of the current and the larger the volume of blood propelled past the obstruction; and if they disappear under any of the circumstances above referred to, they will usually be reproduced by anything which accelerates and increases the force of the heart's action, whether it be mental emotion or bodily exercise. For when a cause of murmur exists, the murmur may not only become soft or inaudible, when the heart's action becomes slow and feeble, but it may increase in intensity under the influence of an excited circulation and increased force in the action of the heart.

Endocardial murmurs are synchronous or nearly so with the ventricular systole, or the ventricular diastole, and hence are sometimes termed respectively systolic or diastolic murmurs, but inasmuch as in some instances they arise from the presence of disease which impedes the onward flow of blood, and in others from disease of the valves which admits of the blood passing back through the auriculo-ventricular, or the arterial orifices, they are sometimes termed respectively obstructive and regurgitant murmurs.

But they may be not only systolic or diastolic and obstructive or regurgitant, they may be organic or else functional in their origin—in other words, they may be due to organic disease of the heart or great vessels, or to simple alteration in the quantity and quality of the blood, combined with functional derangement of the circulatory apparatus. In either case they are essentially connected with the production of an eddy in the current of the blood—a phenomenon which alone is capable of producing vibration such as that of which murmurs are the natural result.

Now, as eddies capable of producing sonorous vibrations do not exist

in a healthy state of the heart and great vessels, the presence of murmurs is a sure indication of some abnormal condition productive of eddies and vibrations. Practically, therefore, a murmur may be regarded as an audible announcement that something has occurred to roughen the surface of the endocardium or the internal coat of the great vessels, to constrict the great vessels or the orifices of the heart, to affect the elasticity of the great vessels, or to render the valves of the heart inefficient, so that they close imperfectly, or admit of regurgitation of the blood.

The mischief may be permanent or of temporary duration, of a structural or of a functional nature, and it may be either external to the organs of circulation or within the cavities of the heart or arteries. If the cause of endocardial murmur be external to the cavities of the heart and great vessels, it must be of an organic nature, and must produce its effect by exerting pressure upon them.

Thus, inflammation of the pericardium, when very acute and of long duration, is apt to impair the elasticity of the coats of the arteries, and, by the exudation which attends it, may lead to constriction of the great vessels at their origin. So, again, malformation of the chest walls and tumours in the thoracic cavity, by compressing the heart or the great vessels, may alter their shape, and excite undue vibrations and murmurs; and so also may the pressure of consolidated lung on the pulmonary artery or some of its branches, and of enlarged bronchial glands or any other intrathoracic tumour.

Endocardial murmurs, due to organic or structural mischief within the organs of circulation, are very numerous. They may be attributable to malformation of the heart, and to the consequent meeting of conflicting currents of blood, to disease in the valves, the chordæ tendinæ, or the papillary muscles, or in the orifice of one of the great vessels, or in the great vessels themselves. The mischief in each case may be of a different nature. The more common causes of endocardial mischief, are, 1st, inflammation, which produces vascular injection of the valves and other parts of the endocardial membrane, followed by infiltration of their texture, and by the deposition of fibrinous granulations on their surface—changes which usually lead to permanent thickening, rigidity, and contraction of the valves, and of the tendinous cords, or to roughening of their surface, but which may result in the cohesion of two or more adjacent tendinous cords, or of two valves, or of two parts of the same valve, by means of coagulable lymph, or even in the softening and

ulceration of the parts, and so to the rupture of a valve or a tendinous cord. The most frequent results, therefore, of this form of disease, are insufficiency of the valves, or roughening of their surface, with constriction of the orifices of the heart, and consequent obstruction to the circulation. No diseases are more provocative of this form of mischief than acute rheumatism and Bright's disease of the kidneys. 2ndly. Chronic degeneration of structure, whereby atheromatous or calcareous matter is deposited in the valves, or in the coats of the artery, leading to impaired contractility of the artery, and to thickening and roughening of the surface of both valves and vessels. 3rdly. Enlargement and dilatation, whether aneurismal, or otherwise, of the walls of the heart, whereby the orifices of the great vessels often become enlarged to such a degree that the valves prove inefficient to close them, or the papillary muscles so much affected as to lose their regulating power over the auriculo-ventricular valves. 4thly. The presence of coagula in the cavities of the heart—a cause, which, presumably, never comes into operation except towards the close of life, when the circulation is slow and languid. 5thly. The sudden rupture of a valve, in consequence of a blow or some violent straining effort.

Drs. Blakeston, Walshe, and others, maintain that dilatation of the ventricle, by rendering it "more spherical and less convergent to its arterial outlet," may cause misdirection of the blood-current, and thus excite murmur. But the idea is founded entirely on misapprehension. If the ventricle at the moment of contraction were partly empty, then might conflicting or eddying currents occur, and the theory would be entitled to some consideration. But, inasmuch as the idea of a vacuum in the heart during life is a simple absurdity, the cavities of the heart being necessarily at all times filled with blood to the full extent of their dilatation, it follows that contraction of their walls can have no other effect than to act upon the mass of blood within, and force it through any outlet which offers; and, inasmuch as the contracting or propelling force is exerted on all sides, and the current, if the auriculo-ventricular valves are sound, can only be in one direction, the shape of the ventricle cannot possibly exercise the slightest influence in the production of murmur. The murmur in the cases referred to is more probably referable to impaired elasticity and contractility of the artery, and to the eddying of blood consequent thereupon.

Endocardial murmurs, due to functional or inorganic causes, do not so readily admit of explanation. It seems impossible to doubt that

murmurs do sometimes arise in cases in which no structural mischief exists; for not only are they met with during the progress of rheumatism, typhus fever, and other disorders unattended by any general symptoms of cardiac disease, but they are often excited by hæmorrhage and other causes productive of anæmia and spanæmia. Further, they are known to disappear completely under circumstances which improve the general health and yet do not exercise any specific influence on the heart. A question, however, arises respecting their causation. It is commonly asserted that these functional murmurs are directly referable, in most cases, to an impoverished and spanæmic condition of the blood; and, in proof of this position, the facts are cited, that these murmurs often follow venesection or hæmorrhage, and are usually met with in pale, anæmic persons. There cannot be a doubt as to the facts themselves; but I am inclined to question the inference drawn from them. True that murmurs often arise under circumstances productive of anæmia and spanæmia, and that these murmurs are usually of functional origin, or, in other words, unconnected with structural disease. But it is equally true that functional murmurs are not unfrequently met with in florid persons, who present no appearance either of spanæmia or anæmia, and it is also true that they are often absent in anæmic persons, and even in persons blanched by hæmorrhage.* Further, it is certain that nervous excitement, which produces palpitation, will often given rise temporarily to a murmur in persons who are not spanæmic,† and that

* A case of this kind is at present, (January 7, 1862) under my care, in the person of Catherine Ward, aged fifty, who is in the Pepys Ward of St. George's Hospital. Although thoroughly blanched, as the result of profuse hæmatemesis, no murmurs of any kind accompany the sounds of the heart.

† This has been often forced on my attention during the examination of persons about to assure their lives. Only last month I met with a remarkable case in point. A gentleman, aged thirty-nine, presented himself for examination. His life had been refused at one office in consequence of his having a cardiac murmur, and he was extremely nervous. A systolic murmur intensely loud at the left apex, and also at the right and left base of the heart, led me in the first instance to consider that his heart was seriously diseased, and that his life was uninsurable. He declared however that he had not had a day's illness for above twenty years, and that he was not short-breathed, and never suffered from palpitation except when he was nervous; and as the area of præcordial dulness was not increased and the impulse of the heart was not of a heaving character, as it ought to have been had he suffered long from regurgitant mitral and obstructive aortic disease, but was rather of a knocking, nervous character, I thought it possible that the murmurs might be functional. He was therefore requested to come to me another day, and after a few interviews, when his nervousness had subsided, the murmurs entirely disappeared.

even in the individuals in whom this is observed, bodily exercise, though it produces increased action of the heart, and forcible impulse, may fail to excite a murmur.* Again, it is true that in many cases of chorea, in which there is neither anæmia nor spanæmia, a functional systolic murmur may occur both at the base and apex of the heart—a murmur, which, at the apex at least, cannot be referred to the condition of the blood, but must be attributable to regurgitation of blood through the auriculo-ventricular opening, and therefore must be connected with a disordered action of the apparatus which regulates the valve. If then, neither anæmia nor spanæmia, nor both combined, will necessarily produce a functional murmur; if functional murmurs may arise in cases in which neither anæmia nor spanæmia exists; and, further, if functional murmurs are met with which are obviously connected with irregular action of the valves, may not all functional murmurs be referable to irregularity in the action of the valvular apparatus, consequent on perverted innervation and disordered action of the elastic tissue of the valves? Any excess or deficiency in the elasticity of the tissue on which the action of the valves and the large vessels depends, would necessarily lead to an eddying of the blood, and so to the production of murmurs. It is certain that, even when spanæmia and anæmia exist, functional murmur is commonly confined to cases characterised by palpitation—a result of nervous irritability—and that profuse venesection, hæmorrhage, and other causes of spanæmia, are also productive of palpitation, and of exalted nervous excitability. Therefore, without denying that an anæmic and spanæmic condition of the blood must necessarily facilitate the production of vibration and murmur, it seems to me probable that these functional murmurs are more closely connected with perverted innervation and disordered contractility of the valves and large vessels than with a mere alteration in the condition of the blood.†

* The forcible propulsion of healthy blood by an hypertrophied heart has been stated by Dr. Walshe (loc. cit., sec. 1303) to be capable of generating direct murmur. The frequent, nay, the usual absence of direct murmur in cases of simple hypertrophy, however fully developed, is sufficient to negative the theory alluded to, and it is probable that the murmur which arises in some of the cases alluded to is connected with some irregularity in the action of the valvular apparatus, or with some deficiency in the elasticity or contractility of the aorta whereby an eddy is created in the current of the blood.

† The mechanism of these functional murmurs is rendered intelligible by Dr. Broadbent's recent dissections of the valves, whereby it is shown that a layer of elastic tissue enters into their composition, the fibres of which run chiefly in a direc-

But, whatever the mechanism of these functional endocardial murmurs may be, the practical question in regard to them, is how to distinguish them from organic murmurs. Their intensity, quality, and duration, are often appealed to as evidence of their functional origin; great loudness, harshness, and prolongation of a murmur, being regarded as proof of its organic nature, whilst feebleness, smoothness, and shortness, are cited as evidence of its functional nature. So again, persistency is often mentioned as a characteristic of organic murmurs, intermission, or variableness of functional murmurs. But however feasible these distinctions may appear in theory, they certainly do not invaribly hold good in practice. It is true that a murmur, *cæteris paribus*, will be loud in proportion to the amount of obstruction from which it arises; it will be harsh in proportion to the roughness of the injured surface, and prolonged in proportion to the extent of that surface; and, as the obstruction is usually greater, and the surface rougher and of larger extent in the case of organic murmurs than it is in the case of inorganic or functional murmurs, the intensity, roughness, and prolongation of a murmur afford *primâ facie* evidence of its organic nature. But it is equally certain that, *cæteris paribus*, a murmur will be loud in proportion to the force and velocity of the circulation; so that the most intense murmur may become slight, if it does not wholly disappear, when the circulation becomes languid; the roughest murmur may become smooth; the most prolonged murmur may become short. And, as it is impossible to measure precisely the relative influence of the various circumstances which at any given moment are modifying the character of a murmur, so it is impossible, from the mere character of a murmur, to draw a positive conclusion respecting the amount of organic changes which produce it, or even to decide whether it is of organic, or of functional origin. So also in regard to persistency. Organic murmurs are usually persistent, and functional murmurs are not so; but organic murmurs are extremely variable in their character, not unfrequently changing at

tion towards the free borders of the valves, and, which being on the auricular surface of the auriculo-ventricular valves and on the ventricular surface of the semilunar valves, has a constant tendency to close the valves by drawing the free borders of the valves towards their attached borders. (See 'Med.-Chir. Trans.,' vol. xliv, p. 45-6.) In a natural state of the tissues and circulation, this elasticity of the valves is simply sufficient to induce closure of the valves when unopposed by the current of the blood; but it is quite conceivable that any undue irritability of this elastic tissue might prevent the thorough closure of the auriculo-ventricular valves, and thus form an obstacle to the semilunar valves falling back evenly against the walls of the arteries.

each successive systole or diastole of the heart; and sometimes, under the conditions above referred to, they may for a time intermit or cease altogether, whilst, on the other hand, functional murmurs often persist uninterruptedly for a considerable length of time, and do not exhibit more changeableness of character than organic murmurs.

But, although the mere character of a murmur will not at once suffice to afford conclusive evidence as to the presence or absence of organic disease, certain circumstances often coexist, which, taken together, may be regarded as affording almost positive proof on the subject. Thus functional murmurs are always systolic, and therefore a diastolic murmur is necessarily of organic origin; they are much more common at the base than at the apex of the heart, and are usually louder at the second left costal cartilage, and thence in the track of the pulmonary artery, than they are at the second right costal cartilage and in the track of the aorta. Thus, if a systolic murmur, audible at the base of the heart be especially loud at the second right cartilage, and be scarcely distinguishable at the second left cartilage; if it be harsh in character, and persistent in duration, and if at the same time there be entire absence of venous hum in the neck, there cannot be a doubt as to its being referable to organic disease; for functional murmurs are never persistent, and are rarely harsh for any length of time; and systolic basic murmur connected with organic disease is almost always louder at the second right than at the left second costal cartilage, being due to disease at the aortic orifice, or in the aorta itself, and not to disease in the pulmonary orifice or in the pulmonary artery. Nevertheless, it must be confessed that it is often impossible at a single examination to determine whether a murmur is functional or organic, and the history of the case, and the course which the murmur has pursued, must be carefully inquired into, and repeated examinations had recourse to, before a positive opinion is given.

There are other functional murmurs which are obviously of arterial and not of cardiac origin, but which, nevertheless, may occasion some difficulty to the inexperienced auscultator. Thus, a murmur evidently of arterial rhythm is frequently heard under the clavicles, especially under the left clavicle. This murmur, which is sometimes audible during tranquil respiration, but more commonly after forced expiration and inspiration, has been referred by many authors to the pressure of a tuberculized and hardened lung, and has been cited as evidence of that form of disease. Nothing, however, can be more erroneous. Undoubtedly

its existence is due to pressure; and as no cause of pressure normally exists during tranquil respiration, its presence under these circumstances is very suggestive of tubercle. But the pressure requisite for its production may be exerted in perfectly healthy persons, sometimes by the fully inflated lung, sometimes by the forcible action of the respiratory muscles. Out of one hundred healthy persons who presented themselves before me for examination, for the purpose of life assurance, it existed on the left side alone in nine, and on both sides in three instances before the effort of blowing the spirometer, whilst after that effort had been repeated three times it was heard for a short time on that left side alone in fifty-six instances, and on both sides in nineteen instances. In other words, it was heard in no less than 75 per cent. of all the cases examined.

The points then which it is all important to ascertain, are, first, the existence or non-existence of a murmur; secondly, if it exists, whether it is referable to organic disease or functional derangement? These being determined, the next matter for decision is as to the precise part of the heart or great vessels from which the murmur arises. This brings us to a consideration of the various signs which indicate the seat of any given endocardial murmur.

It will be remembered,* that all the arteries of the heart are seated behind a portion of the chest-walls seldom exceeding an inch in diameter, and that the extremity of an ordinary stethoscope will therefore cover the entire space in which they are clustered. Further, it will be remembered that the four sets of valves do not occupy opposite corners of this space, but that, on the contrary, the pulmonary semilunar valves lie partly in front of the aortic valves, and the tricuspid valves in front of the mitral. It must be obvious, then, from the position of the valves, that little information is obtainable respecting the orifice which is the seat of murmur, by merely examining this particular region. If we wish to arrive at a decision on that point, we must endeavour to do so by noting the position of the murmur, the period of the heart's action at which it is heard, and the direction in which it is transmitted, and partly also by giving due attention to the concomitant general symptoms, and to the presence or absence of certain phenomena in the arteries and veins.

What, then, are the precise grounds for an opinion as to which of the four orifices of the heart is the seat of endocardial murmur? Before

* See part iii, chap. i, p. 451.

attempting to clear up this question, it may be well to make a few prefatory remarks respecting the different endocardial murmurs. Theoretically, each orifice of the heart may be the seat of an obstructive and a regurgitant murmur; and as there are four orifices of the heart—two on the right side, and two on the left—so there may be two obstructive and two regurgitant murmurs on each side of the heart, or, altogether, eight distinct murmurs; of these four are systolic and four diastolic, and their precise nature will be seen in the subjoined table:

Period of heart's action.	*Seat of murmurs.*		*Cause of murmurs.*
Systole	Left side of heart	Aortic	Obstruction to onward flow of blood through the aortic orifice or through the aorta.
		Mitral	Regurgitation of blood through the mitral valve into the left auricle.
	Right side of heart	Pulmonary	Obstruction to onward flow of blood through the pulmonary orifice or through the pulmonary artery.
		Tricuspid	Regurgitation of blood through the tricuspid orifice into right auricle.
Diastole	Left side of heart	Aortic	Regurgitation of blood through aortic orifice into the left ventricle.
		Mitral	Obstruction to the flow of blood from left auricle into left ventricle.
	Right side of heart	Pulmonary	Regurgitation of blood through the pulmonary orifice into right ventricle.
		Tricuspid	Obstruction to the flow of blood from right auricle into right ventricle.

Practically, however, murmurs on the right side of the heart are extremely rare, the left side being almost exclusively the seat of those varieties of disease by which eddies capable of giving rise to sonorous vibrations, are formed in the current of the blood; and even on the left side of the heart there is one murmur, viz., the diastolic or obstructive mitral murmur, which is of comparatively rare occurrence, the force with which the blood passes from the auricle into the ventricle being ordinarily insufficient to excite sonorous vibration. So that, without reference to any special physical signs, a murmur, if systolic, is almost certainly aortic or mitral; if diastolic, probably aortic.

Exceptions, however, are met with occasionally in respect to the non-occurrence of disease on the right side of the heart, and it is therefore necessary to consider in detail the signs by which the various murmurs may be severally recognised. The systolic murmurs first claim our attention.

1. *Systolic aortic murmur.*—The systolic or obstructive aortic murmur is produced by the vibrations resulting from an obstruction to the onward flow of the blood in its passage from the left ventricle through a roughened or narrowed aortic orifice, or a roughened aorta. It is usually high pitched, loud and prolonged, is heard of maximum intensity at midsternum on a level with the third intercostal space, and may be traced upwards along the course of the aorta towards the second *right* sterno-costal articulation, and not unfrequently is audible in the carotid and subclavian arteries, and on the left side of the spinal column, opposite the second, third, or fourth vertebræ. Its intensity decreases as it is traced towards the second *left* sterno-costal articulation, and as sonorous vibrations, are not easily transmitted in a direction opposed to the current of the blood, it loses greatly in force towards the apex of the heart. When it is heard of greater intensity at the second right sterno-costal articulation than over the aortic valves, it is partly, if not wholly, referable to disease of the arch of the aorta. This murmur, as already stated, is sometimes met with, as a consequence of functional derangement, and is not necessarily connected with organic disease. On the other hand, it is a frequent result of aneurism of the aorta, and may be almost certainly regarded as resulting from disease of the aorta, when it is not audible over the aortic valves, but is heard in some part of the chest where cardiac murmur would not ordinarily exist.

2. *Systolic pulmonary murmur.*—This murmur, which is caused by the vibrations resulting from an obstruction to the onward flow of blood in its passage through the orifice of the pulmonary artery, is of very rare occurrence as a result of organic disease, but not uncommon in connection with functional derangement. It is to be recognised by its superficial character, by its being of maximum intensity at the third left sterno-costal articulation, and extremely faint, if not quite inaudible at the second right or aortic cartilage, and by its being imperceptible on the back, in the track of the aorta, as also in the carotid and subclavian arteries.

3. *Systolic mitral murmur.*—This murmur, which is the commonest of all organic murmurs, is referable to the vibrations occasioned by the regurgitation of blood from the left ventricle through the mitral orifice into the left auricle. It is seldom high pitched, but it varies greatly in quality, loudness, and duration. It is heard of maximum intensity at or about the left apex, being transmitted along the chordæ tendineæ of the

mitral valve; it loses in force, if it be not altogether inaudible at the ensiform cartilage, or, in other words, at the right apex and also at the base of the heart; but it is often audible in the left axilla, and near the inferior angle of the left shoulder-blade. It partially obscures, or entirely replaces the first sound at the left apex, and, if very intense, it may do so even at the right apex; but, under ordinary circumstances, the first sound will remain clear, or only slightly obscured, at the right apex, and at the base, even when it is wholly replaced by a murmur at the left apex.

An excessively loud second sound, which is of greatest intensity at the third left sterno-costal articulation, is often heard in cases characterised by systolic mitral murmur. This, as was pointed out by Skoda, arises from the fact, that in consequence of the continued regurgitation of blood from the left ventricle, an impediment is caused to the onward flow of blood through the lungs; the pulmonary system of vessels thus becomes congested, the pulmonary artery thickened and dilated, and the right side of the heart dilated and hypertrophied. The blood, therefore, is driven with increased force into the pulmonary artery, which contracts with extraordinary energy, and causes more than usually forcible recoil of blood on the semilunar valves, giving rise to an abnormally loud sound. The existence, therefore, of greater intensification of the second sound at the left base than at the right,—at the pulmonary than at the aortic cartilage,—affords strong confirmation of the murmur at the apex being due to mitral regurgitation. It should be mentioned, however, that it is not of constant occurrence in connection with mitral regurgitation, inasmuch as a serious impediment to the pulmonary circulation must have long existed before the condition of the vessels is developed which is necessary to its production.

4. *Systolic tricuspid murmur.*—This murmur, when referable to organic disease of the valve, is of rare occurrence, though not so much so as the systolic pulmonary murmur. It is caused by the vibrations resulting from the regurgitation of blood from the right ventricle through the tricuspid orifice into the right auricle. It is usually soft and of low pitch, is heard of maximum intensity at or about the ensiform cartilage, and is almost inaudible at the left apex. It is generally accompanied by dilatation of the right auricle, and distension of the jugular veins, and not unfrequently by visible pulsation of these vessels at each systole of the ventricle. Pulsation of the jugulars, however, is not a necessary

consequence of tricuspid regurgitation; for it is not observed, unless the ventricular contraction is tolerably forcible, and the veins are so much distended as to render their valves inefficient; or, in other words, unable to prevent the reflux of blood. So also tricuspid regurgitation is not invariably accompanied by a murmur, as is shown by the fact that not unfrequently pulsation of the jugular veins is observed when no murmur exists at the right apex of the heart. Indeed, there are good grounds for believing that in many instances of tricuspid regurgitation the back current is not sufficiently forcible to give rise to murmur when the valves are healthy.

5. *Diastolic aortic murmur.*—This murmur results from the vibrations occasioned by the regurgitation of blood through the aortic orifice into the left ventricle, as a consequence of insufficiency of the aortic valves. The murmur is usually of a blowing character, of variable pitch and loudness and of long duration, is heard of maximum intensity at midsternum, opposite the third intercostal space, and it is usually transmitted to a greater extent along the great vessels than it is towards the apex. Nevertheless, when very intense, it is often audible at the apex, and, in some instances, it is more audible towards the right apex than it is towards the left—a fact which is probably explicable by the vibration being transmitted along the septum of the ventricles against which the force of the eddy caused by the regurgitating blood would be principally directed.

The murmur may entirely mask or replace the normal second sound, or it may be heard at its commencement, or towards its close. Most commonly, unless it be very intense, a distinct sound is heard superadded to the murmur towards the close of the diastole; and, if the stethoscope be shifted to the third left intercostal space, the clear sound resulting from the flapping down of the pulmonary semilunar valves may usually be heard. Sometimes, however, owing to the condition of the surrounding parts, an aortic regurgitant murmur is more distinctly audible at the left than at the right side of the sternum—at the pulmonary than at the aortic cartilage—and thus, without due attention, may lead to error.

Skoda believes that, when a distinct sound is superadded to the murmur, the murmur is not referable to regurgitation, but is due solely to friction of the blood against the coats of a roughened aorta during the systole of the vessel. But the fact that a diastolic murmur, accompanied by a distinct sound, as described by Skoda, is sometimes

heard when no systolic aortic murmur exists, appears to me to be fatal to his theory, as it is difficult to conceive that roughness of the vessel which would not occasion murmur during the rush of blood which accompanies the diastole of the vessel—in other words, during the systole of the ventricle—should do so during the systole of the vessel. Moreover, the same character of murmur exists when the aortic pulse and the pulsation of the carotids leave no room for doubt as to the existence of aortic regurgitation.

6. *Diastolic mitral murmur.*—This murmur is produced by the vibrations caused by an obstruction offered at the mitral orifice to the even flow of the blood from the left auricle into the left ventricle. It is variable in its duration, of low pitch, and seldom very loud; it is heard of maximum intensity at or about the left apex, and decreases in force towards the right apex and as it is traced upwards towards the base. It rarely masks, though it may somewhat obscure the second sound. It is not frequently met with alone, but usually exists in connection with systolic mitral murmur—a circumstance which is explicable by the fact that there can be few conditions of the mitral orifice which will cause obstruction to the onward flow of blood without interfering with the action of the mitral valves sufficiently to admit of regurgitation. It should be understood, however, that a mitral diastolic murmur is comparatively rare, even in cases of considerable constriction of the mitral orifice—a fact which is doubtless referable to the weakness of the stream in which the blood passes from the auricle to the ventricle. In this case, as in that of mitral regurgitation, the second sound of the pulmonary artery, as pointed out by Skoda, is apt to become exceedingly intense, in consequence of the thickening and increased force of the contraction of the pulmonary artery, which results from the obstruction to the pulmonary circulation.

7. *Diastolic pulmonary murmur.*—This murmur, which is produced by the regurgitation of blood through the pulmonary orifice into the right ventricle is quite a pathological curiosity. I have met with it in one instance only, but it was recognised during life by its position, by the direction in which it was heard, and by the condition of the radial pulse. In the instance referred to it was of a blowing character, low pitched and soft, and much prolonged. It was of maximum intensity at the third left sterno-costal articulation, and could be traced upwards to the second left cartilage, a little above which it suddenly disappeared. It was scarcely audible at the second right cartilage, was inaudible in

the carotid and subclavian arteries, and was not accompanied by the characteristic pulse of aortic regurgitation.*

8. *Diastolic tricuspid murmur.*—This murmur, which originates in an obstruction to the flow of blood from the right auricle into the right ventricle, is, perhaps, the rarest of all cardiac murmurs. I know nothing of it from personal experience, but theoretically it should be heard of maximum intensity at or about the ensiform cartilage, and should lose in force as it is traced towards the left apex and upwards towards the base of the heart. A diastolic murmur, inaudible at the base of the heart, very faintly audible at the left apex, but distinctly heard at the ensiform cartilage, might be fairly attributed to the tricuspid orifice.

We will now pass on to the consideration of murmurs which are unconnected with endocardial mischief, and originate in the pericardium or membranous envelope of the heart.

Exocardial or pericardial murmurs are produced by a mechanism altogether different from that which gives rise to endocardial murmurs. Closely applied to and investing the heart is a fibro-serous membrane, which, on reaching the root of the great vessels, is reflected on itself, and forms a close sac. This sac is termed the pericardium. On its internal surface it is lined by a smooth and glistening serous membrane, the two layers of which in their natural state glide smoothly and noiselessly over each other at each successive systole and diastole of the heart. But in disease the condition of the parts is changed, and their mechanism altered. Inflammatory exudation on the serous surface of the membrane, the deposition of tubercular or cancerous matter in the membrane itself or on its free surface, and the formation of coagula within its cavities as the result of the outpouring of blood from an aneurismal tumour, will, one and all, produce roughening and irregularity of its serous surface. And when this is the case, the movement which before was noiseless, at once becomes accompanied by the sound of friction, occasioned by the attrition of the two opposed and roughened surfaces. Sometimes the friction sound is of a grazing quality; it is more frequently rough, harsh, and rasping; sometimes it resembles the creaking of leather as it is heard in a new saddle in riding; but whatever its tone or musical variety, it has the peculiarity of being almost invariably a to-and-fro sound accompanying the whole of the heart's action, and superadded to or masking its natural sounds. Unlike the double murmur of valvular disease, which usually arises from permanent mischief, and therefore

* A similar case is recorded by Dr. Wilks in 'Trans. Path. Soc.,' vol. viii, p. 121.

is necessarily persistent, the to-and-fro sound of pericardial friction results from causes of a transient nature, and endures therefore only for a limited period. Arising, as already stated, from the rubbing together of the two roughened layers of the pericardium, it is necessarily put a stop to by anything which prevents the continuance of this rubbing. Hence, it may cease from one of two causes: either from adhesion of the two layers of the membrane by means of interposed lymph, or from their total separation by the effusion of a large quantity of serum. In the former case the cause of its cessation is a permanent one; consequently, when adhesion is firm and universal, the friction sound can never recur; in the latter it will probably reappear as the serum is reabsorbed and the roughened surfaces come again into apposition, and will cease only when permanent adhesion has taken place.

It sometimes happens that exocardial friction sound does not possess its distinctive character of a to-and-fro sound. Thus, when the two apposed surfaces of the pericardial membrane are thickly coated with lymph, and adhesion is beginning to take place, the freedom of motion necessary to the production of distinct friction sound ceases to exist. The sound of rubbing is then replaced by a peculiar continuous churning sound, resulting from the slight motion which still occurs towards the apex of the heart, between the two thickly coated layers of membrane; or, more especially around the roots of the great vessels, by a clicking sound produced by their occasional momentary separation—a sound which is only heard at intervals, inasmuch as its cause is not in operation at every action of the heart.

But there is yet another condition under which the friction sound produced by exocardial disease might not be recognised if its to-and-fro character were alone appealed to. Cases are not uncommon in which pericardial friction sounds may be heard during the systole of the heart alone,* or even, though this very rarely occurs, during the diastole alone, in either of which cases the sound, being synchronous with the valvular sounds, may simulate endocardial valvular murmur.

Even when a double sound, the characteristic to-and-fro sound, exists, it may be so nearly synchronous with the normal valvular sounds of the heart, and in character may approximate so closely to a double valvular murmur as to render it difficult to decide whether it be of

* For a remarkable case in point, see the records of the case of Peter Broad, in 'Hospital Case-book' for November, 1861, and 'Post-mortem Book' for February, 1862.

exocardial or endocardial origin, or, in other words, whether it be a pericardial friction sound or an endocardial valvular murmur.

By what means, then, can a decision be arrived at? There is no one character by which the true source of the murmur can be recognised; but there are several circumstances which, viewed collectively, will rarely fail to lead to a correct diagnosis. Thus, whilst exocardial murmurs are generally of a rubbing quality, endocardial murmurs are essentially of a blowing character. Exocardial murmurs usually convey the impression of being more superficial in situation than endocardial murmurs; their seat of maximum intensity does not correspond with that of endocardial murmurs; their force does not vary, as they are traced from point to point after the manner of endocardial murmurs; but, on the contrary, they are often limited to one spot, and they are not propagated along the course of the great vessels as are some of the endocardial murmurs. They are apt to vary from day to day, or even from hour to hour in their character, intensity, and position, and not unfrequently are more marked when the patient leans forward, or, if the patient be young and the cartilages of the ribs elastic, when forcible pressure is exerted on the chest walls by means of the stethoscope. They continue throughout the whole period of the heart's action, and therefore, are seldom perfectly synchronous with the sounds of the heart, but appear to begin before, or else to follow at an appreciable interval after the ventricular systole, and they are apt to cease, after a time recommence, and ultimately disappear in a manner which is never noticed in the case of murmur resulting from endocardial mischief. The heart's sounds are more muffled and appear more distant than in the case of endocardial disease, and the lungs being pushed aside by the distended pericardium, the space over which the respiratory sounds are inaudible is wider than when the heart is hypertrophied, as a result of valvular mischief.

Thus, then, it will be admitted that auscultation alone affords many means of distinguishing between exocardial and endocardial murmurs, and when to these are added the existence of friction fremitus, of dulness on percussion in the pericardial region, and of other signs which other methods of examination enable us to recognise, it will be apparent that in the majority of cases inattention alone can lead to difficulty in diagnosing the true nature of the murmur. Nevertheless, when the murmur is single and confined to the systole of the heart, or is simply clicking in its character, the diagnosis is extremely difficult, and, at a

first examination, is almost impossible. But even in these cases the variableness of the signs observed day by day cannot fail within a very short time to point to a correct conclusion.

Pericardial friction varies greatly in duration. It may cease after it has existed for a few hours only, in consequence of adhesion of the two roughened surfaces, or it may endure for many weeks if not for months, especially about the root of the great vessels.* More commonly it continues for several days, is then checked by the outpouring of serous exudation in amount sufficient to separate the two layers of the pericardium, and when it reappears after the absorption of this fluid it ordinarily continues about a week or ten days.

Pericardial friction may be limited to one portion of the pericardial membrane or may extend over its entire surface, and the area over which the friction sound can be heard will vary in a corresponding degree. When the entire surface is affected and has only recently become so, the friction sound is usually heard of maximum intensity towards the apex where the heart is uncovered by lung; but when inflammation has existed for some time and has led to copious effusion into the pericardium, not only are the edges of the lungs pushed aside, but, if the patient be in a semi-recumbent posture, the fluid will gravitate towards the apex of the heart, and under those circumstances the friction sound will be heard of maximum intensity at the base.

It has been attempted to determine the amount and nature of the effusion into the pericardium by reference to the extent and character of the murmur. But observation has proved the impossibility of doing so. The friction sound is more influenced by the energy of the heart's action, and by the extent of movement between the two surfaces of the pericardium, than it is by the mere amount or character of the exudation. So, when the heart is contracting vehemently, a slight amount of exuded lymph will often occasion more intense friction sound, than a much larger quantity of exudation when the systole of the heart is weak.

In some instances, a murmur referable to pleural friction is caused by the action of the healthy heart, and being necessarily cardiac

* Dr. Walshe (loc. cit., p. 258) reports a case in which friction sound was audible at the "lower part of the sternum" for upwards of three months, "long after the man's discharge from hospital and apparent restoration to health." I have never met with such a case, nor can I find a record of any other; and I am inclined to doubt whether in the instance referred to by Dr. Walshe the sound may not have been of endocardial origin.

in rhythm, it is apt to be mistaken for pericardial friction sound. But there are several circumstances which ought to excite suspicion as to its origin, and when suspicion is aroused, the diagnosis is seldom difficult. Thus, the murmur does not extend over the præcordial region, but is limited to its confines, and usually remains fixed about its left border; it is commonly accompanied by the signs of pleurisy at the back of the chest, and it ceases, or nearly so, when the patient holds his breath—an act which does not check the continuance of pericardial friction.

Venous Murmurs.

The larger veins, more especially the large veins of the neck, are apt to become the seat of murmur of a low pitched humming character. This was first pointed out by Dr. Ogier Ward, and is now generally admitted. Nevertheless, Skoda, Kiwisch, and other observers, have at various times maintained that venous hum originates in the artery—a circumstance which is the more curious, because the fact of its being connected with the veins, admits of easy proof. Thus—1st. The least pressure on the jugular vein, above the point where the stethoscope is applied and the murmur is heard, causes the murmur to cease immediately. 2ndly. The venous murmur, which is a continuous murmur, and the arterial murmur, which is intermittent, sometimes coexist in the same patient, and may be distinctly recognised at the same spot and at the same moment; indeed, in a large proportion of cases, the venous murmur may be heard coincident with the normal sound of arterial pulsation. 3rdly. In some instances, the murmur may be heard along the superior longitudinal sinus, especially towards the torcular Herophili, where there is no artery, and, therefore, cannot be an arterial sound. 4thly. The murmur in the neck is increased in intensity by any cause which accelerates the flow of blood through the jugular veins, and is weakened, or becomes inaudible when the venous current is retarded or arrested. Thus it is increased by an erect posture, and by the act of inspiration, which draws the blood on towards the right side of the heart; it is weakened by a recumbent posture, and lessened or absolutely arrested by any disease which leads to turgidity of the jugular veins with partial arrest of the circulation through them—causes which do not materially influence the production of an arterial sound or murmur.

It must be admitted, then, that the veins are sometimes the seat

of murmur; but that they are not so normally, is evident from the fact that venous murmur does not always exist, and in the majority of persons cannot be heard, except under the influence of pressure by the stethoscope.* It may be desirable, therefore, to investigate the character of this murmur, and the causes from which it originates.

In its character it differs remarkably from an arterial murmur, and therefore, in a diagnostic point of view, is not likely to occasion difficulty. Instead of being intermittent, and of a high pitch, as arterial murmurs usually are, and synchronous with the systole, or with the diastole of the heart, or double, accompanying both of these actions, the venous murmur is always single, continuous, and of a low humming, cooing, or roaring character, somewhat resembling the whispered pronunciation of the word "who." It varies in intensity from one moment to another, and, in certain instances, is not only marked by a regular increase or swell, which is synchronous with the heart's systole, and is probably dependent on the pressure of the contiguous artery, but it rises at irregular intervals, almost to the character of a musical tone.

Sex appears to exert little influence on its occurrence. It is met with in men almost as frequently as in women; and though most common in childhood and the earlier periods of life, it may show itself at all ages, under conditions favourable to its existence. It is most common in the jugulars, especially in the right external jugular, and in the subclavian and innominate veins; but it may be sometimes heard in the superior longitudinal sinus, and is often present in the femoral and other distant veins. Dr. Herbert Davies† and

* Dr. Herbert Davies, who is an advocate for the extreme frequency of venous murmur, is constrained to admit that "the sound will be observed only in certain positions of the neck, and with certain degrees of pressure to be found by repeated trials." (Loc. cit., p. 355.) The necessity, however, for such positions and such pressure is all I contend for. No one would deny the possibility of inducing a murmur by well-regulated pressure, whether in an artery or in a vein, provided only the circulation be sufficiently active; but I would maintain that neither in the one nor in other does a murmur exist without the exercise of pressure, or the existence of some cause calculated to excite an eddy in the blood.

† Loc. cit., pp. 355-358. Dr. Davies reports venous murmur to have been present in 765 out of 802 children from fourteen months to fifteen years of age, or, in other words, in about 95 per cent.; in 129 out of 150 healthy males between the ages of seventeen and twenty-seven, or, in other words, in about 86 per cent.; in 46 out of 53 healthy females between the ages of sixteen and twenty-eight, or, in other words, in about 86 per cent.; and in 8 out of 67 persons of both sexes between the ages of fifty and ninety, or in about 12 per cent.

Winterich,* who have examined a large number of individuals of all ages and both sexes with a special view to this inquiry, report its existence in a vast majority of the population—a conclusion from which I am forced to dissent.

What, then, is the cause of this venous hum? My own investigations lead me to believe, that in most cases it is referable solely to pressure on the veins, whether excited by the stethoscope or by any other means. With a view to a solution of this question, I cut channels of various widths and depths in the extremities of different stethoscopes, so as to fit over veins of different sizes, without exerting any pressure on them. Thus, when the stethoscope was adjusted carefully to the part to be examined, I was enabled to listen to the circulation in the vein without much risk of producing murmur by pressure—a result which is unattainable by any other method. Proceeding in this manner, I ascertained the absence of venous murmur in 148 out of 196 healthy persons between the ages of fourteen and fifty-nine; or, in other words, I found it present in only 24 per cent., and, probably, in many of these some pressure was exerted, notwithstanding my precautions.

Without attempting to discuss the various theories which have been broached on the subject of venous murmur, I will endeavour to point out the conclusions at which I have arrived after a long series of experimental investigations. Briefly, then, it may be stated, that pressure, whether by the stethoscope or by any other means, will produce venous hum in most cases in which the venous circulation is sufficiently active to excite sonorous vibrations as the result of an eddy in the blood. 2ndly. That venous murmur is seldom met with, unless pressure be exerted on the vein;† but that, in a few instances, it occurs independently of any discoverable pressure. 3rdly. That the rarity of its occurrence without obvious pressure and the frequency of its

* See 'Med.-Chir. Review' for 1852, vol. ix, p. 501. Winterich gives the following as the per-centage of venous murmurs in healthy persons:

Age.	Males.	Females.	Age.	Males.	Females.
	Per cent.	Per cent.		Per cent.	Per cent.
1— 5	97	98	30—40	80	86
5—10	94	95	40—50	77	78
10—15	89	95	50—60	72	75
15—20	86	88	60—70	68	71
20—25	82	88	70—80	40	39
25—30	80	86			

† See Note (*), p. 509.

occurrence under the influence of pressure, justify the opinion that it is essentially due, like arterial murmur, to an eddy in the blood, resulting from some abnormal cause—whether that be the pressure of the stethoscope in the case of the veins of the neck and the extremities, of spicula of bone in the longitudinal sinus, of tuberculous, cancerous, or other deposits, in the case of the veins of the chest—or in one or all of them the rupture of the attachments of one of the valves, or an inordinate contractility of the elastic tissue, which enters into their composition, whereby they are prevented falling back evenly against the coats of the veins. This opinion is confirmed by the fact, that venous murmur, if audible in one vein, is not necessarily audible in other large veins. Thus, it may be audible in one jugular vein, inaudible in the other; or audible in both jugulars, but inaudible in the subclavian or the femoral veins; or audible in the subclavian or axillary veins, but inaudible in the jugulars. 4thly. That, unless the necessary velocity of circulation exists, the eddy which is caused by pressure or by any other means will not be sonorous. 5thly. That excessive pressure, by arresting or retarding the venous circulation, will arrest or prevent the occurrence of murmur. 6thly. That the production of sonorous vibration in the veins is somewhat dependent on the thinness and elasticity of the coats of those vessels, and that as the veins are thinner and more elastic, and the circulation usually quicker in children than in adults, and the tissues in a condition to feel the effects of pressure more readily, venous murmur is more constantly discoverable in children than in adults. 7thly. That anæmia and spanæmia are not necessarily productive of venous hum, any more than they are of functional arterial murmur. This is proved by the fact, that cases of anæmia and spanæmia are occasionally met with, in which not only does no venous murmur exist, but pressure, however ingeniously applied, cannot be made to produce it. Further, it constantly happens that venous hum is met with in persons who are florid and in robust health, and in whom there is no pretence for saying that they are either anæmic, hydræmic, or spanæmic.* Nevertheless, as in the case of functional arterial murmurs, so also in respect to venous hum, anæmia and spanæmia ordinarily intensify the murmur; in part, probably, by modifying the innervation of the parts, and interfering with their contractility, and in part by leading to a condition of the blood in which vibration is more readily excited.

* Thus Dr. Herbert Davies found it present in no less than 46 out of 50 picked healthy men of the Coldstream Guards, whose ages ranged from twenty-one to twenty-seven.

PART IV.

CHAPTER I.

PERICARDITIS, ITS PATHOLOGY, SYMPTOMS, AND TREATMENT.

THERE are few complaints which more surely abbreviate human life, and none which give rise to more suffering and discomfort than diseases of the heart and great vessels. Occurring in the young as well as in the old, they constitute in one or other of their varieties the principal cause of sudden death, and even when they do not prove instantly fatal, they "lay their own hard conditions on the continuance of a man's life, and almost settle beforehand the manner of his death." Interfering as they do with the supply of blood to the various organs of the body, they lead to derangement of all the functions, and after months, or it may be years of dyspepsia, breathlessness, and palpitation, the patient becomes dropsical, or a confirmed asthmatic, and dies, suffocated by the effusion which takes place into the chest and pericardium, as a result of the impediment to the circulation through his heart and lungs.

Like other organs, the heart and great vessels are subject to a variety of diseases, each one of which runs a different course, and produces its characteristic traces and symptoms. Thus the pericardial membrane may be exclusively the seat of disease, or the valvular apparatus may be damaged, or the muscular tissue of the heart may alone be affected, or one side—usually the left side of the heart—may be the seat of disease; or the structure of the aorta or great vessels may be altered to a greater or less extent. And as a result of these various affections, the pericardium may become adherent to the heart, the tissue of the heart may become firmer or softer than natural, the walls thickened or attenuated, the cavities enlarged or lessened in size, the orifices widened or contracted, the valves inefficient, the aorta or other great vessels roughened on

their internal surface, or dilated, contracted, or otherwise altered in form. It is essential, therefore, to a clear understanding of these various affections, that they should be considered separately and their source and progress carefully examined.

To begin with pericarditis or inflammation of the pericardium, the external covering of the heart.

Pericarditis, like endocarditis and other serous inflammations, is essentially of constitutional origin—a local manifestation of a disease which pervades the entire system. Sometimes, indeed, it occurs, as the result of traumatic injury, or as a consequence of irritation, excited by the spread of disease from contiguous organs, but these are quite exceptional cases, and do not invalidate the truth of the statement, that under ordinary circumstances it is one of the local results of a blood disorder, and is connected with general derangement of the system.

There is scarcely a form of constitutional malady which is not sometimes accompanied by pericarditis; but those with which it is most commonly associated are rheumatic fever, Bright's disease of the kidneys, and pyæmia; and of these the first named is beyond all comparison, the most frequent cause of its occurrence. For although this cardiac complication may be entirely prevented, by treating the disease by alkalies, after the plan pointed out in my work on rheumatism, yet it appears from the statistics which have been published by various observers, that under the ordinary methods of treating acute rheumatism, pericarditis occurs once in every 5·97 cases.

Rheumatic pericarditis was formerly supposed to be due to metastasis, or in other words to the retrocession of inflammation from the external parts, and its consequent transfer to the membranes of the heart. But recent observations have shown that exocardial or endocardial inflammation may occur as the first, and for some time the only local symptom of the disease;* that it sometimes *precedes by several days*, the access of articular redness and swelling; and that, even in cases in which it does not take place until after inflammation of the joints has been set up, it is rarely preceded or accompanied by subsidence of the previously existing

* Five instances of this sort have fallen under my own observation. In two cases the patient laboured under pericarditis two days, in another three days, and in another five days prior to the appearance of articular inflammation, whilst in another—the case recorded at p. 324, ed. iii, of my treatise on 'Rheumatism, Rheumatic Gout, and Sciatica,'—the heart appears to have been attacked seven days before rheumatism manifested itself externally. For reference to many other cases, see p. 141-2, of the same treatise.

articular mischief. In other words it has shown that in the great majority of cases, no connexion can be traced between the two sets of actions beyond their origin in one common source of mischief, in one poison which excites inflammation, now at one spot, now at another; at one time attacking several joints simultaneously, or in succession, and then the investing or lining membranes of the heart; at another, reversing the order of attack, and exciting inflammation, first of the heart, and then of the articular structures.

Pericarditis is incidental to all stages of acute rheumatism, but arises less frequently towards the close of the disease, when tending to a favourable termination, than it does at its beginning, or during its progress; it supervenes most frequently and most extensively in cases characterised by the severity of their general symptoms, in which, therefore, there is a great abundance of the *materies morbi*, or a peculiar susceptibility of its influence; it is a more frequent accompaniment of acute rheumatism when occurring in youth or childhood than when occurring in those more advanced in years; it is more commonly met with in women than in men, more frequently in the irritable than in those of a phlegmatic temperament, more usually in those in whom the fibrous structures about the joints are chiefly affected than in those who suffer principally from synovial inflammation. Further, it is peculiarly prone to attack the pale and weakly, who have been reduced by previous illness, or exhausted by injudicious treatment, and in whom the heart is unusually irritable.*

Non-rheumatic pericarditis may arise in connection with constitutional disorders, or with local irritation excited by disease in contiguous organs. Amongst the former, the most frequent are diseases of the kidneys, pyæmia, scarlatina, variola, scurvy, cancer, pneumonia, pleurisy, and peritonitis; amongst the latter may be mentioned hydatid-cysts, or abcesses in mediastinum or other parts, which make their way into the pericardial sac; inflammation on the lungs or pleura, which may possibly spread to the heart by contiguity of structure; and cancerous, tuberculous, and other deposits, which in some instances appear to excite pericardial inflammation, by acting as local causes of irritation. Thus then, from the circumstances of the case, it is obvious that non-

* For full details on all these subjects consult my work on 'Rheumatism, Rheumatic Gout, and Sciatica,' ed. iii, chap. vi, pp. 140-162; and also refer to chap. ix, pp. 257-284 of the same work, where the statistics relative to rheumatic disease of the heart are fully and carefully worked out.

rheumatic pericarditis must be more frequent in adult life, or even in persons advanced in years, than it is in childhood or in youth, and that it is more likely to be met with in men than in women. It is especially prone to arise in the unhealthy, and, like rheumatic pericarditis, it occurs more particularly in the pale and weakly.

Rheumatic and non-rheumatic pericarditis differ in many important particulars. Thus, rheumatic pericarditis is attended by more violent symptoms than non-rheumatic pericarditis; the former is almost always accompanied by pericardial pain, disturbance of the heart's action, and urgent distress; the latter often occurs in a latent form, without any local symptoms calculated to direct attention to its existence; the former obviously places the life of the patient in jeopardy, and not unfrequently proves rapidly fatal; the latter seldom occasions any great increase of constitutional disturbance, and in most instances plays a subordinate part in producing a fatal termination of the disorder. Nevertheless, as non-rheumatic pericarditis is usually one of the final complications of an otherwise fatal disorder, its mortality, if considered numerically, is far in excess of rheumatic pericarditis. The former terminates in death in the majority of cases, the latter proves fatal only in about one out of every six cases.* But, whether pericarditis be rheumatic or non-rheumatic in its origin, the inflammation is of the same character, and runs a similar course, modified only by the constitutional peculiarities of the patient. The pericardium being a fibro-serous membrane, inflammation of its inner or serous coat is essentially of an adhesive character, and leads to an effusion of serum, or of plastic lymph, or of both these products, in varying proportions; or sometimes, more especially in unhealthy subjects,† of a serous effusion more or less tinged with blood, and mixed with curdy, ill-concocted lymph, which has a tendency to assume a puriform character.

But the pericardium is not only a serous membrane, it is also a shut sac, the two opposed surfaces of which are in contact with each other, and kept in constant motion, the one upon the other, by the natural

* Out of 39 cases of rheumatic pericarditis which I noted in St. George's Hospital between the 1st of January, 1845, and the 1st of May, 1848, seven proved fatal, giving a proportion of 1 to 5·8 cases, a result which accords very closely with the observations of Louis, as recorded in his 'Mémoires Anat. Path.,' p. 291.

† I say in "unhealthy subjects" because these differences in the products of the same disease are due to the differences of constitution and to circumstances tending to influence the patient's strength; and the changes which subsequently take place in the morbid products are also governed by the same peculiarities.

action of the heart. Hence, although, when it becomes inflamed, the inflammation may be sometimes controlled by judicious treatment before it has involved the whole extent of the membrane, yet more generally the constant attrition of the two inflamed surfaces keeps up and aggravates the original mischief until it has spread over the entire surface of the heart.

The effects which follow an attack of pericarditis vary according to the extent of the disease and the nature of its products in each particular instance. When the membrane has been only partially implicated, lymph may be poured out in quantity insufficient to cause adhesion between the two layers of membrane, and then by degrees it may be gradually reabsorbed, until a white patch on the heart only remains, where a thick coating of lymph had previously existed; or it may be effused in larger quantities, and the inflamed surfaces may be glued together at the seat of inflammation; or serum, mixed with only a small quantity of lymph, may be effused, distending the sac of the pericardium, and keeping its two surfaces more or less asunder, in which case, adhesions take place less readily, and those which do occur, are partial and irregular. When the entire membrane becomes inflamed, the changes produced are proportionably extensive. If the effusion consist chiefly of coagulable lymph, the two surfaces may become agglutinated together throughout their whole extent; if it consist of lymph and serum, the liquid part may be gradually absorbed, and universal adhesion may take place as before; whilst, if it consist of serum mixed with pus, or with shreds of curdy lymph of low vitality, or if the serum be very copious, and be not readily absorbed, adhesion sometimes fails to take place, the sac of the pericardium becomes permanently distended, and the heart's action greatly embarrassed. The former results are met with in the vigorous and healthy, the latter are observed chiefly in the scrofulous, weakly, and unhealthy. In the first class of cases, if the patient survives the first shock of the attack, he may experience little ill effect from the lesion his heart has sustained. But in the second class of cases there is no effort at recovery, and the unhappy patient lingers on a victim to palpitation and frightful paroxysms of dyspnœa and suffocation, until, after the lapse of a few weeks—sometimes in the course of a few days—his life and his sufferings are terminated rather suddenly by death.

It must not be imagined that death occurs only in cases accompanied by an abundant liquid effusion; for, although these are generally the most unfavourable instances, yet fatal results are sometimes met with

even when little else than lymph has been poured out. Nor does the fatal issue occur at any particular period of the attack. Rarely, indeed, does it take place at a very early date—seldom before the seventh or eighth day; but sometimes it takes place within thirty-six hours, and in other instances not until after adhesion has taken place. Thus the morbid appearances observed on dissection vary both with the character of the inflammation and with the period at which the attack has proved fatal. If death has occurred early, traces of inflammation may be often observed, even before cutting into the pericardial sac. Lymph and serum, and sometimes pus, may be found effused into the anterior mediastinum, while the pericardium itself is distended with fluid, or, if not much distended, is soft and pulpy to the touch. And no sooner is its cavity laid open, than the reason of this abnormal condition is apparent. In the former cases it will be found to contain an enormous collection of serum, sometimes clear, more often turbid, generally containing flakes of lymph, and not unfrequently tinged with blood; and when this is allowed to escape, the surface of the membrane will be seen to be highly vascular, and covered over a greater or less extent by a coating of recent coagulable lymph. In the latter cases a small quantity only of liquid effusion will be found, but a large amount of plastic lymph, often tinged or mottled with blood, will be seen deposited in successive layers on the internal surface of the sac. And a very peculiar appearance this lymph presents. The constant motion and attrition of the parts prevent its forming a smooth surface, and its appearance varies according to the amount of serum present, and to the vitality of the more solid exudation. If it be of low vitality it adheres very loosely to the membrane which exudes it; much of it is detached by the motion of the heart, and floats in loose shreds in the surrounding serum, while that which still remains adherent to the membrane, is rough, loose, and shaggy. If it be more highly organizable, and serum be present in sufficient quantity to keep the two sides of the sac asunder, and thus prevent their immediate adhesion, then it is thrown by the action of the heart into rough and irregular transverse ridges. If, on the other hand, there be little serum present, and the membrane be covered with a thick coating of highly plastic lymph, then is the tendency to adhesion great, very slight lateral motion of the membrane can take place, and a spongy or honeycomb, or tripe-like appearance is produced by the repeated contact and separation of the two inflamed surfaces.

These are the appearances observed when the patient falls an early

victim to the attack, but when he dies, as he does sometimes, at a later stage of the complaint, soon after the pericardium has become adherent to the heart, there are other points worthy of observation. The medium of adhesion will be found to consist of plastic lymph, still soft, and easily broken down, and which on more than one occasion I have known to give way during life on the speedy recurrence of pericardial inflammation. But in this lymph may be seen here and there some bloody spots or streaks, or anastomising red lines, the first indications of its commencing organization.

And if a still longer period elapses before death, the organization of the plastic lymph will be found completed, and the pericardium will be seen either firmly and uniformly glued to the heart, or attached to it by bands, or a network of fine adhesions, having a smooth glistening surface, and presenting the characters of serous membrane; whilst, if the attack of pericarditis has been severe, exocardial adhesions will be found gluing the outer surface of the pericardium to the anterior surface of the chest, and in some instances to the anterior margins of the lungs.

It has been suggested that the lymph effused in pericarditis is often thoroughly reabsorbed, so that the pericardium is restored to the condition of health, and perfect recovery takes place. This I do not believe to be the case. Doubtless it may be reabsorbed to a very great extent, so that a thin layer only may be left where a copious and thick deposit had previously existed; but I quite agree with Dr. Latham and Dr. Watson that adhesion more or less general between the two layers of the pericardium is the most favourable issue we are justified in expecting when inflammation has been extensively diffused over the membrane. In pericarditis the constant attrition of the two inflamed surfaces keeps up and aggravates the existing inflammation, so that it endures for a lengthened period. The lymph is usually poured out in large quantities, and in healthy persons, when its vitality is great, shows a strong disposition to become organized; and as the two surfaces of membrane, each loaded with a layer of this plastic medium, are constantly in apposition, it is difficult to conceive how they can fail to become adherent. But our *post-mortem* records show clearly and indisputably that a small and partial effusion of lymph may sometimes accompany pericarditis, and that not unfrequently the lymph is curdy and but slightly plastic. In both these cases adhesion may fail to take place. In the first because the lymph, even if plastic, is not in quantity sufficient to restrain the to-and-fro motion of the two layers of the

membrane; in the last because it is from its nature but little disposed to form adhesions, and is almost invariably accompanied by a copious effusion of serum, which keeps the two surfaces of the pericardium asunder until by continued washing the lymph is rendered shreddy, and for the most part inapt for the purpose of adhesion. In such cases, doubtless, it is gradually absorbed to a great degree, and some loose membranous bands, or a few white patches only may be left, such as have been well described by Mr. Paget in vol. xxiii of the 'Medico-Chirurgical Transactions.' But in ordinary cases, when extensive pericarditis occurs in healthy persons, a large quantity of highly plastic lymph is usually poured out, and then so rapidly does adhesion take place and organization of the plastic medium commence, that I look upon its complete absorption as next to impossible.

The course and duration of pericarditis are subject to great variations. Most commonly the disease runs a steady course, and subsides in from ten to twenty days, leaving no symptom of its having existed beyond some increased irritability of the heart; but in some instances the symptoms, both general and physical, may continue for many weeks, and the physical signs, especially a clicking friction sound about the root of the great vessels, may continue even for months. So also in regard to fatal cases. The ordinary duration of fatal cases is from five to ten days, but instances are on record of death from pericarditis within twenty-seven hours;* and, on the other hand, I have known it terminate fatally after nine weeks.

The physical signs of pericarditis vary with the stage of the disease and the amount and character of the inflammatory products. The first, and for some time the only pathognomonic sign of its occurrence is the peculiar sound of friction I have endeavoured to describe,† superficial in its situation, audible, perhaps, over the whole præcordial region, but inaudible along the aorta and great vessels, varying in its quality, pitch, and duration according to the nature and amount of the effusion and the energy of the heart's action, but almost always preserving its distinctive character of a to-and-fro sound produced by the rubbing together of the two roughened surfaces of the pericardial membrane. At an early period of the attack there is no change in the area of dulness on percussion, and no alteration in the position of the apex beat, and these are

* See Andral 'Anat. Path.'

† For a full description of this sound and of the means of distinguishing it from murmurs the result of endocardial disease, see *ante*, pp. 504-5.

points which should be carefully noted, in order that any subsequent change may be accurately ascertained. For after a time, as inflammation progresses and effusion takes place into the pericardial sac, not only does the friction sound cease,* but the heart is displaced and other characteristic signs are produced. The first of these signs is the occurrence of a dull sound on percussion in the præcordial region. In the normal condition the præcordial dulness is seldom above an inch and a half, or two square inches in extent, as the lungs lap over the remainder of the heart and yield a clear sound on percussion. But when effusion takes place into the pericardial sac, the space occupied by the heart and its envelope increases, the lungs are pushed aside, and the chest is found to yield a dull sound on percussion, where it had previously furnished a well-marked resonance. From the recumbent position which the patient ordinarily maintains, the fluid gravitates towards the base of the heart and the root of the great vessels, and in this direction, therefore, the extension of præcordial dulness is first perceived; but as the effusion increases, the whole pericardial sac becomes distended, and the pyramidal form of the pericardial dulness becomes apparent. At the same time the heart sinks backwards and its apex is tilted upwards and to the left,† so that it may beat as high as the fourth intercostal space; the costal expansion over the pericardial region is diminished, and vocal fremitus cannot be felt as usual at the right edge of the sternum, especially if the patient lies over on the right side. Sometimes the dulness produced by pericardial effusion is found as high as the first left rib, sometimes it extends beneath the whole length of the sternum, except about an inch at the top.‡ Not unfrequently it reaches an inch or an inch and a half to the right of the sternum, and occasionally I have known it extend from nipple to nipple over the whole of the anterior surface of the chest. Sometimes, indeed, extension of the præcordial

* This holds good generally, but not universally. In a case in St. George's Hospital, under the care of Dr. Seymour, the distended pericardium reached from nipple, and contained upwards of two pints and a half of fluid; and yet pericardial friction sound was heard up to the last. This was evidently attributable to the existence of enormous hypertrophy of the heart, and to the fact that the patient maintained a semi-erect posture, leaning forwards and resting her elbows on her knees.

† This holds good only when the patient lies flat on his back, and when the heart is healthy; for when it is hypertrophied or dilated, and especially when the right cavities are so affected, and when the pericardium is adherent to the anterior parietes of the chest, this upward and outward tilting of the apex is not always observed.

‡ Dr. Walshe mentions a case in which the distended pericardium reached "about a thumb's breadth *above* the clavicle." Loc. cit., p. 591.

dulness may be occasioned by enlargement of the heart, by pneumonic consolidation of the anterior portion of the left lung, or by collections of fat, mediastinal tumours, and other extraneous causes; but dulness of pyramidal form occurring suddenly in a previously healthy person is symptomatic of effusion, and affords a tolerably accurate measure of its amount. Its absence, however, must not be regarded as certainly indicative of the absence of effusion, as the dulness which otherwise would have resulted from the presence of effusion may be marked by the intervention of a portion of emphysematous lung. In such a case the chest may even yield unusual resonance.

Thus, then, if the cessation of the friction sound be dependent on distension of the pericardial sac, percussion will usually indicate the fact; and, in proportion as the effusion increases in quantity, so will the limits of the dulness be extended, and so also will the heart's action become more and more embarrassed, its impulse more feeble and irregular, and its sounds more distant, and less audible. This is just the reverse of what happens when the friction sound ceases in consequence of adhesion of the two layers of membrane; for as absorption progresses, and adhesion takes place, the præcordial dulness decreases in extent, the heart's impulse becomes steadier, its sounds louder and clearer, and the pulse firmer and more regular.

It may sometimes happen, that we miss listening to the heart until so much fluid has been effused into the pericardium that a friction sound is no longer audible. In hospital practice patients often are not seen until after the stage of attrition has passed away, and even in private practice cases sometimes occur, in which, either from the rapidity with which effusion has taken place, or from the absence of pain and other general symptoms of cardiac distress, the stage of attrition is entirely overlooked. In some such instances an inclination of the body forward, or firm pressure exerted over the region of the heart, serves to bring the two layers of the pericardium into apposition, and thus enables us to detect exocardial friction. But it sometimes happens that even these measures are ineffectual; the posterior surface of the heart is alone coated with lymph, and friction sound cannot be heard. In all these cases percussion is peculiarly valuable; it affords information not otherwise attainable. I have known præcordial dulness for days the only physical sign of the mischief which had occurred, and, taken conjointly with the distance, feebleness, and irregularity of the heart's sound, it has enabled me to trace accurately the extent to which effusion had proceeded. Sometimes, however, there are other signs to

assist us in these cases; for the integuments in the præcordial region may become œdematous, and the intercostal spaces wider and fuller than natural; and in children, in whom the elasticity of the chest walls is great, the whole præcordial region may be more prominent than usual, or the left edge of the sternum may be considerably raised. Sometimes, again, though the friction sound may have ceased, the displacement of the heart resulting from the effusion, or the pressure of the effused lymph on the great vessels may occasion systolic murmur at the base of the heart; and the occurrence of such a murmur coincidently with extension of the area of dulness, and with muffling of the heart's sounds at the lower part of the cardiac region, will sometimes afford confirmatory proofs of the existence of pericardial inflammation.

Another physical sign by which the existence of pericarditis is sometimes marked is a peculiar thrill, communicated to the chest, and from the chest to the hand when placed on the situation of the heart. This strange sensation is most perceptible between the cartilages of the second and third, and the third and fourth ribs on the left side of the chest. Like the friction sound, it results from a vibration occasioned by the attrition of the two roughened surfaces of the pericardium, and hence it imparts to the touch the same information which is conveyed by the friction sound to the ear. Its appearance and disappearance are also regulated by the same circumstances on which the commencement and the cessation of friction sound depend, but unlike the friction sound, it is by no means always, or indeed, generally present in cases of pericarditis. In every case accompanied by this thrill, in which I have had the opportunity of examining the condition of the parts after death, the outer layer of the pericardium has been found glued, as it were, to the parietes of the chest by lymph effused into the anterior mediastinum; and if, as I believe, this consolidation of the parts, and the consequent formation of a good conducting medium, is essential in pericarditis to the production of this tremor, then is the infrequency of its occurrence explained, as is also the fact that it usually accompanies the whole of the heart's action; whereas, the purring tremor communicated to the chest by extensive disease of the valvular apparatus is usually felt only with the systole of the heart when the organ is brought into contact with the anterior parietes of the chest. I do not recollect having ever met with this observation in books, but I have verified it in several instances, and am satisfied of its correctness.

Accompanying this thrill in certain instances is an undulatory

movement visible on the chest, in the same position in which the friction thrill is felt, viz., in the second and third, or third and fourth left intercostal spaces. Whenever the eye detects this motion, it is almost certain that friction thrill will be sensible to the touch;* but it does not follow, that when friction thrill can be felt, this undulatory movement will be perceptible on the chest. This, doubtless, is attributable solely to the difference in the amount and character of the effusion, For as the undulation is a motion communicated to the walls of the chest by the movement of fluid in the pericardial sac, it obviously cannot be present when the products of inflammation are chiefly solid. The value of these signs, however, both of friction and of undulation is greatly diminished by the fact that they do not occur until an advanced period of the attack, when the real character of the disease can hardly be mistaken.

As recovery takes place, the effused products are gradually reabsorbed, and a retrogression of the physical signs is observed. The bulging of the cardiac region subsides; the area of dulness on percussion decreases in extent; the friction sound reappears, and then ultimately ceases; the systolic murmur is no longer heard; the heart sounds become clearer and less distant; the apex resumes its natural position; the impulse regains its force and character, and the respiratory sounds begin to encroach upon the space hitherto occupied by the distended pericardium.

For the detection of pericarditis we have to rely chiefly upon the physical signs; but, in many instances the general symptoms are of great importance, by exciting alarm for the safety of the patient, directing attention to the seat of mischief, and giving information which may guide us in our prognosis. The invasion of the disease is sometimes, but not invariably marked by chilliness or rigors; the skin becomes hot, and, in cases of rheumatic pericarditis, perspires freely, the respiration emitting a peculiarly disagreeable acid odour. The aspect of the patient is changed;† his countenance, which only a short time before

* In making this statement, I presuppose that the undulatory motion has recently arisen, and is not due to the action of a dilated and hypertrophied heart which has contracted adhesions to the anterior walls of the chest.

† Dr. Hope in noticing this peculiar anxiety of countenance remarks "The sardonic expression and peculiar contortion of the features attending the worst cases of pericarditis are occasioned by the sympathy subsisting between the respiratory nerves of the face and those of the heart. An impression is conveyed to the spinal cord through the pneumogastric nerves, and is reflected to the face through the portio dura."

may have been calm and tranquil becomes anxious, and his manner and deportment show but too clearly that serious mischief has supervened. Sometimes he is restless, more generally he lies quietly flat on his back, and evinces inability or much disinclination to lie on his left side. The breathing, which had been unaffected, becomes hurried and shallow, and often accompanied by a short, dry cough; he complains of uneasiness and oppression at the epigastrium, and of pain in the præcordial region, increased on full inspiration, as also by pressure with the fingers in the intercostal spaces, or under the cartilages of the false ribs on the left side. He suffers from palpitation, not unfrequently feels faint, and sometimes experiences a sharp pain, almost resembling a paroxysm of angina, shooting through the chest to the scapula, or upwards to the clavicle or the left shoulder. His sleep is fitful and disturbed; the pulse, which at first is full and hard and frequent, ranging from 120 to 140 in the minute, becomes weak and irregular both in force and rhythm; the tongue is furred; the bowels often torpid; the stomach sometimes, though rarely irritable; the urine high-coloured, loaded with lithates, and, in many cases, albuminous, and as the disease progresses, the voice may become feeble, spasmodic dysphagia may supervene, and œdema of the feet and ankles may occur.

These symptoms are apt to vary in different cases; and, in some respects, the variation is explicable by a difference in the amount and nature of the inflammatory products. The more solid the character of the inflammatory exudation, and the smaller the amount of serum poured out, the less constrained will be the posture of the patient, the less disposition will be manifest to syncope, and the longer, *cæteris paribus*, will his pulse retain its force and regularity. On the other hand, the larger the quantity of serum poured out, the more the pericardial sac is distended, the greater is his reluctance to move or change his posture, the greater the disposition to fainting, and the more feeble, irregular, or intermittent his pulse. Indeed, when the effusion is very great, whatever position he may have assumed, whether on his back or side, or sitting erect, or leaning forwards with his arms upon his knees, so fearful is he of accelerating his heart's action, embarrassed as it is by the amount of liquid, that he cannot be induced to change his posture.

These symptoms are extremely striking, and might be deemed sufficient of themselves to indicate the existence and nature of the mischief, but unfortunately they are not very distinctive, nor are they

very constant in their occurrence. Generally, indeed, when the heart is attacked, some pain, dyspnœa, irregularity of pulse, or some other indication of existing disease, draws attention to the seat of mischief. But I have repeatedly seen cases in which most acute pericarditis—more frequently non-rheumatic than rheumatic—has been set up, and has continued for some days without the supervention of any symptoms likely to direct attention to the action which was going on, and instances not unfrequently occur in which, from first to last, such general symptoms are altogether absent.*

I have hitherto made no mention of one peculiar train of symptoms which sometimes accompany pericarditis, because, in the first place, their occurrence is rare, and, in the second, they are apt to divert attention from the real seat of mischief, and are, therefore, deserving of special notice. I allude to phenomena resulting from disturbance of the cerebro-spinal functions. It has been already stated that a great alteration is usually observed in the expression of the patient, and that excessive restlessness oftentimes supervenes as soon as inflammation of the heart is set up. In some instances, this excitement of the nervous centres proceeds to a very much greater extent. The restlessness and anxiety pass into delirium—into quiet or into low, muttering delirium, often attended with stupor; or, on the other hand, into wild, uncontrollable delirium—into the delirium of mania, accompanied sometimes by tetanic or choreic spasms, or by convulsions which terminate either in extreme exhaustion, or, in death, by coma. Such cases have been long recognised by the profession, and instances in point have been placed on record by several accurate and observant physiologists. In many respects they are very remarkable. They present all the symptoms usually observed in cases of meningeal or cerebral inflammation, or in cases of inflammation of the spinal cord, yet are seldom connected with any structural change within the cranium or the spinal column which can be regarded as indicative of inflammatory action. Sometimes, indeed, on dissection after death, the cerebral veins have been found gorged, and some little serum has been discovered effused under the arachnoid, or into the ventricles of the brain, or into the sheath of the spinal cord; but this has not been a constant occurrence, and, even had it been so, it would only have shown that in this, as in other forms of violent delirium, the circulation in the nervous centres is apt to be interfered with, and serum poured out in consequence.

* Many such cases are recorded in the 'Post-mortem and Case-book' in the museum of St. George's Hospital.

To what, then, are these cerebro-spinal symptoms attributable? Are they to be regarded as tokens of threatening mischief in the nervous centres? or as direct and primary results of cardiac inflammation? or as expressions of alarm experienced by the system generally, and by the sensorium—the source of the system's consciousness—at the interference with its nutrition, resulting from the poisoned condition of the blood? *

To me, I confess the latter, appears the only satisfactory explanation.

At one time all cerebral symptoms which occurred in the course of acute rheumatism and other acute diseases,† were referred to inflammation of the brain or its membranes, resulting, as was supposed, from metastasis of the morbid action to the cerebral structures, in consequence of the subsidence of articular or other inflammation. But their independence of metastatic action is attested by their not infrequent occurrence without the subsidence of existing local inflammation; and their independence of mischief developed within the cranium is abundantly proved by dissection after death, which, even when the case has terminated fatally, fails in most cases in affording the slightest evidence of cerebral mischief.‡

The second suggestion is more in accordance with sound pathology, but, nevertheless, is not quite satisfactory. Soon after the discovery that cerebral disturbance may arise in the course of acute rheumatism and other acute diseases, without the concurrence of cerebral inflammation, delirium was so often found associated with active cardiac inflammation, that many persons were led to regard disorder of the sen-

* The substance of the following remarks on this subject were first published in my work on 'Rheumatism, Rheumatic Gout, and Sciatica.'

† I refer to cases of acute rheumatism and rheumatic pericarditis as affording typical examples of this form of affection of the nervous centres, not as presenting any special peculiarities. In all cases of pericarditis the constitution is deranged, and the blood altered in character, and to this fact I believe the symptoms to be primarily referable, whether they occur in connexion with rheumatic, uræmic, pyæmic, or any other form of pericarditis.

‡ In proof of this I would refer to the case recorded at pp. 307-8, ed. iii, of my treatise on 'Rheumatism, Rheumatic Gout, and Sciatica,' as also to the valuable cases reported by Dr. Richard Bright in his account of spasmodic diseases accompanying affections of the pericardium; by Dr. G. Burrows, in his work on 'Disorders of the Cerebral Circulation;' by Dr. Latham in his 'Clinical Medicine;' by Dr. Todd in his 'Lumleian Lectures;' and by Dr. Watson in his 'Practice of Physic.' Andral, Bouillaud, Davies, Rostan, Stanley, and others have reported similar symptoms connected with pericarditis when occurring without any accompanying rheumatism.

sorial functions as invariably connected with mischief occurring in the central organ of the circulation. The old-fashioned doctrine of metastasis to the brain, exploded under the influence of pathological research, and the heart was in every case pronounced to be the "fons et origo malorum." By some, its anatomical relations with the cerebrum, were pointed to, in explanation of the symptoms observed, and the delirium was attributed to irritation conveyed to the brain by the phrenic* and pneumogastric† nerves, as a consequence of inflammation of the pericardium or endocardium. By others, sympathetic irritation was considered inadequate to explain the symptoms, which were therefore attributed to disturbance of the cerebral circulation occasioned by the embarrassment of the heart's action which results from the access of cardiac inflammation.‡ But neither of these interpretations appears to me correct, inasmuch as delirium, convulsions, and coma, are always rare and exceptional phenomena, even when pericarditis and endocarditis terminate fatally; arise not unfrequently in cases distinguished by less than the average severity of their cardiac symptoms, and in which, therefore, presumption favours the belief that there is no unusual irritation of the cardiac nerves; and occur sometimes when dissection after death proves the heart and its membranes to be quite free from disease, and when, therefore, the non-existence of such a cause of irritation is placed beyond all doubt.§

It is different, however, in regard to the third suggested cause of cerebro-spinal symptoms. The blood in every case of pericarditis is poisoned by the presence of a morbific matter, and the nutrition of the brain is interfered with in consequence; and, although delirium, convul-

* Dr. R. Bright and M. Bouillaud.

† Dr. Hope, op. cit.

‡ Dr. Watson's 'Practice of Physic,' ed. i, vol. ii, p. 276; and Dr. Burrows on 'Disorders of the Cerebral Circulation,' p. 212.

§ In a review of the first edition of my work on 'Rheumatism' it was suggested that the detachment of fibrinous vegetations from the valves on the left side of the heart, and their impaction in the vessels supplying the brain, may be the cause of cerebral disturbance in these cases. This supposition, however, is simply gratuitous, and without foundation. In the valuable records of fibrinous plugging of the cerebral vessels, published by Dr. Kirkes in vol. xxxv of the 'Med.-Chir. Trans.' hemiplegia, and not delirium, resulted from the cause alluded to, and extensive softening of the cerebral structure was the characteristic pathological phenomenon. It is true that delirium is sometimes met with in connection with embolic plugging of the cerebral vessels, but it is so only when the plugging takes place during the existence of acute disease, and it occurs so seldom that it must be regarded as a consequence of the altered condition of the blood, rather than as a result of mere obstruction to the circulation.

sions, and coma, may result from cerebro-spinal inflammation, yet an altered condition of the circulating fluid is equally, if not more energetic in their production.* Every one, for instance, knows how certainly excitement or profound coma is caused by the ingestion of inordinate quantities of spirituous liquors, and by the action of belladonna and other poisonous agents; how frequently delirium results from the deleterious influence of urea, and how often it accompanies typhus fever, erysipelas, and almost every exanthematous disorder. Yet in all these cases, dissection after death has shown that such symptoms afford not the slightest ground for the presumption of cerebral congestion or cerebral inflammation. The brain has been found paler and drier than natural, or of a darker colour, and marked by an increased number of bloody puncta, and sometimes a small quantity of serum has been discovered under the pia mater, or in the ventricles, as a result of an impeded cerebral circulation. Rarely, however, has there been any trace of inflammatory action, or of any other organic lesion. In fact, experience has shown that, whenever the blood is poisoned or altered in character, as it is in all the cases referred to, there may occur, without any local inflammation, every shade and variety of cerebral disturbance, from slight wandering or flightiness, to violent maniacal delirium, accompanied or unaccompanied by convulsions or tetanic spasms, and terminating in recovery, or in death by coma.

A distempered condition of the blood then, I conceive to be the true proximate cause of the sensorial disturbance occasionally observed in the course of pericarditis.† For although this disturbance, when occurring in the course of acute rheumatism and in other disorders, is sometimes associated with inflammation of the heart or other organs, yet instances are not wanting in which, by taking place without the concurrence of any internal inflammation, it asserts its independence of all local action, and ranks itself amongst the symptoms known to be dependent on a vitiated condition of the circulating fluid.

But, admitting the altered condition of the blood to be the primary cause of the brain's disturbance, yet as its character is always altered in pericarditis, and head symptoms seldom occur, there must be other

* For full and copious illustrations of this important fact, see Dr. Todd's 'Lumleian Lectures' for 1850.

† Dr. Todd has urged this view most clearly and forcibly in his admirable lectures on "Delirium and Coma," delivered before the Royal College of Physicians, but he has not dwelt sufficiently on the causes which give effect to the operation of the poison in certain cases, and which by their presence prevent cerebral disturbance in others.

influences at work, which determine the occurrence of delirium in certain cases, and its total absence in others. By looking carefully to the circumstances under which cerebral symptoms are most apt to arise in different disorders, we may glean important information on the subject. It is well known that persons of a nervous, excitable disposition are more apt to experience ill effects from any interference with their functions than are others of a more vigorous and less irritable temperament. Moreover, it has been ascertained that nervous susceptibility is most fully displayed when the constitution has been deranged by habits of intemperance, or by long-continued ill-health. Not only are persons more prone under such circumstances to suffer severely from local injuries, but they are apt to exhibit symptoms of irritation from causes which, in persons of a healthy constitution and less excitable habit, would hardly give rise to any disturbance. Thus it is that in habitual drunkards, whose constitutions are shattered, or whose nervous systems have long been unduly exalted or depressed, comparatively small potations, on the one hand, or on the other a brief abstinence from their accustomed stimulants, or any temporary depressing cause, will seldom fail to induce an attack of delirium tremens. Hence, also, the frequency of traumatic delirium, and of the delirium which so constantly accompanies erysipelas in persons whose constitutions have been severely taxed. The excess or the deficiency of the accustomed stimulus in the case of the drunkard, the shock and the loss of blood in the case of the wounded man, and the poison of the disease in the last instance, prove sufficient to disturb the relationship subsisting between the blood and the nervous centres. With a brain participating in the general mal-nutrition of the body; a heart weak, ill-nourished, ill-supplied with nervous stimulus, and hardly capable of maintaining a proper circulation; and a blood vitiated or impoverished, it is not difficult to conceive that a slight additional cause of irritation or depression may prove sufficient to disturb the brain's equilibrium, and that an attack of delirium may supervene, whenever, by the presence of some fresh morbific matter, by an increase in the watery part, or a diminution in the coloured corpuscles of the blood, or, indeed, by any material alteration in the character of the circulating fluid, the nutrition of the nervous centres is still further interfered with. Nor is it to be wondered at that, from the same cause, an attack of pericarditis or pleuro-pneumonia should in some cases determine an accession of cerebral symptoms. The shock resulting from the occurrence of inflammation in such vital organs as the heart or lungs

must surely so far influence the circulation as to cause the blood to be sent to the brain less forcibly and less regularly than before; indeed, we have proof of such an influence in the weak, irregular, and intermittent pulse, by which the invasion of pericarditis is often accompanied; and this, in a person already predisposed, by the vitiated condition of the blood, would probably disturb the nutrition of the brain sufficiently to give rise to symptoms of undue excitement or undue oppression.

Thus, then, it would appear that in all cases in which cerebral disturbance presents itself in the course of pericarditis, the altered condition of the blood is its primary or proximate cause.

And the same holds good in regard to cases which are attended, not only by disturbance of the intellectual faculties, but by symptoms indicative of spinal irritation. Just as delirium may result from the direct action of the poisoned blood on the brain, so convulsions and other spasmodic affections which are sometimes met with in pericarditis and endocarditis may take their origin in spinal irritation, excited by the action of the same morbific agent. In the first-named cases, dissection after death very generally fails in revealing any trace of mischief within the cranium; and in the last the spinal cord and its membranes are found equally free from organic lesion.

But although this must be admitted, there seems little reason to doubt that the cardiac inflammation exerts considerable influence in the production of cerebro-spinal symptoms, for they are seldom observed in mild cases of pericarditis, and still more rarely in cases of acute rheumatism unaccompanied by pericarditis or endocarditis. Indeed, the history of recorded examples of these affections renders it extremely probable that, although primarily due to the influence of the morbid condition of the blood, they are more or less connected with irritation of those branches of the phrenic and pneumogastric nerves which are distributed over the inflamed parts; for it has been observed that, in the cases most remarkable for choreic or tetanic convulsions and other symptoms of spinal irritation, the inflammation has not been confined to the internal surface of the pericardium, but has extended to its external surface, and to the diaphragmatic pleura, where branches of the phrenic and pneumogastric nerves are distributed most abundantly.*

* For cases in illustration of these facts, see my work on 'Rheumatism, Rheumatic Gout, and Sciatica,' ed. iii, pp. 296-315; also Dr. R. Bright's essay on 'Spasmodic

Thus, then, as delirium and choreic spasm are invariably connected with great susceptibility of the nervous centres, and occur almost invariably in persons of a weak, excitable habit, they are always indicative of extreme danger, even when unattended by cerebral inflammation. That recovery may take place, under proper management, I am satisfied, both by personal experience and by the testimony of others who have watched and noted cases in point; but when the delirium is violent and of long continuance, the result cannot be otherwise than doubtful.

Indeed, the issue of pericarditis is always doubtful, and the prognosis, therefore, should be guarded. *Cæteris paribus*, the prognosis should be more unfavourable in non-rheumatic than in rheumatic cases; more unfavourable in cases accompanied by a copious effusion into the pericardium, with great irregularity of the heart's action, than in those in which a smaller quantity of serum is poured out, and the heart is less embarrassed; more guarded in those accompanied by much constitutional depression than in those marked by tolerance of remedial measures; more cautious when, together with the cardiac inflammation, there coexists inflammation of the lungs or pleura, than when the respiratory organs are unaffected; and much more unfavourable in cases complicated by cerebral disturbance than in those in which the intellect remains unclouded.

It sometimes happens that relapse occurs, so that just as adhesion is taking place friction sound recommences, and is rapidly followed by extension of the præcordial dulness and other signs of pericardial effusion. In these cases, the irregularity of the heart's action is usually very great, and the prognosis unfavourable. I know of no statistics on the subject, but as far as my own experience enables me to judge, I should imagine that the mortality in these cases is at least twice as great as in primary attacks.

The question as to the remote prognosis of pericarditis, or, in other words, as to the effect produced by adhesions of the pericardium, is one on which authors are not agreed. Some persons have represented adhesion of the pericardium as a lesion productive of early death, whilst others have maintained that it has little tendency to disturb the action of the heart, or to shorten life.* And both parties can cite cases in

Diseases Accompanying Affection of the Pericardium," 'Med.-Chir. Trans.,' vol. xxii; Bouillaud's 'Traité des Maladies du Cœur;' Dr. Burrows on 'Disorders of the Cerebral Circulation,' pp. 210-212; and Dr. Hope on 'Diseases of the Heart.'

* Thus Dr. Wm. Budd reports that he has "seen a great number of cases of adhe-

support of their view. The fact appears to be that, under ordinary circumstances, adhesions of the two layers of the pericardium, whether partial or universal, derange the heart's action far less than might be expected. I have repeatedly, on dissection, met with old adhesions of the pericardium in persons who, for years before their death, had been in excellent health, and during life had not exhibited any symptom of diseased heart. But sometimes the inflammatory action appears to act upon the muscular tissue of the heart, and produces weakness of the walls of the heart, with a tendency to yield or dilate under pressure; and sometimes when the coating of effused lymph is very thick, its contraction is so forcible that it presses upon the coronary arteries, impedes the circulation, and interferes with the nutrition of the heart. In these cases, the heart becomes dilated, or hypertrophied, or atrophied, or in some instances, possibly, hypertrophied and dilated, according to the precise amount of influence exerted by these several causes of cardiac derangement. Further, endocarditis is a frequent accompaniment of acute pericarditis, and results in the production of valvular mischief, which surely leads to hypertrophy and dilatation of the heart. In other cases, again, the heart becomes adherent to the anterior walls of the chest, by means of the effused lymph, and then its action is so much interfered with that mischief cannot fail to ensue. Therefore as, on the one hand, many instances occur in which adhesions of the pericardium, whether partial or general, appear not to exercise any seriously pernicious influence over the heart, or to modify its structure or action; and as on the other, in many cases of pericarditis, agencies are at work which have a tendency to produce alteration in its condition, it seems fair to conclude that any structural alteration of the heart which is found associated with adhesion of the pericardium should be attributed to one or the other of those accidental agencies, rather than to adhesion of the two layers of membrane.

The management of a case of pericarditis is at all times difficult, and before it can be undertaken with a reasonable prospect of success, it is necessary to understand what are the pathological conditions to be treated, the ends for which means are to be found. First, then, there is the morbid condition of the blood, the primary source of all the mischief. This obviously must be corrected as far as possible. Secondly,

sion of the pericardium (often general) of long standing in which the heart was in all other respects natural, and its functions during life perfectly performed." ('Library of Medicine,' vol. v, p. 195.)

there is the local inflammation; and this must be subdued at all hazards, if we hope to save our patient from immediate death. Thirdly, there are the products of inflammation; and these must be got rid of, if we wish to prolong his days, and to save him from the consequences of a damaged heart. The treatment which is best adapted to fulfil the first of these objects will depend upon the precise diathetic condition of the patient; but in the case of rheumatism it will be the administration of the alkalies and alkaline salts, in full doses, as pointed out in my treatise "On Rheumatism, Rheumatic Gout, and Sciatica." The second calls for bleeding, calomel, and opium; and the third for blisters, diuretics, and absorbents.

Of all the remedies for the cure of inflammation of the heart, and especially of rheumatic inflammation, venesection is that most generally adopted. M. Bouillard has employed it very largely in France; and even here it has numerous and strenuous advocates. Observation, however, has not led me to form a favourable estimate of its curative power. In some few instances I have known it useful in expediting the action of other remedies, and in moderating the force and frequency of the pulse, when the patient has been robust, and the heart's action turbulent and excessive; but in general it has afforded very little relief. It has usually failed in subduing the pain, and has produced only a temporary impression on the pulse. On the other hand, the arguments against its indiscriminate employment are numerous, practical, and weighty. It certainly is not necessary for the cure of the disease, whether it be of rheumatic or non-rheumatic origin, as I rarely have recourse to it, and yet have effected many cures without it; it is not productive of safety to the patient, for M. Bouillard lost six out of eighteen patients attacked by the disease; whereas, out of eighty-nine cases, of which I have notes, in which it was not employed, three only proved fatal, one of which occurred in a weak, debilitated person, and was complicated by low, quick-spreading inflammation of the pleura. And, lastly, it is often prejudicial to the patient, by exhausting his strength, rendering him liable to relapses, and altogether protracting his recovery. There also appears strong reason for doubting whether, as suggested by Dr. Watson, "bleeding to such an extent as to bring the heart's action to a pause in deliquium" may not tend to favour the deposition of fibrin upon the valvular apparatus.

General bloodletting, then, should be employed in rheumatic pericarditis under precisely the same circumstances as in cases of acute

rheumatism uncomplicated by cardiac inflammation. As a remedy to be exclusively relied upon it is quite unavailing, and often extremely dangerous; as an expedient to be employed in aid of other remedies it is occasionally of much service. If a patient be robust and plethoric, with a pulse characterised by extreme fulness or hardness, it may not only be had recourse to in moderation, but may be repeated until some impression is produced on the circulation. In such persons, it assists in allaying the inflammation, and favours the action of mercury, opium, and other remedies. But in ordinary cases its employment is unnecessary, and therefore inexpedient. It tends to diminish the red globules in the blood, when they are already below the healthy standard; it renders more irritable the already irritable and excited heart, and it favours, as I believe, the formation of fibrinous deposits in the valves. Moreover, if carried beyond the exigencies of the case, it may cause an adhesive inflammation to assume a serous or suppurative character, and may prevent that peculiar and most valuable action of mercury whereby the extent of inflammation is limited, and its products absorbed and got rid of. But although bleeding from the arm is seldom advisable, local bloodletting is often serviceable. Leeches may be placed over the region of the heart, or blood may be abstracted by cupping; and thus more obvious and more immediate benefit will be obtained, and a greater impression produced on the disease—as manifested by the pulse, the stethoscope, and the sensations of the patient—than by the most copious and repeated general bloodletting. Not unfrequently the præcordial pain ceases, the heart's sounds become clearer, and the pulse softer, even while the leeches are doing their work.

It is sometimes a question whether leeching or cupping is the more appropriate remedy in these cases; and some persons recommend the employment of the former, whilst others as strenuously advocate the latter. My own experience inclines me to give a decided preference to leeches. They are quite as easy of application, and are free from the objection very properly urged against cupping, of causing pressure upon the ribs at the spot where, in the inflamed condition of the heart, the least pressure and the least percussion cannot fail to be productive of mischief; and although this difficulty may be in some measure overcome by applying the cupping-glasses between the left scapula and the vertebral column, I still incline most strongly to leeches; for I have never known cupping produce the same amount of benefit that I have often seen result from leeching. Nor is it difficult to suggest an explanation

of the fact. By cupping, blood may be abstracted as copiously, and almost as rapidly as by venesection from the arm; whereas leeches do their work more slowly, and by their gradual and continued drain tend, perhaps, to cause some revulsion from the deeper-seated structures. Be this as it may, they certainly afford an amount of relief out of all proportion to the quantity of blood abstracted through their agency, and incomparably greater, in the majority of cases, than can be obtained by venesection from the arm, or by cupping, as ordinarily practised. If the force and fulness of the pulse be such as to require bloodletting for its relief, or if the patient be so plethoric as to render it desirable to let blood with the view of expediting the action of remedies, then bleeding from the arm is the most appropriate treatment. If, on the contrary, the patient be pale and weakly, and the pulse not more than ordinarily forcible, then, if bloodletting be deemed advisable for the relief of the præcordial pain and anguish, leeching, and not general bleeding, should be had recourse to. Cupping should be reserved for those cases in which a copious bleeding is required, and in which blood does not flow freely from the arm.

Mercury, like bleeding, is sometimes a valuable remedy in pericarditis, and particularly so in rheumatic pericarditis. Powerfully antiphlogistic in the influence it exerts, it assists in moderating the intensity, and in limiting the extent of inflammatory action; and having done so, it operates as bloodletting does not—it promotes the absorption of the matter effused. Having first of all checked the progress of the disease, it subsequently lends its aid to the process of reparation. It is obvious, then, that no case of pericarditis occurring in a strong and healthy person, can be safely treated without mercury. The fact of there being inflammation of the pericardium, implies the occurrence of structural changes of a grave and momentous character—changes which must be arrested, if life is to be preserved, and which, when arrested, must be modified or got rid of, if life is to be prolonged. Changes, too, they are which occur most rapidly, and are slow and difficult of removal, so that no time must be lost in pressing to its fulfilment the treatment considered most efficacious against their extension. For this purpose mercury, of all known remedies, is that on which most reliance can be placed. It does not supersede other remedial agents, but it comes most powerfully and beneficially to their aid. It sustains the good effect produced by bloodletting; it calms the violence, alters the character, and circumscribes the limits of the local inflammation; it stimulates the

absorbents to the business of repair, and promotes the continuance of the natural secretions at a time when they are checked, and well-nigh suspended by the shock the system has sustained.

But to ensure these effects, the remedy must be pushed until its impression on the constitution is unequivocally declared by the occurrence of ptyalism. Three or four grains of calomel combined with opium, in sufficient quantity to prevent its running off by the bowels, may be given every four hours, and if it be considered desirable still further to hasten the access of salivation, mercurial inunction may also be had recourse to. In such a case, a drachm, or a drachm and a half of the strong mercurial ointment may be rubbed in, night and morning, on the abdomen or the inside of the thighs; or a blister may be applied to the region of the heart, and mercurial dressings applied to the blistered surface, and covered with a bread-and-water poultice. Thus, sometimes, in the course of the second, and generally within three or four days, a full mercurial action will be produced. Then, and not until then, have we any guarantee that the medicine has found its way into the system, and that its beneficial influence will be displayed. But as soon as salivation has commenced, and even sooner in some instances, the symptoms manifestly improve; the pain and anguish begin to subside; the pulse becomes quieter and steadier, and the heart's sounds clearer. The cessation of the to-and-fro sound of friction in one case; or, in another, its recurrence coincidently with the decrease of præcordial dulness, denotes the absorption of fluid from the pericardium; whilst, if there be mischief within the heart, the diminished intensity of the endocardial murmur, and the greater regularity of the heart's action, give intelligence of improvement in that quarter. These are facts to which I can testify from repeated observation; and I have so often seen all antiphlogistic remedies employed, and yet no sensible amendment produced until after ptyalism has commenced, that I cannot doubt as to the cause of the improvement.

In this, as in all other serous inflammations, it will sometimes be found difficult to obtain the constitutional effect of mercury. It may be administered freely for a considerable time, and may be guarded carefully by full doses of opium, and yet no mercurial fetor be perceived, no evidence of mercurial action obtained. But it must not, therefore, be inferred that its exhibition is useless. As opium, when acting beneficially in delirium tremens, and acute rheumatism, may be given in

enormous doses without occasioning either stupor or constipation, so, when the constitution is sound, and inflammation violent and extensive, mercury, whilst exercising a beneficial influence over the course of the disease, may sometimes prove tardy in producing salivation. It seem as if, under such circumstances, its whole power were expended in allaying the diseased action; and it fairly admits of question, whether the difficulty experienced in producing salivation, may not be taken, *cæteris paribus*, as a test of the extent and activity of the morbid action, and of the necessity for the early and active administration of mercury to control and arrest its course. Certain it is that, in my experience, the difficulty of inducing salivation has varied, *cæteris paribus*, according as the patient has been strong and healthy, the inflammatory symptoms unusually high, and the inflammation extensive, and of that peculiar character which tends to the effusion of plastic and highly organizable lymph.

Mercury, though generally beneficial in pericarditis, and especially in rheumatic pericarditis, is not equally so in all constitutions. In robust and habitually healthy persons, it seldom gives rise to much constitutional irritation or depression, and when the inflammation is extensive, it produces its specific action slowly, and acts beneficially on the course of the disease. In such cases, therefore, it can hardly be employed too fearlessly or too vigorously. But in the weakly, the irritable, and the unhealthy, and especially in those who are suffering from Bright's disease of the kidneys, its constitutional effects often supervene rapidly, are extremely violent in their character, and, in some instances, frightfully depressing; and hence it is productive of injurious consequences, by favouring a tendency to serous or suppurative, instead of to adhesive inflammation. To such persons, therefore, it cannot be administered too cautiously. If, under the circumstances, it is deemed expedient to obtain mercurial action, the remedy should be so administered as that the system shall not suffer from its operation; it should be exhibited in small doses repeated at long intervals, and at the least symptom of its action, its administration should be suspended.

Some persons have attempted to undervalue the curative influence of mercury in pericarditis,* and others, though not denying its efficacy in many forms of inflammation, have yet contended that the cure of pericarditis occurring in connection with rheumatism may be

* See some papers published in the 'Medical Times' for 1849, by Dr. John Taylor, of Huddersfield.

safely entrusted to other remedies.* Now, although, as already stated, I do not counsel the indiscriminate employment of mercury, and in some rare instances do not administer it even in the most cautious manner, yet I cannot accord my assent to a mode of practice which would deprive us of what, in many cases, proves our most powerful ally. Other remedies very often suffice in non-rheumatic cases, and even in that form of rheumatic pericarditis which occurs in weakly or cachectic persons; indeed, the administration of mercury is seldom of much avail in such cases, and is often prejudicial to the patient's safety, especially when the kidneys are diseased. But I am fully persuaded that, in the acute and sthenic form of pericarditis, as it presents itself in persons of a strong and healthy constitution, nothing is of greater service than the remedy in question. No such case can be treated safely without its administration. Recovery may take place in certain instances without it, as indeed without any other remedy, but that is an argument which applies to almost every disease, and to every kind of treatment; and there is no other remedy of whose curative influence experience and observation have supplied such abundant and unequivocal proofs as of mercury in active serous inflammation. Its efficacy, however, varies remarkably, according to the class of cases in which it is employed, as also to the mode in which it is administered, and it is not surprising, therefore, that persons who have exhibited it largely in all cases of pericarditis should have met with some in which it has been not inoperative only, but actually prejudicial to the safety of the patient. In most of such cases the blame should not be charged upon the remedy, but upon those who have injudiciously administered it; for, given in due season, and with proper regard to the exigencies of the case, no remedy is more powerful, and few are so trustworthy.

Opium is, of all remedies, that which comes most powerfully in aid of bloodletting and mercury. Roused and excited as the vascular and nervous systems are by the violence of the morbid action, and the pain which attends it, all remedies prove comparatively useless, if unaccompanied by the sedative influence of this narcotic. The constant pain, with the absence of sleep and the constitutional irritation consequent thereupon do more, I believe, to exhaust a patient's strength and counteract the action essential to his recovery than bloodletting and mercury can do to promote them. Therefore, in every case of pericarditis, and espe-

* See a paper by Dr. J. Risdon Bennett in the 'Lancet' for December 6th, 1851.

cially in rheumatic pericarditis, opium in full doses is indispensable. It should be given not only in doses adequate to restrain the purgative action of the calomel, but in quantity sufficient to assuage the pain and allay the irritability. From two thirds of a grain to a grain, or even more, should be prescribed, either alone or in combination with calomel, every four or five hours; and in the intervals, if there be much pain, it is expedient to administer eight or ten minims of the tincture, or of Battley's sedative solution.* To the weak and irritable, to whom mercury is of little service, if not positively noxious, it proves peculiarly valuable. It not only subdues pain, but allays irritability, and procures sleep; and I am satisfied that many of my patients would have fallen victims to the disease had not their strength been husbanded by its sedative influence.

It is possible, however, that opium exercises some more directly curative influence. In these cases of pericarditis, the two inflamed surfaces are in constant motion, and rub against one another; their innervation is exaggerated, their irritability exalted. Now, although opium cannot, of itself, prevent the continuance of this friction, it can blunt the sensibility of the inflamed membrane, and thus prevent that excess of irritation whereby the course of inflammation is prolonged, and its products made to assume an unhealthy character. In several instances of pericarditis, in which, in spite of venesection and mercury, inflammation has continued unabated, whilst the constitutional irritability has been excessive, and the heart's action rapid and violent, I have seen the mercury omitted and opium administered alone, with the happiest and most speedy results. The pain has ceased, the patient has obtained sleep, his irritability has subsided, the pulse has fallen in frequency, and has increased in steadiness; and the stethoscope, no less than the general symptoms of the disease, has testified to the reality of the improvement. The same train of symptoms has also been observed in other cases, in which, from the weak and cachectic condition of the patients, I was fearful of inducing mercurial action, and therefore trusted exclusively to opium with alkalies, diuretics, and repeated blistering. The sedative apparently contributed as much to the patients' recovery, as it manifestly did to their comfort.

Another most important remedy in pericarditis is a large blister applied to the chest. In the early stage of the attack, especially in the

* In these cases opium is usually preferable to the salts of morphia.

young and vigorous, I am more inclined to confide in leeches; but, when once effusion has taken place, blistering is, of all local remedies, the most serviceable. Indeed, its efficacy appears to vary, in some measure, according to the amount of liquid effusion, its virtue being most unequivocally displayed when the amount of fluid is greatest. In such cases an immediate diminution in the præcordial dulness, together with greater clearness in the heart's sounds, is often observed as the result of a large and efficient blister.

Whilst applying leeches and blisters, and, in some instances, pushing mercury to salivation, the other general indications must not be lost sight of. Though pericarditis or endocarditis be present, they are due to the same cause, and require the same general treatment for their relief, as does the disease with which they are connected. Thus, in rheumatic pericarditis, alkalies are still necessary to counteract and get rid of the *materies morbi*, to which all the mischief owes its origin, and without the removal of which it is difficult to conceive that a cure can be effected. And not only so, they afford most powerful aid to blisters and mercury, in acting as diuretics, and removing the fluid products of inflammation. In endocarditis, alkalies and the neutral salts, particularly the salts of ammonia, prove eminently useful; for, by helping to maintain the solubility of the fibrin,* and so preventing its deposition on the valves, they guard against a lesion which, by the consecutive changes to which it gives rise, leads surely and rapidly to an untimely death.

In all cases, without exception, rest and abstinence are of the utmost importance. No treatment can be satisfactory in its issue, unless perfect rest be enjoined, and every cause of excitement carefully guarded against. This has been repeatedly forced upon my attention in the strongest possible manner. In several instances in which patients have worked themselves into a state of excitement, in consequence of the dread inspired by the thoughts of being leeched, or cupped, or blistered, every symptom has been aggravated, in spite of the remedies employed. Indeed, so strikingly was this the case in one instance, that I was obliged to suspend all local treatment, and to trust entirely to mercurials and diuretics, with opium in full and repeated doses. If any symptoms of sinking arise, diffusible stimulants must be administered according to the exigencies of the case.

When delirium or choreic spasms occur, stimulants and sedatives

* See Dr. Richardson's 'Astley Cooper Prize Essay' for 1856.

must be freely employed; but it should ever be borne in mind that the symptoms alluded to are only the secondary effects of a great and general constitutional disorder, and the general treatment required for the relief of that disorder must on no account be neglected. In rheumatic cases, alkalies and the neutral salts must still be administered in full doses; and in renal cases, diuretics, purgatives. Blisters and counter-irritation are also serviceable.

In certain instances of pericarditis all ordinary treatment proves unavailing to induce absorption of the effused fluid, and the pericardium remains enormously distended for weeks after all traces of inflammation have disappeared. In these cases the patient suffers urgently from dyspnœa, and medicine offers no prospect of relief. It has, therefore, been proposed to puncture the pericardium and draw off the fluid by means of a trocar, and cases have been reported in which this practice has been employed with a certain amount of success. The immediate relief has been very great, and in a few instances recovery has ensued.

I have no personal experience of this mode of proceeding, as no case has occurred in my own practice requiring the adoption of such an extreme measure, but in the event of its being had recourse to, the patient should be placed in a recumbent posture, and the trocar should be introduced between the fourth and fifth ribs, about half an inch from the left edge of the sternum, and the fluid withdrawn slowly, lest the heart should be embarrassed by the sudden removal of the pressure to which it had so long been subjected. Theoretically, I am inclined to believe that the less formidable operation of withdrawing a certain quantity of the fluid by means of a grooved needle would effect the desired object of relieving the heart, and would enable absorption of the remaining fluid to take place.

There is yet one point on which a few observations are needed. It has been already pointed out how readily in most instances the occurrence of pericardial adhesion may be determined when the course of pericarditis is carefully watched, and it might perhaps be imagined that the detection of pericardial adhesions is at all times an easy matter. Indeed, I know that an impression of this kind prevails amongst those who are not skilled in physical diagnosis. But nothing can be further from the truth. In most instances in which the patient is not seen for some time after pericardial adhesions have taken place, their diagnosis is a physical impossibility. Sometimes, as already pointed out, they do not interfere with the action of the heart, nor do

they induce any structural change, nor give rise to any characteristic alteration of the sounds; and even when, directly or indirectly, they occasion atrophy, hypertrophy, or dilatation of the heart, their existence must be mere matter of conjecture, inasmuch as they are not accompanied by any change in the action or sounds of the heart which can be regarded as in any way indicative of their existence. But although this holds good in the majority of instances, it is otherwise in regard to the exceptional cases in which lymph has been effused in the anterior mediastinum, and the pericardium has contracted adhesions to the anterior walls of the chest. Patients in whom this complication has occurred almost always present symptoms of the disease they have undergone which ought not to be overlooked by the practised physician. Thus, the heart is necessarily retained in the position it occupied at the time when adhesion took place, and as the apex is usually tilted upwards and slightly outwards during the acute stage of pericarditis, it is not unfrequently retained in that position afterwards, and may be felt and seen pulsating there. Further, the heart being retained so closely and so extensively in apposition with the chest walls, its action gives rise to an undulatory movement in the cardiac region, and each systole produces a slight retraction of the parietes in the fourth interspace, just above the apex. Important corroborative evidence is also afforded by the effect of respiration. When the heart is not agglutinated to the chest walls, the lungs encroach upon the præcordial region during full inspiration, its resonance on percussion increases, the intercostal spaces expand, and the sounds of respiration are audible there; but when the heart has contracted adhesions to the anterior walls of the chest, the lungs cannot push in front of the heart, and hence there is little or no expansion of the intercostal spaces in the præcordial region, the dulness on percussion remains uninfluenced even by the fullest inspiration, and extends further upwards than even in cases of cardiac hypertrophy, and respiratory sounds are altogether absent. Add to this a peculiar tumbling action which the heart acquires under these circumstances—an action which is most remarkable when the heart is hypertrophied and dilated—and the evidence of adhesion of the two surfaces of the pericardium to each other and of the external layer of the pericardium to the chest walls, is complete.

CHAPTER II.

ENDOCARDITIS—ENDO-PERICARDITIS—CARDITIS.

Endocarditis, or inflammation of the lining membrane of the heart, is a more common, and practically, therefore, a more important form of disease than pericarditis. Originating in an irritating, unhealthy condition of the blood, it is apt to be set up in acute rheumatism, and in the whole class of diseases already referred to as associated with the production of pericarditis; nay, more, it not unfrequently happens that endocarditis occurs coincidently with pericarditis, as the result of the same agency, and that endocarditis or endo-pericarditis, is accompanied by carditis or acute inflammation of the muscular structure of the heart itself.

The pathology of endocarditis is somewhat obscure, in consequence of the rarity of cases which prove fatal in the earlier stages of the disease, and of the difficulty, therefore, which is experienced in investigating its earlier pathological effects. It appears, however, from the few opportunities I have had of inspecting after death the changes which it produces in the human subject, as also from the valuable experiments of Dr. Richardson, who induced endocarditis by injecting lactic acid into the peritoneum of dogs,* that the first effect of inflammation of the endocardium is the production of intense vascularity of the membrane, with a swollen condition of the valves, and, occasionally, the exudation of transparent lymph, or the deposit of fibrin on their surface. At a subsequent stage the vascularity of the surface begins to subside, the valves are still swollen, but the exuded matter within them is more solid than before, and any existing fibrinous deposits are more firmly adherent to the surface. At a still later stage, the abnormal vascularity entirely disappears; the exudation within the valves is partially and irregularly absorbed, so that the edges

* I had the privilege of witnessing these experiments of Dr. Richardson, and the results were most remarkable. Not only was acute articular rheumatism produced, but endocarditis which was recognised by auscultation during life, and resulted in the production of its usual *post-mortem* appearances, was observed in several cases. For full particulars, see Richardson 'On the Coagulation of the Blood,' p. 371, *et seq.*

of the valves become thickened and beaded; the fibrinous deposits become firmly agglutinated to the valves, and the natural transparency of the membrane is replaced by whiteness and opacity.

The changes just described are those which occur in the milder forms of the disease; but they convey no adequate idea of the mischief produced by the more formidable attacks, nor of the extent to which fibrinous deposition may proceed, nor of the transformations through which the fibrinous deposits or vegetations may pass. Therefore, as fibrinous deposits constitute the most common form of endocarditic valvular lesion—especially in connection with rheumatism—it may be desirable to examine the subject a little more in detail.

Fibrinous vegetations* are sometimes found in all the different chambers of the heart, and they are especially apt to be so in the left auricle, but in most instances they are confined to the valvular apparatus, or its immediate vicinity, and the mitral and aortic valves—the valves, which are constantly bathed in highly fibrinised arterial blood—are those which are peculiarly liable to suffer. The right cavities, however, with the tricuspid and pulmonary semilunar valves, are sometimes, though rarely, affected.†

Fibrinous vegetations are often very numerous, and commonly vary in size from that of a pin's head to that of a millet seed. In one case they are isolated and distinct from each other, at another, partially confluent; and when several spring from a common base, they may form a mass of considerable size. Sometimes when fibrinous accretion has taken place rapidly, as it does in certain conditions of the blood, their form is changed, and the action which in other states of the system might have resulted in the deposition of small warty granules along the edges of contact of the valves gives rise to the production of long, filamentous growths, or of large pedicled masses, forming pendulous tumours, which hang loosely into the ventricle, and are moved to and fro by the current of the blood.‡ When the vegetations assume the former character, they are usually confined to the valves, and chiefly affect their edges of contact; so that in the sigmoid valves they are arranged in a double crescentic

* Many of the following remarks are taken from the chapters "On Inflammation of the Heart," contained in my work on 'Rheumatism, Rheumatic Gout, and Sciatica.' My views on the subject remain unaltered, and I therefore venture to quote them here.

† For cases in point, see 'Edin. Med. and Surg. Journ.,' vol. v.; also, a case recorded by my late colleague, Dr. Wilson, in the 'Lancet' for 1844, vol. ii, p. 217.

‡ Some of these masses, measuring from half an inch to an inch in length, are preserved in the museum of St. George's Hospital.

form;* but when their growth is more luxuriant, as in the instances last alluded to, they are more widely distributed over the endocardial membrane. The surface of the valves against which the current of the blood is directed is often thickly studded with them; on the edges of contact of the valves they form festoons or fringes; the chordæ tendineæ attached to the mitral valve are sometimes loaded with an abundant crop of them; and, occasionally, in the different chambers of the heart, more especially in the left auricle, they are scattered profusely over the entire surface of the lining membrane. Judging from my own experience, the cases in which these last forms of vegetation occur are just those in which, either from some constitutional peculiarity, or from some other cause equally beyond our ken, these accretions manifest a strong tendency to decay, and in which arise the formidable erosions and ulcerations, to which I shall presently have to refer.

The microscopic examination of these fibrinous deposits establishes the identity in nature of their different forms, and shows that they are sometimes granular, but more generally imperfectly fibrinous in texture, presenting somewhat of a laminated appearance; and that throughout their structure are numerous granules and oil globules in varying proportions. In many old-standing cases, cartilaginous and calcareous matter is found in the valves, and in the fibrinous vegetations, as the result of earthy or atheromatous degeneration which has taken place subsequently to the attack in which the valves were injured, or the fibrin first deposited.

In colour and consistency these concretions vary just as much as in size and in position. At first, they are of a pink or reddish colour, soft, and easily broken down, and can be readily detached from the smooth surface of the membrane on which they are deposited; subsequently, they become less coloured, and of a much firmer consistence, but still admit of being separated from the membrane, whilst, after a still longer period, they become perfectly colourless, and so firmly adherent, that they can be removed only by tearing the membrane to which they are attached. At a still later date, these warty growths or bead-like accretions cease in most cases to exist, as such, upon the valves; they become by degrees incorporated with the structure of the valve, and, merging gradually into one another, until the divisions between the several granules are effaced, they are ultimately replaced by a laminated ridge of fibrin. This at first is marked by serratures corresponding to the divisions between the original granules; but after a time it also loses

* For explanation of this, see Dr. Watson's 'Lectures,' ed. i, vol. ii, p. 267.

all traces of its origin or mode of formation, and becomes smooth and polished like the other parts of the endocardium.

Such are the changes which ensue when there is a tendency to the repair of these valvular lesions; but when it happens that a tendency exists to further disorganization, the changes which occur are of a different nature. Ulceration may take place, and produce a perforated condition of the valves, or a broken-down, ragged state of their edges, and it may even spread to the chordæ tendineæ, which may thus be eaten through, and rendered useless.* Cases, too, are on record, though I have never met with one, in which suppuration of the substance of the heart has taken place, and has caused perforation of the septum ventriculorum.† And even when none of these more violent local actions take place, very serious mischief may nevertheless occur; for fibrinous deposits sometimes excite unusual irritation of the valves, with gradually induced thickening, corrugation, and contraction; the motion of the valves becomes more and more limited; adhesion of their flaps may ensue, or earthy or atheromatous degeneration may take place, and the valves, strained by the unwonted force and frequency of the heart's action, and rendered weak and brittle by the changes they have undergone, are no longer able to offer resistance to the pressure of the blood, and are lacerated or slit up.

It has already been stated that the mischief produced by pericarditis does not necessarily induce permanent derangement in the action of the heart; and there is, I think, presumptive evidence to show that some thickening of, or deposit on the valves may take place as the result of endocarditis, and may be entirely got rid of by absorption. Thus, seeing how readily such lesions are repaired in other parts of the body, there can be little doubt but that when the results of endocarditis are confined to redness and œdematous swelling, the mischief may entirely disappear with the gradual cessation of inflammatory action. Even when lymph has been poured out on the surface or in the substance of the valves, there is no impossibility in the lymph being reabsorbed, and the damage completely repaired. In most cases, however, we should not be justified in anticipating such a happy result. Experience has shown that not unfrequently the process of repair is effected

* In vol. ix, pp. 131-153 of the 'Path. Trans. of Lond.,' my colleague, Dr. Ogle, has given an admirable record of twenty-one cases of ulceration of the valves of the heart, which occurred in St. George's Hospital. The paper is worthy of careful perusal.

† For cases in illustration of this fact, see Watson, loc. cit., ed. i, vol. ii, p. 287, and a report by Mr. Avery in 'Trans. Path. Soc., Lond.,' vol. ii.

imperfectly, the valves remaining thicker and more rigid than natural. And when, as sometimes happens, the valves are coated with plastic lymph, and become more or less agglutinated together, or one of them, perhaps, is folded on itself, and its apex glued either to the adjoining surface of the aorta, or to some other portion of its own area, then the damage which has been sustained is irreparable, and the disease must tend rapidly to a fatal termination.

When fibrinous deposits have taken place on the valves, the question of repair is even more uncertain, though presumption favours the belief that in some instances it may be effected more or less perfectly. Thus, dissection has shown that the process of reparation does often advance to a considerable extent; and observation has proved that functional, if not organic repair, may occasionally be completed.* On three several occasions I have had reason to believe that the removal of fibrinous deposits has been effected partially, if not wholly. In the instances alluded to, the accession of heart disease was marked by all the general and physical signs of endocarditis, and systolic mitral murmur was most intense. Nevertheless, on the rapid subsidence of inflammation, the bellows murmur gradually ceased. In two of these cases, the sound commenced so suddenly, increased so rapidly, and was accompanied by such well-marked general and local symptoms of endocarditis, that there could be no more doubt as to the occurrence of inflammation, and of some injury to the valves, as a result of that inflammation, than would attach to any fact announced through the stethoscope, but not submitted to ocular demonstration. Doubtless, the murmur in each of these instances is explicable on the supposition that the valves were merely inflamed and swollen, or that lymph had been effused on their surface; but so constantly are fibrinous deposits associated with endocardial inflammation, that the existence of such deposits in the cases under consideration is rendered extremely probable.† I am bound to

* For some excellent remarks on this subject, see Dr. Ormerod's 'Gulstonian Lectures.'

† Two other sources of this adventitious valvular sound might be suggested, viz.: 1st. The anæmic or spanæmic condition of the patient. 2ndly. The irregular contraction of the structures connected with the valvular apparatus. Neither of these explanations, however, is applicable to the cases in question. For in the first place the patients were not anæmic, nor had they been subjected to depletion, nor was the sound audible in the course of the great vessels. Secondly, if it had been due to irregular contraction of the structures connected with the valves, and not to mischief occurring in the valves themselves, it should have evinced some irregularity

admit, however, that the cases are few in which the process of repair can be thoroughly carried out. Fibrinous vegetations are only indirect products of inflammatory action; they are deposited from the blood, and are produced, not only during active inflammation, but during a state of mere irritation.* Hence, I fear, there is little chance of complete recovery, when obstruction to the circulation is dependent upon this form of lesion; for when once irritability of the valves has been excited, their constant motion, and the presence of adventitious deposits must tend to keep up that irritability until mischief has proceeded beyond the possibility of complete repair.

The physical signs of endocarditis, though not quite so certain and unmistakeable as those of pericarditis, are nevertheless sufficiently characteristic. If we know the previous condition of the heart, they enable us to discover, at an early period, the inroad of endocardial inflammation, and to point out with certainty where mischief has been set up, and where it is still occurring. The only difficulty which can arise is in discriminating between murmurs which are referable to organic valvular mischief and those which take their origin in functional disturbance, and, if we are unacquainted with the previous condition of the heart, in determining whether a murmur results from existing endocardial inflammation, or from old-standing valvular disease.

The nature of the lesions produced by endocarditis having been already described, it is unnecessary to do more than draw attention to the fact that they generally consist of greater or less derangement of the valvular apparatus, and chiefly, though not invariably, affect the valves on the left side of the heart. There may be either swelling, or thickening, or puckering, or rigidity of the valves, or deposits of lymph or fibrin on their edges or surface, or perforation, or tearing, or breaking down of their substance. In all these cases, the current of the blood must be more or less interfered with; in some the onward current must be obstructed; in some a reflux of blood must take place through the imperfectly closed opening, and in others the same lesion may cause obstruction to the onward current, and also permit of regurgitation. In all, however, an eddy must be produced, together with a greater or

both as to its character and continuance, whereas it remained constant throughout several days, though steadily increasing in intensity.

* This was beautifully illustrated in the course of Dr. Hope's experiments on the ass. As the circulation became sluggish, a deposition of fibrin, forming a fleshy vegetation, took place at the spot where the heart's valves had been irritated.

less degree of vibration; and the result of such vibration is the production of a sound, or, as it is often called, a murmur, accompanying the rhythm of the heart. If, when there is much obstruction, the heart's cavities be enlarged, and its muscular walls strong, so that a large current of blood is driven forcibly past the obstruction, the murmur produced will be more intense than it would be if the ventricle were not dilated, and its walls not thickened—than it would be if a smaller quantity of blood were driven less forcibly past the same obstruction. Again, if the obstruction be very great, and the action of the heart extremely feeble, the eddy produced may not be sufficiently strong to produce sonorous vibrations capable of reaching the ear through the chest walls; and so with every variety of lesion. The character of the sound itself, as well as its intensity, must obviously vary according to the exact nature of the mischief, and the relative power and proportion of the obstructing and the propelling forces.

Thus, then, by the commencement of a bellows sound or murmur we are apprised of the access of endocardial inflammation; and by the position of the sound, by the direction in which it is heard, by the period of the heart's action at which it occurs, and by the state of the arterial pulse, we are enabled to judge with amazing accuracy of the site of the lesion from which it originates. If the murmur be synchronous with the systole of the heart, it must obviously accompany the egress of blood from the ventricle, and therefore must be referable either to obstruction at the aortic orifice or to regurgitation through the mitral valve.* If it be contemporaneous with the diastole of the heart, it must accompany the entrance of blood into the ventricle, and therefore must be due either to obstruction at the mitral orifice, or to regurgitation through the aortic outlet. But then comes a question as to the means of distinguishing the two systolic murmurs from one another, and of discriminating between the two diastolic murmurs. How can we refer each to its own particular valve? Partly, as already stated, by its position, partly by the direction in which it is heard, and partly by the character of the arterial pulse.

If a murmur† accompanying the systole of the heart be heard more

* In this instance, and, indeed, throughout the next few paragraphs, I refer to valvular disease on the left side of the heart only, as being by far more common than similar disease on the right side.

† The following remarks are not intended to supersede the more detailed enumeration of the physical signs and general symptoms of valvular disease, which will be found at pp. 500-4 and 561-4.

distinct at the base of that organ and along the track of the aorta than it is towards the apex of the heart, then it is due, in all probability, to a vibration caused in the onward current of the blood by obstruction at the aortic outlet of the ventricle, and this is rendered still more probable if the pulse at the wrist be small. If, as I have often had occasion to observe, more especially in cases of old-standing valvular mischief, the murmur be heard along the track of the larger vessels, if it be perceptible in the carotids, and, as it sometimes is, at the wrist; and if, accompanying this murmur, a thrill be felt, not only over the region of the heart, but even in the radial artery, there can no longer be a doubt on the subject, as obstruction in the course of the arterial circulation could alone give rise to such phenomena.

Again, if a systolic murmur be heard at the left apex of the heart more distinctly than at the base of that organ, and if, under these circumstances, there be irregularity in the arterial pulse, and great inequality in its force and fulness, then is the disease situated at the mitral orifice. In this case, as in the last, the murmur is produced by the passage of the blood through the roughened, or the rigid and contracted ventricular orifice, and the pulse is rendered irregular and unequal by the constant variation in the quantity of the blood which at each contraction of the ventricle regurgitates, or makes its way back through the imperfect valves into the left auricle. And as in the last case, so also in this, a vibration is often communicated to the chest, causing a thrill or purring tremor perceptible to the touch—a thrill, however, which, unlike that which occurs when there is obstructive disease of the aortic valves, is occasioned by a backward, instead of by an onward current of blood, and, consequently, is not transmitted to the same extent along the aorta and the various arterial tubes.

The diagnosis, then, between the two systolic murmurs, is clear and satisfactory, and that between the two diastolic murmurs is equally simple and conclusive. If, which is not very frequently the case, the mitral valve be so diseased, and the current of the blood so strong as to cause sonorous vibration during the passage of the blood from the left auricle into the left ventricle, then will the murmur be heard most distinctly about the fourth left intercostal space, and thence towards the apex of the heart; the pulse, owing to the obstruction, will be irregular in rhythm and small, though variable in size, and, unless either the quantity of blood ejected at each systole of the heart be insufficient to cause the aortic valves to flap distinctly, or their sound be drowned

by the adventitious murmur—a circumstance of rare occurrence—the natural short, smart clack occasioned by their sudden tension will be heard superadded to or accompanying the murmur.

But, if the abnormal sound arises from the reflux of blood through defective aortic valves, the signs produced will be very different. Not only will the sound be loudest from about the middle of the sternum opposite the third intercostal space, upwards towards the base, instead of downwards towards the apex of the heart, but, instead of being heard together with the natural second sound, it will almost or entirely replace it. Moreover, it will be heard along the aorta and the larger arterial trunks, and will be accompanied by a most singular and characteristic pulse. This, the pulse of aortic regurgitation, derives its peculiarity from mechanical causes. It is not the small pulse of aortic obstruction, nor the unequal and irregular pulse of mitral regurgitation; but, in its most marked and most striking character, it is the unsustained pulse of unobstructed arteries. There is in this case no impediment to the onward current of the circulation, no lack of blood to fill the vessels, and no deficiency of force to propel it; but, from the insufficiency of the aortic valves, and the consequent reflux of blood into the ventricle, the prolonged swell which at each systole of the heart is naturally imparted to the blood in the vessels is not sustained; the successive waves of blood, therefore, are short and abrupt, and hence the pulse is jerking, and gives a sensation as if successive balls of blood were being shot suddenly under the finger. So strong is this reflux, and so strong the jerking to which it gives rise when it exists in any marked degree, that the motion occasioned by it may be seen even at the wrist, and may be felt in almost any part of the body.*

Each of the sounds, then, produced by endocarditis, when existing separately, and in a well-marked degree, admits of easy diagnosis, and may soon be recognised even by an unpractised ear. But they are sometimes masked by other sounds, and are often found more or less commingled, two or more of them occurring in the same case; and thus their diagnosis may be greatly complicated. The pericardium may be inflamed coincidently with the setting up of endocardial

* In illustration of this fact, Dr. Watson details the particulars of a case, respecting which he says:—"The shock of this man's artery was plainly to be felt through his clothes by one's hand laid lightly upon the bend of his arm. His wife told me that for four or five years past this jarring blow had made it uncomfortable for her to take his arm when they were walking together. The same kind of jerking impulse was strikingly perceptible in the femoral arteries, and in the carotids." (Loc. cit., p. 256.)

inflammation, and then a deep-seated systolic murmur may be heard at the same time that the ear is assailed by the loud superficial to-and-fro sound of friction. Sometimes there is a double endocardial murmur, the one accompanying the systole, and the other the diastole of the heart, and this may be occasioned either by disease of a single valve, or by disease of two separate valves; either by disease of a single valve of such a nature as both to cause obstruction and admit of regurgitation, or of two separate valves, the one giving rise to the obstructive, the other to the regurgitant murmur. Thus, not unfrequently a loud systolic aortic murmur coexists with an aortic diastolic murmur, and occasionally a systolic mitral murmur occurs coincidently with an aortic diastolic murmur. Sometimes there is even a greater complication, and a systolic murmur, arising in part from mitral regurgitation, in part from aortic obstruction, is followed by a diastolic murmur, occasioned partly by mitral obstruction, and partly by aortic regurgitation. Difficulty, also, repeatedly arises from the fact, that one morbid sound may be so loud and prominent as to mask and render almost inaudible another, which, under other circumstances, would have announced in plain and fearful tones the existence of disease at some other part. But in all these cases, the form or forms of disease which exist may be discovered, and their combinations pointed out by careful attention to the rules I have laid down, by careful attention to the character and position of the abnormal sound, to the direction in which, and the period of the heart's action at which it is heard, and to the state of the arterial pulse.

Our diagnosis, however, is not in all cases so certain as could be desired. In practice many difficulties present themselves which cannot be resolved solely by attention to the sounds. Sometimes, for instance, at the commencement of an attack, it is difficult to determine whether a sound, evidently abnormal, but of an indistinct, indefinite character, be referable to endocardial or exocardial mischief, and it is only by the effect of firm pressure in the cardiac region, and by the variation in the character of the sound observed in the course of repeated examinations, conducted while the patient is in different postures, that it is possible to arrive at a correct conclusion.

So, also, it sometimes happens that an ill-developed, indefinite sound, having its origin in the heart or its membranes, so closely accompanies the respiratory movements as to make it doubtful whether it may not arise from the lungs or pleuræ. Here, again, the position of the sound

does not afford any trustworthy information, but the question may be set at rest by making the patient hold his breath, when, if it be connected with the respiratory organs, it will instantly cease, but will continue as before if referable to the heart.

Again, if a murmur exists when a patient is first examined, it is difficult, and in some instances impossible, to decide whether it be an old murmur or one of recent origin. If it be aortic or mitral in position, systolic in rhythm, soft and blowing in character, and be unaccompanied by evidence of cardiac hypertrophy, it is probably referable to recent mischief; whereas, if it be diastolic in rhythm, harsh and rough in its character, and attended by indications of hypertrophy of the heart, it may be regarded, with even greater probability, as due to disease of old standing. If the heart be hypertrophied, and the murmur systolic, the mere softness or roughness of the murmur affords very uncertain evidence as to its date, and it is impossible to arrive at a trustworthy conclusion. Presumption, however, favours the belief that it is referable to disease of old standing, the only exception being when the murmur, though systolic, is pulmonary in its seat, in which case it is probably of functional origin, and recent in its accession.

Again, in many instances of acute disease, it must be doubtful whether an endocardial murmur may not be due to functional causes. The question is one the decision of which admits not of delay, as on it depends the nature of the treatment and the well-being of the patient. Here our powers of discernment are taxed to the utmost; for it is only by reference to the appearance of the invalid, the treatment which has been adopted, the nature of the symptoms by which the accession of the murmur has been accompanied, and by the position and character of the sound itself, that it is possible to arrive at a sound conclusion. According as the patient is pale and ensanguine, as the murmur varies with the posture of the patient, and is constantly or only occasionally present; as it is heard most distinctly at the apex or at the base of the heart, and in the track of the aorta or of the pulmonary artery; as it is soft or blowing, or harsh and rasping; as it is more or less superficial; and as its accession has been attended or unattended by pain and dyspnœa, by fluttering or irregularity of pulse, or by evidence of increased cardiac action, so must be our judgment.* No certain rule can be laid down to enable the practitioner to estimate correctly the importance of each

* For full details respecting the characters of functional murmurs, see *ante*, pp. 493-5.

indication; but any one who is cautious and watchful for the accession of cardiac inflammation will not fail to give a significant interpretation to præcordial pain, and increased or irregular cardiac action; whilst, on the other hand, he will not overestimate the importance of a systolic murmur, if it occurs in a pale and weakly person who has been sweated profusely, or has undergone venesection, or has been otherwise subjected to loss of blood, more especially if the murmur be not accompanied by præcordial pain, or by increased or irregular action of the heart, and be heard most distinctly in the track of the pulmonary artery.

Thus, by paying attention to the circumstances which characterise each case, we may generally arrive at a correct conclusion, and if we are sometimes unable to do as much as might be wished towards arresting the progress of disease, and repairing the damage it has occasioned, it is at least satisfactory to be able to detect its existence, and to feel that, being aware of its nature, we are in a position to do all that can be done for its alleviation and cure.

The general symptoms of endocarditis ordinarily resemble those of pericarditis so closely that it will be unnecessary to recapitulate them, and the reader is referred to the section on pericarditis for full information on the subject; suffice it to say that pain is a less prominent symptom of endocarditis than of pericarditis, and that there is seldom much distress of breathing, or much disinclination to change of posture. Pressure in the præcordial region is unattended by pain, and so is a deep inspiration. There is usually less fluttering and irregularity and acceleration of the pulse, and less tendency to syncope. In other respects it is generally impossible to draw any diagnostic inference from the general symptoms, and recourse must be had to the distinctive features revealed by a physical examination.

Not unfrequently, endocarditis is unaccompanied by symptoms calculated to direct attention to the seat of mischief, and would escape detection altogether if recourse were not had to the stethoscope. But in certain cases symptoms arise which have no analogue in pericarditis. These symptoms are characterised by sudden collapse, with pulmonary obstruction or cerebral disturbance. The patient becomes suddenly faint and struggles for breath, the countenance is anxious, the face pale and livid; the pulse rises to 140, or even more, and is weak and irregular, both in force and rhythm, the surface is cold, and covered with a clammy perspiration, and delirium or coma supervenes, and is often accompanied by more or less of paralysis. These symptoms are connected with the rapid

formation of enormous fibrinous deposits on the valves of the heart causing great obstruction to the circulation, or with ulceration and breaking down of the chordæ tendineæ, or with the detachment or disintegration of fibrinous deposits. The more formidable of these symptoms are consequent on the detachment of fibrinous deposits from the valves of the heart,* which are then carried by the circulation into the smaller vessels, and cause obstruction of the arterial circulation; or else, on ulceration of the chordæ tendineæ, or on softening and disintegration of the fibrinous deposits. In the two latter cases symptoms are often produced analogous to those connected with pyæmia.

The treatment of endocarditis is essentially the same as that of pericarditis, except in respect to the administration of ammonia or its salts, which, in these cases, should never be omitted. It has been already stated that one of the most formidable lesions connected with endocarditis is the deposit of fibrin on the valves; and Dr. Richardson has ably shown that ammonia possesses the valuable property of keeping it in solution. Therefore, whilst employing the treatment recommended in the previous chapter as calculated to subdue pericarditis and other forms of cardiac inflammation, and administering alkalies and any other remedies which may be requisite for the removal of the disease in connection with which the endocarditis has arisen, the use of ammonia must never be lost sight of. Even if it will not check the progress of disease, it may serve to ward off the formidable mischief which results from the deposit of fibrin on the valves. If there be a tendency to sinking, with weak, fluttering pulse, and more or less delirium, recourse must be had to opium wine and diffusible stimulants.

The immediate prognosis of acute endocarditis is by no means unfavourable. If uncomplicated by pericarditis, the disease seldom proves fatal, unless accompanied by ulceration of the chordæ tendineæ, or by

* Dr. Kirkes' paper on this subject in vol. xxxv of the 'Med.-Chir. Trans.,' is well worthy of perusal. A remarkable case of this sort occurred in February of the present year in the Holland Ward of St. George's Hospital. The patient, a female named Jones, aged twenty-five, was seized with rigors and pains in the limbs on the 14th of February, 1862. On the following day she was admitted into St. George's Hospital, and after lying in a semi-conscious, half-delirious state for seven days, she died on the 21st of February. After death enormous pendulous fibrinous deposits were found attached to the mitral and semi-lunar aortic valves, and a branch of the internal carotid, the hepatic and the renal arteries were found plugged with portions of deposits which had become detached from the endocardial membrane. See the 'Post-mortem and Case-book' for February 21st, 1862, in the museum of St. George's Hospital.

softening and disintegration of the fibrinous deposits, both of which are rare occurrences. But it often arises concurrently with or during the progress of pericarditis, and then seriously aggravates the patient's danger.

Its more remote prognosis is of a far more serious nature. So delicate and so nicely adjusted is the valular apparatus of the heart, that any damage it sustains must necessarily interfere with the due maintenance of the circulation. To overcome the obstruction which the mischief occasions, the heart contracts more forcibly than natural, and as the damage is permanent, the increased labour of the heart is permanent also, and hypertrophy of the walls or dilatation of its cavities are necessarily produced, the relative degree of hypertrophy or dilatation being dependent on the firmness and contractility of the muscular tissue. Of course the rapidity with which the disease runs its course will depend in some measure upon the irritability of the heart, the state of the lungs, the habits of the patient, and other collateral circumstances, but, *cæteris paribus*, it will bear a tolerably constant relation to the degree of impediment offered to the circulation. The more serious the mischief the greater the obstruction to the blood's current, the quicker will hypertrophy and dilatation result, and the sooner will the effects of these lesions be perceived. When the obstruction is slight, though sufficient to induce distinct murmur, the patient may go on for many years with little or no palpitation or apparent cardiac disturbance, or he may suffer constantly from slight palpitation, or may be troubled with it only when the heart's action is augmented by mental excitement, gastric derangement, or active exercise. But if the obstruction be great, no long time will elapse before the patient's sufferings will become very serious. After a few months, or, possibly, two or three years, severe palpitation or dyspnœa will ensue, and the heart, struggling to overcome the resistance, will become gradually more and more dilated or hypertrophied, until, when it is unable any longer to fulfil its functions, the blood will begin to be arrested in its passage through the lungs, and cough and severe dyspnœa will result. Then will follow the terrible array of symptoms connected with an impeded pulmonary circulation—a loading of the capillaries with dark-coloured, imperfectly aerated blood, coldness and lividity of the extremities, blueness and lividity of the lips and face, congestion of the liver and other internal organs, and, as a natural consequence, dropsical effusion into the cavities of the chest, into the abdomen, into the pericardium, and into the cellular tissue of the body. Nor will these fearful changes occur

without producing a proportionate amount of suffering. Excessive palpitation, with præcordial pain and anguish, paroxysms of dyspnœa threatening suffocation, orthopnœa, constant restlessness and impossibility of sleep—these are among the inevitable consequences of such an embarrassed state of the vascular and respiratory systems, and are the symptoms immediately preceding death.

Endo-pericarditis, or inflammation both of the internal and external surfaces of the heart, is not an unfrequent occurrence, though it certainly is less common than has been represented by some authors. Taking rheumatic endo-pericarditis alone, I met with 27 examples of it at St. George's Hospital, amongst 114 cases of recent rheumatic heart disease, 12 of the other cases being instances of pericarditis alone, and 75 of endocardial affection alone.* I cannot refer to the same number of non-rheumatic cases, and cannot speak with equal certainty as to the date of the endocardial mischief; but I have notes of ten cases of endo-pericarditis, among fifty-four cases of non-rheumatic cardiac affection, twelve of the other cases being instances of pericarditis alone, and thirty-two of endocarditis alone. If the relative frequency of the various affections shown by these figures would hold good over an extended series of cases, it would appear that endocarditis, relatively to pericarditis, is more frequent in rheumatic than in non-rheumatic cases.

There is nothing distinctive in the general symptoms of endo-pericarditis, and even the physical signs are often uncertain, inasmuch as the sound of pericardial friction may for a time obscure or render undistinguishable the valvular murmurs induced by endocarditis, whilst in certain cases the effusion of a large quantity of liquid in the pericardium may not only weaken any existing valvular murmur, but may even excite one, and sustain it for many days.

The treatment of these cases is the same as that of pericarditis and endocarditis, and must be varied according to the nature of the disease from which the attack originates.

The prognosis is usually more unfavourable than in cases of pericarditis or endocarditis alone, and, as already pointed out in reference to those diseases, it varies, *cæteris paribus*, with the nature of the primary disease.

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* For full statistical details respecting rheumatic diseases of the heart, see my work 'On Rheumatism, Rheumatic Gout, and Sciatica,' ed. iii, pp. 257-284.

Little is known of acute carditis, except on a very limited scale, in connection with endo-pericarditis; for although some few cases are on record in which inflammation of the walls of the heart has resulted in abscess or purulent infiltration of its tissue, with bulging, or even perforation of the part affected, still such cases have never been diagnosticated during life, and their clinical history has yet to be determined. The general symptoms appear to have been those which are met with in pyæmia and other forms of blood-poisoning.

CHAPTER III.

CHRONIC DISEASE OF THE VALVES AND ORIFICES OF THE HEART.

In the description already given of valvular disease in connection with endocarditis, it has been shown that the valves are often damaged by the deposit of fibrin on their surface or edges. But it must not be inferred that valvular disease is invariably referable to the deposit of fibrin, or that it is always, or even generally, of inflammatory origin. Not only may fibrin accumulate on the valves, as the result of slow deposition from the blood, but malnutrition and chronic textural degeneration may take place in the valvular apparatus, just as in the other tissues of the body. It is especially prone to do so in the rheumatic and gouty diathesis, and in patients who are labouring under Bright's disease of the kidneys;* but it is often met with in after-life in persons whose kidneys are sound, and in whom it is impossible to trace the existence of a rheumatic or gouty diathesis. Hence the conclusion seems inevitable, that chronic disease of the valves may result from other forms of constitutional derangement than those above enumerated, which prove its most frequent source.

Whatever their precise character, these chronic changes in the valves are not accompanied by any special or distinctive symptoms during life. It is not even known whether they necessarily give rise to palpitation, or occasion any uneasiness in the cardiac region; and it is quite certain that physically the murmur which results from chronic disease

* In proof of this refer to 'Med.-Chir. Trans.,' vol. xxxi, pp. 183-213.

is undistinguishable from that produced by active inflammation. Pathologically, too, the results eventually may be much the same, whether the mischief began in active inflammation, or in slow deposition of fibrin, or in chronic degeneration of structure. In either case, the valves may ultimately become thickened, opaque, and puckered, and may be rendered rigid by the presence of atheromatous or calcareous matter; or they may yield under pressure, and become thinner than natural, or may even become cribriform or perforated;* or they may be ruptured or undergo ulceration; or their surface may be rendered irregular, or their edges beaded by the deposit of fibrin from the blood; or one segment of a valve may adhere to another segment of the same or some other valve, or two or more adjoining valves may cohere. In each of these cases, the disease may be of such a nature as to offer obstruction to the onward flow of blood, or to admit of regurgitation through the valves; and an obstructive or a regurgitant murmur will be the result.

So, again, with regard to the apparatus connected with closure of the valves. The valves themselves may be sound, but the papillary muscles may become diseased and inoperative, or the tendinous chords ulcerated, ruptured, contracted, or shortened, or the structure of the columnæ carneæ may degenerate, and their action may become inefficient. In all these cases regurgitation may take place which is not to be distinguished from that which occurs in connection with other forms of disease.

The same holds good with respect to the orifices of the heart. The valves may be sound, but the orifices which they guard may be roughened by calcareous or atheromatous deposit, or may be so much dilated that the valves may prove ineffective to close them, and may admit of regurgitation. In these cases, again, an obstructive or a regurgitant murmur may be produced which is clinically undistinguishable from a murmur resulting from valvular disease.

And yet again, the valve may be defective, not in consequence of active inflammation, or of slow degeneration, but as a result of congenital malformation. In these cases, as in the others, murmurs may exist which will not present any distinctive feature, and which during life can be attributed to their true cause only by reference to the circumstances under which they occur.

Clinically, however, the precise form of lesion is of little importance;

* It is doubtful whether this cribriform reticulated condition of the valves may not be referable to congenital malformation.

the practical question is, whether the disease, whatever it may be, is of such a nature as to offer obstruction to the onward flow of blood, or to admit of regurgitation. If it is not, it is impossible with any certainty to diagnose its existence during life; whereas, if it leads either to obstruction or regurgitation, the existence of such obstruction and regurgitation can be readily ascertained by the aid of the stethoscope, though the precise form of disease which produces the murmur must remain a matter for conjecture.

In all cases in which a permanent endocardial murmur exists, obstruction or regurgitation is unmistakeably indicated, and whether the disease from which it originated was of an acute or chronic character, is perfectly immaterial. Chronic disease of the valves leads as surely to cardiac hypertrophy and dilatation as acute disease has been shown to do.*

The practical question is as to the degree of impediment which is offered to the circulation, and especially to the pulmonary and systemic venous circulation, in consequence of the valvular obstruction or regurgitation. The greater the impediment to the cardiac circulation, the more rapid, *cæteris paribus*, will be the progress of hypertrophy and dilatation of the heart; and the greater the interference with the pulmonary and systemic venous circulation, the more distressing will be the symptoms, and the more rapidly fatal the consequences which ensue. If the injury to the valves be slight, and especially if it be of such a character as not to interfere primarily with the pulmonary or systemic capillary circulation, the patient may go on for many years unconscious of palpitation and of other apparent cardiac derangement; whereas, if it be excessive, and more especially if it be of a nature to impede the circulation through the lungs, or to induce congestion of the systemic capillary circulation, it may speedily give rise to fearful suffering which will terminate only with the patient's death.

It is needless to recapitulate the characteristics of the murmurs which occur at the different orifices of the heart. They have been fully described in a previous chapter, as have also the peculiarities of the radial pulse, by which they are severally accompanied.† But other changes occur in the physical signs to which it is necessary to refer. Thus, valvular disease, though not primarily and immediately productive of alteration in the position and extent of cardiac dulness, on percussion, yet after a time proves so indirectly, by inducing hypertrophy

* See Part iv, cap. ii, p. 556.

† Part iii, cap. iv, pp. 501-4 and 549-51.

and dilatation of the heart. In these cases, the enlargement and consequent dulness extends its limits to the right or to the left, according as the right or left chambers of the heart are affected. From the same cause, the area and force of cardiac pulsation are after a time increased, though the impulse in most well-marked cases is irregular both in force and rhythm.

But beyond this, it is necessary to observe that disease at the various orifices operates very differently in many particulars.*

Obstruction of the aortic orifice is a common form of mischief.† It has little effect in producing engorgement of the pulmonary capillaries, or in occasioning general systemic congestion and dropsy; nay more, the greatest constriction of this orifice may be unattended by these symptoms. It is not until after the left ventricle has become dilated, and the mitral valve consequently so inefficient as to admit of regurgitation, that symptoms of pulmonary and systemic obstruction begin to manifest themselves. Aortic constriction therefore, *cæteris paribus*, is the least rapidly fatal form of valvular disease. In cases of moderate obstruction the character of the pulse is not materially altered; but when the obstruction is great, the pulse is small, though regular in force and frequency. When the action of the heart is forcible, and the obstruction is rough, and of a nature to cause excessive eddying of the blood, it may give rise to thrill at the base of the heart, and in the track of the aorta and its branches.

Obstruction of the mitral orifice is comparatively rare.‡ It necessarily induces dilatation of the left auricle, and not unfrequently hypertrophy also, with pre-systolic auricular impulse. At the same time, the action of the heart becomes rapid and tumultuous, the pulmonary system becomes congested, and the right ventricle and pulmonary artery are dilated and hypertrophied in consequence. It is therefore productive of severe cough, breathlessness, and other peculiarly distressing symp-

* As evidence of the relative frequency with which the various valves are affected, I may refer to the valuable statistics collected by my colleague, Dr. Barclay, from the records in the Museum of St. George's Hospital. He has shown that amongst seventy-nine cases of valvular disease the mitral and aortic valves were both affected in thirty-six instances; the aortic was affected alone in twenty-six, and the mitral alone in seventeen instances. In this series of cases the valves on the right side of the heart were not once affected. (See 'Med.-Chir. Trans.,' vol. xxxi.) I would also refer the reader to Dr. Ormerod's admirable 'Gulstonian Lectures on the Pathology of Valvular Disease of the Heart.'

† For the characteristics of obstructive aortic murmur, see *ante*, pp. 500 and 550.

‡ For the characteristics of obstructive mitral murmur, see *ante*, pp. 503 and 550.

toms, and proves rapidly fatal with congestion and œdema of the lungs, and not infrequently with pulmonary apoplexy. The pulse is small, but not necessarily irregular.

Obstruction of the pulmonary orifice is seldom met with.* It does not influence the radial pulse, nor does it in the first instance produce congestion of the venous system, but it leads after a time to hypertrophy and dilatation of the right ventricle, and in this way ultimately gives rise to regurgitation, through the tricuspid orifice, with turgescence and pulsation of the large veins in the neck. Until this occurs, it does not occasion urgent or distressing symptoms.

Obstruction of the tricuspid orifice is exceedingly rare.† I have only met with one instance in point, and practically therefore can say little about it, but theoretically, it would lead to hypertrophy and dilatation of the right auricle and excessive congestion of the venous system, unaccompanied by any visible pulsation in the neck. Somnolence, headache, and dropsy, would be amongst its most prominent symptoms. The radial pulse would not be primarily influenced by its occurrence.

Regurgitation through the aortic orifice is not an uncommon form of valvular disease.‡ It speedily gives rise to hypertrophy of the left ventricle, but does not produce embarrassment of the pulmonary circulation, until dilatation of the left ventricle has reached a point at which the mitral valve proves unequal to close the dilated auriculo-ventricular opening, and admits of regurgitation through it. Therefore for a considerable length of time it does not materially impede the systemic capillary circulation, and fails to produce dropsy or other distressing symptoms.

It is accompanied by a peculiar and characteristic pulse. It is not the small pulse of aortic obstruction, nor the unequal and irregular pulse of mitral regurgitation; but in its most marked and most striking character it is the unsustained pulse of unobstructed arteries. There is, in this case, no impediment to the onward current of the circulation, no lack of blood to fill the vessels, and no deficiency of force to propel it; but from the insufficiency of the aortic valves, and the consequent reflux of blood into the ventricle, the prolonged swell which

* For the characteristics of obstructive pulmonary murmur, see *ante*, pp. 500.

† For the characteristics of obstructive tricuspid murmur, see *ante*, p. 504.

‡ For the distinctive characters of aortic regurgitant murmur, see *ante*, pp. 502 and 551.

at each systole of the heart is naturally imparted to the blood in the vessels is not sustained; the successive waves of blood, therefore, are short and abrupt, and hence the pulse is jerking, and gives a sensation as if successive balls of blood were being shot suddenly under the finger. When regurgitation exists only in a slight degree, this character is not strongly marked, but so strong is the reflux when the valvular mischief is great, and so strong is the jerking to which it gives rise, that the motion occasioned by it may be seen even at the wrist, and may be felt in almost any part of the body.

Regurgitation through the mitral orifice is, perhaps, the most common form of valvular disease.* It produces hypertrophy of the left ventricle and dilatation of the left auricle, and may lead to systolic auricular impulse at the second intercostal space, by admitting of the transmission of the impulse from the ventricle. Further, when excessive, it may cause a vibration, or thrill, or purring tremor, which will be perceptible on the chest walls in the region of the heart, but which being occasioned by a backward instead of an onward current of blood, is not transmitted to any extent along the aorta or great vessels. When the disease of the mitral valve is of such a nature as to cause obstruction to the flow of blood into the ventricle, as well as to admit of regurgitation from the ventricle, the left auricle usually becomes hypertrophied as well as dilated, and then pre-systolic auricular impulse is sometimes perceptible at the second left intercostal space, occasioned by the systole of the auricle. Mitral regurgitation primarily interferes with the circulation through the lungs, and produces cough, and dyspnœa, and other symptoms of pulmonary congestion. It is in these cases especially that the outpouring of blood into the lungs, constituting pulmonary apoplexy, is most commonly observed.

The pulse is quite characteristic of the disease. It is not the small pulse of aortic obstruction, nor the jerking pulse of aortic regurgitation, but it is irregular in rhythm, and unequal in force and fulness, in consequence of the constant variation in the quantity of blood which at each contraction of the ventricle regurgitates or makes its way back through the imperfect valves into the left auricle.

Regurgitation through the pulmonary orifice is very rare, so that the effects produced by it have scarcely been verified by clinical observation.† Theoretically, however, the small and constantly varying

* For the distinctive characters of regurgitant mitral murmur, see *ante*, pp. 500-1, and 550.

† For the distinctive characters of regurgitant pulmonary murmur, see *ante*, p. 503.

quantity of blood which reaches the lungs at each systole of the ventricle should occasion dyspnœa, whilst as the right ventricle would become hypertrophied and probably dilated, regurgitation through the tricuspid orifice would after a time ensue, and congestion of the systemic and cerebral capillary circulation would take place, producing somnolence, headache, and dropsy. The mischief being on the right side of the heart, the radial pulse would not be materially affected.

Regurgitation through the tricuspid orifice as a result of disease of the tricuspid valve is of rare occurrence, but it is not uncommon as a consequence of dilatation of the right ventricle, in many of which latter cases it is unattended by murmur.* When it occurs, the right ventricle becomes hypertrophied, the right auricle dilated, the venæ cavæ distended, and there is a strong tendency to congestion of the systemic and cerebral capillary circulation. Not unfrequently the larger veins are so much distended that their valves become incompetent to prevent the reflux of blood, and then venous pulsation is visible in the neck, and venous thrill may be perceptible to the touch; but if the regurgitation be slight, or if the right ventricle be very weak, venous pulsation may not occur, and venous thrill will almost certainly be absent. The pulmonary circulation remains unobstructed, and the radial pulse is not materially affected, but somnolence, headache, and dropsy are more constant and distressing than in any other form of valvular disease except obstruction of the tricuspid orifice.

It will be observed that the symptoms which result from valvular disease are mainly dependent on one circumstance, viz., the impediment produced in the pulmonic and systemic capillary circulation. So long as there is no serious obstacle to the onward flow of blood through the heart, and no regurgitation of blood through the mitral and tricuspid valves, producing engorgement of the lungs and congestion of the systemic capillaries, no urgent or distressing symptoms arise. Hence it is that aortic and pulmonary obstruction, and aortic and pulmonary regurgitation, are less rapidly productive of distress than the same forms of disease at the mitral or the tricuspid orifice, and hence also it is that the existence of hypertrophy and dilatation of the various chambers of the heart—a circumstance which in certain forms of valvular disease has been shown to exercise an important influence in producing reflux of blood through the mitral and tricuspid valves—is found to have a marked effect on the progress and duration of valvular disease. Each form of lesion runs its own special course, and in its early stages

* For the distinctive characters of regurgitant tricuspid murmur, see *ante*, p. 501.

gives rise more or less to its own special train of symptoms, the distinctive features of which are referable principally to the greater or less degrees of pulmonic or systemic capillary congestion. But in their more advanced stages all forms of valvular mischief result in the production of a certain amount of capillary engorgement, and, therefore, have many symptoms in common. Those which more prominently attract attention are, oppression at the chest, breathlessness, and speedy exhaustion on exertion, a general sense of lassitude, headache, restless and disturbed sleep, with frequent starting, and frightful dreams, cough, palpitation, dropsy, and occasional pain in the region of the heart, sometimes amounting to severe angina. The rapidity with which cough ensues is mainly dependent on the tendency to pulmonary engorgement, and varies, therefore, with the amount of mitral obstruction or regurgitation; whereas the rapidity with which headache, restlessness, disturbed sleep, and dropsy supervene, is proportioned to the amount of systemic capillary congestion, and varies, therefore, with the extent of tricuspid obstruction, or tricuspid regurgitation. In the more advanced stages of all forms of valvular disease, but especially when there is extensive tricuspid regurgitation, the kidneys and other internal organs are apt to become gorged with blood, and an albuminous condition of urine may result.

As the symptoms above described may arise from either obstructive or regurgitant disease of a single valve, so they may be produced even more speedily by a combination of obstructive and regurgitant valvular disease. It matters little whether the obstruction and regurgitation result from disease of a single valve, or from obstructive disease of one valve, and regurgitant disease of another. In either case the impediment to the cardiac and the capillary circulation is necessarily great, and the resulting mischief is rapidly induced. In these cases murmurs are audible, referable both to obstruction and regurgitation; in short, there are double murmurs, the one due to the eddying of the onward current of blood, the other to regurgitation through an ill-closed, patulous orifice. These murmurs under ordinary circumstances correspond very closely with the same murmurs when occurring singly. Thus, the position and character of a systolic mitral murmur are usually the same whether the murmur exists alone, or is followed by the murmur of mitral obstruction. But in some instances of double murmurs the systolic murmur is so prolonged as to mask and render inaudible the diastolic murmur which follows; in others, in which two

distinct murmurs occur synchronously at different orifices, the one may mask or overpower the other, so that, for instance, a mitral regurgitant murmur may render a tricuspid regurgitant murmur inaudible. In others, again, the coexistence of obstruction and regurgitation may materially modify the character of a murmur, so that, for instance, the occurrence of extensive mitral regurgitation may so far weaken the stream of blood flowing into the aorta, as to diminish or even put a stop to a murmur which would otherwise have resulted from obstruction at the aortic outlet.

But there are still further difficulties in the way of a diagnosis of valvular disease. The heart may be displaced by pleuritic effusion, or by some other form of intra-thoracic disease, or it may be hypertrophied and dilated. Thus, before any inference can be drawn as to the source of a murmur, the position of the heart itself must be determined, and due allowance made for its altered size, and for any displacement it may have undergone.

Again, the seat at which the various murmurs are heard of maximum intensity may undergo material alteration, in consequence of changes in the texture of surrounding parts. Thus, when the anterior margin of the right lung is emphysematous, an aortic murmur may be heard better at the left base than at the right, and this is more particularly the case when the left lung is partially solidified. The reverse holds good in regard to a pulmonary murmur, when the anterior margin of the left lung is emphysematous and the right lung solidified.

Again, if the patient be not seen until after the heart's action has become turbulent and irregular, it is sometimes impossible to determine the rhythm of a murmur, and equally so, therefore, to feel certain as to its nature. But these are difficulties which rarely occur, and, although cases are occasionally surrounded by causes of doubt and perplexity, calculated to baffle even the most experienced observer, yet, in the majority of instances, due attention to the rules already laid down, relative to the diagnosis of the various murmurs—to the character and position of the abnormal sound, to the direction in which, and the period of the heart's action at which it is heard, to the state of the arterial pulse, and to the nature of the accompanying symptoms—will enable a cautious and intelligent practitioner to arrive at a diagnosis which *post-mortem* investigations will fully verify.

The prognosis of valvular disease is a subject beset with the greatest difficulty. It has been already stated, that, *cæteris paribus*, it is most

unfavourable in cases of mitral and tricuspid regurgitation, and least so in cases of aortic obstruction. But these bald facts afford no measure of the average rate of progression of the disease, nor any clue to a test by which its duration in any given case can be estimated. The stethoscope informs us of the existence of a murmur, and careful observation will generally enable us to arrive at a correct conclusion as to the fact of that murmur being dependent on functional or organic derangement. But an organic valvular murmur is not inconsistent with a considerable prolongation of the term of life, and the intensity of a murmur does not afford trustworthy evidence as to the amount of the organic disease, or as to the degree to which the circulation is obstructed. Nay, an excessive amount of disease, by producing excessive obstruction, and so diminishing the force of the blood's current, may cause the cessation of valvular murmur, whilst, on the other hand, tricuspid regurgitation, which is the form of disease which proves most rapidly fatal, is precisely that which least constantly gives rise to murmur. The facts appear to be what careful observation might lead one to infer, viz., that the rapidity with which any form of valvular disease will prove fatal must depend upon—

1st. The precise character of the disease; that form of disease which is most rapidly productive of systemic or pulmonic capillary congestion being that which will prove most rapidly fatal.

2ndly. The extent of the disease; the obstruction being greater, and the effects of obstruction more rapidly induced, in proportion as the disease is extensive.

3rdly. The state of the heart as regards dilatation and hypertrophy; valvular disease being more rapidly fatal when it occurs in a heart which is already dilated and hypertrophied than when it is set up in a healthy heart, inasmuch as dilatation and hypertrophy of the heart have a tendency to induce tricuspid and mitral regurgitation, and so, indirectly, to promote systemic and pulmonic capillary congestion.

4thly. The state of the heart as regards its textural integrity; inasmuch as valvular disease runs a less rapid course when the heart is texturally sound, and therefore little prone to dilatation, than when its muscular fibre is weak or in a state of degeneration, so that its cavities readily dilate under the extra strain put upon them, and thus very speedily admit of increased regurgitation.

5thly. The textural integrity of the arteries; inasmuch as when the coats of these vessels are loaded with atheromatous or calcareous

deposits, their elasticity is impaired, and a great extra strain on the heart results.

6thly. The condition of the blood; inasmuch as unhealthy blood by leading to malnutrition of the heart and of the tissues generally, with congestion of the systemic capillaries, not only weakens the heart and renders it irritable, but at the same time subjects it to an extra strain, and so conduces to dilatation of its cavities.

7thly. The state of the lungs and the other viscera; for the reason that disease or irregular action of any of the principal organs of the body is itself productive of, more or less, impediment to the circulation, and becomes a serious hindrance to the action of the heart when that organ is in any way deranged.

8thly. The regularity of the various secretions; the fulness of the vessels and the poisoned condition of the blood which result from defective excretion being provocative of deranged cardiac action.

9thly. The mode of life which the patient pursues; the strain on the heart being infinitely smaller, and the tendency to dilatation and so to increased regurgitation and capillary congestion, far less when the patient leads a quiet life and avoids everything which is likely to excite the action of the heart, than when he adopts an opposite course, and, either by bodily exercise or emotional excitement, makes a large demand on the action of the damaged organ.

Accordingly, if the heart be healthy at the date of the occurrence of valvular mischief, if the extent of that mischief be not excessive, if the blood be of a normal character, if the viscera be healthy, and the secretions free, and the patient's mode of life regular, temperate, and sedentary, there is scarcely any limit to the time to which life may be prolonged; whereas, if the heart be hypertrophied and dilated at the time when the valvular lesion occurs, if the blood be spanæmic, if the patient's lungs or other viscera be unsound, or his secretions irregular or defective, or, again, if he leads a laborious life, or habitually takes active, straining exercise, then, even though the valvular lesion be of precisely the same character and extent as in the former instance, the disease would run a rapid course, and prove fatal probably within two or three years. In any and all cases life is apt to be arrested suddenly by syncope.

The treatment of valvular disease of the heart is necessarily palliative, not curative. Its aim or object is to regulate the action of the heart, by moderating undue action, or, by supplying the stimulus essential to more forcible action in a weakened organ, to control the tendency to local congestion, and to mitigate or remove the symptoms resulting

from cardiac derangement. More than this it cannot effect; for nothing will avail to remove organic valvular lesion. Practically, therefore, the points to be decided are—1st, whether the action of the heart is too forcible, or too feeble; 2ndly, whether any pulmonary or other local congestion calls for immediate relief; 3rdly, whether dropsy is producing urgent distress; and 4thly, whether any, and if so, which, of the excretory organs are defective in action. If the patient be plethoric, the heart's action turbulent, and its impulse forcible, moderate venesection, or the abstraction of blood by cupping between the shoulders, followed by dry cupping in the same situation, will often afford immediate relief to the palpitation and oppression at the chest. But though the moderate abstraction of blood is serviceable in these cases, repeated or profuse venesection is dangerous, as calculated to excite irritability of the heart, impoverish the blood, and induce dropsy. And, if the patient be pallid and of lax fibre, and the heart's impulse be feeble, then general venesection is not admissible, notwithstanding the existence of oppression at the chest, and of uneasiness in the region of the heart. The utmost which can be safely done by bloodletting in such cases, will be effected by the application of a few leeches to the præcordial region; and very generally relief will be obtained even more readily by purgatives, aided by dry cupping, mustard poultices, or turpentine fomentations.

When active congestion of the lungs exists, venesection, or cupping, dry cupping, blisters, and sinapisms, are imperatively called for, and expectorants may be administered in aid of other remedies. But it must ever be borne in mind in the treatment of this form of pulmonary congestion, that the mischief we have to combat is essentially cardiac, not pulmonary, and that remedies which will relieve the cardiac distress will at once mitigate or remove the oppression on the lungs. Hence, although in some few instances ipecacuanha, or tartarized antimony, may be useful, and, in others, squills, senega, and the more stimulating expectorants, our chief reliance must be placed on bloodletting, dry cupping, counter-irritation, and the use of internal remedies calculated to stimulate increased excretion, and to regulate the action of the heart. If the bowels be costive, a brisk purgative must be given; if the liver be engorged, and the conjunctivæ yellow, the administration of a few doses of calomel or blue pill, in combination with or followed by a purgative, will be essential to the action of other remedies; if the kidneys be congested, and the urine scanty and albuminous, dry cupping across the loins, or a warm bath, will often prove extremely serviceable, removing all trace of

albumen, and promoting free diuresis; but if they fail, saline and other diuretics must be administered. Dyspnœa and paroxysmal shortness of breath may be relieved by a cough drop containing æther and lobelia, stramonium, hydrocyanic acid, belladonna, or hyoscyamus, whilst, if the stomach be much deranged, as it usually is in cases of heart disease, owing to the obstruction in the portal circulation, occasional doses of blue pill with antacids, light vegetable bitters, and warm carminative medicines must be had recourse to. Sometimes in these cases hæmatemesis occurs, or hæmorrhage takes place from the bowels, and gives considerable relief.

The action of the heart must be carefully regulated. A belladonna plaister should be applied to the cardiac region, whilst recourse is had to the internal administration of the veratrum viride, aconite, hyoscyamus, and other remedies which exert a sedative influence over the heart. Opium, which allays the nervous irritability, on which palpitation in some measure depends, may also be given, if the patient be excited and unable to obtain refreshing sleep. But, inasmuch as opiates are prone to derange the stomach, and interfere with or arrest secretion, their administration should be avoided if possible. If the heart be weak, and its impulse feeble and irregular, no treatment can be satisfactory which does not involve the administration of digitalis. It stimulates the heart, and regulates its action, augments the tone and contractility of the vessels, increases the flow of urine, and exerts a restorative and calming influence on the system which is not to be attained by any other means.*

Seldom, however, are these measures of more than temporary service in patients who are pallid, and whose heart's impulse is feeble. Under these circumstances, iron is needed to give effect to the other treatment. In no form of disease are the effects of the judicious exhibition of this medicine more strikingly displayed. If taken for some months in combination with diuretics and light bitter infusions, whilst, at the same time, the condition of the liver and alvine evacuations is carefully attended to, and the force and regularity of the heart's action sustained by the administration of digitalis, the results are often most satisfactory. The palpitation subsides, the dyspnœa and breathlessness pass off, the pulse becomes more regular, and of better strength, the appetite improves, and the aspect and sensations of the patient betoken returning health.

An essential element in the treatment of cardiac disease is the careful

* This is contrary to received doctrines, but it is true, nevertheless, in proof of which I would refer the reader to pp. 592-3 of this treatise.

regulation of the patient's mode of life. Excitement of all kinds must be systematically avoided, and so must active exercise, and whatever puts a strain upon the heart and induces the slightest palpitation or shortness of breath; the bowels must be regulated so that there may be no straining at stool; and care should be taken to avoid stooping and all other postures which may lead to mechanical interference with the heart's action, by inducing upward pressure of the abdominal viscera. The diet should be light and nutritious, but moderate in quantity, so as not to induce distension of the stomach, and wine and spirits may be taken in quantity sufficient to aid digestion, but not to accelerate the action of the heart.

After a time, however, the cardiac and general derangement will probably attain a point at which effusion of the watery parts of the blood will take place into the subcutaneous cellular tissue as a relief to the congested venous system, and dropsy will occur and will urgently claim attention. The fluid necessarily gravitates into the most dependent portions of the body, and thus œdema of the feet and ankles will take place before swelling is perceived in any other part of the body. So long as the swelling is slight, and confined to the feet and ankles, little heed need be taken of it. The remedies which are applicable to the relief of the other symptoms will probably, after a time, effect its removal. But, when anasarca shows a disposition to increase, and extends upwards into the legs and thighs, there is reason to apprehend that if the venous congestion be not speedily relieved, serous effusion will take place into the lungs, or into the pleura, the pericardium, or the peritoneal sac; or, that the skin of the legs, distended to an extreme degree, will crack and ulcerate, or even slough, and that thus very serious aggravation of the patient's sufferings and danger will occur. Under these circumstances the first consideration is how to get rid of the fluid accumulations which have already taken place, and to put a stop to their recurrence. Up to this time the treatment has been palliative, and the remedies which have been employed have been administered with a view to gentle and gradual action. But when dropsy has occurred to a considerable extent and effusion into the lungs or into the serous cavities is imminent, there is no time for dallying or for the exhibition of remedies which cannot be expected to effect their object for many days, or even weeks. Relief, if it is to be obtained at all, must be purchased by vigorous and immediate action. Pulmonary and renal congestion must be relieved by repeated dry cupping, aided, if the pulse

be forcible, by the local abstraction of a few ounces of blood; the various excretory organs must be stimulated to the utmost, and the strength of the patient husbanded by sleep, and supported by food and stimulants. Hydragogue cathartics, such as gamboge, elaterium podophylline, the bitartrate of potash, and the pulvis jalapæ compositus of the pharmacopœia, should be given on alternate days, to ensure a copious watery flux from the bowels; and, if the liver be congested, and the kidneys healthy, a pill, containing squill, digitalis, and blue pill, should be administered twice or three times a day; the action of the kidneys should be solicited by diuretics, of which the nitrate of potash, acetate of potash, and acetate of ammonia, the iodide of potassium, digitalis, squills, cantharides, nitric æther, juniper, scoparium, erodium cicutarium, triticum repens, and chimaphila, are usually the most serviceable; and the skin's action may be cautiously stimulated by a vapour or hot air bath. At the same time, probably, stimulants will be needed to counteract the depressing influence of the watery flux which will be excited by this treatment, and nothing answers better than gin, hollands, and whiskey. Not only do they act as general stimulants, but, in many instances, they promote the action of the kidneys, and tend, I believe, to obviate the disposition to further serous effusion, by rousing the capillary circulation.

Sometimes, when anasarcous distension has reached a certain point, the tension of the venous system appears to be so great as not to admit of relief by internal remedies. In these cases, our utmost diligence will not avail to relieve the circulation and repress the continuance of effusion; the medicines fail utterly to induce diuresis, or to tranquillise the action of the heart; the breathing becomes exceedingly oppressed, inflammatory redness begins to show itself on the legs, and it becomes obvious that, unless relief be speedily afforded, the patient must succumb to his malady. In these cases scarification and acupuncturation of the legs have been proposed, and are often practised. But repeated observation at St. George's Hospital and elsewhere has taught me that they are dangerous expedients, especially when the kidneys are diseased, and should only be resorted to as a last resource. The former, especially, is almost certain under such circumstances to be followed by erysipelatous inflammation and sloughing; and in the majority of cases, unless the kidneys are sound, the same results ensue after the employment of acupuncturation. Further, I believe they are rarely necessary. Before distension has reached the point which justifies the adoption of mechanical

relief, the skin usually cracks, and an oozing of fluid at first glutinous, but afterwards watery, takes place; and if this spontaneous relief is judiciously promoted by warm flannels and other means, the fluid which has accumulated in the cellular tissue will be evacuated as surely, yet more gradually and more safely than by any artificial means. Nevertheless, if this weeping of the legs does not take place spontaneously, and mechanical relief is necessary, recourse may be had to acupuncturation. It is far preferable to scarification, and when the kidneys are sound, may be employed with a fair prospect of affording relief. If the punctures are made at a considerable distance from each other, there will not be any great risk of inflammation, and the serum which has collected in the cellular tissue will drain off in large quantities.

The congested condition of the stomach and intestines consequent on the obstruction to the portal circulation renders digestion weak, impedes nutrition, and deranges the biliary and intestinal secretions. The diet, therefore, should be light and nutritious; and, as the system is enfeebled by long-standing disease, stimulants in full doses are almost always needed.

CHAPTER IV.

HYPERTROPHY OF THE HEART.

The term "hypertrophy," as applied to the heart, is meant to express augmentation in the bulk and weight of that organ—an increase in its muscular tissue. The increase which takes place is not in the thickness of the primative fasciculi of the muscular fibre, but in their number, which sometimes increases so prodigiously, that a heart which should weigh from nine to ten ounces, has been known to weigh upwards of forty ounces.

Hypertrophy may be either simple or eccentric. In the former case the walls of the affected cavities may become thickened, whilst the cavities themselves remain of their natural size; in the latter, both the walls and the cavities enlarge, but retain their normal relative proportions. When the hypertrophous tissue is of a weakly, unhealthy character, or has undergone partial degeneration, the walls often yield, and the cavities become dilated to an extent which is not otherwise observed;

and, in these cases, the disease is not simply hypertrophy, but rather a combination of hypertrophy and dilatation. And inasmuch as when the heart has long been diseased its nutrition is apt to be interfered with and its muscular structure to undergo degeneration, this combination of hypertrophy and dilatation is the form in which hypertrophy is most commonly met with in practice.

A very contracted state of heart is sometimes observed after death—a state, in which the walls appear to be thicker than natural, whilst, at the same time, the cavities are smaller. This was formerly termed "concentric hypertrophy;" but, it is, in fact, a mere *post-mortem* appearance, resulting from the forcible contraction in *articulo mortis* of a healthy or a simply hypertrophied heart. Cruveilheir informs us that it is invariably met with in the bodies of criminals who have been guillotined, and it is also observed in persons who have been suddenly killed by accident, and occasionally in cases of phthisis and anæmia.

Whatever the form it assumes, hypertrophy may affect the entire heart, or may be confined to any portion of that organ. In most instances it is accompanied by dilatation, and involves both sides of the heart, the left side being that on which the hypertrophy is most apparent; but when it is local, its most common seat is the left ventricle, and after that the right ventricle; the left auricle is its next frequent seat, and then the right auricle. The aortic and pulmonary outlets increase in width, according as the right or left ventricle is affected, the semilunar valves enlarge in a corresponding degree, the mitral and tricuspid valves and the carneæ columnæ become hypertrophous, and the coronary arteries and nervous ganglia increase in size.* When hypertrophy exists in a marked degree, the form of the heart and its position in the chest are remarkably altered. Instead of being conical in shape, it becomes globular, or more or less imperfectly square; the natural projection of the apex disappears, and the entire organ is sometimes even broader than it is long. Its position undergoes a corresponding change; it lies more horizontally or transversely than natural in the chest, so that its apex pulsates considerably to the left, instead of to the right of the left nipple, and, in extreme cases, it may extend from the second intercostal space above, to the seventh, or even the eighth rib below. The precise form and position, however, which an hypertrophied heart assumes, in any particular case is regulated by

* See the monogram on the 'Nerves of the Heart,' by my colleague, Dr. Robert Lee.

the extent and position of the hypertrophy; the enlargement taking place in one direction or another, according as the hypertrophy is general or is confined to either ventricle or auricle.

It is sometimes difficult to determine by mere inspection whether a heart is or is not hypertrophied; and, in order that a just estimate may be formed, it is necessary that some conclusion should be arrived at respecting its size and weight in health. From the nature of the case, it is impossible to fix a healthy standard; but for all practical purposes the weight of a healthy heart may be regarded as averaging rather more than nine ounces, or as varying in different cases from eight to ten ounces, the weight being less in women than in men, but in both sexes increasing gradually as age advances. Between the ages of thirty and forty-nine the thickness of the walls of the

	In men.	In women.
Left ventricle, average at the base .	0·43 inches	0·36 inches.
„ „ at the middle	0·45 „	0·39 „
„ „ near the apex	0·31 „	0·28 „
Septum of the ventricles at the middle	0·43 „	0·39 „
Right ventricle average at the base	0·16 „	0·15 „
„ „ at the middle	0·11 „	0·11 „
„ „ near the apex	0·08 „	0·08 „

Nevertheless, these figures can only be regarded as giving approximate results, for not only do the relative size and weight of the heart vary in different persons, but the walls of a perfectly healthy heart vary considerably in thickness in different cases and at different ages.*

The causes of hypertrophy, whatever their precise nature, have one element in common: they all operate as incentives to increased cardiac action. This, in truth, is their essential character. Without an increased demand on the force of the heart's action, hypertrophy would never arise; whereas it is an intelligible and necessary result of any long-continued and abnormally forcible cardiac pulsation. Hence it is that, in the majority of cases, hypertrophy implies the existence of obstruction to the current of the blood; for in such cases the heart is stimulated to increased exertion, with a view to adapt itself to the altered mechanism of the circulation; and the result of that unwonted force of action is the same in the heart as in other muscles, viz., increased development of structure.

* The above are Bizot's admeasurements given in Dr. Stokes' work on 'Diseases of the Heart,' note, pp. 257-8.

The sources, however, of increased cardiac action are very numerous. They may be mental or emotional; they may originate in the habits of the individual; they may exist either in the heart itself or in the vascular system; they may be traced to the condition of the blood, or they may arise in connection with a disordered state of the lungs, kidneys, or other organs. Long-continued and frequently recurring mental excitement, producing palpitation; the continued and inordinate use of stimulants; excessive and violent bodily exercise; disease of the valvular apparatus of the heart; disease of whatever character affecting the vascular system, whether constriction of the vessels, consequent on pressure from without, or on deposits within their coats; or dilatation, aneurismal or otherwise, of the arteries, especially of the aorta near the heart; or diminished elasticity of their coats, produced by atheroma, whether in the larger arteries or in the capillary system of vessels; an impediment to the capillary circulation such as is produced by the condition of blood which exists in Bright's disease, and other forms of constitutional disorder; or disease, especially of a chronic nature, in the lungs, kidneys, or other organs, calculated to interfere with the capillary circulation.

When the causes of increased cardiac action are of a general character, such as mental emotion, undue indulgence in stimulants, and excessive exercise, the hypertrophy is also general, and affects all parts of the heart. When, on the contrary, they are of a local character, such as valvular disease, or disease of the lungs, hypertrophy is first developed in those portions of the heart which are immediately behind the seat of obstruction. Thus, when the obstruction is seated at the aortic outlet, or in the aorta or its branches, the left ventricle becomes hypertrophied; when the lungs are diseased, and the pulmonary circulation is impeded, the right ventricle suffers. Hence it is that hypertrophy is developed in the left ventricle more frequently than in other parts of the heart; for not only does valvular disease occur almost universally on the left side of the heart, but disease of the large arteries, and obstruction of the capillary circulation would also react primarily upon the left ventricle.

After what has just been stated it may at first sight appear difficult to explain the fact that the right side of the heart not unfrequently becomes hypertrophied, even when the lungs are perfectly sound, and when no obstruction exists at the pulmonary orifice. But a little consideration will serve to explain the apparent anomaly. Any impediment to the flow of blood from the left cavities of the heart must necessarily, to

some extent, retard the passage of the blood into those cavities, or, in other words, impede its flow from the lungs. The necessary result is, that the pulmonary capillaries become congested, and an unwonted strain is thrown on the right ventricle; and when the right ventricle has become hypertrophied and dilated, a check is also received to the flow of blood from the right auricle, which, together with the vena cava, becomes distended in consequence. In some instances, the effects of obstruction at the aortic outlet may be traced even further backwards, and evidence adduced to show that the impediment is felt throughout the whole circle of the circulation, so that at length the left ventricle may have to contract with additional force to overcome an obstruction of the capillary circulation, which has arisen as a direct consequence of disease at its aortic outlet.

The physical signs of hypertrophy of the heart necessarily vary with the seat and amount of the affection. Thus, when hypertrophy is general, yet moderate in degree, inspection will seldom afford evidence of its existence in adults; but in early life there may be a fulness and prominence of the præcordial region, with widening, but not bulging of the intercostal spaces. The action of the heart is regular, the extent of visible impulse is ordinarily increased, and the apex pulsates somewhat to the left of the nipple, and as low as the seventh rib; the character of the impulse is that of forcible heaving; the area of dulness on percussion is extended, and the parietal resistance increased; the first sound is dull, muffled, and prolonged, and in some instances is almost inaudible at the apex, though it may still be heard at the base, and especially at the right base and over the aorta; the post systolic silence is almost wholly absent, and the second sound is loud, but deficient in clearness.

In hypertrophy with dilatation, when extensively developed, there is an increased tendency to fulness and prominence of the præcordial region, with widening of the intercostal spaces. The action of the heart is still regular, but the extent of visible impulse is enormously increased, reaching sometimes from the third intercostal space quite down to the epigastrium; the apex may be seen beating as low as the eighth or ninth rib, and usually to the left of the nipple, its impulse being sometimes of a knocking character, but more commonly heaving, and so forcible as to raise the head of the auscultator when applied to the stethoscope, or even to shake the bed on which the patient lies. The parietal resistance on percussion is increased, and so also is the

area of dulness, which may extend vertically from the second or third intercostal space as low as the seventh or eighth rib, and laterally from an inch or even more to the right of the sternum to two inches or more to the left of a line drawn vertically from the left nipple. The first sound is loud and usually prolonged, but wanting in clearness—its prolongation and clearness being determined by the relative amount of the hypertrophy and the dilatation; the post systolic silence is shortened, and the second sound is loud and full. The sounds are transmitted over an unusually extended surface of the chest, and may not only be heard on the back, but may be audible even to the right of the spine.

When, as often happens, hypertrophy is almost confined to the left ventricle, the physical signs do not notably differ from those already described, except that the displacement of the heart, the apex-beat and the area of dulness on percussion are perceptible further to the left of the nipple, in the direction of the left ventricle, than they are in cases of general hypertrophy, and that though the first sound may be dull, muffled, or even inaudible at the left apex, it may be distinctly heard at the right apex. When, on the other hand, hypertrophy is confined to the right ventricle—a very rare occurrence—the heart is felt pulsating against the ensiform cartilage, immediately below, and to the left of it; the area of dulness extends considerably to the right of the sternum, and the first sound, though dull and muffled at the right apex, is sufficiently distinct at the left apex. At the base of the heart the second sound is fuller and more intense than usual. When, in connection with hypertrophy and dilatation of the right ventricle, the tricuspid valve becomes inefficient and admits of regurgitation, a tricuspid regurgitant murmur is sometimes, but by no means always, audible, and there is distension and occasionally pulsation of the veins of the neck.

The symptoms produced by hypertrophy of the heart are various, and depend more upon the coexistence of dilatation than upon the extent of the hypertrophic enlargement. When dilatation is slight in amount, and the hypertrophy considerable, the patient generally wears the aspect of health, his face being florid; but he suffers from dyspnœa, palpitation, and oppression at the chest, on violent exertion, as on running up stairs, or walking quickly up hill, though he can take moderate exercise without inconvenience. The radial pulse is natural in frequency, regular in rhythm, strong, full, and resisting; the action of the carotids is visible, especially after any excitement; and he experiences a throbbing

in the head and neck, with a singing in the ears, under the same circumstances. He generally prefers lying with his head high, has frequent startings in his sleep, and dreams more than a man in health; but his nights are not restless, his intellect remains unclouded, his digestion is good, though his bowels are apt to be costive, and he seldom suffers from pain in the chest.

As the disease progresses, and dilatation and regurgitation ensue, other symptoms begin to manifest themselves. The face becomes purple, the lips are somewhat livid, there is almost constant oppression at the chest, the patient suffers habitually from short dry cough, and feels weak, short-breathed, and unequal to the slightest exertion; he is unable to lie down in bed at night, is restless and uncomfortable, and his sleep is disturbed with frequent starting; pain, amounting almost to angina, occurs from time to time in the region of the heart, the palpitation is distressing, and, together with throbbing in the vessels of the head and neck, is induced or augmented by mental excitement, active exercise, and the act of stooping. As the systemic congestion increases, and the obstruction of the blood current is felt further and further backwards along the course of the circulation, the pulse becomes irregular both in force and rhythm, and dyspepsia and loss of appetite are gradually induced. Ultimately dropsical effusion may take place into the pericardium, the cavities of the pleura, and into the cellular tissue of the body. These latter symptoms, however, are attributable to regurgitation consequent on dilatation, and not primarily to mere hypertrophy; for simple hypertrophy does not of itself give rise to systemic congestion; indeed it may exist for a long series of years without inducing dyspepsia or portal congestion, or dropsical effusion, even to the extent of œdema of the feet and ankles.

It has been stated that sanguineous apoplexy is an occasional result of hypertrophy of the left ventricle, and statistics have been published by Andral and others, in proof of this position. But the result of Rochoux's investigations are completely at variance with Andral's conclusions, and my own experience in the dead-house of St. George's Hospital, inclines me to believe that there is no special connection between the two forms of disease, beyond their connection with an atheromatous condition of the vessels.

Hypertrophy of the heart is commonly met with in persons who have passed the meridian of life; and atheromatous and calcareous degeneration of the vessels, which is the essential cause of sanguineous apoplexy, and which, as already pointed out, constitutes a not unfrequent cause of

hypertrophy, is most frequent amongst the same class of persons. Therefore, although statistical observations may point to a connection between hypertrophy and apoplexy, a more extended range of observation would seem to indicate that the connection is not that of cause and effect, but rather that of a common origin, both being the offspring of disordered conditions of the circulating apparatus commonly met with in middle and advanced life.

I do not wish to imply by the above statement that the influence exerted on the cerebral circulation, by abnormal conditions of the heart, plays no part in determining the occurrence of apoplexy. Nobody, who has noted the distended state of the veins of the head and neck in cases of dilatation of the right cavities of the heart, and the violent throbbing of the carotids in cases of hypertrophy of the left ventricle, can for a moment doubt that the cerebral circulation must feel the effects of these forms of heart disease. In like manner it must be obvious that when there co-exists hypertrophy of the left ventricle and dilatation of the right, with tricuspid regurgitation—an unusually forcible driving onwards of the blood along the arteries, and an equally forcible obstruction to the return of blood through the veins —the conditions exist which must necessarily put the greatest possible strain on the cerebral vessels, and, if possible, induce rupture and hæmorrhage. But it is certain, that when the vessels of the brain are healthy, this condition of the heart is often met with without the occurrence of apoplexy, and it is equally certain that when apoplexy occurs, the heart may be hypertrophied, or small and atrophied, but that the cerebral vessels are invariably diseased. The conclusion therefore seems inevitable, that the essential cause of apoplexy is not hypertrophy of the heart, nor dilatation of its right cavities, but a diseased condition of the vessels, with which hypertrophy and dilatation are often found associated, and that the strain put on the vessels by the existence of these forms of disease is only instrumental in a secondary degree in producing the cerebral symptoms.

There is yet another form of affection which has been regarded by some authorities as the result of hypertrophy. I allude to enlargement of the thyroid gland and dilatation of the inferior thyroid arteries, an affection which is most common in, though not necessarily confined to women. The action of the heart in these cases is rapid and violent, the eyeballs are prominent, and the enlarged gland pulsates, and presents the usual physical signs of aneurismal varix, which may subside as the gland becomes more solid. Dr. Parry, of Bath, and Drs. Graves

and Stokes, of Dublin, have directed their attention specially to this malady, and the latter especially has declared it as his opinion that it originates in functional disturbance of the heart, followed by dilatation and hypertrophy of that organ, enlargement of the inferior thyroid arteries, and dilatation of the jugular veins.*

The rarity of its occurrence in connection with hypertrophy is sufficient proof that it cannot be viewed as a primary result of that affection, and the evidence in support of its connection with functional palpitation is even less satisfactory. My own impression is that it is a disease *sui generis*, unconnected with hypertrophy, and equally so with functional palpitation, except in so far as palpitation or hypertrophy may be caused by the disturbance in which the enlargement of the thyroid originates. The principal point of interest connected with its occurrence is the violent pulsation in the thyroid, which is liable to be misinterpreted by careless practitioners, and regarded as aneurism.†

The prognosis of simple hypertrophy is not unfavourable, provided the patient leads a quiet life, and is moderate in his diet and exercise. I have no means of ascertaining its average duration under these circumstances, but instances of it are under my inspection at the present time, and have been so above twelve years, in which the patients are still apparently in good health, and the disease has made little progress. But it is otherwise when the patient leads an irregular life, or a life of constantly recurring excitement, or is forced to gain his daily bread by the sweat of his brow. In each of these cases, the heart, after a time, almost necessarily becomes dilated, regurgitation ensues and then pulmonary congestion, and other secondary affections arise, which too surely lead to grievous suffering, and terminate only in a premature death.

A question has been started as to the possibility of recovery from simple hypertrophy, and authorities of equal weight and number have ranged themselves on either side of the controversy. My own conviction is, that the complete removal of hypertrophy is well nigh impossible; but I am fully convinced, that by careful attention to hygienic rules for a lengthened period, its effects may be moderated, and kept within such bounds that the patient shall be scarcely conscious of its existence, and shall live on for a long series of years in the enjoyment of tolerable health.

* Stokes, loc. cit., pp. 296, 7.

† A case in point is referred to by Dr. Stokes in which a day had actually been appointed for the operation of tying the carotid artery.

The treatment of hypertrophy requires the exercise of sound judgment, common sense, and extreme patience. If a cure is within the range of possibility, it can only be effected very gradually, by judicious dieting, by careful avoidance of all excitement, by the regulation of the bodily exercise and general mode of life, and by the administration of such medicines as are calculated to moderate the heart's action without exerting a depressing influence on the general health. The occasional abstraction of a few ounces of blood may be necessary, if the heart's action has been over excited, whether by mental emotion, or injudicious conduct on the part of the patient; but even in this case, the greatest caution should be exercised in withdrawing the vital fluid, lest the quality of the blood be impoverished thereby, the patient weakened, and the irritability of the heart increased. The application of a few leeches to the præcordial region, or the abstraction of a few ounces of blood by cupping, will have a tranquillising effect as great as that of general bloodletting; and the loss of even a small quantity of blood will be unnecessary in cases in which the physician pursues a judicious mode of treatment, enjoins a proper course of life, and has his injunctions honestly carried out by his patient.

The administration of medicines which act merely as sedatives on the heart can have little influence in controlling the progress of hypertrophy; nay, more, if carried beyond a certain point, such remedies may actually increase the tendency to the disease, by rendering the heart less able to cope with the obstruction it has to encounter, and thus leading at once to dilatation of its cavities, and to still further impediment in the capillary circulation. The objects which have to be attained by medicine are, 1st, to keep up free excretion from the bowels and kidneys, so as to drain off superfluous fluid without impoverishing the quality of the blood; 2nd, to prevent congestion of the capillary system; and, 3rd, to control undue excitement of the heart, so that its work may be effected at the least expense of labour. Hydragogue purgatives, diuretics, and cardiac sedatives are, therefore, the class of remedies to be employed, and their efficiency depends upon the care and judgment with which each is made use of. The pulvis jalapæ compositus, and the compound gamboge pill of the pharmacopœia, podophyllin, and saline and aloetic purgatives are especially useful; and amongst diuretics may be mentioned the acetate and nitrate of potash, squills, foxglove, and broom. Digitalis, however, must be employed very cautiously, as it augments the contractility of the heart to such a

degree that, if given in full doses in cases of hypertrophy, it is apt to induce tonic spasm and sudden death.* Of sedatives, belladonna applied externally, and administered internally, is that on which most reliance can be placed, and to this may be added aconite, the veratrum viride, and hydrocyanic acid, each of which, under certain conditions of the circulation, may prove in turn of essential service.

Moderate exercise in the open air is essential to the patient's health, and, therefore, to the well-being of his heart; but it must be carefully regulated, in order that it may never be so active as to accelerate the circulation, or induce shortness of breath or palpitation. Food, whether fish or flesh or fowl, should be eaten sparingly and slowly, and the meals should be taken regularly. Soups and all excess of fluids should be avoided, as calculated to produce flatulent distension of the stomach and repletion of the vascular system, and wine and other stimulants should be taken, if at all, in very small quantity.

CHAPTER V.

DILATATION OF THE HEART.

In the section devoted to hypertrophy of the heart, allusion was made to the frequent existence of dilatation of its cavities, and it was stated that a mixed condition of hypertrophy and dilatation is the form of disease which most commonly occurs as a result of any impediment to the flow of blood, whether in the heart itself, in the large arteries, in the systemic capillaries, or in one or more of the principal organs of the body. But dilatation of the cavities of the heart sometimes takes place without any concurrent hypertrophy of its walls; nay, more, its cavities occasionally become enlarged, even whilst its walls are undergoing attenuation.

Thus, then, three forms of dilatation of the heart are met with in practice: 1st, dilatation of the cavities and hypertrophy of the walls—a mixed condition of hypertrophy and dilatation termed active dilatation; 2nd, dilatation of the cavities, with normal thickness of the walls—simple dilatation; 3rd, dilatation of the cavities, with thinning of the walls—attenuated dilatation.

It is difficult to draw a distinct line of demarcation between these

* For facts in proof of this, see pp. 592-3, of this treatise.

different forms of disease. The one passes gradually into the other by imperceptible gradations, which it is impossible to define, either anatomically or clinically, and all that can be fairly stated respecting them is that, in proportion as the dilatation exceeds the hypertrophy, so does the complaint lose the character of hypertrophy, and assume the distinctive features of dilatation. The characteristics of hypertrophy, and of hypertrophy with dilatation, have been already discussed. Those which more peculiarly appertain to simple and attenuated dilatation it will presently be my endeavour to define.

But first let us investigate the causes of dilatation. These may be divided into two distinct classes; the first comprising all causes which affect the nutrition of the heart—causes which, in some instances, induce wasting of the muscular tissue of the organ, and in others impair its tonicity and contractility; the second, embracing all causes which excite the irritability of the heart, or make an extra demand on its action—causes which, if the nutrition of the organ were not disturbed, would lead to hypertrophy of its walls, but which, under existing circumstances, inevitably occasion yielding of its walls, and produce dilatation or enlargement of its cavities. Amongst the former may be mentioned chronic congestion of the heart, which first induces induration of the heart, and then gives rise to defective contractility and yielding of its walls;* fatty degeneration of the muscular tissue; and malnutrition of the heart, whether induced by a diseased condition of the coronary arteries, or by general systemic disturbance and a depressed condition of the blood; amongst the latter, strong mental exertion and nervous excitement, violent exercise, disordered conditions of the blood, various organic diseases of the internal organs, and of the arteries, and disease of the valvular apparatus of the heart.

The causes of dilatation being sometimes general and sometimes local, the seat of dilatation varies accordingly. All the chambers of the heart may be affected when the nutrition of the entire organ is impaired, or when its muscular tissue has undergone degeneration; whereas, one cavity only may enlarge when the malnutrition or degeneration is of a local character,† or when one portion only of the heart has been subjected

* For full details respecting the operation of this cause, consult an admirable paper by Dr. Jenner, in 'Med.-Chir. Trans.,' vol. xliii.

† Disease principally confined to one of the coronary arteries is a frequent cause of local malnutrition; for "although the coronary arteries communicate at the base and apex of the heart, the communication is not very free, and each can do little more than supply its respective regions." See a paper by Mr. Swan in 'Lond. Med. Gazette,' November, 1848.

to extra strain or pressure. But as the causes of dilatation are more commonly general than local, so dilatation usually affects both sides of the heart—the right in a more marked degree than the left; and when it is confined to one side of the heart, the right is that which almost always suffers. The reverse of this holds good when dilatation is combined with hypertrophy; for this combination very generally results from valvular disease or arterial obstruction, both of which are infinitely more frequent on the left than on the right side of the heart.

As the cavities of the heart dilate, their orifices commonly enlarge, and so also do the valves and the tendinous chords. This, in most cases, obviates the danger which would arise from insufficiency of the valves, if the stretching and enlargement of the valvular flaps did not keep pace with the dilatation of the orifices. But in some instances in which the auricle and ventricle on the same side become dilated, the enlargement of the valves is not commensurate with the dilatation of the orifices, and then the valves, though not diseased, become incompetent to close the widened orifice, and regurgitation takes place. This may occur on either side of the heart, but it is more frequent on the right side than on the left.

The tissue of a dilated heart is usually said to be soft and flabby. This is true in most instances, but by no means invariably, the exceptional cases being those in which the walls of the heart have yielded, as a result of impaired contractility of their fibre consequent on previous congestion and induration, under which circumstances fibroid degeneration of the muscular structure is apt to take place.

In these cases, the first effect is thickening and induration of the walls of the heart. Dilatation subsequently occurs, as a result of the diminished elasticity and contractility of the fibre, and after a time the induration is replaced by softening, as an effect of fatty degeneration. If the patient dies during the earlier stage of the disease, the muscular tissue of the heart, when cut, presents a smooth and abnormally compact and homogeneous appearance, and is harder and stronger than natural, and when the ventricles are cut across, their walls do not collapse, but retain the form of the cavity. The muscular fibres are more firmly united together than in health, and under the microscope, their transverse striæ are indistinct, and innumerable granular molecules are to be seen external to and within the sarcolemma. In the later stages of this form of disease, fatty degeneration and softening usually occur, and then the muscular tissue becomes soft and

friable, and the walls of the ventricles collapse the moment they are cut across; the muscular fibre loses its striated character, and assumes an irregularly granular appearance; and innumerable fat globules are seen replacing the granular molecules both within and outside the sarcolemma. In many of these cases the yielding of the walls is excessive, and in some even rupture of the heart may occur, whilst in others the disease may be more advanced at one spot than at another, and bulging or pouching of the walls may take place, constituting what has been termed aneurism of the heart.

A heart which is dilated loses its conical form, and becomes more or less imperfectly square or rounded, the increase of the transverse diameter being much greater than that of its vertical diameter.*

The physical signs of dilatation of the heart are strictly in keeping with the conditions of the heart already described. In the earlier stage of the disease, inspection discovers indistinctness, if not entire absence, of visible cardiac pulsation; palpation confirms the result of inspection, by showing that the impulse is entirely absent; and percussion indicates extension of the præcordial dulness. Nevertheless, there is no fulness or prominence of the cardiac region, and auscultation proves that the action of the heart is regular or slightly unsteady, and that the deficiency of impulse, and the extension of præcordial dulness on percussion, are not attributable to effusion into the pericardium, but rather to the condition of the muscular walls of the heart; for the sounds, instead of being dull, muffled, and distant, as they would be in the event of pericardial effusion, are actually clearer than in health, though altered in

* The following admeasurements of a healthy heart by Bizot, given by Dr. Stokes (loc. cit., p. 257), as referable to subjects between the ages of thirty and forty-nine, will serve as a standard for comparison, viz.:

	In men.	In women.
	English inches.	English inches.
Length	3·82	3·64
Breadth	4·24	3·91
Depth	1·52	1·25
Length of left ventricle	2·61	2·83
Breadth „	4·72	4·10
Length of right ventricle	3·33	2·97
Breadth „	7·40	6·80
Width of the origin of aorta (above the valves)	2·74	2·49
„ pulmonary artery	2·79	2·60
„ mitral orifice	4·29	3·61
„ tricuspid orifice	4·81	4·18

rhythm and character. Thus the systolic sound is clearer, shorter, and more abrupt than natural, and gives the impression of being more superficial in its seat, whilst the second retains its usual features, so that, in some instances, the two sounds appear to be of equal duration.

As the disease progresses, the signs are found to vary according as the heart's tissue is firm, or, on the other hand, soft and flabby. In the former case the cardiac action may remain almost regular until near the close of life, and the cardiac impulse may continue visible; but it is very feeble or even tremulous, is not confined to the apex as in health, but extends over a larger surface, and often manifests a *quasi* undulatory character. It does not convey the impression of heaving, but rather of gentle tapping, and whatever its character may be, the force of successive beats is almost always unequal. The first sound, though shorter than natural, is both louder, clearer, and more prolonged than when the muscular tissue is soft, and has undergone fatty degeneration, and it is markedly clearer and apparently nearer to the surface than in health.

When the fibre of the heart is soft and flabby the cardiac impulse is often imperceptible to the eye and hand; the action of the heart is usually very irregular and often intermittent; reduplication of the sounds are occasionally observed; and the first sound, instead of being louder and clearer, may be actually weaker than in health, whilst the second may be inaudible at the apex. In all cases, however, the first sound is unnaturally short and abrupt, and, *cæteris paribus*, both sounds are transmitted over a less extended surface of the chest than are the sounds of a healthy heart, the precise extent of their transmission being dependent on the existence or non-existence of any hypertrophic thickening of the walls.

In cases of attenuated dilatation the extent and intensity of the præcordial dulness generally decrease in consequence of the overlapping of the lung, the margin of which is often emphysematous, but in simple and in active dilatation the area of percussion dulness gradually increases, the direction in which the increase principally takes place being determined by the seat of dilatation—to the right if the right ventricle is the chief seat of dilatation, to the left if the left ventricle is diseased. The same holds good in respect to dilatation of the auricles. It may be added, that when the right auricle is much dilated, the large veins of the neck are frequently turgid and knotty.

Dilatation of the heart is not necessarily productive of endocardial murmur, nor is it usually accompanied by murmur, unless valvular disease accidentally coexists. But in some instances, as already stated, it gives rise to widening of the orifices of the heart to such an extent that the valves, even when stretched and enlarged to the utmost, prove inadequate to close them thoroughly. Regurgitation, therefore, is one of the results of dilatation when carried to an extreme point, and it occurs most commonly through the tricuspid orifice, less frequently through the mitral, and still less frequently through the aortic and pulmonary orifices. In these cases murmur does not necessarily arise, inasmuch as when the walls of the heart are thus diseased the contraction of the ventricle and the systole of the aorta or pulmonary artery may not be sufficiently forcible to cause a sonorous eddying of the blood. Nevertheless, when regurgitation does occur, it is usually indicated by the general symptoms; and when it takes place through the tricuspid orifice, its occurrence will be marked by turgescence and pulsation of the jugular veins. In some instances, however, in which the ventricle is very weak, the backward stream of blood is not sufficiently forcible to occasion venous pulsation, and the veins are merely turgid and knotty as a result of the impeded circulation.

The symptoms of cardiac dilatation vary with the extent and period of the disorder. In the earlier stage the circulation is weak, the patient feels languid and deficient in energy, is easily fatigued, and finds himself short-breathed and exhausted on active exertion; he suffers from giddiness and frequent palpitation; yawns or sighs without apparent reason; is subject to faintness and giddiness, and is often irritable, low spirited, and desponding. Sometimes he experiences uneasiness—not pain—in the cardiac region; at others there is tenderness or soreness on pressure over the heart, and in others, again, paroxysms of pain occur, amounting, in some instances, to severe angina. The face is either pale and sallow, or mottled and of a purple hue from dilatation and congestion of the capillaries; the eye-balls are often injected, and the eyes watery; the extremities are cold, œdematous towards evening, and, not unfrequently, of a blue or purple colour, and here and there they present spots of livid discolouration, which inflame and slough and cause deep-seated, obstinate, ulcers. The pulse is small, weak, and seldom regular; very commonly it is extremely feeble and irregular; the tongue is pale, flabby, and œdematous; the bowels are usually costive, or alternately costive and relaxed; piles are not of unfrequent occur-

rence, the digestive organs are easily deranged, and flatus is a constant source of annoyance.

In the more advanced stages of the disease the symptoms already named are seriously aggravated. The patient is unequal to the slightest exertion, any attempt at which induces breathlessness and distressing palpitation; his face is of a mottled purple colour; the lips are of a leaden hue; the breathing is oppressed, attacks of dyspnœa, and palpitation frequently occur almost without apparent cause. Tranquil sleep is almost unknown to him, for not only is he unable to lie down in bed, but whenever he doses he is disturbed by startings and frightful dreams. The œdema and swelling, which may have long manifested themselves in the feet and ankles towards evening, become constant, and spread upwards until the thighs, the external genital organs and the abdominal walls become anasarcous, and pit under pressure. At the same time the kidneys become congested, and the urine is scanty, high-coloured, and often albuminous; the circulation through the lungs and liver is also impeded, and symptoms of pulmonary oppression and hepatic congestion begin to manifest themselves. The patient becomes slightly jaundiced, and suffers from biliary derangement, if not from effusion into the cavity of the abdomen; piles frequently occur, and hæmorrhage takes place from various parts of the alimentary canal; a constant, harassing, and oftentimes spasmodic cough occurs, which at first is dry, and subsequently is attended by a frothy, serous, and, possibly, blood-streaked mucous expectoration; the breathing becomes gradually more oppressed, in consequence of the occurrence of pulmonary œdema, or congestive pneumonia, and effusion, which takes place into the cavities of the pleura, augments the dyspnœa and orthopnœa. When severe paroxysms of restlessness and dyspnœa occur the patient presents a pitiable spectacle. Dropsical and helpless, yet struggling for breath, he finds it impossible to rest in bed, and sits for days together in an arm-chair or on the side of his bed, propped up by pillows, with his swollen legs in a depending position, and his brain so oppressed by the imperfectly aërated blood which gorges it, that in the intervals between the severer paroxysms he is drowsy and only semi-conscious. In most instances his death is very lingering, but occasionally his sufferings are terminated suddenly by a paroxysm of angina or by syncope, resulting from failure of the heart's action.

The diagnosis of dilatation is a more difficult problem than is commonly supposed, and is one which even the most practised physician may fail to solve by reference to the physical signs alone. The difficulty

is due to several causes, amongst which may be enumerated, 1st, the frequent existence of emphysema of the lungs, which serves to mask the percussion dulness; 2ndly, the occurrence of pulmonary consolidation, which leads to extension of the dulness, and so complicates the case; 3rdly, the comparatively forcible impulse which is sometimes produced, even by a simply dilated heart, under strong nervous excitement; and 4thly, the feebleness of the impulse of an hypertrophied heart when its cavities are gorged with blood, and the nervous power is exhausted. The difficulty, however, is principally felt on seeing a patient for the first time; for careful attention to the physical signs and general symptoms for a few successive days will generally suffice to unravel the mystery. The distinctive features of the two forms of disease are here subjoined in a tabular form, in order that the principal points of contrast may be distinctly seen. The symptoms of dilatation with hypertrophy necessarily occupy an intermediate place.

Dilatation.	*Hypertrophy.*
Cardiac pulsation entirely absent, or extremely weak, and of a *quasi* undulatory character; apex beat scarcely discernible, not constant in its position, but not materially lowered, and often somewhat to the right of its usual position; impulse, as felt by the hand, feeble and tremulous, often imperceptible; area of percussion dulness greatly increased laterally, not much vertically. Sounds sometimes faint, but often loud, and always clear and abrupt, the first sound being remarkably shortened; the post systolic silence prolonged. Pulse irregular and feeble, each successive pulse being of unequal force; complexion pale or sallow; veins of neck often distended; extremities cold.	Cardiac pulsation forcible; apex beat much lower, and usually further to the left than natural, and constantly perceptible at precisely the same spot. Impulse strong, and of a heaving character. Area of percussion dulness increased vertically more than laterally; sounds dull, muffled, and prolonged, the first more especially so; the post systolic silence shortened, or almost absent; radial pulse strong, full, and regular; visible pulsation of carotids; no distension of the veins of the neck; complexion florid; extremities warm.

Dilatation of the heart is a more serious disease than hypertrophy, and runs a more rapid course. When coexisting with hypertrophy, it augments the danger of the latter form of disease in proportion to the

degree in which dilatation of the chambers of the heart exceeds the hypertrophic thickening of their walls. In like manner, the coexistence of hypertrophy with dilatation lessens the immediate danger of the latter form of disease, by rendering the heart more able to fulfil its functions. In its active or complicated form, or even in its simple or attenuated form, dilatation may continue for many years without producing fatal consequences, provided due attention be paid to the medical treatment of the patient, and to the careful regulation of his mode of life; but if his habits are not placed under proper control, the character of the blood improved, and the tone of the system upheld, the patient may have a brief and miserable existence. Death sometimes occurs suddenly in a paroxysm of angina, or from syncope, the result of failure of the heart's action; but more commonly it takes place slowly, with dropsical effusion and congestion of the pulmonary and systemic capillaries. When once dropsy has supervened, relief will be only temporary, and life can seldom be prolonged beyond one or two years.

The only satisfactory treatment of dilatation in its earlier stages consists of measures calculated to regulate the chylopoietic viscera, to improve the condition of the blood, and of the system generally, and so indirectly to impart tone to the heart without exciting its irritability. In the later stages, when dropsy has supervened, our aim must be to relieve the capillary circulation, and to obviate mischief which would render all other treatment useless.

In the first instance, quinine and other vegetable bitters may be needed, in combination with taraxacum and the mineral acids, or small doses of the alkalies, to promote digestion and ensure a proper action of the liver ; and if further assistance in this direction is desirable, occasional doses of blue pill, podophyllin, and ox-gall may be given at night, and followed by a warm aloetic draught in the morning. But of all remedies, iron, digitalis, and belladonna are those which exert the most remarkable effect on a dilated heart, and contribute most surely to a successful issue. Aconite, which is so useful in cases of hypertrophy, and has been often recommended in dilatation, is dangerously depressing in its action, and if given at all, must be closely watched. The administration of iron is so manifestly indicated by the aspect and general symptoms of the patient, and is so universally acknowledged as an essential element of treatment, that it is unnecessary to do more than urge the expediency of persevering in its exhibition for a lengthened period—not for a few weeks or even months, but, with

trifling intervals, for several years. In many instanses, I have known the physical signs of dilatation improve materially, and the general symptoms almost wholly disappear after a course of iron has been steadily persisted in for the space of three or four years. Belladonna, again, administered internally, and applied externally, is so generally recognised as a remedy on which reliance can be placed for quieting the irritability of the heart and checking palpitation, that lengthened laudation of its virtues would be superfluous. But it is otherwise in respect to the use of digitalis. Most authorities concur in speaking of this drug as valuable in the treatment of hypertrophy of the heart, but most dangerous, and therefore only to be employed with extreme caution, in the management of dilatation, their views being founded on the commonly received but erroneous impression that digitalis exercises a depressing influence over the action of the heart, and therefore leads to accumulation or coagulation of blood in its cavities, if not to actual paralysis of its muscular structure.* But these views are based on an imperfect knowledge of its action, and are utterly inconsistent with fact. Experiment and observation may be alike appealed to in proof that digitalis stimulates the muscular fibre of the heart, and augments the contractility of the capillaries. When it kills, it does so not by producing paralysis of the heart, but by giving rise to tonic contraction and spasm of that organ. Such being the case, it is a most valuable remedy in the treatment of dilatation, and is dangerous only when administered in hypertrophy. Whenever the pulse is feeble and irregular, and more especially when, from any cause, its feebleness and irritability are temporarily increased, digitalis is of all known remedies the most useful. By stimulating the muscular tissue of the heart, it allays the irritability of that organ and moderates its action, whilst at the same time it augments the tone and contractility of the vessels, increases the flow of urine, and exerts a restorative and calming influence over the system, which is not attainable by any other means.†

* For instance, Dr. Walshe, when speaking of the treatment of dilatation, says:—"The exhibition of digitalis requires the utmost caution: slackening the circulation, as it does, it promotes either coagulation within the heart, or, in a less degree of its action, accumulation of blood in the cavities, whereby they may be still further passively dilated. If the power of the ventricles be seriously impaired, digitalis cannot be given without excessive risk, and had much better be altogether avoided." (Loc. cit., p. 664.)

† The grounds for the opinion above given respecting the action of digitalis may be briefly stated:—1st. During many years I have observed that the cases of heart disease most benefited by digitalis have been those in which the heart has been weak and

In the later stages of the disorder, when cough, dyspnœa, and dropsy have resulted from the stagnation of the pulmonary and systemic capillary circulation, recourse must be had to the remedies which experience has shown to be most serviceable in removing these several symptoms. These have been discussed in the sections devoted to valvular disease of the heart, and it is needless to recapitulate them here. Suffice it to say, that dry cupping between the shoulders and across the loins, and the use of mustard poultices and turpentine stupes, should never be neglected when the lungs are congested; that squill, senega, nitric æther, and ammonia, usually prove the most serviceable expectorants; that hyoscyamus, belladonna, hydrocyanic acid, and æther, lobelia, and the liquor opii sedativus, are the remedies which can be most relied on for controlling the frightful paroxysms of dyspnœa and pain in the cardiac region which are sometimes attendant on this form of disease, and that hydragogue purgatives and diuretics are necessary to drain off superfluous fluid, relieve the congested capillaries, and get rid of the dropsical swelling.

dilated, and the pulse feeble and irregular. In these the pulse has become stronger and steadier, and less frequent under its action. 2ndly. In the only cases in which I have known death to occur suddenly during the administration of digitalis, the heart has been hypertrophied and firmly contracted. This may have been a coincidence, but, viewed in connection with the results of experiments to which I shall presently refer, it is, at least, a suspicious fact. 3rdly. Dr. Dickenson has pointed out ('Med.-Chir. Trans.,' vol. xxxix), and I have repeatedly verified his observation, that digitalis, if given in full doses, induces violent uterine contraction, and checks uterine hæmorrhage; and, inasmuch as its action in staying menorrhagia and uterine hæmorrhage is permanent, it seems fair to conclude that it gives tone to the capillaries and increases their contractility. 4thly. This view is borne out by what I have long since observed relative to its action in arresting hæmoptysis, viz., that whilst effecting the object required, it does not weaken but rather increases the force of the pulse though it lessens its frequency. 5thly. When patients die of delirium tremens, the pulse is usually rapid and fluttering before death, and the heart is found weak, flaccid, and distended with blood afterwards. These are just the cases in which, on the commonly received doctrine as to the action of digitalis, the drug ought necessarily to prove fatal, and yet modern experience has shown that in these cases it is tolerated even in excessive doses. My impression is that its remedial action in these cases depends on its stimulating the heart, subduing its irritability, and increasing the tonicity and contractility of the heart and the capillaries, so that the brain is better supplied with blood, and the effusion of its more fluid parts, which gives rise to the "wet brains" of habitual drunkards is avoided. 6thly. It has been proved by experiments on animals (Dr. H. Jones) that when death is induced by digitalis the heart is not flaccid and distended with blood, as is commonly supposed, but, on the contrary, empty, contracted to the utmost, and in a state of tonic spasm. All these facts confirm my view as to the action of digitalis, and if it is correct its practical importance in relation to the treatment of cardiac dilatation can hardly be over estimated.

From the earliest period of the disease the patient should be encased in flannel, and be otherwise kept warm, and should lead a tranquil life, avoiding fatigue and all active exertion. Cheerful society, frequent change of scene, and regular but moderate exercise in the open air, are important elements in the treatment. The diet should be light, nourishing, and easy of digestion, and soups and liquids should be avoided as much as possible, as calculated to excite flatulence and distension of the stomach, and so to interfere mechanically with the action of the heart. Diffusable stimulants are always needed. A glass or two of wine, or brandy and water may be taken at luncheon and dinner, so long as the legs are not œdematous; but, as soon as anasarca supervenes, whisky, hollands, or gin, may be advantageously substituted, as stimulating increased action of the kidneys.

CHAPTER VI.

ATROPHY OF THE HEART.

Atrophy of the heart, as its name implies, is the reverse of hypertrophy, and results in the diminution of the muscular tissue of the heart, and of the nerves and vessels which supply it. Every part of the organ decreases in size, its walls become thinner, its cavities contract, its valves become smaller and more attenuated, and the entire bulk of the organ may be so reduced that the atrophied heart of an adult may weigh only four and a half or five ounces, instead of nine or ten.

The structure of an atrophied heart is not necessarily diseased; indeed, in simple atrophy the fibre is not diseased; the transverse striæ of the muscular tissue are clearly marked, and the only perceptible difference between an atrophied and a healthy fibre is, that the former is paler than natural, and of lessened tonicity. Not unfrequently, however, the muscular fibre of the heart, when atrophous, becomes markedly fatty, and the transverse striæ disappear. Under these circumstances, the muscular tissue is soft and inelastic, and the case comes under the category of a softened or fatty heart, which will be described in a future chapter.

Atrophy of the heart, when simple and uncomplicated, may be the

result of a deficiency in the supply of blood to its tissue, consequent on the tight embrace of an adherent pericardium, or on narrowing or obstruction of the coronary arteries, or else of the comparative inactivity of the organ consequent on the lessened amount of work it has to perform. Just as the voluntary muscles, when little used, dwindle and become weak, so the muscular tissue of the heart under the same conditions wastes, and the bulk of the organ shrinks. This is mostly observed in connection with cancer and other wasting disorders which do not create any serious impediment to the circulation. By leading to a diminution in the bulk of the body, and in the quantity of blood in circulation; by impoverishing the quality of that blood, and by necessitating repose and freedom from exertion, they not only render the nutrition of the heart imperfect, but necessarily produce a decrease in the energy of its action, and a corresponding diminution in its bulk.

This condition of the heart is not characterised by any pathognomonic physical signs or general symptoms. The pulse of course is small, the impulse weak and of small extent—often imperceptible—and the area of percussion dulness lessened. The sounds of the heart are clear, and, unless the muscular tissue be fatty, are not remarkably altered in rhythm. Palpitation is seldom observed, and when it occurs, it is of temporary duration and attributable to accidental causes, rather than to the structural condition of the heart.

Atrophy of the heart can hardly be regarded as an idiopathic disease, but rather as a consequence of another disorder. The treatment is necessarily that of the general malnutrition out of which the atrophy has arisen. When it has occurred in connection with the tight embrace of an adherent pericardium, with obstruction of the coronary arteries, or with cancer or other malignant disease, its cause is persistent, and its treatment hopeless; but when it has taken its origin in a cause of protracted yet temporary waste and bodily inaction, its cause is removable, and whatever will tend to rectify the constitutional derangement, and restore tone and vigour to the body, will tend also to stimulate the nutrition of the heart, and cause it to be effected more perfectly. In one case which I had under observation for the space of three years after the patient's comparative restoration to health, the alteration produced in an atrophied heart was most remarkable.

CHAPTER VII.

INDURATION AND CONGESTION OF THE HEART.

In the section devoted to dilatation of the heart it was stated that induration of the cardiac tissue occasionally precedes and is the cause of dilatation of the cavities of the heart, and reference was made to a paper by Dr. Jenner, in which the subject is discussed. The principal interest attaching to induration is that of its bearing on the production of dilatation, but as it is an affection which is often overlooked, and the pathology of which is not commonly understood, it may be desirable to inquire into it a little more fully.

Induration of the heart results from long-continued congestion of its muscular tissue. Hence it may arise from any cause which occasions an impediment to the egress of blood from the right ventricle, and thus, by producing distension of the right auricle, and venous congestion, impedes the return of blood from the coronary sinus. "If the impediment to the exit of the blood from the sinus be slowly produced, be moderate in degree, and permanent, or be frequently repeated, then induration, toughening and thickening of the walls will ensue, and permanent dilatation of its cavities will be the final result; the over-distension of the walls of the cavities being the immediate cause of the dilatation—the induration, toughening, and thickening being the cause of the permanence of the dilatation."*

On dissection, the appearance and condition of a heart which has once undergone induration will vary with the date of such induration, and with the changes which have subsequently ensued. If examined whilst induration still exists, the muscular tissue will be found harder and tougher than natural, and smoother and more homogeneous in appearance, and when the ventricles are cut across, the walls will not collapse, but the cavities will retain their rounded appearance, and the columnæ carneæ will stand out in bold relief. And in proof that this is an abnormal condition, the microscope will show that the transverse striæ are very indistinct, and that the indurated fibre has assumed an

* 'Med.-Chir. Trans.,' vol. xliii.

irregularly granular appearance, innumerable protein granules existing both within and external to the sarcolemma.

But after a time fatty degeneration is prone to occur; the granular matter is replaced by oil globules, and the muscular fibres of the heart, damaged by the interference with their nutrition which long-continued congestion induces, are apt to undergo the same change; and thus it happens that if the heart be not examined until some time after the first accession of induration, the heart may often be found soft instead of indurated, and innumerable fat globules will be visible under the microscope both between and within the sarcolemma.

The causes which give rise to an accumulation of blood in the right cavities of the heart have been discussed in the section of dilatation, and it is needless to recapitulate them here. But it is important to remark that persistent congestion of the heart occurring coincidently with increased cardiac action appears to be the condition which is most influential in the production of induration.

I am not aware that the physical signs of induration of the heart have been described apart from the signs of dilatation; and inasmuch as fatty degeneration of the heart generally takes place before the malady terminates fatally, opportunities do not often occur of verifying by dissection the observations made at the bedside during life. But in three instances in which I have been enabled to do so, the peculiarity which has attracted attention has been the extraordinary clearness and shortness of the first sound when viewed in connection with the force of the impulse. The sounds have been those of a dilated heart: the impulse that of an hypertrophied heart rather than of one which is simply dilated.

The general symptoms are essentially those of dilatation of the heart, and treatment is that which is appropriate in dilatation.

CHAPTER VIII.

SOFTENING OF THE HEART—FATTY DEGENERATION.

Softening* of the heart is a disease which, even up to the present time, has not assumed a distinct place in our nosology. Originating in defective or perverted nutrition, it is sometimes connected with fatty degeneration of the muscular tissue, sometimes with an interstitial deposit of fat, and sometimes, again, with malnutrition of the sarcous elements, unattended by the deposit of fat. Many authors have treated these several varieties of cardiac lesion as different diseases, each requiring a separate description, and thus have seriously complicated a subject which it should be our aim and object to simplify. It is undoubtedly true that each form of malnutrition is characterised at the outset by its own peculiar pathological changes; but inasmuch as all the varieties are productive of very similar symptoms, and usually terminate in greater or less degrees of fatty degeneration of the tissue of the heart,† there is no sufficient ground, pathologically or clinically, for regarding them as separate diseases. They are all results of malnutrition of the heart's tissue, and clinically are almost identical in significance.

Our ideas as to the causation of softening of the heart are by no means clear or satisfactory. We know that it is of frequent occurrence, arises under circumstances of mental disquietude and bodily ill health, and is productive of symptoms analogous to those already described as connected with simple dilatation of the heart. But in most cases we are utterly ignorant of the primary cause of the malnutrition which softening indicates. And for the present little more can be done than

* The term "softening of the heart," as employed in this section, is not intended to include the acute softening of the heart's tissue, which in some rare instances results from active inflammation and suppuration, nor that which is observed in typhus fever and other febrile blood disorders, in which all the tissues are similarly affected.

† Many observers have long since described the characteristic features of softening and fatty degeneration of the heart, but we are principally indebted to a paper by Dr. Quain, in vol. xxxiii of the 'Med.-Chir. Trans.,' for directing attention to the pathological condition of the muscular tissue of the heart from which these symptoms originate; see also Dr. Ormorod's 'Observations on Fatty Degeneration of the Heart,' and Mr. Paget's 'Lectures.'

to chronicle the history of the disease, to note its symptoms, and point out the treatment which experience has hitherto proved most productive of benefit.

Softening of the heart may occur at all ages, and in persons of both sexes, and in all ranks of society. It is seldom met with in children, and may be regarded as essentially a disease of middle and advanced life. The well-fed and idle are not less subject to it than the ill-fed or the hard-working artisans. It is not necessarily connected with general obesity, nor, on the contrary, with general leanness of the body, but is met with in persons apparently in fair condition, as well as in the stout and the thin. In health, a certain amount of fat is always found on the surface of the heart, and the deposit of an increased quantity of fat in this situation, and its interstitial deposit amidst the fibres, may be regarded simply as fatty hypertrophy, analogous to that observed in other structures of the body. If excessive in degree, it may impede the action of the heart mechanically, and thus give rise to breathlessness and oppression of the breathing, just as is observed in the fatted animals at a Smithfield show; but otherwise it is consistent with the maintenance of good health, and is not for a considerable length of time accompanied by changes in the muscular fibre of the heart. By degrees, however, the pressure of the fat leads to malnutrition of the muscular fibres, and thus they become atrophous, lax, and flaccid, and often undergo fatty degeneration. Under these circumstances the heart is softened.

In other cases, softening of the heart appears to be connected with atheromatous or calcareous degeneration of the coronary arteries, just as in the brain this same condition of the vessels is found to be associated with softening of the cerebral structures. In either case, the disease in the vessels leads to a defective supply of arterial blood, and so to malnutrition of the tissues.* In other instances, again, pericardial adhesions appear to produce the same result and operate in a similar manner in its production, viz., by constricting the vessels which supply the structure of the heart with blood, and so impairing its nutrition.

But in the majority of cases no local cause of malnutrition can be discovered, nor can any trustworthy evidence be obtained of any peculiar condition of the blood; and the conclusion is inevitable, that the degen-

* It is questionable whether the atheromatous condition of the vessels here alluded to is not itself one of the results of the systemic derangement from which the softening of the heart originates.

eration of structure is referable to defective formative action, consequent on impaired vital energy. In some instances, no other organ except the heart presents any trace of degeneration; and it might, perhaps, be supposed that if any generally pervading force were at work producing impaired nutrition, its effects should be manifested elsewhere than in the heart. But local effects of very limited extent are often produced by a cause which pervades the entire system, and there is nothing more astonishing in malnutrition being confined to the heart, than in the strange limitations constantly observed in the action of the gouty, the smallpox, and other poisons. Tuberculous disease of the lungs, fatty degeneration of the liver and kidneys, atonic gout and an atheromatous condition of the vessels, inducing apoplexy, are frequently found associated with a softened heart; and so also is the tendency to a deposit of fat which is observed in asthenic and unhealthy persons. Indeed, their association is so frequent, that it must be more than accidental. It seems to point to a similarity of cause, and renders it still more probable that exhausted nervous power, or defective vital energy, and an impoverished condition of the blood, is the starting-point of the mischief, which issues in malnutrition of the heart, with softening of its muscular fibre.

Microscopically, the structure of a softened heart differs very materially from that of a healthy one. The muscular fibre is diseased, the sharpness of its outline is lost, its transverse striæ disappear, and its sarcous elements are converted into granular and oily matter. When degeneration is far advanced, oil globules are seen scattered between the fibres, as well as within the sarcolemma; and if many adjoining fibres are diseased, the muscular nature of the structure can be scarcely recognised.

To the naked eye, a softened heart presents a pale, mottled, or dirty-yellow appearance, very different from the flesh-red colour of health. It is flabby to the touch; its texture is soft and friable, so that it yields to the slightest pressure, and may be torn without difficulty; and its elasticity is so utterly destroyed, that when the ventricles are cut across, their walls collapse immediately.*

Softening, however, does not necessarily affect all parts of the heart; indeed, the reverse is commonly the case, for the disease is almost always partial. The ventricles are more prone to attack than the auricles, and the left ventricle more frequently than the right. Further, the fibres

* For full details on all these points, consult Dr. Ormerod's 'Observations on Fatty Degeneration of the Heart.'

immediately beneath the endocardium and the pericardium are usually attacked before the intervening tissue; and the muscular structure towards the apex of the heart before that which is seated towards the base. Hence it is that in an atrophied and softened heart, extraordinary thinning of the ventricular walls is generally observed towards the apex, and that when rupture of the heart takes place, as it does sometimes in connection with this condition of the tissue, the laceration most frequently occurs in that situation.

The physical signs of softening of the heart are nearly identical with those of attenuated dilatation. The area of percussion dulness is unchanged, unless the heart be hypertrophied or dilated, or large masses of fat be deposited on its surface; the impulse is weak or imperceptible, the apex beat indistinct and often invisible; the action of the heart is sometimes regular, more frequently irregular and intermittent; the first sound is short, feeble, and toneless—sometimes inaudible—the first systolic silence prolonged; the second sound clear and relatively loud and prolonged. If the left ventricle be more affected than the right, the first sound will be heard louder, and of fuller tone at the right than at the left apex, and the second sound both louder and clearer at the second left costal cartilage than at the corresponding position on the right side. The pulse is weak and unequal, both in force and rhythm; it is often slow, and when, as sometimes happens, every alternate pulsation is imperceptible at the wrist, it may not exceed 24 or 28 in a minute. If endocardial murmurs occur, they are referable to coexistent valvular disease, and not to mere atrophy or softening of the heart.

The general symptoms are quite in accordance with what theoretical considerations would lead us to anticipate. In the earlier stages of the disorder, the heart may perform its functions tolerably, the physical signs may be so little altered as not to attract attention, and the patient may not be conscious of ill health, or even of local discomfort. By degrees, however, he begins to complain of languor and disinclination to active exertion. He is pale, and easily put out of breath, irritable, peevish, and low spirited; he sometimes suffers from pain or uneasiness at the heart, with fluttering, giddiness, loss of memory, and temporary loss of sight; there is a constant tendency to sigh, without any emotional cause to explain it; the pulse is very weak, sometimes regular, more commonly irregular both in force and rhythm; the tissues of the body become soft and flabby, the digestive organs are easily deranged, excessive flatulence is a frequent source of trouble, and the bowels are usually torpid.

As the disease proceeds, the breathlessness increases, until the patient is unequal to the slightest exertion; paroxysms of dyspnœa resembling asthma supervene, and are often accompanied by serious oppression or acute pain in the cardiac region; orthopnœa is an occasional, but by no means constant symptom; distressing palpitation or fluttering of the heart occurs on the slightest excitement, and whenever the chylopoietic viscera are deranged, or the stomach is distended with flatulence; and, on some of these occasions, attacks of giddiness and coma occur, during which the patient may fall to the ground insensible.

In most instances these attacks are of a syncopal nature, and of very brief duration, their immediate cause being a deficiency in the supply of blood to the brain, consequent on the feebleness of the heart's action; but in some cases they are of an apoplectic character, accompanied by stertorous breathing, and followed by paralysis, the immediate cause of the seizure being an outpouring of blood into the brain, consequent on an atheromatous condition of the vessels, which leads to brittleness and rupture whenever anything occurs to check the venous circulation, and so induce stagnation in the cerebral capillaries. But inasmuch as in these cases the strain on the vessels is due to venous obstruction and capillary distension, and not to the unwonted force of the cardiac propulsion or arterial action, the smaller vessels usually give way; so that the quantity of blood effused is seldom large, and if paralysis ensues, it is not generally of long duration.* Unlike what happens in cases of apoplexy connected with hypertrophy of the left ventricle, and excite-

* In a case of this sort, in which I had the opportunity of inspecting the brain after death, there were six clots in different parts of the brain, each of a different date and in a different stage of discolorization. The patient, a gentleman, aged forty-six, had fallen into pecuniary difficulties, and consequently had been subject to great anxiety during the last eighteen months of his life, and during the last nine months he had experienced five slight apoplectiform syncopal seizures, followed by temporary loss of power. In three out of the five attacks the insensibility was only of a few minutes' duration and the loss of power was so slight that he went to his office on the following day. On the other two occasions he was nearly a week before he had entirely recovered power, and on the sixth and last occasion, an enormous quantity of blood was effused, and he died within twenty minutes from the commencement of the attack. The previous attacks had been so slight that they were pronounced by his medical attendant to have been attacks of "serous apoplexy," but the *post-mortem* examination revealed the existence of six clots, one corresponding to each of the apoplectic seizures. Three of the clots were on the surface of the hemispheres, very small and quite discolourised. The vessels were atheromatous, and here and there calcified, and the heart was soft and flabby, and, under the microscope, presented well-marked evidence of fatty degeneration.

ment of the arterial circulation, the patient when attacked is pale, and has a feeble pulse, and presents more or less lividity of the lips—symptoms which ought to serve as indications of the true nature of the disorder.

In extreme cases of cardiac softening, œdema of the feet and ankles occurs; the pulse may be rapid, or may fall to 26 or 30 in a minute, owing to the failure of certain systoles of the heart to communicate a pulsation to the radial artery,* and the breathing may be alternately quick and slow, or even gasping. Sometimes, as in a case related by Dr. Stokes,† it may observe a peculiar rhythm, the respirations gradually increasing in force and duration, until they attain a prolonged gasping character, and then as gradually decreasing until they are no longer perceptible and the patient apparently has ceased to breathe, and may be almost regarded as dead, when, after a lengthened interval, a faint inspiration takes place, and a fresh series of gradually increasing and then decreasing inspirations occur.

There is yet one symptom which requires notice, not so much on account of its intrinsic importance, as in consequence of the diagnostic value, which has been falsely attached to it. I refer to the existence of fatty atrophy of the cornea, constituting what has been termed "arcus senilis," to which attention has been especially directed by Mr. Canton, in connection with fatty heart. It is as its name implies, a frequent accompaniment of old age; and as softening and fatty degeneration of the heart is also a disease to which persons advanced in years, are peculiarly liable, it is not surprising that the two lesions should be sometimes found coexisting. But in the dead-house of St. George's Hospital I have repeatedly found a well-marked arcus senilis, when the heart has been perfectly healthy, and have as frequently noted hearts thoroughly softened and fatty in persons whose eyes presented no trace of an arcus senilis. The conclusion, therefore, is inevitable, that, arcus senilis is a sign of little value in the diagnosis of fatty degeneration of the heart.

Indeed, the diagnosis of a softened or fatty heart is by no means easy, more especially if it is wished to distinguish between simple and

* This was observed in an interesting case of the disease under consideration, which I saw in the year 1860, in consultation with Dr. C. J. B. Williams and Mr. Gardner. For many weeks the pulse did not exceed 28 or 30, though the heart could be heard beating 56 or 60 in a minute. A similar case occurred under my care in St. George's Hospital, in December, 1861, in the person of a woman named Cross, aged thirty-six, in Roseberry Ward.

† Loc. cit., p. 324.

fatty softening, and to draw a distinction between these varieties of disease, and simple or attenuated dilatation. In fact, in uncomplicated cases it is impossible during life to discriminate between them, and, even were it not impossible, it would be practically useless. They are all connected with feebleness of constitution, impaired nervous energy, and defective nutrition, and are characterised by fluttering and palpitation of the heart occurring without any increase in the area of dulness on percussion, and with feebleness of the cardiac impulse, a feeble and often irregular pulse, and shortness of and want of tone in the first sound of the heart—conditions which, whatever the precise form of atrophy in which they originate, require in each instance a similar plan of treatment.

The prognosis must necessarily be unfavourable, not because softening of the heart need prove rapidly fatal, but because it is a form of disease which may cause death suddenly, either from rupture of the heart, or from syncope, or from the occurrence of a paroxysm of angina pectoris, or if it does not prove suddenly fatal will in most cases run on in spite of treatment until it terminates in gradual sinking from asthenia.

Having already described the treatment to be pursued in cases of dilatation of the heart—a condition which is usually connected with softening and fatty degeneration of the muscular tissue—it is needless to recapitulate the directions concerning it. Suffice it to say, that iron in its various forms, quinine, and other vegetable bitters, digitalis, and the mineral acids, are the most efficacious medicinal agents, and that freedom from anxiety, thorough repose of mind, entire avoidance of fatigue, gentle and regular exercise in the open air, careful attention to the state of the skin, and a generous and stimulating diet, taken at regular intervals, and in moderate quantity, are the hygienic means best calculated to aid the effect of remedies, the object being to impart tone to the system, improve the condition of the blood, and so induce a more healthy nutrition of the heart. It is impossible to insist too strongly on the avoidance of all actions which are calculated to put any unwonted strain on the heart, or to accelerate its action. Weakened as that organ is, and unfit for the due performance of its function, it may yet suffice to carry on the circulation if perfect repose of mind be enjoined, and all violent bodily exertion avoided. But strong emotional feelings, or a slight straining muscular effort, either of which would make an extra demand on the energy of its contraction, might suffice to overwhelm it, and endanger life. The bowels should be so regulated, as to render straining at stool unnecessary. Stooping should be

avoided, as tending to press the abdominal organs upwards, and so interfere mechanically with the heart's action; and bodily exercise, though taken regularly, should be extremely quiet, and in amount always short of producing fatigue. The paroxysmal pain in the region of the heart, and the syncopal seizures, which are apt to occur during the course of the disease, must be treated on general principles by diffusible stimulants and anti-spasmodics internally, and by warmth and counter irritation to the lower extremities.

CHAPTER IX.

RUPTURE OF THE HEART.

Spontaneous rupture of the heart is one of the curiosities of medical pathology, and assuredly one of the accidents least amenable to medical treatment. Occurring only under circumstances productive of structural alteration in the muscular walls of the heart, it may be regarded as a consequence of some pre-existing mischief, rather than as itself a special form of disease. Aneurism of the walls of the left ventricle, hydatids, and carditis leading to abscess and perforation, may prove occasional causes of its occurrence, but its most frequent origin is atrophy of the heart, with attenuation of the walls and fatty degeneration of the muscular fibre.* When the heart is thus weakened, or prepared, as it were, for giving way, any strong emotional paroxysm, such as sudden alarm, or a violent fit of passion; any violent straining effort, and any suddenly induced congestion of the thoracic viscera may prove the exciting cause of its occurrence. But, in some instances on record, it appears to have taken place independently of any undue cardiac excitement, and to have resulted simply from the inability of the atrophied and degenerated tissue to sustain the strain which it has had to encounter, even during the tranquil action of the heart—a strain

* Cruveilhier, in his 'Anatomie Pathologique du Corps humain,' speaks of apoplectic effusion of blood into the walls of the heart as an occasional cause of rupture; and Dr. Walshe and other authors have adopted his views on the subject; but I am inclined, with Dr. Stokes, to regard the clots to which Cruveilhier refers as themselves the result of a previous partial laceration of the muscular tissue, rather than as the cause of the final rupture.

which is often excessive, in consequence of the impediment to the circulation created by the atheromatous condition of the vessels.

The statistics of spontaneous rupture of the heart are too limited to admit of any trustworthy generalisations. It appears, however, from the few cases on record,* that it occurs more frequently in males than in females, and is almost confined to the more advanced periods of life —a fact which might have been anticipated by reference to the nature of the structural causes in which it originates. The rent may be small or large, smooth or jagged at its edges, or may run directly, obliquely, or sinuously through the walls of the heart. If it takes place suddenly, as is commonly the case, the rent will probably be nearly direct; whereas, if it occur slowly, the blood may insinuate itself between the muscular fibres, and thus may work its way tortuously to the surface. Sometimes, as below stated,* the laceration takes place in the right ventricle, sometimes through the septum of the ventricles, in others through the walls of both ventricles, but most frequently it occurs in the left ventricle, about the middle of its anterior wall, or parallel to the septum, though in some rare instances, instead of being parallel, or nearly so, to the septum, it may run transversely across the muscular fasciculi.†

Death may occur suddenly as the result of the rupture, and usually does so; but sometimes the rent may be blocked with coagula, and the patient may survive for some hours, or even, as in a case related by Corvisart, may linger on for twenty-three days. One case, indeed, is on record ‡ in which, near a recent laceration of the heart, an older one

* In a paper published in vol. i of the 'Dublin Jour. of Med. Science,' Dr. Townsend refers to twenty-five cases collected by himself and to nineteen collected by Bayle; sixteen are recorded in the twelve volumes at present issued by the Pathological Society of London, and a few cases are to be found scattered through the various periodicals. Out of the sixty cases above referred to, eight were instances of rupture in the right ventricle, and fifty of rupture in the left ventricle, and two of rupture of both ventricles. The rupture in one of the two last-named instances was through the septum of the ventricles, in the other through the edge of the septum and across the right ventricle. Dr. Walshe refers to fifty-two mixed idiopathic and traumatic cases referred to by Gluge in his 'Pathological Anatomy,' and of these thirty-seven were examples of rupture of the left ventricle, eight of the right ventricle, two of both ventricles, two of the right auricle, and three of the left auricle. Dr. Stokes states (loc. cit., p. 466) that spontaneous rupture of the auricles may take place, but I can find no record of rupture of the auricles, except as the result of external violence, and if it ever occurs it must be exceedingly rare.

† A case in point is recorded by Dr. Wilks in vol. viii, p. 156, of 'Trans. Path. Soc. Lond.'

‡ See Rostan's "Mémoire sur les Ruptures du Cœur," 'Journ. de Médecine,' Juillet, 1820.

was found, firmly plugged with fibrin; but this solitary instance, which admits of easier explanation, scarcely justifies a presumption that recovery is possible.

The immediate cause of death is not mere loss of blood, which, in consequence of the inelastic, unyielding nature of the pericardium, is often small in quantity; but it is due, probably, in part to the loss of blood, partly to the shock occasioned by the rupture, and partly also to the pressure exerted upon the heart by the blood which has escaped into the pericardial sac.

There are no recognised physical signs of rupture of the heart, and even the general symptoms are not very characteristic; for they are such as may result from the rupture of an aneurism, and from other diseases of the heart and great vessels. In some instances a sudden agonising pain is felt in the region of the heart; the patient utters a piercing scream, puts his hand to his left breast, and dies. In others, in which life is not arrested so suddenly, acute pain occurs at the heart, and is followed by collapse, marked by extreme anxiety of countenance, pallor, cold clammy sweats, a fluttering pulse and faintness. At length insensibility supervenes, a few convulsive twitches of the muscles take place, and death speedily follows. In some instances on record, the same suddenly occurring and agonising pain has been felt in the region of the heart, followed by collapse, with its attendant symptoms; but after a time a remission has occurred, and the patient's recovery has not seemed hopeless until another similar paroxysm has ensued. When these symptoms recur, life is seldom prolonged beyond a few minutes, and death very commonly takes place suddenly.

The indications for treatment in cases of rupture of the heart are, to counteract collapse and sustain the circulation, and with this view, the patient should be placed in a recumbent posture, and stimulants and slight sedatives should be administered internally, and warmth applied to the surface of the body. But practically, in the great majority of cases, life is extinct before medical aid can be obtained, and even if it happen otherwise, the physician can do little towards averting the catastrophe. The damage which the heart has sustained is of a nature which does not admit of removal, and, under the circumstances, it is generally found impossible to rally the patient from the shock and to maintain the circulation.

Rupture of the valvular apparatus of the heart, which is not very uncommon as a result of straining efforts or violence, is still less so as a consequence of endocarditis. In the one case the aortic valves are those which usually give way; in the other the auriculo-ventricular valves, with their tendinous chords and the papillary muscles. And inasmuch as inflammatory and other changes, from which rupture of the valves and of the tendinous chords arises, are most common on the left side of the heart, it follows that, practically, rupture may be regarded as almost confined to the valvular apparatus of the left cavities of the heart.* For the same reason it is observed more frequently in the mitral valves and their apparatus than it is in the aortic valves.

The symptoms attendant upon rupture of a valve or a tendinous chord are extremely variable. Not unfrequently after death we find rupture of the valves or of the apparatus connected with them in persons who, during life, have never presented any symptoms characteristic of such a formidable lesion; whilst in others, again, the symptoms of rupture are well marked and characteristic. The patient is suddenly seized with acute pain in the heart, followed by excessive palpitation, irregular action of the heart, and dyspnœa; his countenance is deadly pale and expressive of extreme anxiety, the skin cold and clammy, the pulse weak and fluttering.

This variation in the symptoms which accompany rupture of the valves is strictly in keeping with what is observed in rupture of other internal viscera, and does not admit of satisfactory explanation; but in the cases under consideration there is reason to believe that the difference is partly referable to the circumstances under which the rupture takes place. If it occurs suddenly, whilst the heart's action is regular, and the patient is in his normal condition, it can scarcely fail to induce a sudden accession of alarming symptoms; whereas, if it occurs during an attack of endocarditis, by which the heart's action is greatly embarrassed and the patient is seriously distressed, the additional pain at, and disturbance of the heart may not be such as would excite attention under the existing excitement of the vascular and nervous system. Increased cardiac pain there probably would be, attended by tumultuous action of the heart and extreme irregularity of the pulse; but in a

* It may be remarked that rupture of the valvular apparatus which guards the right cavities of the heart occurs sometimes, though rarely. The late Dr. Todd has recorded an interesting example of spontaneous rupture of the chordæ tendineæ of the tricuspid valve in the 'Dublin Quarterly Journ. Med. Science,' new series, vol. v.

large proportion of cases there is none of the intense cardiac anguish, and suddenly occurring dyspnœa and collapse, which are commonly observed when rupture occurs independently of acute disease of the heart.

The diagnosis of rupture of the internal portions of the heart is by no means satisfactory. Not only are the general symptoms extremely variable, but even when most marked and most severe they are such as may arise in rupture of the heart, and in various forms of disease of the heart and great vessels. The same uncertainty attaches to the indications derivable from the general symptoms. Nevertheless, if the general symptoms and physical signs are viewed together, and thus made to illustrate each other, a tolerably correct conclusion may be arrived at in many instances. Thus, for instance, if, in a person previously in good health, a loud diastolic murmur arises suddenly at the base of the heart coincidently with the setting up of the acute cardiac and general symptoms above described, there can be little doubt as to the occurrence of rupture of the valvular apparatus. Even in a person known to have old-standing mitral disease, but in whom no acute disease is present, a sudden accession of cardiac anguish and other serious heart symptoms occurring coincidently with intensification or marked alteration in the character of the mitral murmur would be strongly in favour of rupture of some portion of the valvular apparatus, and so also would the fact of a murmur which before was systolic only becoming suddenly diastolic as well as systolic. But in cases accompanied by acute endocarditis, the character of the symptoms which accompany the disease, and the existence of loud endocardial murmurs, so complicate the diagnosis, that there are seldom sufficient grounds for a positive opinion as to the occurrence of rupture.

The treatment in these cases can only be palliative. The laceration or giving way of the valvular apparatus results, in most instances, from old-standing disease which does not admit of removal, and the rupture itself is equally beyond the scope of remedies. The only objects to be attained, therefore, are to sustain the patient under the shock he has received, and, at the same time, to relieve pain and tranquillise the nervous system. Diffusible stimulants and sedatives in full doses are the principal means by which these ends are to be accomplished, and to these may be added the local application of sinapisms or turpentine stupes, or of linseed poultices sprinkled with morphia, or smeared with hemlock or belladonna.

In connection with rupture of the internal portions of the heart, may be mentioned the pouching or circumscribed dilatation of the walls, which has been termed aneurism of the heart. Primarily referable in most instances, if not in all, to chronic disease of the endocardium, and of the muscular tissue of the heart, the immediate cause of its occurrence may be the giving way of the endocardial membrane, consequent on ulceration or external injury, but more commonly it appears to be produced by the sudden and extreme tension to which the enfeebled or damaged walls of the heart are subjected during violent straining efforts. It is rarely observed, except in the left ventricle,* and even then is a rare and exceptional occurrence, so exceptional indeed, that it was met with only four times amongst the 2161 patients examined in the dead-house of St. George's Hospital during the ten years ending December 31st, 1850.

Its symptoms are extremely variable, their precise character probably being dependent on the extent of the pouching and the rapidity with which it takes place. If it takes place slowly and is not excessive in degree, the general symptoms are simply those of dilatation, combined or not, as the case may be, with symptoms of hypertrophy. If it takes place suddenly, as the result of the giving way of the endocardium, consequent on ulceration or external violence, the symptoms are severe, and resemble those already described as accompanying rupture of the valves or of the tendinous cords.

Of the physical signs nothing definite can be stated, beyond the fact that they are sometimes those of dilatation and hypertrophy alone, sometimes of dilatation and hypertrophy combined with valvular disease. In neither case is there any sign by which to recognise the existence of the disease under consideration.

Death may occur suddenly, either from sudden failure of the heart's action, as in certain cases of dilatation, or from rupture of the aneurismal sac, or the patient may sink gradually from exhaustion, with all the symptoms heretofore described of dilatation of the heart.

* Nevertheless it does sometimes occur in other parts of the heart. In the year 1848 a case of aneurism of the right auricle of the heart occurred in St. George's Hospital. The case is recorded at p. 57 of the 'Post-mortem and Case-book' for that year, and the preparation is preserved in the museum.

CHAPTER X.

ANGINA PECTORIS.

ANGINA PECTORIS is not a disease of the heart or great vessels, but rather an assemblage of symptoms which occur in many forms of cardiac disorder. In themselves, however, they are so striking and important, and they have been so often described as constituting a special form of disease, that it will be desirable not only to point out their character, but to investigate their pathological history, and trace them to the lesion in which they originate.

The symptoms of angina pectoris in its simplest and most fully developed forms are at once striking and characteristic. The patient is suddenly seized with acute, agonising pain in the præcordial region, shooting through to the back, and extending sometimes up the neck, but more commonly along the left arm to the elbow, or even as far as the fingers. The pain, which is always sudden in its accession, is generally of a stabbing or lancinating character, but oftentimes it is said to be quite indescribable, and to produce a sense of intense oppression, or of impending suffocation—a feeling as if death were about to occur. As soon as it commences, the patient becomes deadly pale; his countenance is expressive of extreme anxiety and suffering, the skin is bedewed with drops of cold perspiration, and the pulse falters, and may be almost imperceptible. Fearful of augmenting the agonising "breast pang," which threatens to put an end to his existence, he ordinarily avoids breathing deeply, so that the respiration is at once shallow and hurried, and in some instances, after a time, there may be not only hurried breathing, but lividity of the face and total inability to lie down. The least motion aggravates his sufferings, so that, whatever he may be doing at the time of his seizure, he is unable to stir or otherwise assist himself. Yet, with all this evidence of intense suffering and nervous derangement, his consciousness is usually undisturbed, and the spinal as well as the cerebral functions remain unaffected. There is no spasm, no convulsion, no delirium, no paralysis. Not un-

frequently the rhythm of the heart remains undisturbed, and the patient does not experience palpitation, but sometimes the action of the heart is so much deranged that syncope, or even sudden death, occurs.

The original pain may last a few minutes only, or may recur at intervals, for some hours, or it may persist uninterruptedly, though with variable severity, for the space of half an hour or an hour. It usually subsides as suddenly as it came, and if it does not occasion death by syncope, it leaves the patient pale and exhausted.

Until the first accession of the disease, and during the intervals between the attacks, the patient may be apparently in good health; there is rarely much palpitation or dyspnœa—in the best marked cases absolutely none—and there is no pain or uneasiness in the region of the heart until the sudden occurrence of a breast pang announces the existence of fearful cardiac derangement, and threatens the immediate extinction of life.

The first attack is generally induced by violent bodily or mental excitement. Thus the effort of walking briskly up hill, a sudden fright, and a paroxysm of rage, have often proved the harbingers of anginal mischief, and when an attack has once occurred, there is too much reason for anxiety as to the future. For as the disease progresses, the paroxysms usually recur at shorter and shorter intervals, and not only become more severe in their character, but are excited by the slightest exercise or mental emotion, so that the least attempt to walk up an incline, the least straining on going to stool, the acts of stooping and coughing, and even a sudden movement of the arms, are all liable to induce an attack, as are also severe mental exercise or emotional excitement.

The pathological history of the disease may be briefly told. There is no form of cardiac or aortic disease with which angina pectoris has not been found associated; neither is there any form of structural mischief with which it is invariably or even generally present. The older writers, who did not employ the microscope, affirm that it is sometimes observed in cases in which the heart and arteries are healthy, as proved by dissection after death; but knowing, as we now do, how impossible it is, without the aid of the microscope, to determine whether the structure of the heart is healthy, it is obvious that no reliance can be placed on those statements, the more so as I believe no case is on record in which a genuine attack of angina has been traced to a heart which the microscope has proved to be structurally sound.

But, though angina may occur in connection with many forms of cardiac and arterial disease, there are two forms, which are closely allied pathologically, with which it is specially prone to arise. I refer to calcification or obstruction of the coronary arteries, and to fatty degeneration of the muscular tissue of the heart. Its connection with the former has long been recognised, and the microscope has revealed its connection with the latter. And, inasmuch as obstruction of the coronary arteries always results in malnutrition of the muscular tissue of the heart, and is generally followed by fatty degeneration, there are good grounds for believing that in malnutrition, if not in fatty degeneration of the texture of the heart, we have discovered one of the pathological elements of the disorder.

It is obvious, however, that something more is needed than malnutrition or fatty degeneration of the heart to induce an attack of angina; for innumerable instances of ill-nourished and damaged hearts are met with in which symptoms of angina do not make their appearance. That this additional element is a nervous element, and that the par vagum and the filaments of the sympathetic nerve distributed to the heart play an important part in the production of the attack, must, I think, be conceded. The suddenness of the seizure, the intensity and peculiar character of the pain which accompanies it, the rapidity with which it subsides, the treatment by which it is relieved, and the patient's perfect freedom from uneasiness when the paroxysm has passed off, are all symptoms which point to its nervous origin. The only question which can arise is as to the precise nature of the affection.

Heberden* and most of the older authorities have regarded the paroxysm as due to cardiac spasm, and Dr. Latham, adopting this view, has argued it with much ingenuity.† But I entertain considerable doubts as to its soundness. In the few instances in which I have been enabled to examine after death persons in whom true angina has existed during life, the ventricles of the heart have been always found relaxed and distended with blood—a condition the very reverse of that which would have obtained had cardiac spasm been the cause of death. Moreover, whatever the state of the coronary arteries, and whatever the condition of the heart in other respects, its muscular tissue has always been soft and atrophous and usually in a state of fatty degeneration—a condition in which the contractility of the muscular fibre is

* 'Medical Trans.,' vol. iii, p. 3.

† Loc. cit., p. 385.

at its minimum, and in which, therefore, it is most improbable that muscular spasm would occur. Nor are the results of my experience inconsistent with that of other observers. Even before the introduction of the microscope, the researches of Dr. Parry, of Bath, had led him to believe that angina is connected with an enfeebled state of the heart;* and as far as I am aware, no case of true angina has been reported in which the microscope has shown the muscular tissue of the heart to be healthy and possessed of its full contractile power.

This being the case, and it being difficult to comprehend how spasm of even a few minutes' duration, could fail to arrest the action of the heart and destroy life, the conclusion seems inevitable, that angina is attributable to some other cause than spasm of the heart. My own experience induces me to agree with Dr. Parry, in regarding the disease as syncopal in its nature, and connected with an enfeebled heart and an undue accumulation of blood in its cavities; and the pain as of a neuralgic character, referable, probably, to cardiac obstruction, and overdistension, consequent on the sudden failure of action in some portion of the heart's tissue, resulting from the organic changes which have taken place. And this view is confirmed by the fact that when, in the later stages of most forms of heart disease, the power of the heart begins to fail, and blood accumulates in its cavities, symptoms resembling angina arise, less acute indeed, and less sudden in their accession, and more protracted in their duration, but still essentially anginal in their character, and often so spoken of by systematic writers.

The prognosis of angina pectoris is necessarily unfavourable. In some instances the first attack proves fatal; in many more a second or a third attack; whilst in others, and perhaps in the majority of instances, the patient experiences a succession of attacks, each paroxysm being more severe than the former one, until, after a period of variable duration, sometimes extending to six or eight years, but more commonly from six to twelve months, an attack occurs in which the heart's action is arrested and life becomes extinct. Experience, however, has led me to believe that, in some instances at least, if the circumstances of the patient admit of his being placed under the requisite régime, and if due care be exercised in the treatment of the case, the condition of the heart may be so far ameliorated, that the severity of the attacks may

* Dr. Parry's work, 'An Inquiry into the Symptoms and Causes of the Syncope Anginosa, commonly called Angina Pectoris,' will well repay perusal, though published as long ago as 1799.

be mitigated, and the fatal issue of the disease almost indefinitely postponed.

The treatment of angina pectoris is fairly divisible into two parts, viz., that which will assist in mitigating and removing the paroxysms, and that which will obviate their recurrence. Diffusible stimulants and sedatives are the remedies which are especially needed during the attack, and of these, opium, brandy, æther, chloroform, and ammonia, are the most efficient. Forty to sixty drops of laudanum, or of the liquor opii sedativus may be given at the beginning of the attack in conjunction with æther, chloroform, ammonia, and brandy, and the dose may be repeated if the suffering continues. At the same time mustard poultices and hot fomentations should be applied to the præcordial region, and hot bottles or sinapisms to the extremities. If the patient be gouty, or the stomach acid and distended with flatus, a full dose of soda, magnesia, and ammonia, with six or eight drops of oil of peppermint or cajeput oil, will often prove of essential service as a precursor to or an accompaniment of the stimulants; and a brisk purge of colocynth and calomel may be given if the liver is out of order. Digitalis in full doses would probably prove useful, but as yet I have no experience of its action during the paroxysms, though in two instances I have prescribed it in combination with iron during the intervals between the attacks, and the patients have apparently derived much benefit from its employment.*

Medicine, however, can do little more than mitigate the severity of an attack, and thus it is that the prophylactic treatment of angina pectoris becomes of great importance. If we are unable to do as much as could be wished for our patient's relief during the continuance of a paroxysm, we can at least point out how he may render himself less obnoxious to attack, even if he does not escape altogether. Tranquillity of mind, bodily repose, a strict regulation of the quantity and quality of the food, a careful management of the secreting organs, especially of the bowels, which ought never to be allowed to be costive, and close attention to the general health, are the points which have to be principally attended to, the injunctions laid down respecting these matters in the section on softening of the heart being applicable to the treatment of angina. Change of scene and gentle travelling often prove of essential service; but in these cases, even more than in cases of fatty heart, it is necessary

* For details respecting the action of digitalis in cases of feeble heart, see pp. 592-3, of this treatise.

to subdue all emotional feelings, and regulate the movements of the body. The least mental excitement, and the slightest bodily exertion, are so apt to be followed by an attack of angina, and the attacks are so severe, and so likely to prove fatal, that the patient must be taught as he values his life, to avoid whatever may be likely to induce them. Neglect of these rules will inevitably lead to speedy death; whereas, by careful management, the attacks may be staved off, and the fatal catastrophe averted for many years. It is now above six years since I had the pleasure of a consultation with my friend, Dr. Bence Jones, in the case of a gentleman who was suffering severely from angina pectoris in its purest form. The attacks at that time occurred on the least excitement, or after the slightest exercise; but our patient, whose mental and bodily powers were at that time overtaxed, very wisely consented to abide by our advice and withdraw from his multifarious engagements, and led a life of perfect repose for above twelve months. The result has been that he is still alive and apparently in perfect health, and is so far improved, that he now rarely experiences any pain in his heart, and can walk and take a considerable amount of gentle exercise with impunity. The heart's action is regular, the sounds clear, the first sound unusually short.

It should, perhaps, be added, that many of the attacks described under the title of "angina pectoris" are not true examples of that disorder, but should rather be entitled "cardiac asthma." They are accompanied from the first by palpitation, dyspnœa, and lividity of the face, and are often connected with great derangement of the stomach, liver and bowels. In these cases a brisk emetic of sulphate of zinc or mustard, followed by a purge of colocynth and calomel, will often render all other treatment unnecessary; and when subsequent treatment is needed, hydrocyanic acid, and belladonna both externally and internally, will be found useful adjuncts to antacids, stomatics, and diffusible stimulants.

CHAPTER XI.

CYANOSIS.

The term cyanosis, or morbus cæruleus, is employed to denote a peculiarly blue discolouration of the skin, which experience has proved to be symptomatic of various malformations or derangements of the heart or great vessels.

Cyanosis was formerly ascribed to a direct admixture of arterial and venous blood, consequent on the existence of an abnormal communication between the two sides of the heart, or between the aorta and pulmonary artery. But dissection has shown that, provided there is constriction of the pulmonary artery, cyanosis may occur in cases in which no communication exists between the two sides of the heart, or between its great vessels; that in other cases patency of the foramen ovale, or a deficiency of some portion of the ventricular septum permits the admixture of venous and arterial blood, and that, nevertheless, if the pulmonary artery be of normal size and unobstructed, cyanosis is not thereby produced; and, further, that whatever malformation or disease of the heart exists, obstruction in or contraction of the pulmonary artery is almost always present when cyanosis is strongly marked. Hence it would appear that disease of the heart and great vessels, of whatever nature, calculated to impede the flow of blood through the pulmonary artery, and produce obstruction to the systemic venous circulation, may prove a predisposing cause of cyanosis, and that, in some instances at least, the existence of such obstruction is essential to its production.

It needs, however, little argument to prove that something more than a mere impediment to the flow of blood through the pulmonary artery and the existence of systemic venous engorgement is necessary to the production of well-marked cyanosis. Cases are of daily occurrence in which obstruction to the circulation through the right side of the heart gives rise to extreme venous congestion, and yet is not productive of cyanosis. The missing link in the chain of causation is furnished, I believe, by the condition of the skin and of the capillaries on the surface. In cases of cyanosis the skin is usually thin, and the capillaries abnormally

large; hence, when obstruction to the pulmonary and systemic venous circulation causes imperfectly aërated blood to flow throughout the system, and still more so when, in consequence of some congenital malformation, a small portion only of the blood is subjected to the aërating influence of respiration, a far darker hue is necessarily imparted to the surface than would be produced in persons whose integuments are much thicker and whose capillaries are less dilated.*

But, although cyanosis in its minor degrees is often found associated with the various forms of cardiac and pulmonary derangement, which have the effect of obstructing the onward flow of the blood, and so, of causing pulmonary and systemic venous congestion, yet, practically, its more thoroughly developed forms may be regarded as connected with malformation or disease or injury of the heart or great vessels, of a nature to admit of the admixture of venous and arterial blood, and cause the distribution of venous blood to the systemic capillaries. Amongst the pathological conditions productive of this result may be mentioned a patulous ductus arteriosus; an open foramen ovale; a deficiency, from whatever cause arising, of part of the septum of the ventricles, or of the divisions between either of the cavities on the two sides of the heart; a heart formed of one auricle and one ventricle only, in which case (the aorta and pulmonary artery often arising from a common trunk), the mixed arterial and venous blood contained in the ventricle is necessarily distributed throughout the system; transposition of the aorta, which vessel may arise from the right ventricle alone, or partly from the right ventricle, and partly from the left,† and possibly transposition of the great vessels—the aorta arising from the right ventricle, and the pulmonary artery from the left;‡ the venæ cavæ, as usual, emptying themselves into the right auricle, and the pulmonary veins into the left.

Many of these conditions of the heart and great vessels necessarily lead to the distribution of venous blood to the systemic capillaries, and, therefore, are *always* associated with greater or less degrees of cyanosis; but, others, such as an open foramen ovale, and a partial deficiency in the septum of the ventricles, are not always productive of this result. This, probably, is referable to the fact, that when the

* For details on all these points see Dr. Peacock's admirable treatise on 'Malformations of the Heart,' where the subject is fully and carefully worked out.

† For case in point see 'Med.-Chir. Trans.,' vols. xi, p. 296, and xxx, p. 131.

‡ For cases in point see 'Med.-Chir. Trans.,' vol. xxv, p. 1.

pressure on either side of the septum is equal, there is not a great tendency to intermixture of the two currents of the blood. In short, when the pathological conditions under consideration produce cyanosis, they do so, probably, in consequence, partly of the size of the opening through which the admixture of blood takes place, but chiefly in consequence of the existence of some obstruction to the passage of the blood through the pulmonary artery—a condition which leads to the forcing of venous blood through the abnormal opening into the left chambers of the heart, and thus renders it impossible that the circulation should be carried on without a large distribution of venous blood to the systemic capillaries.

Cyanosis is usually a congenital affection, but in a certain proportion of cases (about 20 per cent.), the symptoms first manifest themselves in early childhood, and in others are developed in adults.* In the last-mentioned instances there is reason to believe that ulceration and perforation of the septum between the ventricles or auricles has taken place, or that the septum has been ruptured by external violence, or by some straining effort, or that some similar circumstance has led to the enlargement of an already patulous foramen ovale.

The physical signs attendant upon cyanosis must necessarily vary with the precise condition of the heart and arteries to which the cyanotic discolouration is due; and all that can be fairly stated is, that the action of the heart is usually as forcible, if not more so than in health, and that hypertrophy and dilatation of the right ventricle are almost always present.

Deficiency of animal heat is the only general symptom constantly present beyond the discolouration of the surface. The intellect is not impaired by the circulation of the imperfectly oxyginated blood, and the various functions are seldom much disturbed. The extremities of the fingers are usually clubbed, and the nails adunc. Bronchorrhœa and bronchial hæmorrhage are not uncommon symptoms, and exertion and excitement of any kind are apt to induce excessive palpitation, with dyspnœa, faintings, or even convulsions. The occurrence of pneumonia, or of any impediment to the respiration, aggravates the cyanotic discolouration and the symptoms associated with it.

* Of seventy-one cases reported by Stille ('Amer. Journ. Med. Sci.,' No. 3, new series, viii; quoted by Dr. Walshe), forty were congenital; and of 101 cases analysed by Dr. Peacock (loc. cit., p. 118) seventy-four were instances in which the affection, if not actually congenital, appeared immediately after birth. In nineteen of the remainder the symptoms manifested themselves within two years after birth.

The prognosis in these cases is always unfavourable, though it necessarily varies, according to the precise nature of the abnormal pathological condition on which the symptoms depend. In some instances the mischief is of such a character, and the cyanosis so intense, that the functions of life cannot be sustained beyond a few months, or, possibly, a few years; but in others, in which the symptoms appear almost equally severe, life may be prolonged until middle age,* or even to an advanced period of life. I know of no special sign or general symptom by which the probable duration of any given case can be determined.

The treatment of cyanosis resolves itself into the prevention of dyspnœa and palpitation, and the maintenance, as far as may be, of the temperature of the body. Mental excitement and active bodily exercise, as in running or straining, almost invariably induce palpitation and dyspnœa, and, therefore, must be carefully avoided; warm clothing should be had resourse to, a generous and stimulating diet adopted, and a free action of the abdominal organs maintained. The conditions on which the cyanosis depends are of course irremediable.

CHAPTER XII.

MALPOSITIONS AND DISPLACEMENTS OF THE HEART.

There are two conditions under which the heart is found to occupy an abnormal position in the chest. The first of these is congenital malposition; the second displacement as a consequence of disease.

Congenital malposition is of little importance, except in so far as it may serve to mislead the unwary practitioner, and induce him to believe that the heart is displaced as a consequence of disease. But, in this point of view, it is very essential to be able to arrive at a correct diagnosis, and, fortunately, in most instances, we have the means of doing so; for when the heart is congenitally misplaced, the abdominal viscera are usually transposed, the spleen being on the right side of the abdomen, and the liver on the left. If no such transposition of the

* In a case reported by Dr. Spitta in the 'Med.-Chir. Trans.,' vol. xxix, the patient, who had been intensely cyanotic from birth, survived in tolerable health unto the age of forty, when she was suddenly seized with faintness and convulsions and shortly expired.

abdominal viscera exists, a careful physical examination of the chest, and a jealous inquiry into the history of the case is needed, in order to ascertain that the displacement is not due to any former or existing disease, but is attributable to congenital malposition.

Displacement of the heart as a consequence of disease is of frequent occurrence. It has been already shown how disease within the heart or its membranes may cause the organ to occupy a higher or a lower position than natural in the chest, or to extend further to the right or left than natural; but disease external to the heart and its membranous covering may prove even more effective in producing displacement. The attachments of the heart are such as to admit of the organ being elevated or depressed by external pressure, or being drawn or pushed to one side or the other; and the extent to which this alteration of position occurs is sometimes quite remarkable.

The causes external to the heart which operate in producing displacement of that organ upwards are ascites, ovarian dropsy, and various abdominal tumours which push up the diaphragm, and at the same time force up the heart; but the same result may be brought about by enlargement of liver or spleen, or by the pressure of the gravid uterus. In some few instances a similar displacement may be produced by the upward traction exerted on the heart as a result of the cicatrization of tuberculous cavities in the upper part of the lung, or of the contraction of pleuritic exudation.

The cause which most frequently forces the heart downwards is emphysema of both lungs; but it must be remembered that hypertrophy and dilatation of the right side of the heart are constant accompaniments of extensive emphysema, and that epigastric pulsation may be due to that cause more than to displacement the result of external pressure. Tumours of whatever character, developed in the upper part of the chest, may also exercise a downward pressure.

Pleurisy and pneumothorax are the forms of disease which operate most powerfully in driving the heart out of its position towards the one side or the other; and their effect is most conspicuous when the disease exists on the left side, and the heart is driven towards the right. Hydrothorax or dropsical effusion into the pleura generally occurs on both sides of the chest, and, therefore, fails to displace the heart sideways; but intra-thoracic tumours, and emphysema on one side of the chest, may, and do sometimes, have that effect. Great diminution in the bulk of the contents of one side of the chest, whether induced by the pressure

and subsequent absorption of pleuritic fluid, or by the contraction of one lung, consequent on tuberculous disease, is sometimes productive of lateral displacement of the heart towards the affected side, partly as a result of the hypertrophic development of the other lung, which pushes the heart over towards the opposite side, but possibly also in some measure as a result of the traction exerted on the heart during the contraction of the pleuritic exudation.

In all cases of displacement of the heart, the sounds will be heard loudest and the impulse will be felt strongest over that portion of the chest walls under which the heart lies, and it is by these signs and by the dulness on percussion in that situation, and by the absence of all these signs in the position ordinarily occupied by the heart, that we are enabled to ascertain the fact of its displacement, and judge of the direction in which it has taken place.

The effects of displacement of the heart cannot be separated from the effects of the disease to which the displacement is due, and the treatment of displacement is in like manner that of the disease by which it is caused. It should be understood, however, that the action and sounds of the heart are not seriously affected by its displacement. Its action, although accelerated, is usually regular, and its sounds remain clear.

CHAPTER XIII.

FIBRINOUS CONCRETIONS IN THE HEART—EMBOLISM.

Fibrinous concretions within the heart are formed under two very different conditions, and are productive of very different results. Sometimes they result from the slow deposition of fibrin on the valvular apparatus, or on other parts of the heart, as described in the section on endocarditis; sometimes apparently from the coagulation of the blood, especially in the right cavities of the heart. The one form of concretion is quite independent of any retardation of the circulation, and is associated with a hyperinotic condition of the blood, and probably, also, with some local irritation; the other is almost exclusively observed

towards the close of life, when the circulation is becoming languid, or when from any serious cause any obstruction exists to the current of the blood through the heart.

The cardiac disturbance and valvular murmurs ordinarily produced by the slow deposition of fibrin on the valves have been already described in the section on endocarditis, and it is only necessary further to remark that this variety of fibrinous concretion is sometimes productive of mischief elsewhere than in the heart, and gives rise to a singular set of symptoms which have been recognised as resulting from "embolism." To Dr. Kirkes we are indebted for having directed attention to this fact, and for having pointed out that portions of these concretions may become detached, and carried with the current of the circulation to different parts of the body, producing plugging of the arteries, and local arrests of the pulmonary and systemic circulations, serious cerebral disturbance, and paralysis.* But, inasmuch as the slow deposition of fibrin from the blood almost always takes place on the left side of the heart, it follows that in these cases the detached portions of the concretions are usually conveyed along the systemic circulation, and are ultimately arrested by the systemic capillaries; and the points at which experience has shown that plugging of the vessels is most likely to occur, are in the brain, spleen, kidneys, and liver. In a remarkable case, which recently occurred in St. George's Hospital, under the care of my colleague, Dr. Page, the vessels of all these organs were found plugged with fibrin, resulting from the detachment of fibrinous concretions from the left cavities of the heart.†

The symptoms in these cases necessarily vary, according as there happens or does not happen to be any co-existent endocarditis or other active inflammation, and also according to the function of the organ the circulation of which is arrested. Softening of the brain and hemiplegia are two of the most frequent results of this form of mischief, when the fibrin is detached, as it usually is, from the left side of the heart; local pulmonary congestion is its immediate consequence when the fibrin is detached from the right cavities of the heart. In either case death is almost inevitable. In some instances disintegration of the fibrinous concretions takes place, and the disintegrated matter, finding its way into the circulation, causes symptoms analogous to those observed

* 'Med.-Chir. Trans.' vol. xxxv.

† See 'Post-mortem and Case-book' for February 21st, 1862, in the museum of St. George's Hospital, under the name of Jones.

in cases of pyæmia, in addition to the special symptoms referable to the disturbance of the organ or organs in the vessels of which the detached fibrin is impacted. These, however, are exceptional cases.

We are indebted to Dr. Richardson for directing our attention to the other form of fibrinous concretion, which is not the effect of slow deposition of fibrin from the blood, but results from the coagulation of the blood itself.* It is most commonly observed on the right side of the heart, and more frequently commences in the right auricle than in the right ventricle. The coagulated blood forms mechanical adhesions to the walls of the cavity, and, as its quantity increases, portions of the coagula extend through the auriculo-ventricular opening, causing obstruction to the onward flow of blood into the ventricle, preventing the valves from closing the opening, and thus producing reflux of blood into the auricle. Unlike the concretions which result from slow deposition from the blood, these concretions when first formed are more or less blood-stained, of gelatinous consistency, and entangled in, rather than attached to, the structure of the heart; but they speedily lose their colour, and become firmer, more consistent, and opaque, and contract adhesions, either mechanical or organic, to the walls of the cavity in which they are seated. Externally they are grooved by the continued passage of the blood, or, if they have contracted adhesions to the walls of the cavity, they are indented externally by the surrounding structures, and in their centre are perforated in consequence of the current of blood having made its way through them. Their structure is usually laminated, and not unfrequently their centre is softened and is found to consist of disintegrated fibrin.†

The symptoms which result from this form of fibrinous concretions in the heart, are necessarily obscure. Primarily there may be nothing more than suddenly occurring cardiac dyspnœa, with violent palpitation, and feeble and irregular pulse, faintness, gasping respiration, lividity, twitching of the muscles, and all the usual evidences of obstruction to the circulation on the right side of the heart, and to the return of blood from the systemic capillaries.‡ But, after a time, wandering or actual delirium ensues, and, when the clot extends into the ventricle through the

* Richardson on the 'Coagulation of the Blood.'

† For full details on all these points, see Richardson, loc. cit.

‡ A case which remarkably illustrates these facts occurred under my care in St. George's Hospital, in the year 1857, and is reported in 'Trans. Path. Soc. Lond.,' vol. ix, p. 99.

auriculo-ventricular opening, a tricuspid murmur may be set up; and if portions of the clot become detached, and are carried with the blood into the pulmonary artery and produce obstruction or plugging of that vessel, symptoms of pulmonary distress and intense congestion of the lungs at once supervene. The patient ordinarily dies from syncope or from coma, sometimes preceded by convulsions.

Whether recovery ever takes place from this form of fibrinous concretion, is a question which cannot be satisfactorily answered, inasmuch as its occurrence would at once preclude the possibility of verifying the existence of concretion. But, inasmuch as these concretions usually accompany some otherwise serious malady, or are formed in the later stage of a patient's existence, when the circulation is becoming languid, it is scarcely possible to conceive that treatment can avail to produce their reabsorption. When there is reason to suspect their formation, stimulants should be resorted to, and alkalies, especially ammonia—which has been shown by Dr. Richardson to possess a remarkably solvent power—should be freely administered. Dry cupping between the shoulders might also prove serviceable in relieving congestion, and therefore should be employed.

Whilst discussing the symptoms of fibrinous concretions in the heart, and the "embolism"* which their detachment sometimes produces, it may be well to append a few remarks respecting the formation of fibrinous coagula in the arteries. Fibrinous coagula may form in the vessels at the spot where they are found after death, quite independently of local inflammation,† but, except as the result of inflammation of the vessel, coagulation in the small arteries is rare. In the larger vessels, however, extensive fibrinous coagula are often formed in connection with aneurismal dilatation, and occasionally it happens that some portions of the coagula escape from an aneurismal sac, and are carried with the blood into the smaller vessels, where they produce plugging and obstruction just as when they are detached from the heart. In a practical point of view, the fact of the existence of this source of embolism is of the greatest moment, inasmuch as it renders necessary the

* A term derived from *εμβολον*, a plug, and applied to the plugging of the vessels produced by fibrinous concretions, whether formed in the vessels or detached from the heart, and carried with the blood to the point at which obstruction takes place.

† A remarkable case of this sort, which occurred in St. George's Hospital, was reported by me in the 'Med. Gazette' for 1847, vol. ii, p. 187. A similar case was reported by Dr. Dickinson in vol. xii, p. 81, of 'Trans. Path. Soc. of London.'

greatest caution in handling an aneurismal tumour,* but otherwise, the effects produced by plugging of the arteries, are regulated by the amount and seat of obstruction, and not by the source whence the fibrinous plug is derived. Pathologically, it is right to recognise the possibility of its occurrence, in order that if the fibrinous plug does not appear to have been formed at the spot where it is found, nor to have been detached from coagula within the heart, a diligent search may be instituted for an aneurismal pouch, or some other source of fibrinous deposits.

CHAPTER XIV.

FUNCTIONAL DERANGEMENT OF THE HEART.

Having discussed the various forms of cardiac disease, it now only remains to notice those derangements of the heart and arteries which although they result from mere functional disturbance, and are unconnected with organic mischief, are yet productive of great uneasiness and distress to the patient, and are liable to be mistaken for the result of disease in the heart or great vessels.

Briefly, then, it may be stated, that there are many forms of functional disturbance of the heart which may simulate organic cardiac disease. These are characterised by palpitation or fluttering of the heart, with or without valvular murmur, and by irregularity, feebleness, or otherwise altered rhythm of the pulse. They are of common occurrence, and depend upon a variety of causes, which operate at different periods of life, but especially during adolescence and middle adult life. Intimately connected with perverted innervation, and often induced, therefore, by excessive mental exercise and sedentary occupation, great anxiety, and strong mental emotions, they are, nevertheless, very generally found associated with local or general derangements confessedly productive of nervous irritation or nervous exhaustion. Amongst these, may be specified the uterine and ovarian excitement which so generally attends the commencement and cessation of the

* In Virchow's 'Arch. für Path., Anat., und Phys.,' vol. xii, p. 410, 1857, quoted under the head of "Treatment of Aneurism by Manipulation," in vol. iii of Holmes' 'System of Surgery,' a case is recorded in which embolism was produced in this way.

catamenial discharge in women, and often results in spinal irritation and aggravated hysteria; excessive venery, and the vicious indulgence of masturbation by the youth of both sexes; the influence of certain poisons in the blood, such as strong green tea and tobacco, an excess of spirituous liquors, and the *materies morbi* of gout and rheumatism; spanæmic and anæmic conditions of the blood, and certain derangements of the stomach and liver, characterised by the existence of flatulence and acidity. In adult life, especially, the last-named cause is a fertile cause of functional palpitation. Wearied and exhausted by a long day's work and anxiety, during which, perhaps, they have taken nothing to eat, men sit down to dinner, with their minds still upon the stretch, and not only partake too heartily of the food, but swallow it quickly, without proper mastication. If their minds were at rest, and their bodies in vigorous exercise, they might possibly digest it, but leading, as they do, a sedentary life, it is impossible that the digestion should be otherwise than deranged. At first mere weakness of the stomach and slight irregularity of the bowels, are complained of; but, after a time, the most careful dieting will not suffice to rectify the chronic derangement which has taken place, and which has issued in flatulence, acidity, habitual disorder of the bowels, turbidity of the urine, palpitation, and other symptoms of general derangement.

Of all the causes of palpitation of the heart, none demand more serious attention, or call for more remark in a practical work, than the functional disturbances now under consideration. Those persons only who have suffered severely from functional palpitation, or, who have had examples of it brought before them in the course of their professional ministrations, can have an idea of the painful character of its symptoms, or of the degree to which it often simulates organic disease of the heart. The palpitation itself is often greater than that which accompanies diseases of the heart, except, perhaps, in its later stages; the cardiac action is excessive both in force and frequency, often irregular, and sometimes intermittent, and the carotids and other large arteries pulsate with a force apparently exceeding that of the heart itself. The pulse also may be unequal and irregular in the highest degree; at one moment slow, at another extremely rapid; at one moment full and forcible; at the next so weak as to be almost imperceptible; at one moment regular; at the next irregular, or even intermittent. The apex of the heart beats in its normal position; but, never-

theless, the cardiac impulse can be seen and felt over an unnaturally large surface. If the heart be healthy, the impulse will be simply more forcible, and more widely felt than natural, or, possibly, will have somewhat of a knocking character. If it be hypertrophied, the impulse will be more distinctly heaving; whilst, if the heart be thin and weak, the impulse, though still felt over an extended area, will be more feeble, and of a flapping character. The area of dulness on percussion varies with the pre-existent condition of the heart, and is seldom modified to any sensible degree by an attack of functional palpitation; but, in some instance, in which the paroxysms of palpitation have been severe and prolonged, the dulness has been said to extend to the right of the sternum, in consequence of the long continued distension of the right cavities of the heart.* Theoretically, this is, perhaps, conceivable, but I have never been able to verify the observation in cases in which the heart was healthy; and knowing, as I do, the practical difficulties which beset the attempt to determine the exact limits of the heart, I cannot but feel that the possible extension of præcordial dulness in these cases is a sign which at least cannot be relied on. The sounds of the heart, unless modified by pre-existing disease, are simply louder, clearer, and more abrupt, than natural; but, not unfrequently, the first sound attains a metallic quality at the apex,† and may even be reduplicated, or the second may be reduplicated at the base. Sometimes, again, systolic murmur is heard both at the base and apex of the heart, and this holds good as well when the patient is apparently healthy as when he is anæmic or spanæmic. This has been repeatedly forced upon my notice during the examination of persons about to insure their lives, in about one eighth of whom the nervous excitement produced by the ordeal through which they have to pass, and the forcible effort of blowing the spirometer, induces temporary systolic murmur at the base, and in a certain, though far smaller proportion at the left apex. In some cases the murmur is extremely loud, and a systolic thrill may be felt in the carotids and larger arteries. These instances, however, are rare.

The general symptoms are indicative of great distress. The patient complains not only of palpitation, but of a fluttering and thumping, as

* Walshe, loc. cit., p. 570.

† This metallic ringing quality of the first sound is characteristic of excessive cardiac excitement, and is doubtless attributable to the energy of the muscular contraction, and the consequently increased tension of the auriculo-ventricular valves.

if the "heart were about to jump into the mouth" There is a sense of fulness and of deep oppression in the præcordial region, with pain, breathlessness, and tendency to faintness—the pain being usually of a dull aching character, but sometimes sharp and stabbing. Not unfrequently the pain is relieved by pressure, but the patient is unable to lie on the left side, because that position induces increase of palpitation, or aggravates the existing "soreness at the heart." He suffers from frequent giddiness, with pain and heat in the head, singing in the ears, flushing of the face, and coldness of the extremities, and from time to time, when the paroxysms are severe, and faintness or actual syncope supervenes, the skin is bedewed with a clammy perspiration. Add to these symptoms dyspepsia, with excessive flatulence, acid eructations, restlessness at night, depression of spirits and pertinacious nervous solicitude as to the nature and issue of his malady, and it will be readily concluded that the sufferings induced by serious functional palpitation are scarcely exceeded by any form of cardiac disease.

The question then arises as to how these cases of functional palpitation are to be distinguished from cases in which palpitation is due to organic mischief? It may seem strange, but it is nevertheless true, that one of the evidences least likely to deceive is the constant complaint which the patient makes of palpitation, and the morbid anxiety which he manifests respecting it. The sufferer from organic disease of the heart seldom complains of his cardiac disorder, but calls attention to the shortness of breath or to some other symptom resulting from it, whereas the sufferer from functional palpitation is ready at all times with a detail of his sufferings, of which palpitation is always the most prominent. Another fact of some importance is, that functional disturbance of the heart is often relieved by active exercise, and aggravated by the patients remaining long in one posture, and as this is the reverse of what is usually observed in cases of organic disease, it is of value in a diagnostic point of view. Unfortunately, however, when the patient is either anæmic or spanæmic he is unable to take exercise, however sound his heart may be, and thus in some instances the diagnostic value of this sign is materially lessened. In short, the diagnosis of functional palpitation is by no means easy, and in difficult cases can only be determined by repeated physical examinations. The impulse of a palpitating heart may be violent, its rhythm irregular or intermittent, its systolic sounds accompanied by murmur, and nevertheless the heart may be sound, and these symptoms may all disappear as soon as the palpitation subsides.

On the other hand the temporary disappearance of the local and general symptoms of cardiac disturbance will not alone suffice to indicate with certainty the functional character of the disorder; for in many forms of organic cardiac mischief the symptoms may be temporarily subdued, and the heart may for a time regain its regularity both in force and rhythm.

The most difficult point to be determined in doubtful cases is as to whether the palpitation is due to hypertrophy of the heart, or simply to functional disorder. The question, of course, is complicated in many instances by the coexistence of hypertrophy and functional palpitation, but even in uncomplicated cases it is not always easy of solution. Indeed it is often impossible to arrive at a conclusion from the result of a single interview; and no careful practitioner would venture to express a positive opinion until he had made repeated examinations. The principal points to which we have to look for guidance in our diagnosis, are the results of percussion, the position of the apex beat, and the relative force of the heart's action, and of the radial pulse. A healthy heart under the influence of palpitation may be felt pulsating violently over an extended surface of the chest; and although in such cases the impulse is usually quicker or more abrupt than in cases of hypertrophy, yet it may be difficult, if not impossible, to distinguish its force and extent from those of an hypertrophied heart. In the one case, however, the area of percussion dulness will not be increased, or, if temporarily extended, will resume its proper limits when the palpitation has subsided; the position of the apex beat will remain unchanged, and the radial pulse will be weak in comparison with the apparent force of the cardiac action; whereas, when hypertrophy exists, the area of percussion dulness is permanently enlarged, the apex beats lower than usual, and far to the left of its normal situation, and the radial pulse is permanently full and forcible.

The treatment applicable to functional palpitation varies according to the circumstances of each case, whereas the principles on which it is founded are always the same. Our first aim must be to tranquillise the excitement of the circulation; our next to guard against the recurrence of the malady by getting rid of its exciting cause, or removing the patient from its influence, by regulating his mode of life, and giving tone to the various organs. With a view to alleviate the violence of a paroxysm and shorten its duration, a mixture of anti-spasmodics, stimulants, and sedatives is especially useful, and amongst these may be

mentioned the ammoniated tincture of valerian, the fœtid spirits of ammonia, sal volatile, camphor, chloroform, æther, brandy, opium, belladonna, and henbane. Administered in full doses and at brief intervals, these remedies will usually cut short a paroxysm which without them might have lasted for many hours, or might even have been protracted, with occasional intermissions for several days; and as, when judiciously administered they are not productive of general disturbance, but rather obviate the mischief which often results from long continued and violent palpitation, there cannot be a doubt as to the propriety of employing them. But it should be clearly understood that diffusible stimulants and anti-spasmodics will not always suffice to get rid of the palpitation. In some instances—especially in gouty persons, and in those who are suffering severely from dyspepsia—the tongue is furred, and the breath offensive, and there are acid eructations, and other evidences of stomach derangement. In such patients the remedies already alluded to will be of little avail until the stomach is emptied, and the disordered secretions corrected or got rid of: the first step to be taken under these circumstances is to evacuate the contents of the stomach by an emetic—nothing answers better than sulphate of zinc or mustard and water—and then to administer a calomel purge, followed by rhubarb and magnesia, and subsequently by bismuth, soda, colchicum, hydrocyanic acid, cajeput oil, or whatever may seem most likely to counteract the acidity of the stomach, allay its irritability, and disperse the flatulence. The first action of an emetic, or the first full bilious evacuation of the bowels will often afford very great relief, and this may then be sustained by the action of the very stimulants and anti-spasmodics which previously had proved inoperative.

But something more is needed than to mitigate or subdue an existing paroxysm of palpitation. Though the first violence of the attack may be overcome, the patient will still experience the pain, the fluttering, or the discomfort from which he has long suffered, and will be as anxious as ever respecting the sensations over which he has long brooded and despaired. The first step, therefore, which the physician should take when the paroxysm has subsided, is to examine the heart carefully, and then to assure the patient that however great his sufferings, the attack was purely functional and unconnected with organic disease. He should point out how common such attacks are found to be in persons pursuing sedentary occupations or otherwise placed under circumstances unfavourable to their general health, how inevitably they arise in certain states of the blood, and in certain disordered

conditions of the secretions, and how essential it is that the patient should have repose of mind, and lead a life in which cheerful society, change of air, careful dieting, moderate yet sufficient bodily exercise, and due regulation of the secretions will one and all contribute their quota towards the maintenance of the general health. The patient should be made to understand that no medicine can avail, so long as over-fatigue and anxiety of mind, or excessive indulgence of the sexual desires are permitted to exert their baneful influence, and that even under the most favourable circumstances time will be an essential element in his cure. If the stomach is the principal organ at fault, the mineral acids, taraxacum, and the vegetable bitters, or the alkalies in combination with hydrocyanic acid, small doses of colchicum, and warm carminatives will often prove serviceable, and these may be administered either with or without valerian, camphor, chloric æther, and the other anti-spasmodic and stimulating remedies of which mention has been already made. In most instances of functional palpitation, iron proves a remedy of great value, and so does the daily use of the shower bath; and there are few cases in which a belladonna plaster will not be found useful in tranquillising the action of the heart. When the patient passes restless nights, and is irritable and nervous during the day, opium, henbane, or hop, are remedies which cannot be safely neglected. Venereal indulgence, and the use of tobacco, strong tea, and other substances which are apt to induce or aggravate palpitation, must of course be abandoned.

The prognosis in these cases must be regulated by the circumstances under which the patient is placed. In young persons it is almost invariably favourable, but in persons advanced in years the heart itself is often texturally diseased, the excretory organs are sluggish in their action, and the patient, therefore, is in a condition unfavourable to recovery, and although, if he will consent to follow the plan of treatment best calculated to improve his general health, he may shake off the nervous irritability on which the functional palpitation depends, it is scarcely probable that he will be enabled to do so, if his circumstances compel him to keep up the strain on his mind and body which have contributed so largely to the production of his disorder. Hence it often happens that persons advanced in years, who begin to suffer from functional palpitation, are led to regard their malady as hopeless, and, brooding over the pain and discomfort they experience, too frequently become confirmed hypochondriacs.

A few remarks should, perhaps, be added respecting the alterations

in the rhythm of the heart, which are often referred to functional causes. There cannot be a doubt that many perversions of the cardiac rhythm are observed in persons in whom the stethoscope fails to reveal any evidence of disease, beyond that furnished by the altered rhythm itself; nay, more, experience has shown that every variety of perverted rhythm may long exist in persons whose heart's action is tranquil, who are not conscious of cardiac disturbance, and who, moreover, go through life and attain to mature age, without the occurrence of any disagreeable symptoms referable to the condition of the heart. Irregularity and intermission of the heart's action is the most common of these varieties of perverted rhythm, extreme feebleness of action is another, and extreme infrequency of action is, perhaps, the least common variety. The first variety is seldom productive of much inconvenience; but the last two are often accompanied by fits of giddiness, faintness, or actual syncope, which are always alarming, and, if unattended to, may even prove fatal. My own experience does not enable me to state positively that these different varieties of altered cardiac rhythm do not result from mere functional disturbance; but I confess to feeling extremely sceptical as to their being really attributable to the cause assigned. I do not wish to imply that the rhythm of the heart may not be temporarily affected by stomach derangement, nervous excitement, and other functional causes; but without microscopic investigation of the structure of the heart in each particular instance, I should rather be inclined to regard these perversions of rhythm, unless of temporary duration, as resulting from organic or textural cardiac mischief; for they are one and all met with in cases in which the heart is structurally diseased, and are never observed in children in whom the heart is presumably sound. Further, in every instance of so-called functionally altered rhythm in which I have had the opportunity of examining the heart after death, traces of textural disease have been discovered by the microscope; and it seems inconsistent with the history attaching to many cases in point, that there should exist any permanent functional derangement. True that irregularity and intermission of the pulse, which have been noticed for months, or possibly for years, may gradually subside as the patient's health and strength improve; but this surely affords no proof of mere functional disorder, but is equally, if not more intelligible on the supposition that the blood has become healthier and more stimulating in character, and the heart more vigorous and more susceptible of stimulation. And knowing as we do how long persons whose

hearts are structurally diseased may go on in the apparent enjoyment of health, how readily disease of the heart's texture is overlooked if the aid of the microscope is not resorted to, and how seldom, until of late years, the microscope has been employed to ascertain the existence of cardiac disease, presumption, I think, is in favour of the belief that in those cases which have been recorded of persistently perverted cardiac action unconnected with structural disease of the heart, the non-discovery of organic disease has been attributable to the imperfection of the means employed to verify the fact, and not to the non-existence of disease. Practically, however, it must be borne in mind that every form of altered cardiac rhythm may exist in cases in which our present means of diagnosis fail during life to afford any proof of organic disease, and in which, even after death, the aid of the microscope must be invoked, if structural disease is to be discovered.

CHAPTER XV.

DISEASES OF THE AORTA.

Aortitis.

Inflammation of the aorta is a singularly rare disease, so rare, indeed, as to possess little practical interest. I have never met with an instance of it myself, and can find records of only a very few cases; and from the anatomical description given of some of these, there seems reason to doubt whether the disease was indeed what it is represented to be. Its origin is involved in much obscurity. Strange as it may appear, the few cases on record prove that it is seldom associated with endocarditis, and point rather to some peculiar alteration in the condition of the blood, as the starting point of the arterial mischief. Stranger still is the inference deducible from the particulars of certain cases which have been placed on record, that in some instance at least no obvious effect is produced by aortitis, except irritability, restlessness, and general uneasiness!

Thus, then, as there are no reliable physical signs of aortitis, as its general symptoms are so variable and uncertain that it is impossible to diagnose its existence during life, and as its anatomical characters, also,

are so obscure, even after death, that it is difficult in all cases to determine its existence, it is obviously useless to attempt a description of the disease, or to lay down rules for its treatment. Its history has yet to be written, its symptoms and physical signs investigated and described, and the best method of treating it ascertained.

Dilatation of the Aorta.

The aorta is subject to changes of structure analogous to those already described as affecting the valves of the heart—changes which, whether induced by inflammation or resulting from slow degeneration of the tissues, impair its natural elasticity and contractility, destroy the smoothness of its internal surface, and render its coats brittle. Pathological research has shown that these changes are connected with the deposit of various abnormal matters in the coats of the artery, more especially in its inner coat. It is probable, however, that all these deposits originate in an exudation of an albuminoid character, which subsequently undergoes fatty, atheromatous, cartilaginous, or calcareous degeneration, and thus assumes the variety of forms which are met with on dissection after death. Sometimes the deposits are seen as small, isolated, opaque, yellowish specks, slightly elevated above the inner coat of the artery; sometimes several of these isolated specks coalesce, and present themselves as hard, opaque masses projecting considerably above the surface of the inner coat of the artery; and sometimes, again, the aggregation of these morbid deposits is such as to destroy the inner coat of the vessel, so that its surface is rendered rough or even ragged.

The seat and extent of these deposits vary greatly. They usually exist in largest quantity, and, in some instances, are almost wholly confined to the ascending and transverse portions of the arch of the aorta, but it is not uncommon to find them to a greater or less degree throughout the whole arterial system. In some instances they are only dotted here and there over the surface of the vessel; in others they are thickly spread over a large area, and greatly thicken the coats of the vessel, and occasionally they are set so closely, and are of such a calcareous nature that they render the coats of the vessel hard, inelastic, and brittle.

These changes are of very frequent occurrence in advanced life, and this has led some pathologists to regard them as essentially connected with

senile decay of the tissues. But there are not sufficient grounds for this opinion. Many of the most remarkable instances of degeneration of the coats of the aorta which have come under under my notice have been in persons not yet passed the prime of life; and aneurism, which is a form of disease essentially connected with these structural changes in the artery, is more common before than after the age of fifty. Therefore it must be admitted that although degeneration of the coats of the arteries, like degeneration of other tissues of the body, is most liable to occur in advanced life, yet that the causes which set it in operation may be fully developed at almost any period of man's existence.

The results which flow from this form of disease are of great practical importance. The loss of elasticity and contractility of the artery consequent on the altered structure of its coats may cause it to yield to the dilating force of the blood thrown into it at each systole of the heart, and so to become permanently dilated and enlarged; its loss of elasticity, by increasing the resistance to the stream of blood, and its loss of contractility, by impairing its power of propulsion, may impede the arterial circulation, throw additional labour on the heart, and give rise to hypertrophy, or to hypertrophy and dilatation of that organ; the roughening of its internal surface may occasion a murmur synchronous with the systole of the heart, and the brittleness of its coats may prepare the way for the occurrence of aneurism.

Dilatation sometimes affects the whole circumference of the aorta, so that the artery is uniformly enlarged, but, very commonly, the dilatation is more marked in the anterior and right portion of the ascending aorta, and in the upper surface of the transverse portion of the arch—the parts against which the current of the blood is directed with the greatest force; and sometimes the dilatation is even more circumscribed, and assumes a sacculated form, so that distinct pouches project from the external surface of the vessel, in consequence of the coats having yielded more rapidly at some points than at others. These pouches form the commencement of that variety of disease to which the title of aneurism has been applied.

Considerable dilatation and roughening of the coats of the aorta may take place without occasioning murmur; nay, more, the vessel may be sacculated, and may even present a distinct aneurismal pouch, without proving the source of murmur.* This is partly dependent on the

* This fact may be illustrated by reference to the case of James Howard, aged thirty-four, who was admitted under my care in the King's Ward of St. George's

character of the blood, and the nature of the roughening; partly on the force of the blood's current, and partly on the particular portion of the vessel, which happens to be the seat of roughening and dilatation. If the circulation be languid, the blood healthy, the roughening extensive, rather than prominent, and the seat of roughening such that the current of the blood is not directed against it, there may not be, and, probably, will not be a murmur. But, when on the other hand, the blood is anæmic or spanæmic, and the circulation active, and when the current of the blood is directed against a portion of the vessel at which there exists a distinct prominence or roughness of a nature to cause a forcible eddying of the blood, a murmur can scarcely fail to accompany each systole of the heart.

In many of these instances the aortic valves are thickened, and give rise to a systolic murmur, which it is difficult to distinguish from the murmur occasioned by dilatation and roughening of the aorta; but when a murmur is due to the latter cause alone, it will usually be heard louder at the second right cartilage and in the track of the aorta than it is over the semilunar valves, and not unfrequently the pulsation and thrill of the dilated aorta will be felt on placing the finger above the sternal notch, and pressing it downwards, behind the sternum. Sometimes, indeed, the pulsation may be seen as well as felt above the sternal notch, and also above the clavicles, and the murmur audible over the arch of the aorta is propagated along the great vessels in the neck.

When pulsation and thrill are clearly perceptible above the sternal notch, and also in the large vessels of the neck, a suspicion of sacculated aneurism may often arise, and the diagnosis may not be easy. Simple non-sacculated dilatation of the aorta, especially when the internal surface of the vessel is roughly calcified, may give rise to the most intense

Hospital, on April 2nd, 1862. This man had an aneurism the size of a large orange which sprang from a part of the ascending and transverse portions of the arch, and involved the origin of the arteria innominata. This aneurism had pushed its way forward, had caused erosion of the sternum, and presented itself superficially as a pulsating tumour. On dissection, the aneurismal sac just referred to was found half filled with old laminated fibrin, the aorta was extensively diseased in other parts besides that which formed the seat of the aneurism, and at about an inch above the semilunar valves there was a large empty pouch about the size of a hen's egg which had a smooth opening into the aorta about the size of a shilling. Yet, in this case, during the whole time that the patient was under observation there was not the slightest roughness or murmur, either over the aortic valves, in the track of the aorta, or over the aneurismal tumour For full details of the case, see 'The Post-mortem and Case Book' for April, 1862, in the museum of St. George's Hospital.

pulsation, thrill, and corresponding murmur, which, even under ordinary circumstances, may be felt and heard an inch or more beyond the limits of the dilated vessel; and when these phenomena are intensified by the existence of anæmia or spanæmia, or by nervous excitement, it is difficult, if not impossible, to determine at one examination whether circumscribed or aneurismal dilatation of the vessel exists. But careful and repeated examinations will seldom fail to settle the question. If sacculated aneurism exists in a position and of an extent to cause pulsation and thrill on the parts above referred to, percussion could scarcely fail to indicate the fact, nor could there fail to arise, within a limited period, some signs of pressure referable to the aneurismal sac. These of course would be absent if the symptoms were due to simple peripheric dilatation of the vessel.

Aneurism of the Aorta.

Aneurism of the aorta may be defined as a circumscribed dilatation of an artery, consequent on disease in one or more of its coats. Its local character, and that alone, distinguishes it from simple dilatation of the aorta, with which it is pathologically allied. For whether the vessel be generally dilated, or whether its coats have yielded to pressure at one or more points, the change is referable to the impaired elasticity and altered character of the tissues of which the vessel is composed. And, inasmuch as the conditions which produce these alterations in the coats of a vessel do not operate on the aorta alone, but exert their influence throughout the entire system, aneurism of the aorta is usually found associated with a diseased condition of the other arteries.

Thoracic aneurisms are of different varieties and different forms, and occur at different parts of the thoracic aorta.

The three coats of the vessel altered in structure, and less elastic than in health, may remain unbroken, and, yielding under the pressure of the blood from within, may constitute the walls of the aneurismal pouch; or the inner, or the middle, or the outer tunic may give way, leaving the other two coats to form the covering of the tumour; or the two inner coats of the vessels may be ruptured, and the outer tunic only may form the covering of the sac; or yet, again, the two inner coats may crack or rupture, and blood, escaping through the fissures, may separate the outer from the middle tunic, producing a so-called "dissecting aneu-

rism."* Practically, however, these anatomical divisions of aneurismal tumours are of little value; for not only may one variety merge into another as the disease progresses, but the symptoms they severally produce are seldom distinguishable during life. In all cases the whole of the tunics of the artery may be ultimately destroyed, and the walls of the tumour may consist of nothing more than the condensed tissue of the parts on which the aneurism presses.

The forms which aneurismal swellings assume are various, and so also are the sizes to which they attain. The swelling may involve the whole circumference of the vessel, or it may arise from one part only of its walls, the opening into the aneurismal sac being often no larger than a shilling; it may be fusiform or globular, or simply sacculated, or it may be irregularly sacculated, and its size may vary from that of a walnut to the bulk of a child's head.

The contents of the sac are also varied. They may consist entirely of solid fibrin, which is usually found in a laminated form, or they may be wholly fluid; but more commonly they are partly solid and partly fluid, the sac being lined by layers of fibrin which are firmer and denser in proportion as they are nearer to the walls of the sac, and soft and loose textured, and scarcely decolourised where they are in contact with the blood. The firmer the fibrin, and the more thickly it lines the cavity, the greater the strength it affords to the sac, and the less rapid is the progress of the disease; so that in some instances, in which the tendency to the firm coagulation of fibrin is great, the sac becomes nearly filled by coagula, the tumour is rendered solid and resistent, the tendency to further enlargement is stayed, and the aneurism is cured.†

Thoracic aneurisms arise most commonly from the ascending portions of the arch of the aorta, less frequently from the transverse portion of the arch, still less frequently from its descending portion, and quite as seldom from the descending thoracic aorta below the arch. This is explicable by the fact that the commencing portion of the aorta is that on

* For full particulars of a remarkable case of dissecting aneurism of the aorta which fell under my care in St. George's Hospital, in the person of Felix Ridout, aged thirty-two, see Hospital 'Post-mortem and Case Book' for December, 1861. The preparation is preserved in the museum of the hospital.

† For details of a case in which two aneurisms existed in one patient and were cured spontaneously, see 'Trans. Path. Soc.,' vol. ix, p. 167. Other cases of spontaneous cure are recorded in the 'Post-mortem and Case-books,' for 1854 and 1855, in the museum of St. George's Hospital.

which atheromatous and calcareous degeneration of the coats of the vessel most frequently takes place, and in which the strain of the impulsive action of the heart is necessarily most felt. Dr. Sibson,* who has taken infinite pains in collecting and analysing cases of thoracic aneurism, informs us that amongst 703 cases, of which he obtained the histories, or found specimens in various museums, no less than 87 were instances of aneurism of the sinuses of Valsalva, 193† of the ascending aorta alone, 112 of the ascending and transverse portions of the arch conjointly, and 28 of the entire arch; so that some portion of the ascending aorta was involved in no less than 420 cases, or in considerably more than half of the whole; whilst the transverse portion of the arch was affected alone in 120 cases, and conjointly with the descending aorta in 20 cases; the descending part of the arch alone in 72 cases; the descending thoracic aorta below the arch in 71 cases.

There is yet another point of considerable interest in connection with the origin of aneurismal tumours. It has long been noticed that they do not arise indifferently from all parts of the circumference of the aorta, but spring most frequently from those parts of the vessel against which the current of the blood is more particularly directed. In this respect they follow a law which is in constant operation in all rivers, and leads to the pouchings or aneurismal dilatations so constantly observed on their banks. The extent to which this law determines the position of aneurisms is clearly shown by Dr. Sibson's statistics. He informs us that aneurisms of the sinuses of Valsalva most frequently effect the right coronary sinus, and least frequently implicate the left; that in fifteen instances aneurism of the ascending aorta originated in the anterior aspect of the aorta, in 46 in the right, and in 8 only in the left side, and that in 19 cases the upper aspect of the transverse aorta was the seat of aneurism; its posterior, or, more properly, its right aspect in 41; and its anterior, or, more properly, its left aspect in 19 cases. These facts are important in a practical point of view; for when once aneurismal dilatation has commenced, the tendency of the tumour would naturally be to increase in the direction in which the vessel has yielded, and the only means by which that tendency would be modified is the existence of mechanical obstacles to its enlargement in that direction; and as the organs which present the obstacles to its extension

* For full statistical details respecting thoracic aneurism, see Dr. Sibson's 'Medical Anatomy,' Fasc. v.

† Of these 193 no less than 52 were examples of dissecting aneurism.

must themselves suffer pressure from the aneurismal sac, and as the effects or symptoms produced by aneurism are in strict relation to the amount and seat of that pressure, it follows that the precise seat of aneurismal dilatation will, in some measure, determine the nature of the symptoms, and conversely, that the seat and nature of the symptoms of pressure may assist in deciding the question as to the point of origin of the aneurism.

The effects produced by aneurism vary not only with the seat of the tumour, but with its size and consistency, and the rapidity of its enlargement. A small sac may exert pressure on more important organs than a large one, and may therefore give rise to more urgent symptoms; but it will produce less extensively diffused signs of pressure. A soft aneurismal pouch, or, in other words, a pouch containing little coagulated fibrin, will press less forcibly, and therefore will be less liable to produce ulceration and erosion of the harder structures than one which is partially filled with coagula. An aneurism which commences gradually and progresses slowly will excite less distress—less urgent symptoms of pressure—than one which, even though much smaller, has been rapidly developed, and has not given time for the surrounding structures to accommodate themselves to its presence.

Thoracic aneurism may simply push aside the surrounding parts without materially interfering with their function, or it may exert an amount of pressure calculated to impede the exercise of their function; or it may excite congestive inflammation, ulceration, or erosion of the parts; or it may destroy them, by causing arrest of the circulation in them, with gangrene as its natural consequence; or it may press on the nerves, and produce excessive pain, and a variety of reflex spasmodic actions.

The parts which are liable to suffer are, the lungs and their appendages, the heart and great vessels, and the bony structures which form the anterior walls of the chest, the bones of the spine, the œsophagus, the recurrent nerve, the sympathetic and the thoracic duct; and their liability to be affected stands much in the order in which it is given above. It is worthy of note, however, that an aneurism rarely presses upon the heart, unless it be an aneurism of the sinuses of Valsalva, or of the very commencement of the aorta, and that, just in accordance with what might have been expected from their positions, and from the direction in which they are given off, aneurisms of the ascending aorta seldom produce dysphagia, or laryngeal stridor, but cause cough and dyspnœa,

and press upon the right lung or the right bronchus, at least four times as often as on the left.* Aneurisms of the transverse portion of the arch are more apt to cause dysphagia and interruption to the radial pulse, and are extremely prone to induce pressure on the trachea and recurrent nerve, giving rise to dyspnœa, orthopnœa, and stridor; whilst aneurisms of the descending portion of the arch are more constantly accompanied by pain in the back, and erosion of the vertebræ, than are aneurisms of other portions of the arch, and more frequently occasion dysphagia and deficiency of breathing on the left side of the chest. Aneurisms of the descending thoracic aorta below the arch still more commonly induce pain in the back and pressure on the left bronchus, but much less frequently give rise to dysphagia.

The effects of pressure on the various organs within the thorax is sufficiently obvious. When the lungs or their appendages are pressed upon, congestion, inflammation, or gangrene of the pulmonary tissue may be set up, and violent cough and hæmoptysis may result; the walls of the trachea or of one of the main bronchi may be eroded and perforated; and hæmoptysis, to a greater or less extent, may take place, according as the bleeding results from intense congestion of their lining membrane, or from oozing from the aneurismal sac, or from actual bursting of the aneurism.

When the aneurism presses upon the heart, it may force the heart out of its natural position, and, interfering with the proper action of the valves, may give rise to the occurrence of obstructive or regurgitant murmur; when it presses upon the larger arteries, it may not only push them out of their position, and, by altering their forms, occasion murmurs, but it may impede or arrest the circulation through them, and thus lead to their becoming plugged with fibrin, and produce vertigo, headache, loss of memory, disturbed sleep, delirium, and a variety of cerebral symptoms. When it presses upon the larger veins, it may prevent the return of blood to the right cavities of the heart, induce somnolence and congestion of the whole venous system, and favour the production of dropsical effusion. Thus, not unfrequently the superficial veins of the chest become turgid, and the face, neck, and one or both

* Dr. Sibson's statistics give forty-one as the number of cases in which pressure was exerted on the right lung or the right bronchus, and ten only as the number of cases in which the left lung alone was affected. In seven instances both lungs suffered. For full statistical details on the seat of pressure in the different varieties of aneurism of the aorta, see Sibson, loc. cit.

of the upper extremities œdematous, in consequence of pressure on the superior cava, or on the innominate veins.

When the sternum or any portion of the bony framework of the chest is pressed upon, erosion of the part takes place, the soft structures yield, and the aneurismal sac, pushing forward through the opening which the erosion has caused, forms a prominence or tumour, which is plainly visible, and usually pulsates synchronously with the systole of the ventricle. When the spine is pressed upon, the bones are eroded, paraplegia may be produced by pressure of the sac, and hæmorrhage may take place into the vertebral column.

When the œsophagus is pressed upon, dysphagia is produced, ulceration and perforation of the coats of the tube may take place, and the symptoms may closely resemble those of œsophageal stricture. In some of these instances, blood oozes from the aneurismal sac into the œsophagus, trickles down into the œsophagus, and either passes away by the bowels, or is rejected by vomiting.* When the recurrent nerve is pressed upon, cough of an irritative and spasmodic character is usually produced, œdema of the lungs may take place, and sudden death may occur from closure of the glottis.

When the sympathetic is subjected to pressure, contraction, or dilatation of the pupil on the affected side is produced, and in some instances ptosis of the eyelids also.†

When the thoracic duct is pressed upon, rapid wasting necessarily ensues.

In addition to the above symptoms produced by direct pressure, thoracic aneurism is apt indirectly to induce hypertrophy, or hypertrophy and dilatation of the heart. The diseased condition of the coats of the aorta, and the loss of elasticity and contractility consequent thereupon, form an obstacle to the flow of blood through the aorta, which causes hypertrophy of the left ventricle; whilst the impediment to the pulmonary circulation, occasioned by the constantly recurring congestion of the lungs, gives rise to hypertrophy and dilatation of the right cavi-

* See details of a case which I reported in vol. i, p. 79, of 'Path. Soc. of Lond.,' in which an aneurism of the arch of the aorta burst both into the trachea and œsophagus.

† A case illustrating both these facts has just proved fatal under my care in St. George's Hospital, see 'Hospital Post-mortem and Case-book,' for April 16, 1862, name, Howard. For detailed account of the influence produced by aneurismal pressure on the sympathetic nerve, see an admirable paper by my colleague, Dr. Ogle, in 'Med.-Chir. Trans.,' vol. xli.

ties. In many cases, however, the arterial disease, when of limited extent, and not affecting the commencement of the aorta, does not appear to offer much obstruction to the circulation, and the heart, therefore, remains of its natural size.

It will be obvious, then, that there is no pathognomonic sign of thoracic aneurism, no symptom to which the disease invariably gives rise, and none which certainly indicates its existence. If the aneurismal sac be small, be developed slowly, and be deeply seated in the chest, it may scarcely attract attention during life, and may first proclaim its existence by bursting, and causing fatal hæmorrhage. Even when of considerable magnitude, an aneurism may fail to interfere materially with the functions of the surrounding parts, and, if deeply seated, may be discovered for the first time on dissection after death.

The diagnosis, therefore, of this form of disease, is necessarily involved in much obscurity. Sometimes, more especially, when the aneurism pushes forward, and, causing erosion of the ribs or sternum, presents itself as a pulsating tumour on the chest, the physical signs and general symptoms are such as to leave little doubt as to the nature of the mischief; though even in this case, as I shall presently show, the most cautious observers may sometimes be mistaken. Sometimes, again, even when no tumour can be seen, or pulsation felt, the physical signs may be so peculiar, and the general symptoms produced by the pressure of the aneurismal sac so striking, that the nature of the lesion may be pretty clearly indicated. But in all cases, during the early period of the disease, and in some even up to the fatal issue of the malady, the symptoms are extremely obscure; and it is only by a careful consideration of the physical signs and of the peculiarities attaching to the general symptoms that it is possible to arrive even approximately at a conclusion. It will be necessary, therefore, to pass carefully in review the physical signs and principal general symptoms met with in these cases.

The physical signs produced by an aneurismal tumour vary greatly.

Inspection may inform us of turgescence of the superficial thoracic veins, or of the veins of one or both upper extremities, accompanied by œdema, and more rarely by paralysis, as the result of obstruction to the thoracic circulation, consequent on the pressure of the aneurismal tumour; or of deficiency of expansion in certain portions of the chest, resulting from the pressure of the aneurismal sac on a large bronchus; or of local pulsation, or of bulging caused by the outward pressure of the aneurism; or even of a distinct prominence or tumour formed by

the aneurismal sac, which has caused erosion of the bony framework of the chest, and has thus presented itself upon the surface. The tumour thus formed is usually somewhat conical in shape, and when it is large and its base is extensive, the skin which covers it becomes tense, thin, and of a smooth, glazed appearance, and ultimately red, or even ulcerated. It usually pulsates synchronously with the systole of the heart, though pulsation may be absent if the sac be filled with solid fibrin; whilst, on the other hand, if there be few coagula in the sac, the tumour may be seen expanding as well as pulsating with each systole of the heart.

The position which the tumour occupies on the chest depends not only on the precise part of the vessel from which the aneurismal sac arises, but on a variety of accidental circumstances which determine the direction in which its enlargement takes place. In a diagnostic point of view, therefore, it affords a very uncertain basis for an opinion respecting the seat of the aneurism; but it may be stated generally, as the result of extended observation, that when the aneurism springs from the ascending aorta, and pushes forward in the chest, the tumour usually shows itself to the right of the sternum, somewhere between the third rib and the clavicle; that when it involves the transverse portion of the arch, or the commencement of the descending portion, the tumour more frequently lies to the left of the sternum, rising sometimes above the clavicle, and at others occupying the infra-clavicular or mammary regions; and that when it springs from the aorta below the arch it seldom produces bulging anteriorly, but rather causes erosion of the vertebræ, and, pushing its way to the left, induces prominence of the left side. In the few instances in which aneurism of the descending aorta below the arch pushes forward in the chest, and causes prominence on its anterior surface, the tumour is usually seated to the left of the sternum, between the third rib and the clavicle. Even when there is no distinct tumour, the existence of localised pulsation in the infra-clavicular regions, when unattended by visible and abnormal pulsation of the carotids, and by the peculiar condition of the arterial pulse which usually accompanie permanent patency of the aortic valves, is very significant of the existence of aneurism; so also is distension of the veins on one side only of the neck, or in one of the upper extremities, or distension of the veins of both sides of the neck, if unattended by evidence of tricuspid regurgitation, or of dilatation of the right side of the heart. These symptoms must be caused by local pressure on the veins, and there is nothing so likely to produce that pressure as an aneurismal tumour.

Palpation is of service in enabling us to distinguish the character of the movement imparted to the chest walls, or to the local tumour at each systole and diastole of the heart. When the sac contains firm coagula of fibrin, the hand takes cognisance of its solidity, and its motion is felt to be single and impulsive, conveying the impression of a violent throbbing or heaving; but when the quantity of fibrin which it contains is small, its want of solidity is at once perceived, and distinct expansion as well as impulse is felt at each systole of the heart. In some of these cases, when the walls of the sac are thin, and its contents fluid, the movement is quite undulatory, and in others the impulse and the recoil of the artery convey to the hand the impression of a double impulse, the diastolic impulse being sometimes the most forcible. In other instances the application of the hand apprises us of the fact that the impulse over the seat of abnormal pulsation is more forcible than that felt over the apex of the heart, and thus lends additional weight to the supposition that the abnormal pulsation is referable to aneurism. Further, palpation often reveals the existence of thrill or purring tremor, which is usually confined to the local bulging or tumour, but is sometimes felt over a large portion of the chest, and may be perceptible above the clavicle and in the course of the large vessels. Again, when the finger is placed above the sternal notch, it informs us of any increase in the pulsation of the transverse portion of the arch of the aorta, and of any displacement of the arch upwards. So also, when the vocal fremitus is stopped, either by the pressure of the tumour on a main bronchus, or by the intervention of the tumour between the lung and the chest walls, palpation makes us acquainted with the fact, and guides us to the seat of mischief; and, lastly, the hand enables us to compare the condition of the arteries on the two sides—the radial, brachial, subclavian, and carotid, and thus, by detecting obstruction to the circulation through one or more of these vessels, not unfrequently directs us to the seat of aneurism.

Mensuration may inform us of an increase in the size of one side of the chest, consequent on the presence of an aneurismal tumour, or of an increase in the distance from one point to another, caused by local prominence or bulging; or it may indicate a diminution in the expansive movement of the chest over the part where the presence of the aneurismal sac prevents the inflation of the lungs.

Percussion may afford very valuable information, or it may fail altogether in throwing any light on the seat or nature of the mischief.

If the patient be thin, and the lungs sound, and, if the aneurism is seated superficially, then percussion will discover the existence of the tumour, even though it be no larger than a walnut. But, when the aneurism springs from the posterior surface of the vessel, and pushes backwards on the chest, the most careful percussion may fail to detect it, unless it be of large size. Even when as large, or larger than an orange, percussion will sometimes fail to indicate its presence, if the patient be stout, and the chest walls thick; and this difficulty will be increased by the existence of emphysema, or of tubercular or pneumonic consolidation of the lung. Practically, the dulness caused by aneurismal dilatation of the aorta is most readily discoverable on the anterior surface of the chest, and to the right of the sternum, and therefore, in connection with aneurism of the ascending portion of the arch, and the commencement of the transverse portion. But dulness, referable to aneurismal tumour, may also be discovered to the left of the sternum, especially above the level of the second rib; and aneurisms, which are of considerable size, and push towards the surface of the chest, may cause dulness on percussion between the scapulæ, or in the posterior and lateral regions. There is nothing, however, in the character of the percussion dulness to distinguish it from that produced by other tumours in the chest, and its indications, therefore, must be interpreted by reference to coexistent physical signs and general symptoms. It is important to remark, however, that the area of superficial dulness is no guide in these cases to the size of the aneurism, inasmuch as the tumour being usually spheroidal, or conical in shape, a small portion of it only approximates to the chest walls, and it would be dangerous to employ the forcible percussion which would be necessary to elicit a dull sound from the more deeply seated portions; for it must ever be borne in mind, that the least violence applied to an aneurismal sac is productive of extreme danger. Not only may it cause extension of the disease, or actual rupture of the sac, but it may dislodge a small portion of the coagulated fibrin, and give rise to embolism and speedy death.*

Auscultation, like percussion, sometimes furnishes almost conclusive evidence of the existence of aneurism, and at others fails altogether in throwing light upon the subject. When the aneurismal sac presses

* For a case in point see Virchow's 'Arch. für Path. Anat. und Phys.,' vol. xi, p. 410, 1857, quoted in Holmes' 'Surgical Dictionary,' under the head of "Treatment of Aneurism by Manipulation."

on the heart, it may interfere mechanically with the closure of the valves, and may thus give rise to a systolic or diastolic murmur, and may also occasion systolic murmur, by pressing upon some portion of the aorta or pulmonary artery. But the aneurismal sac itself may be the seat of murmur, and is so not unfrequently. If it be small, deeply-seated in the chest, and filled with firm coagula; if the opening into it be smooth and narrow; or, if, even though the opening be large, the sac be smoothly lined with fibrin, no murmur or other abnormal arterial sound may be heard; and if the sac does not compress any of the larger bronchi, no stethoscopic evidence of its existence may be discoverable. In these cases, the sounds, though often louder and more intense, are usually identical in character with those heard over healthy arteries, and are not such as would attract attention in the absence of more specific evidence of disease. On the other hand, if the aneurismal tumour be seated superficially in the chest, if it be only partially filled with coagula, and if the opening into it be rough and of tolerable size, a murmur will be not unfrequently audible; whilst, if it presses upon a large bronchus, the respiratory sounds may be excessively weak, and the respiratory sound prolonged over the portion of lung supplied by that bronchus; or respiration may be almost inaudible in that part, and exaggerated in the adjacent parts; or, if the lung be more or less condensed, the respiratory sounds may be bronchial in character, and accompanied by râles and rhonchi. It should be clearly understood, however, that a murmur is not necessarily present even when the aneurism is seated superficially. Only last week I had a patient under my care in St. George's Hospital, in whom a large aneurism of the ascending and transverse portions of the arch had caused erosion of the sternum, and presented itself as a pulsating tumour on the surface of the chest, and, although considerable expansion of the sac took place at each systole of the heart, not the slightest murmur was audible.*

The character of the sounds over aneurismal tumours is extremely variable. In one instance, the sounds, though louder and more intense, may be identical in character with those heard at the base of the heart:† in another, although two sounds may be audible, they may both be weaker than the corresponding sounds at the base of the heart; in another, no sound may be audible at all analogous to an

* See 'Hospital Post-mortem and Case-book' for April 16, 1862; name, Howard.

† This was so in the case of Howard, above referred to.

arterial sound, but the ear takes cognisance of a dull, muffled noise, which conveys the impression of an impulse or pulsation. And when, as often happens, though far less frequently than is commonly supposed, the arterial disease gives rise to murmur, an equal diversity is observed in the results of a stethoscopic examination. There may be systolic murmur alone, or a diastolic murmur alone, or there may be a double murmur—systolic and diastolic; in which latter case the systolic portion of the murmur may be louder than the diastolic, or the diastolic portion louder than the systolic. The systolic is the commonest form of murmur; it may be soft and blowing, or harsh, rasping, whirring, or roaring in character, whilst in pitch, duration, and intensity, it is subject to every possible variation. The diastolic murmur is of comparatively rare occurrence, and is apt to vary, or even disappear from time to time; and, although it is seldom loud or harsh, yet, in many of its qualities, it is just as variable as the systolic murmur. A double murmur occurs not unfrequently, but it possesses nothing of a distinctive character, and can be distinguished from a valvular murmur only by reference to its position and to the direction in which it is heard.

The murmurs produced by aneurismal tumours, like the murmurs which are excited by valvular disease of the heart, may originate in any cause which is capable of produing forcible eddying of the blood. Roughening of the artery, and especially of that portion of it which forms the entrance to the aneurismal sac; the rapid flow of blood into the sac through a roughened opening, or the pressure of the sac on some portion of the aorta, thereby producing narrowing of the vessel, may all, or either of them, contribute to the production of a systolic murmur. One or other of these causes very commonly exists, and hence, the frequency of systolic murmur as an accompaniment of aneurism. But it is otherwise in respect to a diastolic murmur. This can only be produced by the forcible reflux of blood from the aneurismal sac into the artery, and its presence, therefore, denotes that the sac is only partially filled with coagula, and, therefore, admits a free current of blood into its interior; that its orifice is rough, and that a considerable portion of its contents are expelled at each systole of the vessel—conditions which comparatively seldom obtain. This, therefore, explains the comparative infrequency of double aneurismal murmur, and of the feebleness of the diastolic, as compared with the systolic murmur. The still greater infrequency of a diastolic murmur alone, is explicable by the fact that

the orifice of the sac can rarely present a roughened surface to the current of blood as it flows out of the sac without presenting an equally roughened surface to it as it enters. In all cases, of course, violent cardiac action, arterial excitement, and an anæmic or spanæmic condition of blood, increase the tendency to murmur, whereas feebleness of the heart's action in like manner diminishes it, or may even cause it to cease. When the murmur is very intense, it may be audible not only to the patient himself, but even to by-standers, without the aid of the stethoscope or of immediate auscultation, but it does not possess a distinctive character. It is only when a murmur does not exist over the heart, or, if existing in the heart, is of mitral or tricuspid origin, and, therefore, is not transmissible along the great vessels, that the discovery of a murmur in some portion of the thoracic aorta becomes suspiciously significant of the existence of aneurism. Indeed, it must be admitted that the diagnosis of thoracic aneurism, by reference solely to the physical signs, is in many cases impossible, and that, even under favourable circumstances, it must often be involved in great obscurity, in consequence of the physical signs being almost devoid of a distinctive character.

This statement is contrary to the opinions formerly entertained relative to the physical diagnosis of thoracic aneurism. Thus, Dr. Hope asserts* that "the cylinder is scarcely less capable of affording decisive indications of pectoral, than of ventral aneurism," and then, after stating † that "pulsation attends every species of dilatation of the aorta," he implies that aneurismal murmur is a constant attendant on aneurismal dilatation. But modern experience does not bear out Dr. Hope's assertion. Out of thirty-three cases, of which I have notes,‡ twelve were unattended by either murmur or pulsation, in fourteen only was a pulsation perceptible, in ten only was there the slightest tremor, and in nineteen only was there any murmur; whilst, out of twelve cases reported by Dr. Greene,§ four cases presented no pulsation, and six cases were unattended by murmur. In all these cases the existence of aneurism was suspected during life, and repeated stethoscopic examinations were made, with a view of obtaining some confirmation of the

* Hope on 'Diseases of the Heart,' p. 442.

† Loc. cit., p. 447.

‡ These do not include cases in which the aneurism pointed externally, and in which, therefore, the tumour could be seen and felt.

§ In an able article in the 'Dublin Quarterly Journal' for 1846.

diagnosis; so that it was not for want of careful examination that no clue could thus be obtained to the real nature of the lesion.

A question therefore naturally suggests itself, as to whether there is any combination of circumstances which justifies us in diagnosing the existence of thoracic aneurism. This can only be answered in general terms.

In many cases the presence of aneurismal dilatation is manifested by symptoms which can scarcely be mistaken. The heart's action is, perhaps, so greatly disturbed, as to direct attention to the seat of mischief, and then extended dulness on percussion, abnormal impulse, aneurismal murmur and tremor, together, perhaps, with tumescence of the superficial thoracic veins, and obstruction of the pulse, very clearly indicate the nature of the disease; and, even if aneurismal murmur be absent, as it is sometimes even in cases where distinct impulse can be felt, still careful attention will generally reveal the nature of the case, and the physician, aware of serious mischief, may forewarn his patient of his perilous condition.

But it often happens that the arterial tumour, of no great size, produces none of these obvious symptoms, and that, even when certain physical signs are present, their character is not distinctive. In such cases the tumour is overlapped by the lung, and does not occasion dulness on percussion. It is often no larger than a walnut, and does not give rise to external impulse; it is unaccompanied by murmur, and the stethoscope, therefore, whether applied in front or on the back, affords no precise information on the subject, and if the heart's action is not much interfered with, the principal physical signs of aneurism are absent.

But it is otherwise in regard to the general symptoms of the disease. The different organs are so situated in the chest, that the primary divisions of the bronchi, the trachea, the œsophagus, the spine, the nerves, the heart, and the large vessels, can hardly all escape the pressure of a tumour occupying a central position in the upper part of the thoracic cavity. Accordingly, cough, hæmoptysis, dyspnœa, and wheezing, alteration in the voice, difficulty in swallowing, pain in the chest, back, or neck, modifications of the radial pulse, ptosis of the eyelid, and alterations of the pupil, are symptoms which often present themselves under these circumstances, and possess distinctive features which will usually point to a correct diagnosis.

Cough very generally, if not always, exists and present peculiarities which, though not pathognomonic, are yet sufficiently marked to excite

a suspicion of aneurismal pressure. It is generally paroxysmal in the first instance, and frequently loud, shrill, and ringing. It is severe at one time, almost absent at another; relieved by certain postures, aggravated or immediately induced by others. When accompanied by sputa, as it generally is at some time during the progress of the disease, the matters expectorated afford no clue to the real nature of the existing mischief. They may consist of a thin, semi-transparent, serous fluid; or of glairy, or else of a white, frothy mucus; in some instances they may be pneumonic in character, whilst in others, or in the later stages of the disease, they may consist of muco-purulent matter of varying colour and consistence. These are occasionally streaked or mixed with blood, arising from pressure on the pulmonary veins, or from hæmorrhage during ulceration of the bronchial tubes, or from gradual oozing from the sac itself, when it has ulcerated through and communicates directly with the cavity of the air tubes; and sometimes the quantity of blood which escapes in this way amounts to several ounces in the day. In a remarkable case recorded by Dr. Gairdner, hæmoptysis to the extent of several ounces occurred four years and eight months before the patient's death.*

Dyspnœa is usually but not always present, its presence being regulated by the existence or non-existence of irritation of the bronchi, or of the phrenic, the recurrent, or the pulmonary nerves. In many cases, when present, it is severe from the first, and its cause is so obscure, that its presence appears unaccountable. It is paroxysmal and often very violent in its character, yet seldom lasts above one or two hours; it is quite independent of atmospheric changes, and is much relieved by certain postures, which vary, of course, according to the exact position of the tumour. It may be attended by little or no cardiac disturbance; the patient may be entirely free from pain, and may inflate his chest without fear or difficulty; and, though a sense of oppression at the chest is almost constant, and suffocation occasionally seems imminent, yet the respiration is generally vesicular, though possibly more feeble on the one side than on the other. This symptom may, of course, be complicated by the presence of tubercular disease of the lung; but the coexistence of such disease with aneurism is rare,† and cannot be regarded as likely in most cases to interfere with the diagnosis; and if bronchitis, pneumonia, or pleurisy occur, or if emphysema supervenes

* In the 'Med.-Chir. Trans.,' vol. xlii.

† See pp. 665-6 of this treatise.

and aggravates the dyspnœa, a due regard to the history of the case, and to the order in which the symptoms arose, will generally serve to show the true relationship in which these changes stand to the original dyspnœa resulting from the aneurismal pressure.

The wheezing is sometimes of so peculiar and singular a character as hardly to be confounded with that produced by any other cause. It is markedly stridulous, and comes on gradually, without fever or other constitutional disturbance; it increases progressively, yet is not accompanied by emaciation, or by the unhealthy aspect of malignant disease; it is relieved or aggravated by the position of the patient, and though it frequently accompanies the inspiratory effort, it is usually best marked during the act of expiration. Cough may have preceded it, and is generally one of its concomitant evils; but the patient's health is not necessarily interfered with, and neither the cough, the dyspnœa, nor the wheezing, are much relieved by ordinary treatment. Indeed, being referable to one and the same cause, these symptoms are oftentimes relieved or aggravated by the same circumstances. They are generally relieved by a free venesection, as also by spontaneous hæmorrhage, which causes an actual diminution in the size and bulk of the aneurismal tumour, and thus lessens the pressure on the trachea and bronchial tubes. When the tumour is pressing on the anterior part of the trachea, they are alleviated by an upright posture, or rather by the patient's leaning somewhat forwards with his elbows resting on his knees. This, in six instances, of which I have notes, was the easy, and, therefore the favourite position of the patient. In some instances, the patient may endeavour to obtain relief by throwing his head forcibly and suddenly backwards—an action which in some measure dislodges the tumour, and for a time lessens the occlusion of the windpipe. In other instances, where the aneurism presses on the larger bronchi, the patient is relieved by assuming a semi-erect position, with his body inclined to the right or to the left side, according as the tumour presses upon the left or right bronchus. When this is the case, the mere attempt to lie on the affected side induces a severe paroxysm of cough and dyspnœa, and the stethoscope shows that the air has much freer access to the lung when the patient lies on the non-affected side. In some instances the position of the aneurism in the chest may thus be predicted with considerable accuracy during life.

But the symptoms just mentioned, though sometimes arising from one and the same cause, may be produced by causes totally different.

While the wheezing is excited by the alteration in the form of the air tubes, or, as is sometimes the case, by œdema and turgescence of the submucous cellular tissue of the trachea and larger bronchi, caused by the constant pressure and irritation of the tumour, the cough and dyspnœa may be mainly attributable to other secondary causes. As the sac enlarges and becomes filled with coagula, the pulmonary vessels or the aorta may be subjected to pressure, or the heart's action may be directly interfered with; the phrenic nerve may be irritated, or the pneumogastric or some of its branches may be pressed upon; and thus I am satisfied that the symptoms in question, when most severe, are produced not unfrequently. And according as they are due to one cause or another, so do they vary in their character. Sometimes abdominal breathing is greatly deficient; at others it is exceedingly violent and spasmodic. In one instance, during a severe paroxysm, the respiratory efforts succeed each other in rapid succession; in another they are slow, deep, and forcible, effected by the joint action of all the muscles of respiration; and whilst in one case the cough is hardly attended by any difficulty of breathing, in another dyspnœa is the more prominent feature. In one case, of which I have notes, I am satisfied that death, which occurred suddenly, without any hæmorrhage, was the immediate result of suffocation, induced by pressure on the par vagum. Several instances are on record in which the *post-mortem* appearances—the absence of any material disease of the heart, or of any other organic lesion calculated to give rise to sudden death—leave little doubt as to the agency of this cause.

The modification which the character of the voice undergoes not unfrequently resembles that which accompanies chronic laryngitis. But, unlike the voice of chronic laryngitis, the voice, when modified by aneurismal pressure, is by no means constantly or even generally of the same character. At one time it is small, faint, tremulous, and whispering; at another, in the same patient, it is attended only by some little hoarseness; at another, again, it is shrill and harsh, or resembles the voice of laryngeal ulceration. Possibly these modifications in the tone of the voice may be referable to variations in the action of their exciting cause in different cases, or even in the same case at different periods; each modification being produced by a change in the capacity or shape of the windpipe, as a result of displacement of, or pressure on, the trachea; or by a greater or less degree of atrophy or irregular action of the laryngeal muscles, consequent on compression, more or less com-

plete, of the recurrent nerve; or by œdema of the glottis and adjacent parts, consequent on local congestion, which dissection has shown to have occurred in several instances where the return of the blood to the heart has been much retarded. Whether these variations of tone can be each referred accurately to its corresponding pathological condition of the air passages, and what each precise condition may be, experience does not yet enable us to determine; but it is quite certain that they sometimes occur with great rapidity, are apparently unconnected with corresponding constitutional changes, are altogether uninfluenced by external agency, and appear to be affected solely by the position of the patient and by the varying condition of his circulating system; so much so, that the whole character of the voice is sometimes altered by depletion. Thus, if viewed attentively day after day for some little time, this symptom alone may be made to afford very valuable information. If any doubt should exist on the matter, it can be readily cleared up by means of the laryngoscope, which, in cases of aneurismal pressure, will show that the larynx is quite free from disease.

Dysphagia is a common, and often a prominent symptom of thoracic aneurism. It existed in nine out of the twelve cases recorded by Dr. Greene, and in seventeen out of thirty-three cases of which I have notes. Fortunately, it presents several distinctive peculiarities. Unlike the dysphagia produced by scirrhus of the œsophageal tube, it is unaccompanied by the rapid wasting, the sallowness of complexion, and the peculiar aspect characteristic of such disease; it is often rapid in its commencement, uncertain in its progress, and varies greatly from day to day in its completeness and severity. One day the patient may be unable to swallow the smallest quantity of solid food, and with difficulty takes nourishment enough, even in a fluid state, to satisfy the cravings of hunger; whilst, on another day, or, perhaps, only a few hours afterwards, he may be able to swallow, without pain or difficulty, whatever his inclination induces him to attempt to eat. Thus, I have seen a person eat a mutton-chop in the evening, who in the morning could hardly take a drop of milk. The difficulty of swallowing is often, and indeed generally increased by whatever tends to accelerate the circulation. It is, therefore, relieved by perfect rest, and, in many cases, also by free venesection. When a hard-working man is admitted into the hospital, suffering from dysphagia, arising from the cause now under consideration, the relief afforded by the use of the lancet, and by the freedom from exertion which he there enjoys, is often so remark-

able and so immediate, that he fancies a rapid cure is to be effected. Nor is this the only way in which relief is to be obtained. Depending, as the symptoms do, on pressure of the sac, the dysphagia may be alleviated by whatever takes off the pressure and alleviates the spasm arising from its irritation—a spasm occurring not only at the immediate seat of obstruction, but occasionally much higher up the tube. Accordingly, it not unfrequently happens that dysphagia is present only when the patient is in a recumbent posture, and that, even when it is constantly present, an inclination of the body forwards will enable him to take that with ease which he would be otherwise utterly unable to swallow. In one instance I saw very marked relief from the patient remaining in this posture about an hour before the time for his meals. After such a preliminary arrangement he could swallow without much difficulty; whilst, without such a preparation, he could scarcely swallow even the smallest quantity of sopped bread. How different these from the symptoms presented by malignant disease of the œsophageal tube!

The diagnosis between the pressure from an aneurismal tumour and that produced by any other mass of disease within the thorax is, to some extent, more difficult and complicated. But even here certain differences exist which should lead in most cases to a correct conclusion. The previous history and present aspect of the patient, and the existence of disease, whether malignant or scrofulous in other parts of the body, would cause a suspicion of similar mischief within the thorax; whilst the absence of such disease, the existence of an hereditary tendency to aneurism,* the constant variation in the amount of the dysphagia, the peculiar character of the accompanying cough and dyspnœa, the wheezing and aphonia, the palpitation, headache, occasional vertigo, and other symptoms of disturbed circulation, together with the observed effects of remedies and change of posture, would excite suspicion of aneurismal pressure.

Dysphagia is a symptom of peculiar significance in relation to the existence of thoracic aneurism. Not only does it denote pressure on the œsophageal tube, but so seldom does such pressure result from aneurism of the ascending portions of the aorta, that its presence may be regarded as almost certainly indicative of disease of the tranverse or

* I am acquainted with a gentleman whose paternal grandfather, uncle, and father have all died from the effects of aneurism, and whose sister at the time when I last saw him was labouring under that disease.

descending portions of the arch.* But it must be understood that pressure on the œsophagus is not always productive of dysphagia, and that the degree of dysphagia is not necessarily an index to the amount of pressure. Strange as it may appear, aneurismal pressure may even cause ulceration of the coats of the œsophagus without giving rise to notable dysphagia; and, on the other hand, dysphagia may be excessive in cases in which the evidence of pressure as derived from inspection after death is comparatively slight; so that local nervous irritability, exciting spasm, appears, in some instances at all events, to be an essential element in the production of this symptom.

Pain in the chest and back is a not unusual accompaniment of aneurism, and is a symptom which often arrests attention. Its cause generally appears to be irritation of the nerves, produced by pressure of the aneurismal sac; for though it is usually more severe when erosion of the sternum or vertebral column is going on, it exists to a very considerable degree in instances where the bones are found uninjured after death. It is not attributable, therefore, to the process of ulceration. The patient, probably a healthy-looking person, without appreciable disease of the spine, complains of pain at one particular spot—pain at once so constant and severe, as to render him restless and miserable during the day, and utterly unable to sleep at night. He dreads certain postures, as inducing or aggravating his suffering, and he particularly avoids lying on his back. He moves with the greatest caution, and can hardly be prevailed upon to take active exercise, so certainly is his pain increased by so doing. The expression of his countenance is indicative of suffering, and there is that in his manner and ordinary gait which would prevent a cautious practitioner from treating the case lightly. The pain itself, which is usually accompanied by a distressing sense of oppression at the chest, and occasionally by constriction across the epigastrium—the result of irritation of the phrenic nerve—is often referred to a spot in the back, a little below the junction of the neck and thorax; sometimes it extends to one of the shoulders, and shoots down the corresponding arm; and, sometimes, though principally affecting the back, it will dart with great violence along the neck. In most instances it is constant, but is aggravated in paroxysms which seem independent of external causes. It is often described as a dull, heavy, aching, or gnawing, or occasionally as a lancinating or a burning pain, and sometimes the patient, in dreadful

* Dr. Sibson's statistics give 2 per cent. as the proportion of cases in which dysphagia is occasioned by aneurism of the ascending aorta, and from 21 to 33 per cent. as the proportion for other portions of the arch.

agony, declares his back is breaking. In six instances I have seen this agonising pain accompanied by occasional dysphagia, and followed by hæmoptysis, or by slight hæmatemesis, the only obtainable sign of disease; the stethoscopic, as well as the other general symptoms of aneurism, being altogether absent. Indeed, pain in the back—pain, be it remembered, of a peculiar character, invariably referred to the same spot, and not increased by external pressure—is so often, for a time at least, the only discernible symptom of aneurism, that, whenever such pain is met with in practice, it ought never to be treated lightly. Nor am I alone in counselling extreme caution in cases such as these. "The description given of this pain by the patients," says Dr. Greene, "was always the same, so that its character could be recognised, and there was a degree of earnestness in the description very unlike that of persons who are merely feigning. I mention this circumstance because from their long complaining of this pain without any apparent cause to account for it, four of these patients had been condemned as malingerers."

Viewed, then, simply by itself, this pain is an unusual and a striking symptom. Its paroxysmal character, its aggravation by exercise, and by whatever tends to excite the circulation, its marked increase when certain postures are assumed, the relief which is often obtained by venesection, and by all measures tending to tranquillise the action of the heart, and its want of connection with any obvious cause, are circumstances sufficient to excite the suspicion of any careful practitioner. And although pain may be excited in the same situation by disease of the vertebræ, by malignant disease within the thorax, by enlargement of the bronchial glands, dependent on cancerous or scrofulous deposits, and, indeed, by anything causing pressure in the same situation, still, when once suspicion has been excited, it is seldom that, from the history or symptoms of the case, some facts may not be elicited calculated to assist in confirming the diagnosis.

The pulse is not necessarily interfered with in these cases; but according as the arteria innominata or the left subclavian is pressed upon or implicated in the aneurismal swelling, so of course does the pulse vary. Sometimes it is altogether absent at one or both wrists; sometimes it is weaker or later on one side than the other; sometimes it is remarkably weak at both wrists, and quite out of character with the impulse and sounds of the heart; and sometimes, again, it may be unequal and attended by a peculiar thrill in the one arm, whilst it is soft and regular in the other. These peculiarities, though sometimes produced by irregular distribution of the vessels and by other causes

than aneurismal pressure, are yet sufficiently suggestive of mischief to direct attention to its real seat, and thus afford valuable accessary evidence.

These symptoms, then, together with hæmoptysis and hæmatemesis, which, until the fatal event takes place, present nothing distinctive, beyond their obvious want of connection with their usual exciting causes, I take to be the most important of the general symptoms of thoracic aneurism. Others there are, which it is unnecessary to do more than indicate. Such, for instance, are the phenomena arising from obstructed thoracic circulation; turgescence of the superficial thoracic veins, or of the veins of one or both upper extremities, accompanied by œdema, and more rarely by paralysis; and such again are the phenomena referable to interference with the cerebral circulation, the sudden startings at night, the frightful dreams, the occasional syncope. These and many others, though valuable as accessary to, and confirmative of other more characteristic symptoms, are yet hardly sufficient of themselves to excite a suspicion as to the real cause of mischief. They simply indicate the presence of some foreign matter within the chest, but afford no clue to its nature.

It is constantly asserted that, in the diagnosis of aneurism, we have the physical signs alone to depend upon; that the general symptoms I have described may be met with separately, and under every variety of combination, independently of pressure from an aneurismal tumour; and, consequently, that they cannot be regarded as diagnostic marks of the presence of the disease in question. But although it cannot be denied that each and all of the general symptoms already enumerated may possibly occur independently of aneurismal pressure, yet I am convinced, by close and careful observation, that no more uncertainty attaches in many cases to indications derivable from this source than to those obtainable by means of the stethoscope. Without undervaluing the assistance of auscultation in the diagnosis of thoracic aneurism, I do not hesitate to affirm that, in at least half the cases met with in practice, more especially in the early stage in which they first attract attention, more real information is to be derived from a careful consideration of the general symptoms than from the imperfect revelations of the stethoscope. In some instances, indeed, auscultation gives us all the information we require; but, in many, even up to the moment of death, it furnishes nothing but negative information. It tells us, perhaps, of an obstructed bronchus, or shows us that no organic disease of the heart or lungs exists to account for the hæmoptysis which is

frequently occurring; but it often fails to do more than this, for the aneurismal pouch may be smoothly lined with coagula, and may not occasion a murmur, and the heart's action may not be greatly disturbed. Many cases have fallen under my observation at St. George's Hospital and elsewhere, in which the existence of aneurism was correctly diagnosed by means of the general symptoms alone, the stethoscope having failed to afford any positive evidence on the subject. Indeed, when the general symptoms are well marked, the nature of the disease must certainly suggest itself to any one who has had much experience in such cases. The appearance of the patient, the curious succession and alternation of the symptoms, their strange and varied combination, and their aggravation and alleviation by causes which exert no influence under other circumstances, must one and all awaken suspicion as to the real nature of the mischief.

The variableness of the symptoms is the point which, according to my experience, is most deserving of attention. There are few, if any, of the general symptoms of thoracic aneurism which may not be produced by the pressure of any diseased mass within the thorax; but, when they are attributable to aneurismal pressure, they are generally distinguishable by the feature just alluded to. A diseased mass, whether malignant, scrofulous, or of whatever nature, having broad and firm attachments in the chest, arising, that is, from a broad extended surface, as such masses do almost invariably, would not be influenced by change of posture sufficiently to effect any material diminution in the amount of pressure, whether on the spine, the trachea, the œsophagus, the bronchi, or other parts; but an aneurismal tumour, arising, as it does, from a narrow base, and attached only to a vessel which admits of considerable motion, will have its position altered considerably by gravity alone, if the patient leans sufficiently forward; and thus the spine, the œsophagus, the trachea, and the larger bronchi may be to some degree, relieved from pressure. Accordingly, on several occasions, I have heard patients suffering from aneurismal tumours declare that, although they could eat, drink, and breathe without much difficulty in one position, yet that their pain was immediately aggravated, the dysphagia increased, or a severe paroxysm of dyspnœa induced whenever they attempted to assume another.

But let me not be misunderstood. I do not wish to imply that an aneurism cannot exist within the thorax without producing evident symptoms of its existence, nor even that when it does give rise to general symptoms, they may not prove insufficient grounds on which to

base a certain diagnosis. Unfortunately, too many cases arise in practice which show that our means of diagnosis of thoracic aneurism are very uncertain, even when we call the stethoscope in aid of the general symptoms of the disease. But I am convinced that the real nature of the disorder is sometimes overlooked, by reason of a too implicit reliance on its physical signs, and a too ready neglect of its general symptoms. In many cases recorded as symptoms of aneurism without corresponding thoracic symptoms, certain facts incidentally mentioned prove that symptoms existed which should have served to awaken suspicion. And although there are many exceptions to this rule, yet experience shows that in the majority of cases phenomena are present sufficiently marked to direct attention to the seat and nature of the mischief; and this, after all, is the practical question. It is not whether in some few instances the obscurity of a case may not mislead even the most wary, but whether, by close attention to the history and progress of the symptoms, we may not usually obtain glimpses of light sufficient to enable us, in some measure, to penetrate the surrounding darkness. If our suspicions are but excited, they will cause us to speak cautiously, and prevent our committing the inexcusable blunder of treating the symptoms lightly and as of little consequence.

There cannot be a doubt that undue importance is often attached to the stethoscopic signs of thoracic aneurism, and that the inexperienced practitioner is apt to be thereby led into grievous error. Not only are stethoscopic signs absent in many doubtful and obscure cases, but instances are occasionally met with in which they occur independently of the lesion of which they are considered to be the characteristic exponents. This fact may be illustrated by a case which fell under my observation in the year 1847:—The patient a poor woman, aged forty-eight, was complaining of dropsy, dyspnœa, palpitation, and sickness. She had long suffered from dyspnœa and palpitation, and since the previous March, when increasing oppression at the chest and palpitation prevented her taking exercise, she had suffered from œdema of the lower extremities. The only other symptoms she complained of were of a dyspeptic character, with constipation, flatus, pyrosis, and vomiting. She had never had rheumatism, nor had she ever received a blow or injury on the chest.

When first seen she was unhealthy in appearance, but not much emaciated. There was frequent cough, with thick, muco-purulent expectoration, and excessive dyspnœa, indicated by the quickness of the respiration and lividity of the lips. Her legs were slightly anasarcous. She was complaining of pain and pulsation, referred to the upper part

of the sternum, and to a spot just to the right of the sternum, between the second and third ribs. Distinct pulsation could be felt in that situation, and there was an imperfectly defined tumour. There was extended dulness on percussion in the præcordial region, reaching almost up to the right clavicle; the heart's action was tumultuous, and rather irregular; its impulse was increased, and its apex was pulsating between the sixth and seventh left ribs. Its sounds were dull and distant, and at the apex were unattended by appreciable murmur. From about on a level with the lower border of the fourth rib upwards to the clavicle, and for nearly three inches to the right of the sternum, was heard a loud, rough murmur, synchronous with the pulsation felt between the second and third ribs, as also with the systole of the heart. It manifestly accompanied the first sound of the heart, but was prolonged over, and much obscured the second sound. It was heard loudest on the sternum, at about the level of the upper border of the second sterno-costal articulation, but it was exceedingly loud over the whole of the space already alluded to, and could be heard distinctly on the back. There was no perceptible difference between the pulse at either wrist, but on both sides it was small, weak, and irregular. The superficial veins on the right side of the chest were turgid.

Taking the murmur in connection with the dulness on percussion, the distinct, and to some extent circumscribed pulsation between the second and third ribs on the right side of the sternum, and the obviously obstructed cardiac circulation, all who saw her inferred the presence of an aneurismal tumour. The physical signs were implicitly relied on, to the exclusion of the general symptoms; or rather, the general symptoms which were present were not allowed their due weight in determining the nature of the existing mischief. The dyspnœa was not characteristic of aneurism; there had never been dysphagia, nor pain in the back, nor alteration of the voice, nor a harsh ringing character with the cough, nor hæmoptysis, nor any of the other general symptoms of aneurism; but, in spite of the dyspepsia, pyrosis, and vomiting, the physical signs were deemed sufficient to determine the aneurismal nature of the disease.

The patient lingered on for about ten weeks; the dyspnœa, palpitation, and vomiting continued, the anasarca increased, and she sank, after remaining comatose about three or four hours.

The *post-mortem* investigation revealed the uncertainty of physical signs when viewed independently of general symptoms. In the anterior

mediastinum was a mass of malignant disease, reaching from about the level of the fourth rib nearly up to the clavicle, and extending considerably to the right of the sternum. This mass was pressing in some measure on the heart itself, and also on the large vessels; and while this pressure produced alteration in the form of the aorta, which, roughened by abundant atheromatous deposits, gave rise to the murmur so distinctly heard, pulsation was communicated to the malignant mass, and thus was felt to the right of the sternum. The sickness and the dyspeptic symptoms had been produced by malignant disease of the stomach, affecting chiefly the cardiac extremity, and a small, malignant mass was found in the smaller lobe of the liver.

It may perhaps be suggested that a careful physical examination would have sufficed to establish the true nature of the mischief, inasmuch as the pulsation of an aneurism conveys an impression of expansion, whereas that communicated by a cancerous tumour does not. This holds good in most instances, but not universally. When the aneurismal pouch is filled with thick layers of firm fibrin, the pulsation is not accompanied by expansion. On the other hand, some malignant osteosarcomatous tumours are known to give rise to expansion as well as pulsation. The unhealthy appearance of the patient, and the wasting which had occurred, when coupled with the dyspeptic symptoms, were more surely indicative of the true state of the case, and might possibly have served to establish a correct diagnosis.

It would be difficult, however, to cite a more convincing case in proof of the necessity of extreme caution in the diagnosis of thoracic aneurism, and more especially of the importance of close attention to the general symptoms.

The three features by which intra-thoracic aneurism may be most commonly distinguished from malignant growths are, the variableness in the amount and direction of the signs of pressure, the absence of distension of the superficial veins, and the freedom from local inflammatory action. The first has been already alluded to, and although it is not a constant feature of aneurism, it is, when present, very distinctive. The second is also very significant; for whereas malignant growths are rarely developed in the chest without invading the lung, and causing pressure on or obstruction of the larger veins, with consequent enlargement of the collateral veins, aneurismal tumours, being soft and yielding, and varying constantly in the force of their pressure, do not usually create that amount of obstruction which renders necessary the establishment of a collateral venous circulation. The third is also very charac-

teristic, inasmuch as malignant growths very generally involve the root of the lung, implicate the nerves which supply it, and thus, by interfering with its nutrition, excite inflammatory action.*

The course of aneurism is extremely variable. The disease commences in an insidious manner, and for a length of time—often for many months—progresses slowly, and without occasioning any notable distress. After a time, however, symptoms are developed—sometimes rather suddenly, as if from a sudden increase in the bulk of the aneurismal tumour, and then the progress of the disease is usually more rapid.

In some rare instances, spontaneous cure is effected by the filling of the sac with firm coagula of fibrin; but almost invariably the disease proves fatal within two or three years from the date of its commencement.

It may destroy life by exhaustion, consequent in part on the inability to take food which results from pressure on the œsophagus, and in part on the local pain, constitutional irritation, and general distress, induced by pressure of the aneurismal sac; or it may prove fatal by inducing pericarditis, pleurisy, pneumonia, or gangrene of the lung;† or by pressing upon the recurrent nerve, and thus giving rise to spasm or paralysis of the glottis. But in the vast majority of cases its fatal issue is referable to rupture of the aneurismal sac. This may take place slowly or suddenly. In most instances, the walls of the sac are gradually destroyed, and some oozing or slight outpouring of blood occurs some time before the fatal event; and this holds good as well when the tumour points externally, as when it bursts internally;‡ but sometimes rupture of the sac takes place suddenly, the quantity of blood poured out is enormous, and in less than three minutes the patient is hurried into eternity.

When the hæmorrhage takes place into the trachea, or into a bronchus, the blood is ejected by coughing; if it takes place into the œsophagus, the blood may either trickle down into the stomach and pass away by the bowels, or may be rejected by vomiting; whilst if it occurs into any of the other organs, it will produce symptoms varying accord-

* See a paper by Dr. Budd in 'Med.-Chir. Trans.,' vol. xlii.

† A remarkable case of pulmonary gangrene, induced by pressure of an aneurism fell under my care in St. George's Hospital in 1859. For particulars, see 'Path. Trans.,' vol. xi, p. 62.

‡ Dr. Stokes (loc. cit., p. 584) refers to a case in which "successive ruptures of the sac took place with intervals of several days," each rush of blood being "attended with the best-marked syncopal coma and convulsions;" and Dr. Thorburn ('Brit. Med. Jour.' for Oct. 18, 1862) relates a case in which two distinct ruptures of the sac took place into the pericardium, and the patient survived the first rupture for a period of twelve days.

ing to the functional peculiarities of those organs, but will not make its appearance externally.

There is scarcely a spot at which, or an organ of the body into which rupture of an aneurismal sac may not take place. The bronchi, the trachea, the pericardium, the œsophagus, the pleural cavities, the lungs, the pulmonary artery, the heart, the mediastina, the venæ cavæ, and the spinal column, have all been known to receive the blood effused from an aneurismal tumour; and when the tumour presents itself on the surface of the chest, it may even burst externally.*

Dr. Stokes has stated† that "the morbid condition which most often accompanies aneurism is that of tubercle;" "that of the general morbid conditions which accompany aneurism, tubercular phthisis is the most common;"‡ and again, that "pulmonary disease is often present."§ Hence, although he has disclaimed the intention of implying that the combination of pulmonary tubercle with thoracic aneurism is of frequent occurrence,‖ an opinion has arisen that there is some sort of connection between the two forms of disease. But in fact no opinion can be more erroneous. Since the year 1841, when the practice was first adopted at St. George's Hospital of preserving a record in the museum of the appearances observed in all *post-mortem* examinations, there have been examined after death forty cases of thoracic aneurism, excluding aneurismal dilatation of the heart. Of these forty cases, six only presented any trace of tubercular disease, and in several of these the tubercle was in a quiescent or retrograde condition. Amongst eighteen other cases of which I have obtained notes, two only presented any trace of tubercle.

In the 'Transactions of the Pathological Society of London,' there have been recorded fifty-seven cases of thoracic aneurism. In two instances only is there any mention of the existence of tubercle. In fourteen of the cases the condition of the lungs is not remarked upon; in the remaining forty-one cases these organs were free from tubercle.

Rokitansky gives us the result of his examination of 108 cases of thoracic aneurism. He states that in five only was any tubercle discovered, and that even in those five the tubercle was in a retrograde condition, and possibly may have existed for many years, and have been deposited before the commencement of aneurism. He further expresses a very strong opinion against the coexistence of the two forms of disease.

* For full statistics on these points, see Dr. Sibson's 'Med. Anat.,' fasc. v; and Dr. Crisp's treatise on 'Diseases of the Blood Vessels.'

† Loc. cit., p. 578. ‡ Loc. cit., p. 599. § Loc. cit., p. 592.

‖ See 'Med. Times and Gazette' for December 26th, 1856, p. 644.

He says: * "The arterial disease upon which spontaneous aneurism depends is, in its more highly developed grades, very rarely associated with tuberculosis. The immunity is, perhaps, based upon the exhaustion of the materials for tubercle, due to the deposition of a solidified blastema out of arterial blood. A more decided immunity is brought about by aneurism in the proximity of the heart, involving the endogenous coagulation of great fibrinous masses, and a consequent hydræmia through defibrination of the blood."

Dr. Crisp, in his treatise on diseases of the blood-vessels, has recorded eight fatal cases of thoracic aneurism, in which a post-mortem examination was obtained, and in all of these the lungs were found free from tubercle. He has also collected 132 cases, and only mentions phthisis in connection with two of them.

Dr. Green, of Dublin, has recorded twelve cases of aneurism of the arch, in four of which some tubercle existed.

Dr. Walshe has reported two cases of tubercle amongst fourteen cases of thoracic aneurism which fell under his observation, but one of these two is spoken of as "a rather doubtful example of tubercle."

Thus, then, it will be admitted that statistics afford a very strong support to the views I have enunciated; for the numbers above quoted show only twenty-one cases of tubercle to 389 cases of thoracic aneurism, or 1 case of tubercle to 18·5 cases of thoracic aneurism; in other words, a proportion of about 5 per cent.; whilst even if Dr. Crisp's 132 collected cases are excluded from the calculation, there will still be 1 case only of tubercle to 12·2 cases of aneurism; in other words, a proportion only of about 8 per cent.

Before quitting the subject of thoracic aneurism, some reference should, perhaps, be made to the means of distinguishing aneurism of the innominata from aneurism of the arch of the aorta. In an essay by Dr. Holland on the diagnosis of these aneurisms,† it is stated that the physical signs and general symptoms of aneurism, including a diminution in the force of the arterial pulsation in the arm, head, and neck, are met with in these cases most frequently on the right side, whereas they occur most frequently on the left in cases of aneurism of the transverse portion of the arch. But I am inclined to believe that an unqualified assertion of this kind conveys more than is really warranted by facts. From the position of the vessel, it is probable that the pulsation would

* 'Path. Anat.,' vol. i, p. 315.

† See 'Dublin Quarterly Journal,' vol. xii.

be first perceived on the right side, and that it would appear earlier, and when it first presents itself, would be seated higher in the chest than if it was caused by aneurism of the arch; and it is also probable that the arteries on the right side of the neck, and in the right upper extremity would pulsate less strongly than those on the left. But it is certain that in some instances pulsation which results from aneurism of the arch makes its appearance in the same position as that occupied by aneurism of the innominata, and that pulsation is sometimes feebler in the arteries on the right side, just as in cases of aneurism of the innominata. In these facts, therefore, there are not sufficient grounds for a positive diagnosis between the two forms of disease. But there is another distinction which was pointed out by Dr. Holland which is of vast importance in a diagnostic point of view, viz., that pressure upon the right subclavian and the right carotid arteries diminishes or arrests the pulsations of an aneurism of the innominata, but exercises no influence over the pulsations of an aortic aneurism. In this fact, and in this alone, we have a means of distinguishing between the two forms of disease.

The treatment of aneurism is a subject on which various opinions have been entertained, and, which, therefore, requires careful consideration. Formerly, under the advice of Valsalva, the patient was placed upon scanty diet, and was subjected to full and repeated venesections, with the view of moderating the action of the heart and arteries. But modern experience and pathological research have not only pointed out the unsoundness of the theory on which this practice was founded, but have demonstrated beyond dispute its practical futility. The patient's health and strength are soon impaired, the quality of the blood is impoverished, and the irritability of the circulating organs increased by repeated bloodletting, whilst, at the same time, his digestive organs are weakened, and his nervous apprehensions so constantly excited as to render sleep almost impossible. Accordingly, the result was found to be, in the majority of cases, that the unfortunate patient soon fell into ill health, and succumbed much more rapidly than if he had not placed himself under medical treatment.

Indeed, in this as in other cases, the dictates of common sense are of greater worth than any theoretical considerations. The objects to be obtained are the relief of pain and the arrest of the disease, and these ends, if attainable at all, must be sought by means which will not seriously impair the general health. Aneurism, though incurable by medical art, yet sometimes undergoes spontaneous cure, when the

health and strength are properly sustained; and even though it should ultimately prove fatal, its existence is compatible with the enjoyment of good health, and by careful management its fatal issue may be greatly protracted. Nothing can be more baneful, and therefore more unjustifiable, than treatment which theoretically aims at the cure of the disease, and practically undermines the health and strength of the patient.

The precise means to be adopted for the patient's relief must be regulated according to the circumstances of the case. If the heart's action is tranquil, and its valvular apparatus sound, there will be little need for medical interference, provided the general health is good, and the various functions of the body are regularly performed. Repose of mind, entire absence from violent exercise, and careful dieting are the only means calculated to benefit the patient, and these should be earnestly enforced. If there be pain sufficient to interfere with sleep or render the patient irritable and exhausted during the day, sedatives and anti-spasmodics must be given, and their constipating effects must be counteracted by gentle aperients; whilst if flatulence is induced by derangement of the stomach or liver, the action of those organs must be promoted by slight alteratives, and by warm carminative and stomatic medicines.

If, as sometimes happens, the sounds of the heart are free from murmur, and the force of the heart's action is less than natural, so that there is reason to suspect the existence of a feeble, fatty heart, the general treatment must be regulated according to the maxims already laid down for the management of that variety of cardiac lesion, and the only special treatment required would be such as had relation to the relief of pain, or of dyspnœa, or some other urgent symptoms.

If again, as is most commonly the case, the action of the heart is increased, and a physical examination informs us of the existence of valvular disease, or of organic changes in the muscular structure of the heart, the treatment must be that already described as adapted to the relief of those particular forms of cardiac disease, with the addition of such remedies as may be specially needed for the subjugation of thoracic pain, or other urgent symptoms.

The treatment of the various forms of cardiac disease have been fully discussed in a former part of this treatise, and it is therefore unnecessary to do more than glance at the remedies specially required in aid of the general treatment there recommended.

Bloodletting, which oftentimes affords remarkable relief to the pain

excited by aneurismal pressure, requires, as already stated, much care and circumspection in its employment. If the patient be of a full habit, the heart hypertrophic, and the circulation excited, venesection to a moderate amount may be had recourse to occasionally. In such cases, not only will the general health sustain no injury from the loss of blood, but the vascular system will be evidently relieved, the thoracic pain alleviated, and the spasm, on which the dyspnœa and other symptoms are in some measure dependent, will also be subdued. The very practice, however, which in these cases proves remedial, has an opposite effect when the patient is of spare habit, or of feeble constitution; and it is also likely to prove prejudicial by disturbing the circulation, if the heart's action is regular and normal. It is a palliative, not a curative measure, and should be employed only when required by the circumstances of the case.

It often happens, however, that local bleeding is useful, even in cases in which general venesection would not be admissible. There is frequently so much of pain and tenderness over an aneurismal sac, especially when it is rapidly extending, that the existence of local congestion, if not of subacute, local inflammation, seems extremely probable. Be this as it may, there cannot be a doubt that the application of three or four leeches over the tumour is often productive of great and immediate relief; and as the loss of blood is not at all commensurate with the relief obtained through its agency, and is not calculated seriously to affect the general health, the remedy may be repeated, from time to time. It oftentimes enables us to relieve our patient when sedatives and other remedies have failed.

Amongst the measures most commonly productive of relief from pain may be mentioned the external use of various sedative lotions,* or of sulphuric æther, or chloroform; or the local application of ice, or better still, of a bladder containing a freezing mixture. When cold to the surface is grateful to the patient, the relief which is thus afforded is sometimes most remarkable. Counter-irritation in the immediate vicinity of the sac, whether by blisters, issues, or stimulating applications, is sometimes of essential service, especially when there is much local tenderness; a strong iodine lotion is also serviceable when there is infiltration of the surrounding parts, arising probably from vascular obstruction.

When the aneurismal sac presents itself externally, and it is found, on examination, that its walls are very thin, and its contents fluid, it is

* The form which I find most useful in these cases is that of the extract—as of opium, belladonna, aconite, or conium—dissolved in glycerine.

sometimes necessary to protect it from the risk of external violence, by applying some thick covering as a shield to the part. In this case, if there is much pain, a piece of linen smeared with the extract of belladonna or conium may be placed on the part, and, over this, may be placed the shield thickly lined with finely carded wool.

Acetate of lead, gallic acid, tannin, and other remedies of a styptic character, have been recommended in these cases, under the idea that they would promote coagulation of fibrin, and thus conduce to the cure of the aneurism.* But the assumption that they exercise any influence of the kind is simply gratuitous, and is unsupported by a single fact. There is no reason why they should induce coagulation in the aorta more than in any of the other large arteries; and if they had a tendency to produce coagulation of blood in the living body, it is obvious that their administration would not be consistent with a patient's safety. Therefore, as they cannot do that which their administration is intended to effect, and as they probably may derange the system, and act prejudicially to the general health, their administration should be avoided.

If there is any exception to the rule that the administration of specifics is useless, or worse than useless in the treatment of thoracic aneurism, it is in reference to digitalis, aconite, and the veratrum viride—medicines which tranquillise the heart's action, and render the circulation slower without deranging the stomach, or otherwise interfering with the general health. It is well known that the deposition of fibrin from the blood is more prone to take place when the circulation is sluggish than when it is active; and it is quite conceivable, that in an aneurismal sac, where a certain degree of stagnation of the blood must necessarily exist, and where, therefore, the tendency to the separation of fibrin is always manifest, a diminution of ten or fifteen pulsations of the heart in a minute might greatly promote the filling up of the sac with fibrinous coagula. In the only two instances I have ever met with of spontaneous cure of thoracic aneurism, every care was taken to tranquillise the action of the heat, and prevent the least quickening of the circulation; and theoretical considerations justify the statement that, without those precautions, the sac, probably, would not have become plugged with fibrin, and the patients would not have recovered.

It has been suggested that when stridor exists, tracheotomy may be resorted to with a fair prospect of affording relief. Dr. Gairdner, of Edinburgh, has advocated the performance of the operation as soon as it is ascertained that the "laryngeal symptoms are the source of the more

* See Walshe, loc. cit., p. 772.

immediate danger." I have no personal experience of the operation in these cases; but I am not indisposed to admit the cogency of Dr. Gairdner's reasoning, and to recommend the adoption of the practice in appropriate cases, provided the temporary prolongation of life is of importance. It is no argument against having recourse to its assistance to assert, that when the stridor has been mistaken for the result of laryngeal disease, and tracheotomy has been performed in consequence, it has failed in attaining the desired object. Of course it must fail when the patient is suffering directly from pressure on the trachea below the seat of the artificial opening; but we know that even when the trachea is not much occluded, pressure on the nerves below will often occasion serious interference with the innervation of the larynx, and induce loss or alteration of the voice, and paroxysms of suffocative dyspnœa. Under these circumstances there is risk of sudden death from disease of the larynx—a risk which would be avoided by an opening in the windpipe; and, therefore, although I would not counsel the indiscriminate employment of tracheotomy in these cases, I see no objection to its performance in appropriate cases, provided the patient be made to understand that it can only afford very temporary relief, and that he has a wish to prolong his earthly existence, even at the risk of additional suffering.

Dr. Stokes* has suggested that there is yet another case in which surgical interference might relieve the patient, viz., when excessive pain results from the pressure of the aneurismal sac against the clavicle. In some of these cases semi-dislocation of the bone takes place spontaneously, and the relief which follows is "extraordinary." He argues, therefore, that when this yielding does not take place spontaneously, "the division of the attachments of the clavicle to the sternum, if it could be effected with safety, would be followed by alleviation of the external pressure." The reasoning is sound, but as the operation is somewhat formidable, and the relief it would afford questionable, it is doubtful whether any surgeon would be found to undertake it, or any patient willing to submit to it. If these practical difficulties can be overcome, I see no theoretical objection to its being tried.

The most important part of the treatment of thoracic aneurism has yet to be discussed. It consists in the management of the daily routine of the patient's life. No remedies can be of the slightest avail if the patient is permitted to take active exercise, to subject himself to mental and emotional excitement, or to indulge, without check, in the pleasures of the table. The diet should be simple, yet generous, as calculated

* Loc. cit., p. 596.

to sustain the patient's strength, and promote the formation of healthy fibrin; but, it should be carefully regulated as to quantity, and every article of food should be avoided which is likely to induce flatulence and distension of the stomach. Wine may be given in moderate quantity when the patient is weak, and the heart feeble; indeed, it may be recommended in all cases in which the circulation is not accelerated by its administration; but malt liquor of all kinds should be eschewed, as having a tendency to excite acidity and derangement of the stomach in persons who are not taking active exercise. The patient should be kept as quiet as possible, all bodily exercise and mental excitement being carefully avoided. The sofa should find him its occupant during the day, and reading, or quiet, unexciting conversation should be his employment. Fresh air, of course, is serviceable, but even the jolting of a carriage may prove mischievous, and if the patient is very anxious to go out, he should do so in a bath chair, with C springs. On no account should he walk up stairs or stoop, or do anything which either directly or indirectly is calculated to accelerate the circulation, or put any strain on the heart or arteries. Purgatives, therefore, should be constantly taken in small quantities, with a view to maintain a free action of the bowels, and prevent the necessity of straining at stool.

Dissecting Aneurisms.

The clinical history of dissecting aneurism, is involved in much uncertainty; indeed, from the very nature of the disease it is obvious that the precise feature of each particular case must vary according to the extent of the mischief, and the portion of the vessel implicated. In all the cases on record, however, there are certain points of resemblance, which deserve special notice. Thus the symptoms commence suddenly, with severe pain in the chest or abdomen, producing cold sweats, vertigo and syncope; and when the patient recovers from the collapse and loss of consciousness with which the attack is ushered in, he usually suffers from nausea or vomiting, followed by more or less febrile disturbance. The pain along the course of the injured vessel is often very severe, and accompanied by violent throbbing; and as the lacerated coats of the vessels offer obstruction to the current of the blood, and produce an eddy, a loud systolic murmur is audible over the seat of mischief and along the course of the circulation. Further, the blood effused between the coats of the artery may constrict the aorta at a point beyond the seat

of laceration, and destroy its elasticity and contractility, and it may even exert sufficient pressure to block up the orifices of the arterial branches which spring from the injured vessels. Thus, in a case recorded by the late Dr. Todd,* in which the arteria innominata and the renal arteries were obstructed, cerebral symptoms and suppression of urine were the natural results; and in a case which occurred a few months since under my care at St. George's Hospital,† in which the arteria innominata, the common carotid, and the left subclavian were pressed upon, not only was there partial obstruction of the pulse, but headache, faintness and vertigo on the slightest exertion were prominent features. This man—a policeman—was attacked whilst on duty on the 11th of December, and died on the following day.

The sudden supervention of the symptoms above described, especially when accompanied by an intense systolic murmur which had not previously existed, might be sufficient to excite suspicion as to the nature of the mischief, and if, further, as in my case in St. George's Hospital, a pulsating tumour were to make its appearance coincidently with the setting up of these symptoms, there could be little room for doubt on the subject. Nevertheless, even under these circumstances, there is little to be done beyond endeavouring to tranquillise the circulation, and at the same time to mitigate the patient's sufferings, by the administration of sedatives.

Varicose Aneurisms of the Thoracic Aorta.

It sometimes happens that a communication takes place between the arch of the aorta and the auricles, the ventricles, the vena cava, or the pulmonary artery. The characteristic features of this form of disease are such as would be expected from disease of the aorta, complicated by sudden rupture of its coats, and an admixture of arterial and venous blood. Pain—sudden pain—generally marks the accession of the disease, and this is accompanied by a sensation as if something had given way at the heart, and is followed by faintness or actual syncope, dyspnœa, and orthopnœa. The patient's chief complaint is of chilliness and debility, a constant fluttering at the chest, and a sense of impending suffocation. The principal physical signs are a "superficial, harsh, and peculiarly intense sawing or blowing sound, accompanied by an equally

* 'Med.-Chir. Trans.,' vol. xxvii, p. .

† See case of Felix Ridout, recorded in the 'Post-mortem and Case-book' for December, 1861, in the museum of St. George's Hospital.

marked purring tremor heard over the varicose orifice, and in the current of the circulation beyond it"—a sound which "is continuous, but is loudest during the systole, less during the diastole, and still less during the interval." * When the communication is with the pulmonary artery, the lungs rapidly become gorged, lividity of the lips ensues, the circulation on the right side of the heart is obstructed, and anasarca of the lower extremities sets in; when the communication is with the vena cava, the face and upper part of the body become livid, the veins of these parts are distended, and œdematous swelling takes place along the course of the internal jugular and subclavian veins.

Mr. Thurnam's paper* contains a large collection of these rare cases, with a full detail of their symptoms, and a few have been recorded by Dr. Hope and others. From these it is obvious that, even with the exercise of the greatest care, the diagnosis of this disease is very uncertain. If in a person known not to have had arterial murmur, an intensely harsh systolic murmur and purring tremor were suddenly to supervene, and their accession were to be marked by the general symptoms above described, the diagnosis of a varicose aneurism might be made without much fear of mistake. But when there has not been an opportunity of examining the patient prior to the setting in of the new train of symptoms, the diagnosis must usually be involved in uncertainty. Nevertheless, the true nature of the lesion may sometimes be made out by the aid of a careful physical examination, as will be evident by reference to an interesting case reported by Dr. Wade in vol. xliv of the 'Med.-Chir. Trans.'

Aneurism of the Abdominal Aorta or its Branches.

Abdominal aneurism is a rare disease, and one of which the diagnosis is often difficult. Occurring usually in early adult life, and more frequently in the male than in the female sex, it is often unaccompanied by cardiac disease, and by any alteration in the structure of the arteries, the single perforation which forms the entrance to the aneurismal sac being the only arterial lesion.

At the commencement of the disease there may be no discoverable physical signs of its existence. If the sac be small, and especially if it arises from the posterior aspect of the vessel, neither the eye nor the hand will be able to detect it; percussion will fail to throw any

* See a valuable paper on this subject by Mr. Thurnam in 'Med.-Chir. Trans.,' vol. xxiii, p. 323.

light on the subject, and, as abdominal aneurisms are often unattended by murmur, auscultation may also fail to afford the slightest information. Nor will the general symptoms prove much more characteristic. Pain—dull, deep-seated, permanent pain—with occasional attacks of agonising, paroxysmal pain in the back and in the abdomen, unaccompanied by symptoms of general ill health, and by any commensurate constitutional disturbance, aggravated by exertion, and relieved by change of posture, especially by lying flat on the belly, constitutes almost the only symptom during the early stage of the malady. And, although the occurrence of persistent pain, of the character of that described, is sufficient to excite suspicion as to the nature of the disease, still, in the absence of physical signs and other general symptoms, it is scarcely sufficient to justify a positive diagnosis.

Even when the disease is more advanced, and the sac is larger, the diagnosis is by no means easy. There may be abdominal tumour, pulsation, and murmur, and, nevertheless, no aneurism. The tumour may be referable to a cancerous growth, or to some other form of abdominal disease; the pulsation may be communicated to the tumour from the aorta, and the murmur may be caused by the pressure exerted on the aorta by the tumour. On the other hand, aneurismal swelling may exist, and there may be neither discoverable pulsation nor murmur. It will be necessary, therefore, to examine in detail the physical signs and general symptoms which will enable us to arrive at a correct diagnosis.

When the sac has attained to any size, it is usually discoverable by the hand, and sometimes forms a prominence visible to the eye. If the patient is of a spare habit of body, and the abdominal walls are thin, the hand may be able to trace the outline of the sac, and so convey a tolerably accurate idea as to its bulk; but if the patient be stout, the abdominal walls thick, or the abdomen much distended with flatulence, it will be impossible to form even an approximate estimate of its size. Gentle pressure with the hand, and gentle percussion, will be utterly useless as a means of diagnosis, and forcible manipulation and heavy percussion would not only be dangerous, but may be unbearable in consequence of the general tenderness of the parts. Add to this the fact that even after the firmest pressure the percussion note will surely be influenced by the intestinal resonance to a degree which would interfere with any accurate definition of the limits of the tumour, and it will be obvious that heavy pressure and forcible percussion are unjustifiable diagnostic expedients.

The aneurismal tumour may or may not pulsate, and may or may not convey the sensation of pulsation to the patient. Further, the amount and force of the pulsation is not proportioned to the size of the tumour; a large tumour may pulsate feebly or not at all; a small tumour may convey an extensively diffused and a powerful impulse. If an impulse is communicated to the abdominal walls, the hand placed over the sac will take cognisance of it, and it will be generally found to be single, and synchronous with the systole of the heart and the diastole of the vessel; but in some rare instances in which the sac has arisen high up in the abdomen, a jogging action has been observed in the tumour, which has conveyed the impression of a second impulse synchronous with the diastole of the heart and the systole of the vessel. When the tumour pulsates, it is usually felt to expand also—laterally, as well as in its antero-posterior diameter—and not unfrequently a thrill will be felt in the tumour, and a murmur heard accompanying the systole of the heart. In some few instances a diastolic thrill is also present.

Murmur, like pulsation, is a symptom which varies greatly. Whatever the size of the aneurismal tumour, it may or may not be accompanied by murmur, and even when murmur exists it may vary in character and position, and may be greatly influenced by the position of the patient. In one case there may be nothing more than a dull, muffled sound; in another there may be a prolonged bellows murmur; in a third a harsh, yet abrupt murmur. In some instances the murmur will be faintly, and in others plainly audible, and occasionally it may be audible even at a distance from the patient. Sometimes it will be most plainly audible on the abdominal walls immediately over the aneurismal tumour; sometimes along the left vertebral groove; sometimes it will be audible when the patient is in a recumbent posture, but inaudible when he is erect. Practically, however, it must always be remembered that abdominal aneurism may exist, and may even prove fatal without giving rise to perceptible swelling, pulsation, or murmur, and that in these cases the general symptoms will be the only guides to a correct diagnosis.

The chief, nay, almost the only important symptom which presents itself under these circumstances has been already alluded to, viz., pain in the region of the aneurismal tumour—dull, deep-seated, persistent pain—aggravated occasionally by attacks of plunging agonising pain in the back, or in the abdomen, following the track of the nerves pressed upon by the tumour, aggravated by exertion, and relieved by change of posture, especially by an erect posture, or by lying flat on the belly.

This pain is usually unaccompanied by fever and by any serious symptoms of ill health, and by any commensurate constitutional disturbance. Sometimes, indeed, when the aneurism becomes diffused, severe irritative fever may be excited, and even when the aneurism does not become diffused, there may sometimes be excessive constipation caused by the pressure of the aneurism, and there may be œdema of the legs, as a result of pressure on the larger veins. But these are rather exceptional occurrences; and in the vast majority of cases there is either an almost entire absence of general symptoms, or severe neuralgic pains in the back or abdomen form the only token of existing mischief.* The absence of peritoneal inflammation, and of ascites, of distension of the superficial veins of the abdomen, and, indeed, of all symptoms indicating deep-seated pressure, is most remarkable in cases of abdominal aneurism, even in cases in which the aneurismal sac is so large as to cause visible pulsation, and to be felt without difficulty by the hand.

It is obvious, then, that ventral aneurism may excite a suspicion of the existence of disease, the nature of which may not easily be recognised: on the other hand, experience has shown that certain forms of disease may simulate abdominal aneurism. It will be desirable, therefore, to point out the marks by which a correct diagnosis may be arrived at.

The diseases for which it is most likely to be mistaken, are lumbago and lumbar or psoas abscess; the diseases by which it may be simulated are various forms of abdominal tumours, such as encephaloid growths or tuberculous masses in the omentum or mesentery, encephaloid enlargement of the left lobe of the liver, malignant growths in the pancreas or pylorus, and simple, yet violent nervous pulsation of the aorta.

It is only in its earlier stages that abdominal aneurism can simulate lumbar or psoas abscess. For a time, however, the symptoms of both forms of disease present many points of resemblance. Not unfrequently the situation of the mischief is the same in both instances; in both there is lumbar pain, and in both there may be more or less loss of power over the leg on the affected side; but, in the former, the neuralgic pain is usually far more severe than in the latter, and there is less tenderness in the loins on pressure, and, usually, far less lameness. In the one, the general health may be good, and, indeed, very generally is so, whereas in the other the patient will generally have experienced

* It should be understood that what is here stated applies as well to aneurisms of the larger branches of the abdominal aorta as to those arising from the aorta itself. At present we do not possess any positive means of distinguishing between the ifferent forms of abdominal aneurism.

rigors and other symptoms of constitutional disturbance, and is usually delicate or unhealthy in appearance, has lost flesh and appetite, and not unfrequently presents traces of tubercles in the lungs. When swelling occurs, all doubt on the subject is necessarily at an end; for whereas in the former case it usually presents itself high up in the belly, probably in the left epigastric region, is irregularly roundin its outline, more or less firm and resistent to the touch, and often pulsates, and yields a murmur on auscultation, in the latter it exhibits an elongated form, passing from above downwards towards the groin, is even on its surface, and soft and fluctuating to the touch, and neither pulsates nor furnishes a murmur. The fact that the pain is confined to one side of the loins, is deep-seated, and not affected by stooping, and is unaccompanied by loaded urine, and other rheumatic or gouty symptoms, ought to enable the physician to distinguish it from lumbago.

Encephaloid tumours are much more perplexing. They are often more or less globular, yet irregular in their outline, soft, or partially fluctuating to the touch, and when they lie over the aorta, or press upon one of its larger branches, they may pulsate forcibly, and may furnish a loud murmur on auscultation. It is not surprising, therefore, that on some few occasions, even experienced physicians should have mistaken these tumours for ventral aneurism, of which, swelling, pulsation, and murmur, are important features; nor can it be doubted that the inexperienced and the hasty are almost invariably deceived as to the nature of the complaint. In some instances, as when the tumour is soft and semi-fluid, fixed in its position, and receives an impulse from the aorta, which causes it not only to pulsate, but to expand, there may be nothing beyond the history of the case and the cachetic appearance of the patient, to guide us to a correct diagnosis. The presence of murmur would not necesarily denote the existence of aneurism, nor would its absence indicate the non-existence of aneurism. If the pulsation is diastolic as well as systolic, this will strongly favour a belief in the existence of aneurism, though it must not be considered an infallible sign, as is proved by a case referred to by Dr. Stokes.*

Perhaps the only distinguishing mark between aneurismal and non-aneurismal pulsating tumours in the abdomen is the cessation or non-cessation of impulse, according as the position of the tumour is changed. An aneurismal tumour will necessarily continue to pulsate, whatever the position of the patient or of the tumour; whereas, a non-aneurismal tumour, which derives its pulsation only from an adjacent vessel, will

* Loc. cit., 644.

cease to pulsate if it can be removed from contiguity with that vessel. Hence, in some instances in which malignant, tuberculous, or other abdominal tumours admit of a certain amount of motion, it is sometimes possible, by careful manipulation, or by changing the patient's posture, as by placing him on his hands and knees, to remove the tumour from the vessels sufficiently to prevent the transmission of impulse. The same may be said in respect to the existence of a murmur; but this is of less diagnostic importance, inasmuch as, whether the tumour be aneurismal or otherwise, murmurs may be absent in every conceivable position, and even in cases of aneurismal tumour may sometimes be made to cease or reappear by change of posture.*

Another point, however, of considerable importance, is the position of the tumour. The upper portions of the abdominal aorta are more prone to suffer aneurismal dilatation than the lower portions of the vessel, and as the aorta lies on the left side of the spine, aneurismal swellings almost invariably make their appearance on the left side, and usually high up in the belly. Tubercular and malignant masses are often developed in the lower part of the abdomen, and lie in the centre rather than on the side of the abdominal cavity, and thus the position in which the tumour first presents itself may afford corroborative proof as to its nature.

The diagnosis between abdominal aneurism and the violent nervous or sympathetic pulsation of a healthy aorta will be pointed out in the section on "Aortic Pulsation," and it is needless, therefore, to discuss it here. Suffice it to say, that if due care be exercised in making the examination, and no malignant or other tumour lies between the vessel and the abdominal walls, there ought not to be much difficulty in determining the nature of the complaint.

The duration of abdominal aneurism is a question which must ever remain involved in obscurity, inasmuch as the date of its commencement cannot be ascertained. Its progress, however, like that of thoracic murmur appears to be very uncertain. There are cases on record which have proved fatal within six months from the commencement of the symptoms, and there are others in which the symptoms have endured above three years. There are even cases which prove that the disease may undergo spontaneous cure.†

* See a case reported by Dr. Corrigan in vol. ii of 'Dublin Medical Journal.'

† See St. George's Hospital 'Post-mortem and Case-book' for 1858, p. 91. The case was one of aneurism of the aorta and cœliac axis in a patient of sixty-nine years of age. The preparation is in the museum of the hospital.

Abdominal aneurisms sometimes attain an enormous size; and even when only of moderate dimensions, they frequently lose their original shape, and becoming diffused by the outpouring of blood into the omentum, or into the subperitoneal tissue, they form a large, irregular mass of the abdomen. In this case death occurs gradually from exhaustion and slow irritative fever. More frequently death occurs suddenly, as in cases of thoracic aneurism, by rupture into the surrounding parts, the abdominal cavity, the omentum, the bowels, the pelvis of the kidney, the lungs, the pleura, or the mediastinum. In a few rare instances death has been known to result from exhaustion, without rupture of the sac.

The treatment of abdominal aneurism scarcely calls for any remark, as the same general principles are applicable as were laid down in reference to thoracic aneurism. The only variation which is needed is the free employment of sedatives and antispasmodics, which is rendered necessary by the agonising pain, sometimes endured by the unfortunate victims to this form of disease. Dr. Walshe has suggested the employment of pressure "either on or above or below the sac;" but, it would be impossible, from the position of the parts, to use pressure effectually, and, not improbably, it might occasion mischief.

Contraction of the Aorta.

Contraction of the aorta is a form of disease which is seldom met with, and therefore is a subject of comparatively little interest to the practical physician.

In phthisis, and in all diseases which lead to a diminution in the quantity of blood in circulation, and in all diseases, whether in heart or lungs, which retard the flow of blood to the aorta, the calibre of the vessel gradually diminishes; but this contraction, which results from a tendency in the vessel to accommodate itself to the altered conditions of the system, is not productive of manifest symptoms, and cannot be regarded as a disease.

Morbid contraction usually occurs as the result of faulty development, and its effects become manifest during the closure of the ductus arteriosus, or within a few days after birth; but sometimes, though very rarely, it takes place in after-life as a result of atheromatous or calcareous deposit in the coats of the vessel, or of constriction caused by lymph poured out in pericarditis.

Personally I have had no experience of this form of disease; but in the 'British and Foreign Medico-Chirurgical Review' for April, 1860,

Dr. Peacock has given an able and elaborate analysis of forty cases, recorded by various authors, from which it appears that when the obstruction is not referable to the presence of tumours, coagula, or inflammatory exudation, the contraction is always situated at or near the point of junction of the ductus arteriosus and the aorta, the constriction being referable to contraction and thickening of the internal tissues, or else to the formation of a more or less complete septum. The defect probably originates, as suggested by M. Raynard, in the faulty development of the portions of the bronchial arches, which form the continuation of the aorta from the origin of the left subclavian to beyond the insertion of the ductus arteriosus; and the contraction of some portion of that space is sometimes so great as to prevent the closure of the duct after birth, and to make that passage the channel by which the blood either wholly or in part reaches the descending aorta. Below the seat of contraction the vessel is generally dilated, the dilatation sometimes occurring quite abruptly. Above it is also usually dilated, and the coats thickened, atheromatous, or calcified, and not unfrequently these changes extend to the larger portion of the arch. In ten instances the canal of the vessel was entirely obliterated, the obstruction extending usually from half a line to a quarter, but in one instance to half an inch; in thirty the obstruction was incomplete. In 73·7 per cent. of the cases the patients were males; in 22·3 only females, their ages varying from that of twenty-two days to that of ninety-two years.*

* Dr. Peacock, loc. cit., p. 484, gives the following table indicative of the age and sex of the patients affected with aortic contraction or obliteration:

Age.	Females.	Males.	Age.	Females.	Males.
4	1	0	38	0	1
7	1	0	39	1	0
14	0	2	40	0	2
17	1	0	42	0	1
19	1	0	45	0	1
20	1	0	48	0	2
21	0	1	50	1	1
22	0	2	56	1	0
24	0	1	57	0	2
25	0	2	69	0	1
27	0	2	92	0	1
28	1	0	Young	0	1
29	0	1	Middle-aged	0	1
32	0	1	Not stated	0	1
35	0	1			
37	1	0	Total	10	28

Age and sex not stated, 1; child, 22 days.

In the nine cases in which the aorta was completely obliterated, the ages were 22 days, 7 years, 14, 21, 42, 45, 50, and 57 years; and 1 middle-aged, the precise age not stated.

In twelve cases the patients died of diseases which had little connection with the aortic obstruction, in eight or nine death occurred suddenly, and was directly traceable to the condition of the aorta, and in six out of thirty-six cases, in which the cause of death is clearly stated, cardiac asthma and dropsy, with their usual complications, proved the immediate cause of the patient's death. The circulation was carried on by means of anastomosing branches of the subclavian arteries, and the aortic branches below the seat of constriction. Thus, in some instances, there were anastomoses of the posterior scapular branch of the transversalis colli, derived from the thyroid axis, with the posterior branches of the aortic intercostal arteries; or of the superior intercostal artery, derived from the subclavian, with the aortic intercostals; or of the branches of the internal mammary with the intercostals and the ascending epigastric. The valves and endocardium were diseased in more than one fourth of the cases, and the heart, in consequence of the obstruction to the circulation, was dilated and hypertrophied.

The symptoms which have been observed in these cases somewhat resemble those of aneurism, viz., pain, dyspnœa, palpitation and violent throbbing of the carotid and temporal arteries, accompanied by intense systolic murmur and well-marked thrill. It is obvious, therefore, that something more is needed than systolic murmur and arterial thrill to justify the diagnosis of aneurism; and the points on which reliance must be placed are, first, the absence of tumour and of dulness on percussion over the seat of pain and murmur; secondly, the absence of the signs of pressure which are usually produced by aneurismal and other tumours; thirdly, the dilated condition of the anastomosing vessels before referred to; fourthly, the violence of the pulsation observed in the carotids, temporal, subclavian, and the before-mentioned anastomosing arteries, as contrasted with the feeble pulsation of the vessels which supply the lower extremities. A permanent harsh systolic aortic murmur, following the impulse of the heart, and of greater intensity over the upper part of the sternum under the clavicles, and on each side of the neck in front, than over the central organ of circulation, and accompanied by a thrill in the second intercostal space on either side of the sternum, by violent throbbing of the carotid and temporary arteries, by dilatation and pulsation of the smaller anastomosing vessels, and by very feeble or imperceptible pulsation of the vessels supplying the lower extremities, affords just grounds for diagnosing the existence of a constricted or obliterated thoracic aorta;

and if these symptoms be unattended by bulging of the thoracic walls, by dulness on percussion, and by the phenomena indicative of morbid pressure within the chest, which would probably exist if aneurism were present, the diagnosis would be greatly confirmed. Anasarca is seldom observed in these cases, but when the obstruction is the result of disease in the coats of the vessels, and occurs in the abdominal aorta, gangrene of the extremities has been known to follow.

It is difficult to determine how long a person may live in the enjoyment of apparent health whilst suffering from this morbid condition of the arteries, inasmuch as the precise date of the commencement of the mischief cannot be positively ascertained; but it may be pointed out that in one of the cases collected by Dr. Peacock, the patient had attained the age of ninety-two, and that considerable contraction of the aorta has been discovered after death in many instances, in which the patients had reached the middle period of life without presenting any symptom calculated to direct attention to the the organs of circulation.*

Functional Pulsation of the Aorta.

Functional pulsation of the aorta is an affection of considerable practical importance, inasmuch as it is extremely distressing to the patient, and is apt to mislead the unwary practitioner into the belief that the vessel is organically diseased, and that aneurism is the form of mischief he has to encounter.

The occurrence of this affection has been referred to a variety of causes, amongst which may be mentioned hysteria, dyspepsia, and spinal irritation, acute gastritis, and the use of strong green tea and tobacco. But inasmuch as these agencies are frequently in full operation without producing aortic pulsation, and as aortic pulsation is often observed in cases in which there is no evidence of their action, the conclusion seems inevitable that however much any one of these agencies may seem to aggravate or even excite the pulsation, when a tendency to it already exists, some peculiar predisposition to aortic pulsation, connected probably with perverted innervation, must be a condition precedent to its occurrence.

Functional pulsation sometimes affects the thoracic aorta, but much more frequently the abdominal portion of the vessel. When the

* Dr. Peacock's paper above referred to will well repay a careful perusal.

thoracic aorta is affected there may be oppression in the upper part of the chest, with dyspnœa, violent throbbing in the upper sternal region, and visibly increased action of the large arteries in the neck, and yet with all this evidence of morbid action there may be no organic disease of the vessel. The absence of organic mischief, however, can only be determined by a careful inquiry into the history of the case, and by the aid of physical diagnosis. The mode in which the pulsation arose and the special nature of the symptoms which accompany it, coupled with the age, sex, and appearance of the patient, and with the absence of any extension of the dulness on percussion, of arterial thrill and murmur, and of evidence of pressure on the thoracic organs will generally indicate the true nature of the malady, and prevent the experienced physician from falling into error.

When the abdominal aorta is the subject of pulsation there is usually considerable uneasiness and throbbing in the epigastrium, with a feeling of nausea or faintness. Pulsation may not only be perceptible to the patient, but visible to the bystander. The hand, placed along the course of the vessel, perceives a forcible yet abrupt impulse, synchronous, or nearly so, with the systole of the heart; but if the physician is cautious in his examination he will be struck with the extraordinary elongation of the pulsation, and with the absence of circumscribed tumour, of dulness on percussion, of diastolic impulse, of lateral expansion, and of aneurismal thrill and murmur. Tumour, indeed, there may be, and dulness on percussion, and systolic murmur also, if a malignant growth or other mass of diseased structure chances to lie over and compress the throbbing aorta; and when this is the case, and pain, which is a constant attendant on these forms of disease, is present, a positive diagnosis is difficult, if not impossible. Under these circumstances even the lateral expansion of an aneurismal tumour may be simulated by the impulse communicated to the diseased mass; and there may be diastolic pulsation and diastolic murmur also. Even when no tumour is present, murmur may be excited in a very thin person by forcible pressure with the stethoscope. Nevertheless, when no tumour exists, and no disease of the vertebra, of a nature to push forward the artery and excite arterial murmur and thrill, the diagnosis in most cases is not likely to be mistaken by experienced practitioners. The history of the case, the age, sex, and appearance of the patient—for this form of complaint is most common in women—the mode in which the pulsation commenced, the peculiar character of the concomitant symptoms and the

absence of the physical signs already described as accompanying abdominal aneurism can leave little doubt as to the nature of the malady.

This functional pulsation of the aorta is often said to be difficult of removal, and Dr. Walshe even quotes a case to prove that it is "susceptible of complete cure." Judging from my own experience in the matter, I cannot understand how any doubt has ever been entertained on the subject; for amongst the many examples of it which have fallen under my notice at St. George's Hospital and elsewhere, I have never met with one which did not yield readily to appropriate remedies. The treatment, however, to be efficacious, must be directed against the functional derangements which happen to be present in each particular instance; and no single remedy or set of remedies can avail for this purpose. As a general rule, the patients are weakly, nervous, and more or less dyspeptic, and therefore the administration of some of the preparations of iron, combined with quinine or strychnine, valerian, and camphor, diffusible stimulants, and antispasmodics, and aided in their action by the shower-bath or dripping-sheet, or a cold douche to the spine, constitute an essential element of success. But even these remedies will fail in producing the desired effect, if the stomach is deranged or the bowels costive; and in this case the frequent exhibition of the compound sagapenum or galbanum pill, in combination with aloes, podophyllin, belladonna, and nux vomica, is often of essential service. Sometimes, again, an emetic is serviceable under these circumstances, more especially when it is followed by brisk purgation and by the administration of bismuth, soda, magnesia, and hydrocyanic acid, in a light bitter infusion calculated to give tone to the digestive organs. Opiates internally are seldom of much service, and may even prove mischievous, by deranging secretion and producing constipation; but I have seen marked benefit result from the application of an opium or belladonna plaster down the spine and over the seat of pulsation in the abdomen. Digitalis, aconite, the veratrum viride, and other vascular sedatives and medicines, such as hyoscyamus and conium, which have the reputation of allaying nervous excitement, are far less efficacious than might be anticipated, and fall far short of the remedies first referred to in producing a satisfactory result; and bloodletting, whether by venesection or by leeches, should not be had recourse to, unless unquestionable symptoms of gastritis are present.

The diet should be light, yet nutritious; all articles such as tea and coffee, which are apt to occasion nervousness and palpitation, should be

strictly forbidden, and so also should food, of whatever nature, which would be likely to create acidity or flatulence. Malt liquor should be avoided, and should be replaced by wine or brandy. Ice and cold drinks are often very serviceable; moderate exercise, change of air and scene, and constant occupation and amusement, without any strain on mind or body, are useful adjuncts to the treatment.

Diseases of the Pulmonary Artery.

Diseases of the pulmonary artery are rare, and their variety is equalled by the obscurity in which they are at present involved.

Inflammation of the vessel is said to have been observed in various distempered conditions of the blood; but, I am not aware of any recorded example of the disease in which conclusive evidence is adduced of the existence of local inflammation. Cases in which fibrinous coagula have formed in the pulmonary artery have been carefully observed by several excellent pathologists, but many of them have been shown to be unconnected with inflammation of the vessel; and the narratives of those which are said to have been dependent on acute inflammation leave considerable doubt in my mind as to whether the disease was not spontaneous coagulation of the blood, occurring independently of arterial inflammation. Personally, however, I have had no experience in the matter, and those who are interested in the subject will do well to refer to Mr. Paget's papers 'On the Obstructions of the Branches of the Pulmonary Artery',* and to Dr. Norman Chevers' admirable work,† in which all that is known respecting the morbid conditions of the pulmonary artery is carefully recorded.

Dilatation of the pulmonary artery, when of slight extent, is not of unfrequent occurrence in connection with diseases productive of long continued impediment to the circulation through the lungs. In no disease is it seen more strikingly than in emphysema, in which dilatation of the right cavities of the heart is of constant occurrence. The main trunk of the artery and its various branches are all apt to suffer to a greater or less extent; the latter especially being often

* 'Med.-Chir. Trans.,' vols. xxvii and xxviii.

† 'Collection of Facts illustrative of the Morbid Conditions of the Pulmonary Artery,' by Dr. Norman Chevers (London, 1851), pp. 79-98.

almost varicose in appearance, and more or less thickened and opake. Dr. Hope* and Dr. Stokes,† both make mention of a case in which dilatation had proceeded to such an extent as to cause insufficiency of the pulmonary valves. In my own practice an instance has never occurred in which regurgitation has taken place through the pulmonary valves, as the result of simple dilatation;‡ and, although theoretically there is no inherent impossibility in such an occurrence, yet its extreme rarity is unquestionably proved by the almost entire absence of recorded cases. In a case detailed by Dr. Alfred Taylor, in the 'Medical Gazette,' vol. xxxvi, p. 19, the pulmonary artery measured four metres and five lines immediately above the semilunar valves, and yet there was no reason to doubt the efficiency of the valves. Unless regurgitation takes place, there is no means of diagnosing the disease, as the enlargement does not proceed to an extent sufficient to cause dulness on percussion, and is not productive of any characteristic general symptoms.

Aneurism of the pulmonary artery is an exceedingly rare disease, but has been met with well developed. Drs. Fletcher§ and Blakeston‖ have put one case on record; another has been detailed by Skoda, and a few other cases have been collected or referred to by Dr. Norman Chevers.¶ Lividity of the face, dyspnœa, cough, dysphagia headache, and pain in the chest and epigastrium, are the principal symptoms which have been observed in these cases, and pulsation between the second and third left ribs, accompanied by a superficial rough systolic murmur, a purring thrill, and dulness on percussion in the same situation, are the principal physical signs which have been recorded. But the physical signs and general symptoms above enumerated are not of constant occurrence, nor are they distinctive of pulmonary aneurism. Dysphagia, as far as I am aware, has only been observed in a single instance.** Dyspnœa has not always been a constant symptom, and

* Hope on 'Diseases of the Heart.'

† Loc. cit., p. 168.

‡ The only instance of persistent insufficiency of the pulmonary valves which has fallen under my observation resulted from fibrinous vegetation on the valves. It is possible, however, that, shortly before death, a double pulmonary murmur may sometimes arise from the presence of fibrinous clots, extending from the right ventricle into the artery.

§ 'Med.-Chir. Trans.,' vol. xxv.

‖ On 'Certain Diseases of the Chest,' p. 98.

¶ Loc. cit., pp. 118-121.

** A case recorded by Dr. Harlan in his 'Medical and Physical Researches,' and quoted by Dr. Norman Chevers.

lividity, as in Dr. Fletcher's case, has been wholly absent. So also in regard to the physical signs. Even if they were all present they might be produced by a tumour in the anterior mediastinum pressing upon and causing a murmur in the pulmonary artery, and at the same time receiving an impulse from it. Possibly, however, the murmur in this case might not convey the impression of being seated superficially to the same degree as that which accompanies pulmonary aneurism.

Contraction and obstruction of the pulmonary artery are far more frequent, and, therefore, more important lesions than pulmonary aneurisms. They may result from congenital malformation, from endocarditis, from the pressure of aneurismal and other tumours, and from the formation of clots of fibrin in the vessels. In all cases a systolic murmur, superficial in seat, is heard over the base of the right ventricle, and in the course of the pulmonary artery; the complexion is usually livid, or of a cyanotic tinge; the pulse is habitually small and quick, yet regular; there is shortness of breath, which is aggravated in paroxysms, and the patient assumes a horizontal posture either habitually or during the paroxysms,—a fact, the true diagnostic importance of which was first pointed out by Dr. Norman Chevers. In all other forms of disease of the heart or great vessels the patient breathes easier when the shoulders are raised; but in this the dyspnœa results from insufficiency of the supply of blood to the lungs, and to the system generally, and, hence, a recumbent posture affords relief by removing the impediment which gravity would offer to the action of the heart and thus by promoting the supply of blood to the brain.

The treatment in these is precisely that already described as applicable to cases of cyanosis.

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